The Hip and Pelvis in Sports Medicine and Primary Care

Peter H. Seidenberg Jimmy D. Bowen David J. King *Editors*

Second Edition



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ISBN 978-3-319-42786-7 ISBN 978-3-319-42788-1 (eBook) DOI 10.1007/978-3-319-42788-1

Library of Congress Control Number: 2016954321

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The registered company is Springer International Publishing AG

The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface to the First Edition

What a great opportunity it is to participate in the body of information advancing the study of musculoskeletal medicine. As a physician, the readers can attest that didactic presentations of musculoskeletal complaints are at a minimum during undergraduate training. The advancement of individual clinical understanding of this field many times is left to the practitioner. Out of imagination, passion, or frustration, we the musculoskeletal practitioners seek to improve our abilities to provide better clinical diagnostic endeavors. The hip and pelvis is an area in musculoskeletal and sports medicine that provides continued mystery. It is the last great bastion of the unknown. Our hope in bringing together many excellent clinician authors is to provide the basis for improved approach to the patient and athlete who have complaints involving the hip and pelvis. Each chapter begins with a clinical case which is probably similar to the patients you see in your practices. Each chapter provides an approach to the diagnosis of hip and pelvis pain and dysfunction that hopefully is easily applicable to your daily activities as a practitioner. Most importantly, we hope that the material contained within this book helps you provide improved care, satisfaction, and function for your patient athletes.

Jimmy D. Bowen

Preface to Second Edition

It is hard to believe 7 years has passed since our first edition of this text. Initially, we wanted to present a comprehensive, clinically based approach to a subject matter that was relatively unknown or misunderstood by all but the most adept musculo-skeletal providers or evaluators. With this new edition, it is recognized that awareness and expertise in this area have greatly increased over the years since the first edition's publication. Much-needed research, education, evaluators and ultimately the suffering athletic patients. It could be argued that we are still scratching the surface and/or approaching the end of the beginning in the understanding of the hip and pelvis in sports and primary care.

Part of this recognition necessitated the addition of an accomplished orthopedic provider specializing in this area as an additional editor. We are very fortunate to have Dr. David King providing his guidance and expertise in the new edition.

The goal of this edition was not to change the unique format of the presentation within the text, but to bring the information up to date and make it more informative within the sphere of continually advancing medical enterprise. We hope that readers will find this helpful as they continue to improve the care and function for their clients, patients, and athletes.

State College, PA, USA Cape Giardeau, MO, USA St. Louis, MO, USA Peter H. Seidenberg Jimmy D. Bowen David J. King

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Chapter 1 Epidemiology of Hip and Pelvis Injury

Brandon D. Larkin

Clinical Pearls

- Injuries to the hip and pelvis are common among both athletes and the general population.
- The incidence and etiology of hip and pelvis injury vary depending on patients' age, gender, anatomy, injury history, and the sport in which they participate.
- Hip and pelvis injury and pain are most common in adolescents and older adults.
- Field-based explosive and contact sports carry the highest risk of hip and pelvis injury.
- Women are twice as likely to suffer from hip pain as men.
- A history of previous injury is the single most important risk factor in injury of the hip and pelvis, followed by age and hip muscle weakness.

1.1 Case Presentation

1.1.1 Chief Complaint and History

A 17-year-old female high school basketball player presents with pain in the lateral aspect of the right hip that radiates down the lateral thigh. She reports a painful "snapping" sensation as she runs down the court. Initially, she noted this pain only while running during practices and games, but it has recently begun to bother her during normal ambulation.

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P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_1

1.1.2 Physical Examination

Examination of the right hip reveals no obvious deformity. There is tenderness to palpation of the greater trochanter. Range of motion of the hip is full in flexion, extension, abduction, adduction, internal rotation, and external rotation. While lying on the left side, passive internal and external rotation of the right hip reproduces symptoms. There is a positive Trendelenberg test bilaterally.

1.2 Introduction

Hip and pelvis injuries are typically not the most common etiology for pain in the lower extremity in athletes nor in the general population. However, many of these conditions carry significant associated morbidity that makes them important in the scope of musculoskeletal care. The diagnosis is often challenging, as hip and pelvis pain is often secondary to numerous pathologic processes. Twenty-seven to ninety percent of patients presenting with groin pain are eventually found to have more than one associated injury [1]. In children and adolescents, those with hip pain have a higher prevalence of pain in the lower back and lower extremity joints, further clouding the diagnosis [2]. Additionally, in patients presenting with hip pathology, the hip is not initially recognized as the source of pain in 60% of all cases [3]. An individual's predisposition to injury and the type of injury sustained vary greatly on the basis of age and type of recreational activity.

Hip pain is often caused by sports-related injury. Ten to twenty-four percent of injuries sustained during athletics or recreational activities in children are hip related [4], and 5–6% of adult sports injuries originate in the hip and pelvis [3, 5]. Pain may result from either acute injury or chronic pathology due to excessive or repetitive activity that places significant demand on the hip and pelvis. The hip bears a tremendous burden during typical weight-bearing activities of daily living. Hip loading is further increased by up to 5–8% during exercise, leading to elevated risk of injury [6]. As a significant element of the body's core musculature, the pelvis also provides an important biomechanical foundation for the lower extremities and is often a hidden contributor to pain in more distal joints.

This chapter will consider the incidence of hip and pelvis pain and injury in the general population as well as in selected subsets. It will also discuss factors that have been shown to increase the risk of injury to this region, including both anatomic features and characteristics of specific sport participation.

1.3 Age

The age of the patient is the single most important factor in determining the etiology of hip and pelvis pain. In very young children, there is rarely a significant acute injury, but several common orthopedic entities involving this region may initially present with exercise-associated pain. As a child grows, skeletal development occurs in a predictable pattern with the appearance of apophyses and epiphyses and their eventual fusion. During growth, these are areas of relative weakness, and avulsion injuries to the developing apophyses are more common than those involving the musculotendinous unit. During adolescence, ossification continues, but the immature skeleton remains more prone to injury as the high physical demands of sports participation exceed the capacity of the musculoskeletal system. Additionally, rapid increases in muscular power related to hormonal changes accentuate the mismatch between muscular and physeal strength.

In children and adolescents, the most common disorder that causes hip pain is transient synovitis. In addition, Legg–Calve–Perthes disease has been shown to have an incidence of 1.5–5 per 10,000 children of ages 2–12 years. Slipped capital femoral epiphysis, with an incidence of 0.8–2.2 per 10,000, is also an oft-encountered etiology for hip pain that usually presents in the early adolescent period. Developmental hip dysplasia, noted in 1.5–20 cases per 1000 births in developed countries, depending on the diagnostic modality used and timing of the evaluation, may lead to hip pain later in life [2]. Each entity should be considered not only in the investigation of hip pain in the limping child, but also in complaints of knee pain in this population. Each is discussed further in this text. (Please see Chap. 7–Hip and Pelvis Injuries in Childhood and Adolescence.)

The epidemiologic data regarding incidence of hip and pelvis injury in children have been studied at length, often in association with investigation of injury incidence at other anatomical sites. Data have been further divided into acute and chronic injury, with acute injury occurring much more commonly in this population. In retrospective studies, injuries to hip and thigh in children encompassed 17-25% of all acute, but only 2.2–4.8% of chronic injuries [7]. Sports injuries to the hip and groin have been noted in 5–9% of high school athletes [1, 5].

Investigation involving primary school through high school-aged individuals in the general population has found an incidence of hip pain in 6.4% [2]. This can be further divided into 4% in the primary school-aged population, compared with 7.8% in the high school group. These data portend a higher risk in the older child of suffering from hip pain. Interestingly, in the same study, 2.5% of the subjects were found to have clinical evidence of hip pathology on examination, the most commonly noted findings being pelvic obliquity, limb length discrepancy, and snapping hip. In only 0.6% of those who reported hip pain was any pathology noted by a physician on physical examination. This may suggest that objectively dysfunctional hips are relatively common in the school-aged and adolescent population, but that these pathologic features do not typically result in pain. One may further conclude that most hip pain in this population is functional, as examination findings are typically lacking in those who do report pain.

Among adults, the spectrum of hip and pelvis injury evolves. As these patients age, the risk of pain from hip osteoarthritis increases substantially. The prevalence of hip and pelvis pain in adults from all etiologies ranges from 2.8 % to 22.4 %, and reports of pain tend to increase with age [2]. In the athletic population, increased age is a risk factor for players of field-based sports in sustaining a groin or hip injury, likely due to decreased elasticity of collagen tissue in older individuals [8, 9].

Over the age of 60, fully 14.3% of adults report significant activity-limiting hip pain [10]. This has an effect beyond the bothersome joint, as those who suffer from hip pain have poorer self-rated overall health scores, as well as increased knee and back pain and reduced muscle power. Climbing/descending stairs and walking tend to elicit the most severe pain [11]. Morning stiffness is also common in the older population with hip pain, affecting 30% of those reporting hip pain [11].

1.4 Sport

Participation in athletic activity of any kind has been shown to increase the risk of hip and pelvis injury, as well as the eventual development of hip osteoarthritis [12]. Men with high long term exposure to sports had a relative risk of developing hip osteoarthritis of 4.5 when compared to those with lower exposure [12]. In those with exposure to high physical loads from both sports and occupation, the relative risk increased to 8.5 for the development of hip osteoarthritis when compared to those with low physical loads in both activities [12].

Overall, hip and groin injuries are more prevalent in athletes participating in explosive or contact sports [7]. Such injuries are seen in a wide variety of sports, including those that feature cutting activities and quick accelerations and decelerations, such as football and soccer, those with repetitive rotational activities, such as golf and martial arts, as well as dancing, running, and skating [1, 6].

By far, dancers possess the highest incidence of hip and pelvis injury among athletes. Ballet dancers are at particularly high risk, as most studies note that the hip is implicated in between 7% and 14.2% of all injuries in this population [13]. Often, these athletes substitute proper technique with exaggerated external rotation of the lower extremity, placing further stress on the hip joint and pelvis.

Runners and soccer players are also at higher risk than other athletes. The incidence of hip and groin injury in these participants has been found to be 2–11% and 5.4–13%, respectively, of all reported injuries [4]. The most common injuries that involve these sites in runners are adductor strains and iliac apophysitis [14]. The injuries to the groin in soccer athletes fall on a spectrum, and may range from mild adductor and hip flexor strains to the often debilitating "sports hernia." Adductor and iliopsoas-related injuries are the most common among professional soccer players, representing almost three quarters of all cases of hip and groin injury [15]. More than half of the injuries to the hip and groin in this population classify as moderate or severe, resulting in a mean absence per injury of 15 days [15].

Seven percent of all injuries to participants in high school football involve the hip and thigh, compared to 20% involving the knee and 18% involving the ankle [16]. Injuries such as hip pointers and thigh contusions are common in this population. Track and field, rugby, martial arts, and racket sports have been implicated as being hazardous to the hip joint itself, specifically for the later development of hip osteoarthritis [3, 12].

1.5 Gender

Injuries of the hip and pelvis are more commonly suffered by women in direct comparisons with men, regardless of age or sport. Most studies note incidences of hip pain in women that are twice that in men. In a study of primary and high schoolaged children, 8.2% of all girls reported hip pain, compared with 4.4% of the boys [2]. In the adult population, the risk of hip pain in women is more than double that in men [10, 17].

In a comparison of injuries in high school basketball athletes, injuries to the hip and thigh ranked third in female students compared with fourth in male students. Incidences of ankle and knee injuries were much more common in both groups, with facial injuries also more common than hip and thigh injuries in boys [18]. Isolated injuries of the pelvis were noted in less than 1% of both genders.

In regard to injuries specific to the groin, the converse is true. There is moderate evidence that men have a higher relative risk of groin injury (2.45) than do women who play the same sport [19].

Specific hip joint pathologies more likely to cause pain differ between genders. In chronic hip and groin pain, men demonstrate a higher percentage of cases of femoroacetabular impingement in comparison with women, who are affected more commonly by tears of the labrum [20].

The etiology of the increased incidence of hip pain in women is likely because of both anatomic and functional factors. The anatomic differences of the lower extremities in women are well described in the literature. Regarding the hip, larger femoral anteversion may predispose women to hip pain. Furthermore, during running, female subjects have a higher degree of hip abduction, hip internal rotation, and knee abduction compared to men [2, 21]. This increased motion is likely to at least partly contribute to higher injury statistics in this region. In addition, acquired anatomic laxity secondary to hormonal changes in pregnancy may contribute to increased incidence of hip and pelvis complaints in women.

1.6 Anatomic Features

Multiple anatomic structures in and around the hip joint and pelvis are known to cause pain, often with significant overlap. Thus, chronic hip and groin pain poses a difficult diagnostic challenge. Recent consensus has settled on classifying entities for hip and groin pain into one of five areas: adductor-related pain, iliopsoas-related pain, inguinal-related pain, pubic-related pain, and hip joint-related pain [22]. Other musculoskeletal causes such as hernias or nerve entrapments are also mentioned.

In a young, active, adult population, hip joint pathology is the most common source of pain, affecting 56% of active individuals in one large-scale study [20]. Further analysis implicates femoroacetabular impingement as the most common

pathology of the joint, affecting 40% of individuals, while labral tears (33%) and osteoarthritis (24%) are also often implicated [20].

While hip joint pathology is more likely to affect individuals with pain in isolation, adductor-related pain or pubic bone stress injury more often occurs in concert with other etiologies [20].

Weakness of hip musculature confers increased risk of overuse injuries of the lower extremity in runners. In a comparison of injured runners to non-injured runners, the injured group showed significant side-to-side differences in strength at the hip [23]. The abductor and hip flexor musculature of the injured leg was significantly weaker, and the adductor musculature was significantly stronger than the muscles of the unaffected side. Additionally, weak adductor muscles are implicated as a risk factor for hip and groin injury specifically, with four times greater risk than those with normal strength [22, 24]. These results suggest that focused strengthening of weak hip muscles may help prevent acute and overuse injury or facilitate injury recovery.

Reinjuries to previously affected anatomic structures about the hip and groin are common. The most prominent risk factor for hip and groin injury identified for athletes in field-based sports is a history of previous injury [22]. Male soccer players with history of injury are at seven times greater risk of sustaining a new groin injury than non-injured players [25]. In a large study of European professional soccer players, fully 15% of injuries to this region represented reinjury, emphasizing the importance of complete rehabilitation and healing of primary injuries to a successful return to sport. [20] In comparison with the primary injury, a reinjury typically results in a longer period of time lost from sport.

1.7 Case Report: Conclusion

1.7.1 Assessment and Plan

The athlete is diagnosed with external snapping hip syndrome, characterized by subluxation of the iliotibial band over the greater trochanter of the femur. Additionally, she exhibits signs of core weakness, which partly contributes to her symptoms. A physical therapy regimen addressing core strengthening and stabilization and iliotibial band flexibility is instituted. Corticosteroid injection into the greater trochanteric bursa can be considered if relief is not obtained through therapy alone.

1.8 Summary

As this chapter has discussed, injury to the hip and pelvis is prevalent in all populations, from the very young to the elderly, among athletes in numerous sports, and in both men and women. Differences in anatomy of the hip and pelvis also factor into injury risk and prevalence. A history of previous injury is the single most important risk factor in injury of the hip and pelvis, followed by age and the presence of hip muscle weakness. Field-based explosive and contact sports also confer higher risk of injury. In the chapters that follow, specific entities affecting this often diagnostically challenging anatomic location will be further explored, including appropriate workup and management strategies that are useful to not only the sports medicine physician but the primary care physician as well.

References

- 1. Morelli V, Weaver V. Groin injuries and groin pain in athletes: part 1. Prim Care. 2005;32(1):163-83.
- 2. Spahn G, Schiele R, Langlotz A, et al. Hip pain in adolescents: results of a cross-sectional study in German pupils and a review of the literature. Acta Paediatr. 2005;94(5):568–73.
- Braly BA, Beall DP, Martin HD. Clinical examination of the athletic hip. Clin Sports Med. 2006;25(2):199–210. vii.
- 4. Boyd KT, Peirce NS, Batt ME. Common hip injuries in sport. Sports Med. 1997;24(4):273-88.
- DeAngelis NA, Busconi BD. Assessment and differential diagnosis of the painful hip. Clin Orthop Relat Res. 2003;406:11–8.
- 6. Bharam S, Philippon MJ. Hip injuries. Clin Sports Med. 2006;25(2):xv-xvi.
- 7. Watkins J, Peabody P. Sports injuries in children and adolescents treated at a sports injury clinic. J Sports Med Phys Fitness. 1996;36(1):43–8.
- Ryan J, DeBurca N, McCreesh K. Risk factors for groin/hip injuries in field-based sports: a systematic review. Br J Sports Med. 2014;48:1089–96.
- Mays PK, McAnulty RJ, Campa JS, et al. Age-related changes in collagen synthesis and degradation in rat tissues: importance of degradation of newly synthesized collaten in regulating collagen production. Biochem J. 1991;276:307–13.
- Christmas C, Crespo CJ, Franckowiak SC, et al. How common is hip pain among older adults? Results from the Third National Health and Nutrition Examination Survey. J Fam Pract. 2002;51(4):345–8.
- Cecchi F, Mannoni A, Molino-Lova R, et al. Epidemiology of hip and knee pain in a community based sample of Italian persons aged 65 and older. Osteoarthritis Cartilage. 2008;16(9):1039–46.
- 12. Vingard E, Alfredsson L, Goldie I, et al. Sports and osteoarthrosis of the hip. An epidemiologic study. Am J Sports Med. 1993;21(2):195–200.
- 13. Reid DC. Prevention of hip and knee injuries in ballet dancers. Sports Med. 1988;6(5):295-307.
- Ballas M, Tytko J, Cookson D. Common overuse running injuries: diagnosis and management. Am Fam Physician. 1997;55(7):2473–84.
- Werner J, Hagglund M, Walden M, et al. UEFA injury study: a prospective study of hip and groin injuries in professional football over seven consecutive seasons. Br J Sports Med. 2009;43:1036–40.
- DeLee JC, Farney WC. Incidence of injury in Texas high school football. Am J Sports Med. 1992;20(5):575–80.
- 17. Tuchsen F, Hannerz H, Burr H, et al. Risk factors predicting hip pain in a 5-year prospective cohort study. Scand J Work Environ Health. 2003;29(1):35–9.
- Messina DF, Farney WC, DeLee JC. The incidence of injury in Texas high school basketball. A prospective study among male and female athletes. Am J Sports Med. 1999;27(3):294–9.
- Orchard JW. Men at higher risk of groin injuries in elite team sports: a systematic review. Br J Sports Med. 2015;49:798–802.
- Rankin AT, Bleakley CM, Cullen M. Hip joint pathology as a leading cause of groin pain in the sporting population: a 6-year review of 894 cases. Am J Sports Med. 2015;43(7):1698–703.

- Ferber R, Davis IM, Williams 3rd DS. Gender differences in lower extremity mechanics during running. Clin Biomech. 2003;18(4):350–7.
- 22. Weir A, Brukner P, Delahunt E, et al. Doha agreement meeting on terminology and definitions in groin pain in athletes. Br J Sports Med. 2015;49:768–74.
- Niemuth PE, Johnson RJ, Myers MJ, et al. Hip muscle weakness and overuse injuries in recreational runners. Clin J Sport Med. 2005;15:14–21.
- 24. Engebretsen AH, Myklebust G, Holme I, et al. Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med. 2010;38:2051–7.
- 25. Arnason A, Sigurdsson SB, Gudmundsson A, et al. Risk factors for injuries in football. Am J Sports Med. 2004;32 Suppl 1:5S–16S.

Chapter 2 Physical Examination of the Hip and Pelvis

Devin P. McFadden and Chad A. Asplund

Clinical Pearls

- Hip and pelvis injuries are common in both sports medicine and primary care practices.
- The hip and pelvis are often viewed as a "black box" because of the complex anatomy and overlapping pain referral patterns.
- Use of a systemic physical examination will assist the clinician in demystifying this region of the body and narrowing the differential diagnosis.
- A thorough lower extremity neurologic examination should be included in the evaluation of the hip and pelvis.
- Special tests are used in concert to gather a more complete picture of the patient's biomechanical deficits.

2.1 Case Presentation

2.1.1 Chief Complaint and History

T.R. is a 20-year-old male NCAA Division I cross-country runner who presents to the sports medicine clinic complaining of "right hip dislocation." He has noticed lateral hip pain over the past 2 months which has been gradually increasing in severity. He

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[©] Springer International Publishing Switzerland 2017 P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_2

states that it feels as though his hip is "going out of place." Initially, the patient noticed the pain only at the end of a run. Now, he complains of a constant dull ache that sharpens during runs. He has no radiation of the pain and no complaints of paresthesias. His running shoes are only 2 months old, and he used his prior pair for only 4 months. A change to his training regimen—the addition of hill workouts—corresponded to the onset of his pain. His first scheduled meet of the season is in 4 weeks.

He denies fevers, chills, night sweats, anorexia, or weight loss. He has no gastrointestinal symptoms or genitourinary symptoms. He denies a history of back pain.

He has no personal history of cancer. He had a left distal tibial stress fracture 2 years ago that healed without complication. There is no family history of cancer or rheumatologic disorders other than osteoarthritis in his grandparents.

T.R. takes ibuprofen prn. He is a non-smoker, uses alcohol socially, and does not use recreational drugs or dietary supplements.

2.1.2 Physical Examination Results

T.R.'s vital signs are within normal limits. He is a well-developed, well-nourished, Caucasian man. He is in no acute distress, alert, and oriented to person, place, and time with normal affect.

The abdomen is non-distended and non-tender, with normal active bowel sounds. There is no abdominal mass.

A back examination reveals no tenderness to palpation of the lumbar spinous or transverse processes. There is no sacroiliac (SI) joint tenderness and no tenderness of the lumbar musculature. The back demonstrates full range of motion in all planes and the Stork test is negative. No pelvic obliquity is present.

On neurological examination, T.R. demonstrates normal gait, mildly positive Trendelenburg on left and grossly positive Trendelenburg on right. The straight leg raise is negative, and hip abduction strength is 4+/5 right and 5/5 left. The remainder of lower extremity strength testing is 5/5 bilaterally, sensation is intact, and lower extremity reflexes are +2 and symmetrical.

Right hip examination reveals no obvious deformity. T.R. is able to reproduce an audible, palpable pop by flexing and abducting the hip. Log roll is negative. Hip range of motion tests' results are as follows:

- Extension: 20° bilaterally
- Flexion: full bilaterally
- · Abduction and adduction: equal and full bilaterally
- · Internal rotation and external rotation: full and equal bilaterally

There is no tenderness at the anterior superior iliac spine (ASIS), anterior inferior iliac spine (AIIS), ischial spine, iliac crest, or lesser trochanter. There is tenderness just posterior to the greater trochanter. Ober's test is positive; Stinchfield, piriformis, and Gaenslen's tests are negative. Leg lengths are equal. The FABER (Flexion, ABduction, External Rotation), modified Thomas, and Ely's tests are all negative. The popliteal angles are equal at 30° bilaterally.

2.2 Introduction

Hip and pelvis injuries are seen commonly in sports medicine and primary care clinics. In fact, certain groups of athletes such as runners, dancers, and soccer players have been identified as being at particularly high risk of injuring these regions [1], likely secondary to the extremes of motion and high-level forces exerted on the hip during participation in these sports. Studies suggest that up to 10-24% of all pediatric patients as well as 5-6% of all adult patients presenting with musculo-skeletal complaints have involvement of the hip [1]. Yet despite the commonplace nature of hip and pelvis injuries, many still view the evaluation of this area as a proverbial "black box." Such a view may result from the combination of complex anatomy and overlapping pain referral patterns that often present as hip complaints. As such, the clinician is required to maintain a broad differential diagnosis of both musculoskeletal and non-musculoskeletal etiologies. The following chapter strives to arm the reader with a systematic method for evaluation of this potentially intimidating area.

2.3 Anatomy

The hip derives its stability from the fact that, unlike the shoulder, it is a true ball-andsocket joint, with the femoral head held snugly in place by the pelvic acetabulum. Yet despite its stable construction, the hip maintains a considerable deal of flexibility in the frontal, sagittal, and transverse planes of motion. Because of its unique design, the partially sperical articulation of the femoral head with the acetabulum is ideally designed to recieve and distribute the forces of daily activity. This fact is critical, as studies have shown that forces ranging from three to five times a patient's bodyweight are transmitted to the hip with simple tasks such as walking or running [2].

The bony joint of the hip is derived from the articulation of the head of the femur and the convergence of the ilium, ischium, and pubis bones of the pelvis to form the acetabulum. These bones, which are commonly referred to as innominate bones, collectively comprise the "hip bone." Like the shoulder, the articular surface of the acetabular cartilage possesses a thickened rim, or labrum, at its periphery which serves to deepen the acetabulum and lend additional support without significantly sacrificing flexibility. When one considers the extra support provided by the three ligaments which surround and enmesh the joint capsule as well as the small ligamentum teres which connects directly to the femoral head, it is easy to see why the hip is one of the most stable joints in the body [3]. The innominate bones also articulate anteriorly at the symphysis pubis, while the posterior articulation of the pelvic girdle is completed by the sacrum and coccyx, providing the connection of the distal appendicular skeleton to the axial skeleton and torso.

The muscular anatomy of the hip is typically divided for simplicity into the medial adductor region, the anterior flexor region, the lateral abductor region, and the posterior extensor region. This breakdown oversimplifies the muscular anatomy

insofar as such a strict classification does not take into account the ability of the hip to internally and externally rotate. However, it serves as a convenient way to conceptualize the cooperative muscle groups that mobilize the hip joint. When evaluating the individual muscles, it is important to remember that the musculature of the "hip" includes origins as proximal as the lumbar spine and insertions as far distal as the tibia. The major muscles from each of the cardinal groupings and their respective functions will be discussed in greater detail later in this chapter.

2.4 History

One of the primary difficulties in definitively diagnosing a patient who presents with hip pain is the sheer number of possible sources of this seemingly simple complaint. Aside from the articulation of the femur and acetabulum (i.e., hip joint proper), potential causes of pain include a variety of local bony and soft tissue sources, several potential peripheral nerve palsies and radiculopathies referring pain from the lumbar spine. In light of the many potential etiologies of hip pain, it is important to consider a broad differential diagnosis before devising a treatment strategy.

Further complicating matters is the significant effect of age on the differential diagnosis of hip pain. Developmental dysplasia of the hip, Legg-Calve-Perthes disease, and slipped capital femoral epiphyses all have their own classical age ranges for presentation in the pediatric population (see Chap. 7) [4]. In addition, a mechanism of injury which likely would cause tendinopathy at the musculotendinous junction in a skeletally mature individual is far more likely to cause an apophysitis or apophyseal avulsion fracture in children or adolescents with open growth plates as their tendons are frequently stronger than the apophyses to which they attach. Fortunately, the incidence of all the previously cited pediatric hip disorders decreases as a patient reaches skeletal maturity [5]. Systemic causes of intra-articular hip pathology, such as transient synovitis and septic arthritis, are also more common in pediatric populations. Such examples illustrate the importance of assessing constitutional symptoms as well as musculoskeletal complaints [6, 7]. Because of these variables, accurate assessment of both the patient's chronologic and physiologic age is an elementary yet essential part of any evaluation of the hip.

Non-musculoskeletal diseases, particularly those involving the genitourinary and gastrointestinal systems (e.g., pelvic inflammatory disease, appendicitis), have also been known to masquerade as vague hip discomfort. Due to complex pain referral patterns differentiation between pain originating from the hip and that originating in the groin can be difficult. Emphasizing this point, the 2014 Doha agreement on terminology and definitions in groin pain in athletes recommends classification of groin pain in three categories: defined clinical entities for groin pain (adductor related, iliopsoas related, inguinal related, and pubic-related), hip related groin pain, and other causes of groin pain. This requires the clinician to maintain an open mind and broad differential for musculoskeletal and medical conditions with any complaint in the region of the hip [8]. A final yet critical point entails the possible diagnosis of cancer, as hematopoietic and metastatic tumors frequently invade the hip region [9], making the presence of a long-standing limp or recent weight loss an integral part of the patient history.

Despite the complex interplay of biomechanical, developmental, and systemic contributions to "hip pain," the responsible healthcare practitioner should find that a detailed, thorough, and systematic history and physical examination can efficiently narrow an initially broad differential diagnosis.

As with any musculoskeletal complaint, the patient presenting with hip pathology should always be questioned regarding the onset, provoking and alleviating factors, quality, radiation, severity, and timing (duration) of symptoms (OPQRST). Characterization of the pain as sharp or dull, constant or intermittent, severe or mild can also be helpful. Other historical points of emphasis should include the presence or absence of neurological signs and symptoms and a clear description of any clicking or snapping of the joint.

One of the primary objectives of the history with any joint complaint should be to determine whether the injury is acute, chronic, or acute-on-chronic in nature. Detailed questions regarding athletic involvement, exercise habits, training regimen and modifications, equipment use, and nutritional practices often yield useful information to help elucidate the mechanism of injury. One must also inquire about any previous or concurrent injuries sustained to the back or lower extremities, as injury to either the ipsilateral or contralateral leg, knee, ankle, or foot can cause a compensatory alteration to gait pattern and potentially contribute to sustained hip pain (see Chap. 3) [10]. Once one determines the nature of the injury, considering the progression or regression of symptoms along with behavioral modifications preceding those changes may help unmask the precise biomechanical "culprit" of the disease process, which is an imperative step in healing the "victim" and preventing recurrence.

The presence of radiating symptoms can be helpful in accurately diagnosing hip pain as well. The hip serves as a conduit through which all the nerves innervating the lower extremity must pass; as such, it is a frequent location of nerve injury. The surface area and skin distribution affected can vary widely with these complaints and should be definitively delineated to ensure an accurate diagnosis. Sciatica presents with its classic nerve impingement syndrome, but smaller nerves such as the ilioinguinal nerve may also be damaged and should also be included in the differential diagnosis. If neurological symptoms are present, the healthcare professional must differentiate between functionally predominant and sensory-predominant symptomatology and pursue an immediate diagnosis more aggressively when functional decline is apparent (SOR-C).

Finally, the value of an accurate past medical history and medication list must never be underestimated, as a history of osteoporosis or recent steroid treatment, for example, can alter the differential diagnosis and treatment plan significantly.

2.5 Physical Examination

The evaluation of hip complaints should begin before the physician has even entered the room. An astute physician can gather vital information simply by observing the patient's affect, posture, and gait pattern as he or she is escorted to an exam room. In doing so, the physician should be able to identify obvious muscular atrophy or weakness, pelvic obliquity, and abnormal scoliotic or lordotic curves resulting in gross postural abnormalities [11]. Knowledge of normal gait biomechanics and frequently encountered compensatory reactions to traditional disturbances is essential for integrating this information into the clinical picture (see Chap. 4). For example, a patient with a Trendelenburg gait most likely has hip abductor weakness, but the cause may also be related to a tight iliotibial band or coxa externa saltans (snapping hip) [10]. Likewise, a hyperlordotic lumbar curve may indicate a compensatory reaction employed to preserve balance in a patient with flexion contractures of one or both hips.

2.5.1 Range of Motion

Range-of-motion testing can be very informative and, therefore, should constitute a distinct part of the standard hip exam. Normal parameters for range of motion have been well defined, giving the practitioner a reliable standard against which to compare collected data (Table 2.1). When performing this portion of the exam, it is important to pay special attention to abduction and internal rotation, as these are the most commonly compromised motions in many pathological conditions involving the hip (SOR-C).

The majority of the range-of-motion testing can easily be performed with the patient supine. One can assess internal and external rotation by having the patient lying with his or her legs slightly separated and passively rolling the entire lower extremity as if performing a log roll. An alternate method involves flexing the patient at the knee and rotating the leg around the vertical axis of the femur (Figs. 2.1 and 2.2). This method may make measurements easier, but it is important to remember that pivoting the ankle in one direction causes the hip to rotate in the opposite plane. For example, moving the ankle laterally, while using this method, causes internal rotation at the hip. Abduction (Fig. 2.3) and adduction (Fig. 2.4) are performed by anchoring the patient's pelvis with one hand while moving one leg at a time through the transverse plane with the other. When the hip begins to rotate

Motion	Flexion	Extension	Abduction	Adduction	Internal rotation	External rotation
Range in degrees	110-120	0–15	30–50	30	30–40	40–60

Table 2.1 Normal parameters for hip range of motion [12]

Fig. 2.1 Internal rotation



Fig. 2.2 External rotation



Fig. 2.3 Abduction



Fig. 2.4 Adduction





Fig. 2.5 Extension

(despite the added support provided by the examiner) the full range of motion in that plane has been reached. Hip flexion should be tested in the supine position by having the patient draw both knees to his or her chest, as flexion at the knee eliminates hamstring tightness as a potential limiting factor for this exam. Extension, on the other hand, is best performed with the patient in the prone position by raising the selected thigh from the exam table (Fig. 2.5).

2.5.2 Palpation

Palpation constitutes another significant portion of the exam. The musculature, tendinous origins and insertions, bony prominences (e.g., the greater trochanter), bony articulations (including the SI joint (Fig. 2.6) and pubic symphysis), bursae, and apophyses all must be palpated to the extent possible. The examiner must be attentive to any snapping or popping throughout the range of motion. While this usually indicates benign tendinous friction over a bony prominence, it can at times indicate an intra-articular lesion or free-floating loose body [13]. This information may be obtained by palpating the portion of the joint being assessed with the free hand while performing the range-of-motion testing as detailed above.

2.5.3 Neurologic Testing

The hip and pelvis channel numerous nerves from the back to the groin and lower extremity. Accordingly, a thorough neurologic exam is essential even when neurological involvement is not suspected. Strength testing of the lower extremity must



Fig. 2.6 SI (sacroiliac) joint palpation

Strength test value	Meaning of the value
1/5	No signs of muscle firing
2/5	Visible twitching or fibrillations of the contracted muscle group without any movement
3/5	Active movement when gravity is eliminated
4/5	Active muscle activity against resistance with decreased strength
5/5	Indicating normal strength

Table 2.2 Strength testing values

include each of the major muscle complexes that mobilize the hip and knee. As described previously, these muscle complexes can be divided into four cardinal muscle groups: the flexors (e.g., iliopsoas and rectus femoris), the extensors (e.g., gluteus maximus and hamstrings), the abductors (e.g., gluteus medius and gluteus minimus), and the adductors (e.g., adductor longus, adductor brevis, adductor magnus, pectineus, and gracilis.) Strength should then be graded on a scale from 1 to 5 (Table 2.2).

After palpating the muscle bellies and tendinous junctions of the individual muscles, the examiner may proceed to test the strength of each muscle grouping. In order to test the flexor group, the examiner places his or her hand over the seated patient's thigh and asks the patient to push upward against his or her hand while offering resistance. Similarly, to test the hip extensors, the patient is placed in a prone position and instructed to raise his or her thigh from the exam table as resistance is applied from behind the knee. Abduction and adduction may be assessed

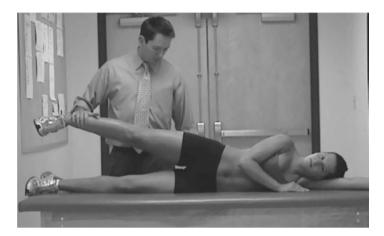


Fig. 2.7 Abduction strength testing

from the supine position with knees extended. The patient is instructed to separate the legs as the examiner offers resistance from the lateral malleoli and then to squeeze the legs together as resistance is applied to the medial malleoli. These latter tests may also be performed with the patient in the lateral decubitus position with the hips neutral. In this scenario, the patient is instructed to abduct the upper thigh to 30° and strength testing proceeds as above for the elevated leg (Fig. 2.7). The authors suggest that this technique may offer a greater degree of sensitivity to subtle deficits of strength (SOR-C).

2.5.4 Special Tests

One may employ a number of special tests to narrow the differential diagnosis after history, range-of-motion testing, neurologic testing, and palpation have been completed. Despite variability in sensitivity and specificity (Tables 2.3, 2.4, 2.5, 2.6, 2.7, and 2.8), as well as significant crossover, such tests can be helpful when employed within the context of the previously obtained information.

Trendelenburg's sign is a test used to determine whether the patient has adequate hip abductor strength, particularly of the gluteus medius. To perform this test the patient is instructed to stand on both feet and slowly raise one foot off of the ground without additional support. If the patient has adequate abductor strength, then the iliac crest of the raised leg should remain parallel with or elevated slightly in relation to the contralateral side (Fig. 2.8). In addition, the patient should maintain an upright posture without significant tilt of the upper trunk, which would indicate a compensatory mechanism to help the patient maintain his or her balance (Fig. 2.9). A positive Trendelenburg sign is defined as either a compensatory tilt of the torso (vide supra) or a drop of the contralateral iliac crest (Fig. 2.10), indicating that the ipsilateral hip

Test/lead author (year published)	Pathology	SN/SP ^a
1. Trendelenburg's Sign	l i i i i i i i i i i i i i i i i i i i	
Bird (2001) [14]	Gluteal tear/tendinopathy	73/77
Woodley (2008) [15]	Gluteal tear/tendinopathy	23/94
Lequesne (2008) [16]	Gluteal tear/tendinopathy	97/96
2. Ely's Test		
Marks (2003) [17]	Flexion contracture (rectus femoris spastisity)— pedatric CP patients only	56–59/64–85

 Table 2.3 Physical exam tests for muscle/tendon pathology

^aSN sensitivity, SP specificity

Test/lead author	Pathology	SN/SP ^a
1. FABER (Jansen or Patrick's test)		
Martin (2008) [18]	Labral tear, FAI, arthritis, dysplasia	60/18
Troelsen (2009) [19]	Labral tear	42/75
Maslowski (2010) [20]	Labral tear, FAI, arthritis, avascular necrosis	81/25
2. Impingement (FADIR)		
Reiman (2015) [21]	Labral tear, FAI, arthritis, chondral defects	94/8
Reiman (2015) [21]	Labral tear, FAI, arthritis, chondral defects	99/7
3. Stinchfield (resisted straight leg ra	aise)	
Maslowski (2010) [20]	Labral tear, FAI, arthritis, avascular necrosis	59/32
4. Scour (quadrant) test		
Maslowski (2010) [20]	Labral tear, FAI, arthritis, avascular necrosis	50/29
5. Thomas test		
McCarthy (1995) [22]	Labral tear, arthritis, loose bodies	89/92

 Table 2.4
 Physical exam tests for intra-articular pathology

^aSN sensitivity, SP specificity

Table 2.5 Physical exam	Test/lead author	Pathology	SN/SP ^a		
tests for osteoarthritis of the hip	1. Trendelenburg's sign				
	Youdas (2010) [23]	Osteoarthrisis	55/70		
	2. FABER (Jansen or Patrick's test)				
	Sutlive (2008) [24]	Osteoarthritis	57/71		
	^a SN sensitivity. SP specificity				

SN sensitivity, SP specificity

Test/lead author	Pathology	SN/SP ^a
1. Fulcrum test		
Johnson (1994) [25]	Femoral shaft fracture	93/75
Kang (2005) [26]	Femoral shaft fracture	88/13
2. Patellar pubic percussion test	· · · · · · · · · · · · · · · · · · ·	
Bache (1984) [27]	Femoral neck fracture	91/82
Adams (1997) [28]	Femoral Fx at the neck or proximal/ trochanteric Fx	94/95
Tiru (2002) [29]	Femoral neck fracture	96/86

 Table 2.6
 Physical exam tests for femoral shaft fracture

^aSN sensitivity, SP specificity

 Table 2.7 Physical exam tests for neurologic disease

Test/lead author	Pathology	SN/SP ^a
1. Straight leg raise		
Vroomen (1999) [30]	Ipsilateral lumbar herniated disk disease	85/52
Vroomen (1999) [30]	Contralateral lumbar herniated disk disease	30/84
2. Piriformis (FAIR) test	,	
Fishman (1982) [31]	Sciatic nerve impingement	88/83
3. Lateral pelvic compression test	,	
Nouraei (2007) [32]	Meralgia parasthetica	95/93.3

^aSN sensitivity, SP specificity

Table 2.8 Physical	exam tests	for pelvic	disorders
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Test/lead author	Pathology	SN/SP ^a
1. Suppine to sit (long sitting tes	t)	
Bemis (1987) [33]	Pelvic dysfunction, pelvic malrotation	17/38
2. Standing flexion test		
Levangie (1999) [34]	SI joint dysfunction	17/79
3. Gillet test		
Dreyfuss (1996) [35]	SI joint dysfunction	43/68
Levangie (1999) [34]	SI joint dysfunction	8/93
4. Gaenslen sign		
Russel (1981) [36]	SI joint dysfunction	21/72
Dreyfus (1996) [35]	SI joint dysfunction	68/35
van der Wurff (2000) [37]	SI joint dysfunction	71/26
5. Compression test		
Russel (1981) [36]	SI joint dysfunction	7/90
Ozgocmen (2008) [38]	Rt sided sacroiliitis	22/83
Ozgocmen (2008) [38]	Lt sided sacroiliitis	27/93

^aSN sensitivity, SP specificity

Fig. 2.8 Negative Trendelenburg sign



abductors are unable to contract with adequate force to maintain a level pelvis. Instability of the pelvis from other etiologies may also create a positive Trendelenburg's sign resulting from increased tensile forces on the bony structures of the hip. Therefore, diagnoses causing pelvic instability, such as Legg–Calve–Perthes disease or acetabular fractures of any etiology, may be considered as alternate causes of a positive test [39].

Ely's test is used to assess the flexibility of the rectus femoris. To perform this test, the patient is instructed to lie in the prone position with legs fully extended. The examiner then passively hyperflexes the knee to the extreme of its range of motion taking care to avoid rotation or extension of the hip joint, and observes the ipsilateral hip for vertical separation from the exam table (Fig. 2.11). If the hip is forced to lift off of the table, then the test is considered positive, suggesting a rectus femoris contracture. Again, the examiner must cautiously avoid any extension or rotation of the hip which can cause false positive results by eliciting pain from other areas [9].

The *Thomas test* [11] and *modified Thomas test* are used to assess hip flexor flexibility, particularly of the iliopsoas muscle. To perform the Thomas test, the patient is placed in a supine position and instructed to flex one leg and pull it to the chest. A flexion contracture would be indicated by passive flexion of the contralateral straight leg lifting off of the exam table (Figs. 2.12 and 2.13). A more informative version, the modified Thomas test, is performed by having the patient sit on the end of the



Fig. 2.9 Compensated Trendelenburg sign

exam table and pull a single leg to his or her chest. The patient is then instructed to lie back on the exam table while maintaining the knee against the chest wall, as the examiner watches carefully to insure that the patient does not fall. Once again, a flexion contracture of the iliopsoas is indicated by the contralateral thigh rising off of the table. In the modified Thomas, however, the patient may also demonstrate a rectus femoris contracture via extension of the contralateral knee from its passively flexed position, making this a higher-yield test [10].

Ober's test is useful for evaluating the iliotibial band, tensor fascia lata, and greater trochanteric bursa. The patient is placed in a lateral decubitus position with hips and knees each flexed to 90°. Initially, the examiner passively abducts and extends the upper leg until the thigh is in line with the torso, followed by passive adduction until the extremity returns to a natural position (Fig. 2.14). A positive test is indicated by a leg maintained in relative abduction, in contrast to a negative test in which the leg may rest on the table without causing significant discomfort [40]. Inflexibility indicated by a positive test suggests excessive tightness of the iliotibial band, whereas focal pain overlying the trochanter points towards a possible trochanteric bursitis.

The FABER test, sometimes referred to as Jansen's test or Patrick's test, was designed to isolate hip joint, SI joint, or iliopsoas pathology. The most commonly





Fig. 2.11 Ely's test

Fig. 2.10 Positive Trendelenburg sign



Fig. 2.12 Negative Thomas test



Fig. 2.13 Positive Thomas test

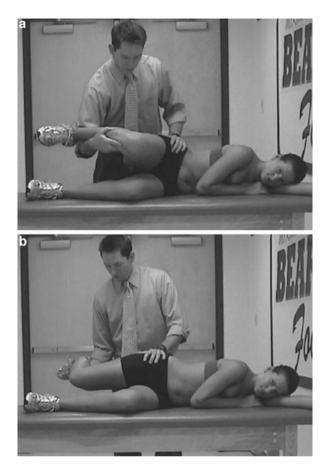


Fig. 2.14 Ober test. (a) Start. (b) End

used name of this test is an acronym for the positioning of the hip during the test (i.e., FABER). The patient lies supine and one leg is placed in a *f*lexed, *ab*ducted, *ex*ternally *r*otated position, as if creating the number 4, with the foot of the leg being tested resting on the contralateral knee (Fig. 2.15). From this position, the examiner places gentle downward traction on the ipsilateral knee. Pain or a decreased range of motion indicates a positive test. A study by Broadhurt and Bond published in 1998 demonstrated a sensitivity of 0.77 and specificity of 1.0 when using the symptom of SI pain during the FABER test to indicate SI dysfunction [13]. The examiner should note, however, that a restricted range of any of the individual planes of motion being tested in the FABER results in decreased specificity of the test, as any individual restriction would be expected to decrease range of motion in this composite motion exam as well, thereby leading to false positive results [41, 42].

The *piriformis* or *FAIR* (*flexion*, *adduction*, *internal rotation*) *test* is performed with the patient in the lateral decubitus position, with the upper leg flexed to 60° and



Fig. 2.15 FABER test

the lower leg maintained in full extension. The examiner places one hand on the patient's shoulder and, with the other hand, exerts mild pressure on the flexed leg at the knee. A positive test is defined as classical "shooting" pain elicited by direct impingement of the sciatic nerve by the tight piriformis muscle. In 1992, Fishman and Zybert showed that when used to demonstrate sciatic nerve impingement, these symptoms have a sensitivity of 0.88 and a specificity of 0.83 in comparison with electrodiagnostic studies as a gold standard [31]. It is important to distinguish this classical description, however, from other sources of pain which may result from the added pressure placed on the hip joint during the exam.

The *impingement* or *FADIR* (*flexion, adduction, internal rotation*) *test* is similar to the *piriformis test* but performed in the supine position. The examiner stands on the side of the leg to be tested with the patient lying supine and passively flexes the knee and hip to 90°. The hip is then passively adducted and internally rotated with over-pressure, closing the space between the acetabulum and femoral head. Pain, locking, or catching in the region of the abutment indicates a positive test. A positive result has proven to be useful for ruling out intra-articular pathology but poor at ruling it in, with sensitivities and specificities of 0.94–0.98 and 0.07–0.08, respectively, in multiple studies [21].

The *Log Roll test* [12] is a simple but useful test for demonstrating acetabular or femoral neck pathology. To perform the test, the practitioner passively internally and externally rotates both fully extended legs of the supine patient. Pain in the anterior

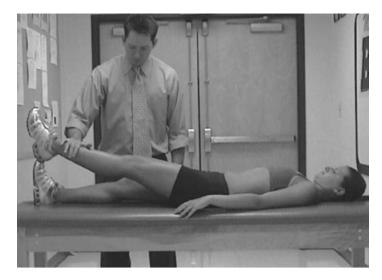


Fig. 2.16 Stinchfield test

hip or groin is considered positive. When significant bony injury is suspected, this test can be used to offer a preliminary assessment of bony integrity in order to guide subsequent exam maneuvers and minimize risk of further injury (SOR-C).

The *Stinchfield test* is performed with the patient in the supine position with the symptomatic hip flexed to 20° and the knee maintained in full extension. A gentle downward pressure is then exerted on the distal end of the elevated leg (Fig. 2.16). Pain in the anterior hip or groin indicates a positive test and may suggest femoral fracture, acetabular injury, or osteoarthritis of the affected hip [43].

The *Scour test* or *Quadrant test* is used for investigation of possible labral pathology. To perform this test the examiner axial loads, adducts, and flexes the hip to its end range of motion with the patient in a supine position. If performed correctly, the ipsilateral knee should point to the patient's contralateral shoulder. From this position the leg is taken in an arc-like motion to the point of full abduction. Any positive exam, defined as pain, apprehension, or catching of the hip during the maneuver, is presumably caused by either labral pathology or a loose body within the hip joint [11]. The authors suggest that this maneuver is analogous in technique to the McMurray test for the knee, lending a useful method for conceptualizing the exam.

The *straight leg raise* test classically has been used in descriptions of patients suffering from lumbar herniated disk disease, but it can also be used to differentiate various types of hip pathology from those of gluteal etiology. To perform the exam, the patient is placed in a supine position, and the examiner passively flexes (raises) one leg at a time while maintaining full extension at the knee. If the patient experiences pain or is too inflexible to perform an adequate exam, then the knee may be slightly flexed and continued flexion of the hip attempted. If the examiner is unable to further flex the hip despite this modification, then a pathology of the buttock such

as ischial bursitis or an abscess, rather than intra-articular hip pathology, is likely. Pain radiating distally in either leg represents a positive test and suggests some form of sciatic nerve irritability, either at the level of the piriformis or possibly at the site of a more proximal lumbar disk herniation. A meta-analysis by Vroomen and Knottnerus in 1999 suggested that the ipsilateral straight leg raise was the most sensitive physical exam maneuver used to rule out a herniated disk, with a pooled sensitivity of 0.85 compared to a specificity of just 0.52 [30]. The contralateral straight leg raise, on the other hand, was the most specific exam technique for identification of a herniated disk, with a specificity of 0.84 in contrast to its poor sensitivity of 0.30 [30]. For this reason, when used, the straight leg raise should always be evaluated bilaterally (SOR-B).

An accurate *leg length assessment* is critical in the evaluation of hip pain, as a significant discrepancy sometimes may represent a masked "culprit" masquerading as secondary pathology. These secondary problems, or "victims," are destined to recur unless the primary etiology is addressed appropriately. While it may be difficult for the practitioner to definitively diagnose an anatomic leg length discrepancy [43], it is equally important to make the diagnosis of a functional leg length discrepancy due to its effects on the athlete's kinetic chain. One proposed method for determining anatomical leg length requires the patient to stand fully erect with his or her feet 6–8 in. apart as the examiner measures the distance from the ASIS to the medial malleolus of each lower extremity [44]. Confounding factors such as the patient shifting weight to alleviate pain and the potential for asymmetrical soft tissue distribution, however, sometimes decrease the reliability of the obtained measurements. In addition, even in cases in which the measurements are reliable, this method offers no information as to where the discrepancy arises, thus limiting its clinical utility.

The *Weber–Barstow maneuver* was subsequently designed to address these limitations. In this technique, the patient is asked to lie supine with both knees and hips flexed to approximately 45°. The patient is then instructed to reset the pelvis by pushing off of the table and gently lowering himself or herself back down (Fig. 2.17).

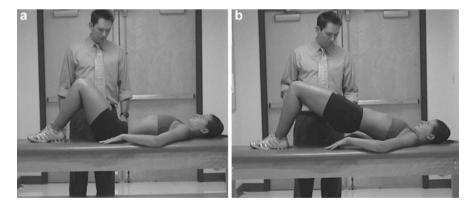


Fig. 2.17 (a) Start for resetting the pelvis for leg length evaluation. (b) Pelvic bridge for resetting pelvis



Fig. 2.18 Measuring position for leg length discrepancy following the Weber–Barstow maneuver

The examiner can then assess the length of the femur and tibia individually by aligning the medial malleoli and examining the profile of the knees from both the front and side. A vertical discrepancy between the level of the knee joints most visible from the front would indicate a discrepancy of the structures distal to the knee (i.e., tibia), while an anterior–posterior discrepancy more apparent from the lateral view indicates that the discrepancy lies proximally (i.e., femur). After making these observations, the examiner then passively extends the knees and uses the medial malleoli as landmarks to indicate whether a discrepancy appears to be present (Fig. 2.18). Similarly, the examiner can compare the position of the patellas and ASISs to evaluate for proper and symmetrical body alignment.

The *prone knee flexion test* can be used as a confirmatory examination when the Weber–Barstow suggests that a discrepancy may exist. It is performed with the knees flexed to 90° and the patient in a prone position. The clinician's thumbs are placed transversely across the soles of the feet distal to the calcaneus bilaterally, and the heights of the thumbs are compared. A discrepancy in thumb position would suggest a tibial length discrepancy. Unfortunately, the inter-examiner reliability of the prone knee flexion test has only been found to be 0.21–0.26 [45, 46].

The *supine to sit test* or *long sitting test* is employed to differentiate functional versus anatomical leg length discrepancy. To perform this test, the patient is placed in the supine position with legs fully extended and the medial malleoli aligned. The patient is then instructed to rise to a sitting position without moving his or her legs. The examiner observes this motion, paying particular attention to the medial malleoli. If the patient is unable to rise without one leg shifting proximally to the other, then there is likely some degree of pelvic dysfunction or malrotation contributing to any discrepancy present (Fig. 2.19). Bemis and Caniel evaluated 51 asymptomatic individuals and found the test to have a sensitivity of 0.17 and specificity of 0.38 [33]. However, a limitation of this study was that all of the subjects were asymptomatic.

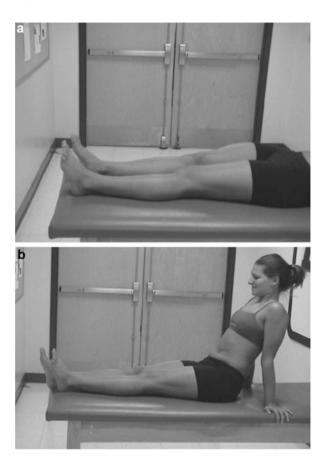


Fig. 2.19 Long-sit test. (a) Supine and (b) seated

The *standing flexion test* is also used to assess for pelvic dysfunction. The posterior superior iliac spines are palpated while the patient stands vertically and then maximally flexes at the waist. A positive test is marked by migration of one side cephalad (cranially) and suggests SI joint hypomobility. Cohort studies have found inter-examiner reliability of the standing flexion test to range from 0.08 to 0.68 [45, 47–51].

The *Gillet test* is another assessment of the SI joint. The patient stands with feet separated by about a foot, and the examiner's thumbs are again placed on the posterior superior iliac spines. The patient is then instructed to balance himself or herself on a single leg while pulling the opposite leg toward his or her chest wall. The maneuver is performed bilaterally, and a positive finding is noted if the posterior superior iliac spine on the flexed side migrates vertically or remains still, indicating inadequate SI flexibility or hypomobility. Dreyfuss et al. determined the sensitivity and specificity of this particular test to be 0.43 and 0.68, respectively, making it a poor screening tool if SI dysfunction is suspected [35].

The *Gaenslen sign* is also used to elicit symptoms of SI disorders. This test is performed by positioning a supine patient on the edge of the table with both legs flexed to his or her chest. The patient is then instructed to allow the outside leg to hang off of the side of the table as the examiner stabilizes the patient's torso. Pain in the SI region with this maneuver indicates a positive test. Sensitivity and specificity of this test have varied widely in multiple studies, with ranges from 0.21 to 0.71 and 0.26 to 0.72, respectively [35–37].

Passive abduction and *resisted adduction* are useful maneuvers for differentiating pubic symphysis pathology from other midline pelvic symptoms [52]. The exam is performed with the patient in the lateral decubitus position and 90° flexion of the knees and hips. Pain at the pubic symphysis with either maneuver from this position is considered a positive test and may be indicative of osteitis pubis.

The *lateral pelvic compression test* is also performed from the lateral decubitus position with knees and hips flexed. To perform this test, the examiner places direct downward pressure to the greater trochanter. Once again, midline pain overlying the pubic symphysis is consistent with bony injury or osteitis pubis [12].

The *fulcrum test* is performed with the patient seated on the exam table with legs hanging from the edge. The examiner's fore-arm is positioned under the patient's thigh for use as a fulcrum as pressure is applied to the ipsilateral knee by the examiner's spare hand. In this manner, the examiner moves up and down the entire shaft of the femur attempting to elicit any point tenderness which may indicate a stress fracture of the overlying bone [11]. Either sharp pain or apprehension indicates a positive test.

In the Patellar Pubic Percussion Test (PPPT) the practitioner stands next to a supine patient, and from the side to be tested places a stethoscope over the pubic tubercle while tapping on or applying a tuning fork to the ipsilateral patella. A femoral fracture reduces the propagation of sound waves resulting in a diminished volume transmitted to the stethoscope when compared to the contralateral side (if uninjured). The test has utility both as a screening exam and a diagnostic test with pooled sensitivity and specificity values of 0.95 and 0.86, respectively, across three studies [27–29, 53].

Finally, *Craig's test* [11] is used to test for femoral torsion. The normal range of femoral anteversion, or forward projection of the femoral neck, changes throughout life from an average range of $30-40^{\circ}$ in infants to $8-15^{\circ}$ in adults. Excessive anteversion, or less commonly femoral retroversion, can be problematic and presents most frequently in the pediatric population. To perform the exam, the patient is instructed to lie prone on the exam table with the knee of the side being tested flexed to 90° . From this position the examiner palpates the greater trochanter of the flexed knee and internally and externally rotates the hip to find the position in which the greater trochanter is most lateral. The degree of femoral anteversion can then be estimated using a goniometer with the stationary arm perpendicular to the floor and the moving arm at the angle of the leg.

2.6 Case Report: Conclusion

2.6.1 Assessment

- External snapping of the right hip secondary to inflexibility of the iliotibial band and tensor fascia lata which is secondary to core instability, specifically gluteus medius weakness.
- Poor hamstring flexibility bilaterally.

2.6.2 Plan

Physical therapy to address the above biomechanical deficits with specific attention to the core instability. Cross train to maintain cardiovascular fitness. Decrease weekly running mileage in half and decrease running pace by 1 minute per mile.

2.6.3 Follow-up

After 4 weeks of physical therapy, the patient's pain had resolved. Once the patient became completely pain free, his running mileage and pace were gradually increased. When he was able to run on level ground at his previous pace, hills were slowly re-introduced. He was back competing at pre-injury levels by his second competition, which was 6 weeks after presentation.

References

- 1. Scopp M. The assessment of athletic hip injury. Clin Sports Med. 2001;20(4):647-59.
- Hurwitz DE, Foucher KC, Andriacchi TP. A new parametric approach for modeling hip forces during gait. J Biomech. 2003;36(1):113–9.
- 3. Jenkins DB. The bony pelvis, femur, and hip joint. In: Jenkins DB, editor. Hollinshead's functional anatomy of the limbs and back. Philadelphia: W.B. Saunders; 1998. p. 239–48.
- 4. Adkins III SB, Figler RA. Hip pain in athletes. Am Fam Physician. 2000;61(7):2109–18.
- 5. Metzmaker JN, Pappas AM. Avulsion fractures in the pelvis. Am J Sports Med. 1985;13(5):349–58.
- Vijlbrief AS, Bruijnzeels MA, van der Wouden JC, van Suijelkom-Smit LW. Incidence and management of transient synovitis of the hip: a study in Dutch general practice. Br J Gen Pract. 1992;42(363):426–8.
- Tolat V, Carty H, Klenerman L, Hart CA. Evidence for a viral aetiology of transient synovitis of the hip. J Bone Joint Surg. 1993;75(6):973–4.
- 8. Weir A, Brukner P, Delahunt E, et al. Doha agreement meeting terminology and definitions in groin pain in athletes. Br J Sports Med. 2015;49:768–74.
- Hage WD, Aboulafia AJ, Aboulafia DM. Orthopedic management of metastatic disease: incidence, location, and diagnostic evaluation of metastatic bone disease. Orthop Clin North Am. 2000;31(4):515–28.

- Geraci Jr MC, Brown W. Evidence-based treatment of hip and pelvic injuries in runners. Phys Med Rehabil Clin N Am. 2005;16(3):711–47.
- Magee DJ. Pelvis. In: Magee DJ, editor. Orthopedic physical assessment. 3rd ed. Philadelphia: WB Saunders; 1997.
- 12. Seidenberg PH, Childress MA. Evaluating hip pain in athletes. J Musculoskel Med. 2005;22(5):246–54.
- 13. Allen WC, Cope R. Coxa saltans: the snapping hip syndrome. J Am Acad Orthop Surg. 1995;3:303.
- 14. Bird PA, Oakley SP, Shneir R, et al. Prospective evaluation of magnetic resonance imaging and physical exam findings in patients with greater trochanteric pain syndrome. Arthritis Rheum. 2001;44:2138–45.
- Woodley SJ, Nicholson HD, Livingstone V, et al. Lateral hip pain: findings from magnetic resonance imaging and clinical examination. J Orthop Sports Phys Ther. 2008;38:313–28.
- Lequesne M, Mathieu P, Vuillemin-Bodaghi V, et al. Gluteal tendinopathyin refractory greater trochanter pain syndrome: diagnostic value of two clinical tests. Arthritis Rheum. 2008;59:241–6.
- 17. Marks MC, Alexander J, Sutherland DH, Chambers HG. Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. Dev Med Child Neurol. 2003;45:763–8.
- Martin RL, Irgang JJ, Sekiya JK. The diagnostic accuracy of a clinical examination in determining intra-articular hip pain for potential hip arthroscopy candidates. Arthroscopy. 2008;24:1013–8.
- 19. Troelsen A, Mechlenburg I, Gelineck J, et al. What is the role of clinical tests and ultrasound in acetabular labral tear diagnostics. Acta Orthop. 2009;80:314–8.
- 20. Maslowski E, Sullivan W, Forster Harwood J, et al. The diagnostic validity of hip provocation maneuvers to detect intra-articular hip pathology. PM R. 2010;2:174–81.
- Reiman MP, Mather III RC, Cook CE. Physical exam tests for hip dysfunction and injury. Br J Sports Med. 2015;49:357–61.
- 22. McCarthy JC, Busconi B. The role of hip arthroscopy in the diagnosis and treatment of hip disease. Orthopedics. 1995;18:753–6.
- 23. Youdas JW, Madson TJ, Hollman JH. Usefulness of the Trendelenburg test foridentification of patients with hip joint osteoarthritis. Physiother Theory Pract. 2010;26:184–94.
- Sutlive TG, Lopez HP, Schnitker DE, et al. Development of a clinical prediction rule for diagnosing hip osteoarthritis in individuals with unilateral hip pain. J Orthop Sports Phys Ther. 2008;38:542–50.
- Johnson AW, Weiss Jr CB, Wheeler DL. Stress fractures of the femoral shaft in athletes-more common than expected. A new clinical test. Am J Sports Med. 1994;22:248–56.
- 26. Kang L, Belcher D, Hulstyn MJ. Stress fractures of the femoral shaft in women's college lacrosse: a report of seven cases and a review of the literature. Br J Sports Med. 2005;39:902–6.
- 27. Bache JB, Cross AB. The Bardford test: a useful diagnostic sign in fractures of the femoral neck. Practitioner. 1984;228:305–8.
- Adams SL, Yarnold PR. Clinical use of the patellar-pubic percussion sign in hip trauma. Am J Emerg Med. 1997;15:173–5.
- 29. Tiru M, Goh SH, Low BY. Use of percussion as a screening tool in the diagnosis of occult hip fractures. Singapore Med J. 2002;43:467–9.
- Vroomen PC, de Krom MC, Knotterus JA. Diagnostic value of history and physical examination in patients with sciatica due to disc herniation; a systematic review. J Neurol. 1999;246(10):899–906.
- Fishman L, Zybert P. Electrophysiologic evidence of piriformis syndrome. Arch Phys Med Rehabil. 1992;73:359–64.
- 32. Nouraei SA, Anand B, Spink G, O'neil KS. A novel approach to the diagnosis and management of meralgia parasthetica. Neurosurgery. 2007;60:696–700.
- Bemis T, Caniel M. Validation of the long sitting test on subjects with iliosacral dysfunction. J Orthop Sports Phys Ther. 1987;8:336–45.

- 2 Physical Examination of the Hip and Pelvis
- Levangie PK. Four clinical tests of sacroiliac joint dysfunction: the association of test results with innominate torsion among patients with and without low back pain. Phys Ther. 1999;79:1043–57.
- Dreyfuss P, Michaelen M, Pauza K, et al. The value of the medical history and physical examination in diagnosing sacroiliac joint pain. Spine. 1996;21:2594–602.
- Russel AS, Maksymowych W, LeClercq S. Clinical examination of the sacroiliac joints: a prospective study. Arthritis Rheum. 1981;24:1575–7.
- 37. van der Wuff P, Hagmeijer R, Meyne W. Clinical tests of the sacroiliac joint. Man Ther. 2000;5:30–6.
- Ozgocmen S, Bozgeyik Z, Kalcik M, Yildrim A. The value of sacroiliac pain provocation tests in early sacroiliitis. Clin Rheumatol. 2008;10:1275–82.
- Hardcastle P, Nade S. The significance of the Trendelenburg test. J Bone Joint Surg. 1985;67(5):741–6.
- 40. Gajdosik RL, Sandler MM, Marr HL. Influence of knee positions and gender on the Ober test for length of the iliotibial band. Clin Biomech. 2003;18(1):77–9.
- Broadhurst N, Bond M. Pain provocation tests for the assessment of sacroiliac dysfunction. J Spinal Disord. 1998;11:341–5.
- Ross MD, Nordeen MH, Barido M. Test–retest reliability of Patrick's hip range of motion test in healthy college-aged men. J Strength Cond Res. 2003;17(1):156–61.
- 43. McGrory BJ. Stinchfield resisted hip flexion test. Hosp Phys. 1999;35(9):41-2.
- 44. Rhodes DW, Mansfield ER, Bishop PA, Smith JF. The validity of the prone leg check as an estimate of standing leg length inequality measured by X-ray. J Manipulative Physiol Ther. 1995;18(6):343–6.
- 45. Riddle D, Freburger J. Evaluation of the presence of sacroiliac joint dysfunction using a combination of tests: a multicenter intertester reliability study. Phys Ther. 2002;82:772–81.
- O'Haire C, Gibbons P. Inter-examiner and intra-examiner agreement for assessing sacroiliac anatomical landmarks using palpation and observation: pilot study. Man Ther. 2000;5:13–20.
- Touissaint R, Gawlik C, Rehder U, Ruther W. Sacroiliac dysfunction in construction workers. J Manipulative Physiol Ther. 1999;22:134–9.
- Touissaint R, Gawlik C, Rehder U, Ruther W. Sacroiliac joint diagnosis in the Hamburg Construction Workers study. J Manipulative Physiol Ther. 1999;22:139–43.
- Vincent-Smith B, Gibbons P. Inter-examiner and intra-examiner reliability of the standing flexion test. Man Ther. 1999;4:87–93.
- Potter N, Rothstein J. Intertester reliability for selected clinical tests of the sacroiliac joint. Phys Ther. 1985;65:1671–5.
- Flynn T, Fritz J, Whitman J, et al. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation. Spine. 2002;27:2835–43.
- 52. Nuccion S, Hunter D, Finerman G. Hip and pelvis. In: DeLee J, Drez D, Miller MD, editors. DeLee and Drez's orthopedic sports medicine. 2nd ed. Philadelphia: Saunders; 2003.
- 53. Reiman MP, Goode AP, Hegedus EJ, et al. Br J Sports Med. 2013;47:893-902.

Chapter 3 Functional and Kinetic Chain Evaluation of the Hip and Pelvis

Per Gunnar Brolinson, Mark Rogers, and Joseph Edison

Clinical Pearls

Some common signs of kinetic chain dysfunction:

- Abnormal muscle firing sequences on muscle testing
- Poor proprioception
- Need for frequent manual medicine or manipulation
- "Weak" phasic muscles on exam
- Easy fatigability of phasic muscles
- Chronic musculoskeletal pain

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© Springer International Publishing Switzerland 2017 P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_3

Dedicated to the memory of Kevin Granata, PhD, Virginia Tech, 4–16–07, husband, father, and pre-eminent biomechanical researcher.

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3.1 Case Presentation

3.1.1 Chief Complaint and History

B.L. is an 18-year-old varsity college third baseman at a Division I university with the complaint of right hip and right low back pain (LBP). He noted waxing and waning discomfort for the past 2 years, and for the past 6 months, his pain has been progressively worsening resulting in altered batting mechanics and reduction in hitting power and efficiency. His initial injury occurred, while playing high school baseball, when he was sliding into second base and collided with another player. That player's knee struck the posterior aspect of B.L.'s right hip. He was unable to finish playing the game and took 3 weeks off from competitive baseball secondary to the right hip pain. He gradually resumed activities and returned to baseball, but was never completely free from pain. He sustained a second injury about 1 year later consisting of an axial load through the right femur after falling down on his right knee and ultimately landed again on the right posterior hip. He was diagnosed with "right hip spasm" and placed on muscle relaxers and given a course of physical therapy. He improved but never really felt 100 % healthy. Prior to coming to college, he had had no diagnostic workup.

When initially seen in our sports medicine clinic, he complained of his "typical" right low back and right posterior hip discomfort. He had some radiation of pain into the low back and posterior hip regions but denied true radicular symptoms.

3.1.2 Physical Examination

Nerve root tension signs were negative, but straight leg raise did produce some mild upper hamstring and posterior hip discomfort. Neurovascular and motor exams were normal. The right hamstring was tight. He had reduced motion in internal and external hip rotation with some reproduction of hip and low back discomfort at extremes of motion. Functional structural evaluation revealed a positive standing forward flexion test (Fig. 3.1) with an associated sacral torsion as well as restricted motion and tissue texture changes noted at the lumbosacral and thoracolumbar regions. He had multiple tender points noted in the right quadratus lumborum. His gait was normal. Anteroposterior pelvis x-ray with a frog leg view of the right hip was obtained and was normal. X-rays of the sacrum and coccyx were normal. He was initially treated with anti-inflammatory medicine, manipulation to correct the dysfunctions noted on exam, and was seen in the training room for functional therapeutic exercise and modalities as indicated.

He had a partial response to this treatment but continued to experience episodic discomfort. A lumbar spine magnetic resonance image (MRI) was obtained to evaluate him for potential disk pathology and revealed signal change and mild bulging of the L4 disk centrally which was not felt to be clinically significant. Despite aggressive

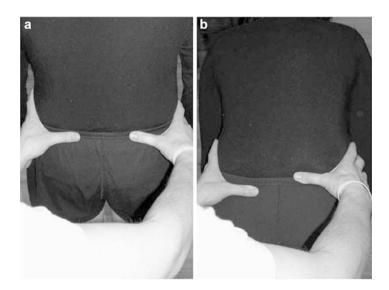


Fig. 3.1 Standing flexion test in neutral (*left*) and forward (*right*) flexion. Note elevated right thumb with forward flexion while monitoring PSIS, signifying a positive standing flexion test on the right

conservative management and transient improvement in symptoms following manipulation, he continued to demonstrate a recurrent dysfunctional pattern in the hip, pelvis, and low back area. Ultimately, a magnetic resonance (MR) arthrogram of the right hip was obtained demonstrating a right hip labral tear anteriorly and inferiorly with an associated paralabral cyst.

3.2 Introduction

A *kinetic chain* can be described as the sequencing of individual body segments and joints to accomplish a task. It generally functions from a base of support proximally and then proceeds distally, but this is entirely dependent on the task at hand. Because of the unique nature of sport and the tremendous demands that most sporting activities place on the spine, pelvis, and hip, the ability to recognize kinetic chain disorders related to these specific structures and their interactions with related components of the musculoskeletal system is important for sports medicine practitioners. Because of the complexity of the anatomic and biomechanical interactions as well as neuromuscular control issues, evaluation and accurate diagnosis are often problematic.

The hip and pelvis serve as a force transfer link between the lower extremities and torso, and as such, is an at-risk region for athletic injury. The evaluation and treatment of hip and pelvis dysfunction is controversial. One issue is the broad categorization and terminology utilized for the anatomic etiologies of the pain by various health care practitioners. There is no specific or salient historical issue or single clinical examination technique that is both sensitive and specific for the diagnosis of hip and/or pelvis dysfunction. To date, imaging studies do not always distinguish the asymptomatic from symptomatic patient population, nor is there a gold standard for the treatment of the symptom complex associated with these problems in the active patient population [1].

As noted in Chap. 1, sports injuries to the hip and groin region have been noted in 5–9% of high school athletes [2, 3] and, according to NCAA Injury Surveillance System from 2004–2009, in 2.2–14.7% of collegiate athletes [4, 5]. These injuries occur most commonly in athletes participating in sports involving side-to-side cutting, quick accelerations and decelerations, and sudden directional changes. The sports medicine practitioner must diligently evaluate hip and pelvis pain and carefully monitor the athlete's response to initial conservative management. This is paramount not only because of the difficulty in making an accurate diagnosis, but also because 27–90% of patients presenting with groin pain have more than one coexisting injury [6–8]. This emphasizes the need for a thorough and comprehensive functional biomechanical evaluation of the region.

3.3 Natural History

The clinical evidence suggests that hip and pelvis dysfunction may not simply be an acute process that resolves with time alone. Typically, hip and pelvis pain and dysfunction are often a recurrent problem similar to other chronic musculoskeletal conditions that may have symptom-free periods interspersed with exacerbations. Therefore, physicians must approach hip and pelvis dysfunction with this mindset and be aware of its potential episodic, recurrent, and chronic nature. They should initially seek treatment methods that are active and physical in nature to help restore the body's normal balance of regional and segmental joint motion, posture, and neuromuscular control, with appropriate functional strength and flexibility (SOR=B).

There are studies indicating that physical activity is a risk factor for the development of osteoarthritis of the hip and pelvis. Unfortunately, osteoarthritis of the hip is relatively common in athletes, second only to osteoarthritis of the knee. It may be the result of chronic overuse or secondary to specific traumatic events, such as transient subluxation or chondral injury [9, 10]. Kujala et al. performed a retrospective review on former elite male athletes [11]. Using a registry of Finnish male athletes who competed at an Olympic or other international level between 1920 and 1965, the study looked at relative risks of development of chronic disease. Although the participants had significant improvement in health with respect to coronary artery disease, diabetes, and hypertension, an increase in the development of osteoarthritis was noted in these athletes. The cohort was divided into endurance sports (runners and cross country skiers), mixed sports (soccer, basketball, and ice hockey), and power sports (boxing, wrestling, and weight lifting). The relative risks for osteoarthritis were 2.42, 2.37, and 2.68, respectively [11], with a 2–3 fold increase risk in females [12]. In another retrospective study, Spector et al. evaluated 81 female ex-elite middle long-distance runners and tennis players [12]. In comparison with 997 age-matched controls, the athletes demonstrated a relative risk of 2.5 for hip arthritis, and 3.5 for knee arthritis. A cross-sectional study performed by Lindberg et al. demonstrated a 5.8% incidence of hip osteoarthritis in 286 ex-soccer players compared with a 2.8% incidence in controls [13].

Contradicting this information are studies in long-distance runners, which fail to demonstrate an increased risk for osteoarthritis. Lane et al retrospectively studied 41 long-distance runners averaging 5 h/week of running over 9 years, concluding that there was no increased risk of osteoarthritis in runners [14]. Konradsen et al. evaluated 58 ex-long-distance runners who averaged more than 20 km/week of running over 40 years and compared them to age, weight, and occupation-matched controls. Radiographically, the athletic cohort had no significant changes suggestive of osteoarthritis when compared with controls [15].

Multiple studies have demonstrated a risk of hip osteoarthritis for professional soccer players that may be as high as 13.2%, or 10.2 times that of the general population, even in the absence of identifiable injury to the joint [13, 16, 17]. Other studies have shown other significant increases in risk in rugby players [18], javelin throwers [19], high jumpers, track, and field sports [20]. Osteoarthritis is also common among former National Football League (NFL) players, with 62% reporting some arthritic problem, compared to 32% of general male population, in a 2009 NFL Player's Care Foundation study [21]. This association of hip osteoarthritis with significant athletic activity has been demonstrated in female as well as male athletes [12, 22, 23].

Although it is not a consensus opinion of these articles, excessive microtrauma from exercise and cumulative overuse can potentially increase the risk of developing joint injury and osteoarthritis. This risk is dependent on the amount, type, and intensity of the exercise, as well as the genetics, joint structure, fitness, and body habitus of the individual [24, 25]. Other specific risk factors include high loads, sudden or irregular impact [26], and preexisting abnormalities such as dysplasia [13, 27]. More recently, labral tears of the hip have been implicated in early osteoarthritis [28].

In summary, the clinician should view athletic hip and pelvis pain and dysfunction as a common injury with a potentially episodic nature that can affect the athlete's ability to function in both sports and personal life. Treatment must be focused on complete functional recovery and prevention, not just elimination of acute pain, as there appears to be significant risk for the development of hip arthritis if the pain and resultant kinetic chain dysfunction is left untreated (SOR = B).

3.4 Functional Anatomic Concepts and Neuromuscular Control

Arthrokinetic responses are transmitted through the neuromuscular system as proprioceptive data processed by the central nervous system (CNS). These responses are separate from stretch reflexes, though some of the same pathways are utilized. The four nerve types responsible for transmitting afferent information from the joint are globular (static and dynamic mechanoreceptors), conical (dynamic mechanoreceptor), fusiform (mechanoreceptor), and plexus (nociceptor) [29].

The gamma loop mechanism functions in the following fashion. A dynamic load applied to the tendon stretches the spindle muscle fibers. This activates the afferent nerve fibers which synapse in the anterior horn (we are skipping the numerous interneurons for simplicity) on the alpha motor neurons in the same and adjacent spinal segments, simultaneously inhibiting the antagonist muscle groups. If the capsule or ligament becomes stretched beyond what its programming allows for as a normal range of motion (or if too rapid a stretch occurs), inhibitory signals are sent to the agonist muscle responsible for loading the joint in the plane in question and stimulatory signals to the antagonist musculature [29].

An *engram* is a memorized series of muscle activation patterns (MAPs), for example, tying your shoes, or changing lanes when driving a car. They free up your conscious mind from the task at hand, allowing you to focus on other tasks simultaneously. The development and "burning in" of successful engrams as well as kinetic chain movement patterns (a specific sequence of engrams resulting in a motion) result in successful athletic performance. Injuries and overload can happen when there is compensation for dysfunction (motion loss) in the earlier (temporally speaking) components of the kinetic chain and can lead to injury in the later components, as the tissues either cannot handle the load or the neuromuscular system fires inappropriately [29].

Neuromuscular imbalance in the postural musculature, either due to hypertonicity or inhibition, allows microtrauma to begin to insidiously accumulate. With repetitions of these dysfunctional MAPs, dysfunctional kinetic chains and engrams develop that "burn in" the dysfunctional, although usually asymptomatic, pathways even more. Pain and/or pathology usually will begin in the local stability system, which cannot maintain its functioning, thus perpetuating the loop [29]. Tendons and ligaments lose their tensile properties over time. Proprioceptive inputs become less reliable and actually can become harmful as MAPs and their kinetic chains are thereby altered, leading to abnormal loading of bone and the supporting soft tissues.

3.5 Clinical Biomechanics

Much of our understanding of the biomechanics of the hip joint has been obtained through simple static diagrams, gait analysis, and through the insertion of forcemeasuring implants. The muscles about the hip joint are generally at a mechanical disadvantage because of a relatively short lever arm and a production of forces across the joint that are several times body weight. It has been calculated that level walking can produce forces of up to six times body weight and that jogging with a stumble increases these forces to up to eight times body weight [30]. Although forces, when measured in vivo, tend to be less than the calculated values, one can anticipate potentially greater loads during vigorous sports athletic competition [31]. The structures about the hip are uniquely adapted to transfer such forces. The body's center of gravity is located within the pelvis, anterior to the second sacral vertebra; thus, the loads that are generated or transferred through this area are important in virtually every athletic endeavor [32].

The normal hip joint is capable of a flexion and extension arc of approximately 140°, but one study has shown that slow-paced jogging used only about 40° of this arc [33]. This increases somewhat as pace increases. Analysis of electromyographic. (EMG) activity shows that the rectus femoris and iliac muscles are very active with swing-phase hip flexion, while the hamstring muscles act eccentrically to control hip flexion and decelerate knee extension [34]. It is of note that, when running, the body is propelled forward primarily through hip flexion and knee extension rather than by push-off with ankle plantar flexion.

Sahrmann has described a hip lateral rotation (HLR) movement impairment that she has observed in people with LBP [35]. The impairment is described as early coupling [36, 37] of the primary hip rotation motion with lumbopelvic rotation during a clinical test of active HLR in prone position [38]. The HLR test was performed with the patient in the prone position, the knee flexed to 90°, and the hip in neutral rotation and neutral abduction/adduction. At a self-selected movement speed, patients laterally rotate the hip as far as possible toward the opposite leg, and then return it to the starting position. This is done both actively and passively and the amount and quality of the motion is noted by the clinician [35]. The relationship between LBP and repeated early coupling of hip and lumbopelvic rotation may be of particular importance in people who put rotational demands on both the hip and lumbopelvic region [35]. Passive tissue stiffness about the hips has the potential to contribute to early motion of the lumbopelvic region during HLR [37].

Patients may, therefore, demonstrate lumbopelvic-coupled movement early during HLR because they have a greater amount of passive stiffness in the hip musculature. The difference in the pattern of movement during the HLR test may be the result of an interaction of biomechanical factors, such as passive tissue stiffness, and motor control factors, such as timing and magnitude of muscle activity. Identifying how movement patterns differ during this clinical test is important because it provides information that can assist the clinician in treatment of a person with hip and pelvis pain and dysfunction.

Atraumatic instability can occur because of overuse or repetitive motion. This is a common complaint in athletes who participate in sports involving repetitive hip rotation with axial loading (i.e., figure skating, golf, football, baseball, martial arts, ballet, gymnastics, etc.). The history provides the greatest clues to the diagnosis because patients can usually describe the motion that causes the pain, such as swinging a golf club during a drive or throwing a football. These repetitive stresses may directly injure the iliofemoral ligament or labrum and alter the balance of forces in the hip. These abnormal forces cause increased tension in the joint capsule, which can lead to capsular redundancy, painful labral injury, and subsequent microinstability. On physical examination, patients will usually experience anterior hip pain while in the prone position with passive hip extension and external rotation [39, 40]. Once the static stabilizers of the hip, including the iliofemoral ligament and labrum, are injured, the hip must rely more on the dynamic stabilizers to maintain stability during activity. It is hypothesized that when capsular laxity is present, the psoas major, a dynamic stabilizer of the hip, contracts to provide hip stability. Over time, this condition can lead to stiffness, coxa saltans, or flexion contractures of the hip [41, 42]. In addition, because of the origin of this muscle from the lumbar spine, a chronically contracted or tightened psoas major may be a major contributor to LBP. Thus, hip instability or capsular laxity can trigger a whole spectrum of disorders that the physician must take into consideration when considering various treatment options [37].

The relationship between hip rotation motion, hip stability, and LBP is important because external forces must be sequentially transmitted from distal body segments to more proximal ones during movement. Movement at the hip could, therefore, influence movement and loading at the lumbar spine. When performed repeatedly, such hip movement could result in excessive loading on tissues in the low back region, and eventually LBP [35].

In 2001, Vleeming et al. [43] described their integrated model of joint dysfunction. This functional description comes from extensive study of the sacroiliac joint (SIJ) over the past 10–15 years, and is the most studied and supported model for sacroiliac joint dysfunction (SIJD). It integrates structure (form and anatomy), function (forces and motor control), and the mind (emotions and awareness) on human performance. Integral to the biomechanics of SIJ stability is the concept of a selflocking mechanism. The SIJ is the only joint in the body that has a flat joint surface that lies almost parallel to the plane of maximal load. Its ability to self-lock occurs through two types of closure—form and force.

Form closure describes how specifically shaped, closely fit contacts provide inherent stability independent of external load. Force closure describes how external compression forces add additional stability (Fig. 3.2). It had long been thought that only the ligaments in this region provided that additional support. However, it is the fascia and muscles within the region that provide significant self-bracing or self-locking to the SIJ and its ligaments through their cross-like anatomic configuration. Ventrally, this is formed by the external abdominal obliques, linea alba, internal abdominal obliques, and transverse abdominals, whereas dorsally the latissimus dorsi, thoracolumbar fascia, gluteus maximus, and iliotibial tract contribute significantly. In addition, there appears to be an arthrokinetic reflex mechanism by which the nervous system actively controls this added support system. These supports are critical in asymmetric loading, when the SIJ is most prone to subluxation. The important concept to gain from this understanding of integrated function with regard to treatment and prevention of LBP is that SIJD is a *neuromyofascialmusculoligamentous* injury [1].

The relationship of the abdominal musculature and the erector muscles of the spine, along with their role in stabilization of the lumbosacral spine, is being studied extensively because of the high incidence of LBP in our society. Decreased spinal mobility and trunk muscle strength have been identified in patients with recurrent LBP [44]. These muscles must also be considered for their role in conditions that affect pelvic tilt

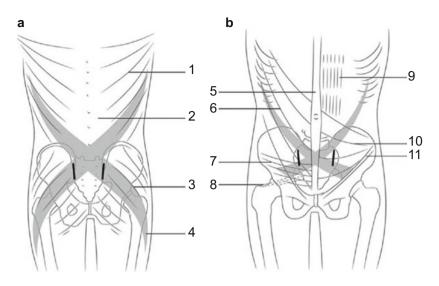


Fig. 3.2 The cross-like configuration demonstrating the force closure of the sacroiliac joint. The SIJ becomes stable on the basis of dynamic force closure via the trunk, arm, and leg muscles that can compress it, as well as its structural orientation. The cross-like configuration indicates treatment and prevention of low back pain with strengthening and coordination of trunk, arm, and leg muscles in torsion and extension rather than flexion. The crossing musculature is noted. (a) (1) Latissimus dorsi; (2) Thoracolumbar fascia; (3) Gluteus maximus; (4) Iliotibial tract. (b) (5) Linea alba; (6) External abdominal obliques; (7) Transverse abdominals; (8) Piriformis; (9) Rectus abdominis; (10) Internal abdominal obliques; (11) Ilioinguinal ligament. From Brolinson PG. Sacroiliac joint dysfunction in athletes. Curr Sports Med Rep. 2003;2:47–56; used with permission

and the hip joint. The transversus abdominis has been shown to be the key muscle to functional stability of the lumbosacral pelvic region to generate stability and retraining of the core, because of its observed patterns of firing before and independent of the other abdominal muscles. Most recently, a study by Richardson et al. [45] appears to show that these clinical benefits focusing on the transversus abdominis occur as a result of significantly reduced laxity in the SIJ. The balance of the muscles of the upper thigh, particularly the adductor muscles, with those of the lower abdomen requires further study. Conditioning programs have traditionally focused on strengthening of the extremities. Only recently have there been rehabilitation programs designed to address the power and endurance of the trunk and postural muscles [46, 47].

Vleeming et al. [48] defined the posterior layer of the thoracolumbar fascia as a mechanism of load transfer from the ipsilateral latissimus dorsi and the contralateral gluteus maximus. This load transfer is critical during rotation of the trunk, helping to stabilize the lower lumbar spine and pelvis. This was demonstrated through cadaveric and EMG studies [49]. The stretched tissue of the posterior thoracolumbar fascia assists the muscles by generating an extensor influence and by storing elastic energy during lifting to improve muscular efficiency [1].

In recent years, intramuscular EMG studies of the hip flexor muscles during human locomotion have revealed a separate role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis, and hip [49-51]. In 1995, Vleeming et al. presented evidence that the iliacus muscle was selectively recruited in the standing position with extension of the contralateral leg, and in standing, maximal ipsilateral abduction, significantly higher levels of activation in the iliacus muscle, when compared with the psoas muscle, were found [48]. This suggested preferential action of involved single-joint muscles when possible to achieve local pelvic control. In another study, Anderson et al studied walking and running and found that the iliacus muscle was the main "switch muscle" during low-speed walking [49]. Therefore, it is the key to reversing lower extremity motion from extension to flexion. In a later study, they reported that the iliacus and sartorius muscles performed a static function needed to prevent a backward tilting of the pelvis during trunk flexion sit-ups [51]. Also, with static supine leg lifts, there was progressively more activation of these muscles with increasing elevation of the extremity; they recognized that a change in pelvic tilt influenced activation of the iliacus and sartorius muscles. A backward pelvic tilt combined with a hypolordotic back decreased activation of these muscles, whereas forward pelvic tilt combined with a hyperlordotic back increased activation of these muscles. This suggests an important and separate role of the iliacus from the psoas in function and dysfunction of the low back and pelvis region [50].

Recent studies show there is both a functional and anatomic connection between the biceps femoris muscle and the sacrotuberous ligament [52–54]. This relationship allows the hamstring to play an integral role in the intrinsic stability of the pelvis and SIJ. It appears that the biceps femoris, often found to be short on the pathologic side in LBP, may actually be a compensatory mechanism via the previously described arthrokinetic reflexes to help stabilize the SIJ. In healthy individuals, a normal lumbopelvic rhythm exists, during which the first 65° of forward bending is via the lumbar spine, followed by the next 30° via the hip joints. Increased hamstring tension prevents the pelvis from tilting forward, which diminishes the forward bent position of the spine, which results in reducing the spinal load [54]. Normalization of the lumbopelvic rhythm is an essential component to treatment of LBP, hip, pelvis, and SIJD [1].

In the normal gait cycle (see Chap. 4: Gait Assessment), there are combined activities that occur conversely in the right and left innominates and function in connection with the sacrum and spine (Fig. 3.3) [55]. As one steps forward with the right foot, at heel strike the right innominate rotates posterior and the left innominate rotates anterior. During this motion, the anterior surface of the sacrum is rotated to the left and the superior surface is level, while the spine is straight but rotated to the left. Toward mid-stance, the right leg is straight and the innominate is rotated anterior. The sacrum is rotated right and side-bent left, while the lumbar spine is side-bent right and rotated left. At left heel strike, the opposite sequence will occur and the cycle is repeated. Throughout this cycle, there is a rotatory motion at the pubic symphysis, which is essential to allow normal motion through the SIJ. Several authors [56, 57] have suggested that pubic symphysis dysfunction in walking is one

3 Functional and Kinetic Chain Evaluation of the Hip and Pelvis

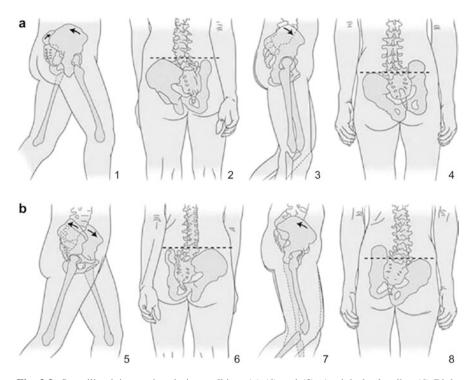


Fig. 3.3 Sacroiliac joint motion during walking. (a) (1) and (2): At right heel strike. (1) Right innominate has rotated in a posterior and left innominate in an anterior direction. (2) Anterior surface of sacrum is rotated to left and superior surface is level, while spine is straight but rotated to the left. (3) and (4): At right mid-stance. (3) Right leg is straight and innominate is rotating anterior. (4) Sacrum has rotated right and sidebent left, while lumbar spine has side-bent right and rotated left. (b) (5) and (6): At left heel strike. (5) Left innominate begins anterior rotation; after toe-off, right innominate begins posterior rotation. (6) Sacrum is level but with anterior surface rotated to the right. The spine, although straight, is also rotated to the right, as is the lower trunk. (7) and (8): At left leg stance. (7) Left innominate is high and left leg straight. (8) Sacrum has rotated to the left and side-bent right, while lumbar spine has side-bent left and rotated right. From Brolinson PG. Sacroiliac joint dysfunction in athletes. Curr Sports Med Rep. 2003;2:47–56; used with permission

of the essential or leading causes of the development of hip and pelvic dysfunction. In static stance, the lumbar spine regionally extends (i.e., lumbar lordosis), the sacrum regionally flexes, with the base moving forward and the apex moving posterior. During forward bending, both innominates go into a motion of external rotation and out-flaring. This combination of motion during forward bending is called nutation of the pelvis. The opposite occurs in backward bending, which is called counternutation. As the sacrum goes into extension with the base moving posterior and the apex anterior, the innominate components internally rotate and in-flare. This motion is clearly demonstrated and illustrated by Kapandji [58].

The model of suboptimal posture, though incomplete, has shown to be effective when used as a model to guide treatment [59-61]. Posture can be defined as the size,

shape, and attitude of the musculoskeletal system with respect to gravitational force [62]. Subtle departure from ideal posture has been implicated as an important biomechanical factor in athletes with regard to injury because it results in increased mechanical stress throughout the body. Posture must always be evaluated as part of the biomechanical evaluation. The size, shape, and attitude of three cardinal bases of support should always be included—standing surface, the feet, and the base of the sacrum [1].

Muscles respond to dysfunctional joints in a predictable, characteristic pattern. This pattern is not random and occurs irrespective of the clinical diagnosis or specific regional injury. Tonic or postural muscles are facilitated and hypertonic, which maintain a low level of tone nearly all the time. These muscles tend to utilize more fibers of an oxidative nature to avoid fatigue. Phasic or dynamic muscles are inhibited, hypotonic, or "weak" (pseudoparesis). They exhibit quicker, shorter bursts of activity with phases of rest in between and more often utilize the glycolytic pathway fibers [33]. The specific response pattern of the muscles in the lower half of the body is seen in Fig. 3.4.

Tonic muscles will increase their resting tone and become less pliable. Phasic musculature will become less responsive and weak. Both responses will carry negative impact for the kinetic chain resulting in compensatory phenomenon. This muscle dysfunction, referred to as neuromuscular imbalance, is characterized by a change in the sequence of MAPs. This has been described both as an upper crossed syndrome and a lower (pelvic) crossed syndrome that when combined produce a layered syndrome that can be appreciated throughout the body (Fig. 3.2). Superficial and deep EMG analysis reveals that there are delays in the activation of phasic muscles, a decrease in amplitude, and recruitment of phasic muscles, and that normal input can have an inhibitory effect [63].

Triggers of muscle imbalance patterns include muscle disuse, repetitive movements, development of inflexibility, and pain. Of these, pain seems to be the single dominant factor in the maintenance of these patterns. Muscle imbalance should be suspected any time there are abnormal firing sequences on range-of-motion testing, poor balance, recurrent somatic dysfunction, "weak" or easily fatigable phasic muscles on clinical exam, history of recurrent injury or other overuse injury in the same region, chronic pain, and postural imbalance. It is critical to understand these muscle imbalances because they may be a dominant factor in the cause of musculoskeletal pain and/or a major factor in the continuance of the pain. Failure to rehabilitate these patterns is sure to be a significant factor in recurrent injury [63].

Initially the pseudoparesis, as described above, may be seen as a CNS inhibition, not a true weakness. Over a prolonged time period of inhibition, the muscles actually may become weak. Attempts at strengthening the "weak" muscles only increase inhibition. Physiologically, a decrease in recruitment is seen with added resistance. These muscles actually may not appear grossly weak on initial testing but they are seen to fatigue quickly and demonstrate poor endurance. This can lead to poor motor control or neuromuscular instability, in which there is marked irregularity in sensory-motor balance. It is important to remember that treatments and rehabilitation must be directed at the cause of inhibition, the neural reflex, first as most likely this will be a major factor in recurrent injury.

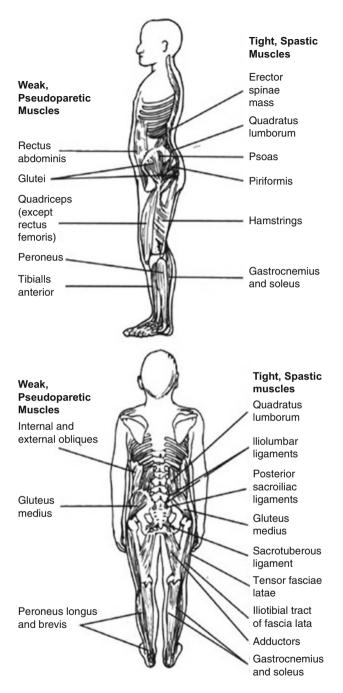


Fig. 3.4 Muscle imbalance caused by biomechanical stressors. From Kuchera ML. Treatment of gravitational strain pathophysiology. In: Vleeming A, Mooney V, Dorman T, et al., editors. Movement, stability, and low back pain: the essential role of the pelvis. New York: Churchill Livingstone; 1997. p. 477–99; used with permission

3.6 Common Hip and Pelvis Dysfunctions

In a review from by Rankin et al, between 2006–2011 in nearly 900 soccer, rugby, football, and running athletes, they found the largest etiology of hip and pelvic pain was 56% joint related. They further delineated between specific etiologies (joint, adductors, iliopsoas, and stress related injuries) and compared between males and females. In females, 77% were related to the joint specifically, followed by 17% iliopsoas, 4% pubic bone stress related injuries, and 3% adductor injuries. While in men, 45% were joint related, followed by 22% adductor, 19% pubic bone stress related, and 6% iliopsoas [64].

Strains of the adductor group (adductor longus, magnus, and brevis; gracilis; pectineus; and obturators) are the most common causes of acute groin pain in athletes. Their primary function is stabilization of the lower extremity and pelvis in the closed kinetic chain, as well as adduction of the thigh in the open kinetic chain and assisting in femoral flexion and rotation [65]. Strains are more common with eccentric loading. The adductor longus is most frequently affected, at the musculotendinous junction, likely because of its lack of mechanical advantage [66].

Among soccer players, incidence rates ranging between 10% and 18% have been reported [55, 67, 68]. Risk factors associated with increased incidence of strains include decreased hip range of motion, decreased adductor strength, and prior injury with 32–44% of injuries classified as recurrent [69–72]. In addition, biomechanical abnormalities of the lower limb, such as leg-length discrepancy, imbalance of the surrounding hip musculature, and muscular fatigue, have also been postulated to increase the risk of adductor strain [71]. Although there have been no controlled clinical studies proving these latter elements to be causative, prevention programs focused on ameliorating some of these abnormalities have been shown to be effective in professional hockey players [73].

In 2002, National Hockey League (NHL) statistics demonstrated that adductor strains occurred 20 times more frequently during training camp as opposed to the regular season, implying that deconditioning might contribute to these injuries, and therefore, functional sport-specific strengthening programs may be preventative. Such strengthening of the musculature of the hip, pelvis, and lower extremities has long been thought to be an important part of adductor injury prevention programs [74]; recently, these programs have been documented to be effective in preventing groin injuries in soccer and hockey players [72, 75]. In one study, Tyler et al. [72] presented their strengthening and injury prevention programs focused on decreasing adductor weakness (with a goal of keeping at least 80% of abductor strength). They found that adductor strengthening significantly reduced injury in NHL players.

Strains and tendonitis of the iliopsoas muscle usually occur at the musculotendinous junction during resisted hip flexion or hyperextension. Iliopsoas bursitis can occur alone or in conjunction with strain. The two conditions commonly occur concomitantly and are essentially identical in their clinical presentations [76]. The iliopsoas bursa is the largest bursa in the body. It communicates with the hip joint in 15% of people and can be a source of significant groin pain. Bursitis results from overuse and friction as

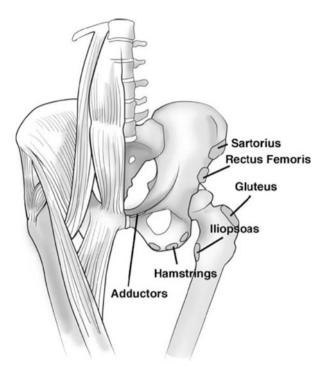


Fig. 3.5 Attachment of the muscles in the groin region. From Morelli V, Espinoza L. Groin injuries and groin pain in athletes: part 1. Prim Care Clin Office Pract. 2005;32:163–83; used with permission

the tendon rides over the iliopectineal eminence of the pubis (Fig. 3.5). This condition occurs in activities requiring extensive use of the hip flexors including soccer, ballet, uphill running, hurdling, and jumping. Iliopsoas bursitis is characterized by deep groin pain that sometimes radiates to the anterior hip or thigh and is often accompanied by a snapping sensation. If this is severe enough, the athlete may exhibit a limp [77]. Because of poor localization and reproducibility of the pain, the average time from the onset of symptoms to diagnosis has been reported from 32 to 41 months [76].

Pain may also be reproduced when the flexed, abducted, externally rotated hip is extended and brought back into a neutral position (extension test). During this maneuver, the iliopsoas is stretched as the hip is extended and symptoms are reproduced. Another diagnostic test is to have the supine athlete raise his or her heels off the table to about 15°; in this position, the only active hip flexor is the iliopsoas, and provocative testing as such will elicit pain [78]. Tenderness may be felt during this maneuver by palpating the psoas muscle below the lateral inguinal ligament at the femoral triangle [79]. The biomechanical abnormalities that may contribute to the injury must be sought and corrected as previously described.

High hamstring strains occur most commonly in dancers, hurdlers, runners, water skiers, and other athletes who place excessive stress on the stretched hamstrings [80]. Muscle fatigue may increase the risk of injury [66]. It must be remembered that the

ischial tuberosity may not fuse until the third decade [32] and that some of these injuries may actually represent an apophysitis rather than a true strain [32]. Patients usually present with posterior thigh pain and can have radiation to the groin as well. The diagnosis may be easily made with pain on palpation directly over the muscle insertion on the ischial tuberosity; however, it must be remembered that the adductor magnus also originates from the ischial tuberosity and that injuries to this muscle must also be considered in the differential diagnosis and rehabilitation treatment plan. These injuries occur usually with the hip extended and knee flexed when the sartorius undergoes a sudden contraction. Because the anterior superior iliac spine (ASIS) ossification center fuses relatively late, commonly at 21–25 years of age, a diagnosis of both strain and apophysitis must be entertained in these age groups [6].

Because rectus femoris is the only member of the quadriceps muscle group that spans two joints, it is subject to more stress and subsequently more injuries. Muscular injuries often result from the explosive hip flexion experienced in kicking or sprinting. Clinically, there is swelling and tenderness over the anterior thigh or at the anterior inferior iliac spine (AIIS) if the injury occurs at the tendon–bone interface. Conservative treatment is effective for most acute partial ruptures; however, small subsets of these patients go on to have chronic pain and disability [6].

Osteitis pubis, or inflammation of the symphysis pubis, is seen commonly in runners [81], hockey players [79], and soccer players. Shearing forces across the pubic symphysis may result in symphysis inflammation or even joint disruption [82]. Repetitive adductor traction on the symphysis has also been proposed as a possible mechanism [83]. Cutting and twisting forces may transmit even greater forces to the pubic symphysis in athletes lacking ideal ranges of hip flexibility [84, 85]. Although no published studies have addressed the role of biomechanical abnormalities of the lower limb (e.g., leg-length discrepancies or excessive pronation play), in the genesis of osteitis pubis, it is intuitive that such abnormalities might increase forces acting across the os pubis and thus increase susceptibility to the condition.

The symptoms may be initially indistinguishable from an adductor strain and may be aggravated by kicking and running. Symptoms may increase in severity if athletic activity is not reduced. A clinical review [79] noted that in athletes who have documented osteitis pubis, adductor pain occurred in 80%, pain around the pubic symphysis in 40%, lower abdominal pain in 30%, and hip pain in 12%, while scrotal pain, previously described as a classic complaint, was found in only 8%. Physical examination usually reveals tenderness over the pubic symphysis [8]. Pain can often be provoked by active adduction if the distal symphysis is involved or by partial sit-ups if the proximal portion is involved.

The two most common stress fractures of the groin region are femoral neck stress fractures and pubic ramus fractures. These are often seen in distance runners, endurance athletes, or military recruits and are caused by repetitive overuse and overload. Additional risk factors include relative osteoporosis in young female athletes who have nutritional or hormonal imbalances, changes in shoes or training surface, sudden increases in intensity or duration of training regimens, and muscle fatigue, which may reduce shock-absorbing abilities of the hip and pelvis region [86, 87].

An estimated 1% of stress fractures occur at the femoral neck. Although most femoral neck fractures are non-displaced at presentation, diagnostic delay is common, especially as initial radiographs are often normal. There has been a reported average diagnostic delay of up to 14 weeks [88]. A decreased range of motion at the hip has also been detected in athletes diagnosed with pubic bone stress injury [89].

During walking or running, the loads on the femoral head can exceed three to five times body weight. These loads occur because of gravity and the torque on the medial side of the hip joint, and are counteracted by contraction of the gluteus medius and minimus muscles. The force on the femoral head is transmitted through the neck to the femoral shaft, creating stresses and strains in the femoral neck secondary to compression and bending. If the abductor muscles fatigue and are unable to provide the normal compensatory tension, the tensile stress in the femoral neck will increase. Muscle fatigue also plays a role in gait alterations that affect the position of the body's center of mass and alter the stress and strain patterns within the femoral neck [90].

Pelvic biomechanical studies lend support to this view of increased stress to the central pubic bone area. These studies consider that during weight-bearing loads the superior pubic rami and the pubic symphysis act as a compression strut linking the femur to the posterior pelvic structures and spine [91], with the centers of rotation being near the pubic symphysis. Therefore, the pubic symphysis area is the region of the anterior pelvis most vulnerable to the stressors of athletic activity. Having a hip joint range-of-motion restriction will contribute to dysfunction, resulting in a greater stress across the superior pubic ramus and pubic symphysis. This, in turn, may lead to increased stress through this vulnerable area and increased likelihood of the athlete having chronic groin injury consistent with a pubic bone stress injury [92].

Although first reported by Patterson in 1957 [93], it is only in the last 15 years that acetabular labrum tears have become more widely recognized as a cause of hip and groin pain. In studies of patients presenting to a sports medicine center with chronic groin pain, 22% were found to have labral tears [94, 95]. In addition, 55% of athletes with mechanical hip pain also were found to have underlying labral tear [95]. Awareness and clinical suspicion of this condition among healthcare providers is important, especially in those athletes who have not responded to the prescribed treatment for the more familiar causes of hip and groin pain; early diagnosis and appropriate management lend themselves to improved outcomes [96].

The labrum is a fibrocartilaginous rim which encompasses the acetabulum, effectively deepening the socket, much like the glenoid labrum of the glenohumeral joint [96]. It can vary in form and thickness. It has three main surfaces: an internal articular surface, an external surface which is in contact with the joint capsule, and a basal surface which is attached to the acetabular bone and transverse ligaments. Its distal edge is free, forming the lateral limit of the acetabulum. Anteriorly, the labrum is equilaterally triangular in shape; posteriorly, it is square with a rounded distal surface, making it more bulbous.

In contrast to the glenoid in the shoulder, the acetabulum in the hip is much deeper and, therefore, provides substantially more stability to the hip joint. Thus, the deepening of the acetabulum that is provided by the labrum is thought to play less of a role in hip joint stability. The acetabular labrum, however, may enhance stability by providing negative intra-articular pressures within the hip joint with joint distraction, thereby adding a "sealing" function to the joint [97]. The sealing function of the labrum also may enhance the lubrication mechanism of the joint by preventing direct contact of the joint surfaces and more evenly distribute the applied forces across the cartilaginous surface [98]. The role of the acetabular labrum in load transmission was further examined in a cadaveric biomechanical study by Konrath et al. [99], in which they found no significant changes with regard to contact area, load, and mean pressure after removal of the labrum. From these findings they concluded that removal of the acetabular labrum does not predispose to premature hip osteoarthritis.

Femoroacetabular impingement (FAI) has been described as a cause of hip and pelvis pain as well as early development of osteoarthritis in young athletes [100-102]. There have been two mechanisms described which cause impingement between the femoral head and the acetabulum. The cam-type involves an abnormally shaped, nonspherical femoral head with decreased offset at the anterolateral head-neck junction, leading to impingement on the normal acetabulum and medial displacement of the labrum with flexion and internal rotation. The pincer type causes impingement of the normally shaped femoral head on a retroverted or abnormally deep acetabulum. Both types occur on a spectrum and can coexist [65]. However, these anatomical changes may also be asymptomatic, but may lead to other alterations of one's biomechanics about the hip and pelvis and affect the kinetic chain. In asymptomatic NFL and NCAA football, elite hockey, and Army recruits, they have found up to 55% had cam-type, 48% had pincer-type lesions, and 68% were found to have labral injury [103]. Anderson et al studied group of senior athletes, that were asymptomatic, found to have evidence of FAI in 83% of hips of the roughly 1100 x-rayed, 66.7% isolated cam, 8.5% isolated pincer, and 24.8% mixed [104].

Most tears are caused by relatively atraumatic mechanisms such as twisting or pivoting during athletic activity, by chronic degenerative disease, or associated with developmental dysplasia [28, 105], as only about one-third of athletes recall a specific traumatic event as the cause of symptom onset [106].

Athletes commonly present with diffuse, poorly localized groin pain, night pain, pain with pivoting or walking, and mechanical symptoms in the hip and pelvis area; some may present with a painful snapping hip syndrome.

On examination, passive and/or active range of motion may not be limited but pain may be present at the extremes. There are a number of clinical tests that have been reported to reproduce pain, clicking, or locking sensations in the hip and pelvis, specifically they are as follows:

- The impingement test—performed by inducing hip flexion, adduction, and internal rotation, especially in anterior–superior tears (FADIR) [65, 106, 107].
- Passive hyperextension, abduction, and external rotation, particularly with posterior tears [106, 107].
- Acute flexion of the hip with external rotation and full abduction, followed by extension, abduction, and internal rotation (anterior tears) [108].

• Extension, abduction, and external rotation brought to a flexed, adducted, and internally rotated position (posterior tears) [108].

In one study, a clicking sensation of the hip was both sensitive (100%) and specific (85%) in predicting labral tears [65]. On physical examination, the internal rotation/flexion/axial compression maneuver (Scour Test) was 75\% sensitive but only 43% specific [92], and the flexion/abduction/external rotation (FABER) test was found to be 88% sensitive [109].

In 2006, Burnett et al. [110] reported an average time from the initial onset of symptoms to the definitive diagnosis of 21 months (range, 2–156 months; median, 12 months). In addition, an average of 3.3 health-care providers (range, 0–11 health-care providers) had been seen prior to the establishment of a definitive diagnosis. Therefore, clinicians must keep in mind that these physical maneuvers are imperfect and must maintain a high index of suspicion for such injuries.

Although plain radiographs and computed tomography may show hip dysplasia, arthritis, and acetabular cysts in patients with acetabular labrum tears, they cannot be counted as reliable tools for diagnosing the condition itself [106, 107, 111–113]. Even when arthrograms are obtained as well, there does not seem to be an appreciable improvement in the ability of these investigations to detect labral tears [112]. They are, however, useful for excluding other types of hip pathology. MRI, by virtue of its superior soft tissue contrast and ability to directly depict the labrum, has shown more promise in detecting labral tears over the last two decades [96].

The use of MR arthrography to evaluate labral tears in patients yielded a sensitivity of around 80 % [110]. However, in 27 % of the patients who had an arthroscopically verified tear, preoperative MR arthrography failed to detect the lesion. Despite this limitation in sensitivity, this test frequently confirms the diagnosis and reliably rules out other uncommon conditions (e.g., osteonecrosis, stress fracture, neoplasm) that could present with hip symptoms suggestive of labral disease [110].

Conservative treatment is usually tried for at least 6 weeks before definitive surgical intervention. This is done to insure that mechanical symptoms are not due to snapping hip syndrome or other functional pathologies as identified above and that any associated soft tissue injuries are given a chance to heal [64]. A guided steroid injection may also be utilized as a diagnostic and therapeutic procedure to help clarify exact etiology as well.

3.7 Evaluation

In the clinical evaluation of the patient with suspected kinetic chain dysfunction resulting in hip and pelvis pain with associated altered biomechanics, we have found the following schema to be helpful (Fig. 3.6).

The physical examination begins with observation of the athlete both statically and dynamically. One should evaluate the patient in standing, supine, and prone positions, and assess symmetry of the heights of the iliac crests, anterior superior iliac spine (ASIS), posterior superior iliac spine (PSIS), ischial tuberosities, gluteal folds,

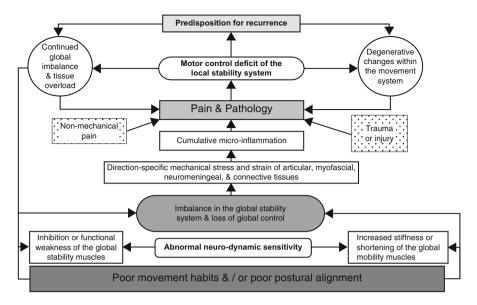


Fig. 3.6 Kinetic chain dysfunction. From Kerger S. Exercise principles. In: Steven J, Karageanes J, editors. Principles of manual sports medicine. Philadelphia: Lippincott, Williams & Wilkins; 2005. p. 65–76; used with permission

and greater trochanters, as well as symmetry of the pubic tubercles, sacral sulci, and inferior lateral angles. Next, determine if there is any leg-length discrepancy. One should realize that true anatomic leg-length discrepancies will generally cause asymmetry and pain, whereas a functional leg-length discrepancy is usually the result of SIJ, hip, or pelvic dysfunction [1].

Leg-length discrepancies allow for an unequal transmission of forces across the spine and pelvis during weight-bearing activities. These forces can be amplified in sport because of the rapid acceleration of body mass or repetitive stress transmitted. Although traditional orthopedic teaching is that a minimum of 1-2 cm difference is essential to cause dysfunction, many in the sports medicine community feel that an anatomically short (or long) leg is clinically relevant and consider differences of as little as 4 mm to be significant [114].

Assess posture for increased lumbar lordosis, which can result from sacrum, pelvis, and/or hip dysfunction. Dynamic observation assesses for any asymmetry during both gait and specific motions characteristic of the patient's sport. SIJ pain, pathology, and restriction may cause a decrease in stride length, leading to a limp or cause reflex inhibition of the gluteus medius, leading to a Trendelenburg gait [1].

The examiner should then look for decreases in both passive and active range of motion of the entire spine, hips, pelvis, knees, and feet. Perform the HLR test as previously described. If pain with motion testing occurs, the patient should specifically identify the area of pain and the clinician should then perform a thorough examination of the relevant structures. A neurologic examination for radiculopathy should also be conducted, in addition to evaluating core strength and overall flexibility [1].

There have been numerous functional (motion) and provocative (painproducing) tests reported in the literature; however, none has consistently been shown to reliably diagnose hip and pelvic joint dysfunction [115-119]. We feel that there are two major flaws in how these studies and others like them have been carried out. Dreyfuss et al. [120] assumed that pain production is an essential pre-requisite to dysfunction. We would suggest that hip and pelvic dysfunction can be diagnosed on the basis of motion restriction and tissue texture changes, especially in chronic pain syndromes when pain location can vary greatly because of muscle imbalance and other factors. Also hip and pelvis screening tests should always be followed up with segmental motion testing and tissue palpation. When these tests are used together with a thorough history to create a clinical picture, they become significantly more reliable [119]. A detailed discussion of the numerous tests described for dysfunction is beyond the scope of this review, but the reader is referred to several excellent sources (see Chap. 2: Physical Examination of the Hip and Pelvis) [63, 121-123]. In osteopathic medicine, hip and sacrum joint somatic dysfunction is diagnosed primarily by the standing and seated flexion tests and asymmetry of pelvic and sacral bony landmarks [63].

3.7.1 Functional Diagnostic Imaging

There is no specific gold standard imaging test to diagnose hip and pelvis dysfunction, largely because of the location and complexity of the joint and the associated structures that may make visualization difficult. However, a variety of normal radiographic indices have been described to differentiate normal from abnormal bony anatomy and these play an important role in understanding why some patients develop instability (see Chap. 5: Radiology of Hip Injuries).

3.8 Principles of Treatment

Recurrent hip and pelvis dysfunction and altered postural alignment should provide clue to the physician for the diagnosis of chronic neuromuscular dysfunction or postural imbalance related to kinetic chain dysfunction. Gravitational strain results in a systemic neuromuscular response of postural musculature and muscle firing patterns related to chronic gravitational stress. Other findings may include chronic or recurrent sprains/strains, pseudoparesis, articular dysfunction, myofascial trigger points, muscle imbalance, and ligamentous laxity. Gravitational stress, an obligatory consequence of bipedal posture, is a constant and a greatly underestimated systemic stressor [56, 124]. It is most important that one understands that postural imbalance is a systemic neuromuscular dysfunction.

Initial treatment in hip and pelvis dysfunction must focus on the re-education of the neuromuscular system. This is partially accomplished by seeking optimization of posture and can be achieved through the use of one or more of the following modes of physical manipulation [125]:

- Contoured orthotics worn in the shoes to optimize foot and lower extremity biomechanics
- A flat orthotic of sufficient thickness to level the sacral base
- Joint manipulation and/or mobilization directed to restore resilience to soft tissues and motion of restricted joint segments
- Daily practice of a therapeutic posture for 20 min to counter the bias of soft tissues reflective of the initial posture

During the implementation of the above, a principle-centered, functional rehabilitation program [125] that focuses first on the stretching of tight, hypertonic postural muscles, strengthening of weak phasic muscles, and proprioceptive retraining must be carried out [126–129]. It is critical to remember that muscle imbalances must be eliminated and coordinated movement patterns returned to normal before strengthening of the core can begin effectively (see Chap 10: Functional Therapeutic and Core Strengthening).

3.9 Case Report: Conclusion

Principles of sequencing the exercise prescription:

- 1. Normalization of segmental function through manual medicine and bodywork as clinically indicated
- 2. Sensorimotor balance retraining
- 3. Comprehensive, symmetric, flexibility
 - a. Stretch to symmetry
 - b. Go for overall increase in length
- 4. Re-educate movement patterns-PRECISION
 - a. Normalize firing patterns-slow, precise, minimalist movements
 - b. Quality of movement (neuromotor control) over quantity
 - c. Start unloaded, progressive load, sports specific movements
- 5. Strengthening
- 6. Sport-specific conditioning

B.L., our 18-year-old college third baseman, subsequently underwent arthroscopic debridement of the labrum, and his hip and low back pain dramatically improved with no subsequent evidence of the previously noted dysfunctional movement patterns.

This case illustrates the important contribution of hip labral pathology and the resultant transverse plane motion loss in the hip causing recurrent hip, low back, and pelvis pain. The patient's motion loss on functional examination, as well as recurrent biomechanical abnormalities partially responsive to conservative management, is an excellent demonstration of a dysfunctional kinetic chain pattern which resolved once the primary pathology was identified and treated.

3.10 Summary

So, as sports medicine clinicians, when should we suspect kinetic chain dysfunction? Some common signs are as follows:

- · Abnormal muscle firing sequences on muscle testing
- Poor proprioception
- Need for frequent manual medicine or manipulation
- "Weak" phasic muscles on exam
- Easy fatigability of phasic muscles
- Chronic musculoskeletal pain
- Progressive postural decline
- Symptoms of tendinopathy
- Poor "core" strength

While each one of these elements may not be considered relevant on its own, when you see several of these findings together you should consider a thorough functional biomechanical examination with emphasis on identifying kinetic chain issues and treating them in an integrated manner.

References

- Brolinson PG, Kozar AJ, Cibo G. Sacroiliac joint dysfunction in athletes. Curr Sports Med Rep. 2003;2:47–56.
- DeLee JC, Farney WC. Incidence of injury in Texas high school football. Am J Sports Med. 1992;20(5):575–80.
- Gomez E, DeLee JC, Farney WC. Incidence of injury in Texas girls' high school basketball. Am J Sports Med. 1996;24(5):684–7.
- Marshall SW, Corlette J. Fall sports qualifying report: 2004–2009 academic years. Datalys NCAA Injury Surveillance Program Center for Sports Injury Research and Prevention: Indianapolis, IN. December, 2009.
- 5. Datalys Center Sports Injury Research and Prevention. http://www.datalyscenter.org/fact-sheets/.
- Morelli V, Espinoza L. Groin injuries and groin pain in athletes: part 1. Prim Care Clin Office Pract. 2005;32:163–83.
- Lovell G. The diagnosis of chronic groin pain in athletes: a review of 189 cases. Aust J Sci Med Sport. 1995;27(3):76–9.

- 8. Westlin N. Groin pain in athletes from Southern Sweden. Sports Med Arthrosc Rev. 1997;5:280–4.
- 9. Koh J, Dietz J. Osteoarthritis in other joints (hip, elbow, foot, ankle, toes, wrist) after sports. Clin Sports Med. 2005;24:57–70.
- Olsen O, Vingard E, Koster M, Alfredsson L. Etiologic fractions for physical work load, sports and overweight in the occurrence of coxarthrosis. Scand J Work Environ Health. 1994;20(3):184–8.
- Kujala UM, Marti P, Kaprio J, et al. Occurrence of chronic disease in former top-level athletes. Predominance of benefits, risks or selection effects? Sports Med. 2003;33(8):553–61.
- Spector TD, Harris PA, Hart DJ, Cicuttini FM, Nandra D, Etherington J, Wolman RL, Doyle DV. A radiologic survey of the hips and knees in female ex-athletes and population controls. Arthritis Rheum. 1996;39(6):988–95.
- Lindberg H, Roos H, Gardsell P. Prevalence of coxarthrosis in former soccer players. 286 players compared with matched controls. Acta Orthop Scand. 1993;64(2):165–7.
- Lane NE, Bloch DA, Jones HH, et al. Long-distance running, bone density, and osteoarthritis. JAMA. 1986;255(9):1147–51.
- Konradsen L, Hansen EM, Sondergaard L. Long distance running and osteoarthrosis. Am J Sports Med. 1990;18(4):379–81.
- 16. Shepard GJ, Banks AJ, Ryan WG. Ex-professional association footballers have an increased prevalence of osteoarthritis of the hip compared with age matched controls despite not having sustained notable hip injuries. Br J Sports Med. 2003;37(1):80–1.
- Drawer S, Fuller C. Propensity for osteoarthritis and lower limb joint pain in retired professional soccer players. Br J Sports Med. 2001;35:402–8.
- Lequesne MG, Dang N, Lane NE. Sport practice and osteoarthritis of the limbs. Osteoarthritis Cartilage. 1997;5(2):75–86.
- 19. Schmitt H, Brocai DR, Lukoschek M. High prevalence of hip arthrosis in former elite javelin throwers and high jumpers: 41 athletes examined more than 10 years after retirement from competitive sports. Acta Orthop Scand. 2004;75(1):34–9.
- Vingard E, Sandmark H, Alfredsson L. Musculoskeletal disorders in former athletes. A cohort study in 114 track and field champions. Acta Orthop Scand. 1995;66(3):289–91.
- 21. Weir DR, Jackson JS, Sonnega A. National Football League Player Care Foundation Study of Retired NFL Players. Institute for Social Research, University of Michigan; 2009.
- 22. Vingard E, Alfredsson L, Malchau H. Osteoarthrosis of the hip in women and its relation to physical load at work and in the home. Ann Rheum Dis. 1997;56(5):293–8.
- Lane NE, Hochberg MC, Pressman A, et al. Recreational physical activity and the risk of osteoarthritis of the hip in elderly women. J Rheumatol. 1999;26(4):849–54.
- Buckwalter JA, Lane NE. Athletics and osteoarthritis. Am J Sports Med. 1997;25(6):873–81.
- Gorsline RT, Kaeding CC. The use of NSAIDs and nutritional supplements in athletes with osteoarthritis. Clin Sports Med. 2005;24:71–82.
- Kujala UM, Kaprio J, Sarna S. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. BMJ. 1994;308(6923):231–4.
- 27. Leunig M, Casillas MM, Hamlet M, et al. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. Acta Orthop Scand. 2000;71(4):370–5.
- McCarthy JC, Noble PC, Schuck MR, et al. The Otto E. Aufranc award: the role of labral lesions to development of early degenerative hip disease. Clin Orthop. 2001;393:25–37.
- Kerger S. Exercise principles. In: Steven J, Karageanes J, editors. Principles of manual sports medicine. Philadelphia: Lippincott, Williams & Wilkins; 2005. p. 65–76.
- Wertheimer LG, Lopes SD. Arterial supply of the femoral head: a combined angiographic and histological study. J Bone Joint Surg. 1971;53A:545–56.
- Brand RA, Pedersen DR, Davy DT, et al. Comparison of hip force calculations and measurements in the same patient. J Arthroplasty. 1994;9:45–51.
- 32. Anderson K, et al. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521-33.

- Pink M, Perry J, Houglum PA, et al. Lower extremity range of motion in the recreational sport runner. Am J Sports Med. 1994;22:541–9.
- Montgomery III WH, Pink M, Perry J. Electromyographic analysis of hip and knee musculature during running. Am J Sports Med. 1994;22:272–8.
- 35. Sahrmann SA. Diagnosis and treatment of movement impairment syndromes. St. Louis: Mosby; 2002.
- Panjabi MM, White AA. Biomechanics in the musculoskeletal system. 1st ed. Philadelphia: Churchill Livingstone; 2001.
- 37. Gajdosik RL. Passive extensibility of skeletal muscle: review of the literature with clinical implications. Clin Biomech. 2001;16(2):87–101.
- Gombatto SP, Collins DR, Sahrmann S, et al. Gender differences in pattern of hip and lumbopelvic rotation in people with low back pain. Clin Biomech. 2006;21:263–71.
- Philippon MJ. The role of arthroscopic thermal capsulorrhaphy in the hip. Clin Sports Med. 2001;20(4):817–29.
- Bellabarba C, Sheinkop MB, Kuo KN. Idiopathic hip instability. An unrecognized cause of coxa saltans in the adult. Clin Orthop. 1998;355:261–71.
- 41. Shindle MK, Ranawat AS, Kelly BT. Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. Clin Sports Med. 2006;25:309–26.
- 42. Crowninshield RD, Johnston RC, Andrews JG, et al. A biomechanical investigation of the human hip. J Biomech. 1978;11:75–85.
- 43. Vleeming A, Lee D, Ostgaard HC, et al. An integrated model of "joint" function and its clinical application. Presented at the 4th Interdisciplinary World Congress on Low Back and Pelvic Pain. Montreal, Canada; November 8–10, 2001.
- Hodges PW, Richardson CA. Feedforward contraction of transverses abdominis is not influenced by the direction of arm movement. Exp Brain Res. 1997;114:362–70.
- 45. Richardson CA, Snijders CJ, Hides JA, et al. The relation between the transversus abdominis muscles, sacroiliac joint mechanics, and low back pain. Spine. 2002;27:399–405.
- 46. Leinonen V, Kankaanpaa M, Airaksinen O, et al. Back and hip extensor activities during trunk flexion/extension: effects of low back pain and rehabilitation. Arch Phys Med Rehabil. 2000;81:32–7.
- 47. Sparto PJ, Parnianpour M, Reinsel TE, et al. The effect of fatigue on multi-joint kinematics, coordination, and postural stability during a repetitive lifting test. J Orthop Sports Phys Ther. 1997;25:3–12.
- 48. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, et al. The posterior layer of the thoracoabdominal fascia: its function in load transfer from spine to legs. Spine. 1995;20:753–8.
- 49. Anderson E, Nilsson J, Thorstensson A. Intramuscular EMG from the hip flexor muscles during human locomotion. Acta Physiol Scand. 1997;161:361–70.
- Anderson E, Oddsson L, Grundstrom H, et al. The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. Scand J Med Sci Sports. 1995;5:10–6.
- Anderson E, Nilsson J, Zhijia M, et al. Abdominal and hip flexor muscle activation during various training exercises. Eur J Appl Physiol. 1997;75:115–23.
- 52. Vleeming A, Stoeckart R, Snijders CJ. The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. Clin Biomech. 1989;4:201–3.
- Vleeming A, van Wingerden JP, Snijders CJ, Stoeckart R. Load application to the sacrotuberous ligament: influences on sacroiliac joint mechanics. Clin Biomech. 1989;4:204–9.
- 54. van Wingerden JP, Vleeming A, Stam HJ, Stoeckart R. Interaction of the spine and legs: influence of the hamstring tension on lumbopelvic rhythm. Second Interdisciplinary World Congress on Low Back Pain. San Diego, CA; November 9–11, 1993.
- 55. Renstrom P, Peterson L. Groin injuries in athletes. Br J Sports Med. 1980;14(1):30-6.
- 56. Kuchera ML. Treatment of gravitational strain pathophysiology. In: Vleeming A, Mooney V, Dorman T, et al., editors. Movement, stability, and low back pain: the essential role of the pelvis. New York: Churchill Livingstone; 1997. p. 477–99.

- 57. Greenman PE. Clinical aspects of the sacroiliac joint in walking. In: Vleeming A, Mooney V, Dorman T, et al., editors. Movement, stability, and low back pain: the essential role of the pelvis. New York: Churchill Livingstone; 1997.
- Kapandji IA. The physiology of the joints. In: Vol 3: the trunk and the vertebral column. 2nd ed. New York: Churchill Livingstone; 1994.
- 59. Irvin RE. Reduction of lumbar scoliosis by use of a heal lift to level the sacral base. J Am Osteopath Assoc. 1991;1:33–44.
- 60. Hoffman K, Hoffman L. Effects of adding sacral base leveling to osteopathic manipulative treatment of low back pain: a pilot study. J Am Osteopath Assoc. 1994;3:217–26.
- Kuchera ML, Jungman M. Inclusion of a Levitor orthotic device in management of refractory low back pain patients. J Am Osteopath Assoc. 1986;10:673.
- 62. Irvin RE. Suboptimal posture: the origin of the majority of idiopathic pain of the musculoskeletal system. In: Vleeming A, Mooney V, Dorman T, et al., editors. Movement, stability, and low back pain: the essential role of the pelvis. New York: Churchill Livingstone; 1997. p. 133–55.
- 63. Greenman PE. Principles of manual medicine. 2nd ed. Baltimore: Williams & Wilkins; 1996.
- Rankin AT, Bleakley CM, Cullen M. Hip joint pathology as a leading cause of groin pain in the sporting population: a 6-year review of 894 cases. Am J Sports Med. 2015;43(7):1698–703.
- 65. Macintyre J, Johson C, Schroeder EL. Groin pain in athletes. Curr Sports Med Rep. 2006;5:293–9.
- 66. Verrall GM, Slavotinek JP, Barnes PG, et al. Diagnostic and prognostic value of clinical findings in 83 athletes with posterior thigh injury: comparison of clinical findings with magnetic resonance imaging documentation of hamstring muscle strain. Am J Sports Med. 2003;31(6):969–73.
- Ekstrand J, Gillquist J. Soccer injuries and their mechanisms: a prospective study. Med Sci Sports Exerc. 1983;15(3):267–70.
- Nielsen AB, Yde J. Epidemiology and traumatology of injuries in soccer. Am J Sports Med. 1989;17(6):803–7.
- 69. Ekstrand J, Gillquist J. The avoidability of soccer injuries. Int J Sports Med. 1983;4(2):124-8.
- 70. Tyler TF, Nicholas SJ, Campbell RJ, et al. The association of hip strength and flexibility with the incidence of adductor muscle strains in professional ice hockey players. Am J Sports Med. 2001;29(2):124–8.
- Holmich P, Uhrskou P, Ulnits L, et al. Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. Lancet. 1999;353(9151):439–43.
- 72. Tyler TF, Nicholas SJ, Campbell RJ, et al. The effectiveness of a preseason exercise program to prevent adductor muscle strains in professional ice hockey players. Am J Sports Med. 2002;30(5):680–3.
- 73. Broadhurt N. Iliopsoas tendinitis and bursitis. Aust Fam Physician. 1995;24(7):1303.
- Emery CA, Meeuwisse WH. Risk factors for groin injuries in hockey. Med Sci Sports Exerc. 2001;33:1423–33.
- 75. Nicholas SJ, Tyler TF. Adductor muscle strains in sport. Sports Med. 2002;32:339-44.
- Hoelmich P. Adductor-related groin pain in athletes. Sports Med Arthrosc Rev. 1997;5:285–91.
- 77. Fricker PA. Management of groin pain in athletes. Br J Sports Med. 1997;31:97–101.
- Johnston CA, Wiley JP, Lindsay DM, et al. Iliopsoas bursitis and tendinitis. A review. Sports Med. 1998;25(4):271–83.
- Fricker PA, Taunton JE, Ammann W. Osteitis publis in athletes. Infection, inflammation or injury? Sports Med. 1991;12(4):266–79.
- Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. Am J Sports Med. 1995;23(6):702–5.
- Kujala UM, Orava S, Karpakka J, et al. Ischial tuberosity apophysitis and avulsion among athletes. Int J Sports Med. 1997;18(2):149–55.

- Renstroem AF. Groin injuries: a true challenge in orthopedic sports medicine. Sports Med Arthrosc Rev. 1997;5:247–51.
- Koch RA, Jackson DW. Pubic symphysitis in runners. A report of two cases. Am J Sports Med. 1981;9(1):62–3.
- Williams JG. Limitation of hip joint movement as a factor in traumatic osteitis pubis. Br J Sports Med. 1978;12(3):129–33.
- Paletta Jr GA, Andrish JT. Injuries about the hip and pelvis in the young athlete. Clin Sports Med. 1995;14(3):591–628.
- 86. Roos HP. Hip pain in sport. Sports Med Arthrosc Rev. 1997;5:292-300.
- 87. Rolf C. Pelvis and groin stress fractures: a cause of groin pain in athletes. Sports Med Arthrosc Rev. 1997;5:301–4.
- Clough TM. Femoral neck stress fracture: the importance of clinical suspicion and early review. Br J Sports Med. 2002;36:308–9.
- Egol KA, Koval KJ, Kummer F, Frankel VH. Stress fractures of the femoral neck. Clin Ortho Relat Res. 1998;348:72–8.
- Canale ST, Beaty JH. Pelvic and hip fractures. In: Rockwood Jr CA, Wilkins KE, Beaty JE, et al., editors. Rockwood and Green's fractures in adults. 4th ed. Philadelphia: Lippincott-Raven; 1996. p. 1109–47.
- Beaty JH. Pelvis, hip and thigh. In: Sullivan JA, Anderson SJ, editors. Care of the young athlete. Rosemont: American Academy of Orthopedic Surgeons and American Academy of Pediatrics; 2000. p. 365–76.
- 92. Boyd KT, Peirce NS, Batt ME. Common hip injuries in sport. Sports Med. 1997;24(4):273-88.
- 93. Patterson I. The torn acetabular labrum. J Bone Joint Surg Br. 1957;39:306-9.
- 94. Narvani AA, Tsiridis E, Kendall S, et al. A preliminary report on prevalence of acetabular labrum tears in sports patients with groin pain. Knee Surg Sports Traumatol Arthrosc. 2003;11(6):403–8.
- Groh MM, Herrera J. A comprehensive review of hip labral tears. Curr Rev Musculoskelet Med. 2009;2:105–17.
- 96. Narvani AA, Tsiridis E, Tai CC, et al. Acetabular labrum and its tears. Br J Sports Med. 2003;37:207–11.
- Takechi H, Nagashima H, Ito S. Intra-articular pressure of the hip joint outside and inside the limbus. J Jpn Orthop Assoc. 1982;56:529–36.
- Ferguson SJ, Bryant JT, Ganz R, et al. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech. 2000;15:463–8.
- Konrath GA, Hamel AJ, Olsen SA, et al. The role of the acetabular labrum and the transverse acetabular ligament in load transmission in hip. J Bone Joint Surg Am. 1998;80:1781–7.
- 100. Ganz R, Parvizi J, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;412:112–20.
- Crawford JR, Villar RN. Current concepts in the management of femoroacetabular impingement. J Bone Joint Surg Br. 2005;87-B:1459–62.
- Philippon MJ. Arthroscopy for the treatment of femoroacetabular impingement in the athlete. Clin Sports Med. 2006;25:299–308.
- Harris JD, Frank JM, Erickson BJ, Slikker III W, Salata MJ, Nho SJ. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systemic review. Arthroscopy. 2015;31(6):1199–204.
- 104. Anderson LA, Anderson MB, Kapron A, Aoki SK, Erickson JA, Chrastil J, Grijaiva R, Peters C. Radiographic abnormalities common in senior athletes with well-functioning hips but not associated with osteoarthritis. Clin Orthop Relat Res. 2016;474(2):342–52.
- 105. Ikeda T, Awaya G, Suzuki S, et al. Torn acetabular labrum in young patients. Arthroscopic diagnosis and management. J Bone Joint Surg Br. 1988;70(1):13–6.
- 106. Klaue K, Durni CW, Ganz R. The acetabular rim syndrome: a clinical presentation of dysplasia of the hip. J Bone Joint Surg Br. 1991;73:423–9.

- 107. Leunig M, Werlen S, Ungersbock A, et al. Evaluation of the acetabulum labrum by MR arthrography. J Bone Joint Surg Br. 1997;79:230–4.
- 108. Fitzgerald RH. Acetabular labrum tears. Diagnosis and treatment. Clin Orthop. 1995;311:60-8.
- Laorr A, Greenspan A, Anderson MW, et al. Traumatic hip dislocation: early MRI findings. Skeletal Radiol. 1995;24(4):239–45.
- 110. Burnett RSJ, Della Roca GJ, Prather H, et al. Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg. 2006;88:1448–57.
- 111. Hase T, Ueo T. Acetabular labral tear: arthroscopic diagnosis and treatment. Arthroscopy. 1999;15:138-41.
- 112. Hofmann S, Tschauner C, Urban M, et al. Clinical and radiological diagnosis of lesions of the labrum of the hip. Orthopade. 1998;27:681–9.
- 113. Czerny C, Hofmann S, Neuhold A, et al. Lesions of the acetabular labrum: accuracy of MR arthrography in detection and staging. Radiology. 1996;200:225–30.
- 114. Waters PM, Millis MB. Hip and pelvic injuries in the young athlete. Clin Sports Med. 1988;7(3):513–26.
- 115. Hochschuler SH. The spine and sports. Philadelphia: Hanley and Belfus; 1990.
- 116. Slipman CW, Sterenfeld EB, Chou LH, et al. The predictive value of provocative sacroiliac joint stress maneuvers in the diagnosis of sacroiliac joint syndrome. Arch Phys Med Rehabil. 1998;79:288–92.
- 117. Van der Wurff P, Hagmeijer RHM, Meyne W. Clinical tests of the sacroiliac joint: a systematic methodological review. Part I: reliability. Man Ther. 2000;5:30–6.
- 118. Van der Wurff P, Hagmeijer RHM, Meyne W. Clinical tests of the sacroiliac joint: a systematic methodological review. Part II: validity. Man Ther. 2000;5:89–96.
- 119. Cibulka MT, Koldehoff R. Clinical usefulness of a cluster of sacroiliac joint tests in patients with and without low back pain. J Orthop Sports Phys Ther. 1999;29:83–92.
- 120. Dreyfuss P, Dreyer S, Griffin J, et al. Positive sacroiliac screening tests in asymptomatic adults. Spine. 1994;19:1138–43.
- 121. Ward RC, Jerome JA, Jones III JM. Foundations for osteopathic medicine. 2nd ed. Baltimore: Lippincott Williams & Wilkins; 2002.
- 122. Magee DJ. Orthopedic physical assessment. 3rd ed. Philadelphia: WB Saunders; 1997.
- Morelli V, Espinoza L. Groin injuries and groin pain in athletes: part 2. Prim Care Clin Office Pract. 2005;32:185–200.
- 124. Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br. 1999;81(2):281–8.
- 125. Kuchera ML. Gravitational stress, musculoligamentous strain, and postural alignment. Spine: State of the Art Reviews. 1995;9:463–90.
- 126. Brolinson PG, Gray G. Principle-centered rehabilitation. In: Garrett WE, Kirkendall DT, Squire DH, editors. Principles and practice of primary care sports medicine. Philadelphia: Lippincott Williams and Wilkins; 2001. p. 645–52.
- 127. Jones PS, Tomski MA. Exercise and osteopathic manipulative medicine: the Janda approach. PM&R: State of the Art Reviews. 2000;14:163–79.
- 128. Comerford MJ, Mottram SL. Functional stability re-training: principles and strategies for managing mechanical dysfunction. Man Ther. 2001;6:3–14.
- 129. Schlink MB. Muscle imbalance patterns associated with low back problems. In: The spine in sports. Los Angeles: Robert & Watkins; 1996.

Chapter 4 Gait Assessment

Adam M. Pourcho, Sean Colio, and Jimmy D. Bowen

Clinical Pearls

- The understanding of gait and its effects on the athlete is paramount to the diagnosis, treatment, rehabilitation, and eventual return to activity following injury.
- While walking and running are endeavors of all athletic activities, efficient walking and running is not necessarily universally present. Recognition of subtle differences in normal and abnormal gait is key to a complete orthopedic assessment.
- With any injury of the lower extremity, the entire kinetic chain must be taken into account. This usually starts at the feet and extends to the hip, pelvis, and spine.
- Understanding the variations that occur in gait with age and between genders will help sports clinician promote the continued athletic performance and participation of the patient.

4.1 Case Presentation

4.1.1 Chief Complaint

A 22-year-old female long distance runner presents with right anterior knee pain and complains of right foot placement changes during running.

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[©] Springer International Publishing Switzerland 2017 P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_4

4.1.2 History of Present Illness

She has been a competitive runner for 8 years, at times running up to 60–80 miles a week. During the last 8 years, she has had a number of bilateral lower extremity injuries including patellofemoral knee pain, iliotibial band friction syndrome, plantar fasciitis, peroneal tendonitis, right groin pain, and sacroiliac joint dysfunction. These ailments were resolved previously with relative rest, nonsteroidal antiinflammatory agents (NSAIDS), and manual physical therapy. About 2 years ago, she began insidiously catching her right foot on her left heel while running, occasionally falling to the ground. After relative rest and treatment, she had been able to return to running but has continued to have problems regarding right foot placement and right peri-patellar knee pain. She has no symptoms with other activities, such as activities of daily living or cross training athletic activities, including elliptical trainers and road biking. The persistent right foot catching, peri-patellar right knee pain, and inability now to push off correctly have affected her to the point that she has not run in the last 3 months.

4.1.3 Review of Systems

She denies any neurological symptoms, neck or back problems, and is currently not taking any medications except for occasional NSAIDS. She denies a mechanism of injury, mechanical symptoms in the knee, or effusion. She has no history of internal derangement or previous ligamentous injury to her knee.

4.1.4 Physical Examination

Her physical examination reveals normal spinal alignment and range of motion without pain. Her pelvic and spinal static alignment demonstrates no imbalances. She demonstrates frontal plane genu valgus of less than 10° bilaterally that are symmetric (Table 4.1 and Fig. 4.1). During relaxed double stance, she has 9° more frontal plane rear foot eversion (calcaneal valgus) on the right versus the left. She had a positive dynamic valgus during single leg squat (Fig. 4.2a). While walking, she demonstrates more right hip transverse plane internal rotation and more right foot pronation than on the left. During running, there was an increase of hip internal rotation and adduction with increased genu valgus, as well as a heavy landing at heel strike, with a rear foot strike pattern. Interestingly, the right foot supinated during plantar flexion of stance phase with internal rotation and forefoot transverse plane adduction during push off, causing her foot to catch. She compensated for this by circumduction of her right lower extremity so that she could clear her foot during swing phase. This caused her to be off balanced during running and she consistently

Region	Sagittal	Frontal/coronal	Transverse/rotational	
Foot	Toe flex/ext	Pronation/supination	Adduction/abduction	
Ankle	Plantar flexion Dorsiflexion	Varus/valgus		
Tibia			Internal/external torsion	
Knee	Flex/extension	Varum/valgum		
Femur			Internal/external rotation	
Hip	Flex/extension	Adduction/abduction		
Pelvis	Ant/post tilt	Elevated/depressed	Rotation	
Spine	Lordosis/kyphosis	Lateral scoliosis	Rotational scoliosis	

Table 4.1 Anatomic plane classification

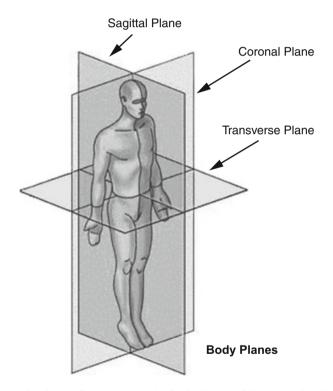


Fig. 4.1 Anatomic planes of the human body. Sagittal plane divides body into left and right. Coronal/frontal plane divides it into front and back or anterior and posterior. Transverse/axial plane divides body into cranial and caudal portions. This picture is in the *public domain* in the USA

lunged to the right. Her hip, knee, and ankle ranges of motion were normal and symmetric. She was noted to have adductor tightness on the right compared to the left. She was also noted to have decreased strength on manual muscle testing of the right hip abductors and external rotators (Fig. 4.2b). The remainder of her neurological and vascular examination was normal.



Fig. 4.2 (a) Patient has a positive dynamic valgus test. Notice the internal rotation of the femur resulting in valgus at the knee (*arrow*). (b) Resisted strength testing for the hip external rotators

4.1.5 Testing

Radiographic images of her lumbar spine, hips and pelvis, and knees were all normal. Scanogram of her pelvis to ankles demonstrated no anatomic leg length discrepancy. An electrodiagnostic study (EDX) of her right lower extremity and lumbar spine was normal.

4.2 Gait Assessment

Individual anthropometric differences, turn over speeds, stride lengths, arm swing, and endurance, all contribute to the overall performance and success of the athlete. Correct treatment of the athlete with a lower extremity or spine injury begins with an assessment of his/her gait. Walking and running are two of the most obvious and fundamental actions of life. It is important for the sports medicine clinician to appreciate the complexities of gait, the determinates of an efficient gait, the differences in gait relative to age and gender, the importance of symmetry in gait, and the identification of impairments and their functional impact on athletic performance [1]. The ability of the sports practitioner to visualize and discern different gait patterns is key to understanding the complexities of gait, can give insight into possible injury mechanisms, and provides possible rehabilitative avenues for a successful return to sport [2].

4.3 The Elements of Gait

An analysis of gait begins with understanding the components of the gait cycle. A single gait cycle has been defined as the period from initial heel contact until the initial heel contact on the ipsilateral leg [3]. Walking is the act of falling forward and catching oneself [4]. The tasks of walking and running involve forward propulsion, which is accomplished by an inverted pendulum gait. This is accomplished when the body is vaulted over a stationary limb with each step, moving continuously in the direction of travel. This involves each limb in turn either advancing forward or providing support to the contralateral advancing limb. The assessment of these activities takes time, practice, and technical skill [4]. It begins with an understanding of the definitions, phases, and determinants of gait.

Each foot has two main phases during the gait cycle: a *stance phase* where the foot is in contact with the ground and *swing phase* where the foot is off the ground [5]. When allowed to walk at a self-selected walking speed, stance phase is around 60% of the gait cycle, while swing phase accounts for approximately 40% [5]. During walking, one foot is always in contact with the ground. It involves periods of single limb support (SLS) and double limb support (DLS). Running, on the other hand, involves periods of flight in which neither foot is in contact with a surface, called *flight phase* (Fig. 4.3). Walking and running demonstrate a reversal of the percentage of stance and swing phase. It is therefore intuitive that the slower the



Fig. 4.3 Two of three runners in flight phase. Picture by Kevin Lewter

Initial contact/loading response	Initial double support stance phase beginning with initial contact. Some literature may include initial contact as separate phase of cycle			
Mid-stance	First half of single support representing the time the opposite limb leaves the floor until body weight is aligned over the forefoot			
Terminal stance	Second half of single support representing the time the opposite limb makes contact with the floor and the body weight moves ahead of the forefoot			
Push off	Late stance when there is an ankle plantar flexion moment advancing the limb into swing phase. Some literature references include this as part of pre-swing phase			
Pre-swing	Final double support representing the time of initial contact of the contralateral limb to ipsilateral toe-off			
Initial swing	Initial third of the swing phase representing the time from toe-off to when the swing limb foot is opposite the stance limb			
Mid-swing	Middle third of swing, time the swing foot is opposite the stance limb to when the tibia is vertical			
Terminal swing	Final third of the swing phase, time from tibia being vertical until initial contact			

Table 4.2 Normal gait cycle

walking speed, the longer the time spent in DLS. Conversely, faster walking speeds result in less time spent in DLS and running involves no DLS phase and includes a *flight phase*.

The basic unit of walking and running is the gait cycle; also known as stride. The gait cycle involves a pattern of acceleration and deceleration which is controlled by contraction of muscles. It is important to remember that the entire body is involved in the gait cycle, with movements occurring in the cardinal planes simultaneously (Fig. 4.1) It also involves functional and temporal variables [2]. The functional aspects of the gait cycle are weight acceptance and support during the stance and swing phases [2]. There are eight phases of the gait cycle beginning with the initial contact of the loading response and ending with terminal swing before the next initial contact (Table 4.2 and Fig. 4.4a-h) [3]. It is important to understand the temporal-spatial gait parameters including the difference between stride time and step time. Stride time is defined as initial contact on one limb to initial contact on ipsilateral limb and step time is defined as initial contact on one limb to initial contact on the contralateral limb. The stride length is the distance covered during one stride and gait velocity is determined by dividing the stride distance by the time taken. Walking speed is the distance travelled per unit of time. Cadence is the number of steps taken in a decided timeframe [1-3, 6].

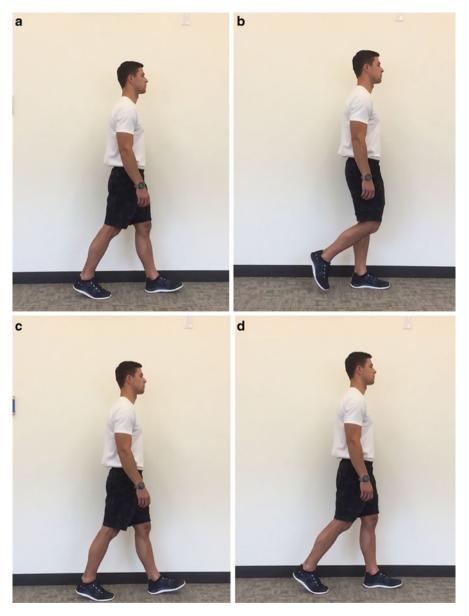


Fig. 4.4 (a) Initial contact on the right foot, the beginning of the loading response. (b) Mid-stance on the right foot. (c) Terminal stance on the right foot. (d) Push off, beginning of pre-swing on the right foot

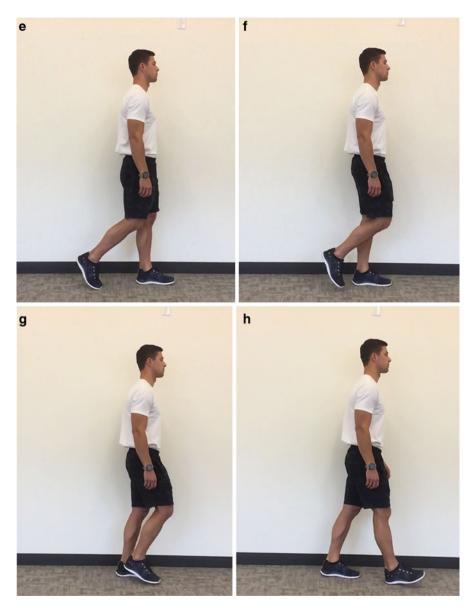


Fig. 4.4 (continued) (e) Pre-swing on the right foot. (f) Initial swing on the right foot. (g) Mid-swing on the right foot. (h) Terminal swing on the right foot

4.4 Kinematics of Gait

In order to analyze gait, it is important to have a good understanding of the kinematics of gait. Kinematics is the science of motion. In athletic movement, it is the study of positions, angles, velocities, and accelerations of individual body segments and joints during movement. The kinematics of gait of the individual during walking or running is evaluated by observing the body as composed of body segments (links) and joints (connections) between segments. Body segments, for the purposes of describing human motion, are considered to be rigid bodies. These include the foot, leg (shank), thigh, pelvis, thorax, hand, forearm, upper arm, and head. Joints or links between segments include the ankle (both talocrural and subtalar joints), knee, hip, wrist, elbow, and shoulder. The kinematics of the individual or system is determined by taking into account the orientation and position of each segment. Position describes the relative location of the body segment or joint space. Each body segment has a center of mass (COM) or center of gravity (COG). Furthermore, the whole system, the body, also has a COM or COG. As segments move, their positions in time and space affect the balance and energy use of the system as the whole body COM distribution changes [7]. Kinematics is best evaluated through quantitative and not qualitative means. Three-dimensional (3D) gait analysis provides information relative to degrees of movement of the joint and relationships to the body segments in terms of frontal, sagittal, and transverse arrangements. The flexion-extension, abduction-adduction, and internal-external rotation relationship of the segments and joints are evaluated, as well as the side to side, front to back symmetry within an individual. The COM for the segments and the COM of the body are expressed in terms of vertical, anterior to posterior, and medial to lateral relationships [8, 9]. The body's COM reaches its highest point in stance when the speed is minimal [10]. During normal walking, for example, the maximums of COM and height are during double stance phase. 8 During running, however, the maximums of COM and height are during flight at maximum velocity [10].

4.5 Muscle Function During Gait

The muscles play a pivotal role in energy conservation and joint movement in normal gait. Electromyographic studies have demonstrated that during running and walking most muscle activity occurs at the start and end of swing phase [11]. This would suggest that the main function of the muscles during gait is to accelerate and decelerate the body. The remainder of the energy expenditure is contributed by passive forces through the limbs and joints [12].

4.6 Walking Gait

4.6.1 Stance Phase

Because the stance phase in walking is around 60 % of the gait cycle, there are two periods of DLS, when both feet are in contact with the ground at the same time [8]. This occurs once at the beginning and once at the end of stance phase.

At initial contact, the knee extensor and flexor muscles contract simultaneously to decelerate the limb and correctly position it before accepting weight. The hip extensors also eccentrically contract to slow the forward movement of the leg [11].

During loading response, the ankle dorsiflexors contract eccentrically at heel strike, again slowing down the limb and forward momentum of the body [2, 12]. The gluteus medius will contract isometrically to stabilize the pelvis and femur [12].

As the COM reaches its highest point during mid-stance, the gluteus medius and minimus muscles contract isometrically to stabilize the pelvis and femur, preventing pelvic drop against gravity [11, 12]. Also during mid-stance, the gastrosoleus complex contracts eccentrically stabilizing the foot and ankle [11, 12]. Many investigations have demonstrated that weakness or inhibition of the hip abductors and external rotators is associated with patellofemoral disorder (PFD) and knee pain, as well as destabilization of the femur in space [13–17]. Therefore, athletes suffering from PFD need a comprehensive rehabilitation program that stresses hip abductor strengthening. In late stance, the plantar flexors contract concentrically to propel the body forward in preparation for toe off and swing phase [11, 12]. The fibularis (peroneus) longus and brevis also contract concentrically to transfer weight from lateral to medial, again in preparation for toe off [18]. Just prior to toe off, the hip flexors contract again, this time concentrically, to unload the pelvis and prepare it for forward propulsion [11, 12].

The ligaments of the hip play a role in stabilization during normal gait. The iliofemoral, ischiofemoral, and pubofemoral ligaments all act to limit medial rotation of the femur during gait. In stance, the COG passes behind the center of rotation of the hip joint, causing the three ligaments to become taut and allowing static stance without supporting muscular contraction [19]. If the mobility of the hip is reduced, there will be an increased moment of the ipsilateral knee, contralateral hip, and the lumbar spine to compensate, possibly leading to injuries in these areas [20].

4.6.2 Swing Phase

During the pendulum motion of the non-weight bearing leg, most of the lower limb muscles are physiologically inactive [11, 12]. At the start of swing phase, the ankle dorsiflexors will contract concentrically to allow clearance of the foot as the leg swings freely forward [4]. Weakness of dorsiflexors results in a foot slap gait, resulting in increased energy expenditure as the individual compensates with hip hiking or excessive hip and knee flexion, to achieve foot clearance during swing phase [21].

At terminal swing, the muscles contract eccentrically to decelerate the leg and prepare for weight acceptance. The hamstrings contract eccentrically slowing both hip flexion and knee extension [12, 22].

4.7 Running Gait

During running, the amount of time in stance and swing phase is inverted compared to walking, with approximately 40% of time spent in stance phase. Furthermore, it includes a *flight phase* in which both feet are airborne twice in each gait cycle [23]. While runners may run at different rates, running consists of periods of acceleration and deceleration similar to walking gait. During late flight, the quadriceps and hip flexors contract eccentrically to prepare the limb for ground contact and shock absorption during heel strike [11, 23]. This helps stabilize the knee by restraining the posterior movement of the tibia during knee flexion. The hamstrings and hip extensors contract concentrically to extend the hip during the lateral half of swing phase and the first half of stance phase [23]. The hamstrings also contract eccentrically to slow the limb down just prior to heel strike and initial contact [12]. Acute hamstring strains are most likely to occur when the hamstring is lengthened, during eccentric contraction, in the terminal swing phase of the gait cycle [24-27]. The gastrosoleus complex and hamstrings have important concentric and eccentric functions, while the knee extensors function concentrically during running. Similar to walking gait, the ankle dorsiflexors concentrically contract during swing to provide clearance for the foot and contract eccentrically during initial contact to control lowering of the forefoot [8, 11, 23].

4.7.1 Energy Consumption

During normal walking, energy is consumed in three different ways [28]. First, there is the energy consumption of moving the entire body mass through the desired distance and time. Second, there is the energy consumption due to the work of moving the trunk up and down with each step. Third, there is the general basal body metabolism. There is an optimal speed that varies for each individual, where the combined metabolic rate is most efficient for travel [28]. It is also logical that the faster one moves, the more energy one consumes.

4.8 Kinetics of Gait

In order to completely understand and analyze gait the practitioner must have an understanding of the *kinetics of gait* [2]. *Kinetics* is the study of forces acting on bodies to cause motion [3]. In order to understand kinetics, one must understand

Newton's three lows of motion; a body will change velocity only if a force is applied to it; the change in velocity is proportional to the force; and a force applied to an object will result in an equal and opposite reaction. This means that for every force there is a reaction force that is equal in size, but opposite in direction [2]. In the context of gait analysis, the expression of the forces exerted on the foot during contact with the ground called the *ground reaction force* (GRF). While the body is exerting a force on the ground via gravity and body weight, the ground is exacting an equal and opposite force against the body.

The GRF is determined by three directions perpendicular to one another: horizontal, side to side, and vertical forces are taken into account. The center of the distribution of these forces is called the *center of pressure* (COP). Based on the COP, the net force vector of the GRFs affecting each body segment can be determined. The perpendicular distance between the GRF and each body segment defines the ultimate moment arm that when multiplied by the applied force creates rotation about each segment's axis. Using inverse dynamics, the forces on each segment can be calculated from the foot through the entire body.

Normal walking is almost effortless. This efficiency is made possible by minimizing the displacement of the body's COM during ambulation [28]. Vertical displacement is far more relevant than lateral displacement when considering optimization of this efficiency [28]. During walking, the COM normally travels along an inverted pendulum with a sinusoidal, vertical (up and down), and horizontal (side to side) path with each step [10]. The COM is normally 5 cm anterior to the second sacral vertebrae and slightly higher in males on average [9]. The vertical and horizontal displacements of the COM describe a figure of eight within a relatively small 5 cm square during walking [9]. Vertical displacement is defined as change in the height of an individual's head as it lowers during weight acceptance and unloading, and heightens during stance [29, 30]. A series of maneuvers described by Saunders and colleagues, the six determinants of gait, are the mechanism by which the body minimizes the COM displacement during walking [29] (Table 4.3). Without the combined actions of the determinants of gait, the average total vertical displacement of the COM would double causing an increase in energy expenditure and a decrease in gait efficiency [29].

1. Pelvic rotation	Pelvis rotates alternatively to the right and to the left in relation to the line of progression in transverse plane about a vertical axis
2. Pelvic tilting	Pelvis tilts downward during <i>swing phase</i> (on swing leg) along the frontal plane and around the sagittal axis. (Maximal tilt is at mid-swing)
3. Knee flexion (stance phase)	Knee flexion in stance phase functions as shock absorber, minimalizes displacement of COM, and decreases energy expenditure
4. and 5. Foot and knee mechanism	Function synergistically to allow maximal extremity length in the early stance phase and maintenance of COM in late stance phase
6. Lateral displacement of COG	Acts to shift COM over the weight bearing extremity in the horizontal plane

Table 4.3 The determinates of gait

4.9 The Six Determinants of Gait

1. *Pelvic rotation in the transverse/axial plane:* Decreases the drop in COM during double limb support. The pelvis rotates anteriorly 4° during swing and posterior 4° during stance phase. This results in a decrease in the amplitude of displacement along the COM's path. The thorax and pelvis rotate in opposite directions, to maintain balance of COM. Weakness or inflexibility in these core areas will increase the amplitude of displacement of the COM and cause less efficient progression of gait and increased expenditure of energy [7, 10]. During pelvic rotation, the other joints of the lower limb are involved in rotation as well. The greater the joint's distance from the trunk, the greater is the rotation (i.e., the tibial rotation is three times the rotation of the pelvis) [10].

2. *Pelvic tilting in the frontal/coronal plane*: Slight pelvic obliquity reduces the peak COM during single limb support. During swing phase the hip of the swing limb is lowered, the knee is flexed, and the ankle is dorsiflexed to allow toe clearance of the swing leg. Flexion of the lower limb joints and lowering of the hip keeps the COM from moving up and down less than 5 cm (2 in.) during normal gait [2, 23, 29].

3. Flexion of the knee during stance phase: At initial contact the knee is ideally flexed $0^{\circ}\pm 5^{\circ}$. At loading response just after heel strike, the knee begins to flex $15^{\circ}-20^{\circ}$. This knee flexion during stance phase provides a shock-absorbing mechanism at the beginning of stance phase, reduces the height of COM and thereby reduces the energy expenditure that would be present if one had to stop and restart the gait cycle [29].

4. and 5. *The interchange of knee, ankle, and foot motions*: The three rockers of sagittal plane foot movement occur at heel strike, during foot-flat, and at toe off. Dorsiflexion of the foot at heel strike produces a net lengthening of the leg to maximize the step length. The eccentrically contracting ankle dorsiflexors serve to provide shock absorption during foot-flat, serving to reduce the length of the leg until the pelvis passes in front of the ankle [1]. The plantar flexors concentrically contract during toe off increasing the leg length thus, limiting the amount pelvic drop. Inability to flex or lower the hip or flex the knee or ankle will be seen during the qualitative evaluation as *circumduction* of the relatively longer limb during swing phase or *vaulting* during stance phase. This results in a noticeable increase in vertical displacement of the head (bobbing) and a decrease in gait efficiency [29].

6. Lateral pelvic shift in the frontal/coronal plane: The pelvis is laterally displaced over the stance leg during each step. This balances the COM of the trunk over the stance limb. Furthermore, this results in alignment of the tibia into a vertical position during stance. There is, however, a net loss of energy during this determinant of gait secondary to the slight upward movement of the body. This is a necessary step in the gait process providing balance in bipedal gait. The relative moment of lateral displacement can be altered secondary to changes in foot placement and relative weakness of the hip abductors [29].

4.10 Age Differences in Gait

Gait differences are seen between different ages and genders. The *determinants of gait* change within an individual depending on age, segment and joint maintenance, health, strength, and flexibility. From 8 to 10 months of age most normal developing children begin to walk by supporting themselves with stationary objects. The two major skills of balance and support are slowly improved as the child progresses towards supported walking [31]. The normal determinants of gait are seen between 8 and 45 years of age, but, may be seen as young as 3 years old and well into the eighth decade in some individuals [29].

Two times during each gait cycle both hips simultaneously lift during midstance. Toddlers are more variable from step to step than older children and adults [31]. The recovery of mechanical energy of the COM in each gait cycle is similar for older children and adults [32]. However, when compared to normal adult ambulation, children demonstrated greater angles of hip flexion during walking [33]. Moreover, there are also significant differences in knee and ankle patterns when comparing normal child gait to normal adult gait [31, 33]. During normal adult gait, the hip vaults over the stance leg like an inverted pendulum. The inverted pendulum model accurately predicts the general pattern of mechanical energy fluctuations of the body during walking and optimal walking speed [34]. The sinusoidal hip oscillations seen in adults are lacking in toddlers but are developing in children [33]. Furthermore, the forward energy and displacement of the COM in toddlers is usually irregular, due to the lack of coordination of muscle contractions and joint movement [33]. Newly locomotive toddlers do not implement the classic inverted pendulum mechanism, but they do develop this over the first few months of ambulation as muscle coordination and strength for unsupported walking improve. In summary, the unique features of supported walking in children which distinguish it from adult ambulation include: increased hip flexion, decreased knee extension, increased ankle dorsiflexion, decreased walking speed, increased variability, and decreased smoothness of gait.

As adults age, it is not unusual to observe decreased walking speed and step length. In fact, many of the gait alterations observed in aging patients can be attributed to the overall reduction in walking speed [35]. There is evidence that physiological changes associated with aging contribute to these observations [36–39]. Several changes are noted such as: reduced peak hip extension, increased anterior pelvic tilt, and reduced ankle plantar flexion and power generation [35]. Neuromuscular changes such as weakness of ankle dorsiflexors, ankle plantar flexors, knee extensors, hip flexors, and hip extensors have all been suggested as reasons for reduction in both the stride length and cadence seen with aging [40–42].

Falls in the aging population have many contributing factors such as loss of balance and visual, vestibular, cognitive, and sensory disturbances. Falls can also occur in the absence of these factors. A dramatic decrease in ankle power

output during terminal stance phase represents a consistent finding in age and impaired gait. This limits gait speed, length of steps, and may cause variable stride characteristics that may be linked to falls in adults [43]. Although current literature has failed to pinpoint a single contributing factor between fallers and non-fallers, physiologic changes seen in aging patients such as increased peak external hip flexion moment in stance phase, reduced peak hip extension moment, reduced knee flexion moment in pre-swing, and reduced knee power absorption in pre-swing have been implicated as potential causative factors [44].

4.11 Male and Female Gait

Relative weakness and reduced ability to stabilize the hip, pelvis, and trunk are reported in female athletes when compared to age matched male counterparts [45–49]. This theoretically results in reduction of endurance of side bridging activity and decreased hip abduction and external rotation isometric strength. Females may be more vulnerable to large external forces experienced during athletics, especially in the transverse and frontal planes. Therefore, females may be predisposed to excessive motion in the hip and trunk compared to men, potentially permitting their entire lower extremity to move into positions frequently associated with non-contact injuries. Multiple gait analysis studies have established that females display greater hip internal rotation and adduction during athletic tasks (Fig. 4.5) [50–52]. This functional positioning has been demonstrated to be associated with increased injury in retrospective and cross sectional studies [53].

For example, female athletes have a 2.3–9.7 times higher life time incident of anterior cruciate ligament (ACL) tears than males [54]. Electrodiagnostic testing has revealed that significant differences in neuromuscular control between males and females may explain this observation [55]. When analyzing gait, females had overall decreased activation of the vastus medialis obliques along with decreased and earlier activation of the biceps femoris, resulting in an overall quadriceps heavy landing [54, 55]. ACL injury prevention and neuromuscular strengthening programs have reported at 24–82% reduction in ACL injuries and a 50% reduction in ACL injury risk [54, 56]. Consequently, athletes and coaches should consider incorporating neuromuscular training into their regular training routine.

4.12 Case Assessment and Treatment

The female runner in the case presentation demonstrated weakness of the proximal stabilizers of the pelvis with a positive standing dynamic valgus (Fig. 4.2a, b) resulting in an abnormal running pattern. Relative weakness of the external rotators and abductors of the right hip placed the right hip in a more internally rotated and



Fig. 4.5 (a) Female and male walking (*front*). Note the relative hip internal rotation and adduction on the stance leg in our female athlete. (b) Female and male walking (*back*). Note the relatively lower COM and the relative increase of hip internal rotation and adduction on the stance leg in our female athlete

adducted position during running. This resulted in a lateral pull on her patella and increased patellofemoral knee pain. It has been well demonstrated that weakness of hip external rotators is the primary contributor to peri-patellar knee pain [13, 14, 16]. Furthermore, this athlete demonstrated a heavy heel strike gait pattern, which has been shown to increase a dynamic knee valgus moment during running, likely contributing to her anterior knee pain [57].

Initially, the athlete was held out of running and was given a routine of open and closed chain exercises designed to improve the functional motion and stability of her spine, pelvis, and hips through all plans of movement. Then she was placed on a series of exercises that emphasized concentric, isometric, and eccentric progressive strengthening in a functional fashion needed to reduce her proximal muscle weakness. This exercise routine specifically targeted the gluteus medius and hip external rotators. After 8 weeks of three to five times a week of conditioning, she was released to a progressive return to running program. During her video running analysis, she was initially started on a treadmill. Her pace cadence was increased by 10 % using a metronome to promote a faster turn around and shorter stride, thereby, making her land more on her mid-foot instead of her heel [58]. For example, if her cadence was 100 steps per minute at pace she was made to run 110 steps per minute at pace using a metronome to pace her. Once she could run consistently on a treadmill, she was

converted to track running. As long as she continued to keep her hip abductors and core strong and ran more on her mid-foot, her impaired movement pattern did not return. She is currently running without difficulty or anterior knee pain.

4.13 Summary

Gait evaluation is an essential part of the assessment of the injured athlete. The implementation of an efficient and effective gait analysis into clinical practice takes time and patience. Through systematic understanding of the anatomy, neuromuscular interaction, kinematics, and kinetics involved in human locomotion, a more comprehensive clinical evaluation can be achieved. An understanding of differences in gait, both between genders and age, can help provide a more accurate diagnosis and lead to a more effective treatment for the athlete.

References

- 1. Pease W, Bowyer B. Human walking. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.
- Kerrigan D, Croce U. Gait analysis. 1st ed. New York: McGraw-Hill Medical Publishing Division; 2005.
- 3. Kaufman K, Sutherland D. Kinematics of normal human walking. 3rd ed. Philadelphia: Lippincott, Williams & Wilkins; 2006.
- 4. Magee D. Assessment of gait. 6th ed. Philadelphia: Elsevier; 2013.
- Shumway-Cook A, Woollacoot M. Motor control: translating research into clinical practice. 4th ed. Philadelphia: Lippincott, Williams & Wilkins; 2011.
- 6. Olney S. Gait. 5th ed. Philadelphia: F.A. Davis Company; 2011.
- 7. Birrer R, Buzermanis S, DellaCorte M. Biomechanics of running. 1st ed. New York: McGraw Hill; 2001.
- 8. Novacheck T. The biomechanics of running. Gait Posture. 1998;7(1):77-95.
- 9. Adams J, Perry J. Gait analysis: clinical applications. 1st ed. Baltimore: Lippincott, Williams & Wilkins; 1994.
- Inman V, Ralston H, Todd F. Human locomotion. 1st ed. Baltimore: Lippincott, Williams & Wilkins; 1994.
- Boakes J, GT. R. Muscle activity during walking. 3rd ed. Philadelphia: Lippincott, Williams & Wilkins; 2006.
- Anderson F, Pandy M. Individual muscle contributions to support in normal walking. Gait Posture. 2003;17(2):159–69.
- Souza R, Powers C. Differences in hip kinematics, muscle strength, and muscle activation between subjects with and without patellofemoral pain. J Orthop Sports Phys Ther. 2009;39(1):12–9.
- Noehren B, Pohl M, Sanchez Z, Cunningham T, Lattermann C. Proximal and distal kinematics in female runners with patellofemoral pain. Clin Biomech (Bristol, Avon). 2012;27(4):366–71.
- 15. Heiderscheit B. Lower extremity injuries: is it just about hip strength? J Orthop Sports Phys Ther. 2010;40(2):39–41.
- 16. Khayambashi K, Mohammadkhani Z, Ghaznavi K, Lyle M, Powers C. The effects of isolated hip abductor and external rotator muscle strengthening on pain, health status, and hip strength

in females with patellofemoral pain: a randomized controlled trial. J Orthop Sports Phys Ther. 2012;42(1):22–9.

- Pal S, Besier T, Beaupre G, Fredericson M, Delp S, Gold G. Patellar maltracking is prevalent among patellofemoral pain subjects with patella alta: an upright, weightbearing MRI study. J Orthop Res. 2013;31(3):448–57.
- 18. Mueller M. The ankle and foot complex. 5th ed. Philadelphia: F.A. Davis Company; 2011.
- 19. Levangie P. The hip complex. 5th ed. Philadelphia: F.A. Davis Company; 2011.
- Sadeghi H, Sadeghi S, Allard P, Labelle H, Duhaime M. Lower limb muscle power relationships in bilateral able-bodied gait. Am J Phys Med Rehabil. 2001;80(11):821–30.
- Jamshidi N, Rostami M, Najarian S, Menhaj M, Saadatnia M, Salami F. Differences in center of pressure trajectory between normal and steppage gait. J Res Med Sci. 2010;15(1):33–40.
- Sutherland D, Olshen R, Cooper L, Woo S. The development of mature gait. J Bone Joint Surg Am. 1980;62(3):336–53.
- Novacheck T. Walking, running, and sprinting: a three-dimensional analysis of kinematics and kinetics. Instr Course Lect. 1995;44:497–506.
- 24. Puranen J, Orava S. The hamstring syndrome--a new gluteal sciatica. Ann Chir Gynaecol. 1991;80(2):212–4.
- Thelen D, Chumanov E, Sherry M, Heiderscheit B. Neuromusculoskeletal models provide insights into the mechanisms and rehabilitation of hamstring strains. Exerc Sport Sci Rev. 2006;34(3):135–41.
- 26. Ali K, Leland J. Hamstring strains and tears in the athlete. Clin Sports Med. 2012;31(2):263-72.
- 27. Orchard J. Biomechanics of muscle strain injury. NZ J Sports Med. 2012;30:92-8.
- Rose J, Morgan D, Gamble J. Energetics of walking. 3rd ed. Baltimore: Lippincott, Williams & Wilkins; 2005.
- 29. Sauders J, Inman V, Eberhart H. The major determinants in normal and pathological gait. J Bone Joint Surg Am. 1953;35-A(3):543–58.
- 30. Nuber G. Biomechanics of the foot and ankle during gait. Clin Podiatr Med Surg. 1989;6(3):615-27.
- 31. Johnson E, Spiegel M. Ambulation problems in very young children. JAMA. 1961;175:858-63.
- 32. Cavagna GA, Franzetti P, Fuchimoto T. The mechanics of walking in children. J Physiol. 1983;343:323–39.
- Statham L, Murray M. Early walking patterns of normal children. Clin Orthop Relat Res. 1971;79:8–24.
- Ivanenko Y, Dominici N, Lacquaniti F. Development of independent walking in toddlers. Exerc Sport Sci Rev. 2007;35(2):67–73.
- Kerrigan D, Todd M, Della-Croce U, Lipsitz L, Collins J. Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments. Arch Phys Med Rehabil. 1998;79(3):317–22.
- DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. J Appl Physiol (1985). 2000;88(5):1804–11.
- Judge J, Davis R, Ounpuu S. Step length reductions in advanced age: the role of ankle and hip kinetics. J Gerontol A Biol Sci Med Sci. 1996;51(6):M303–12.
- McGibbon C, Krebs D. Age-related changes in lower trunk coordination and energy transfer during gait. J Neurophysiol. 2001;85(5):1923–31.
- Riley P, DellaCroce U, Kerrigan D. Effect of age on lower extremity joint moment contributions to gait speed. Gait Posture. 2001;14(3):264–70.
- Bendall M, Bassey E, Pearson M. Factors affecting walking speed of elderly people. Age Ageing. 1989;18(5):327–32.
- Chang R, Dunlop D, Gibbs J, Hughes S. The determinants of walking velocity in the elderly. An evaluation using regression trees. Arthritis Rheum. 1995;38(3):343–50.
- 42. McGibbon C. Toward a better understanding of gait changes with age and disablement: neuromuscular adaptation. Exerc Sport Sci Rev. 2003;31(2):102–8.
- Hausdorff J, Rios D, Edelberg H. Gait variability and fall risk in community-living older adults: a 1-year prospective study. Arch Phys Med Rehabil. 2001;82(8):1050–60.

- 44. Kerrigan D, Lee L, Nieto T, Markman J, Collins J, Riley P. Kinetic alterations independent of walking speed in elderly fallers. Arch Phys Med Rehabil. 2000;81(6):730–5.
- 45. Leetun D, Ireland M, Willson J, Ballantyne B, Davis I. Core stability measures as risk factors for lower extremity injury in athletes. Med Sci Sports Exerc. 2004;36(6):926–34.
- 46. Bohannon R. Reference values for extremity muscle strength obtained by hand-held dynamometry from adults aged 20 to 79 years. Arch Phys Med Rehabil. 1997;78(1):26–32.
- Cahalan T, Johnson M, Liu S, Chao E. Quantitative measurements of hip strength in different age groups. Clin Orthop Relat Res. 1989;246:136–45.
- McGill S, Childs A, Liebenson C. Endurance times for low back stabilization exercises: clinical targets for testing and training from a normal database. Arch Phys Med Rehabil. 1999;80(8):941–4.
- 49. Nadler S, Malanga G, DePrince M, Stitik T, Feinberg J. The relationship between lower extremity injury, low back pain, and hip muscle strength in male and female collegiate athletes. Clin J Sports Med. 2000;10(2):89–97.
- Ferber R, Davis IM, Williams 3rd DS. Gender differences in lower extremity mechanics during running. Clin Biomech (Bristol, Avon). 2003;18(4):350–7.
- 51. Lephart S, Ferris C, Riemann B, Myers J, Fu F. Gender differences in strength and lower extremity kinematics during landing. Clin Orthop Relat Res. 2002;401:162–9.
- 52. Malinzak R, Colby S, Kirkendall D, Yu B, Garrett W. A comparison of knee joint motion patterns between men and women in selected athletic tasks. Clin Biomech (Bristol, Avon). 2001;16(5):438–45.
- Ireland M, Willson J, Ballantyne B, Davis I. Hip strength in females with and without patellofemoral pain. J Orthop Sports Phys Ther. 2003;33(11):671–6.
- Micheo W, Hernández L, Seda C. Evaluation, management, rehabilitation, and prevention of anterior cruciate ligament injury: current concepts. PM R. 2010;2(10):935–44.
- Hewett T, Zazulak B, Myer G, Ford K. A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes. Br J Sports Med. 2005;39(6):347–50.
- 56. Steffen K, Emery C, Romiti M, Kang J, Bizzini M, Dvorak J, et al. High adherence to a neuromuscular injury prevention programme (FIFA 11+) improves functional balance and reduces injury risk in Canadian youth female football players: a cluster randomised trial. Br J Sports Med. 2013;47(12):794–802.
- Lieberman D, Venkadesan M, Werbel W, Daoud A, D'Andrea S, Davis I, et al. Foot strike patterns and collision forces in habitually barefoot versus shod runners. Nature. 2010;463(7280):531–5.
- Heiderscheit B, Chumanov E, Michalski M, Wille C, Ryan M. Effects of step rate manipulation on joint mechanics during running. Med Sci Sports Exerc. 2011;43(2):296–302.

Chapter 5 Radiology of Hip Injuries

Donald J. Flemming and Eric A. Walker

Clinical Pearls

- It is extremely important to provide the interpreting radiologist with a detailed and accurate history in order to optimize the opportunity to recognize subtle abnormalities.
- Do not focus on just the hip joint and femoral head when interpreting plain radiographs of the hip. The search pattern should also include the sacroiliac joints, lumbar spine, pubic symphysis, obturator foramen, and the adjacent soft tissues.
- If intraarticular pathology of the hip is suspected, magnetic resonance imaging (MRI) should be performed on a high field MRI (3 Tesla) with small field of view imaging
- Magnetic resonance arthrography (MRA) is the preferred method of assessment of cartilage and labral pathology of the hip.
- Stress fractures present on MR as a linear band of signal replacing the normal bone marrow.
- If femoroacetabular impingement (FAI) is suspected, radiography should include an AP view of the pelvis and cross-table lateral view of the symptomatic hip.

5.1 Case Presentation

Athletic injuries of the hip and groin are not as common as those of the extremities but are important to diagnose and treat accurately because they can be associated with prolonged rehabilitation times and significant disability [1]. Imaging plays an

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P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_5

important role in the accurate diagnosis of these injuries but should not supplant a careful history and physical examination. Many factors must be considered prior to requesting a radiologic study including the age of the patient, duration, type and location of symptoms, and likely source of injury because the optimal exam may be different if the injured tissue is bone, muscle, cartilage, or tendon.

A physician evaluating an injured athlete has a tremendous advantage over a treating provider compared to 30 years ago because of the relatively recent explosion of cross-sectional imaging techniques including ultrasound (US), computed tomography (CT), and, most importantly, magnetic resonance imaging (MRI). It is vital that the referring clinician understands the advantages and limitations of these various modalities to ensure that the most cost-effective and accurate diagnosis can be rendered. The importance of providing a detailed and accurate history to the interpreting radiologist cannot be over-emphasized.

5.2 Modalities

5.2.1 Radiography

Despite the proliferation of advanced imaging modalities, the radiograph continues to be an important examination for the evaluation hip and groin pain [2]. This study is frequently referred to as a plain film but there is nothing simple about this examination despite its humble moniker. In fact, significant findings may be underappreciated by clinicians who are unaware of recently described subtle radiographic manifestations of important sources of hip pain such as femoroacetabular impingement. The advantages of radiography include high spatial resolution, relative low cost, high specificity, and wide availability.

It is absolutely paramount that the examination be properly exposed and positioned. The proper examination of the hip should include an anteroposterior (AP) view of the pelvis and a lateral view of the hip. The AP view of the pelvis allows for side to side comparison which may aide in the detection of subtle pathology. Some authors recommend that the AP view be obtained in the weight bearing position and although there are potential advantages to this approach, no study has been performed to confirm the superiority of this technique over standard radiography. Regardless, the femurs should be internally rotated to optimally evaluate the femoral neck on the AP examination (Fig. 5.1a). There are two options for lateral radiography. The frog leg lateral view (Fig. 5.1b) is preferred for general diagnostic evaluation of the hip but a cross-table lateral view (Fig. 5.1c) should be obtained if femoroacetabular impingement is a clinical concern.

An organized approach to the evaluation of the radiograph increases the likelihood of detection of pathology. The interpreter must remember to include the sacroiliac joints, lumbar spine, pubic symphysis, obturator foramen, and soft tissues in their scan pattern and not to focus on just the femoral head and hip joints.

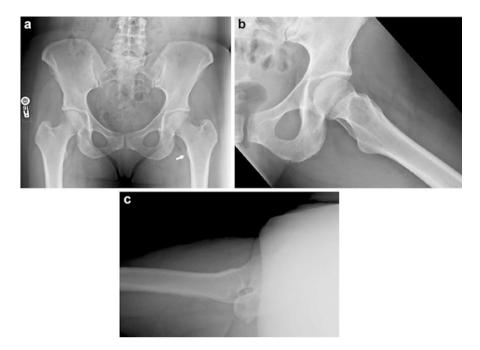


Fig. 5.1 (a) Normal AP view of the pelvis. The curves formed by joint surface of the femoral head and the acetabulum are parallel. The femurs are internally rotated as indicated by the fact that the lesser trochanter (*white arrow*) is barely visible. (b) Normal frog leg lateral view of the hip. (c) Normal cross-table lateral view of the hip

5.2.2 Ultrasound

The availability of ultrasound (US) scanners in sports medicine practice is rapidly expanding allowing both diagnositic imaging and guidance for therapeutic interventions. The technique requires that the operator be knowledgeable and the learning curve can be quite steep. US is best used in the assessment of soft tissue disorders such as muscle and tendon pathology. For example, the diagnosis of a snapping iliopsoas tendon is rapidly and easily confirmed with ultrasound.

5.2.3 Computed Tomography

Computed tomography (CT) is most commonly used to evaluate suspected or known fractures. However, the development of helical CT in modern scanners has led to the ability to perform reconstructed images in multiple planes. Complex oblique images are now obtainable on-the-fly on many workstations. Some investigators have taken advantage of this capability and have shown that CT arthrography can yield useful information in patients that are not suitable for MR evaluation regardless of the reason.

It is extremely important that referring clinicians understand that radiation dose is not insignificant with this modality. Techniques not requiring radiation such as MR should be considered before CT particularly in young patients.

5.2.4 Magnetic Resonance Imaging

The introduction of clinical magnetic resonance imaging (MR) has revolutionized the field of musculoskeletal imaging and has improved our understanding of injuries to the athlete. Although expensive and time-consuming, MR offers a global evaluation of the hip including assessment of bone, cartilage, muscle, and tendons. The two most critical aspects leading to successful diagnosis of pathology are the quality of the examination and the experience of the interpreter.

Not all MR examinations or scanners are equal and it is extremely important that the referring clinician has an open line of communication with the radiologist to ensure that the appropriate examination is done with the appropriate equipment. One consideration is often whether the patient should be scanned on an open low field magnet or a closed high field system. In general, if the question is fracture or muscle injury, either a low field or high field system will yield equivalent results. However, if the question pertains to intraarticular pathology such as cartilage defects or labral tears, then the examination should be performed on a high field system (ideally 3 Tesla) with a small field of view.

5.2.5 Magnetic Resonance Arthrography

Magnetic resonance arthrography (MRA) is the preferred examination for the assessment of cartilage pathology in the hip joint [3]. Some investigators feel that high quality, small field of view imaging is sufficient for detection of pathology but most radiologists prefer MRA. Diagnoses can be rendered more confidently when the joint is distended by contrast. Although MRA may be superior to non-contrast examination, it does have some negative aspects. The examination is typically performed following the direct administration of contrast into the joint which requires an image guided introduction of a needle into the joint and this can be painful for some patients. One distinct advantage of direct arthrography is that additional diagnostic information can be obtained if anesthetic is injected into the joint at the same time as contrast. Relief of pain with provocative maneuvers following the intraarticular source of pain. Some radiologists advocate the use of indirect arthrography which involves intravenous injection of contrast with the imaging of the joint following

Diagnosis	X-ray	CT	US	MR	MRA
Stress fracture	+++	-	-	+++	-
Acute fracture	+++	++	-	++	-
Arthritis	+++	-	-	+++	-
Femoroacetabular impingement	+++	++	-	++	+++
Labral tear	-	-	-	+	+++
Tendon injury	+	-	++	+++	-
Muscle injury	+	+	++	+++	-

Table 5.1 Recommended radiology studies

CT computed tomography, US ultrasound, MR magnetic resonance imaging, MRA magnetic resonance arthrography

(-), Not indicated; (+), May be useful; (++), Useful; (+++), Recommended

exercise and a brief delay. While this technique is less painful, it does not distend the joint and is associated with rare but potential allergic reaction to gadolinium contrast.

A summary of recommended examinations is provided in Table 5.1.

5.3 Radiologic Diagnosis of Hip Pain in Athletes

5.3.1 Osseous

Bone is an amazing organ system that develops based on a combination of genetic and physical influences. However, despite its remarkable mechanical and physiologic properties, bone may fail in response to excessive acute or chronic repetitive forces.

5.3.2 Stress Fracture

Stress fractures are a result of excessive force being applied to bone in a chronic repetitive fashion. The normal physiologic response to new stress to a bone is for remodeling to occur but if the osteoblastic response is outpaced by removal of bone by osteoclasts, the bone can mechanically fail. Stress fracture should be considered in the differential diagnosis of any patient that has recently changed the intensity of their physical activity. In addition to poor physical conditioning, risk factors for development of stress fracture include female gender, Caucasian race, smoking, steroid use, tall/thin physique, and low sex hormones [4, 5]. The basicervical portion of the femoral neck is the classic location for stress fracture to occur in the hip or pelvis but other described locations include the pubic rami, sacrum, superior acetabulum [6], medial femoral diaphysis [7], and even the femoral head [8].



Fig. 5.2 Twenty-five-year-old runner with hip pain and completed stress fracture. (a) AP view of the hip in external rotation. The lesser trochanter (*white arrow*) is in profile indicating external rotation which foreshortens the femoral neck. (b) AP view of the hip in internal rotation. The fracture that was not appreciated in external rotation is now easily seen (*black arrowheads*). Note the linear lucency laterally and the condensation of trabeculae medially

Radiographs are usually the first line modality used to evaluate stress fractures. The appearance of a stress injury to bone will be dependent on the temporal nature of the injury and the bone involved. The initial radiographic examination may be completely normal because 30-50% of bone must be resorbed for a lucency to be appreciated on radiography. It is very important to make sure that the femur is properly positioned for the radiographic examination [2]. The femur should be internally rotated approximately $10-15^{\circ}$ to ensure that the femoral neck is adequately evaluated (Fig. 5.2).

The classic radiographic findings that may be appreciated include sclerosis and periosteal reaction [9]. The initial radiographs may be normal. Sclerosis is usually in a linear pattern transverse to the long axis of the involved bone. On occasion, this sclerotic reaction and periosteal reaction can be exuberant leading to concern for tumor. The linear nature of the abnormality and the patient's history will usually be sufficient to dispel concerns about neoplasm. It is not uncommon for subtle findings to be missed particularly when the abnormality is in an unusual location or at a site that may be obscured by bowel gas such as the sacrum. A high degree of suspicion and careful side-to-side comparison of the AP radiograph of the pelvis will increase the likelihood of detection of a stress injury (Fig. 5.3).

MR of the hip should be ordered when a stress fracture is clinically likely and the radiograph is normal. The importance of early detection has been emphasized because the prognosis is poorer if the patient progresses to a complete or displaced fracture. The MR examination should evaluate the entire pelvis with both T1 and a fluid sensitive sequence such as a fat suppressed T2 weighted or short-tau inversion recovery (STIR) sequence.

Stress fractures present as a linear band of signal replacing normal bone marrow [10]. On T1 weighted sequences, the normal bright signal of fat will be replaced by

Fig. 5.3 Twenty-two-yearold active duty military woman with right hip pain after recent increase in miles run per week. AP view of the pelvis shows subtle band of sclerosis at the inferior basicervical portion of the femoral neck (*black arrows*) indicating stress fracture



a linear band of low signal. On fat suppressed sequences, the normal low signal fat is replaced by bright signal that may or may not surround a thin band of low signal. The MR presentation is usually very characteristic (Fig. 5.4).

Two recently described presentations of stress injuries deserve special mention, namely, thigh splints [7] and subchondral stress fracture of the femoral head [8]. Patients with thigh splints present with either medial sided thigh or groin pain. This injury occurs secondary to abnormal forces at the insertion of the adductor muscles on the medial diaphysis of the femur. Radiographically, thigh splints present as solid periosteal reaction along the medial aspect of the femur. The MR correlate for this plain film finding is high signal on fluid sensitive sequences located medial to the cortex of the diaphysis of the femur. This subtle MR finding can be easily overlooked.

Stress fracture in subchondral bone of the femoral head is a relatively newly described condition that may be confused with avascular necrosis (AVN). This diagnosis should be considered when findings mimicking AVN are seen in the femoral head of an athlete without risk factors for osteonecrosis. A correct diagnosis of subchondral stress fracture in these patients leads to the correct treatment (rest) and the prognosis is generally much better than that of AVN.

5.3.3 Acute Fracture

Acute hip or pelvis fracture is uncommon as a result of acute trauma in athletics even in high-energy contact sports. Children, however, are an exception to this rule because of the inherent weakness at the physis and apophyses of growing bone [11]. Radiography with AP view of the pelvis and orthogonal view of the symptomatic hip is usually sufficient to render a diagnosis. Careful side-to-side comparison between the affected and asymptomatic side helps establishing otherwise diagnoses (Fig. 5.5).

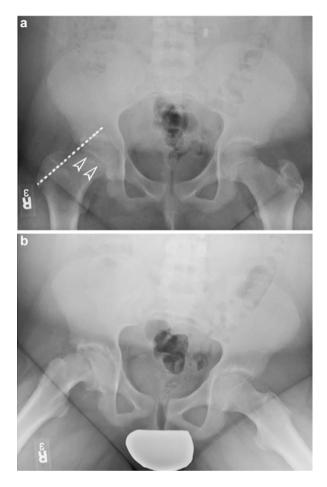


Fig. 5.4 Twenty-one-year-old active duty female with left hip pain after changing physical training regimen. (**a**) AP view of the pelvis shows subtle sclerosis in the left femoral neck (*white arrowheads*). (**b**) Coronal T2 fat saturation sequence clearly demonstrates diffuse edema of the femoral neck (*asterisk*) and medial callus formation (*white arrow*)

Fig. 5.5 Thirteen-year-old boy with left hip pain after being tackled while playing football with fractured left acetabulum. An AP view of the pelvis shows subtle asymmetry in the triradiate cartilage, which is wider on the left (*white arrowheads*) than on the right (*white arrow*) indicating a Salter I fracture of this growth center



Slipped capital femoral epiphysis (SCFE) is an important diagnostic consideration in the pre-adolescent or adolescent athlete. These patients are approximately 11 years of age with risk factors including obesity and black race. SCFE can present bilaterally although it is not common to see both hips symptomatically affected at the same time. Children present with either an acute or chronic history of groin pain. Radiographs are usually abnormal (Fig. 5.6). The involved physis is usually widened and the proximal femoral epiphysis is usually displaced inferiorly and medially relative to the femoral diaphysis [12]. The displacement of the epiphysis is sometimes appreciated on the AP view of the hip if the femoral head is located below a line drawn along the superior border of the femoral neck (Kline's line). This line should normally intersect the superior lateral aspect of the femoral head. A lateral view will confirm the displacement of the femoral epiphysis. MR may be Fig. 5.6 Thirteen-year-old boy with right hip pain and slipped capital femoral epiphysis. (a) An AP view of the pelvis shows subtle widening of the right physis (hollow white arrowheads) in comparison with the left hip. Note the femoral head is below the white dotted line (Klein's line) drawn parallel to the superior aspect of the femoral neck indicating the proximal femoral epiphysis is inferiorly displaced. (b) Frog leg lateral view confirms posterior displacement of the femoral head



useful in the situation when a radiograph is normal in a patient with hip pain at risk for slipped femoral epiphysis. The MR will reveal widening of the physis with increased signal on fluid sensitive sequences in the adjacent metaphysis.

The apophyses about the hip and pelvis are at risk for avulsion and these injuries are being diagnosed with increased frequency as children are exposed to highly competitive athletic activities that require sudden or violent muscle contraction such as soccer, hockey, gymnastics, and sprinting sports [11]. The apophyses at risk include the anterior superior iliac spine (ASIS) (sartorius origin), anterior inferior iliac spine (ASIS) (rectus femoris origin), ischial tuberosity (hamstring origin), ilium (oblique insertion), and lesser trochanter (iliopsoas insertion). Diagnosis is usually readily established with an AP radiograph (Fig. 5.7) although injuries in the ASIS and AIIS may be better appreciated on view of the pelvis obliqued to the side

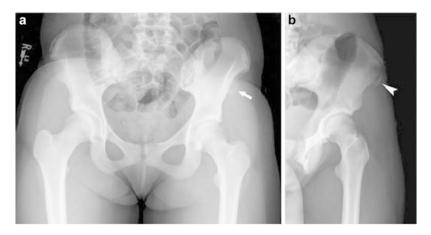


Fig. 5.7 Sixteen-year-old woman with acute onset of pain in left hip and avulsion of the anterior superior iliac spine. (a) AP view of the pelvis shows avulsion of the anterior superior iliac spine (*white arrow*). (b) Avulsion injury is better appreciated on a left posterior oblique view of the left hip (*white arrowhead*)

of injury. Avulsion of an apophysis can be easily missed on MR [13] and may be better appreciated on US or CT if confirmation is required in the setting of a "normal" radiograph.

5.4 Articular Pathology

There is an ever-increasing body of knowledge directed at the early detection of intraarticular injuries of the hip. Although the hip is an intrinsically stable joint, it should be no surprise that cartilage structure of the hip is at risk for tears since contact forces are 3–5 times body weight or higher in athletes. It is very important to remember to evaluate the sacroiliac joints and the pubic symphysis for conditions such as sacroilitis or osteitis pubis. Disease in both of these joints may present as hip or groin pain in the athlete.

5.4.1 Osteoarthritis

Patients with cartilage pathology frequently present with pain that may be associated with locking or clicking. Both the labrum and the articular cartilage are at risk for injury. The labrum is a nerve containing fibrocartilaginous structure that is at the periphery of the joint. Its purpose is to deepen the articular socket and to help maintain the normal negative pressure of the joint. Tears of this structure can occur along its entire length but are most common anterior superiorly and increase the likelihood of damage of the articular surface [2]. Labral tears are associated with the accelerated development of osteoarthritis. While congential and developmental anomalies of the hip are a common cause of early onset osteoarthritis, other potential causes include subclinical joint laxity, history of acetabular or femoral neck fracture, osteonecrosis, prior inflammatory arthritis, and inherited collagen production anomalies.

Osteoarthritis presents radiographically as joint space narrowing with osteophyte formation. Joint space narrowing can be subtle but comparison to the unaffected side can increase detection of subtle alteration in joint dimensions. Joint space narrowing usually occurs at the superolateral aspect of hip. The AP view of the pelvis is usually sufficient for detection of joint space narrowing but assessment of joint dimension should also be performed on lateral views. (Fig. 5.8) Posterior joint space narrowing is associated with a poor clinical prognosis and should not be overlooked on faux views of the acetabulum [14].

Abnormal morphology of the joint, whether from prior slipped capital femoral epiphysis, idiopathic avascular necrosis as a child (Legg–Calve–Perthes Disease) or developmental dysplasia, can lead to accelerated degenerative disease. A surprisingly large number of these conditions can escape detection at an early age and only are recognized when the patient presents with hip pain as an adult.

5.4.2 Developmental Dysplasia

Patients with developmental dysplasia have less osseous coverage of the femoral head than normal patients and are at increased risk for the development of labral tear and early onset osteoarthritis. Developmental dysplasia of the hip (DDH) is frequently detected in infancy but there are subclinical cases that may elude typical screening practices.

Fig. 5.8 Thirty-nine-yearold man with right hip pain and bilateral hip osteoarthritis. AP view of the pelvis shows narrowing of the superolateral right hip joint space with osteophyte formation (*white arrow*). Subtle superolateral joint space narrowing is also seen in the left hip



Radiographic changes of DDH may be very subtle but are extremely important to recognize. Whenever there is poor osseous acetabular coverage of the lateral femoral head, the interpreter of a radiographic examination should consider DDH as a diagnosis. Many measurements have been developed to confirm the radiographic diagnosis of DDH, but the most easily used in clinical practice is the center edge angle of Wiberg [14]. This angle is easily assessed electronically on images in a PACS system. A horizontal line is drawn between the centers of the femoral heads. Perpendicular lines are drawn to this horizontal line extending through the center of the femoral head. A line is then drawn from the center of the femoral head to the lateral margin of the acetabulum. The angle formed between the perpendicular and the lateral margin of the acetabulum is known as the center edge angle and should be between 25–39° in the normal patient [14, 15]. A center edge angle of less than 25° indicates DDH (Fig. 5.9).

5.4.3 Femoroacetabular Impingement

Femoroacetabular impingement (FAI) has recently been recognized as an important source of hip pain [16] but the diagnosis is somewhat controversial. It is critical that all radiologic findings are correlated with physical examination and history to



Fig. 5.9 Thirty-four-year-old man with right hip pain and developmental dysplasia of the hip. AP view of the pelvis shows relative lack of coverage of the lateral aspect of the femoral head that is suspicious for mild developmental dysplasia of the hip (DDH). The center edge angle measures less than 25° confirming DDH

ensure that FAI is the source of the patient's hip pain [17]. Normal motion of the hip leads to impaction of the femoral neck on the acetabulum and labrum. This mechanical impaction leads to labral tears and osteoarthritis. FAI has been separated into two basic types, cam and pincer. Most patients have a combination of both types. Cam impingement is caused by an aspherical femoral head neck junction. The pincer type of impingement is produced by excessive acetabular coverage of the femoral head.

The assessment for FAI begins with the radiograph. A properly positioned AP view of the pelvis and cross-table lateral view are used for primary evaluation [15]. These views are assessed for signs of aspherical femoral head neck junction, acetabular over-coverage, and acetabular rim phenomenon.

The classic sign of cam type of impingement is excessive bone formation at the femoral head neck junction that can be appreciated as flattening of the contour of the superolateral femoral neck on the AP radiograph or of the anterior superior femoral neck on the lateral view. The shape of the femoral head neck junction can assume the shape of a pistol grip as a manifestation of asphericity. The alpha angle is a measurement used to measure asphericity on the cross-table lateral view and is best performed on a PACS workstation. A circle is drawn around the femoral head and a parallel line is drawn through the center of the femoral neck that intersects the center of this circle. A second line is drawn where the circle crosses the cortex of the anterior femoral neck junction. The resulting angle should be less than 55° in a normal patient (Fig. 5.10). Another manifestation of the same phenomenon is femoral offset. This too is measured on the cross-table lateral view. Both of these measurements have poor inter- and intra-observer reliability [18].

Osseous over-coverage of the femoral head is responsible for the pincer type of FAI. Numerous measurements have been devised to assess over-coverage. The simplest measurement is a center edge angle greater than 39° (Fig. 5.11). Subtle forms

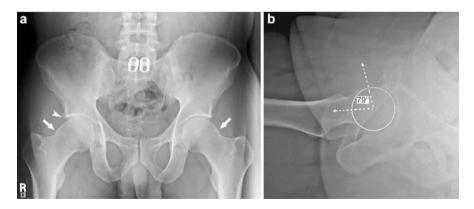


Fig. 5.10 Forty-one-year-old man with left hip pain and cam-type femoroacetabular impingement (FAI). (a) AP view of the pelvis shows bone formation at the superolateral aspect of the femoral neck bilaterally (*white arrows*). Ossification is present in the right acetabular labrum (*white arrowhead*). (b) Cross-table lateral view of the left hip shows asphericity of the femoral head with abnormally wide α [alpha]-angle, which is seen in the cam-type mechanism of FAI

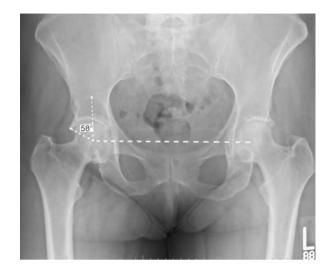


Fig. 5.11 A 48-year-old female with hip pain. Degenerative changes with marginal osteophytes and protrusio acetabuli lead to pincer type femoroacetabular impingement (FAI). In pincer type FAI, acetabular overcoverage limits the range of motion and leads to impaction between the acetabulum and the femur

of pincer impingement can occur particularly involving the anterolateral acetabular rim. All AP radiographs of the pelvis should be assessed for the "cross-over" sign at the anterolateral acetabulum. The anterior acetabular wall should not project beyond the posterior acetabular wall in a properly positioned AP radiograph of pelvis and if it does so, this indicates relative prominence of the anterior lateral acetabulum that can lead to impingement (Fig. 5.12). However, before interpreting a positive crossover sign, one must make sure that the radiograph is properly positioned [15]. The center of the sacrum should be directed at the pubic symphysis to ensure that there is not excessive rotation of the pelvis that may be confounding. Additionally, the distance from the superior aspect of the pubic symphysis should not be about 3.2 cm in a man or 4.7 cm in a female to correctly assess for focal acetabular over-coverage. If the pelvis is too far tilted anteriorly, this distance will widen and be associated with a false positive cross-over sign.

FAI may produce other findings that can be appreciated on radiographs including some manifestations that up until recently were discounted as normal variants. Chronic impaction may lead to separation of the rim from the remainder of the acetabulum producing what has historically been described as the os acetabulum [19] (Fig. 5.13). Impaction may also induce a focal lucency in the subcapital portion of the femoral neck (Fig. 5.14). This juxtaarticular cyst has been called a synovial herniation pit and was initially described as a normal variant to avoid confusion with neoplasm. This juxtaarticular cyst is now recognized as a manifestation of FAI particularly when it is situated in the anterior superior portion of the femoral neck [20]. Finally, chronic impaction may lead to ossification of the labrum itself [21]. This pattern of ossification is different than that of an os acetabulum but the distinction is not that important because both foci of ossification are associated with FAI.

Fig. 5.12 Thirty-six-yearold woman with focal acetabular overcoverage and pincer-type femoroacetabular impingement (FAI). AP view of the pelvis shows anterior wall of the acetabulum (black dotted *line*) crossing over the posterior wall (white solid line) superolaterally. The point of intersection is indicated by the *black* arrowhead. This is the abnormal cross-over sign indicating focal acetabular over coverage

Fig. 5.13 Twenty-nineyear-old man with left hip pain and os acetabulum. An AP view of the pelvis shows an ossicle (*white arrow*) adjacent to the left superolateral acetabulum, which is a secondary sign of femoroacetabular impingement





5.4.4 Labral Tears

Labral tears are now widely recognized as a potential source of hip pain in athletes. Patients present with hip pain that may or may not be associated with clicking or a locking sensation. Patients with DDH are at increased risk of developing labral tears.

Labral tears are best diagnosed using high quality MR arthrography [3] even though the diagnosis may be established by CT arthrography. MR offers a superior assessment of the surrounding soft tissues such as muscle or tendon and therefore may provide important additional information that cannot be obtained by CT. It is

Fig. 5.14 Twenty-sixyear-old woman with hip pain and synovial herniation pit. Cross-table lateral view shows subcortical lucency in the anterior femoral neck (*white arrowheads*) representing a secondary sign of femoroacetabular impingement



important to consult with an experienced hip arthroscopic surgeon to guide the evaluation of patients with potential hip pathology to avoid costly repeat or unnecessary imaging.

Labral pathology can present in many ways on MR including abnormal morphology, abnormal internal signal, and detachment. Paralabral cysts are an important indicator of adjacent labral pathology because they develop from forced extrusion of joint fluid through a defect in the labrum.

5.4.5 Hip Ligaments

Injuries or abnormalities of ligaments and capsule are most commonly recognized in the shoulder and not really frequently considered in the hip because of the inherent stability of the joint. However, subluxation of the hip joint can occur in athletes and tears of the capsule and ligaments of the hip can occur [2]. Subluxation events may also result in disruption of the ligamentum teres. The ligament may be thickened or torn with resulting pain. Diagnosis of capsular or ligamentous pathology is best accomplished with MR. In the acute setting, frank disruption of the capsule or ligament can be seen.

Thickening of the capsule may also be appreciated on MR although the criteria for this diagnosis have not been widely accepted at this time. The cause of capsular thickening is not clear but the finding is more commonly seen in patients with osteo-arthritis. The relationship between capsular thickening and adhesive capsulitis has not been established in the hip but one must wonder if there is a correlate to findings that are more commonly associated with the entity that has been well described in the shoulder.

5.4.6 Tendon

Multiple tendons are present around the hip that may present be a source of chronic or acute hip pain. In general, tendon disease usually is not associated with radiographic findings. An important exception is calcific tendonitis. This entity, also known as hydroxyapatite deposition disease (HADD) is an idiopathic condition that is more commonly seen in the shoulder. However, the deposition of hydroxyapatite crystals into tendons about the hip can be seen and may be associated with pain (Fig. 5.15). Common locations for visualization of HADD near the hip include the soft tissues lateral and superior to the greater trochanter (hip abductors) and the soft tissues posterior to the linea aspera (gluteus maximus).

Tendon degeneration or tear in the hip and groin is best evaluated on MR. The appearance of tendon pathology on MR is dependent on the extent of disease at presentation. Tendons are black on all sequences in the normal situation and the insertion on bone is also black. Acute avulsion or tear of a tendon is readily visible on fluid sensitive sequences (T2 weighted or STIR images) and presents as increased signal (white) at the tendon bone interface. Increased signal may also be seen in the adjacent bone. Chronic avulsion or tear of a tendon can be subtle on imaging but is more readily appreciated if the injury is accompanied by heterotopic bone formation and retraction and atrophy of the involved muscle.

Fig. 5.15 Forty-two-yearold woman with left hip pain and calcific tendinopathy of the hip abductors. AP view of the left hip shows a calcification in the hip abductor tendon immediately superior to the greater trochanter (*white arrow*)



In some cases, tendon degeneration, also known as tendinopathy, may be associated with chronic pain in an athlete. Chronic disease in a tendon manifests as enlargement and internal increased signal on MR. Chronic degeneration of a tendon can easily be overlooked on MR and in fact, many cases of "trochanteric bursitis" actually represent tendinopathy of the hip abductors [22].

5.4.7 Snapping Tendons

On occasion, a tendon may be responsible for a snapping sensation of the hip. If the snapping occurs in the groin, the iliopsoas tendon may be snapping over an osseous prominence on the superior pubic ramus. The tensor fascia lata may snap over the greater trochanter and produce lateral symptoms. Both conditions are readily confirmed with ultrasound [23]. Iliopsoas snapping can be confirmed and successfully treated with fluoroscopic or ultrasound guided injection of the iliopsoas bursa [24].

5.4.8 Athletic Pubalgia

Athletic pubalgia, formerly known as sportsman's hernia, is an important source of groin pain in athletes [25, 26]. Patients may present with either the acute or chronic onset of pain that is worsened by twisting motions or sit-ups. These patients have tears of either the adductor origin and/or the rectus abdominis insertion on the pubic symphysis probably as a result of pubic instability. Pubic instability may lead to plain film findings of osteitis pubis. Osteitis pubis presents radiographically as sclerosis with variable erosion that may simulate infection. The rectus abdominis-adductor complex, also known as the pubic plate, is best visualized on MR but can be easily overlooked by the interpreter that is not searching for the findings of this entity. Avulsion of the adductor origin or rectus abdominis insertion will manifest as increased signal on T2 weighted images at the level of the pubic symphysis [27, 28] (Fig. 5.16). As with all imaging findings, the plain film or MR results must be interpreted in conjunction with findings on physical examination because positive results in asymptomatic individuals are not uncommon.

5.4.9 Muscle

Muscle injuries of the lower extremities are very common. Imaging of these injuries may be required not only for confirmation but also for determining treatment and prognosis. Muscle injuries have been divided into different grades for research purposes but from a practical perspective injuries are divided into minor requiring minimal rest and major for those injuries requiring more protracted recovery [29, 30].

Muscle injuries are usually not visible on radiographs. Heterotopic ossification is the sequela of soft tissue injury that may be visible on plain film. Recognizing het-

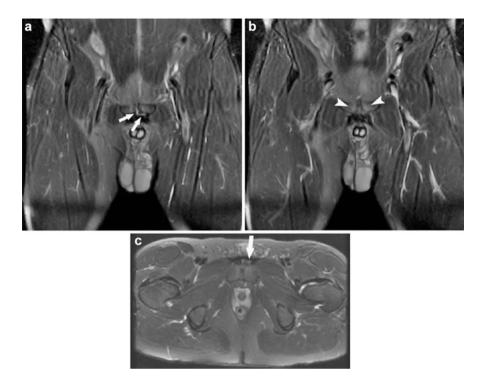


Fig. 5.16 Thirty-eight-year-old man with left groin pain, osteitis pubis, and avulsion of adductor longus. (a) Coronal fat suppressed T2 weighted image of the pelvis shows a linear cleft of high T2 signal (*white arrows*) indicating a tear of the adductor tendon origin. (b) Coronal fat suppressed T2-weighted image of the pelvis shows bone marrow edema (*white arrowheads*) in the pubic symphysis indicating osteitis pubis. (c) Axial fat suppressed T2 weighted image of the pelvis shows high signal (*white arrow*) replacing normal black in the left adductor longus origin indicating a tear

erotopic ossification on radiography is very important to avoid confusion with tumor [31]. Many patients report a history of blunt trauma that is followed by the onset of severe pain and swelling. The initial radiographs may not show any abnormality or findings may be limited to subtle amorphous density. This will rapidly progress over weeks to a classic pattern of zonal ossification that separates this disease radiographically from neoplasm (Fig. 5.17).

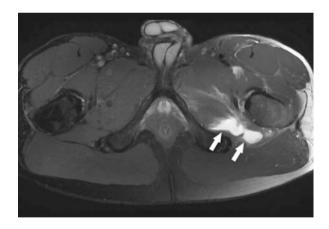
Acute muscle injuries can be rapidly assessed by ultrasound when an experienced user performs the examination. Ultrasound manifestations of injury include interruption of muscle fibers and hematoma [32]. The presence of hematoma is an indication of a higher degree of injury. Chronic injuries of muscle may be subtle on ultrasound with focal muscle atrophy and thickening of the myotendinous junction representing indications of old trauma.

MR best images muscle injuries because it offers a global assessment of muscle, tendon, and bone. MR can accurately assess and characterize both acute and chronic injuries [33, 34]. Synchronous injuries are very common so the entire extremity or

Fig. 5.17 Forty-four-yearold man with right pain and myostis ossificans. Frog leg lateral view of the right hip shows heterotopic ossification in the soft tissues posterior and lateral to the femoral neck (*white arrows*)



Fig. 5.18 Nineteen-yearold quarterback with muscle tear and history of pop and immediate pain in left groin while playing football. Axial fat suppressed T2 weighted image of the left hip shows a hematoma (*white arrows*) in the body of the adductor muscle indicating high grade tear



pelvis should be imaged when there is a clinical concern for muscle injury. Increased signal in a muscle on T2-weighted images is an indicator of acute tear although other causes may produce the same MR findings including contusion, delayed onset muscle soreness, and denervation. The presence of a hematoma is an indication of a high grade injury (Fig. 5.18).

5 Radiology of Hip Injuries

References

- Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29:521–31.
- Armfield DR, Towers JD, Robertson DD. Radiographic imaging of the athletic hip. Clin Sports Med. 2006;25:211–39.
- Naraghi A, White LM. MRI of labral and chondral lesions of the hip. AJR Am J Roentgenol. 2015;205(3):479–90.
- Valimaki VV, Alfthan H, Lehmuskallio E, Loyttyniemi E, Sahi T, Suominen H, Valimaki MJ. Risk factors for clinical stress fractures in male military recruits: a prospective cohort study. Bone. 2005;37:267–73.
- Mattila VM, Niva M, Kiuru M, Pihlajamaki H. Risk factors for bone stress injuries: a followup study of 102,515 person-years. Med Sci Sports Exerc. 2007;39:1061–6.
- Kiuru MJ, Pihlajamaki HK, Ahovuo JA. Fatigue stress injuries of the pelvic bones and proximal femur: evaluation withMR imaging. Eur Radiol. 2003;13:605–11.
- Anderson MW, Kaplan PA, Dussault RG. Adductor insertion avulsion syndrome (thigh splints): spectrum of MR imaging features. AJR Am J Roentgenol. 2001;177:673–5.
- Song WS, Yoo JJ, Koo KH, Yoon KS, Kim YM, Kim HJ. Subchondral fatigue fracture of the femoral head in military recruits. J Bone Joint Surg Am. 2004;86A:1917–24.
- Dorne HL, Lander PH. Spontaneous stress fractures of the femoral neck. AJR Am J Roentgenol. 1985;144:343–7.
- 10. Lee JK, Yao L. Stress fractures: MR imaging. Radiology. 1988;169:217-20.
- 11. Kocher MS, Tucker R. Pediatric athlete hip disorders. Clin Sports Med. 2006;25:241-53.
- 12. Ozonoff MB. Pediatric orthopedic radiology. 2nd ed. Philadelphia: WB Saunders Co.; 1992.
- 13. Bencardino JT, Palmer WE. Imaging of hip disorders in athletes. Radiol Clin North Am. 2002;40:267–87.
- Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis – what the radiologist should know. AJR Am J Roentgenol. 2007;188:1540–52.
- Delaunay S, Dussault RG, Kaplan PA, Alford BA. Radiographic measurements of dysplastic adult hips. Skeletal Radiol. 1997;26:75–81.
- Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res. 2003;417:112–20.
- Frank JM, Harris JD, Erickson BJ, Slikker 3rd W, Bush-Joseph CA, Salata MJ, Nho SJ. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. Arthroscopy. 2015;31:1199–204.
- Clohisy JC, Carlisle JC, Trousdale R, Kim YJ, Beaule PE, Morgan P, Steger-May K, Schoenecker PL, Millis M. Radiographic evaluation of the hip has limited reliability. Clin Orthop Relat Res. 2009;467(3):666–75.
- Reynolds D, Lucac J, Klaue K. Retroversion of the acetabulum: a cause of hip pain. J Bone Joint Surg. 1999;81B:281–8.
- Leunig M, Beck M, Kalhor M, Kim YJ, Werlen S, Ganz R. Fibrocystic changes at anterosuperior femoral neck: prevalence in hips with femoroacetabular impingement. Radiology. 2005;236:237–46.
- Ito K, Leunig M, Ganz R. Histopathologic features of the acetabular labrum in femoroacetabular impingement. Clin Orthop Relat Res. 2004;429:262–71.
- 22. Kingzett-Taylor A, Tirman PF, Feller J, McGann W, Prieto V, Wischer T, Cameron JA, Cvitanic O, Genant HK. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. AJR Am J Roentgenol. 1999;173:1123–6.
- Cardinal E, Buckwalter KA, Capello WN, Duval N. US of the snapping iliopsoas tendon. Radiology. 1996;198:521–2.
- 24. Vaccaro JP, Sauser DD, Beals RK. Iliopsoas bursa imaging: efficacy in depicting abnormal iliopsoas tendon motion in patients with internal snapping hip syndrome. Radiology. 1995;197:853–6.

- 25. Kemp S, Batt ME. The "sports hernia": a complex cause of groin pain. Phys Sportsmed. 1998;26:59–65.
- Palisch A, Zoga AC, Meyers WC. Imaging of athletic pubalgia and core muscle injuries: clinical and therapeutic correlations. Clin Sports Med. 2013;32:427–47.
- Robinson P, Barron DA, Parsons W, Grainger AJ, Schilders EM, O'Connor PJ. Adductorrelated groin pain in athletes: correlation of MR imaging with clinical findings. Skeletal Radiol. 2004;33:451–7.
- Cunningham PM, Brennan D, O'Connell M, MacMahon P, O'Neill P, Eustace S. Patterns of bone and soft-tissue injury at the symphysis pubis in soccer players: observations at MRI. AJR Am J Roentgenol. 2007;188:W291–6.
- Connell DA, Schneider-Kolsky ME, Hoving JL, Malara F, Buchbinder R, Koulouris G, Burke F, Bass C. Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. AJR Am J Roentgenol. 2004;183:975–84.
- Slavotinek JP, Verrall GM, Fon GT. Hamstring injury in athletes: using MR imaging measurements to compare extent of muscle injury with amount of time lost from competition. AJR Am J Roentgenol. 2002;179:1621–8.
- McCarthy EF, Sundaram M. Heterotopic ossification: a review. Skeletal Radiol. 2005;34:609–19.
- 32. Peetrons P. Ultrasound of muscles. Eur Radiol. 2002;12:35-43.
- Boutin RD, Fritz RC, Steinbach LS. Imaging of sports-related muscle injuries. Radiol Clin North Am. 2002;40:333–62.
- Bencardino JT, Mellado JM. Hamstring injuries of the hip. Magn Reson Imaging Clin N Am. 2005;13:677–90.

Chapter 6 Adult Hip and Pelvis Disorders

Peter H. Seidenberg, Michael Pitzer, and Michael Kenneth Seifert

Clinical Pearls

- Hip pain can originate from the hip joint, groin, surrounding musculature, sacroiliac joints, lumbar spine, abdomen, or pelvis.
- To ease in the clinical evaluation of hip pain, it is helpful to divide the hip and pelvis into anterior, lateral, and posterior regions.
- Asymptomatic labral tears are common in athletes. Do not refer an athlete with hip pain and a labral tear for surgical intervention until it has been confirmed that the femoroacetabular joint is the actual source of the patient's pain.
- Athletic pubalgia should be considered in athletes with persistent hip, groin, or pelvic pain despite an adequate trial of conservative therapy.

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6.1 Case Presentation

A 29-year-old male presents to your office with a complaint of intermittent hip pain for the last 18 months, which has been progressively getting worse over the last 2 weeks. He plays softball, baseball, and runs three to five miles several days a week. He also lifts weights 3 days a week. He denies any history of trauma. He states the pain occurred initially only with running and heavy exertion, but now it has progressed to occur with general ambulation. He admits to experiencing pain and tightness down the lateral side of his thigh to his knee. He denies weakness and tingling. He denies a snapping sensation. He denies systemic symptoms or incontinence of urine or stool. He has had no prior interventions besides rest, ice, and antiinflammatory medication. Despite the above measures, when he returns to activity, the symptoms resurface.

Physical examination reveals no erythema, edema, or ecchymosis. He has normal, pain-free full range of motion of his hip although he experiences pain with terminal flexion, abduction, and external rotation. Palpation reveals tenderness over the greater trochanter, the tensor fascia lata, and the gluteus medius. He has negative log roll, flexion abduction external rotation (FABER), Gaenslen's, Gillet, and piriformis tests. Strength is 5/5 with hip flexion, extension, and adduction. Abduction strength is 4+/5. He is neurovascular intact with normal sensation. He has a positive Ober test, Ely's test, and Thomas test. Long sit test is normal. He has a compensated Trendelenburg on the affected side, but negative Stinchfield's and fulcrum tests. He has mild pes planus bilaterally. No imaging studies are performed at this time.

6.2 Introduction

The human hip and pelvis are complex anatomical structures through which a great amount of energy passes during weight bearing and activity. As such, hip and pelvic injuries are common. Hip and pelvic pathology can present as hip pain, groin pain or can even be referred to other areas. Additionally, injury and disease from nearby structures can refer pain to the hip and pelvis. This produces an extensive differential diagnosis, which includes both musculoskeletal and non-musculoskeletal etiologies (Table 6.1). Furthermore, the evaluation is often made difficult because injuries may involve a variety of anatomical structures and may be acute, subacute, or chronic. This chapter reviews the common musculoskeletal etiologies of hip and pelvis pain, symptoms associated with these injuries, diagnostic evaluation, and treatment. The chapter divides hip and pelvis pathology into anterior, lateral, and posterior causes of pain (Table 6.2). The astute physician will remember to perform an examination of the abdomen, spine, knee, and other areas as appropriate because many problems outside the hip and pelvis refer pain to this area.

Hip/pelvis	Thigh	Low back	Buttock	Abdomen/genital
Femoral neck stress fracture	Adductor strain	Sacroiliitis	Gluteal strain	Athletic pubalgia
Pubic ramus stress fracture	Quadriceps strain	Sacroiliac dysfunction	Gluteal contusion	Inguinal hernia
Osteitis pubis	Quadriceps contusion	Lumbar radiculopathy	Piriformis syndrome	Ilioinguinal nerve entrapment
Pubic symphysis dysfunction	Myositis ossificans	Spinal stenosis		Intra-abdominal pathology
Internal snapping	Greater trochanteric pain syndrome	Lumbosacral strain		Intra-pelvic pathology
External snapping	Hamstring strain			Sexual transmitted infection
Labral tear	Femoral hernia			Genital pathology
Femoroacetabular impingement	Meralgia paresthetica			Ectopic pregnancy
Iliopsoas strain or bursitis				
Avascular necrosis of the femoral head				
Osteoarthritis				
Hip dislocation				
Iliac crest contusion				
Coccygeal injury				
Leg length discrepancy				

Table 6.1 Differential diagnosis of hip and pelvic pain

6.3 Anterior Hip and Pelvis Pain

6.3.1 Adductor Strains and Tendinopathy

Adductor strains are generally referred to as a "pulled groin" and are a common cause of hip and groin pain in athletes. The adductor longus, adductor magnus, adductor brevis, adductor minimus, pectineus, and gracilis muscles are all adductors of the hip, contribute to hip flexion and extension, and contribute to internal and external rotation of the hip. In sport, the adductor longus is the most often injured, and may comprise up to 10% of all athletic injuries [1, 2].

An acute adductor strain is caused by a sudden change in direction, sprinting, forced external rotation of an abducted leg, or powerful abduction stress during simultaneous adduction. Acute adductor strains are commonly seen in the sports of hockey and soccer because of the frequent cutting and frequent eccentric contraction of the adductors in these sports [2, 3]. Adductor tendinopathy is a mechanical enthe-sopathy generally due to repetitive strain injuries [4].

Anterior	Lateral	Posterior
Adductor strain and tendinopathy	Greater trochanteric pain syndrome	Hamstring strain
Quadriceps strain	External snapping hip	Ischial bursitis
Quadriceps contusion	Iliac crest contusion	Gluteus medius strain
Myositis ossificans	Meralgia paresthetica	Piriformis syndrome
Iliopsoas strain, tendinopathy, bursitis		Gluteus maximus strain or contusion
Rectus abdominis strain		Sacroiliitis
Pubic symphysis dysfunction		Sacroiliac sprain or dysfunction
Osteitis pubis		Coccygeal injury
Pubic ramus stress fracture		
Femoral neck stress fracture		
Hip dislocation		
Avascular necrosis of femoral head		
Osteoarthritis		
Labral tear		
Femoroacetabular impingement		
Athletic pubalgia		
Internal snapping hip		

Table 6.2 Hip and pelvic pain diagnosis by anatomic region

The differential diagnosis includes avascular necrosis of the femoral head, femoral neck stress fracture, iliopsoas bursitis, osteitis pubis, obturator nerve entrapment, osteoarthritis, pelvic stress fracture, inguinal hernia, and athletic pubalgia.

With an acute injury, athletes will complain of immediate pain piercing into the groin and an inability to continue activity. Delayed ecchymosis and soft tissue swelling may also occur. In chronic injury, athletes will report an insidious onset of groin pain, possibly starting after a change in training that is worse at the start of exercise. Severe pain and dysfunction that occur after a "pop" may suggest adductor tendon avulsion.

Physical examination will reveal tenderness to palpation along the subcutaneous border of the pubic ramus and along the involved adductor muscles and tendons. The patient will have pain with resisted adduction and with passive stretching.

The diagnosis of an adductor strain is usually made clinically. Plain radiographs can be helpful in excluding fractures or avulsions of the hip and pelvis. If the diagnosis is in question, musculoskeletal ultrasonography (Fig. 6.1) or magnetic resonance imaging (MRI) can be used to confirm the diagnosis and fully evaluate the degree of injury [4, 5].

Eccentric strengthening of the adductor muscles is an established preventative measure to protect against groin injuries in soccer players [6]. However, after the injury occurs, treatment depends on the severity of the symptoms. Initially, rest from aggravating factors for 1–2 weeks with ice and oral analgesics provides symptomatic relief. Athletes can begin a stretching program after the inflammation subsides. The



Fig. 6.1 Musculoskeletal ultrasound long axis image of the origins of the adductor muscles on the pubic ramus. Note the separation of the superficial portion of the adductor longus from the pubic ramus (PR). *AB* adductor brevis, *AL* adductor longus. *Arrows* demonstrate partial thickness tear

goal of physical therapy is to prevent atrophy and to regain strength, flexibility, and endurance. Rehabilitative therapy should be instituted as soon as pain allows and should include isometric contractions without resistance followed by isometric contractions against resistance. Prevention and correction of predisposing biomechanical factors should be included in the rehabilitation program. Return to play may take 4 weeks to 6 months depending on the extent of injury. Shorts with directional compression may aid in preventing adductor strains and these shorts may reduce demand during rehabilitation after strains [7]. Athletes with chronic adductor longus strains that have failed several months of conservative therapy may be considered for platelet-rich plasma injection (PRP) [8]. Athletes with adductor avulsion injuries or with chronic tendinopathy that has failed more conservative measures should be referred to orthopedics for consideration for possible surgical intervention.

6.3.2 Quadriceps Strains

The quadriceps muscles are the rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius. The main action of the rectus femoris is to flex the hip and extend the knee, and it is the most commonly injured of the quadriceps muscles [9]. The quadriceps are heavily recruited and frequently overused during jumping, sprinting, cutting, skating, bicycling, and during other explosive movements. Injuries usually occur as a result of a heavy eccentric load. Quadriceps injuries are twice as common in the dominant leg, and risk factors for quadriceps injuries include short height and heavy weight, lack of flexibility, dry field conditions, and a history of quadriceps injuries [10]. The differential diagnosis includes femoral shaft stress fracture, acute compartment syndrome of the anterior thigh, meralgia paresthetica, and femoral nerve injury.

Athletes may report a sensation of "pulling" or "tearing" in the anterior hip with acute injury. Onset of pain is typically after forceful contraction of the quadriceps muscles. The pain is in the anterior thigh and occurs with knee extension or hip flexion.

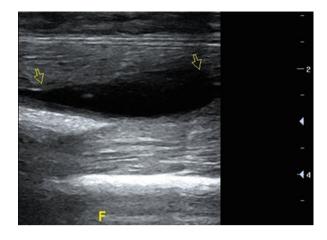


Fig. 6.2 A long axis ultrasound view of a partial thickness tear of the vastus intermedius muscle. *Arrows* demonstrate the tear. *F* femur

The pain often radiates down the thigh and into inguinal area. Physical examination may reveal pain with resisted hip flexion or resisted knee extension. There is often tenderness to palpation of the quadriceps muscles and a palpable defect or mass.

A rectus femoris strain is diagnosed clinically, but if an avulsion injury to the anterior inferior iliac spine is suspected then plain radiographs should be ordered. Ultrasonography (Fig. 6.2) and MRI can aid in the diagnosis and be used to determine severity.

Initial treatment consists of rest, ice, compression, oral analgesia, protected weight bearing, gentle range of motion, and quadriceps exercises. Ice massage and therapeutic ultrasound should be started early in the rehabilitation course. Rehabilitation strengthening exercises are initially concentric and progress to eccentric. An example of this progression is to start the athlete with backward walking, progress to backward running, and later transitioning to forward running. The athlete can return to running when his or her knee range of motion is 80% that of the unaffected side. Depending on the severity of the injury, return to play may take 2–6 weeks. Data is currently lacking on evidence-based return to play protocols following quadriceps injury.

6.3.3 Quadriceps Contusion

Contusions of the quadriceps muscles are a common injury in contact sports including football, rugby, hockey, and martial arts. Quadriceps contusions are caused by direct trauma to the anterior thigh. The differential diagnosis includes femoral shaft fracture, acute compartment syndrome of the anterior thigh, meralgia paresthetica, and quadriceps strains.

Athletes will generally be able to give a history of trauma to the anterior thigh and will describe pain with passive knee flexion and active extension at the knee. Examination often reveals tenderness, swelling, and a hematoma on the anterior thigh.

Pain with knee flexion and a loss of range of motion at the knee is often present on exam. If necessary, the diagnosis can be confirmed with musculoskeletal ultrasound, which will show disruption of the normal muscle fibers and hematoma formation.

Initial treatment is essentially the same as with quadriceps strains including rest, oral analgesia, elevation, and intermittent application of ice with a compression wrap. Keeping the muscle in a lengthened and flexed position for the first 24 h after injury can help maintain flexibility, decrease bleeding, and may shorten the time required for return to play [6]. The athlete should be allowed to weight bear as tolerated. Deep massage and ultrasound should be avoided, as these can increase muscle bleeding. Active flexion should be encouraged and continued frequently. Heat and cold contrasts and strengthening exercises are initiated after the swelling begins to decrease. Return to play may be allowed after the athlete regains quadriceps flexibility and strength equal to 90 % of the unaffected side.

6.3.4 Myositis Ossificans

Myositis ossificans is a pseudosarcomatous lesion that is characterized by bone formation in or adjacent to muscle and can be a post-traumatic complication of a quadriceps contusion [11]. Post-traumatic myositis ossificans occurs after initial muscular bleeding leads to the formation of a hematoma, which later calcifies within the muscle. This process causes pain and reduced flexibility. The incidence of myositis ossificans following muscle contusion is 9-17% [12]. Although occurrence after a contusion is the most common scenario reported by athletes, myositis ossificans can also develop in an athlete with a history of repetitive minor trauma.

The differential diagnosis includes hematoma, abscess, focal rhabdomyolysis, and malignant primary or secondary soft tissue tumors [13]. Post-traumatic myositis ossificans will become clinically suspected when an athlete does not respond to conservative interventions within 3–4 weeks after a contusion. The usual symptoms include a painful and palpable mass with progressive loss of range of motion.

On examination, the athlete may have an antalgic gait and a tender anterior leg mass. Plain radiographs are performed to evaluate the femur for fracture. A bone scan is often used to track myositis ossificans formation. Radiographic evidence of calcification due to myositis ossificans may not be present for weeks following injury and, therefore, a presumptive diagnosis of myositis ossificans is often made after a severe contusion does not show rapid improvement. To help differentiate late presenting myositis ossificans from a sarcoma, computed tomography (CT) scan or MRI is often utilized. Sonographic findings in myositis ossificans have also been described [14] (Fig. 6.3).

Treatment of myositis ossificans is similar to that of a quadriceps strain. The goals of therapy are to restore strength and range of motion. Placing and holding the knee in end of range of flexion immediately after a significant contusion to the quadriceps may shorten the time to return to unrestricted full activity [10]. Early administration of indomethacin may aid in prevention of myositis ossificans; however, this concept

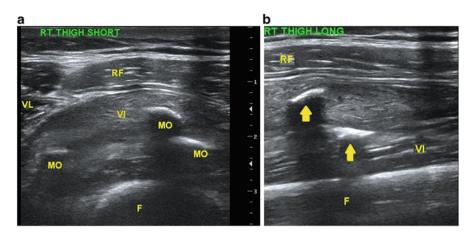


Fig. 6.3 A musculoskeletal ultrasound short axis (a) and long axis (b) views demonstrating heterotopic ossification within the vastus intermedius (VI) consistent with myositis ossificans (MO). RT right, RF rectus femoris, VL vastus lateralis, F femur. Arrows demonstrate myositis ossificans

is extrapolated from indomethacin's use in prevention of heterotopic ossification following hip arthroplasty. At this time, it is uncertain if indomethacin has a substantial impact on the prevention of heterotopic bone formation following quadriceps contusion. Treatment with short term use of bisphosphonates has been suggested in the literature [15]; however, its use is limited by tetratogenicity. Additionally, once the drug is discontinued, the mineralization process appears to resume [16].

Early rehabilitation should include active stretching and strengthening exercises. Extracorporeal shock wave therapy may be considered in the treatment of myositis ossificans [17]. Surgical treatment generally is not indicated, but if necessary, it should be delayed until the mass has matured. Radiographic resolution is not necessary for return to play. However, rehabilitation to achieve full strength and flexibility is mandatory. Wearing protective padding over the affected area should be considered as well.

6.3.5 Iliopsoas Strain

The iliopsoas muscle is a strong flexor of the hip and can be acutely injured when the hip is forced into extension or is blocked during active flexion. This injury is commonly called a hip flexor strain. Tendinopathy may also occur with overuse. It often occurs in soccer players who are hit as they flex at the hip and extend at the knee to kick the ball. It also occurs in weightlifting, uphill running, and with sit-ups. The differential diagnosis includes adductor strains, quadriceps strains, femoroacetabular impingement, osteitis pubis, and athletic pubalgia.

Athletes present with a complaint of sharp, deep groin pain that worsens with active hip flexion or passive hip extension. Physical examination reveals tenderness to palpation in the femoral triangle and increased pain with resisted hip flexion and passive external rotation or extension. The diagnosis is made clinically and imaging is rarely needed. When the diagnosis is not clear, plain radiographs followed by MRI can help establish a diagnosis, with MRI able to distinguish between abnormalities of the involved bones, tendons, and muscles.

Initial treatment involves rest, protection, and oral analgesia followed by rehabilitation. In adults, this injury may result in a partial or complete tear at the musculotendinous junction and these tears take longer to rehabilitate. Corticosteroid injection and surgery may be considered in refractory cases [18].

6.3.6 Iliopsoas Bursitis

The iliopsoas tendon results from the joining of the iliacus and psoas muscles and inserts onto the lesser trochanter of the femur after passing over the protective iliopsoas bursa. The iliopsoas bursa is the largest bursa in the body and communicates with the hip joint in 15% of athletes [19]. This bursitis is associated with sports requiring extensive use of the hip flexors (i.e., soccer, ballet, uphill running, hurdling, jumping) and may be particularly disabling for athletes. It is also associated with degenerative or inflammatory arthritis, infections, trauma, osteonecrosis, and hip replacement [20].

The differential diagnosis includes iliopsoas tendinopathy, FAI and labral tears, athletic pubalgia, AVN, and stress fractures of the femur and pelvis.

Athletes may present with severe, acute, deep groin pain radiating to the anterior hip or thigh. The pain may be great enough to disrupt ambulation. It is often associated with a snapping sensation caused by the iliopsoas tendon snapping over the iliopectineal eminence.

Athletes will assume a position of hip flexion and external rotation to obtain relief [21]. The musculotendinous junction of the iliopsoas lies in the femoral triangle, and deep palpation of the femoral triangle may elicit pain [19]. Pain may be exacerbated by passive hip extension or when the supine athlete raises his or her heels off the table approximately 15°, thereby isolating the iliopsoas [22]. Musculoskeletal ultrasonography will demonstrate an enlargement of the iliopsoas bursa [20] (Fig. 6.4), and MRI reveals a collection of fluid adjacent to the muscle in iliopsoas bursitis [23].

Fig. 6.4 Iliopsoas bursitis: A sagittal oblique view of the anterior hip demonstrates significant fluid within the iliopsoas bursa (*open arrows*). *BUR* bursa, *AC* acetabulum, *FH* femoral head

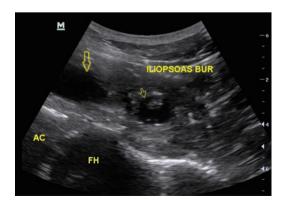




Fig. 6.5 Ultrasound guided aspiration of iliopsoas bursitis. A sagittal oblique view of the anterior hip was utilized to guide aspiration of the enlarged iliopsoas bursa from Fig. 6.4. The *open arrow* points to the needle used in the procedure. The *asterisk* demonstrates the medication that was injected following the aspiration. A acetabulum, *FH* femoral head

Treatment is almost always nonoperative and consists of rest, ice, nonsteroidal anti-inflammatories (NSAIDs), and stretching of the iliopsoas. If symptoms are recalcitrant to conservative therapy, corticosteroid injection with ultrasonographic (Fig. 6.5) or fluoroscopic guidance, release of the iliopsoas tendon near its insertion on the lesser trochanter, or excision of the bursa may be considered [19].

6.3.7 Rectus Abdominis Strain

The rectus abdominis originates on the pubis adjacent to the origin of the adductor longus. As such, injury to the rectus abdominis may easily be confused or coexist with an adductor injury.

The differential diagnosis includes intra-abdominal pathology, sexually transmitted diseases, inguinal hernia, adductor strain, and osteitis pubis.

Athletes typically report deep groin pain that is worsened by tightening or stretching of the rectus abdominis muscle. On physical examination, pain is elicited on palpation of the superior aspect of the pubic ramus, and is exacerbated in the supine patient by a bilateral straight leg raise or resisted sit-up. Imaging is generally not needed to make the diagnosis; however, if necessary, musculoskeletal ultrasound or MRI may demonstrate the injury.

Treatment includes relative rest and oral analgesia followed by activity modification with a physical therapy program that includes core stabilization. Local injection of anesthetic and corticosteroid can be considered in particularly painful cases or in recalcitrant cases, which the authors prefer to perform under ultrasound guidance [24].

6.3.8 Pubic Symphysis Dysfunction

The symphysis pubis plays a key role in energy dissipation and cushioning of impact forces during the human gait, and those forces multiply during sport causing biomechanical strain on the pubic symphysis [2]. Chronic pain at the pubis symphysis results from joint instability. This injury typically occurs in sports where high-speed cutting activity is common and is often present concurrently with an adductor strain. The disorder can also be seen in pregnancy, with widening of the pelvic joints occurring to allow passage of the infant through the pelvis during delivery [25].

The differential diagnosis of pubic symphysis pain includes osteitis pubis, inguinal hernia, athletic pubalgia, and pubic ramus stress fracture.

Athletes may complain of suprapubic pain that is worsened with walking, climbing stairs, turning in bed, getting up from a chair, or lifting. They may report suprapubic pain radiating to the groin or to the sacroiliac region and pain with urination.

The physical examination includes evaluation of the position of the pubic tubercles by palpating their borders to assess right-to-left variation in the frontal plane. Pubic symphysis dysfunction is present if the pubic bones are not level and tension of the two inguinal ligaments is asymmetric. Pain is also evoked by a lateral compression test. Rarely, a palpable groove at the level of the symphysis is detected. Conventional plain radiographs of the pelvis are often helpful in assessing pathologic widening of the symphysis. Flamingo view radiographs may aid in making the diagnosis and should be considered along with conventional plain radiographs. MRI will demonstrate soft tissue injuries and osseous edema and is the imaging study of choice [26].

Treatment is traditionally conservatively with rest, oral analgesia, and pelvic support and compression. A graded exercise program including pelvic stabilization should be initiated as the patient tolerates. Symphyseal injections have also been shown to relieve pain. Referral for surgical intervention is appropriate if there is failure to improve with these conservative measures.

6.3.9 Osteitis Pubis

Osteitis pubis is a painful condition, generally caused by overuse in athletics, which affects the pubic symphysis and surrounding tendinous attachments. More recently, experts have differentiated osteitis pubis into three clinical entities specified as "pubic bone stress injury," "symphysis pubis stress injury," and "traumatic osteitis pubis." [27] Osteitis pubis is commonly reported in sports requiring cutting, twisting, pivoting, excessive side-to-side motion, or multidirectional motions with frequent acceleration and deceleration [28]. For these reasons, osteitis pubis is common in athletics, and it is also common in pregnant and postpartum women, after urologic and gynecologic procedures, and in degenerative and rheumatologic conditions [29]. Athletes with reduced total hip range of motion, reduced hip abduction and adduction strength, and reduced trunk control may be at increased risk for the development of osteitis pubis [30].

The differential diagnosis of osteitis pubis includes pubic ramus stress fracture, pubic symphysis dysfunction, inguinal hernias, athletic pubalgia, proximal adductor pathology, and osteomyelitis.

Athletes describe a gradual onset of pain in the pubic region, which may radiate to the hip, groin, abdomen, proximal medial thigh, testes, and scrotum. The pain is often described as sharp, stabbing, or even burning. Athletes may report worsened pain with striding, pivoting, twisting, climbing stairs, kicking, sit-ups, leg raises, or Valsalva maneuvers. Athletes may also describe an audible or palpable clicking sensation at the symphysis [28].

Physical examination often reveals tenderness over the pubic symphysis and adductor origins of the inferior pubic ramus. Pain may be exaggerated with passive hip abduction, active hip flexion, or active adduction. The lateral pelvic compression and cross-leg tests are often positive. Trendelenburg's test is often positive indicating weak hip abductors, and in severe cases, the athlete may have an antalgic gait with partially flexed hips and knees [28].

Plain radiographs of the pelvis should be obtained. It is important to note that radiographs can lag behind clinical symptoms by as much as 4 weeks. As the disease progresses, reactive sclerosis of the adjacent pubic bones, erosion and resorption of the symphysis margins, and widening of the joint space may appear on the radiographs [28]. If instability is suspected, one-legged standing flamingo views should be performed. Instability is defined as greater than two millimeter height difference between the superior rami of the symphysis [28]. MRI of the pelvis is the most detailed study and can be used to identify acute, subacute, and chronic osteitis pubis. However, marrow edema may be seen in asymptomatic patients, so clinical correlation is necessary to make the diagnosis [1, 27].

Various treatment options for osteitis pubis have been suggested, but most programs start with rest and pharmacologic pain reduction. The goal is to reduce inflammation and remove the provocative activity by modifying training. Athletes should decrease mileage, prevent over-striding, and eliminate downhill running. Oral corticosteroids can be used if the athlete demonstrates intense pain that is limiting their ability to participate in a rehabilitative program [28]. Ultrasound or fluoroscopic guided corticosteroid injection into and around the pubic symphysis may be considered in athletes with refractory symptoms [29]. However, prior to injection, laboratory evaluation should be performed to evaluate for osteomyelitis as a possible cause [27]. A structured physical therapy program should begin when pain and inflammation are reduced. Modalities such as ultrasound and phonophoresis may assist with pain reduction. Leg length discrepancy should also be corrected if found. A graduated exercise program should be utilized with the goal of returning the athlete to a preinjury level of participation. However, patience is required, as this may take 3-6 months or longer, and time to return to play correlates well with the athlete's experienced level of dysfunction [30]. Compression shorts may reduce pain during and after the rehabilitation program. Surgical interventions may be considered if the athlete fails to improve after a long trial of conservative interventions [29]. As some cases take longer than 9 months to improve and there is a high recurrence rate, surgery may also be considered if there is a desire for earlier return to sport or if femoroacetabular impingement coexists [31].

6.3.10 Pubic Ramus Stress Fractures

Stress fractures are common in athletes and military personnel. Pubic ramus stress fractures account for a small percentage of the stress fractures and are considered to be a low risk stress fracture. The etiology of pubic ramus stress fractures has not been fully elucidated, but one common thought is that it often starts with a periosteal reaction at the adductor muscle origin on the pubis as a result of the tensile forces. In general, risk factors for stress fractures include female sex, amenorrhea, smoking, poor nutrition, valgus knee alignment, and leg length discrepancy [32]. Stress fractures are usually seen in distance runners with recent increases in distance or speed as well as in military personnel who enter basic combat training with poorer indices of physical fitness [33].

The differential diagnosis includes femoral neck stress fracture, osteitis pubis, pubic symphysis dysfunction, athletic pubalgia, inguinal hernia, adductor tendinopathy, or referred pain from the genitals or pelvic organs.

Athletes complain of an insidious onset of groin pain that is exacerbated by weight bearing and relieved by rest. It may be localized to the inguinal, peroneal, or adductor regions [34]. Physical examination reveals tenderness of the inferior aspect of the pubic rim, an antalgic gait, and a positive standing sign (frank pain or inability to stand unsupported on the affected leg). Plain radiographs may not be positive for several weeks after the initial injury but are still the preferred initial study due to cost and availability [33]. If the plain radiograph is negative and a stress fracture is still suspected, then advanced imaging with MRI is indicated [32].

Treatment consists of avoiding pain-inducing activities for 4–6 weeks. The athlete should focus on non-weight-bearing activities and stretching of the adductor muscle group and hip joint capsule. This is followed by a gradual functional progression to activity. Most athletes will show a response to treatment in 3–5 months. In addition, evaluation of the athlete's nutritional intake, estrogen status, and training program is warranted.

6.3.11 Femoral Neck Stress Fractures

Femoral neck stress fractures account for approximately 10% of stress fractures, but these injuries can be a potentially career-ending with complications that include avascular necrosis, nonunion, and varus deformity [33]. As such, the clinician needs to maintain a high index of suspicion for this injury. Femoral neck stress fractures are classified as tension or compression, with tension side fractures much more likely to become a displaced fracture, but with compression side stress injuries being more common. Like pubic ramus stress fractures, stress fracture of the femoral neck often occur in distance runners and is usually preceded by a recent change in mileage or intensity. Both intrinsic characteristics of an individual's body and extrinsic factors can precipitate a stress injury. Risk factors include training errors, inadequate footwear, inadequate nutrition, amenorrhea, running on poor surfaces, coxa vara, and femoroacetabular impingement [32, 35, 36].

The differential diagnosis includes avascular necrosis, transient osteoporosis, hip flexor tendonitis or bursitis, hernia, osteitis pubis, and neoplasm [37].

The athlete may describe an aching in the groin, hip, thigh, or knee, which abates shortly after cessation of activity. Nocturnal pain is also common. Pain is associated with exertion and weight-bearing. The athlete often notes a progressive limitation of activity because of the pain.

On physical examination, the athlete may have a positive log roll, a positive FABER test, and the Stinchfield test will cause groin pain [24]. There may also be pain with axial compression and pain with percussion over the greater trochanter. A walking evaluation may reveal an antalgic gait or painful Trendelenburg gait. The single leg hop test will likely be positive, but many feel the hop test should not be performed in patients suspected of having a femoral neck stress fracture for fear of completing the fracture.

If a femoral neck stress fracture is suspected and physical exam findings are suggestive, then it should be considered to be present until proven otherwise. Distinct radiographic findings may not develop until weeks after the initial injury (Fig. 6.6). MRI is used to localize the injury and grade its severity [38]. Additionally, studies have found MRI for the diagnosis of femoral stress fracture to be both 100 % sensitive and specific [39, 40].

The primary goal in management of femoral neck stress fractures should be to prevent complications through early diagnosis and careful treatment. If a stress fracture is suspected, the athlete should remain non-weight-bearing on the affected leg until a full evaluation for a stress injury is completed. Return to play following a stress fracture can take as long as 4–5 months.

Tension side stress fractures, also known as distraction fractures, are more common in older patients and occur on the superolateral side of the femoral neck. These should be referred immediately to an orthopedic surgeon as the preferred treatment is surgical fixation of the fracture.

Compression side stress fractures are more common in younger patients and occur on the inferomedial side of the femoral neck. Compression side femoral neck stress fractures involving more than 50% of the width of the femoral neck should be referred to an orthopedic surgeon for consideration of operative management. If the

Fig. 6.6 An anteroposterior view of the pelvis in a patient with prior history of femoral neck stress fracture demonstrating old callus along the compression side of the right femoral neck



fracture involves less than 50% of the width of the femoral neck, there is low risk for displacement, and it can be treated with prolonged non-weight-bearing until pain-free. Some patients require a short time on bed rest. The athlete should not bear weight until there is evidence of radiographic healing. Frequent radiographs should be performed until complete healing is documented. Supervised gradual return to activity can then occur. Recurrence of pain requires rest for 2–3 days, and then resumption of activity at the last tolerated level of activity. Progression of the fracture and other failures of nonoperative management are indications for immediate orthopedic surgical referral.

A displaced fracture is a combination of both tension and compression fractures, which results in displacement of the femoral head. This type of femoral neck stress fracture is an orthopedic emergency requiring immediate surgical reduction and internal fixation. Exact time lines for return to activity depend upon the nature of the fracture, type of fixation utilized, and surgeon's preference.

6.3.12 Hip Dislocation

A trauma with high energy directed along the axis of the femur when the hip is in the extremes of its normal range of motion is required to cause a hip dislocation. Hip dislocations can be anterior, posterior, or central. Posterior dislocations account for 90% and anterior dislocations account for much of the remainder [41, 42]. The clinician must maintain a high index of suspicion for associated injuries including fractures of the femoral neck, femoral head, and acetabulum.

When a hip dislocation occurs, the athlete is immediately disabled and complains of extreme pain. Attempts to move the hip will increase discomfort. Posteriorly dislocated hips are characteristically held in adduction, internal rotation, and slight flexion. The femoral head may be palpable posteriorly.

A hip trauma series of plain radiographs should be obtained in the emergency room. CT scans are not routinely obtained prior to reduction because of the need for rapid treatment but are usually obtained after the reduction.

Hip dislocations are orthopedic emergencies. Attempts at reduction should not be performed on the playing field. However, it is critical to perform an onfield neurovascular examination, because sciatic nerve injury is observed in 10-14% of patients with posterior dislocations. The athlete should be immobilized and transported to the emergency room for definitive evaluation and treatment [42, 43]. Prompt reduction using proper technique is important in decreasing the incidence of avascular necrosis of the femoral head, sciatic nerve injury, degenerative joint disease, and chondrolysis. The blood supply to the femoral head reaches a minimum level after 24 h after injury, and reduction after 24 h has been shown to cause an increase in osteonecrosis and post-traumatic arthritis. A reduction within 6 h enhances early recovery of the vascularity to the femoral head [44].

6.3.13 Avascular Necrosis of the Femoral Head

Avascular necrosis of the femoral head (AVN) is a cause of pain and loss of function. The pathophysiology of this disease is poorly understood, but it involves thrombus formation in the microvasculature of the bone followed by endothelial cell dysfunction and a disruption of normal angiogenesis [45]. There are both traumatic and atraumatic causes of AVN. Traumatic causes include displaced fractures of the femoral neck and hip dislocation. Atraumatic causes are not as well defined but include systemic corticosteroid use and heavy alcohol intake [46]. The majority of cases of AVN will be diagnosed in persons between the ages of 30 and 60 years and will be males [47].

The differential diagnosis includes osteoarthritis, iliopsoas bursitis, femoroacetabular impingement, and femoroacetabular labral tears.

Athletes usually present with nonspecific groin or hip pain that is worse with weight-bearing and nonspecific hip motion. Pain at rest and night pain also occur. On physical examination, hip range of motion and gait are usually normal except in advanced disease. Plain radiographs of both hips are essential in making the diagnosis. Osteopenia or a mottled appearance with patchy areas of sclerosis and lucency of the femoral head are the earliest radiographic findings but may not be present until 3 months after the inciting injury. As the disease progresses, there is collapse of the involved segment and degenerative change. If AVN is suspected and radiographs are normal, MRI can be used to make the diagnosis and to stage severity. The use of gadolinium increases the likelihood of detection early in the course of the disease.

Management depends on the stage of the disease and should be coordinated with an orthopedic surgeon. Goals of treatment include pain control and improved function. Bone marrow transplants have shown promise in the treatment of AVN [48], but core decompression and arthroplasty are the mainstays of treatment.

6.3.14 Osteoarthritis

Osteoarthritis of the hip is the end stage of many different disorders and the prevalence is on the rise as the population ages. It is the main cause of anterior hip pain in patients over 50 years of age [49]. Patients will complain of hip and groin pain with weight bearing that is relieved by rest. Plain films are helpful in confirming the diagnosis and will show joint degeneration. The disease process is irreversible and the mainstays of treatment are analgesia and surgery. For a more complete discussion of osteoarthritis, see Chap. 14.

6.3.15 Acetabular Labral Tear

Like the shoulder, the hip has a labrum consisting of a fibrocartilaginous rim that serves to deepen the acetabulum. Acetabular labral injuries typically present after an athlete has experienced some form of trauma such as slipping, twisting, or dislocation, and the most common tear location is in the anterosuperior region of the labrum [50]. Labral tears have been identified as precursors of osteoarthritis, and recognition and correction may mitigate progression of hip osteoarthritis [51]. Labral tears are also commonly associated with femoroacetabular impingement which is the next topic in this chapter.

The differential diagnosis includes extra-articular causes of internal snapping hip, iliopsoas tendinosis, iliopsoas bursitis, AVN, and synovial chondromatosis.

The classic symptoms of a labral injury include painful catching or clicking of the hip. The athlete may also experience episodes of sharp groin pain precipitated by pivoting or twisting, and a feeling that the hip is "giving way." Physical examination reveals palpable clicking on Thomas flexion-to-extension, which frequently correlates with the finding of labral tears at arthroscopy [52]. Assessment for anterior labral tears includes moving the hip from full flexion with external rotation and abduction into extension with internal rotation and adduction with reproduction of the patient's symptoms as a suggestive finding. Moreover, in the assessment of posterior labral tears bringing the hip from full flexion with adduction and internal rotation into extension with abduction and external rotation may reproduce the patient's symptoms.

Plain radiographs are the initial study of choice in the evaluation of intra-articular disease due to low cost and high availability. MRI will help in the assessment of soft tissue derangements, but is limited in its ability to detect chondral and labral lesions. Magnetic resonance arthrography (MRA) or hip arthroscopy are the diagnostic investigations of choice in acetabular labral tears [53]. However, there is a high rate of asymptomatic labral tears in young and active individuals [54]. For this reason, the clinical relevance of labral tears on advanced imaging should be supported by history, physical examination, and the use of local anesthetic. Injection of local anesthetic into the joint guided by fluoroscopy or ultrasonography can be useful in diagnosing symptomatic labral tears. If symptoms resolve after injection, an intra-articular etiology is more likely [55].

Conservative intervention with analgesics, guided corticosteroid injection, and/ or physical therapy may be tried. Protected weight-bearing for 4 weeks may result in symptom resolution in a limited number of cases [55]. If conservative measures fail, surgical intervention by hip arthroscopy should be offered and is also the preferred method of treatment by many experts [56].

6.3.16 Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is caused by the abutment of the anterior femoral head–neck junction against the adjacent anterosuperior labrum. Recognition of FAI can be clinically and radiographically difficult. However, familiarity with this disorder is essential as it is thought to progress to osteoarthritis in young adults [57, 58]. CAMtype FAI is commonly found in young athletic males, and is described as an abnormal femoral head–neck junction [59]. Pincer-type FAI is commonly found in middle-aged women, and is described as an over-coverage of the femoral head by the acetabular wall [59]. Mixed-type FAI has also been described. The differential diagnosis includes hip dysplasia, greater trochanteric pain syndrome, iliopsoas tendinopathy, iliopsoas bursitis, athletic pubalgia, quadriceps strains, osteoarthritis, and hamstring tendinopathy.

Patients present with groin pain, loss of function, restricted hip range of motion, as well as grinding or popping [60]. Pain is associated with flexion and internal rotation and will occur after prolonged sitting. In general, physical examination maneuvers on patients with FAI pathology are of high sensitivity, poor specificity, and are limited in their ability to aid in the diagnosis [61]. There may be a decrease in internal rotation of the hip that is associated with pain, and the FADIR (flexion, adduction, internal rotation) impingement test may be positive. A positive painful squat test increases the probability of FAI as the correct diagnosis [62].

Initial radiographs are helpful in identifying morphological cam and pincer deformities and to exclude other possible diagnoses (Fig. 6.7). The findings of a pistol-grip deformity and an abnormal alpha angle are suggestive of cam-type FAI, and a cross-over sign and significant acetabular retroversion are suggestive of pincer-type FAI [24]. MRA is then used to determine the degree of chondrolabral damage [60]. However, asymptomatic cam and pincer deformities are fairly common. As such, the diagnosis of FAI remains a clinical diagnosis that is aided by imaging [60, 63].

Athletes with mild and non-limiting symptoms can be managed with activity modification and close monitoring. Many athletes will experience persistent pain and limited range of motion for which surgical intervention is indicated. Advances have allowed for arthroscopic-based treatment with faster rehabilitation and less restrictions [59]. The goals of treatment include pain reduction, increasing range of motion and function, and preventing further degeneration of the femoroacetabular joint.



Fig. 6.7 Radiographic features of femoroacetabular impingement. This anteroposterior view of the pelvis demonstrates bilateral pistol-grip deformities consistent with Cam lesions (*arrows*) as well as acetabular over-coverage consistent with pincer lesions (*stars*). The left hip also has os acetabuli just distal to the pincer lesion

6.3.17 Athletic Pubalgia

Athletic pubalgia is a controversial overuse injury that goes by many different names including hockey player's syndrome, Gilmore's groin, slap shot gut, the sportsman's hernia, and inguinal disruption. Use of the word "hernia" for this syndrome is common but is a misnomer as the pathology rarely involves herniation of tissues. This injury is more common in men than women and typically occurs in fast-moving sports that involve twisting, turning, and kicking. This injury is common in hockey, soccer, football, baseball, and rugby. The involved pathology involves weakness to the posterior wall of the inguinal canal, external ring dilatation, conjoined tendon damage, and tears in the inguinal ligament [64].

The differential diagnosis includes adductor strains, osteitis pubis, degenerative hip disease, inguinal or femoral hernias, FAI, or referred genital or rectal pain.

Athletes present with an insidious onset of unilateral "deep" groin pain exacerbated by exercise and relieved by rest. In chronic cases, the athlete may have pain with activities of daily living. Sudden movements, sit-ups, and increases in intraabdominal pressure may also worsen the pain.

Athletic pubalgia presents a diagnostic challenge as there is no clear consensus on this clinical entity. Examination usually reveals pain over the conjoined tendon, pubic tubercle, and the deep and superficial inguinal rings. Pain may be reproduced with a resisted sit-up, and there may be observed adductor and hip flexor weakness with dynamic movement [65]. Radiographs may assist to rule out other injuries. A dedicated MRI protocol is the study of choice for athletes with this suspected cause of groin pain, with the protocol detailed to 1.5 T or 3 T systems, coil selection and positioning, and specific sequencing [66, 67]. Diagnostic anesthetic injections of the femoroacetabular joint and the pubic symphysis may aid in the evaluation by ruling out other causes of pain [68].

The goal of treatment is pain reduction and return to sport. Initially, nonoperative treatment with rest, oral analgesics, and physical therapy should be considered. After an initial period of rest, there is a slow resumption of physical activity with supervised physical therapy consisting of core stabilization, pelvic stabilization, strengthening, and flexibility training. Multiple rehabilitative programs have been described [69]. Those who insist on continuing activity will take considerably longer to heal. Athletes who fail conservative treatment should be referred to a surgeon familiar with this disorder for evaluation [70, 71].

6.3.18 Internal Snapping Hip

Snapping hip syndrome, also known as coxa saltans, is a clinical condition with a painful, audible snap occurring during hip flexion or extension. Snapping hip is more common in females and most commonly seen in individuals in their late teens and twenties who are active in dance and running [72]. There is usually no history of trauma. Internal snapping hip is subdivided into intra-articular and extra-articular causes. Extra-articular internal snapping is typically caused by the iliopsoas tendon snapping across the head of the femur, catching on the iliopectineal eminence, or impingement on an overhanging acetabulum [73]. Intra-articular internal snapping hip is most often caused by loose bodies that may arise from labral disease, acetabular or femoral head chondral lesions, idiopathic recurrent hip subluxation, and synovial chondromatosis [74].

The differential diagnosis for internal snapping hip includes external snapping hip, AVN, and athletic pubalgia.

In extra-articular internal snapping hip, the athlete will report an audible and sometimes painful snap with motion of the hip. Athletes are usually able to reproduce the snap with certain hip motions. The snap is felt in the groin or anterior hip. Pain associated with internal snapping hip is insidious in onset. Performance is rarely impaired. In intra-articular internal snapping hip, athletes report a sudden onset of snapping or clicking after trauma. Reproduction of the snap is more difficult, and performance may or may not be inhibited.

The diagnosis of internal snapping hip is made clinically, and a detailed examination is important. The physical examination is performed by placing the athlete in a supine position with the hip flexed, externally rotated, and abducted. The hip is then passively extended, internally rotated, and adducted to reveal snapping as the iliopsoas tendon passes over the femoral head and joint capsule. In extra-articular internal snapping hip, snapping may be prevented by placing significant pressure on the iliopsoas tendon and anterior hip [74–76]. Intra-articular snapping may be uncovered with a scour test.

Plain radiographs are often normal in athletes with snapping hip. However, they are imperative to exclude less common etiologies such as fractures, loose bodies, dysplasia, and synovial chondromatosis. Historically, bursography and tenography have also been used to diagnose snapping hip [74]. However, static and dynamic musculoskeletal ultrasonography have become readily available and can aid in the diagnosis. Static ultrasonography will demonstrate iliopsoas tendon thickening, enlarged bursa, and peritendinous fluid collections. Dynamic ultrasonography will show the moving structures of the hip and reveals an abnormal jerking motion of the iliopsoas tendon corresponding to the athlete's location of pain and audible snapping [77–79]. MRA may be used to evaluate for intra-articular causes of internal snapping hip including labral tears, osteochondral fractures, and loose bodies. In extra-articular snapping, the MRI may show iliopsoas tendon thickening and inflammation of the iliopsoas bursa.

The mainstay of treatment for extra-articular internal snapping hip is nonoperative and involves rest, activity modification, NSAIDs, and physical therapy. Hip flexor stretching and strengthening, pelvic mobilization, and alignment exercises help relieve the pain of internal snapping hip. Core stabilization and pelvic tilt should also be addressed. Other interventions include corticosteroid injection of the bursa and biofeedback to teach the patient how to avoid repetitive hip snapping. If conservative therapy does not adequately relieve symptoms, then referral for surgical management is indicated.

6.4 Lateral Hip and Pelvis Pain

6.4.1 External Snapping Hip

As with internal snapping hip, external snapping hip is also commonly seen in runners and dancers and in the second and third decades of life. External snapping hip is more common than internal snapping hip. It is caused by friction on the greater trochanter of the femur by the iliotibial band (ITB), the anterior border of the gluteus maximus, or the posterior border of the tensor fascia lata (TFL). The ITB is thought to be the most common cause. Ordinarily, the ITB glides smoothly over the greater trochanter with assistance from the underlying bursa. If the posterior aspect of the ITB band is thickened, it will then rub over the greater trochanter and cause a snapping sensation. The bursa may also become painful and inflamed. Other proposed causes of external snapping hip relate to alterations in hip mechanics including decreased angulation of the femoral neck (coxa vara), narrow biiliac width, increased distance between the greater trochanters, and prominent greater trochanters [74, 80, 81].

The differential diagnosis includes internal snapping hip, AVN, acetabular labral tears and athletic pubalgia.

Athletes will report of an audible and often painful snap over the lateral hip with certain movements. The athlete can often voluntarily reproduce the snapping. Ober testing during physical examination will reveal snapping when the affected leg is taken from full extension to 90° of flexion. The examiner's hands should be placed posterior to the greater trochanter in order to feel the snap. If enough force is applied to the greater trochanter to keep the ITB reduced posteriorly, the snapping will not occur with maneuvers.

If the diagnosis is unclear after the history and physical examination, then imaging may be warranted. Plain radiographs are typically normal. Musculoskeletal ultrasonography has been used in external snapping hip to visualize the ITB or gluteus maximus muscle snapping over the greater trochanter [74]. MRI is generally not needed but may show inflammation of the greater trochanteric bursa or thickening of the ITB or gluteus maximus.

The mainstay of treatment for external snapping hip is nonoperative and involves rest, activity modification, NSAIDs, and physical therapy. Physical therapy should include stretching of the ITB, core stabilization, and correction of functional pelvic tilt. Corticosteroid injection of the greater trochanteric bursa can be performed if these measures are unsuccessful or if severe pain limits the athlete's participation in rehabilitation. If conservative interventions do not relieve symptoms, then referral to orthopedic surgery may be considered.

6.4.2 Greater Trochanteric Pain Syndrome

The diagnosis of "greater trochanteric bursitis" has been debated as imaging and surgical pathology often fails to reveal increased bursal fluid and evidence of inflammation. As such, experts are referring to this clinical entity as greater trochanteric pain syndrome (GTPS). This umbrella term can be applied to the involved pathologies of trochanteric bursitis, gluteus medius and minimus tendinopathy and tears, ITB disorders, and TFL disorders [82]. The previously described external snapping hip is on the spectrum of these disorders. Disorders of the gluteus medius and minimus are further discussed later in this chapter in the section on posterior hip and pelvis pain. GTPS is more common in women and is often found in association with or preceded by low back pain [83]. In runners, it is commonly a result of over-use rather than direct trauma, and risk factors include a broad pelvis, leg length discrepancy, and excessive pronation of the foot [84].

The differential diagnosis includes pain radiation from the sacroiliac joint, radicular symptoms of lumbar origin, and piriformis syndrome.

Athletes will report lateral hip pain that may radiate into the buttock or down the lateral aspect of the thigh. They may complain of pain with prolonged standing, lying on the ipsilateral side, climbing stairs, or running. On physical examination, there will be point tenderness over the greater trochanter, the lateral aspect of the hip, and the posterior hip along the gluteus medius and minimus muscles. Pain may be exacerbated by external rotation and abduction of the hip. Clinicians will often find the hip abductors to be weak, the ITB to be tight, and Patrick's test (FABER) causes lateral hip pain.

The diagnosis is made clinically, and imaging is generally unnecessary. Plain radiographs may reveal irregularities of the greater trochanter and tendon calcifications [85]. MRI may show peritrochanteric edema, gluteus medius and minimus tendinosis or tear, and bursal fluid [85]. Similarly, ultrasonography may show tendinopathy or tendon tears, increased bursal fluid, and evidence of enthesopathy [77, 85, 86].

This syndrome is highly responsive to conservative interventions. Initial treatment should begin with ice massage, heat contrasts, and oral analgesics. A rehabilitative program should include TFL and ITB flexibility and mobilization as well as gluteal, hip abductor, and core strengthening exercises. Local anesthetic and corticosteroid injection may be helpful for severe pain or if pain is refractory to other treatment modalities. Other options for refractory cases include extracorporeal shock wave therapy, percutaneous needle tenotomy, platelet-rich plasma or whole blood injection, prolotherapy, and surgical intervention [85]. Leg length and other biomechanical discrepancies should be corrected to prevent recurrence [80].

6.4.3 Iliac Crest Contusion

Iliac crest contusions typically occur in contact sports from a direct trauma to an unprotected iliac crest. This trauma may cause a contusion, hematoma formation, or muscle avulsion. The term "hip pointer" is used to describe an iliac crest contusion that is associated with a subperiosteal hematoma [46, 80].

The differential diagnosis includes fractures of the ilium and avulsions of the nearby soft tissues.

Athletes complain of pain over the iliac crest with ambulation, rotation, and bending at the waist away from the injured side. The athlete may also report numbness or decreased sensation in the lateral buttock and hip if there is damage to the lateral femoral cutaneous nerve, iliohypogastric nerve, or ilioinguinal nerve.

Physical examination may reveal swelling, ecchymosis, and tenderness over the iliac crest extending superiorly into the internal and external oblique muscles. A hematoma may be palpable and a palpable defect along the iliac crest would indicate an avulsion injury. Radiographs are generally unnecessary at the time of diagnosis, but pelvic radiographs are indicated to evaluate for fractures and periostitis in athletes with prolonged or severe symptoms. Ultrasonography is useful in visualizing the subperiosteal hematoma, may demonstrate muscle disruption, and can assist with hematoma aspiration [24] (Fig. 6.8).

Treatment is initiated immediately with ice and compression to minimize swelling and hematoma formation. Rest, activity modification, and oral analgesia may be recommended depending on the severity of symptoms. Abdominal muscle, low back, and flank stretching and strengthening are performed as tolerated. Treatment with local anesthetic and corticosteroid injection is generally considered safe and effective and may aid in earlier return to play [87]. However, prior to resumption of contact sports, trunk range of motion should be pain-free and the athlete should be protected with adequate padding.

6.4.4 Meralgia Paresthetica

Meralgia paresthetica is a mononeuropathy caused by compression of the lateral femoral cutaneous nerve [88]. The most common site of entrapment is at the inguinal ligament. Meralgia paresthetica is more common in males than females and is associated with the sports of gymnastics, baseball, soccer, and body building [89]. It is also commonly found in diabetics, the obese, older patients, and in people who wear tight pants, belts, or girdles [84].

The differential diagnosis includes lumbar radiculopathy and greater trochanteric pain syndrome.

Most athletes will describe pain, numbress, tingling, or burning pain over the anterolateral thigh. The athlete should be questioned about important predisposing factors such as recent weight gain or previous surgical procedure. In athletes, prolonged flexion (marksmen), increased muscle mass (weight lifters), or constrictive clothing may play a role. However, in the athletic population, it is not unusual for no identifiable cause to be found [84].

The diagnosis is made clinically and is based on sensory symptoms in the lateral femoral cutaneous nerve distribution. On physical examination, a positive Tinel's sign is usually present one centimeter inferomedial to the anterior superior iliac crest. The pelvic compression test is a useful and simple clinical test to support the diagnosis of meralgia paresthetica [88]. Nerve block testing may aid in the diagnosis and is considered positive if an anesthetic injected at the site where the lateral femoral cutaneous nerve passes the inguinal ligament causes immediate relief of symptoms [89]. Electrophysiologic testing is technically difficult but may demonstrate prolonged latency or decreased conduction velocity consistent with compression [46, 90]. Radiographs and MRI of the hip and pelvis are useful if there is concern for intra-pelvic and intra-articular compression on the nerve.

Heat, compression avoidance, physical therapy, and NSAIDs have been shown to be effective in the treatment of meralgia paresthetica. Nerve blocks and radiofrequency ablations are also considered effective [91, 92]. However, high quality evidence is lacking to support these interventions. If symptoms are persistent and disabling despite conservative therapy, then surgical intervention may be warranted.

6.5 Posterior Hip and Pelvic Pain

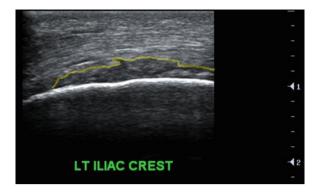
6.5.1 Hamstring Strain

The hamstring is made up of the semitendinosus, semimembranosus, and the long and short heads of the biceps femoris. The three muscles have a common origin at the ischial tuberosity. The hamstring is highly susceptible to injury because the muscle group spans two joints. Hamstring injuries may be the most common injury in sport [93]. Of the muscles in the hamstring, the biceps femoris is the most frequently strained. Complete tears are rare, but have been reported in water skiers, runners, dancers, and power lifters. The most important risk factor for a hamstring strain is a history of a prior hamstring strain [93]. Other risk factors for hamstring injuries include leg length discrepancy, muscle imbalances, insufficient pre-activity stretching, and poor technique [94].

The differential diagnosis includes radicular pain of lumbar origin, referred pain from the SI joint, and avulsion fracture of the ischial tuberosity.

Athletes usually self-diagnose the injury at the time that it occurs. Patients will describe acute onset posterior thigh pain and maybe even a "pop" at the time of injury [95].

Physical examination may show edema and ecchymosis over the affected muscle belly. The examiner should palpate the ischial tuberosity and follow the muscle inferiorly to locate the area of maximal tenderness, size of the area of tenderness, **Fig. 6.8** Ultrasound image over the iliac crest demonstrating muscle fiber discontinuity following iliac crest contusion (outlined in *yellow*). *LT* left



and to determine whether there is a palpable defect in the muscle [95]. If the ischial tuberosity is extremely tender, then an avulsion fracture should be suspected. Pain is exacerbated with resisted knee flexion, and the athlete is often unable to fully straighten his or her knee due to pain.

The diagnosis of a hamstring strain is made by history and physical examination, and imaging is not routinely needed. Plain radiographs should be ordered in athletes with suspected ischial tuberosity avulsion fractures. Musculoskeletal ultrasonography and MRI may be used to assess the severity of injury. MRI is better able to detect small muscular injuries, however ultrasound has the advantage of providing assessment of the muscle dynamically [96]. Either ultrasonography or MRI should be used to confirm if a complete tear of the hamstring is suspected.

Like other soft tissue injuries, initial treatment should consist of ice, oral analgesia, compression wraps, and protected weight-bearing. This is then followed by passive hamstring stretches in the pain-free range. As the symptoms resolve, the athlete progresses to active hip and knee range of motion exercise, then on to hamstring strengthening and isometrics. Early studies showed promise in the treatment of hamstring injuries but there is currently no high grade evidence to support the use of platelet-rich plasma or stem cell therapy for hamstring strains [97, 98]. A complete avulsion of the entire hamstring group from the ischial tuberosity should be referred for possible surgical intervention [99].

6.5.2 Ischial Bursitis

Ischial bursitis, also known as weaver's bottom, occurs after a contusion of the ischial tuberosity, as a complication after injury of the hamstring origin, or from prolonged sitting. The differential diagnosis includes hamstring strain, radicular pain of lumbar origin, referred pain from the SI joint, and avulsion fracture of the ischial tuberosity. Athletes will complain of pain while sitting [100].

On physical examination there will be localized tenderness over the ischial tuberosity. Ultrasound or MRI can be used to confirm the diagnosis [23] (Fig. 6.9).



Fig. 6.9 Ischial bursitis: A long axis ultrasound view of the semimembranosus tendon origin (semim outline in *blue*) at the level of the ischial tuberosity (IT—outlined in *red*). The ischial bursa was found to be enlarged due to bursal effusion (outlined in *green*)

Treatment consists of rest, ice, oral analgesia, hamstring stretching and strengthening, and protection. A doughnut cushion will help alleviate pain while sitting. In recalcitrant cases, aspiration of the bursa and injection of corticosteroid should be considered.

6.5.3 Gluteus Maximus Strain

The gluteus maximus is an extensor of the hip and trunk. Compared to hamstring injuries, isolated strains of the gluteus maximus are uncommon but can occur in sprinters. More often, this muscle is injured through direct trauma [101].

The differential diagnosis includes hamstring injury, ischial bursitis, radicular pain of lumbar origin, and referred pain from the SI joint.

The athlete will report a sharp pain in the buttock with sudden onset, typically during a burst of speed or sudden change in direction. On physical examination, the hip, lumbar spine, and SI joints should be examined. If no tenderness is elicited in any of these areas, then a gluteus maximus strain is suspected.

As this disease process is on the spectrum of GTPS, intervention and rehabilitation are essentially the same. Treatment of a gluteus maximus strain involves rest, ice, and compression. A rehabilitative program should be started with mobilization and range of motion and progresses to strengthening as the athlete tolerates. Return to full participation can occur once the athlete is pain-free and is able to do sports-specific activities.

6.5.4 Gluteus Medius Strain

The gluteus medius functions as a hip abductor. Injuries to this muscle are common in runners. The differential diagnosis includes hamstring injury, radicular pain of lumbar origin, and referred pain from the SI joint. The athlete will often complain of lateral thigh pain near the greater trochanter. There will be pain with palpation just proximal to the tendinous insertion on the greater trochanter and often in the muscle belly itself [102]. Resisted abduction of the hip will provoke pain. Treatment follows the same principles as GTPS with additional emphasis placed upon core strengthening [80].

6.5.5 Sacroiliac Joint Dysfunction

The SI articulation is formed by the sacrum and the ilia, and the major supporting ligaments include the anterior and posterior SI ligaments, the interosseous SI ligament, and the sacrotuberous ligament. Although uncommon, painful tearing and stretching of any of these ligaments can occur. Sports that involve repetitive unidirectional pelvic shear and torsional forces (skating, gymnastics, bowling) put the athlete at risk of SI joint dysfunction [103]. SI joint pain is also well described in rowers [104]. Loss of motion in the SI joint or sustained contraction of the overlying muscles may cause pain.

The differential diagnosis includes radicular pain, piriformis syndrome, gluteus medius strain, ankylosing spondylitis, Reiter's syndrome, and other spondyloar-thropathies. The clinician should have high suspicion for an autoimmune etiology if both SI joints are involved or if the athlete is not responding to conservative interventions.

The athlete typically presents with pain at one SI joint. The pain may radiate to the low back, groin, posterolateral hip, and thigh. The pain pattern may mimic radicular pain from a herniated nucleus pulposus or spinal stenosis.

During examination, care should be taken to ensure that features of ankylosing spondylitis, Reiter's syndrome, and other spondyloarthropathies are not present. There will be unilateral tenderness over the affected posterior superior iliac spine and along the sacral sulcus. The lack of nerve root tension signs and absence of motor, reflex, or sensory deficits help distinguish SI joint dysfunction from nerve root compression lesions. However, SI joint dysfunction can present in conjunction with spinal pathology [105]. FABER, piriformis, and Gaenslen's tests may be positive. Straight leg raise may cause SI pain. Pain is also exacerbated by forward flexion of the trunk with knees extended. A positive one-legged stork test (Gillet test) indicates a lack of mobility on the affected side. Sacral compression medial to the posterior superior iliac spine often causes localized pain. The athlete should also be evaluated for a leg length discrepancy [104].

No gold standard clinical or diagnostic testing exists for the diagnosis of SI joint dysfunction [105]. Plain radiographs and CT imaging may aid in the diagnosis but also show degenerative changes in asymptomatic patients and incur high false positive rates. MRI is the imaging modality of choice in suspected sacroiliitis with negative plain films [106]. Sacroiliac joint injection with an anesthetic may aid in the diagnosis but remains controversial [105]. Such injections should be performed under ultrasound or fluoroscopic guidance.

The goal of treatment is pain reduction and restoration of normal movement. Treatment consists of ice, NSAIDs, ice massage, and heat. A corset, constricting elastic bandage, or SI belt may provide pain relief. The rehabilitation program should include pelvic stabilization exercises and exercises to stretch and strengthen the piriformis muscle. Osteopathic manipulation may assist in reestablishing a neutral pelvis and corticosteroid injections should be considered in recalcitrant pain [24].

6.5.6 Piriformis Syndrome

The piriformis muscle originates on the anterolateral aspect of the sacrum and inserts on the upper border of the greater trochanter of the femur. The piriformis is an external rotator of the hip. Piriformis syndrome is the occurrence of pain at the site of the piriformis muscle secondary to inflammation or spasm. The nearby sciatic nerve may become secondarily involved.

The differential diagnosis includes hip joint disease, SI joint dysfunction, nerve root irritation, spinal stenosis, GTPS, and lumbar herniated nucleus pulposus.

Athletes usually present with a history of blunt trauma to the gluteal or SI region. They complain of pain in the lower SI joint, the greater sciatic notch, and piriformis muscle. The pain may radiate down the posterior buttock into the hip and thigh and is frequently exacerbated by stooping or lifting.

On physical examination, there is typically tenderness over the piriformis muscle. The Lasègue sign will demonstrate tenderness at the gluteal region around the piriformis muscle [107]. Buttock pain is exacerbated by hip flexion and passive internal rotation. Resisted hip external rotation exacerbates the pain. Straight leg raise is occasionally positive with referred pain down the posterior thigh and calf. Patients will often have a positive Gaenslen's test. The FAIR test (flexion, adduction, internal rotation) has been shown to have a sensitivity of 88 % and a specificity of 83 % [108]. Radiographs, MRI, and CT scanning are not needed to make the diagnosis, but may show changes in the piriformis muscle or sciatic nerve [109].

Treatment of piriformis syndrome includes ice massage, NSAIDs, muscle relaxants, ultrasound, electrical stimulation, and physical therapy [103, 110]. Osteopathic manipulative treatment has also been shown to be helpful [111]. Local anesthetic or steroid injections may provide pain relief [112]. Injection of botulinum toxin has been shown to reduce buttock pain and improve hip functionality and quality of life in patients suffering with chronic piriformis syndrome [113]. To prevent recurrence, lumbosacral dysfunction and imbalances in the surrounding musculature must be concurrently treated. If conservative therapy fails or if the patient develops foot drop or gluteal muscle atrophy, then operative treatments are available [103, 110].

6.5.7 Coccygeal Injury

The coccyx is joined to the sacrum by cartilage which forms a synchondrosis. It is susceptible to injury during a fall on the buttocks, when struck from behind, or during a difficult vaginal delivery.

The differential diagnosis is limited in this region but examiners should consider referred pain from the spine or hip, intra-pelvic etiologies of pain, and pilonidal cysts.

Athletes will typically report direct trauma to the upper buttock. Insidious onset of chronic coccygeal pain may also be reported.

Physical examination reveals localized tenderness in the coccygeal region, localized swelling or ecchymosis, and pain exacerbated by sitting. If there is no history of trauma, rectal examination and lower abdominal evaluation are indicated. Plain radiographs are required to rule out an inferior sacrum fracture and to determine whether the coccyx is dislocated or displaced. Ultrasound may demonstrate an inflamed over-riding bursa (Fig. 6.10).

In the event of an acutely dislocated or displaced coccyx, the examiner may reduce the displaced element by inserting a lubricated index finger into the rectum so that the palmar surface rests against the anterior aspect of the coccyx. He or she then palpates the posterior aspect of the coccyx externally, applies a gentle traction on the coccyx, and glides the coccyx into its normal position. If successful, pain relief is usually immediate. However, there is no conclusive evidence as to the efficacy of this approach.

Like other contusions or non-displaced fractures, acute coccygeal injuries are treated with rest and analgesia. Successful treatment of chronic coccydynia has been reported with pelvic relaxation exercises, pelvic floor strengthening, biofeedback, local corticosteroid injection, botulinum toxin injection, and coccygectomy [114–117].

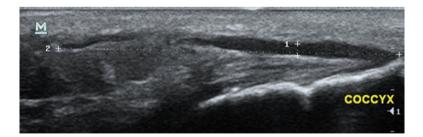


Fig. 6.10 Adventitial bursa over-riding the coccyx. Very light transducer pressure was required to obtain this long axis image. The bursa measured 33 mm by 1 mm

6.6 Other Hip and Pelvic Pain

6.6.1 Leg Length Discrepancy

There are two types of leg length discrepancies: true and functional. In a true leg length discrepancy, the actual length of the two lower extremities is different when measured from the femoral heads to the plantar surfaces of the feet. This may be the result of a varus or valgus deformity of the femoral neck, congenital anomalies of the femur or tibia, or growth disturbances of the femur or tibia [118]. In a functional leg length discrepancy, the athlete's two lower extremities are, in fact, the same length, but pelvic obliquity gives the appearance of a discrepancy. Possible causes of a functional leg discrepancy include contractures at the lumbosacral junction due to scoliosis, post-traumatic deformities of the pelvis, somatic dysfunction of the pelvis and SI joints, and muscle contractures about the hip and knee.

Pain originating in the pelvis, low back, and hip region can be caused or worsened by a disparity in leg length. The evaluation of atraumatic hip and pelvis pain should always include screening for a leg length discrepancy [118–120]. The physical examination and osteopathic manipulation chapters discuss the evaluation of leg length in greater detail.

A more precise measurement of leg length is obtained with a standing AP radiograph film of the pelvis down to the feet. This view should include the upper lumbar spine and the femoral heads. The athlete should stand with his or her feet shoulder width apart with equal weight distribution while the plain film is being taken. Lines are then drawn on the radiograph at the superior sacral ala bilaterally to form a sacral base and at the superior margin of each femoral head. The examiner then draws a line from the sacral base and femoral heads to the base of the film. This method is recommended if the standing measurements have not been accurate. Instead of plain radiographs, some facilities are now using a quick computer tomography scan from the upper lumbar spine to the feet and then comparing measurements from the medial malleolus to the superior margin of the femoral head on each side.

True limb length discrepancies are treated with orthotics and other assistive devices. Functional limb length discrepancies are treated with rehabilitation and manual medicine.

6.7 Conclusion of the Case

The patient was diagnosed with greater trochanteric pain and ITB syndromes. He was instructed to take a 7 day course of NSAIDs. He was referred to physical therapy for gluteus medius, quadriceps, hamstring, and ITB stretching and strengthening with modalities (ultrasound, electrical stimulation, iontophoresis, or phonophoresis) as needed to assist with pain control. A core stabilization program was also emphasized. Additionally, over-the-counter orthotics were recommended for the pes planus with hyperpronation. He responded well and was back to his usual activities in approximately 6 weeks.

References

- Cunningham PM, Brennan D, O'Connell M, MacMohan P, O'Neill P. Patterns of bone and soft-tissue injury at the symphysis publis in soccer players: observations at MRI. AJR Am J Roentgenol. 2007;188(3):W291–6.
- 2. Nicholas SJ. Tyler TF adductor muscle strains in sport. Sports Med. 2002;32(5):339-44.
- Rishiraj N, Lloyd-Smith R, Lorenz T, Niven B, Michel M. University men's ice hockey: rates and risk of injuries over 6-years. J Sports Med Phys Fitness. 2009;49:159–66.
- 4. Pesquer L, Reboul G, Silvestre A, Poussange N, Meyer P, Dallaudiere B. Imaging of adductor-related groin pain. Diagn Interv Imaging. 2015;96:861–9.
- 5. Koulouris G. Imaging review of groin pain in elite athletes: an anatomic approach to imaging findings. AJR Am J Roentgenol. 2008;191:962–72.
- Frizziero A, Trainito S, Oliva F, Aldini NN, Masiero S, Maffulli N. The role of eccentric exercise in sports injuries rehabilitation. Br Med Bull. 2014;110:47–75.
- Am C, Jamison ST, McNally MP, Pan X, Schmitt LC. Hip adductor activations during runto-cut manoeuvres in compression shorts: implications for return to sport after groin injury. J Sports Sci. 2014;32(14):1333–40.
- Dallaudière B, Pesquer L, Meyer P, Silvestre A, Perozziello A, et al. Intratendinous injection of platelet-rich plasma under US guidance to treat tendinopathy: a long-term pilot study. J Vasc Interv Radiol. 2014;25(5):717–23. doi:10.1016/j.jvir.2014.01.026. Epub 2014 Mar 20.
- Mendiguchia J, Alentorn-Geli E, Idoate F, Myer GD. Rectus femoris muscle injuries in football: a clinically relevant review of mechanism of injury, risk factors and preventative strategies. Br J Sports Med. 2013;47:359–66.
- Aronen JG, Garrick JG, Chronister RD. Quadriceps contusions: clinical results of immediate immobilization in 120 degrees of knee flexion. Clin J Sport Med. 2006;16:383–7.
- Nowaczyk P, Murawa D, Zmudzinska M, Wasiewicz J. Myositis ossificans of the quadriceps femoris mimicking sarcoma as a diagnostic and therapeutic problem—case report and literature review. Pol J Surg. 2013;85(9):520–6.
- 12. Ryan JB, Wheeler JH, Hopkinson WJ, et al. Quadriceps contusions. West Point update. Am J Sports Med. 1991;19(3):299–304.
- 13. Yochum AM, Reckelhoff K, Kaeser M, Kettner NW. Ultrasonography and radiography to identify early post traumatic myositis ossificans in an 18-year-old male: a case report. J Chiropr Med. 2014;13:134–8.
- 14. Abate M, Salini V, Rimondi E, Errani C, Marco Alberghini M, Mercuri M, Pelotti P. Post traumatic myositis ossificans: sonographic findings. J Clin Ultrasound. 2011;39(3):135–40.
- Mani-Babu S, Wolman R, Keen R. Quadriceps traumatic myositis ossificans in a football player: management with intravenous pamidronate. Clin J Sport Med. 2014;24(5):e56–8. doi:10.1097/JSM.0000000000034.
- Mavrogenis AF, Soucacos PN, Papagelopoulos PJ. Heterotopic ossification revisited. Orthopedics. 2011;34(3):177. doi:10.3928/01477447-20110124-08.
- Buselli P, Coco V, Notarnicola A, Messina S, Saggini R, Tafuri S, Moretti L, Moretti B. Shock waves in the treatment of post-traumatic myositis ossificans. Ultrasound Med Biol. 2010;36(3):397–409.
- 18. Hureibi KA, McLatchie GR. Groin pain in athletes. Scott Med J. 2010;55(2):8-11.
- Johnston CA, Wiley JP, Lindsay DM, Wiseman DA. Iliopsoas bursitis and tendonitis. Sports Med. 1998;25(4):271–83.
- Di Sante L, Paoloni M, De Benedittis S, Tognolo L, Santilli V. Groin pain and iliopsoas bursitis: always a cause-effect relationship? J Back Musculoskelet Rehabil. 2014;27:103–6.
- 21. Fricker PA. Management of groin pain in athletes. Br J Sports Med. 1997;31(2):91-101.
- Heolmich P. Adductor-related groin pain in athletes. Sports Med Arthrosc Rev. 1997;5(4):285–91.
- Karlsson J, Jerre R. The use of radiography, magnetic resonance, and ultrasound in the diagnosis of hip, pelvis, and groin injuries. Sports Med Arthrosc Rev. 1997;5(4):268–73.

- 24. Seidenberg PH, Sirota M. Hip and pelvis. In: Birrer, O'Connor, Kane, editors. Musculoskeletal and sports medicine: essentials for the primary care practitioner, 4th edition. Philadelphia: CRC Press, 2016.
- Becker I, Woodley SJ, Stringer MD. The adult human pubic symphysis. J Anat. 2010; 217:475–87.
- Herren C, Sobottke R, Dadgar A, Ringe MJ, Graf M, Keller K, Eysel P, Mallmann P, Siewe J. Peripartum pubic symphysis separation—current strategies in diagnosis and therapy and presentation of two cases. Injury. 2015;46:1074–80.
- 27. Beatty T. Osteitis pubis in athletes. Curr Sports Med Rep. 2012;11(2):96-8.
- Vitanzo PC, McShane JM. Osteitis pubis: solving a perplexing problem. Phys Sports Med. 2001;29(7):33–40.
- 29. Choi H, McCartney M, Best TM. Treatment of osteitis pubis and osteomyelitis of the pubic symphysis in athletes: a systematic review. Br J Sports Med. 2011;45:57–64.
- 30. McAleer SS, Gille J, Bark S, Riepenhof H. Management of chronic recurrent osteitis pubis/ pubic bone stress in a premier league footballer: evaluating the evidence base and application of a nine-point management strategy. Phys Ther Sport. 2015;16:285–99.
- Matsuda DK, Ribas M, Matsuda NA, Domb BG. Multicenter outcomes of endoscopic pubic symphysectomy for osteitis pubis associated with femoroacetabular impingement. Arthroscopy. 2015;31(7):1255–60.
- 32. Henning PT. The running athlete: stress fractures, osteitis pubis, and snapping hips. Sports Health. 2014;6(2):122–7.
- Jacobs JM, Cameron KL, Bojescul JA. Lower extremity stress fractures in the military. Clin Sports Med. 2014;33:591–613.
- 34. Pavlov H, Nelson TL, Warren RF, Torg JS, Burstein AH. Stress fractures of the pubic ramus: a report of twelve cases. J Bone Joint Surg Am. 1982;64(7):1020–5.
- Gold M, Anderson CN, Fredericson M, Safran MR, Stevens KJ. Femoral neck stress fractures and imaging features of femoroacetabular impingement. PM R. 2015;7:584–92.
- 36. Markey KL. Stress fractures. Clin Sports Med. 1987;6(2):405-25.
- DeFranco MJ, Recht M, Schilis J, Parker RD. Stress fractures of the femur in athletes. Clin Sports Med. 2006;25(1):89–103.
- Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. Sports Health. 2013;5(2):165–74.
- Quinn SF, McCarthy JL. Prospective evaluation of patients with suspected hip fracture and indeterminate radiographs: use of T1-weighted MR images. Radiology. 1993;187(2):469–71.
- Shin AY, Morin WD, Gorman JD, Jones SB, Lapinsky AS. The superiority of magnetic resonance imaging in differentiating the cause of hip pain in endurance athletes. Am J Sports Med. 1996;24(2):168–76.
- 41. Scudese BA. Traumatic anterior hip dislocation. Clin Orthop. 1972;88:60.
- 42. Walsh ZT, Micheli LJ. Hip dislocation in a high school football player. Phys Sports Med. 1989;17:112.
- Nishina T, Saito S, Ohzono K, Shimizu N, Hosoya T, Ono K. Chiari pelvic osteotomy for osteoarthritis: the influence of the torn and detached acetabular labrum. J Bone Joint Surg Br. 1990;72:765.
- 44. Hougaard K, Thomsen PB. Traumatic posterior dislocation of the hip—prognostic factors influencing the incidence of avascular necrosis of the femoral head. Arch Orthop Trauma Surg. 1986;106:32–5.
- 45. Pouya F, Kerachian MA. Avascular necrosis of the femoral head: are any genes involved? Arch Bone Jt Surg. 2015;3(3):149–55.
- 46. Nuccion S, Hunter DM, Finerman GAM. Hip and pelvis: adult. In: DeLee JC, Drez Jr D, Miller MD, editors. DeLee & Drez's orthopedic sports medicine: principles and practice. 2nd ed. Philadelphia: WB Saunders; 2003. p. 1443–80.
- Assouline-Dayan Y, Change C, Greenspan A, Shoenfeld Y, Gershwin ME. Pathogenesis and natural history of osteonecrosis. Semin Arthritis Rheum. 2002;32(2):94–124; Beatty T. Osteitis pubis in athletes. Curr Sports Med Rep. 2012;11(2):96–8.

- 48. Cia J, Wu Z, Huang L, Chen J, Wu C, Wang S, Deng Z, Wu W, Luo F, Tan J. Cotransplantation of bone marrow mononuclear cells and umbilical cord mesenchymal stem cells in avascular necrosis of the femoral head. Transplant Proc. 2014;46:151–5.
- 49. Roberts WN, Williams RB. Hip pain. Prim Care. 1988;15:783-93.
- Domb BG, Shindle MK, McArthur B, Voos JE, Magennis EM, Kelly BT. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. HSS J. 2011;7:145–50.
- Philippon MJ, Stubbs AJ, Schenker ML, Maxwell RB, Ganz R, Leunig M. Arthroscopic management of femoroacetabular impingement: osteoplasty technique and literature review. Am J Sports Med. 2007;35:1571–80.
- McCarthy JC, Busconi B. The role of hip arthroscopy in the diagnosis and treatment of hip disease. Orthopedics. 1995;18:753–6.
- Naraghi A, White LM. MRI of labral and chondral lesions of the hip. AJR Am J Roentgenol. 2015;205:479–90.
- Lee AJ, Armour P, Thind D, Coates MH, Kang AC. The prevalence of acetabular labral tears and associated pathology in young asymptomatic population. Bone Joint J. 2015;97-B:623–7.
- 55. Byrd JW. Labral lesions: an elusive source of hip pain: case reports and literature review. Arthroscopy. 1996;12:603–12.
- Lage L, Patel J, Villar R. The acetabular labral tear: an arthroscopic classification. Arthroscopy. 1996;12(3):269–72.
- Beal DP, Sweet CF, Martin HD, Lastine CL, Grayson DE, Ly JQ, Fish JR. Imaging findings of femoroacetabular impingement syndrome. Skeletal Radiol. 2005;34:691–701.
- Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement. A cause for osteoarthritis of the hip. Clin Orthop. 2003;417:112–20.
- Nielsen TG, Miller LL, Lund B, Christiansen SE, Lind M. Outcome of arthroscopic treatment for symptomatic femoroacetabular impingement. BMC Musculoskelet Disord. 2014;15:394–9.
- Bittersohl B, Hosalkar HS, Hesper T, Tiderius CJ, Zilkens C, Krauspe R. Advanced imaging in femoroacetabular impingement: current state and future prospects. Front Surg. 2015; 2(34):1–15.
- 61. Reiman MP, Goode AP, Cook CE, Holmich P, Thorborg K. Diagnostic accuracy of clinical tests for the diagnosis of hip femoroacetabular impingement/labral tear: a systematic review with meta-analysis. Br J Sports Med. 2015;49:811–22.
- 62. Ayeni O, Chu R, Hetaimish B, Nur L, Simunovic N, Farrokhyar F, Bedi A, Bhandari M. A painful squat test provides limited diagnostic utility in CAM-type femoroacetabular impingement. Knee Surg Sports Traumatol Arthrosc. 2014;22:806–11.
- Frank JM, Harris JD, Erickson BJ, Slikker W, Bush-Joseph CA, Salata MJ, Nhu SJ. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. Arthroscopy. 2015;31:1199–204.
- 64. Sheen AJ, Stephenson BM, Lloyd DM, Robinson P, Fevre D, Paajanen H, de Beaux A, Kingsnorth A, Gilmore OJ, Bennett D, Maclennan I, O'Dwyer P, Sanders D, Kurzer M. Treatment of the sportsman's groin: British Hernia Society's 2014 position statement based on the Manchester consensus conference. Br J Sports Med. 2014;48:1079–87.
- 65. Ellsworth AA, Zoland MP, Tyler TF. Athletic pubalgia and associated rehabilitation. Int J Sports Phys Ther. 2014;9(6):774–84.
- 66. Khan W, Zoga AC, Meyers WC. Magnetic resonance imaging of athletic pubalgia and the sports hernia—current understanding and practice. Magn Reson Imaging Clin N Am. 2013; 12:97–110.
- Mullens FE, Zoga AC, Morrison WB, Meyers WC. Review of MRI technique and imaging findings in athletic pubalgia and the "sports hernia". Eur J Radiol. 2012;81:3780–92.
- 68. Larson CM. Sports hernia/athletic pubalgia: evaluation and management. Sports Health. 2014;6(2):139–44.
- Hegedus EJ, Stern B, Reiman MP, Tarara D, Wright AA. A suggested model for physical examination and conservative treatment of athletic pubalgia. Phys Ther Sport. 2013;14:3–16.

- Ahumada LA, Ashruf S, Espinosa-de-los-Monteros A, Long JN, de la Torre JI, Garth WP, Vasconez LO. Athletic pubalgia: definition and surgical treatment. Ann Plast Surg. 2005; 55(4):393–6.
- 71. Jakoi A, O'Neill C, Damsgaard C, Fehring K, Tom J. Sports hernia in National Hockey League players: does surgery affect performance? Am J Sports Med. 2013;41:107–10.
- Jacobson T, Allen WC. Surgical correction of the snapping iliopsoas tendon. Am J Sports Med. 1990;18(5):470–4.
- Nelson IR. Keene. Results of labral-level arthroscopic iliopsoas tenotomies for treatment of labral impingement. Arthroscopy. 2014;30(6):688–94.
- 74. Idjadi J, Meislin R. Symptomatic snapping hip: targeted treatment for maximum pain relief. Phys Sportsmed. 2004;32(1):25–31.
- Allen WC, Cope R. Coxa saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3(5):303–8.
- 76. Larsen E, Johansen J. Snapping hip. Acta Orthop Scand. 1986;57(2):168-70.
- 77. Dawes AR, Seidenberg PH. Sonography of sports injuries of the hip. Sports Health. 2014;6(6):531-8.
- Deslandes M, Guillin R, Cardinal E, Hobden R, Bureau NJ. The snapping iliopsoas tendon: new mechanisms using dynamic sonography. Am J Roentgenol. 2008;190:576–81.
- Pelsser V, Cardinal E, Hobden R, Aubin B, Lafortune M. Extraarticular snapping hip: sonographic findings. AJR Am J Roentgenol. 2001;176(1):67–73.
- Farber AJ, Wilckens JH, Jarvis CG. Pelvic pain in the athlete: In: Seidenberg PH, Beutler AI, editors. The sports medicine resource manual. Philadelphia: Saunders Elsevier; 2008. p. 306–27.
- Zoltan DJ, Clancy WG, Keene JS. A new operative approach to snapping hip and refractory trochanteric bursitis in athletes. Am J Sports Med. 1986;14:201–4.
- Board TN, Hughes SJ, Freemont AJ. Trochanteric bursitis: the last great misnomer. Hip Int. 2014;24(6):610–5.
- Mulligan EP, Middleton EF, Brunette M. Evaluation and management of greater trochanter pain syndrome. Phys Ther Sport. 2015;16:205–14.
- Seidenberg PH, Childress MA. Managing hip pain in athletes. J Musculoskelet Med. 2005;22(5):246–54.
- Mallow M, Nazarian LN. Greater trochanteric pain syndrome diagnosis and treatment. Phys Med Rehabil Clin N Am. 2014;25:279–89.
- Klauser AS, Martinoli C, Tagliafico A, Bellmann-Weiler R, Feuchtner GM, Wick M, Jaschke WR. Greater trochanteric pain syndrome. Semin Musculoskelet Radiol. 2013;17:43–8.
- 87. Hall M, Anderson J. Hip pointers. Clin Sports Med. 2013;32:325-30.
- Nouraei SA, Anand B, Spink G, O'Neill KS. A novel approach to the diagnosis and management of meralgia paresthetica. Neurosurgery. 2007;60(4):696–700.
- 89. Cheatham SW, Kolber MJ, Salamh PA. Meralgia paresthetica: a review of the literature. Int J Sports Phys Ther. 2013;8(6):883–93.
- 90. Suh DH, Kim DH, Park JW, Park BK. Sonographic and electrophysiologic findings in patients with meralgia paresthetica. Clin Neurophysiol. 2013;124:1460–4.
- Tagliafico A, Serafini G, Lacelli F, Perrone N, Valsania V, Martinoli C. Ultrasound-guided treatment of meralgia paresthetica (lateral femoral cutaneous neuropathy). J Ultrasound Med. 2011;30:1341–6.
- Philip CN, Candido KD, Joseph NJ, Crystal GJ. Successful treatment of meralgia paresthetica with pulsed radiofrequency ablation of the lateral femoral cutaneous nerve. Pain Pract. 2012;12(5):394–8.
- 93. Mendiguchia J, Alentorn-Geli E, Brughelli M. Hamstring strain injuries: are we heading in the right direction? Br J Sports Med. 2012;46(2):81–5.
- Gabbe BJ, Finch CF, Bennell KL, Wajswelner H. Risk factors for hamstring injuries in community level Australian football. Br J Sports Med. 2005;39(2):106–10.

- Heiderscheit BC, Sherry MA, Silder A, Chumanov ES, Thelen DG. Hamstring strain injuries: recommendations for diagnosis, rehabilitation and injury prevention. J Orthop Sports Phys Ther. 2010;40(2):67–81.
- Kerkhoffs GM, van Es N, Wieldraaijer T, Sierevelt IN, Ekstrand J, van Dijk CN. Diagnosis and prognosis of acute hamstring injuries in athletes. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):500–9.
- 97. Ahmad CS, Redler LH, Ciccotti MG, Maffulli N, Longo UG, Bradley J. Evaluation and management of hamstring injuries. Am J Sports Med. 2013;41(12):2933–47.
- Hamilton B, Tol JL, Almusa E, Boukarroum S, Eirale C, Farooq A, Whiteley R, Chalabi H. Platelet-rich plasma does not enhance return to play in hamstring injuries: a randomised controlled trial. Br J Sports Med. 2015;49:943–50.
- 99. Seidenberg PH, Lynch SA. Hip pain in athletes—when it is not the labrum. Curr Sports Med Rep. 2015;14(5):373–9.
- 100. Roos HP. Hip pain in sport. Sports Med Arthrosc Rev. 1997;5(4):292-300.
- 101. Armfield DR, Kim DH, Towers JD, Bradley JP, Robertson DD. Sports-related muscle injury in the lower extremity. Clin Sports Med. 2006;25(4):803–42.
- 102. Boyd KT, Peirce NS, Batt ME. Common hip injuries in sport. Sports Med. 1997;24(4):273-88.
- 103. Webb CW, Geshel R. Thoracic and lumbar spine injuries. In: Seidenberg PH, Beutler AI, editors. The sports medicine resource manual. Philadelphia: Saunders Elsevier; 2008. p. 285–305.
- Brolinson PG, Kozar AJ, Cibor G. Sacroiliac joint dysfunction in athletes. Curr Sports Med Rep. 2003;2(1):47–56.
- 105. Clavel AL. Sacroiliac joint dysfunction: from a simple pain in the butt to integrated care for complex low back pain. Tech Reg Anesth Pain Manage. 2011;15(2):40–50.
- 106. Tuite MJ. Sacroiliac joint imaging. Semin Musculoskelet Radiol. 2008;12(1):72-82.
- 107. Chen CK, Nizar AJ. Prevalence of piriformis syndrome in chronic low back pain patients. A clinical diagnosis with modified FAIR test. Pain Pract. 2013;13(4):276–81.
- 108. Fishman L, Dombi G, Michaelson C, Ringel S, Rozbruch J, Rosner B, Weber C. Piriformis syndrome: diagnosis, treatment and outcome—a 10 year study. Arch Phys Med Rehabil. 2002;83:295–301.
- 109. Cassidy L, Walters A, Bubb K, Shoja MM, Tubbs RS, Loukas M. Piriformis syndrome: implications of anatomical variations, diagnostic techniques, and treatment options. Surg Radiol Anat. 2012;34:479–86.
- 110. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am. 2004;35(1):65–71.
- 111. Steiner C, Staubs C, Ganon M, Buhlinger C. Piriformis syndrome: pathogenesis, diagnosis, and treatment. J Am Osteopath Assoc. 1987;87(4):318–23.
- 112. Jeong HS, Lee GY, Lee EG, Joe EG, Lee JW, Kang HS. Long-term assessment of clinical outcomes of ultrasound-guided steroid injections in patients with piriformis syndrome. Ultrasonography. 2015;34(3):206–10.
- 113. Santamato A, Micello MF, Valeno G, Beatrice R, Cinone N, Baricich A, Picelli A, Panza F, Logroscino G, Fiore P, Ranieri M. Ultrasound-guided injection of botulinum toxin type A for piriformis muscle syndrome: a case report and review of the literature. Toxins (Basel). 2015;7:3045–56.
- Doursounian L, Maigne JY, Faure F, Chatellier G. Coccygectomy for instability of the coccyx. Int Orthop. 2004;28:176–9.
- 115. Hodges SD, Eck JC, Humphreys SC. A treatment and outcomes analysis of patients with coccydynia. Spine J. 2002;4:138–40.
- 116. Jarvis SK, Abbott JA, Lenart MG, Steensma A, Vancaillie TG. Pilot study of botulinum toxin type A in the treatment of chronic pelvic pain associated with spasm of the levator ani muscles. Aust N Z J Obstet Gynaecol. 2004;44:46–50.

- 117. Perkins R, Schofferman J, Reynolds J. Coccygectomy for severe refractory sacrococcygeal joint pain. J Spinal Disord Tech. 2003;16:100–3.
- 118. Seidenberg PH, Childress MA. Physical examination of the hip and pelvis. In: Seidenberg PH, Beutler AI, editors. The sports medicine resource manual. Philadelphia: Saunders Elsevier; 2008. p. 110–22.
- 119. Geraci Jr MC, Brown W. Evidence-based treatment of hip and pelvic injuries in runners. Phys Med Rehabil Clin N Am. 2005;16(3):711–47.
- 120. McGrory BJ. Stinchfield resisted hip flexion test. Hosp Physician. 1999;35(9):41-2.

Chapter 7 Hip and Pelvis Injuries in Childhood and Adolescence

Mark E. Halstead

Clinical Pearls

- Universal ultrasound screening of infants for developmental dysplasia of the hip (DDH) is not recommended.
- Legg–Calve–Perthes Disease (LCPD) is a common hip disorder affecting children ages 4–8 and is typically felt to be a self-limited condition.
- Slipped capital femoral epiphysis (SCFE) is a common condition affecting middle school and early high school aged children and requires urgent orthopedic attention when diagnosed.
- Transient synovitis of the hip is the most common source of hip pain in children but needs to be distinguished from the more concerning septic arthritis of the hip.
- Snapping hip syndrome is a common source of hip popping that may or may not be painful. It is commonly seen in dancers.
- Avulsion fractures of the pelvis should be strongly considered in the adolescent athlete presenting with an acute injury, a painful pop, and difficulty bearing weight or lifting the leg.

7.1 Case Presentation

A 13-year-old male football offensive lineman presents to clinic with his parents following an acute injury he sustained last night at football practice. He was doing several sprints at the end of his practice and during the second sprint he felt a sharp pain and a pop around the anterior hip and groin. He was unable to bear weight. His

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[©] Springer International Publishing Switzerland 2017 P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_7

school had an athletic trainer who evaluated him and felt he may have "pulled his groin." He was given crutches and iced his hip.

In the office he refuses to bear weight. He is overweight. He has mild pain but significant weakness upon resisted hip flexion. Also has pain endorsed with internally and externally rotating his hip. Tender over the anterior hip joint and anterior superior iliac spine. He is then sent for X-rays.

7.2 Introduction

Disorders of the hip are common in childhood and adolescence. As a child grows older, the most common problems affecting the hip will change. The hip often may be overlooked as a source of pain as disorders may refer pain to the thigh or knee, potentially leading the clinician astray as to the source of the problem, potentially resulting in a delayed or incorrect diagnosis. Clinicians should understand the appropriate anatomy, physical exam techniques and imaging studies of the hip and the common pediatric hip disorders and injuries presented in this chapter.

7.3 Developmental Dysplasia of the Hip

Developmental dysplasia of the hip (DDH) is a general term that refers to conditions of the hip present at birth to early infancy that include a wide spectrum of problems typically affecting the development of the hip joint. This may include problems such as abnormal development of the osseous structures of the hip, subluxation of the femoral head from the acetabulum to complete dislocation of the femoral head from the acetabulum. This condition does not refer to just a click on exam of a stable hip in the newborn. Published estimates of DDH are variable. Incidence ranges from 1.5 to 20 per 1000 newborns [1]. The etiology of DDH is unknown.

Various risk factors have been proposed for the development of DDH. These risk factors include family history of DDH, female gender, first-born child, breech delivery position, postnatal swaddling, oligohydramnios, foot deformity, and large birth size [2].

7.3.1 Diagnosis

Physical exam screening for DDH often is conducted by assessing with the Ortolani and Barlow test. An Ortolani sign is noted as the femoral head moves back into the acetabulum creating a palpable clunk that is typically felt rather than heard. The Barlow test is meant to identify a hip that easily dislocates. Therefore, the Barlow test is intended to dislocate the hip, while the Ortolani test will reduce the hip. These exam maneuvers must be performed one hip at a time and with the diaper removed. There is limited evidence to support re-examination of infants previously found to have normal exams [3]. Other signs on physical exam can include asymmetric gluteal folds, the Galeazzi sign (knee height differences when knees are flexed with feet placed flat on a table), and limited hip abduction.

The American Academy of Orthopedic Surgeons published a clinical practice guideline in 2015, which was endorsed by numerous other organizations, including the American Academy of Pediatrics, regarding screening and management of DDH in infants up to 6 months of age [3]. This evidence based report found moderate support for not performing universal ultrasound screening of newborn infants. Moderate support was noted for screening infants before 6 months of age with an imaging study (X-ray or ultrasound) in infants with one of more of the following risk factors: breech presentation, family history, or history of clinical instability. There was limited evidence supporting obtaining an AP pelvis radiograph instead of ultrasound after 4 months of age. Ultrasound is typically preferred in infants younger than 4 months of age due to limited ossification of the acetabulum and femoral head, limiting the utility of plain radiographs.

7.3.2 Treatment

Various bracing can be used in the infants with DDH. The most commonly used method is through use of the Pavlik harness. The goal of treatment is to maintain the femoral head as close to the acetabulum as possible. In the AAOS review, there was limited evidence to support the use of the von Rosen splint over the Pavlik, Craig, or Frejka splints [3]. Recommendations for duration in the splint vary from 6 weeks to several months [4].

Fig. 7.1 An AP pelvis radiograph in an adult with bilateral hip osteoarthritis secondary to bilateral developmental hip dysplasia (courtesy of Dr. Eric Eutsler, St Louis Children's Hospital)



Complications of delayed treatment can include shortening of the limbs resulting in an abnormal gait, early osteoarthritis of the affected side (Fig. 7.1), and lower back pain [4].

7.4 Legg–Calve–Perthes Disease

LCPD is a self-limiting, idiopathic condition characterized by avascular necrosis of the hip with bone resorption and collapse of the femoral head. This process is followed by reconstitution and regeneration of the femoral head. Originally described over a century ago by the three physicians for whom it is named, it is often just referred to as Perthes disease. Typically normal blood supply and restoration of the femoral head occurs over a period of 2–4 years, although full restoration of the femoral head may not occur in more severe disease [5]. Patients are most commonly affected between the ages of 4 and 8 but may occur as early as age 2 and as late as the teenage years (adolescent Perthes). Males are affected more than females and most cases are unilateral. Bilateral cases occur in 15%. Annual incidence of LCPD varies from 0.9 to 15.6 per 100,000 population ages 0–14 [6].

The etiology of LCPD is still unknown but many possibilities have been proposed that may cause the interruption of blood supply to the femoral head. These include inflammatory conditions, vasculopathies, coagulopathies, insulin-like growth factor-1 pathway abnormalities, genetic predisposition, type II collagen mutations, maternal or passive smoking, vascular occlusion, and trauma [7].

7.4.1 Diagnosis

A child with LCPD presents often with a limp that may or may not be painful. Physical activity often can produce pain. If pain is present, it can be localized to the hip, groin, thigh, or knee. There often is no history of trauma.

Physical exam may be normal outside of the limp but often there is a reduction in hip abduction and internal rotation. Difficulty walking may be present. If the process has been present for a longer time, limited range of motion is more likely and there may be evidence of muscle atrophy. Trendelenburg test may be positive.

Plain film radiographs, an anteroposterior (AP) and frog leg lateral, are often the only diagnostic imaging needed to diagnose LCPD. Findings can include widening of the joint space of the affected hip, lucencies in the femoral epiphysis, and various stages of deformity of the femoral head (Fig. 7.2). These deformities can include flattening, fragmentation, and sclerosis. Further advanced imaging can be conducted with an MRI if LCPD is suspected, as early in this condition, plain radiographs may be normal.

Various classification systems exist for LCPD. The Catterall system is one of the more commonly used systems. Catterall groups are I, II, III, and IV and represent the relationship of the involvement of the femoral head to outcome. Femoral head



Fig. 7.2 An AP view of the left hip demonstrating Legg–Calve–Perthes disease (courtesy of Dr. Eric Eutsler, St Louis Children's Hospital)

involvement is divided from 25% through 100% (total) head involvement. As expected, more head involvement is associated with poorer outcomes although the Catterall system was criticized for poor interobserver reliability [8]. The lateral pillar classification system is a 4 group system (A, B, B/C, or C). The system rates the height of the lateral pillar, which is the height of the lateral aspect of the epiphysis. Group A is no loss, Group B is <50% loss, and group C is >50% loss [8].

7.4.2 Treatment

Treatment goals for LCPD include preventing secondary osteoarthritis of the hip through early diagnosis and keeping deformation of the femoral head to a minimum [5]. Patients presenting prior to age 8 were found to have a better outcome than those presenting after age 8 [9].

Controversy exists as to the most effective management for LCPD. In younger children, nonsurgical management is usually appropriate. Containment of the femoral head with the acetabulum is an important goal of treatment and may be accomplished through abduction casts or braces, which typically is more effective in children younger than age 5. Various surgical treatments exist to accomplish surgical containment of the femoral head although the majority of patients will not need surgical management. A varus osteotomy is one of the more common surgical procedures utilized if surgical management is considered [5].

7.5 Slipped Capital Femoral Epiphysis

SCFE is a condition involving anterior translation and external rotation of the metaphysis of the femur relative to the epiphysis [10]. This condition is considered an urgent orthopedic problem requiring prompt evaluation and treatment. The precise pathophysiologic mechanism of SCFE remains unknown. SCFE affects males greater than females, and overall incidence is around 10 per 100,000 [11, 12]. There is an increased incidence in blacks, Polynesians, Hispanics, and Native Americans compared to whites [11, 12]. Children and adolescents between the ages of 10 and 16 are most commonly affected with an average age of onset around 13 years for boys and 11.5 years for girls [11, 12]. Typically SCFE is associated with obese children and 80% of cases have a body mass index greater than the 95% [13]. The condition is bilateral at onset in about 20% of patients with 90% of slips of the contralateral side occurring within 18 months of the treatment of the initial presenting side [14].

7.5.1 Diagnosis

A typical history for the child or adolescent presenting with an SCFE is that of a limp with groin, hip, thigh, or medial knee pain. One study reported 15% of patients with SCFE had knee pain alone as the presenting complaint [15]. Pain may be vague or significant but often worsens with physical activity in a stable SCFE. Pain may be severe and have marked limp or inability to bear weight with or without history of trauma in unstable SCFE.

Physical examination is often notable for the hip to be externally rotated and flexed. Pain is often increased with passive internal rotation of the hip. Unstable SCFE may present with the patient in a FABER (flexed, abducted, and externally rotated) position of the hip with guarding present with any passive hip maneuvers.

Standard radiographs of an AP pelvis and frog leg lateral are obtained for evaluation for SCFE (Fig. 7.3). Klein's line, extending a line along the superior femoral neck that intersects with the femoral epiphysis, has often been used to diagnose the condition although its sensitivity has been questioned [16, 17]. Steel proposed the metaphyseal blanch sign which is an extra dense area around the physis due to the slip [18]. Other notable findings can include widening or irregularity of the physis and periosteal elevation. Contralateral hip radiographs should be obtained given the possibility of a contralateral SCFE. Advanced imaging such as a bone scan or MRI may help identify the condition in questionable cases although this is often identified with plain radiographs alone.

In cases where presentation occurs before the age of 10, after the age of 16 and age or height is below the 50th percentile, associated endocrine conditions should be considered [19].

Fig. 7.3 An AP pelvis view demonstrating slipped capital femoral epiphysis (SCFE) of the left hip (courtesy of Dr. Eric Eutsler, St Louis Children's Hospital)



7.5.2 Classification

Originally, SCFE was classified as acute (onset of symptoms 3 weeks or less), chronic (symptoms greater than 3 weeks), or acute-on-chronic (acute worsening of symptoms lasting longer than 3 weeks) [20]. Acute slips accounted for 15% of the cases. In 1993, a new classification system was proposed dividing the condition into cases of stable or unstable SCFE [14]. A stable SCFE is considered if the patient is able to walk with or without crutches. Unstable SCFE is a patient who is unable to walk with or without crutches and is associated with increased incidence of AVN. Rates of AVN in stable SCFE are nearly zero, whereas unstable SCFE progress to AVN about 25% of the time [21].

7.5.3 Treatment

The goals of treatment of SCFE are to stabilize the slip, prevent the slip from progressing, and avoiding complications such as AVN. Various methods of treating the slip are utilized and considerable controversy and debate still occurs regarding the ideal method. Methods include single in situ screw fixation, multiple pin fixation, osteotomy, spica cast, or epiphysiodesis. A systematic review of SCFE treatment methods found single screw in situ fixation to be the most effective for stable slips [22].

Controversy exists regarding prophylactic fixation of the contralateral hip. In children with endocrine abnormalities, where the incidence of bilateral SCFE is much higher, prophylactic pinning may be a more reasonable consideration. Unnecessary surgery would often be the case for the majority of patients given the typical 20% estimate of bilateral SCFE in the general population [23].

7.6 Transient Synovitis of the Hip

Transient synovitis of the hip is felt to be a benign, self-limited condition of the hip. It is felt to be the most common cause of acute hip pain in the pediatric aged patient. It typically affects children aged 3-8 and affects boys nearly twice as often as females. Overall annual incidence of the condition is reported at 0.2%, although this is based solely off of European data [24]. It is important to distinguish this condition from septic arthritis of the hip as treatment for septic arthritis requires surgical drainage and intravenous antibiotics [25].

Currently there is no known etiology for transient synovitis. Several publications suggest viral or postviral etiologies as many children have a history of recent gastrointestinal complaints such as vomiting or diarrhea or upper respiratory infection symptoms [24, 26, 27]. Viral synovial cultures have typically been found to be normal. A history of mild trauma may be present.

7.6.1 Diagnosis

Patients typically present with acute onset of anterior hip or groin pain and limited weight bearing ability. Often the hip is held in a flexed, abducted, and external rotated (FABER) position, which maximizes comfort for the patient. The child is generally well appearing, in contrast to the patient with septic arthritis who often is ill appearing. Patients are often afebrile in this condition.

Transient synovitis is often felt to be a diagnosis of exclusion given the broad differential that exists with this presentation. Differential diagnosis includes osteomyelitis, LCPD, SCFE, Lyme arthritis, rheumatoid arthritis, malignancy, septic arthritis, and pelvic abscess.

Plain film radiographs of the hip often are normal but may show slight medial widening of the joint spaces suggesting a joint effusion. Ultrasound is helpful in determining the presence of an effusion but unfortunately does not distinguish between septic arthritis and transient synovitis or other sources of effusion. MRI has been demonstrated to be helpful in distinguishing between transient synovitis and septic arthritis [28].

Kocher et al. proposed an evidence-based algorithm to help predict the likelihood of transient synovitis versus septic arthritis [29, 30]. A history of fever, complete blood cell count (CBC) greater than 12,000/mm³, inability to bear weight and erythrocyte sedimentation rate greater than 40 mm/h are the factors used. A patient with all four criteria had a probability of septic arthritis greater than 99% [29]. Luhmann et al. attempted to validate the Kocher clinical prediction rule and found only a predicted probability of 59% in their study, calling into question the validity and reproducibility of the Kocher algorithm [31]. Their study proposed a threevariable algorithm of history of fever, CBC greater than 12,000/mm³ and prior health-care visit with a predicted probability of septic arthritis at 71%. A 2006 prospective study found fever (oral temperature greater than 38.5 °C) as the best predictor of septic arthritis. A CRP greater than 2.0 mg/dl was also found to be a strong independent risk factor for assessing for suspected septic arthritis [32].

7.6.2 Treatment

Once the diagnosis of transient synovitis has been established, no treatment is required since it is a self-limited condition. Ibuprofen has been found to shorten the course of the condition [33]. Most cases typically resolve within 3–10 days [34]. Recurrence rates have been reported at about 4% [24]. In prolonged courses of this condition, other inflammatory conditions of the hip should be considered. Interestingly, the incidence of LCPD has been demonstrated to be higher than the general population incidence following transient synovitis [24, 35].

7.7 Snapping Hip Syndrome

Extra-articular snapping hip syndrome, or *coxa sultans*, can refer to conditions affecting the lateral hip or the anterior hip where a recurrent pop may be felt or observed with movement of the hip. This has also been described as "dancers hip" as the condition is frequently reported in ballet. This discussion is not intended to include intra-articular pathology such as a labral tear. The lateral hip condition is from the iliotibial band (ITB) moving over the greater trochanter. The anterior condition is thought to occur from the iliopsoas muscle moving over the anterior portion of the femoral head or the iliopectineal eminence. The lesser trochanter has also been described as a source of the snapping. An estimated 5-10% of the population has snapping hip syndrome [36]. Often the condition is bilateral and women are affected more commonly than men [37].

7.7.1 Diagnosis

Patients often will present with a history of painful or painless popping localized deep to the anterior hip and groin or to the lateral hip. When localized to the lateral hip, patients often will describe that the hip pops out of socket. If one asks a patient to recreate this, if they can voluntarily produce the problem, the patient often will rotate the pelvis and a visible and/or audible snapping is seen across the greater

trochanter. Dancers often describe their snapping most commonly when in the "second position."

On physical exam, a snapping may be felt with the iliopsoas condition with simple flexion and extension of the hip. More commonly it is reproduced by starting with the problematic hip in a flexed, externally rotated and abducted position and then internally rotating the hip back into full extension.

Radiographs can be performed to evaluate for other disorders of the bones or joint but in snapping hip are routinely normal. Iliopsoas bursography, with contrast under fluoroscopy, has been described but is not frequently performed. Dynamic ultrasound assessment has been described and may be used to assess and visualize the actual snapping phenomenon and may be able to specifically identify what is truly producing the snapping [38–40].

7.7.2 Treatment

Snapping affecting the lateral hip can be treated non-operatively with rest, avoidance of the activity that produces the problem, stretching of the ITB, and strengthening of the hip girdle. Surgical measures have been described for cases that do not respond to conservative measures including a lengthening of the ITB or via an endoscopic ITB release [41, 42].

Snapping of the iliopsoas should also initially be treated with nonsurgical measures which can include rest, activity modification, and stretching of the iliopsoas. Various modalities may be used for pain control in therapy. Benefit may be obtained through active release techniques. Surgical techniques also have been described including fractional lengthening or release of the iliopsoas [43].

7.8 Avulsion Fractures of the Pelvis

In the skeletally immature athlete, the growth centers in the bone tend to be the weakest link and are most susceptible to injury. In an adult, muscular injuries affecting the pelvis are common. The apophysis, which is a center of growth that provides no longitudinal growth but acts as a muscle attachment site, is frequently injured in the pelvis in the adolescent athlete. The injury is typically caused by rapid muscle contraction, which overpowers the apophysis resulting in the bone being avulsed. Chronic traction of the muscle may cause an "apophysitis" although it is more of a chronic stress injury than a true inflammatory condition as the name would imply.

The majority of pelvic avulsion injuries affect males. A study of 203 avulsion injuries in adolescents found 54% affected the ischial tuberosity, 22% the anterior inferior iliac spine (AIIS), 19% the anterior superior iliac spine (ASIS), 3% the superior corner of the pubic symphysis, and 1% the lesser trochanter [44].

Common sports to experience avulsion fractures include soccer, track and field, gymnastics, football, tennis, fencing, rugby, and wrestling [44]. Essentially any sport that can produce a rapid contracture of a muscle attaching to the pelvic apophyses can produce the injury. The ischial tuberosity apophysis is affected by the proximal hamstring tendons, the ASIS by the sartorius, the AIIS by the rectus femoris, the lesser trochanter by the iliopsoas, and the iliac crest by the transverse abdominis and internal oblique. A summary of the characteristic of the pelvic apophyses can be found in Table 7.1.

7.8.1 Diagnosis

Athletes with acute pelvic apophyseal avulsion injuries present with a history of a sudden onset of localized pain around the anterior hip/groin along with a decreased ability to bear weight. Pain is localized to palpation over the site of the avulsion injury. Weakness is often present, associated with pain to resisted testing of the relevant musculature that attaches to the injured apophysis. Patients often report hearing or feeling a "pop" in the pelvis area. There may be mild swelling. Bruising is not common in the acute avulsion injury.

Overuse injuries of the apophysis typically have a history of insidious onset of pain that often worsens with physical activity. Tenderness is present to palpation over the affected apophysis. Mild weakness may be present, often with pain, to resisted testing of the muscle that attach to the apophysis.

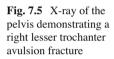
The vast majority of cases can be determined through appropriate physical exam and plain film radiographs, often an AP pelvis and possible frog leg view or oblique view of the affected hip (Figs. 7.4 and 7.5). If the diagnosis is in question, MRI and ultrasound have been reported to be used to help establish the diagnosis [45, 46].

Apophysis	Age of appearance	Age at fusion	Muscle attachment	Common sports seen
Ischial tuberosity	14–16 years	18–21 years	Biceps femoris, semitendinosus, semimembranosus	Soccer, waterskiing, gymnastics, track and field
ASIS	13–15 years	21–25 years	Sartorius	Soccer, track and field, basketball, gymnastics
AIIS	13–15 years	16–18 years	Rectus femoris	Football, rugby, soccer
Iliac crest	13–15 years	15–25 years	Transverse abdominis, gluteus medius, internal oblique	Track and field, soccer
Lesser trochanter	8–12 years	16–18 years	Iliopsoas	Soccer, hockey, football

 Table 7.1
 Characteristics of pelvic apophyseal avulsion injuries



Fig. 7.4 X-ray of the pelvis demonstrating a right anterior inferior iliac spine avulsion fracture





7.8.2 Treatment

Non-operative treatment is the mainstay for the majority of pelvic avulsion injuries. Exceptions to non-operative treatment may be a chronic painful non-unions or the ischial tuberosity avulsion that is displaced greater than 2 cm [47]. Initial treatment consists of crutch use as needed until able to ambulate without a limp. Range of motion can be initiated with progression to light strengthening. Typical healing times can be between 4–12 weeks with the majority of cases healed between 6–8 weeks. A return to sports can be considered when strength has returned to normal, the athlete is pain free and there is evidence of adequate radiographic healing.

7.9 Clinical Case Wrap-Up

The differential for the adolescent presented at the start of the chapter could include SCFE, an avulsion fracture of the pelvis or hip flexor strain. He had X-rays performed to determine if the first two possibilities were present since he is at an appropriate age and has a physical exam and history that would be consistent with the typical presentation for an acute SCFE or avulsion fracture. His X-rays revealed an avulsion fracture of the ASIS and the epiphysis appeared normal with no evidence of slip. He was treated with crutches until normal weight bearing occurred and he was followed up with good radiographic healing of the avulsion fracture by 6 weeks after the injury and allowed to return to sports at that time.

References

- 1. Patel H; Canadian Task Force on Preventive Health C. Preventive health care, 2001 update: screening and management of developmental dysplasia of the hip in newborns. CMAJ. 2001;164:1669–77.
- Shipman SA, Helfand M, Moyer VA, Yamp BP. Screening for developmental dysplasia of the hip: a systematic literature review for the US Preventative Services Task Force. Pediatrics. 2006;117:e557–76.
- American Academy of Orthopedic Surgeons. Detection and nonoperative management of pediatric developmental dysplasia of the hip in infants up to six months of age. http://www. aaos.org/research/guidelines/DDHGuidelineFINAL.pdf. Accessed 31 Oct 2015.
- Alsaleem M, Set KK, Saadeh L. Developmental dysplasia of hip: a review. Clin Pediatr (Phila). 2015;54:921–8.
- 5. Shah H. Perthes disease: evaluation and management. Orthop Clin North Am. 2014;45:87–97.
- Perry DC, Hall AJ. The epidemiology and etiology of Perthes disease. Orthop Clin North Am. 2011;42:279–83.
- Kim HK. Legg-Calve-Perthes disease: etiology, pathogenesis, and biology. J Pediatr Orthop. 2011;31(2 Suppl):S141–6.
- Kim HK, Herring JA. Pathophysiology, classifications, and natural history of Perthes disease. Orthop Clin North Am. 2011;42:285–95.
- 9. Cheng JC, Lam TP, Ng BK. Prognosis and prognostic factors of Legg-Calve-Perthes disease. J Pediatr Orthop. 2011;31(2 Suppl):S147–51.
- 10. Georgiadis AG, Zaltz I. Slipped capital femoral epiphysis: how to evaluate with a review and update of treatment. Pediatr Clin North Am. 2014;61:1119–35.
- 11. Lehmann CL, Arons RR, Loder RT, Vitale MG. The epidemiology of slipped capital femoral epiphysis: an update. J Pediatr Orthop. 2006;26:286–90.
- Loder RT. The demographics of slipped capital femoral epiphysis. An international multicenter study. Clin Orthop Relat Res. 1996;322:8–27.
- 13. Manoff EM, Banffy MB, Winell JJ. Relationship between body mass index and slipped capital femoral epiphysis. J Pediatr Orthop. 2005;25:744–6.
- Loder RT, Richards BS, Shapiro PS, et al. Acute slipped capital femoral epiphysis: the importance of physeal stability. J Bone Joint Surg Am. 1993;75:1134–40.
- Matava MJ, Patton CM, Luhmann S, Gordon JE, Schoenecker PL. Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of initial presentation and treatment. J Pediatr Orthop. 1999;19:455–60.
- 16. Klein A, Joplin RJ, Reidy JA, et al. Roentgenographic features of slipped capital femoral epiphysis. Am J Roentgenol Radium Ther. 1951;66:361–74.

- Pinkowsky GJ, Hennrikus WL. Klein line on the anteroposterior radiograph is not a sensitive diagnostic radiologic test for slipped capital femoral epiphysis. J Pediatr. 2013;162:804–7.
- Steel HH. The metaphyseal blanch sign of slipped capital femoral epiphysis. J Bone Joint Surg Am. 1986;68:920–2.
- Loder RT, Starnes T, Dikos G. Atypical and typical (idiopathic) slipped capital femoral epiphysis. Reconfirmation of the age-weight test and description of the height and age-height tests. J Bone Joint Surg Am. 2006;88(7):1574–81.
- Asdalen R, Weiner D, Hoyt W. Acute slipped capital femoral epiphysis. J Bone Joint Surg. 1974;56A:473–87.
- Zaltz I, Baca G, Clohisy JC. Unstable SCFE: review of treatment modalities and prevalence of osteonecrosis. Clin Orthop Relat Res. 2013;471:2192–8.
- 22. Loder RT, Dietz FE. What is the best evidence for the treatment of slipped capital femoral epiphysis? J Pediatr Orthop. 2012;32:S158–65.
- Castro F, Bennett J, Doulens K. Epidemiological perspective on prophylactic pinning in patients with unilateral slipped capital femoral epiphysis. J Pediatr Orthop. 2000;20:745–8.
- Landin LA, Danielsson LG, Wattsgard C. Transient synovitis of the hip. Its incidence, epidemiology and relation to Perthes' disease. J Bone Joint Surg Br. 1987;69:238–42.
- 25. Haueisen D, Weiner D, Weiner S. The characterization of 'transient synovitis of the hip' in children. J Pediatr Orthop. 1986;6:11–7.
- Kastrissianakis K, Beattie TF. Transient synovitis of the hip: more evidence for a viral aetiology. Eur J Emerg Med. 2010;17:270–3.
- Leibowitz E, Levin S, Torten J, Meyer R. Interferon system in acute transient synovitis. Arch Dis Child. 1985;60:959–62.
- Yang W, Im S, Lim G, Chun HJ, Jung NY, Sung MS, Choi BG. MR imaging of transient synovitis: differentiation from septic arthritis. Pediatr Radiol. 2006;36:1154–8.
- 29. Kocher MS, Zurakowski D, Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence based clinical prediction algorithm. J Bone Joint Surg Am. 1999;81:1662–70.
- Kocher M, Zurakowski D, Kasser J. Validations of a clinical prediction rule for the differentiation of septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg. 2004;86:1629–35.
- Luhmann SJ, Jones A, Schootman M, Gordon JE, Schoenecker PL, Luhmann JD. Differentiation between septic arthritis and transient synovitis of the hip in children with clinical prediction algorithms. J Bone Joint Surg Am. 2004;86-A:956–62.
- 32. Caird M, Flynn J, Leung Y, Millman J, D'Italia J, Dormans J. Factors distinguishing septic arthritis from transient synovitis of the hip in children. A prospective study. J Bone Joint Surg Am. 2006;88:1251–7.
- 33. Kermond S, Fink M, Graham K, Carlin J, Barnett P. A randomized clinical trial: should the child with transient synovitis of the hip be treated with non-steroidal anti-inflammatory drugs? Ann Emerg Med. 2002;40:294–9.
- 34. Do T. Transient synovitis as a cause of painful limps in children. Curr Opin Pediatr. 2000;12:48-51.
- 35. Mukamel M, Litmanovitch M, Yosipovich Z, Grunebaum M, Varsano I. Legg-Calve-Perthes disease following transient synovitis. How often? Clin Pediatr (Phila). 1985;24:629–31.
- 36. Byrd JW. Snapping hip. Oper Tech Sports Med. 2005;13:46-54.
- 37. Lewis CL. Extra-articular snapping hip: a literature review. Sports Health. 2010;2:186-90.
- Winston P, Awan R, Cassidy JD, Bleakney RK. Clinical examination and ultrasound of selfreported snapping hip syndrome in elite ballet dancers. Am J Sports Med. 2007;35:118–26.
- 39. Choi YS, Lee SM, Song BY, Paik SH, Yoon YK. Dynamic sonography of external snapping hip syndrome. J Ultrasound Med. 2002;21:753–8.
- 40. Pelsser V, Cardinal E, Hobden R, Aubin B, Lafortune M. Extraarticular snapping hip: sonographic findings. AJR Am J Roentgenol. 2001;176:67–73.

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- 41. Provencher MT, Hofmeister EP, Muldoon MP. The surgical treatment of external coxa saltans (the snapping hip) by Z-plasty of the iliotibial band. Am J Sports Med. 2004;32:470–6.
- Ilizaliturri Jr VM, Martinez-Escalante FA, Chaidez PA, Camacho-Galindo J. Endoscopic iliotibial band release for external snapping hip syndrome. Arthroscopy. 2006;22:505–10.
- Hoskins JS, Burd TA, Allen WC. Surgical correction of internal coxa saltans: a 20-year consecutive study. Am J Sports Med. 2004;32:998–1001.
- 44. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skeletal Radiol. 2001;30:127–31.
- 45. Blankenbaker D, DeSmet A. The role of ultrasound in the evaluation of sports injuries of the lower extremities. Clin Sports Med. 2006;25:867–97.
- 46. Pisacano R, Miller T. Comparing sonography with MR imaging of apophyseal injuries of the pelvis in four boys. Am J Roentgenol. 2003;181:223–30.
- 47. McKinney B, Nelson C, Carrion W. Apophyseal avulsion fractures of the hip and pelvis. Orthopedics. 2009;32:42.

Chapter 8 Specific Considerations in Geriatric Athletes

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Key Points

- More than 10 million Americans have osteoporosis and an estimated additional 33.6 million have low bone density of the hip
- Risk factors for osteoporosis include physical inactivity, low body weight, low dietary intake of calcium and vitamin D, excessive alcohol use, smoking, certain medications or medical conditions, personal history of low-trauma fractures, family history of osteoporosis or low-trauma fractures, white or Asian race, and female sex.
- Hip fractures increase mortality 10–20% within 1 year. Approximately 20% of patients who sustain a hip fracture need long-term nursing home care. Only 40% of hip fracture patients regain their previous level of independence.
- Screening and preventive measures are still not regularly being recommended and implemented in primary care despite current clinical knowledge about osteoporosis and hip fractures.
- Exercise programs for geriatric athletes should include lower body weightbearing, balance, and flexibility in order to maintain or improve function of the hip and pelvis and to prevent falls and hip fractures.

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P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_8

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8.1 Case Presentation

SA is a 65-year-old white female master triathlete who sustained a displaced right femoral neck fracture after slipping and falling at the pool where she trains. She underwent a right total hip arthroplasty and is following up now 6 weeks postoperatively. She has had no post-operative problems or complications and completed 4 weeks of physical therapy. She would like to start exercising but expresses that she is concerned about falling again.

Her history reveals that she is 17 years postmenopausal and has never taken any hormone replacement therapy or other medications or supplements regularly. She does not have any chronic medical conditions that she is aware of and does not take any medications. She and her husband both retired 10 years ago and they spend much of their time at their country club playing tennis, golfing, and swimming. They also have a condo in the mountains where they go 3–4 times each year to ski, bike, and hike.

She eats what she considers a healthy diet, but avoids dairy products because of lactose intolerance. She has never been a smoker, but drinks 2–3 glasses of red wine with dinner a few nights each week as she has heard it's good for your health. Her menstrual cycles started when she was 15 and she had about 6–8 menstrual cycles per year throughout her adult life. She admits that she strove to keep herself quite thin when she was younger as miniskirts and Twiggy were fashionable.

Family History Her mother had severe kyphosis and at age 80 suffered a hip fracture and was admitted to a nursing home where she subsequently died within a year. No other significant family history is noted.

Physical Exam She is 64 inches tall, but states she has always been 65 inches. She weighs 124 pounds, with a body mass index of 20.6. She is fair-skinned with blue eyes and white hair. She has slightly rounded shoulders and a slightly protuberant abdomen. She walks unassisted without a limp, but with a slow gait. She uses her arms to assist her when rising from a chair. Her surgical site is well healed and her hips have adequate range of motion to permit her to get on and off the examination table without difficulty. It is noted that she moves very cautiously. The remainder of her physical exam is unremarkable.

8.2 Age-Related Physiological Changes

8.2.1 Bone

Bone is a dynamic tissue that is constantly remodeling. In the adult skeleton, bone homeostasis involves a coupled process of bone formation by osteoblasts and bone resorption by osteoclasts; osteoporosis occurs when bone resorption supersedes bone formation. Normally, during childhood, bone formation outpaces bone resorption,

resulting in increasing bone density until approximately age 18 when 90% of bone mass is achieved. In young adulthood, bone mineral content remains relatively stable. In midlife, resorption outpaces formation resulting in net loss of bone mineral content. Women typically undergo an accelerated phase of bone loss that starts about 1 year prior to final menses and lasts approximately 3 years. During this time, there is an estimated bone loss of 7% per year at the femoral neck and 6% per year in the lumbar spine [1].

This gradual loss of bone can lead to osteoporosis which is defined as the reduction of bone mineral density, resulting in an increased risk of fractures. These fractures are especially common at the vertebral bodies, femoral neck, distal forearm, and proximal humerus [2]. Currently, it is estimated that there are 10 million people in the USA with osteoporosis and that there are over 1.5 million osteoporotic fractures annually [3]. The decrease in quantity and quality of bone associated with osteoporosis leads to an increased risk of fracture, especially of the hip and spine. Given the insidious onset, the diagnosis of osteoporosis is frequently overlooked until a fracture has occurred and, in some cases, the diagnosis can be overlooked even after a fracture has taken place. Even with substantial evidence that a prior fracture predicts a subsequent fracture, less than 30% of postmenopausal women and less than 10% of men with prior fracture are treated [4].

There are many known risk factors for osteoporosis. Intrinsic risk factors include being female, advanced age, low peak bone mass, low body weight, white or Asian race, past history of low-trauma fracture, family history of low-trauma fracture or osteoporosis, and low levels of circulating estrogens [3, 4]. Extrinsic factors include cigarette smoking, excessive alcohol intake, low levels of calcium intake, low levels of vitamin D intake, and physical inactivity [3, 4]. Age is one of the most important risk factors for women and is one of the criteria used to determine who should be screened by further imaging.

In addition to the above risk factors, some medications are associated with reduced bone mass in adults (Table 8.1). Some underlying medical conditions such as gastrointestinal diseases (e.g., malabsorption syndromes and inflammatory bowel disease), hematologic disorders (e.g., thalassemia and pernicious anemia), and hypo-gonadal states can also contribute to osteoporosis [3].

The fracture risk assessment tool (FRAX) was developed by the World Health Organization to predict the risk of osteoporotic fracture for a person over the next 10 years and the calculator can be found online at http://www.sheffield.ac.uk/FRAX/. The output is a 10-year probability of hip fracture and the 10-year probability of a major osteoporotic fracture (clinical spine, forearm, hip, or shoulder fracture).

Osteoporosis Evaluation A thorough history is the most important part of the evaluation for osteoporosis. Using the FRAX tool can help quantify a patient's risk factors as it takes into account age, sex, body mass index, previous low-trauma fracture, parental hip fracture, current smoking status, corticosteroid use, alcohol use, rheumatoid arthritis, and other secondary causes of osteoporosis. The FRAX algorithm should be used in conjunction with clinical judgement as there are some items from a patient's history it does not capture such as a personal history of multiple fractures or a significant fall history.

Table	8.1	Drugs	that 1	nay
lead to	o dec	reased	bone	mass

Aluminum
 Aromatase inhibitors
• Anticonvulsants (phenobarbital,
phenytoin, valproate)
Cytotoxic drugs
• Ethanol (excess use)
Glucocorticoids
Gonadotropin-releasing hormone
agonists
• Heparin (long-term use)
• Lithium
• Progesterone (parenteral, long-acting)
• Thyroxine (supra-physiologic doses)

A physical exam, looking for risk factors for falling, such as decreased mobility or strength (e.g., unable to rise from a chair without using hands), decreased proprioception, decreased visual acuity, signs of osteoporosis (i.e., evidence of vertebral compression fractures with loss of height, kyphosis, and overly protuberant abdomen) should be done. The physical exam should also rule out signs of other possible causes of metabolic disease (i.e., cushingoid features, goiter, jaundice, etc).

The information gathered from the history and physical will help identify patients for whom further diagnostic imaging is indicated.

The United States Preventative Services Task Force (USPSTF) recommends screening for osteoporosis in women aged 65 years or older and in younger women whose fracture risk is equal to or greater than that of a 65-year-old white woman with no additional risk factures [5]. (SOR-B) Dual energy X-ray absorptiometry (DEXA) is currently considered the gold standard for measuring BMD [3]. The areal unit of measurement is grams per square centimeter (g/cm²), although it is usually reported as a T-score, which is a standard deviation without units of measurement. The World Health Organization (WHO) has defined osteoporosis on the basis of BMD as measured by DEXA. BMD that is greater than 2.5 standard deviations below the mean for a young, white, healthy female is defined as osteoporosis. BMD 1.0–2.5 standard deviations below the mean is defined as low bone mass (previously called osteopenia).

Other methods used to measure BMD include quantitative computed tomography (QCT) and quantitative ultrasound (QUS). However, DEXA of the hip is the best predictor of future hip fracture and the only imaging recommended for serial evaluations of patients being treated for osteoporosis.

Once the diagnosis of osteoporosis has been made, there are a number of pharmacological and nonpharmacological treatments available. Some of the treatments are also used for the prevention of osteoporosis in high-risk individuals. Nonpharmacological interventions for treatment and prevention of osteoporosis are listed in Table 8.2.

 Table 8.2
 Nonpharmacologic interventions that may help prevent osteoporosis-related fractures

- · Diet with adequate calories, protein, and nutrients
- Weight-bearing exercise
- Strength-training exercise
- · Balance-training exercise
- Tobacco cessation
- · Reducing excessive alcohol intake
- Vision correction
- · Assessment of any medical conditions that may decrease bone density or increase the risk for falls
- Assessment of any medications that may decrease bone density or increase the risk for falls
- · Elimination of tripping hazards in the home, work, and social environments

Pharmacological treatments used to prevent and treat osteoporosis include antiresorptive drugs such as the bisphosphonates (alendronate, risedronate, ibandronate), calcitonin, estrogen, and partial estrogen agonists and antagonists (previously known as selective estrogen receptor modulators or SERMs), such as raloxifene. The bisphosphonates reduce bone turnover and subsequently prevent bone loss. Calcitonin inhibits osteoclasts, and previous studies have shown a decrease in the incidence of vertebral fractures as well as an analgesic effect when administered after acute vertebral fractures [4]. Currently, there is no significant information on the effect of calcitonin on early menopausal BMD, so it is not recommended within the first 5 years of menopause. Estrogen also inhibits bone resorption, increases total hip BMD, and reduces the risk of fracture at the hip, spine, and wrist, and is currently approved for prevention, but not treatment for osteoporosis. Raloxifene is able to exert estrogen-like effects on the skeleton, although not as effectively as estrogen or the bisphosphonates [4]. Calcium and vitamin D supplementation is also important therapy and can be prescribed for prevention of osteoporosis, and should be prescribed for any patients taking bisphosphonates for prevention or treatment of osteoporosis. Not all medications used to treat osteoporosis have data supporting reduced fractures of all kinds (spine, hip, nonvertebral fractures). Specifically, ibandronate (Boniva) does not show reduced hip and nonvertebral fractures. Calcium carbonate and gluconate will require an acidic environment, so patients on PPIs will need calcium citrate supplementation, which is effective in acidic or alkaline environment [6].

The fundamental goal of managing patients at high risk for osteoporosis is to prevent fractures and loss of function, and also to prevent or decrease pain. While there is evidence of increased bone density and decreased risk of fracture with pharmacologic intervention, there are still remaining questions about the effectiveness of interventions in asymptomatic populations [4, 7].

While there is general consensus that women over the age of 65 should have a BMD test, for women under age 65, BMD testing is generally reserved for those considered to be "at risk" for osteoporosis [4, 7]. What defines "at risk" is not universally agreed upon, and any data on using medications to prevent bone loss in

perimenopausal women with normal BMD are extremely limited. Also, the risk factors, testing, and treatment of osteoporosis in populations other than postmenopausal white women need to be further investigated.

8.2.2 Muscle

It is well established that muscular strength declines with age [8]. Sarcopenia is a multifactorial physiologic change in aging that is caused by a loss of type II muscle fibers and therefore, a loss in muscle mass. It is estimated that muscle mass decreases 30-40% in relation to body weight between the ages of 30 and 80 [9]. How much of the muscle mass and strength loss is directly related to aging and how much is related to disuse is unclear.

Studies have shown that resistance training can minimize, and in some case reverse, this loss of strength and muscle mass, but much more research needs to be done in order to determine the best management strategy. At this time, an operational definition of sarcopenia and what is "pathological" and puts a patient at increased risk for a bad outcome such as a fall, fracture, or loss of independence versus what is normal physiological loss of muscle mass and does not increase a patient's risk is not clear. The Foundation for the National Institutes of Health Sarcopenia Project is working on establishing definitions, evidence-based endpoints, and functional limitations to be used in future clinical trials.

8.2.3 Cartilage, Ligaments, and Tendons

Aging leads to a decrease in the quantity and quality of synovial fluid and a decrease in cartilage proteoglycan content. This leads to decreased water content and elasticity of the cartilage in weight-bearing joints, including the hip [10].

Elasticity of the connective tissue of ligaments and tendons declines with age, most likely secondary to changes in collagen, elastin, and water content. This can lead to aging collagen being more subject to overload failure as well as leading to increased stress and force across the joint because of decreased flexibility and decreased range of motion [10].

8.2.4 Balance

Balance can be defined as the ability of an individual to maintain his or her center of gravity within specific boundaries and may be either static or dynamic [11]. The visual, vestibular, and proprioceptive system are all important in maintaining

Table 8.3 Causes of falls in older persons: summary of 12 large studies	Cause	Mean (%) ^a	Range ^b
	Accident and environment related	31	1–53
	Gait and balance disorders or weakness	17	4–39
	Dizziness and vertigo	13	0-30
	Drop attack	9	0-52
	Confusion	5	0-14
	Postural hypotension	3	0–24
	Visual disorder	2	0-5
	Syncope	0.3	0-3
	Other specified causes ^c	15	2-39
	Unknown	5	0-21

^aMean percent calculated from the 3628 reported falls

^bRanges indicate the percentage reported in each of the 12 studies

"This category includes arthritis, acute illness, drugs, alcohol, pain, epilepsy, and falling from bed

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balance, and all can suffer degenerative changes with aging. One of the most serious outcomes of poor balance is falls.

The accumulative exposure of degenerative, infective, and injurious processes to the sensory, motor, and adaptive systems, combined with slowed protective reflexes, leads to a decreased ability to withstand unexpected perturbations with advancing age [11, 12].

At least 18% of the community-dwelling population over the age of 70 has substantial visual impairment from conditions such as cataracts, glaucoma, or macular degeneration [12]. Three case–control studies demonstrated a significant increase in falls and hip fractures among both men and women with impaired vision [12].

Aging has a significant effect on the vestibular system, with an estimated neuronal loss of 3 % per decade after age 40 [11].

Age-associated changes in postural control, muscle strength, and step height can impair a person's ability to avoid a fall after an unexpected trip or while reaching or bending. These changes can be due to arthritis or decreased range of motion secondary to loss of elasticity in muscle, tendon, or ligaments.

The most common causes of falls in older person are listed in Table 8.3. The most common risk factors for falls are listed in Table 8.4.

About one out of three community-dwelling people over the age of 65 sustain a fall each year [12]. About 1% of those who fall sustain a hip fracture, which carries a 1-year mortality rate of 20-30% [12]. Among community-dwelling people who fall and sustain a hip fracture, between 25% and 75% never recover their pre-fracture level of function [12].

Table 8.4 Risk factors forfalls: analysis of 16 studies

Risk factor	Mean RR–OR ^a		
Lower extremity	4.4		
weakness			
History of falls	3.0		
Gait deficit	2.9		
Balance deficit	2.9		
Use assistive device	2.6		
Visual deficit	2.5		
Arthritis	2.4		
Impaired ADL	2.3		
Depression	2.2		
Cognitive impairment	1.8		
Age >80 years	1.7		

^aRelative risk ratio (RR) calculated for prospective studies. Odds ratio (OR) calculated for retrospective studies

From Rubenstein LZ, Josephson KR. Falls and their prevention in elderly people: what does the evidence show? Med Clin North Am 2006;90:807–24; used with permission

8.3 Exercise to Prevent Injury

8.3.1 Weight-Bearing Exercise

A Cochrane review of 18 randomized controlled trials showed that walking is effective in increasing the density of the bone mass in the spine and the hip [7]. Aerobics and weight-bearing and weight-resistance exercises were also found to be effective; however, the review gave no specific information about the amount of walking or exercise needed.

Physical activity provides a positive stimulus for bone formation, but only at the sites of the skeleton that are physically stressed. For example, upper body weight training will not increase bone mass at the hip. Also, the physical activity needs to be continued, as bone mass will return to its previous level if activity returns to the previous level. With a complete lack of activity, such as with immobility or bedrest, bone loss occurs.

Weight training is also an effective way to approach age-related sarcopenia. Many studies have confirmed an increase in muscle strength and muscle mass with weight training, even in the very elderly. Current evidence provides support for resistance training as a primary intervention to prevent muscle and strength loss. Numerous studies involving both men and women over a wide range of ages (40–90) have shown that strength training aids in increasing muscle strength and muscle hypertrophy as well in increasing bone strength and reducing injuries [13].

8.3.2 Flexibility Exercise

Research regarding flexibility exercises in the elderly is limited. It is generally accepted, however, that flexibility training can optimize joint range of motion and musculoskeletal function, and thus reduce injury potential and enhance functional capability [10].

8.3.3 Balance Exercise

Numerous studies have shown that exercise can improve fall risks such as poor balance, gait impairment, and muscle weakness [13]. The fall rate in elderly patients is estimated to be 33% per year, though less than half of these patients admit these falls to their healthcare provider. Effective balance programs are associated with a 50% reduction in falls [14]. Tai Chi, which consists of slow, rhythmic movements that require trunk rotation, dynamic weight shifting, and coordination between upper and lower extremity movements, and other programs that focus on muscle and balance training are effective [9]. Balance training often includes a range of static and dynamic exercises such as standing on one foot, tandem standing, ball games, moving to music, and functional exercises that involve bending, reaching, and transferring weight [12].

8.4 Exercise After Hip Surgery

Senior citizens are the fastest growing segment of the American population, with 40.3 million people over the age of 65 in 2010 [15]. The majority of total hip arthroplasties (THA) done for end-stage osteoarthritis and the majority of open reduction internal fixation (ORIF) surgeries for hip fractures are done in this population.

Hip fractures are one of the most devastating injuries an elderly individual can suffer. While hip fractures in younger patients are usually due to high-impact trauma, hip fractures in the geriatric population usually result from a fall from a standing height. This level of force is not usually enough to fracture a bone of normal density, but can be enough to cause a fracture in osteoporotic bone, especially if a person's ability to reach out and cushion the fall is compromised by slowed reflexes, arthritis, or any other condition that limits their movement.

Women account for 80% of all hip fractures, and the incidence of hip fractures increases dramatically with age, from 2 fractures per 100,000 among white women under age 35 to over 3000 per 100,000 among white women 85 years and older [16].

Hip fractures are repaired surgically in the majority of cases. Either ORIF or THA may be done depending on the exact location and extent of the fracture, the expectation for level of activity after surgery, and any underlying pre-existing pathology of the hip joint. THA is also done for patients with end-stage osteoarthritis and has been shown to greatly improve mobility and quality of life [15].

Exercise following hip surgery should be encouraged, but should also be done in a safe environment. Supervised physical therapy is appropriate until a patient is able to ambulate and transfer safely either with or without an assistive device. Any rehabilitation or exercise program should address any underlying muscular weakness or limited mobility that the patient may have had prior to the surgery. The exercise program should also include weight-bearing and resistance training to prevent continued bone loss from disuse. Exercises that promote balance and proprioception should be incorporated to decrease the chance for future falls and further injury. Any exercise program should enhance psychological well-being by helping the patient overcome any possible fear of falling or further injury. The exercise program should also include enjoyable activities and help the patient return to their prior level of social functioning.

For the first 3 months after THA, patients must observe precautions to avoid dislocating the hip, but should participate in a physical therapy program to increase their strength and improve their gait to the point that they can ambulate without an assistive device. After the first 12 weeks, patients may begin to resume some recreational activities [17].

There is concern that after THA excessive load-bearing will increase the amount of wear on the joint, increasing the chances of early loosening and implant failure. This has led most surgeons to conservatively recommend low-impact activities such as swimming, cycling, and walking, and discourage high-impact activities such as football, handball, basketball, soccer, or hockey [18]. However, there are not many prospective randomized studies on athletic activities after THA in the current literature and not much evidence and information available to assist in counseling patients on sporting activities following THA.

There has been some concern that more active patients were at increased risk for revision surgery because wear on the prosthesis is correlated to number of cycles in vivo, but any harmful effects of sports participation were not noticed until 10 years postoperatively [18, 19]. It is not recommended that patients try to pick up any new activities after their THA that may be technically difficult to learn, as the incidence of injury increases with inexperience. However, for patients who are experienced in some activities and would like to do them occasionally for recreation, there is no general consensus that these activities must be avoided. However, it is recommended that any exercise that is done several times each week to maintain aerobic fitness be a low-impact activity such as level surface walking or bicycling, swimming and golf to avoid excessive wear on the hip joint [20, 21]. Higher-impact activities have historically been discouraged, but with improved prosthetic implants, a younger population, and a greater number of surgeons gaining comfort with revision surgery, there is a trend toward less activity restrictions [22–24].

8.5 Case Follow-up

SA is a white postmenopausal female who has sustained a low-trauma fracture and should be considered for osteoporosis treatment. Treatment includes non-pharmacologic measures such as continuing weight-bearing exercise and adding some strength, balance, and flexibility training. She should also start a first-line medication such as a bisphosphonate as well as calcium and vitamin D supplementation in adequate amounts.

The initial appointment is spent answering her questions about osteoporosis, addressing her fears about the possibility of another hip fracture, discussing measures to prevent falls, and recommending testing and treatment.

She does not like to take pills, but is agreeable to an over-the-counter chewable calcium supplement with vitamin D and is willing to take a bisphosphonate she has heard about that only has to be taken annually intravenously. She is also agreeable to decreasing her wine to only one glass with dinner once or twice each week. She will schedule her baseline DEXA scan to be completed before her next appointment at which time administration of zoledronic acid is planned. She has been given all pertinent information about osteoporosis, her DEXA scan, and zoledronic acid to review prior to her upcoming appointments.

She wants to resume swimming at this time and she is encouraged to do so. Given her underlying osteoporosis and some of the deficits noted on exam (inability to rise from a chair without using her arms), she is referred to physical therapy to be evaluated and instructed in appropriate strength, balance, and flexibility exercises. She states she is hesitant to start exercising outside as she is nervous about falling again, but she is willing participate in the physical therapy program and take Tai Chi classes at the local community center in order to build her strength and stability. At her next appointment, in order to get a better idea of her risk of falling, she will have a functional assessment of her gait, core strength and stability, balance, proprioception, and vision in addition to reviewing her DEXA results and getting her bisphosphonate injection. At that time, she can be advised on how to progress her activity and on the benefits and risks of various activities given her underlying osteoporosis and her hip prosthesis.

References

- American College of Obstetricians and Gynecologists. Practice Bulletin Number 129: osteoporosis. Obstetrics and Gynecology. 2012;120(3):718–34.
- National Osteoporosis Foundation. Clinician's guide to prevention and treatment of osteoporosis. Washington, DC: National Osteoporosis Foundation; 2014. http://nof.org/articles/7. Accessed 10 July 2015.
- Lane NE. Epidemiology, etiology, and diagnosis of osteoporosis. Am J Obstet Gynecol. 2006;194:S3–11.
- Delaney MF. Strategies for the prevention and treatment of osteoporosis during early postmenopause. Am J Obstet Gynecol. 2006;194:S12–23.

- United States Preventive Services Task Force. Recommendations and rationale: screening for osteoporosis in postmenopausal women. Ann Intern Med. 2002;137:526–8.
- Gehrig L, Lane J, O'Connor MI. Osteoporosis: management and treatment strategies for orthopedic surgeons. J Bone Joint Surg. 2008;1362–1374.
- Johnell O, Hertzman P. What evidence is there for the prevention and screening of osteoporosis? WHO Regional Office for Europe (Health Evidence Network report); 2006. http://www. euro.who.int/document/e88668.pdf. Accessed 5 Dec 2008.
- 8. Bemben MG. Age-related alterations in muscular endurance. Sports Med. 1998;25(4):259-69.
- 9. Epperly T, Palmer T. The older athlete. In: Birrer RB, O'Connor FG, editors. Sports medicine for the primary care practitioner, 4th ed.; CRC Press. Taylor and Francis. 2015.
- Micheo W, Soto-Quijano DA, Rivera-Tavares C, et al. The geriatric runner. In: O'Connor FG, Wilder RP, editors. Textbook of running medicine. New York: McGraw-Hill; 2001.
- 11. Matsumura BA, Ambrose AF. Balance in the elderly. Clin Geriatric Med. 2006;22:395–412.
- 12. Rubenstein LZ, Josephson KR. Falls and their prevention in elderly people: what does the evidence show? Med Clin North Am. 2006;90:807–24.
- 13. Escamilla RF. Exercise testing and prescription. In: Speer KP, editor. Injury prevention and rehabilitation for active older adults. Champaign: Human Kinetics; 2005. p. 19–48.
- Falls among older adults: an overview. CDC. http://www.cdc.gov/homeandrecreationalsafety/ falls/adultfalls.html. Accessed 10 July 2015.
- The Older Population. 2010 census briefs. 2010. http://www.census.gov/prod/cen2010/briefs/ c2010br-09.pdf. Accessed 10 July 2015.
- 16. United States Department of Health and Human Services. Bone health and osteoporosis: a report of the surgeon general. Rockville: US Department of Health and Human Services, Office of the Surgeon General; 2004.
- 17. Total hip arthroplasty/hemiarthroplasty protocol 2011. The Brigham and Women's Hospital, Inc., Department of Rehabilitation Services.
- 18. Kuster MS. Exercise recommendations after total joint replacement: a review of the current literature and proposal of scientifically based guidelines. Sports Med. 2002;32(7):433–45.
- 19. Schmalzried TP, Shepherd EF, Dorey JF, et al. The John Charnley award. Wear is a function of use, not time. Clin Orthop Relat Res 2000:36.
- 20. Chatterji U, Ashworth M, Lewis P, Dobson P. Effect of total hip arthroplasty on recreational and sporting activity. ANZ J Surg. 2004;74(6):446–9.
- Swanson EA, Schmalzried TP, Dorey FJ. Activity recommendations after total hip and knee arthroplasty: a survey of the American Association for Hip and Knee Surgeons. J Arthroplasty. 2009;24(6 Suppl):120–6.
- 22. Klein GR, et al. Return to athletic activity after total hip arthroplasty. J Arthroplasty. 2007;22(2):171–5.
- Healy WL, Iorio R, Lemos MJ. Athletic activity after joint replacement. Am J Sports Med. 2001;29(3):377–88.
- Chatterji U, Ashworth M, Lewis P, Dobson P. Effect of total hip arthroplasty on recreational and sporting activity. ANZ J Surg [serial on the Internet]. (2004, June), [cited August 3, 2015]; 74(6): 446–9.

Chapter 9 Hip and Pelvis Injuries in Special Populations

Dorianne R. Feldman, Tiffany Vu, Marlís González-Fernández, and Brian J. Krabak

Clinical Pearls

- Heterotopic ossification is a common complication in disabled populations that can significantly affect hip range of motion, cause pain, and limit functional mobility.
- The most common musculoskeletal injuries among amputee athletes are sprains and strains to the lumbar spine and sacroiliac joint on the uninvolved side.
- Athletes with cerebral palsy typically experience lower extremity injuries that involve the patellofemoral joint which are due to muscle spasms of the surrounding muscles.
- Wheelchair athletes are at a greater risk of long bone fractures due to osteoporosis.
- Disabled athletes require sport- and disability-specific equipment to minimize injury.

9.1 Case Presentation

FM is a 33-year-old South Asian male with a history of a traumatic C5–6 complete spinal cord injury (SCI) who presents with right hip pain and swelling. At the age of 19, FM sustained a C5 posterior vertebral fracture.

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[©] Springer International Publishing Switzerland 2017 P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_9

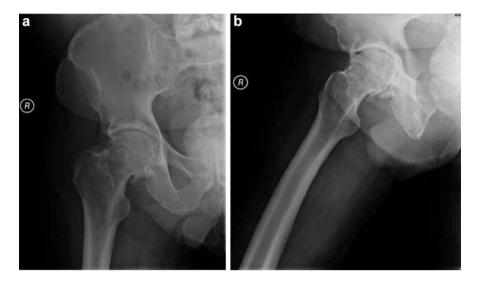


Fig. 9.1 Heterotopic ossification of the right hip. An irregular linear lucency is seen in the subcapital region. (a) AP view of the right hip. (b) Lateral view of right hip

For the last 10 years, he has been actively involved in wheelchair athletics such as swimming, skiing, rugby, and wheelchair racing. The patient has complained of right hip pain and mild swelling for approximately 3 years. He then began noticing increased difficulty with hip flexion. The pain worsened and limited hip range of motion, interfering with transfers, bed mobility, and wheelchair positioning. He began requiring more assistance with all functional mobility skills and activities of daily living, significantly affecting his quality of life and prompting him to seek medical care.

FM complained of pain in the anterior–lateral region of the right hip. Musculoskeletal examination was consistent with C6 tetraplegia. Both lower extremities were insensate and atonic. There was significant atrophy throughout the trunk and bilateral lower extremities. Right hip examination demonstrated warmth to palpation. There was no erythema. Passive range of motion was significantly limited in external rotation, internal rotation, and flexion. Radiographs of the right hip revealed extrinsic bone formation lateral to the subcapital femoral region, most evident on anterior–posterior view (Fig. 9.1).

The differential diagnosis for right hip pain is extensive in both able-bodied and disabled athletes. In the absence of obvious trauma, the differential diagnosis is narrower and includes conditions such as osteoporotic fractures, osteoarthritis, septic arthritis, heterotopic ossification, and hip dislocation. In this case, the most likely diagnosis is heterotopic ossification. Heterotopic ossification (HO) is common in SCI, particularly in athletes. In SCI, the hip is the most common joint affected by HO. Radiography was diagnostic, demonstrating abnormal periarticular bone deposition. Ectopic bone formation is responsible for the limitations in range of motion and pain. In this chapter, we focus our discussion on the most common causes of hip and pelvis dysfunction in the disabled athlete population.

9.2 Introduction

For centuries, able-bodied individuals have engaged in athletic competitions. However, it has only been since the middle of the twentieth century that individuals with physical impairments have been able to participate in competitive sporting events. As a result, functional impairment is no longer a barrier to participation in athletics, and interest in accommodating the needs of those with disabilities continues to grow.

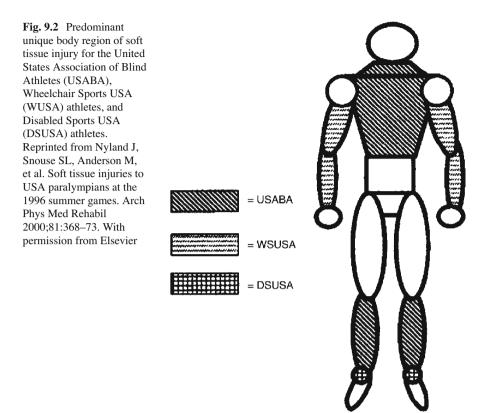
The psychological and physical benefits of exercise are numerous and include improved self-concept, psychosocial attitude, social awareness, social reintegration, perception of well-being, and health [1, 2]. Studies show that exercise can significantly increase psychological well-being in wheelchair athletes [1]. Disabled individuals who participate in athletic activities demonstrate better cardiopulmonary endurance, exercise tolerance, mobility, balance, cardiovascular health, and less obesity when compared to those who do not participate [3]. Engaging in sports may also improve bone mineral density (especially for those who are at a wheelchair mobility level) [4]. Proprioceptive related activities have been associated with improved amputee weight-bearing and gait [5].

It is well known that physical fitness levels are decreased for individuals with disabilities compared to their able-bodied counterparts [2]. Involvement in sports can significantly improve quality of life and life expectancy, which consequently decreases hospital admissions and medical complications [6]. Individuals who are disabled but active have fewer cardiac risk factors including a better lipid profile [7, 8].

Studies have shown that the injury rate and type of injuries are similar for both disabled and able-bodied athletes [9]. According to injury data [10], the most common injuries in parathletes (1976 forward) are sprains, strains, abrasions, contusions, fractures, and dislocations. Location of injury is sport- and disability-related (Fig. 9.2). Lower extremity injuries occur more frequently in ambulatory athletes (visually impaired, amputee, cerebral palsy), while upper extremity injuries are more common in wheelchair athletes. Most injuries required less than 7 days without participation in the sport. [10]

9.3 History of Disabled Athletics

Sir Ludwig Guttmann, a German-born refugee from Nazi occupation, is credited with originating and popularizing organized sports for the disabled. As a British neurosurgeon and director of the National Spinal Injuries Center at Stokes-Mandeville Hospital in London, Guttmann established a comprehensive rehabilitation program for paralyzed patients and incorporated athletics. On July 28th 1948, Guttmann organized a sports competition for World War II veterans with SCI on the same day as opening ceremonies as the London 1948 Olympic Games [11]. All the participants were wheelchair athletes. What began as recreational rehabilitation is considered to



be the origin of athletic competition for individuals with disability. Four years later, Guttmann's event was transformed into an international competition when a Dutch Ex-serviceman joined the games. The Paralympics were born [12].

The Paralympics, which began, in Rome, as an event consisted of 600 athletes from 23 different countries. In 2012 at the London Paralympics, there were 4237 participants from 164 countries and at the Sochi 2014 Winter Paralympics, there were 545 participants from 54 countries [13, 14]. As the number of athletes grows, the training, competition, and criteria for participation have become more rigorous [13]. And as such, it should be expected that injury rates will increase as well. In a study by Derman et al. [15], a total of 475 injuries were reported in 387 athletes at the London 2012 Paralympic Games. Seventy percent of all injuries were acute, with upper limb being the most injured. However, it is still essential to note that there were 9 injuries in the pelvis/buttock, 18 in hip/groin, and 30 in the thigh [15]. As the number of disabled individuals participating in sports continues to rise, it is important that physicians are able to meet the needs of this population.

9.4 Classification System

Athletes are categorized using the International Paralympic Committee's Classification system (Table 9.1). The system classification provides a structure for competition. This system was developed to decrease the impact of impairments on sport performance by determining who is eligible to compete in a certain para-sport and by grouping the eligible athletes in sport classes according to their activity limitation in that particular sport [16]. Each Paralympic sport has its own classification system and is developed by the International Federation (IF) governing that sport. It can be used to re-evaluate an athlete throughout their career as some disease processes changes over time. IF decides how severe an impairment must be in order for athlete to be eligible to compete in that sport. The only exception is the classification for athletes with visual impairments which remains a medical system [16].

Functional classification of an athlete's impairment depends on how much that impairment impacts sports performance. For example, athletes with spinal cord injury resulting in lower extremity paresis can now compete with double above-knee

Impairment	Explanation
Impaired muscle power	Reduced force generated by muscles or muscle groups, may occur in one limb or the lower half of the body, as caused, for example, by spinal cord injuries, spina bifida or poliomyelitis
Impaired passive	Range of movement in one or more joints is reduced permanently
range of movement	Limb deficiency joints that can move beyond the average range of motion, joint instability, and acute conditions, such as arthritis, are not considered eligible impairments
Limb deficiency	Total or partial absence of bones or joints, from birth or as a consequence of trauma (e.g., car accident or amputation) or illness (e.g., bone cancer)
Leg length difference	Bone shortening in one leg from birth or trauma
Short stature	Reduced standing height due to abnormal dimensions of bones of upper and lower limbs or trunk, for example due to achondroplasia or growth hormone dysfunction
Hypertonia	Abnormal increase in muscle tension and a reduced ability of a muscle to stretch, which can result from injury, illness, or a health condition such as cerebral palsy
Ataxia	Lack of coordination of muscle movements due to a neurological condition, such as cerebral palsy, brain injury or multiple sclerosis
Athetosis	Generally characterized by unbalanced, uncontrolled movements and a difficulty in maintaining a symmetrical posture, due to cerebral palsy, brain injury, multiple sclerosis, or other conditions
Visual impairment	Vision is impacted by either an impairment of the eye structure, optical nerve/pathways or the part of the brain controlling vision (visual cortex)
Intellectual impairment	A limitation in intellectual functioning and adaptive behavior as expressed in conceptual, social, and practical adaptive skills, which originates before the age of 18

 Table 9.1
 Paralympic classification [16]

amputees in wheelchair races. Their functional classification considers both impairments to be similar and will have limited problems with propelling a wheelchair, despite having different etiologies [16].

All assistive devices, prosthetics, or other adaptive equipment must be examined to ensure fair competition. For example, an above-knee-amputee skier with either knee disarticulation or hip disarticulation must use a three-track system (ski with two outriggers) [12].

In this chapter, we will discuss the challenges of athletes who use wheelchairs, amputees, and cerebral palsy.

9.5 The Amputee Athlete

The most common musculoskeletal injuries among amputee athletes are sprains and strains to the lumbar spine and sacroiliac joint on the uninvolved side. These injuries are attributed to the mechanical stress of the ground reaction forces during running or ambulation and the asymmetric biomechanical force transmission patterns of the involved and noninvolved sides. In addition, prosthetic alignment (increased hip flexion and ankle plantar flexion) contributes to lumbar spine and pelvic injuries, by causing increased lumbar lordosis [17]. It is important for the sports physician to understand the impact of prostheses and other adaptive equipment on the biomechanics of the specific sport.

9.5.1 Overview

The amputee athlete has a variety of options for adaptive equipment. The new, lightweight, more durable, and better engineered adaptive devices have enabled amputee athletes to attain a better gait pattern [1]. As a result, these athletes can engage in almost any sporting activity. Depending on the type of lower extremity amputation, gait and biomechanics are affected differently. As noted above, it is important that the physician understand (1) the types of amputation, (2) the various adaptive equipment options, (3) biomechanical considerations, and (4) how these factors impact athletic injuries.

Regardless of the level of amputation, there is an automatic change in biomechanics both with and without the use of a prosthesis. The higher the level of amputation, the greater the biomechanical considerations: more energy is required to complete basic functional activities, weight-bearing demands on the residual limb are increased, and the center of gravity is altered, compromising stability and increasing the likelihood of falls. As a result, balance-dependent tasks are more challenging for these individuals [1].

Transfemoral prosthesis has evolved and has locking mechanisms taking up more space distal to the socket [18]. A shorter residual limb has biomechanical and

functional consequences. Longer residual limbs enable greater stability for sitting, augment transfers, and provide a more secure suction for the prosthesis which is important for active athletes [18]. In a study by Baum et al., it was found that there is significant correlation between increased pelvic tilt excursion and smaller limb ratios. Unilateral transfemoral amputees who have shorter residual limbs tend to have an anterior tilt of the pelvis prior to toe-off and a remarkable posterior pelvic thrust to swing the prosthetic leg forward. This exaggerated pelvic tilt is secondary to inability to securely attach the hamstrings and quadriceps due to decreased availability of tendon attachment. Those with longer residual limbs such as a knee disarticulation have a more stable and controlled pelvic tilt similar to the uninjured population [19]. Baum postulated that the gait is not profoundly affected if the residual femoral limb length is greater than 57% of the contralateral intact limb [19]. Thus it is increasingly imperative to understand the type of amputation and prosthetic used in order to anticipate unique biomechanics and gait complications.

Athletes with transtibial amputees also have asymmetric muscle loads around the hip joint specifically, in the adductor and abductor muscle groups which may lead to overuse injuries from running. A study by Kersting et al. [20] revealed that hip and knee joint movements change during running in unilateral transtibial amputees in which there requires a larger power at the hip but reduced power at the knee. Bilateral transtibial amputees were also similarly affected but had a symmetric pattern. Lower-extremity amputee athletes use prosthetics that are adapted for their specific sport. All sports prostheses must be able to withstand the demand placed on them by the athlete. Specific prosthetic components are used for activities such as sprinting, endurance, and jumping. Carbon composite and energy-storing feet, as well as hydraulic, multiaxial, and computerized knee systems improve gait, athletic prowess, and agility. A shock-absorbing mechanism is desirable for endurance activities [12]. Because shock-absorbing devices are heavier, they may reduce speed, and therefore sprinters prefer lighter prosthetic components [12]. Carbon fiber components, particularly feet, have flexible shanks allowing them to deform on loading and recoil at toe-off, increasing energy return [21].

Of note, the residual limb is vulnerable to blistering and swelling at pressuresensitive areas (fibular head, distal tibia, and femoral condyles for transtibial amputees, and ischium for transfemoral amputees) [1]. In athletes with transfemoral amputations, ischial bursitis can occur as a result of weight-bearing patterns and prosthetic socket design. For the same reason, the greater trochanter and femur can also be affected [1]. An improperly fitting prosthesis can further aggravate the situation [17]. Socket irritation can cause prepatellar, infrapatellar, or pretibial bursitis in athletes with transtibial amputations [22]. Amputee athletes can develop residual limb fractures above the prosthesis [22].

Hyperextension frequently precipitates quadriceps tendon injuries. The quadriceps muscle is subjected to extreme forces during sudden extension movements: acceleration, deceleration, landing, and jumping [12]. The repetitive stress of these forces causes micro-tears at or near the attachment point of the quadriceps tendon to the superior aspect of the patella [12]. Most of the time, these injuries are minimal and do not limit competition. When the symptoms persist or progress, pain will

intensify and performance will suffer. Chronic tendinopathy may develop, which could ultimately result in a complete rupture of the quadriceps tendon [12]. Injuries to the uninvolved limb may include plantar fasciitis, Achilles tendonitis, and stress fractures [14–17].

9.5.2 Running

Injuries can affect both the involved and uninvolved extremity. Lower extremity injuries are more common in ambulatory athletes and typically occur during running activities [10]. While engineering has improved knee mechanisms and energystoring feet designs, the abnormal ground reaction force effect on the residual limb remains a concern. In able-bodied individuals, the ankle is the primary shock absorber and also dampens the rotatory effects of the distal leg and knee [1]. Prosthetic devices attempt to imitate these protective mechanisms, but may still endanger the residual limb. Environmental surfaces, irregular terrain, and malaligned prostheses alter biomechanical forces, affecting balance and increasing the risk for falls [1]. It should be noted that amputee athletes who compete without a prosthesis have an increased risk of injury of the uninvolved limb [1].

For running, it is necessary to have sufficient lower extremity muscle strength, power, and motor coordination. Similar to normal gait, the running cycle consists of two phases: stance and swing. As speed increases, the amount of time spent in stance decreases. Because runners with transtibial amputations must use an artificial foot and ankle, their performance is dependent on prosthetic design, regardless of prosthetic choice [21]. Running with a transtibial prosthesis increases knee extension time during stance on the involved limb [21]. This alteration in biomechanics can affect resilience and premature fatigue may develop [1]. Czerniecki et al. [23] found that individuals with transtibial amputations rely heavily on the hip extensors of the affected limb during running for energy production and shock absorption. In a follow-up study comparing able-bodied runners to runners with unilateral transtibial amputations, inherent adaptations in swing phase mechanics were seen. These findings suggest that the uninvolved lower extremity and trunk compensate for the decreased force generation of the stance phase prosthetic limb [24]. Many believe that this energy transformation provides some compensation. Other biomechanical differences in amputee runners include asymmetry of component ankle, knee, and hip forces, all of which increase the risk of injury.

For athletes with transfemoral amputations, running is more challenging because prosthetic choice necessitates a knee component. Gait dynamics change: duration of swing phase increases on the affected side, and toe clearance on the unaffected side is accomplished by hip elevation combined with weight shifting toward the affected side [12]. Computer-enhanced knee components facilitate forward movement of the distal elements approximating normal cadence [12]. This type of prosthesis is a common choice for track athletes because it facilitates swing phase control [12]. However, gait deviations may still occur. These may include excessive vaulting (rising on the

toe of the uninvolved side to assist with forward movement of the prosthesis, minimal knee flexion required), abnormal trunk movement and control, nonreciprocal or symmetric arm movement, and decreased pelvic rotation. Training and modification of prosthetic knee component may alleviate these problems. [1, 12]

9.5.3 Skiing

Specialized adaptive equipment is available for amputees who want to ski. For some amputees a three-track system is required: skis attached to two crutches with a third ski on the uninvolved side. Other adaptations include a multiaxial ankle or an ankle fixed in $15-25^{\circ}$ of dorsiflexion for athletes with transtibial amputations, bilateral transfemoral amputations, or hip disarticulation [1]. Data from the 2002 Winter Paralympic Games indicate that alpine skiers were more frequently injured than sledge hockey or Nordic skiers (64 % vs. 31 % vs. 8 %, respectively); 38 % of alpine skiers had lower extremity injuries; Nordic skiing injuries were located exclusively in the upper extremity, and sledge hockey injuries were more common in the upper extremity (50 %) than in the lower extremity (33 %) [25]. No link has been found between gender and the incidence of ski injuries.

9.5.4 Water Sports

Amputee scuba divers use water-safe prostheses, which are designed to prevent buoyancy. Elastomeric coverings also are available to protect traditional prostheses from limited water exposure. High-level amputees can also swim without a prosthesis [1]. This is important because conventional prosthetic devices are not permitted at governed, international swimming events (although fin attachments are acceptable) [1]. In any case, training to prevent lateral drifting and trunk dysfunction is recommended [1].

9.5.5 Cycling

Specialized terminal devices enable many amputee athletes to compete in cycling events. As in able-body cycling, binding systems that attach the prosthesis and the contralateral cycling shoe to the pedals are available. It may be necessary to adapt the seat width to optimize balance [1]. Toe clips might be contraindicated if the unaffected foot needs to become free to prevent falls. Some high-level amputees can cycle without a prosthesis using only their intact limb [1].

Prosthetics for other sports can be tailored to the individual and the specific sport. [13]

9.6 The Athlete with Cerebral Palsy

Spasticity, athetosis, and ataxia are classic features of cerebral palsy (CP). Other manifestations include reduced flexibility of muscles and tendons and decreased strength (extensors weaker than flexors). Also, joint contracture can develop if these conditions worsen.

The ratio of ambulatory to nonambulatory athletes with CP is about 50:50. Nonambulatory athletes participate in wheelchair sports [22]. Ambulatory ability determines whether an athlete who has CP is at risk for upper (43%) or lower extremity injuries (44%). In contrast, wheelchair-dependent athletes generally sustain upper extremity injuries (65%) [10]. In the London 2012 Paralympics, Willick et al. [26] found that 50.2% of all injuries occurred in the upper limb compared to any other body part. For the purposes of this text, we will concentrate on lower extremity injuries.

Increased muscle tone (spasticity) is the major predisposing factor for athletic injuries in this population [12]. Spasticity can interfere with a person's ability to operate a manual wheelchair, ambulate, fit into clothing properly, or perform transfers and activities of daily living. Other sequelae include contractures, skin breakdown, and pain. The frequent involuntary muscle activity exhibited by this population raises safety issues that are important considerations when organizing competitions and designing training programs.

These issues include falling out of the wheelchair, gait dysfunction with forward movement, and loss of balance/postural control. Athletes with CP typically experience lower extremity injuries that involve the patellofemoral joint and are due to muscle spasms of the surrounding muscles. Classically, there is shortening and tightening of the quadriceps and hamstring muscles, which increases the tension across the joint. Athletes with CP can also have deformities of the ankle and foot (such as equinovarus, equinus, and valgus deformities), which increase the risk for subsequent injuries and pain. Often, athletic involvement is interrupted and orthopedic intervention is needed [22]. Ankle instability, calluses, pressure sores, and metatarsal pain are commonly seen [12, 22].

There is also a tendency for overuse syndromes including muscle strains and chronic knee pain. As stated earlier, there is an increased risk for patellofemoral dysfunction as well as chondromalacia patella [12]. Patella alta (high-riding patella) can be a consequence of decreased quadriceps and hamstring flexibility [12]. Developmental hip abnormalities (coxa valga, acetabular dysplasia, and hip subluxation) may also occur [12, 22]. These conditions are more common in the pediatric CP population [9]. Tight hip flexors and extensors can exacerbate lumbar lordosis, chronic back pain, and spondylolytic changes [22].

For functional and athletic purposes, spasticity must be controlled. Strategies for management include pharmacologic treatment with antispasmodics, positioning, bracing, or physical therapy. Bracing is helpful for tone reduction and ambulation. Pain is usually treated with anti-inflammatory and antispasmodic medications depending on the etiology. Once tone is reduced, stretching is important in preventing contractures and maintaining flexibility. This must be done in a slow, sustained manner to avoid stretch reflex induction [22]. Strengthening programs must also consider the effect of muscle tone in an effort to prevent further muscle imbalance [22].

9.7 The Wheelchair Athlete

Individuals with SCI, CP, amputations, and spina bifida engage in wheelchair athletics. The incidence of lower extremity injuries (specifically the hip and pelvis) is rare because the upper limb is used for transfers and mobility. Despite the limited number of lower extremity injuries, the sports physician should be aware of some specific injuries.

Among those with SCI, osteoporosis is common, increasing the risk of fracture. The distribution of bone demineralization differs in the spinal-cord-injured population where it is predominantly located in the lower extremities and is attributed to disuse [27]. Goktepe and colleagues [4] found reduced trochanter and femoral neck bone density in athletes and individuals with SCI. Bone loss occurs within the first 3 months after the onset of injury [28].

According to Zehnder et al. [29], the incidence of known fracture in complete paraplegic males is 2.2% per year. The average time to onset of documented fracture was approximately 9 years. Fracture incidence for males with complete paraplegia increases from 1% within the first year to 5% per year after 20 years [29]. The rate of complications such as osteomyelitis, pressure sores, or contractures can reach 20–40% [30]. Patients can present with symptoms mimicking a viral illness like fever and general malaise [28]. Pain is less commonly identified because of the lack of sensation [28]. Physical examination may reveal swelling and ecchymosis.

Conservative treatment is usually recommended for nondisplaced fractures. A soft well-padded splint is generally prescribed, in the early stages, for functional mobility [28]. Sitting should be started soon after injury, since it is important to help prevent deformities [28].

Another complication in the SCI population is heterotopic ossification (HO): formation of bone in ectopic sites. This can be devastating with severe activity limitations in 8-10% [11]. The reported incidence of HO in paraplegic patients is close to 30% [31]. HO typically develops within the first 2 months after SCI [32], and most frequently occurs at the hips [27]. Other possible sites include the knees, shoulders, and elbows [33]. The etiology of heterotopic ossification is unknown, although risk factors include complete SCI, older age at time of injury, spasticity, pressure ulcers, and other injuries [33, 34]. HO can continue for more than 6 months, at which time the bone matures.

Symptoms include local swelling with progression to diffuse edema, limited range of motion, and an elevated serum alkaline phosphatase which usually returns to normal or near-normal levels with maturation [33, 35]. There is a low incidence of permanent disability from HO [33]. Radiographic evaluation is helpful in the mature phase, while bone scan can identify HO at earlier stages of development [33] (see Fig. 9.1).

Treatment focuses on halting progression of ossification and maintaining range of motion and function. Etidronate, a bisphosphonate, is the first-line treatment to prevent further ossification [11, 33]. Gentle range-of-motion exercises and radiation therapy are also part of the treatment algorithm. Although radiation therapy is effective, it is not used as frequently [34, 36]. Surgical resection is considered when there is severe restriction of joint range of motion and after maturation has occurred [33]. HO can recur after surgical resection. To prevent recurrence of HO postoperatively, Etidronate, anti-inflammatory agents, radiation therapy, and range-of-motion exercises are incorporated in the treatment plan [33, 34].

9.8 Summary

With the rapidly growing number of disabled athletes, it is important that the physician fully understand the medical, functional, psychological, and social needs of this population.

Despite the risk for musculoskeletal athletic injuries, there is evidence that supports participation in sporting activities for the disabled population. Athletic involvement has been associated with numerous systemic and mental health benefits that are far more important than the risk of injury. In our case, FM developed right hip heterotopic ossification, a common and often debilitating complication of SCI. FM was treated with Etidronate, a bisphosphonate, and gentle range-of motion exercises. The goal of this intervention was to reduce ectopic bone formation and preserve hip mobility. After treatment, FM returned to all previous sporting activities.

References

- 1. Bergeron JW. Athletes with disabilities. Phys Med Rehabil Clin N Am. 1999;10:213, 28, viii.
- Lai AM, Stanish WD, Stanish HI. The young athlete with physical challenges. Clin Sports Med. 2000;19:793–819.
- 3. Curtis KA, McClanahan S, Hall KM, et al. Health, vocational, and functional status in spinal cord injured athletes and nonathletes. Arch Phys Med Rehabil. 1986;67:862–5.
- Goktepe AS, Yilmaz B, Alaca R, et al. Bone density loss after spinal cord injury: elite paraplegic basketball players vs. paraplegic sedentary persons. Am J Phys Med Rehabil. 2004;83:279–83.
- Yigiter K, Sener G, Erbahceci F, et al. A comparison of traditional prosthetic training versus proprioceptive neuromuscular facilitation resistive gait training with trans-femoral amputees. Prosthet Orthot Int. 2002;26:213–7.
- Groah SL, Lanig IS. Neuromusculoskeletal syndromes in wheelchair athletes. Semin Neurol. 2000;20:201–8.
- 7. Dearwater SR, LaPorte RE, Robertson RJ, et al. Activity in the spinal cord-injured patient: an epidemiologic analysis of metabolic parameters. Med Sci Sports Exerc. 1986;18:541–4.
- Brenes G, Dearwater S, Shapera R, et al. High density lipoprotein cholesterol concentrations in physically active and sedentary spinal cord injured patients. Arch Phys Med Rehabil. 1986;67:445–50.

- 9 Hip and Pelvis Injuries in Special Populations
- 9. Dec KL, Sparrow KJ, McKeag DB. The physically-challenged athlete: medical issues and assessment. Sports Med. 2000;29:245–58.
- Ferrara MS, Peterson CL. Injuries to athletes with disabilities: identifying injury patterns. Sports Med. 2000;30:137–43.
- Subbarao JV, Garrison SJ. Heterotopic ossification: diagnosis and management, current concepts and controversies. J Spinal Cord Med. 1999;22:273–83.
- 12. Gottschalk F. The orthopedically disabled athlete. In: Stevenson A, editor. DeLee and Drez's orthopedic sports medicine. 3rd ed. Philadelphia: Saunders; 2003.
- 13. Willick SE, Lexell J. Paralympic sports medicine and sports science—introduction. PM R. 2014;6:S1–3.
- 14. London 2012 Paralympics. http://www.paralympic.org/london-2012. Accessed 2 Sept 2015.
- Derman W, Schwellnus M, Jordaan E, et al. Illness and injury in athletes during the competition period at the London 2012 Paralympic Games: development and implementation of a web-based surveillance system (WEB-IISS) for team medical staff. Br J Sports Med. 2013;47:420–5.
- International Paralympic Committee. Explanatory guide to Paralympic classification. http:// www.paralympic.org/sites/default/files/document/121203164523073_WinterSportLaymens. pdf. Published 1 Sept 2015. Accessed 25 Sept 2015.
- Klenck C, Gebke K. Practical management: common medical problems in disabled athletes. Clin J Sport Med. 2007;17:55–60.
- 18. Smith D, Michael J, Bowker J. Atlas of amputations and limb deficiencies. Rosemont: American Academy of Orthopedic Surgeons; 2004.
- Baum BS, Schnall BL, Tis JE, et al. Correlation of residual limb length and gait parameters in amputees. Injury. 2008;39:728–33.
- 20. Kersting UG, Gurney JK, Lewis S. et al. An individualized musculoskeletal model for the analysis of amputee running. International Symposium of Biomechanics in Sports. 2010;142–144.
- Buckley JG. Biomechanical adaptations of transtibial amputee sprinting in athletes using dedicated prostheses. Clin Biomech (Bristol, Avon). 2000;15:352–8.
- 22. Patel DR, Greydanus DE. The pediatric athlete with disabilities. Pediatr Clin North Am. 2002;49:803–27.
- Czerniecki JM, Gitter A, Munro C. Joint moment and muscle power output characteristics of below knee amputees during running: the influence of energy storing prosthetic feet. J Biomech. 1991;24:63–75.
- 24. Czerniecki JM, Gitter AJ, Beck JC. Energy transfer mechanisms as a compensatory strategy in below knee amputee runners. J Biomech. 1996;29:717–22.
- Webborn N, Willick S, Reeser JC. Injuries among disabled athletes during the 2002 Winter Paralympic Games. Med Sci Sports Exerc. 2006;38:811–5.
- 26. Willick SE, Webborn N, Emery C, et al. The epidemiology of injuries at the London 2012 Paralympic Games. Br J Sports Med. 2013;47:426–32.
- Jiang SD, Dai LY, Jiang LS. Osteoporosis after spinal cord injury. Osteoporos Int. 2006;17:180–92.
- Freehafer AA. Limb fractures in patients with spinal cord injury. Arch Phys Med Rehabil. 1995;76:823–7.
- 29. Zehnder Y, Luthi M, Michel D, et al. Long-term changes in bone metabolism, bone mineral density, quantitative ultrasound parameters, and fracture incidence after spinal cord injury: a cross-sectional observational study in 100 paraplegic men. Osteoporos Int. 2004;15:180–9.
- Chen SC, Lai CH, Chan WP, et al. Increases in bone mineral density after functional electrical stimulation cycling exercises in spinal cord injured patients. Disabil Rehabil. 2005;27:1337–41.
- Meiners T, Abel R, Bohm V, Gerner HJ. Resection of heterotopic ossification of the hip in spinal cord injured patients. Spinal Cord. 1997;35:443–5.
- Banovac K, Williams JM, Patrick LD, Haniff YM. Prevention of heterotopic ossification after spinal cord injury with indomethacin. Spinal Cord. 2001;39:370–4.

- Kirshblum SC, Priebe MM, Ho CH, et al. Spinal cord injury medicine. 3. Rehabilitation phase after acute spinal cord injury. Arch Phys Med Rehabil. 2007;88:S62–70.
- Jamil F, Subbarao JV, Banaovac K, et al. Management of immature heterotopic ossification (HO) of the hip. Spinal Cord. 2002;40:388–95.
- 35. Singh RS, Craig MC, Katholi CR, et al. The predictive value of creatine phosphokinase and alkaline phosphatase in identification of heterotopic ossification in patients after spinal cord injury. Arch Phys Med Rehabil. 2003;84:1584–8.
- 36. Sautter-Bihl ML, Liebermeister E, Nanassy A. Radiotherapy as a local treatment option for heterotopic ossifications in patients with spinal cord injury. Spinal Cord. 2000;38:33–6.

Chapter 10 Functional Therapeutic and Core Strengthening

Gerard A. Malanga, Steve M. Aydin, Eric K. Holder, and Ziva Petrin

Clinical Pearls

- Core strengthening is an integral part of training programs in athletes as well as patients.
- The kinetic chain theory describes how the core connects and influences movement in the rest of the body.
- A core training program includes a focus on balance, strength, flexibility, and correction of asymmetry for optimal benefit.
- Neuromuscular control and proprioceptive feedback play a major role in training the core musculature.
- The long-term benefits of core stability may improve athletic performance and decrease dysfunction in patients with injuries or low-back pain.

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10.1 Case Presentation

An avid 22-year-old male runner presents with a 1-year history of low-back, left buttock, and lateral thigh pain. The pain began insidiously and progressively worsened with time and activity. The athlete occasionally uses non-steroidal antiinflammatory drugs, such as ibuprofen, with minimal benefit. He continues to run 4 to 5 miles per day, despite persistent symptoms of pain in the left lower back, buttock, and lateral thigh. His past medical history is otherwise unremarkable.

On physical examination, he is a thin and healthy appearing male. His lumbar spine exhibits a mild decrease in physiologic lordosis with otherwise full pain-free flexion and extension. However, a mild decrease in left-sided segmental motion is noted. His neurological exam is unremarkable. Straight leg raise is negative bilaterally. Patrick's test is mildly positive on the left. Additional flexibility testing reveals he has mildly tight hamstrings and very tight quadriceps muscles, left greater than right. Ober's test is positive bilaterally, indicating iliotibial band tightness, worse on the left than right. His gait pattern demonstrates increased pronation of his feet during mid stance phase, more noticeable on the left than right. His leg length, measured by resetting the pelvis and observing the ankles and anterior superior iliac spine levels, illustrates that his left leg is shorter than his right leg.

Functional testing of the patient demonstrates weakness of core muscles, with loss of lumbar support, particularly with lateral and prone bridges exercises. Muscle testing of the hip abductors on the left reveals relative weakness.

On radiographic evaluation, lumbar spine magnetic resonance imaging (MRI) demonstrates a small disk bulge at L5/S1. X-rays and MRI of the hip are normal. Bone scan and electrodiagnostic studies are also normal.

He was previously diagnosed with greater trochanteric bursitis, for which he received a corticosteroid injection, with only transient benefit. A secondary diagnosis of sacroiliac (SI) joint dysfunction was determined, for which he received treatment with ultrasound, manipulation, and a fluoroscopically guided injection to the left SI joint, with minimal benefit. A suspected diagnosis of piriformis syndrome was treated with trigger point injections followed by a Botox injection, all without lasting benefit.

After minimal benefit from above treatments, the patient was enrolled into a comprehensive core strengthening program to address physical exam asymmetries, maximize function, and eliminate pain.

10.2 Introduction

The core acts as the stabilizer of the spine and trunk, the generator of power when initiating movement of the limbs, and a point of transition of forces from the lower limbs to the upper limbs, and vice versa, via the abdomen and spine. When this system is altered by injury, the smooth transition of forces generated by the limbs and trunk, i.e., the kinetic chain of motion, becomes disrupted. The kinetic chain theory stresses the importance of core strength [1].

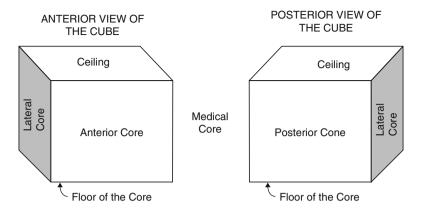


Fig. 10.1 The core is a three-dimensional structure resembling a cube or sphere. Viewing it as a cube, with each side contributing a function, while overlapping with one another (like in a sphere), helps in understanding its function and purpose

In this chapter, we will review the anatomy of the core musculature, the kinesiologic properties of the core, and the kinetic chain. This chapter will also provide tools for evaluation of core stability, and an overview of core stability training programs.

10.3 Anatomy

The core is often described as a box: a matrix of bones, ligaments, muscles, and nerves in a three-dimensional layering of tissues. This anatomical arrangement provides stability to the spine, allows motion in multiple planes, and enables the transference of energy during movement. The core is composed of a floor, a ceiling, front, back, and sides (Fig. 10.1). The superior aspect or ceiling of the core is the diaphragm. The floor is composed of the pelvic floor. The anterior portion of the core is made up of the abdominal muscles, while the posterior aspect is composed of the paraspinals and thoracolumbar fascia. The lateral portions are composed of the lateral hip girdle musculature, and external/internal obliques [2–4].

The muscles and tissues of the core are arranged in layers, each with a different function. These are the inner, middle, and outer layers of the core. The outer layer is made up of the power muscles, such as the rectus abdominis. The middle layer is made up of the abdominal and back-stabilizing muscles. And finally, the inner most layer is comprised of the intersegmental muscles and proprioceptive structures, such as the multifidi and nerves [4] (Fig. 10.2).

Looking at the core as a cube simplifies its complexity. All the described borders are connected to one another and overlap. They all function together as a unit in body mechanics to allow transition of forces and stability (Fig. 10.2).

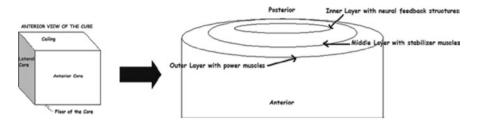
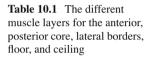


Fig. 10.2 The core is arranged spherically with a complex arrangement of tissues. The three layers that make it up are the outer, middle, and inner. Each one contributes a specific function to the core's action. The outer layer functions by producing muscular power. The middle layer uses its muscles and tissues to stabilize the core. The inner layer functions in providing neural feedback to the body



Anterior core muscles	Lateral core muscles
Rectus abdominis	Gluteus maximus
External oblique	Gluteus minimus
Internal oblique	Gluteus medius
Transversus abdominis	External oblique
Posterior core muscles	Internal oblique
Erector spinae group	Transversus abdominis
Iliopsoas	Ceiling core muscle
Quadratus lumborum	Abdominal diaphragm
Multifidi	Floor core muscle
Thoracolumbar fascia	Pelvic diaphragm/ pelvic floor muscles

The anterior core consists of the rectus abdominis, the external and internal obliques, the transversus abdominis and their aponeurosis (Table 10.1 and Fig. 10.3a). These muscles connect the ribcage and the pelvis. The rectus abdominis is the most superficial muscle and originates on the pubis of the pelvis, inserting onto the cartilage of the xiphoid, fifth, sixth, and seventh ribs. It flexes the lumbar spine, straightens the pelvis, maintains intra-abdominal pressure, and assists in expiration. The external oblique originates on the surfaces of ribs 5 to 12 and inserts on the iliac crest and anterior rectus sheath. When it contracts unilaterally, it causes bending of the trunk to the ipsilateral side of contraction and rotation to contralateral side. Bilateral activation of the external obliques leads to flexion of the trunk, straightening of the pelvis and increases intra-abdominal pressure. The internal oblique lies between the external oblique and the transversus abdominis. It increases intra-abdominal pressure similar to the external oblique. It bends and rotates the trunk to the ipsilateral side. It originates from the thoracolumbar fascia, iliac crest, and anterior superior iliac spine and inserts on the lower borders of ribs 10 to 12, the linea alba, and rectus sheath. The deepest muscle is the transversus abdominis. It originates on the posterior-lateral

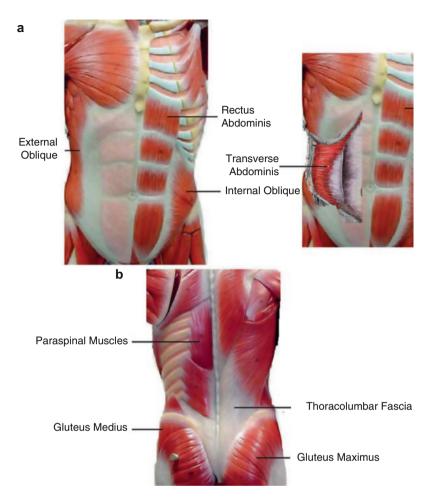


Fig. 10.3 (a) The anterior abdomen and core muscles. (b) The posterior aspect of the torso and muscles

portions of the deep thoracolumbar fascia, anterior iliac spine, and iliac crest. It inserts on the inner surface of the 7th to 12th ribs, the rectus sheath, and the linea alba. It bends the trunk unilaterally to the same side of contraction, and rotates to the opposite side. With bilateral contraction, it assists in flexion of the trunk and lumbar spine, increases intra-abdominal pressure, and assists in expiration. It is noteworthy that all the origins and insertions have connections to the hip, pelvis, and lumbar spine [2, 3].

The posterior portion of the core consists of the erector spinae, iliopsoas, quadratus lumborum, multifidi muscles, and the thoracolumbar fascia. The erector spinae originates at the sacrum, iliac crest, lumbar spinous processes, and lumbar transverse processes (Table 10.1 and Fig. 10.3b) and inserts onto the 2nd to 12th ribs and thoracic vertebral transverse processes. During bilateral contraction, the erector spinae causes the lumbar spine to extend; unilateral contraction leads to lateral bending to the ipsilateral side. The iliopsoas originates on the T12-L5 vertebral bodies, L1-L5 transverse processes and intervertebral disks, as well as the iliac fossa. It inserts on the lesser trochanter of the femur. The iliopsoas causes hip flexion, internal rotation at the hip and tilts the pelvis anteriorly, exaggerating the lumbar lordosis. Quadratus lumborum originates from the iliac crest and inserts on the 12th rib and L1-L4 vertebrae. It has three poles: superior, longitudinal, and inferior. The inferior pole is functional in the core musculature, while the superior and middle poles function during respiration. When the quadratus lumborum contracts unilaterally, it causes the trunk to bend to the ipsilateral side; when it contracts bilaterally, it provides the ability to bear down and increase intra-abdominal pressure [2]. The multifidi originate on the transverse processes and insert superiorly on the spinous process. They function in segmental motion of the lumbar vertebrae and help extend the spine with bilateral contraction. With unilateral contraction, they cause flexion, ipsilateral side-bending, and contralateral rotation of the spine [2, 3, 5, 6].

The lateral borders of the core are composed of the gluteal muscles as well as the lateral portions of the previously discussed anterior core muscles; the obliques, thoracolumbar fascia, and transverse abdominis. The gluteal muscles consist of the gluteus maximus, medius, and minimus (Table 10.1 and Fig. 10.3). The gluteus maximus originates from the dorsal surface of the sacrum and posterior iliac crest, and inserts on the iliotibial band and gluteal tuberosity of the femur. Its primary motion is hip extension with lateral rotation. The gluteus maximus both originate from the ilium and insert on the greater trochanter of the femur. Their primary motion is hip abduction. The gluteal muscle group as a whole has an important role in dynamic stabilization of the hip, pelvis, and trunk during motion [2, 3, 5].

The thoracolumbar fascia acts as a girdle around the abdomen, and consists of three layers (Fig. 10.3). All three layers—anterior, middle, and posterior—function to maintain stability of the spine and trunk while preventing unwanted motion during movement. The anterior and middle portions are intimately connected to the iliopsoas, quadratus lumborum, and intrinsic back muscles such as the multifidi. The posterior layer is the most superficial layer with connections to the transversus abdominis and latissimus dorsi, connecting the abdomen and the upper limb. The various attachments pulling at the thoracodorsal fascia are instrumental in providing tension, removing slack, and providing stability (Fig. 10.4) [2–5].

The abdominal diaphragm serves as the roof of the core and increases the intraabdominal pressure by descending. It provides stability during exercise through coordinated breathing.

The pelvic floor acts as the base of the core and aids in increasing intra-abdominal pressure by contracting and ascending. The pelvic floor and diaphragm primarily work in co-contraction with the trunk muscles (Table 10.1 and Fig. 10.5). When a contraction occurs, an increase in abdominal pressure occurs, which, in turn, causes the lumbar spine to stabilize, reducing unwanted movement [2, 3, 5–8].

Fig. 10.4 The

thoracolumbar fascia is a crucial component of the core. It is connected to many muscles and, when they contract, it is pulled tight like a girdle and provides stability. The transverse abdominis muscle is one of the major players in pulling the thoracolumbar fascia tight during contraction. The arrows indicate the manner in which the fascia is pulled during a contraction

Fig. 10.5 The pelvic floor and diaphragm are major components in the core and help in providing and maintaining an intraabdominal pressure during movements. The floor of the core is made up of the pelvic diaphragm muscles. The roof of the core is made up of the diaphragm

Ceiling Diaphragm Pelvic Floor

10.4 Mechanism of the Core and the Kinetic Chain

To understand the core and its function, the body should be viewed as having an inherent connection among all the different layers of muscles, fascia, and other connective tissues. Patterns of muscle contraction allow for coordinated movement



Fig. 10.6 The kinetic chain theory can often be applied to sports that involve explosive motions. In this example of kicking a soccer ball, the power to kick the ball will often develop in the upper extremities, and transmit downward to the leg during the follow-through. The core contracts prior to all these motions, and stabilizes the body to allow for a fluid transition of the upper body momentum to the lower body. When the core is functional and intact, the maximal and coordinated force from these sources can be achieved

[9]. The motion of an extremity in three-dimensional space occurs freely at the distal portions of the extremity. The proximal limb girdle is anchored to the trunk, while the trunk is anchored to the spine and stabilized by the core. This coupling of musculature of the limbs and trunk allows the body to function as a unit. Thus the motion of one body part influences movement of ipsi- and contralateral distal or proximal portions of the body. These motions are generated from contractile forces within the muscles, which then disperse energy distally and proximally, allowing for motion in a three-dimensional plane. The spine, hip, pelvis, and trunk are the areas of transition of this energy, force, and motion (Fig. 10.6). Therefore, when a force is generated in the lower extremity, the core transmits the force to the trunk. Studies have shown that the generation of forces in the extremities is preceded by unconscious contraction of the core muscles [9, 10]. The core therefore both transmits forces from the extremities proximally and stabilizes the trunk for motion of the limb distally. This coupling of functions makes the core the center of the kinetic chain.

The kinetic chain theory, also known as the *link theory*, describes the connection of musculoskeletal motion in the extremities and trunk. The definition of the kinetic chain, as described in the literature, is the significant interrelationship of muscle activation and translation of forces within the musculoskeletal system [11].

An example illustrating this principle is in the case of a pitcher throwing a fastball. The starting point of the force is the lower extremity; the end point is the upper limb. When the lower limb contracts in the early part of a pitcher's motion, power is generated in the lower limbs. Prior to start of the pitching stride, the abdominal muscles and back muscles contract, to provide stability. As the pitcher steps forward through his stride, the force is transferred from the lower limbs through the stabilized

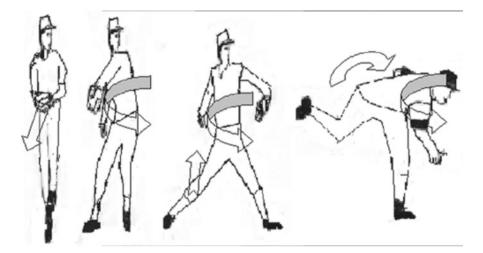


Fig. 10.7 In the case of a baseball pitcher, the kinetic chain functions similarly to that of a soccer player; however, the forces develop in the lower extremities initially, and then transmit upward to the upper extremities. The core once again contracts, stabilizes, and allows for an efficient throw

core, and upwards to the upper extremity where the follow-through is made and the ball is thrown (Fig. 10.7). In an ideal kinetic chain system, no vulnerable points exist where forces can be lost because of instability or weakness. When an interruption in the chain occurs, motion and forces are improperly and inadequately transmitted, and compensation is attempted by other muscle groups. For example, generating the same amount of power to throw a fastball in an athlete with a weak core will lead to straining of the compensatory muscles. As a result, a weak core places a person at greater risk of injury, misalignment, and submaximal performance [4, 9, 10, 12, 13].

To understand this, it is important to see how patterns of motion are interrupted in patients with extremity pain or low-back pain. These injuries can result in altered neuromuscular control of the core and limbs. Athletes with lower extremity instability (i.e., ankle or knee) have a delayed firing pattern of the gluteus medius (which functions in hip abduction and posterior-lateral trunk stability) and the anterior abdominal muscles [13, 14]. A dysfunction of the neuromuscular control of the core interrupts the kinetic chain. The effect of breakdown in one part of the chain results in abnormal and delayed muscle firing patterns, poor neuronal feedback, and poor proprioception. This, in turn, causes increased instability in the spine and other joints [4, 9, 10, 14, 15].

The core is not only responsible for generating or transmitting force; it functions to provide stability to the spine and other joints. Joints require stabilizing forces from muscles and proprioceptive feedback from neurons for optimal function. The early development of arthritis or joint dysfunction often results from misalignment, unwanted motion, and high shearing stress. A strong core with sufficient endurance and optimal neuromuscular control patterns provides spinal stability [9, 16]. The diaphragm, pelvic floor, internal oblique, external oblique, and transversus abdominis increase intra-abdominal pressure when contracting. This creates tension in the spine and thoracolumbar fascia, reducing laxity in the spine and imparting stability

to the lumbar spine [4, 7, 8, 17, 18]. This is analogous to inflating a tire. As air is added to the tire, tension in the walls increases, preventing the tire from sagging, and allowing it to roll firmly and efficiently.

The transversus abdominis and multifidi have been shown to contract prior to movement of a limb. This is thought to provide immediate stability to the core prior to limb movement, occurring through a neuromuscular control feedback mechanism [6, 11]. In cases of injury or in presence of low-back pain, the multifidi and transversus abdominis are inhibited. During muscle contraction of the lower extremity, the transversus abdominis is normally the first to contract. This prevents unwanted trunk movement, and allows the fluid transition of energy/force through the kinetic chain, as described previously. With low-back pain, a delayed activation of the transversus abdominis contraction is noted on electromyography evaluation, relative to limb activation [19, 20].

Pelvic motion is controlled by the paraspinals and anterior abdominal musculature as well as the pelvic attachments of the hip rotators, adductors and abductors and knee flexors and extensors. Thus weakness of the core can create breakdown in pelvic control, leading to breakdown in lower extremity control. Pelvic tilt is important in achieving normal range of motion of the lower extremity and providing an optimal center of balance. The hip rotator muscles are particularly important in dynamic single-leg stance activity such as running, jumping, and throwing [9-11, 21].

Summarily, a strong, balanced, and controlled core is essential in providing stability and proper alignment of the spine, hip, pelvis, and sacrum. Lack of core stability interrupts the physiologic contraction patterns of the kinetic chain as a result of compensatory muscle firing due to alterations in neuronal feedback [1].

For a more comprehensive discussion of the kinetic chain, refer to Chap. 3 of this text.

10.5 Core Dysfunction in Clinical Scenarios

As illustrated thus far, core strength is integral to the dynamic movements of everyday activities and even more so in athletic endeavors. In cases where distal extremity dysfunction develops, there is a greater risk for developing proximal injury due to compensatory mechanisms. A core strengthening program may prevent injury by improving dynamic control of the core and stabilization of motion of the limbs through different planes [7, 22].

Core and hip extensors, primarily the gluteus maximus and medius, play a major role in stabilizing the pelvis during trunk rotation, or when the center of gravity is grossly shifted. The hip abductors function in midstance to stabilize the pelvis, preventing downward inclination (Trendelenburg sign) during single leg stance. When hip abduction weakness occurs, there is an increase in lateral trunk stabilizer firing. Asymmetry in hip muscle strength, flexibility, or endurance may produce delayed firing in the hip extensors and abductors, theorized to predispose to lower extremity injury and low-back pain. This can be attributed to the interruption of neuronal firing patterns, which in turn results in poor muscular control [10]. Hip abductor strength in females athletes has been studied utilizing the Star Excursion Balance Test [23–25] which associates neuromuscular control and decreased lower extremity injury risk.

In cases of injury, such as anterior cruciate ligament (ACL) dysfunction or ankle instability, the firing patterns of the hip and core muscle are disrupted [14, 15, 26]. In response to an ACL injury or knee instability, the hip flexors and knee extensors display altered contraction patterns, in an effort to reduce stress on the ACL and knee joint. A pattern of increased firing of the hip muscles on the ipsilateral side of an ankle injury has been shown [14]. With a chronic injury or without neuromuscular rehabilitation, this results in poor firing patterns on the ipsilateral side of the injury causing a chronic disruption in the kinetic chain [15].

Patients with low-back pain have been shown to develop an abnormal pattern of muscle contraction [20]. The multifidi and transverse abdominis have been shown to function abnormally when low-back pain is present. During muscle contraction with anterior loading, the transverse abdominis is activated first, preventing unwanted trunk movement and thus allowing the transition of energy and forces in a normal pattern of the kinetic chain. However, patients with low-back pain have demonstrated delayed activation of the transverse abdominis relative to the limbs [6–8, 12, 27].

Core strengthening for low-back pain requires focus on the muscles associated with twisting motions and repetitive flexion/extension motions. People with poor musculature control of the trunk and hips are often at greater risk for developing low-back pain due to abnormal or unstable transferences of forces when motion is occurring. Correction of abnormal neuromuscular control with core stability exercises can improve function [6, 27, 28]. Focused core stability exercise appears to be more effective in decreasing pain and improving general function in the short term versus general exercise in chronic low back pain [29] Several studies have shown significant improvement in low back pain in athletes after core stability exercise, however, further research is needed for high quality support [30].

10.6 Assessing the Core Musculature

The comprehensive evaluation of an athletes' core stability is important and should include 5 components: range of motion, strength, endurance, motor control, and function [31]. There are both static and dynamic measures of assessing the core musculature. It is important to note that athletic performance is dynamic, intermittent, and oscillates in intensity and energy requirements. Therefore a static core muscle test may not represent a comprehensive assessment of the core in a healthy athlete [32]. Currently, there are no standardized means of dynamically assessing the core [32].



Fig. 10.8 Maintenance of neutral spine positioning during core assessment is essential. (a) kyphotic posture; (b) neutral spine posture; (c) lordotic posture

No published consensus definition or measurement criteria for core stability exist. In fact, there are currently at least 35 published tests developed to assess core stability. As a group, it has been determined that the core endurance tests have the highest intra-rater reliability [31].

10.6.1 Neutral Spine Position

Neutral spine position should be maintained during examination (Fig. 10.8). Neutral spine position represents normal alignment of the spine, with all three physiological curvatures in place. With regard to core stability, neutral spine position should also describe the point of pain-free positioning, where maximal power can be obtained and balance can be preserved. Maintenance of the neutral spine position is important in day-to-day function and is the position in which training should occur. Neutral spine positioning allows for muscle training and development without increasing load, shear, and damage on the spine and other joints. The eventual goal of core stability training is to improve strength, endurance, balance, and the neuronal output of the trunk and limbs while maintaining a neutral spine position [4].

Table 10.2 Screening testsfor stability and flexibility

Hamstring screens		
Rotation screen		
Sit and reach		
Trunk flexion		
Trunk extension		
Hip extension		
Hip internal rotation		
Hip external rotation		

10.6.2 Flexibility

The examiner should begin by assessing the patient's posture and gait. Flexibility assessment should include examination of both the active and passive range of motion of the trunk, hips, and pelvic girdle, all the while assessing for laxity and stability of joints (Table 10.2; Fig. 10.9). It is important to compare side-to-side for asymmetries. Examination may begin by observing and palpating the spine to assess for anatomical alignment or deformity. The spine should be evaluated in the neutral spine position, which is the position of minimal loading on the spinal joints. Range of motion of the lumbar spine should be assessed in the functional planes of flexion, extension, side bending, and rotation (Figs. 10.10 and 10.11). Hip extension, flexion, internal and external rotation should be evaluated (Fig. 10.12). The major components under scrutiny throughout the evaluation are loss of range of motion or clinical instability, which is defined as the loss of the ability to maintain normal anatomical positions [16].

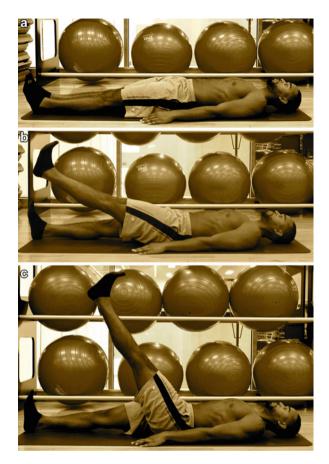
10.6.3 Motor Control

Proper balance is important before initiating a program to strengthen the core. To test for motor control, the examiner can utilize the passive repositioning test for each hip and single leg balance assessment with the patients' eyes open and then closed. With the repositioning tests the athlete will stop their passively moving leg at a target degree range of hip motion [31]. The single leg stance athletic test is useful to assess single limb stability and pelvic control.

10.6.4 Strength

When imbalance is present, dysfunction and damage often occurs on the weaker side [7, 8, 12]. Waldhelm and colleagues described eight isometric tests and one isoinertial test to assess relative core strength [31] (Table 10.3). These isometric

Fig. 10.9 Flexibility and mobility screens—the hamstrings test. Hamstring flexibility can be assessed and compared bilaterally with the athlete in the supine position elevating the leg to 80°–90° of flexion at the hip, without introducing pelvic tilt



tests were completed utilizing a Biodex System 3 Pro. The isoinertial test is simply a timed sit-up test, with the goal being to perform as many sit-ups in 60 s as possible [31].

10.6.5 Endurance

Endurance testing is integral to a comprehensive core assessment. Strength in the absence of endurance of core muscles is insufficient for most sports. This is due to the fact that endurance of muscles in the core allows for extended periods of stability. Since the core is recruited prior to most limb movements in order to brace the body for action, maintaining contraction during the course of the limb movement is required for stability. The goal of core endurance testing is to determine the length of time an athlete can maintain a static core engaging position. The four major tests to evaluate the core's endurance described by McGill and colleagues are the isometric prone bridge, isometric lateral bridge, isometric torso flexor, and isometric torso extensor endurance tests [33–35].

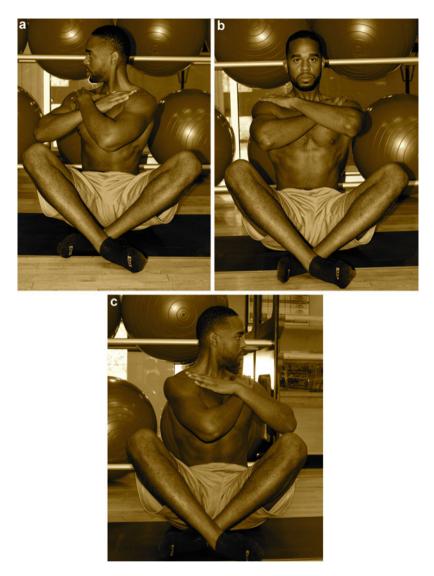


Fig. 10.10 Flexibility and mobility screens—the rotational test. To evaluate the abdominal and lumbar rotators, a seated-rotation position can be utilized. The athlete sits in the "Indian style" position, arms crossed over chest, and rotates left to right. Rotation is compared side to side, looking for asymmetry. (a) right rotation screen; (b) neutral position; (c) left rotation screen

The prone bridge is performed by having the patient lie prone, with forearms and toes on the floor, assessing the anterior and posterior core muscles (Fig. 10.13a). The pelvis should be in a neutral position. Failure of the test occurs when the pelvis moves from a neutral position into lordosis, with anterior rotation of the pelvis. A modified test position if the patient demonstrates inability to secure the standard position, is to position the patient with body weight over bent knees, rather than with the knees in extension and body weight resting on the toes, thus reducing the

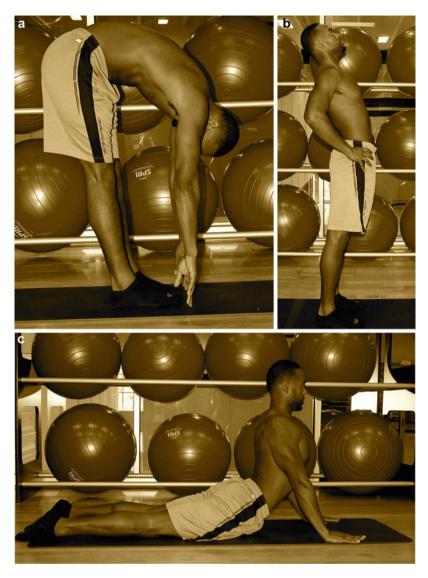


Fig. 10.11 Flexibility and mobility screen-trunk flexion/extension tests

lever arm. Enhanced assessment of the prone bridge can be performed with weights positioned on the back, or the forearms placed further cranially [4, 7, 8, 12, 34].

The lateral bridge assesses the lateral core muscles (Fig. 10.13b). This position assesses the abdominal obliques, quadratus lumborum and hip abductors, while limiting psoas participation and minimizing strain on the lumbar spine [35]. Failure occurs when there is loss of a straight posture and the hip tilts toward the table. An advanced exercise can be completed by further abducting the supporting arm and adding slight rotational movements while holding the position [12].



Fig. 10.12 Active external rotation (ER) and internal rotation (IR) can be assessed by having the athlete lay prone, flex the knee and rotation the leg inward to test ER and outward to test IR of the hip. Passive testing of ER can be completed by having the athlete lay supine with knee and hip flexed to 90 degrees. The examiner then externally and internally rotates the athlete's leg at the hip to assess for range of motion. (**a**) active external rotation; (**b**) active internal rotation; (**c**) passive external rotation; (**d**) passive internal rotation

Strength tests (*All maneuvers completed against resistance)
Trunk flexion*
Trunk extension*
Right hip extension*
Left hip extension*
Right hip abduction*
Left hip abduction*
Right hip external rotation*
Left hip external rotation*
Sit up test

Table 10.3 Strength testing in core assessment

Testing the torso extensors can be performed in prone position, with hip, pelvis, and knees secured on to a platform or table (Fig. 10.13c). The upper body is held out straight, without the support of the table, at 180°. This requires recruitment of the extensor muscles to a neutral position, while trying to avoid placing the spine into

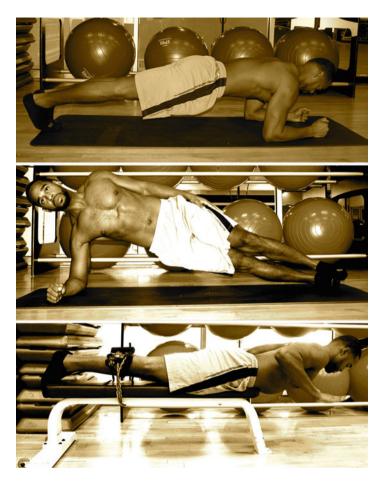


Fig. 10.13 (a) The prone bridge or plank for core assessment, particularly the anterior and posterior core muscles. (b) The lateral bridge or side bridge for lateral core assessment. (c) The torso extension test for posterior core assessment (also assesses components of the anterior and lateral core)

hyperextension. Failure occurs when the 180° posture is lost and the athlete falls into a flexed position [12]. The time for maintaining position should be around 60 s before fatigue is noted and maintenance of the testing posture is lost [12].

Testing the endurance of the torso flexors can be performed by timing how long the patient can hold a position of seated torso flexion (Fig. 10.14) with torso flexed to 60° and the knee/hips flexed to 90° . The toes should be secured under a toe strap or held by the examiner. Failure occurs when the patient's torso falls below 60° of flexion.

Standardized times representing a functional level of endurance are available for each of the described positions. Achieving those standardized times while maintaining a neutral spine is the goal. Training to hold the bridge and torso positions can be the foundation of developing a core strengthening program [4, 12].

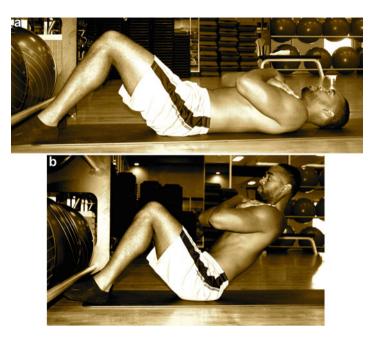


Fig. 10.14 The torso flexion test for anterior core assessment. Also known as the Curl-up test [4]

10.6.6 Functional Tests

"Functional movement is the ability to produce and maintain a balance between mobility and stability along the kinetic chain while performing fundamental patterns with accuracy and efficiency" [36, 37]. Performance in sports related activity is intimately related to one's strength, endurance, coordination, flexibility, and balance. The Functional Movement Screen (FMS) developed by Cook is frequently used as an initial tool to assess functional movement [36, 38, 39]. It is composed of 7 basic movements with a standardized scoring system. The movements include: deep squat, trunk-stability push up, bilateral hurdle steps, inline lunges, shoulder mobility, active straight leg raises, and rotary stabilities (Figs. 10.15, 10.16, and 10.17). It is important to note that in a study by Okada and colleagues that there was no significant relationship determined between the McGill core endurance tests and FMS [36]. It is believed that certain aspects such as mobility and coordination may account for the lack of association between the two [36]. Therefore if a patient or athlete lacks coordination or mobility, he or she may perform poorly on the FMS despite a core with excellent endurance capabilities.

10.6.7 Points for Further Investigation

Medical literature supports the importance of a strong core in the prevention of injury and static isokinetic endurance times [32]. However, currently there is a lack of literature evidence correlating core strength with improvements in sports performance

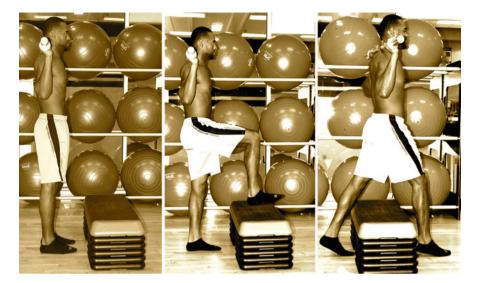


Fig. 10.15 Functional test–hurdle step test. The hurdle step evaluates the hip flexors and core muscles by maintaining a cross bar behind the shoulders while stepping over a hurdle of about 2 ft high, alternately leading with the left and then the right. Observations of balance control and posture should be noted during the process

within the athletic population [40]. This may be a result of the often non-translatable static testing measures used to assess the core to predict athletic performance [32]. Considerations should be made for investigation of core stability and functional movement tests that demonstrate statistically significant correspondence with performance [36]. Further research is required to identify the effects of the core on sports performance utilizing standardized sports specific dynamic core testing [32, 36, 40–42].

10.7 Exercise Programs for Core Stability

The core musculature is activated during all exercise involving either the trunk or the limbs. The degree of core activation during various exercise programs has been studied with surface electromyography (EMG) activity as well as with ultrasound evaluation of muscular thickness after training programs [21, 43–48]. However, surface EMG recordings [43] have shown low to moderate levels of muscle activity as compared to maximal voluntary isometric contraction (MVIC) levels. The levels of EMG muscle activity during standard core training programs are classically thought to be insufficient for significant strength gains, and rather thought to be compatible with endurance and neuromuscular re-education. Thus motor relearning or re-education of proper core muscle activity is the primary goal of performing targeted exercise programs for core stability [4, 22, 43–48].

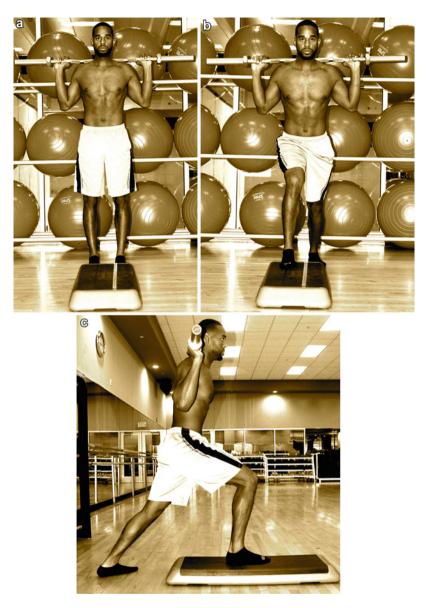


Fig. 10.16 Functional test–narrow lunge test. The narrow lunge is used to assess the hamstrings, quadriceps, pelvic muscle, and gluteal groups. It is performed by walking a plank, and then alternating squats with the left leading and then the right, while having a cross bar behind the shoulders. Observations to balance control and posture should be noted during the process

Various studies have demonstrated that EMG activity of the core musculature is greatest when performed on an unstable surface [21, 42, 44–48]. The greatest MVIC during core stability exercises has been associated with exercises against resistance

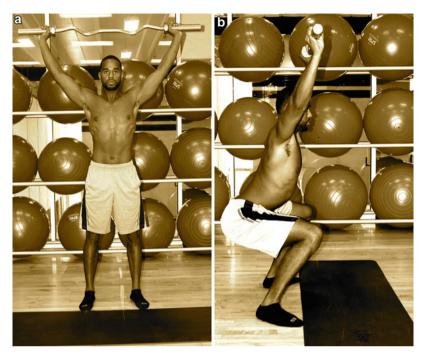
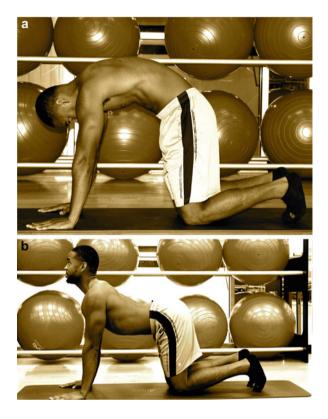


Fig. 10.17 Functional test–deep squat test. The deep squat is used to evaluate hip flexors and extensors, the lumbar spine, and abdominal muscles. It is done by holding a cross bar above the head, feet in a line, squatting down, and then back up. Examiner should pay special attention to maintenance of a neutral spine, balance control, and posture [4, 7, 8, 12]

on an unstable surface, such as single-leg-stance with elastic bands on a rocker board [47]. Multi-joint free-weight exercises have also been associated with a high degree of core muscle activity [44].

Studies have shown that specific populations of athletes or patients are likely to respond to core exercises, as they display movement abnormalities that can be addressed with neuromuscular re-education. Hicks, Fritz, Delitto, and McGill [28] attempted to develop a clinical prediction rule in determining patients who respond to core stability exercise. Prior injury and female sex have been associated with deficits in core neuromuscular control. Changes in movement patterns in low back, ACL, or ankle injury have been associated with changes in hip and core firing patterns (including timing, amplitudes, and endurance), as previously noted. This, combined with the functional improvements found after activities with low MVIC activation, supports the hypothesis that core dysfunction is primarily a neuromuscular control problem rather than a problem of isolated muscle weakness.

The abdominal exercises in a core exercise program can be stressful to the lumbar spine if performed incorrectly. A general core strengthening program should not begin in the first hour of awakening due to the increased hydrostatic pressure in the intervertebral disks after rising [4]. Neutral spine positioning must be learned during early training, to allow the development of control and endurance for future Fig. 10.18 Top image displays "the Cat Stretch" or "Cat Pose." Bottom image displays the "Camel Stretch" or "Camel Pose." The pose begins on hands and knees on the floor with elbows fully extended and hands directly under shoulders and flat on the ground. During exhalation, the chin should be dropped to the chest and the back arched. During inhalation, the chin is raised, head tilted back, the navel pushed towards the ground, and buttocks raised towards the ceiling, leading to extension



stabilization of the spine. Improper technique will result in loading forces distributed asymmetrically, placing a person at an increased risk for pain and dysfunction at a joint or point of motion [12, 33, 49].

After a person has been assessed and deemed safe to participate in an exercise program, an individualized exercise program should be created. Goals and level of participation of a patient with low-back pain will differ from those of an athlete training for a sport. A progressive exercise algorithm is proposed [22, 41, 47, 49], starting from the simplest and most balanced such as the bridge or double leg squat, to a crossed bridge or single leg squat on unstable surface. The goal of the progressive program is to first address any structural asymmetries such as lack of flexibility, and re-establish normal movement of the core and unlearn dysfunctional patterns (Fig. 10.18). New patterns of optimal neuromuscular control of the core are maintained during progressively dynamic and functional movements.

At the beginner level, there should be a focus on maintaining a neutral spine during daily motions, termed kinesthetic or *core awareness*. Neutral spine position is a position of the spine prior to flexion or extension where normal spinal curvatures, including pelvic tilt, lumbar lordosis, thoracic kyphosis, cervical lordosis, and head position are maintained. Popular exercise programs such as Yoga, Pilates, or Tai Chi

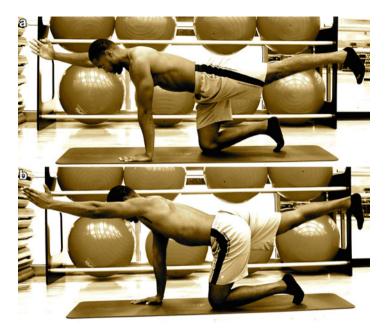


Fig. 10.19 Bird-Dog Exercise: actively strengthens both the anterior and posterior core. The pose begins with athlete on all fours, with spine and neck in neutral position. One leg is slowly extended while reaching forward with the contralateral arm. Both hips and shoulders should remain square while maintaining the neutral spine position

are based on core-stability exercise principles and teach kinesthetic awareness of the core. Correction of asymmetries in flexibility and range of motion should be addressed before moving to an intermediate level.

Intermediate level of training begins when the neutral spinal position can be maintained during simple tasks. At the intermediate level, the goal is to maintain spinal stabilization while increasing challenges to the core musculature (Fig. 10.19). Static exercises with balanced support such as the bridge or plank are performed at the intermediate level.

Advanced training addresses the ability to maintain spinal control with increased complexities of motions and activities (Fig. 10.20). This includes maintaining trunk control on unsteady surfaces and during dynamic or plyometric activity [10, 12, 21, 33, 41, 44] with maximal functional activation and neuromuscular control during motions in all planes: sagittal, frontal, and transverse/rotational plane. These types of exercises are thought to improve learning quick unconscious movements, sharpen reactions to postural shifts, and help with joint stabilization during motion [7, 8, 49]. Ultimately, the advanced athlete progresses to a program with plyometrics as well as functional exercises specific to his sport. Plyometrics is an explosive use of energy through the kinetic chain and produces maximal activation of core muscles and most functional movement patterns (Fig. 10.21) [41, 44].

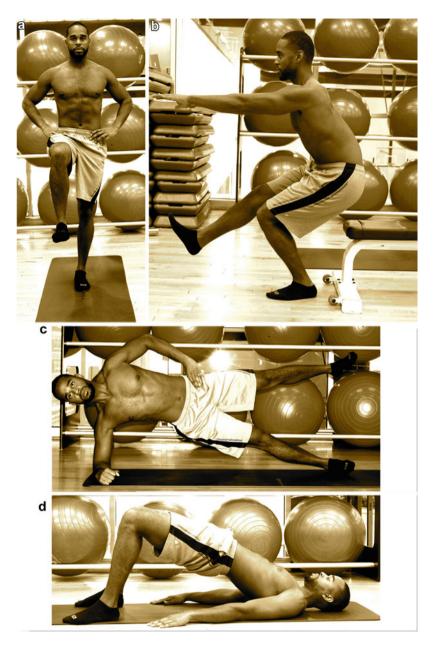


Fig. 10.20 (a) Single Leg Stance is utilized to test strength and static postural and balance control. (b) Single Leg Squat is a dynamic assessment and exercise to improve ankle/foot proprioception, knee/hip stability and core strength. Only consider this advanced test if patient is capable of adequately performing single leg stance test. (c) Side Brige/Side Plank with leg abducted is an advanced core training exercise. (d) Supine Hip Extension targets the gluteus and hamstring musculature (it is important to maintain spine in neutral positioning during this maneuver)

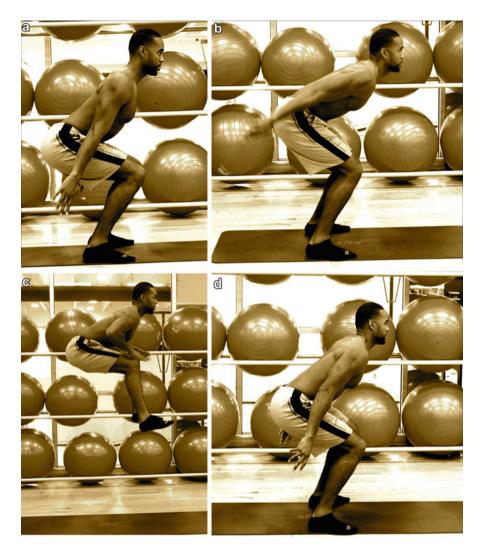


Fig. 10.21 The Double Leg Squat Jump is an advanced plyometric core stabilizing exercise that requires dynamic, high intensity activity, with intact functional movement patterns to allow for proper transference of energy, power, and force through the kinetic chain

10.7.1 Specific Exercises

The goal of the progressive core stability program (Tables 10.4 and 10.5) is to establish automatic motor patterns of postural awareness and core neuromuscular control in the motor cortex, so that conscious effort is not necessary during functional movement [7–10, 12, 33].

Table 10.4Types of corestability exercises

Floor core stabilization
Cat and camel
Plank
Supine bridge
Supine unilateral bridge
Side-bridge
Bird-Dog
Balance exercises
Single leg stance
Single leg squat
Single leg wobble board, toss
Plyometric exercises
Forward/backward/lateral
single or double leg jumps
Bounding
Multi-joint free weight exercises

Table 10.5 Progressive core program example exercises

Beginner
Neutral spine position
Cat and Camel
Flexibility exercises for hips, pelvis and spine
Intermediate
Plank and bridge
Side-bridge
Bird-Dog
Single leg stance
Static endurance exercises (as described in Sect. 10.4)
Advanced
Dynamic exercises (single leg squats, side-bridge with trunk rotation or leg abduction)
Plyometric exercises (jumping exercises)
Sport-specific functional exercises
Multi-joint free weight exercises

10.8 Conclusion

Core stability is necessary to efficiently transmit forces from the limbs to the trunk, provide support to the adjacent joints, and provide proper functional alignment of the limbs and trunk. Dysfunction of core activation patterns has been associated

with injury as well as decreased sports performance. Core stability exercises primarily address imbalances as well as improve neuromuscular activation patterns.

In the clinical vignette, while undergoing physical therapy it was quickly evident that case athlete had deficiencies in his core, which resulted in pain in the more distal structures and a decreased level of function. On follow-up, after entering in a core-training program, he was pain free and better conditioned. Thus, the practical application of the knowledge of core anatomy and biomechanics can be beneficial to the clinician in the treatment of various patients including high-level athletes.

References

- 1. Nadler SF. Visual vignette: injury in a throwing athlete: understanding the kinetic chain. Am J Phys Med Rehabil. 2004;83(1):79.
- 2. Schuenke M, et al. Thieme atlas of anatomy, general anatomy and musculoskeletal system. New York: Thieme; 2006.
- 3. Netter FH. Atlas of human anatomy. 2nd ed. Canada: Icon Learning Systems LLC; 2001.
- 4. Akuthota V, Nadler SF. Core strengthening. Arch Phys Med Rehabil. 2004;85:S86-92.
- 5. Norris CM. Abdominal muscle training in sport. Br J Sports Med. 1993;27(1):19-27.
- Callaghan JP, Patla AE, McGill SM. Low back three-dimensional joint forces, kinematics, and kinetics during walking. Clin Biomech (Bristol, Avon). 1999;14:203–16.
- Barr KP, Griggs M, Cadby T. Lumbar stabilization: core concepts and current literature, part 1. Am J Phys Med Rehabil. 2005;84:473–80.
- Barr KP, Griggs M, Cadby T. Lumbar stabilization: a review of core concepts and current literature, part 2. Am J Phys Med Rehabil. 2007;86:72–80.
- Nadler SF, Malanga GA, DePrince M, et al. The relationship between lower extremity injury, low back pain, and hip muscle strength in male and female college athletes. Clin J Sport Med. 2000;80:89–97.
- Nadler SF, Malanga GA, Feinberg JH, et al. Relationship between hip muscle imbalance and occurrence of low back pain in collegiate athletes: a prospective study. Am J Phys Med Rehabil. 2001;80:572–7.
- 11. Nadler SF, Malanga GA, Bartoli LA, et al. Hip muscle imbalance and low back pain in athletes: influence of core strengthening. Med Sci Sports Exerc. 2002;34:9–16.
- 12. Bliss LS, Teeple P. Core stability: the centerpiece of any training program. Curr Sports Med Rep. 2005;4:179–83.
- Keankaampaa M, Taimela S, Laaksonen D, et al. Back and hip extensor fatigability in chronic low back pain patients and controls. Arch Phys Med Rehabil. 1998;79:412–7.
- Beckman SM, Buchanan TS. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. Arch Phys Med Rehabil. 1995;76:1138–43.
- 15. DeVita P, Hunter PB, Skelly WA. Effects of a functional knee brace on the biomechanics of running. Med Sci Sports Exerc. 1992;24:797–806.
- 16. Panjabi M. The stabilizing system of the spine: I. function, dysfunction, adaptation and enhancement. J Spinal Disord. 1992;5:383–9.
- Vleeming A, Pool-Goudzwaad AL, Stoeckart R, et al. The posterior layer of the thoracolumbar fascia: its function in load transfer from spine to legs. Spine. 1995;20:753–8.
- Solomonow M, Zhou B, Harris M, et al. The ligamento-muscular stabilizing system of the spine. Spine. 1998;23:2552–62.
- Juker D, McGill S, Kropf P, Steffen T. Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Med Sci Sports Exerc. 1998;30:301–10.

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- Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord. 1998;11:46–56.
- Youdas JW, Boor MM, Darfler AL, Koenig MK, Mills KM, Hollman JH. Surface electromyographic analysis of core trunk and hip muscles during selected rehabilitation exercises in the side-bridge to neutral spine position. Sports Health. 2014;6(5):416–21.
- 22. Huxel Bliven KC, Anderson BE. Core stability training for injury prevention. Sports Health. 2013;5(6):514–22.
- Ambegaonkar JP, Mettinger LM, Caswell SV, Burtt A, Cortes N. Relationships between core endurance, hip strength, and balance in collegiate female athletes. Int J Sports Phys Ther. 2014;9(5):604–16.
- Gordon AT, Ambegaonkar JP, Caswell SV. Relationships between core strength, hip external rotator muscle strength, and star excursion balance test performance in female lacrosse players. Int J Sports Phys Ther. 2013;8(2):97–104.
- Filipa A, Byrnes R, Paterno MV, Myer GD, Hewett TE. Neuromuscular training improves performance on the star excursion balance test in young female athletes. J Orthop Sports Phys Ther. 2010;40(9):551–8.
- Zazulak BT, Hewett TE, Reeves NP, Goldberg B, Cholewicki J. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. Am J Sports Med. 2007;35(7):1123–30.
- Saal JA. Dynamic muscular stabilization in the nonoperative treatment of lumbar pain syndromes. Orthop Rev. 1990;19(8):691–700.
- Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Arch Phys Med Rehabil. 2005;86(9):1753–62.
- 29. Wang XQ, Zheng JJ, Yu ZW, Bi X, Lou SJ, Liu J, Cai B, Hua YH, Wu M, Wei ML, Shen HM, Chen Y, Pan YJ, Xu GH, Chen PJ. A meta-analysis of core stability exercise versus general exercise for chronic low back pain. PLoS One. 2012;7(12), e52082.
- 30. Stuber KJ, Bruno P, Sajko S, Hayden JA. Core stability exercises for low back pain in athletes: a systematic review of the literature. Clin J Sport Med. 2014;24(6):448–56.
- Waldhelm A, Li L. Endurance tests are the most reliable core stability related measurements. J Sport Health Sci. 2012;1(2):121–8.
- Shinkle J, Nesser TW, Demchak TJ, McManus DM. Effect of core strength on the measure of power in the extremities. J Strength Cond Res. 2012;26(2):373–80.
- Fredericson M. A systematic approach to core strengthening for improved athletic improvement. Lecture handout, 2007.
- 34. Gilchrist RV, Frey ME, Nadler SF. Muscular control of the lumbar spine. Pain Physician. 2003;6:361–8.
- 35. McGill SM, Childs A, Liebenson C. Endurance times for low back stabilization exercises: clinical targets and training from a normal database. Arch Phys Med Rehabil. 1999;941–44.
- Okada T, Huxel KC, Nesser TW. Relationship between core stability functional movement and performance. J Strength Cond Res. 2011;25(1):252–61.
- Mills JD, Taunton JE, Mills WA. The effect of a 10 week training regimen on lumbo-pelvic stability and athletic performance in female athletes: a randomized controlled trial. Phys Ther Sport. 2005;6:60–6.
- Cook G. Baseline sports-fitness testing. In: Foran B, editor. High performance sports conditioning. Champaign: Human Kinetics; 2001. p. 19–47.
- 39. Cook G. Weak links: screening an athlete's movement patterns for weak links can boost your rehab and training effects. Train Cond. 2002;12:29–37.
- 40. Reed CA, Ford KR, Myer GD, Hewett TE. The effects of isolated and integrated 'core stability' training on athletic performance measures: a systematic review. Sports Med. 2012;42(8):697–706.
- Hill J, Leiszler M. Review and role of plyometrics and core rehabilitation in competitive sport. Curr Sports Med Rep. 2011;10(6):345–51.

- 42. Prieske O, Muehlbauer T, Borde R, Gube M, Bruhn S, Behm DG, Granacher U. Neuromuscular and athletic performance following core strength training in elite youth soccer: Role of instability. Scand J Med Sci Sports. 2015;6.
- 43. Hibbs AE, Thompson KG, French DN, Hodgson D, Spears IR. Peak and average rectified EMG measures: which method of data reduction should be used for assessing core training exercises? J Electromyogr Kinesiol. 2011;21:102–11.
- Martuscello JM, Nuzzo JL, Ashley CD, Campbell BI, Orriola JJ, Mayer JM. Systematic review of core muscle activity during physical fitness exercises. J Strength Cond Res. 2013;27(6):1684–98.
- 45. Yun K, Lee S, Park J. Effects of closed chain exercises for the lumbar region performed with local vibration applied to an unstable support surface on the thickness and length of the transverse abdominis. J Phys Ther Sci. 2015;27(1):101–3.
- Bolgla L, Cook N, Hogarth K, Scott J, West C. Trunk and hip electromyographic activity during single leg squat exercises do sex differences exist? Int J Sports Phys Ther. 2014;9(6):756–64.
- Calatayud J, Borreani S, Martin J, Martin F, Flandez J, Colado JC. Core muscle activity in a series of balance exercises with different stability conditions. Gait Posture. 2015;42(2):186–92.
- 48. Granacher U, Schellbach J, Klein K, Prieske O, Baeyens JP, Muehlbauer T. Effects of core strength training using stable versus unstable surfaces on physical fitness in adolescents: a randomized controlled trial. BMC Sports Sci Med Rehabil. 2014;6(1):40.
- Akuthota V, Ferreiro A, Moore T, Fredrickson M. Core stability exercise principles. Curr Sports Med Rep. 2008;7(1):39–44.

Chapter 11 Manual Medicine of the Hip and Pelvis

Charles W. Webb

The important of injuries to the hip is too much overlooked. To the Sports Physician it should be a subject of the deepest thought.

A.T. Still

Clinical Pearls

- Do not forget to treat sacroiliac joint and the lumbar spine. The supine direct articulatory technique for the lumbar spine is a quick, easy, and very low risk treatment.
- The six muscle groups of the pelvis are the adductors, abductors, external rotators, internal rotators, extensors, and flexors. Always look for a restriction of motion as a potential cause for the discomfort.
- Somatic dysfunction is impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures; and related vascular, lymphatic, and neural elements.
- Somatic dysfunction is found in areas where TART exists. (TART: tissue texture changes, asymmetry, restriction in motion, and tenderness)
- The minor motions, not the major motions, usually become restricted when somatic dysfunction occurs.

11.1 Case Presentation

A 25-year-old married woman, a recreational runner presents herself to the office with the chief complaint of right hip pain. The pain has been present for 3 weeks, worse during her last run after slipping off a curb awkwardly. The pain gets worse with

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P.H. Seidenberg et al. (eds.), *The Hip and Pelvis in Sports Medicine and Primary Care*, DOI 10.1007/978-3-319-42788-1_11

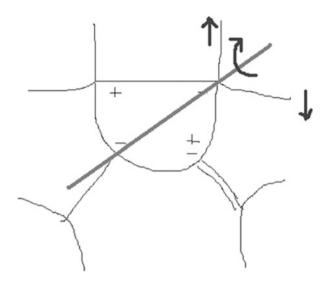


Fig. 11.1 Graphic depiction of the case pelvic findings

activity and with prolonged sitting, it lightens during rest. She is currently training for a marathon. Her last menstrual period was within the last month. She is not pregnant and she uses oral contraception regularly. She is not taking any other medications other than an occasional acetaminophen or ibuprofen for headaches and menstrual pain. Her past medical and surgical histories are negative, and she has never been pregnant. She has no known allergies. She denies tobacco use of any kind, nor any illicit drugs. She admits to having an occasional glass of wine, and she drinks coffee in the mornings.

Physical examination reveals stable vital signs. She has a positive Ober's test on the right. Her popliteal angle is 140° bilaterally. She has a positive modified Thomas test on the right as well as a positive standing and seated flexion test on the right. She has mild tenderness over her right greater trochanter as well as the right sacroiliac (SI) joint. Her sacral sulcus is deep on the left, and the inferior lateral angle (ILA) of the sacrum is shallow on the right. She has good motion of the sacral base on the left, with none on the right and minimal motion of the right ILA. She has decreased internal and external rotation of the hip on the right compared to the left. Patrick/FABER test is negative. Her piriformis muscle is tight on the right with several noted tender points on both the right piriformis and psoas (Fig. 11.1).

X-rays of the hip and pelvis are negative for evidence of fracture, arthritis, and other bone abnormalities.

11.2 Introduction

How do you approach this patient and many more who present themselves to the sports clinic with hip pain? This is a very typical patient seen in many sports medicine clinics. They are usually athletes who are training for a specific event and have pushed themselves to the point where even the most benign mechanism of injury is enough to cause them to "fall off the edge" and have an injury that necessitates a significant decrease in training. The first step in evaluating a patient with this type of presentation is to have a thorough systematic examination process to ensure an accurate pathoanatomic diagnosis. (SOR-C)

Hip pain/pelvis pain in the athlete can be from a multitude of problems. These problems range from simple greater trochanteric bursitis to fracture or abscess in the hip musculature (Table 11.1) [1–8].

Both osteopathic and chiropractic philosophies embrace an approach to wellness through knowledge of interrelationships of structure and function, and a search for the cause of the patient's problems. When applied to addressing pain in the hip and pelvis, one must use a global approach to narrow the differential. Foot and lower extremity misalignment, joint restrictions, muscular imbalances, leg length discrepancy, and sport specific mobility abnormalities can all place abnormal loads on the hip and cause pain. Once the provider is confident that the cause of the problem is musculoskeletal, the search for dysfunction begins [1, 6, 9].

In order to quickly and accurately diagnose hip dysfunction, the sports provider must understand the muscles and ligaments of the hip and pelvis, the lymphatic drainage patterns of the leg, the nerves of the lumbar and sacral plexi in addition to the sympathetic innervations and associated reflexes.

11.3 Functional Anatomy of the Hip and Pelvis

The hip and pelvis are built for support and motion. Composed of the largest bones and muscles in the body forming foundation for locomotion. The body's center of gravity is located in the pelvis, just anterior to the second sacral vertebra. Once the provider understands the anatomy of the hip and pelvis, it is easy to understand how dysfunction (decrease in motion) of the hip can produce not only lower extremity pain, but also low back pain, pelvic pain, and changes in the gait cycle that may lead to other pain syndromes [4, 10, 11].

Functional anatomy relationships of the hip and pelvis are the key to determining the cause of the dysfunction and correcting it. The inominates articulate with the sacrum via the sacroiliac joints. The pubic symphysis acts as an anterior static bar providing stability to the pelvis during both ambulation and sitting. Tension on the ligaments, which cross the SI joint, can cause dysfunction in the leg. Patients with iliolumbar ligament sprains often present thinking they have an inguinal hernia; however, the physical findings are not supportive. Somatic dysfunction of the sacrum, inominates, and lubosacral junction are common causes of hip and pelvic pain in active patients. In a subset of patients the sciatic nerve divides before entering the gluteal region and the peroneal portion passes through the piriformis muscle; in small percentage, it will pass superior to the piriformis muscles. The piriformis muscle is found in the midpoint of a triangle made between the posterior superior iliac spine (PSIS), the coccyx, and the superior aspect of the greater trochanter

 Table 11.1
 Differential diagnosis for hip and pelvic pain

218

Spondylolisthesis	
Slipped capital epiphysis	
Congenital short leg	
Congenital asymmetry of the facets	
Inflammatory arthritis	
Apophyseal injury	

Fig. 11.2 (a) Graphic depiction of the location of the piriformis muscle. (b) Location of the piriformis tender points
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(Fig. 11.2). When the composite sciatic nerve is compressed secondary to piriform is spasm, the superficial nerve bundles are mechanically irritated and the resultant pain can radiate down the leg, usually not below the knee. Pain from nerve root pressure usually radiates below the knee [8, 11-13].

The hip is the term for the composite of the inominate bone, the head of the femur, and the acetabular joint. The acetabulum of the inominate is composed of portions of

Table 11.1 (continued)

the ilium, ischium, and the pubic bone. The femur travels along three axes in the hip joint. The transverse axis is where flexion (130°) and extension (35°) occur. Abduction (55°) and adduction (35°) occur along the anteroposterior (AP) axis, and internal rotation (35°) and external rotation (45°) occur along the longitudinal axis.

It is the minor motions, not the major motions that usually become restricted when somatic dysfunction occurs, and it is the compensations for these minor motion restrictions that become the issue over a period of time. Posterior and anterior glide of the femoral head in relation to internal and external rotation of the hip are the minor motions. Flexion, extension, abduction, and adduction are considered the major motions of the hip [12, 14–16].

As other chapters of this book focus on various treatments of the pelvis and hip in regard to sports medicine and primary care, this chapter will focus on manual medicine techniques. The use of manual therapy is an ancient healing form that has been documented as early as 2,700 B.C. [17]

11.4 Manipulation Basics

Before we can discuss manual treatments, we must first understand some basics about manipulations. Osteopathic and chiropractic manipulations are forms of manual medicine that stress the need for normal and symmetrical motion in the joints. These manipulations are done to enhance or restore motion in a joint. By restoring motion to a joint and homeostasis to the tissues we are allowing the body to function in a more optimum state of health. The growth of manual medicine has been fueled by patient outcome. The rise of modern-day manipulative medicine by osteopathic physicians, chiropractors, and physical therapists has come behind that of efficacy studies such as the RAND study by Dr. Paul Shekelle, who said: "Spinal Manipulation is the most commonly used conservative treatment for back pain supported by the most research evidence of effectiveness in terms of early results and long term effectiveness." (SOR = B) [18].

There are three general considerations that a provider must understand before deploying manual medicine as a treatment option: (1) One technique may treat more than one type of dysfunction; (2) More than one technique may be required to treat a single type of dysfunction; and (3) All techniques work best when applied to a specific diagnosis. The goals of treatment are to enhance the movement of body fluids, modify somatosomatic, viscerosomatic, and viscerosomatovisceral reflexes, provide maintenance treatment to irreversible conditions (osteoarthritis), and to mobilize articular restrictions.

11.5 Methods of Manipulation

There are three distinct methods of manipulation: direct, indirect, and combined. Direct is when the restrictive barrier is engaged in one or more planes of motion normal to the articulation so that the activating force applied may carry the dysfunctional component through the restrictive barrier. Indirect is when the provider moves the dysfunctional component away from the restrictive barrier (the direction that it wants to go) in one or more planes of motion normal to the articulation. The joint, structure, or tissue is moved to the point of balanced ligamentous tension or ease. Combined is a combination of direct and indirect methods. This method is most useful in treating myofascial tissues.

11.6 Activating Forces

There are various activating forces that are used in treating somatic dysfunction. Many are beyond the scope of this text. If further reading is desired, please refer to the references at the end of this chapter. The activating forces discussed here will be discussed in detail in the treatment section of this chapter. *Patient cooperation* is an activating force that is required when treating dysfunction with strain/counter strain (treatment of tender points) and when using muscle energy. The patient is instructed to move his/ her body in specific directions involving various planes of articulation to aid in the mobilizing of an area of restriction. In muscle energy techniques, the activating force is the *patient's contraction of muscles* in a specific direction against the physician's counterforce. A *physician guiding force* is exerted when the physician positions the patient away from the restrictive barrier to a point of release and then guides the tissue or joint somatic dysfunction through various positions that move with ease until the dysfunctional pathway has been completely retraced. Springing is also known as the low velocity moderate amplitude force: the provider makes contact upon the restrictive barrier and with variable degrees of force, springs the structure with intermittent pressures. High velocity low amplitude (HVLA) is used only with direct methods: the restrictive barrier is properly engaged to yield along one of the planes of a joint. A HVLA force is applied to move the joint or tissues through the restrictive barrier. An articulatory procedure is of a low velocity and low to a high amplitude technique where a joint is carried through its full range of motion [9, 14, 19–22].

11.7 Goals of Treatment

The primary goal of treatment is to restore function to the tissues. Hypo or hyper mobility of a joint leads to muscle imbalance, altered movement, and eventually pain syndrome. The few contraindications to manual medicine are listed in Table 11.2.

11.8 Manual Medicine Techniques

There are a plethora of manipulative techniques for the hip and pelvis in the literature. Covering all of these techniques is not practical for this text. The techniques presented here are the author's preferred techniques and the ones that I teach on a regular basis. Considerations for these techniques include the time involved in the clinic to perform, the ease of patient education, and the ease of teaching to other providers. I hope you find these techniques helpful. **Table 11.2**Contraindicationsto manipulation

Conditions
Fracture
Acute rheumatoid arthritis
Joint instability
Infection
Malignancy
Advanced neurological
deficit/urinary incontinence
Severe osteoporosis

11.8.1 Counterstrain [9, 15, 21]

11.8.1.1 Psoas

Indications for psoas strain include hip flexor contracture and anterior hip pain. The psoas tender points are routinely found just medial to the anterior superior iliac spine (ASIS), upon palpation. They are treated using a supine counter strain technique (Fig. 11.3.). The provider places his fingers on the tender points and then flexes the hip to fold the psoas around the tender point. This is the point where the pain from the tender point is gone or 80% of the tenderness is gone. Then the provider holds this position for approximately 90 s or until the tender point melts away under the provider's fingertips. Then the provider moves the legs back to the neutral position. This must be done passively to avoid re-aggravating the tender point. The premise is that the Golgi tendon apparatus of the muscle fibers is being reset.

11.8.1.2 Piriformis

Piriformis strains are restricted internal rotation and posterior hip pain. The piriformis tender points are treated in a similar fashion (Fig. 11.4), but the patient is in the prone position and the hip is extended and rotated to find the position of comfort. This is again held until the point melts away, approximately 90 s, then the hip is passively moved back to the neutral position.

11.8.2 Muscle Energy Techniques [15, 20, 22, 23]

11.8.2.1 Anteriorly Rotated Inominate, or the Inferior Pubic Shear

Indications are osteitis pubis, groin strain, low back pain, hip or pelvic pain, and restriction in hip extension.

Findings show that the ASIS is inferior, the pubic bone is inferior, and the PSIS is superior on the affected side. There is a positive flexion test on the affected side; the pelvic rock test will also be positive (restricted) on the affected side.

Fig. 11.3 Treatment of psoas tender points



Fig. 11.4 Treatment of piriformis tender points



To treat, the patient is asked to lie supine on the table; the provider stabilizes the opposite ASIS with one hand then flexes the knee and the hip. The provider then leans on the patient's knee to flex and abduct the thigh to the barrier (ligamentous tension). The patient is instructed to push against the provider's equally applied force for 3–5 s, then instructed to relax, pause, and asked to loosen up/ take up the slack. This is repeated 3–5 times or until the ASIS/pubes are equal.

11.8.2.2 Posterior Rotated Inominate, or Superior Pubic Shear

Indications are osteitis pubis, groin strain, low back pain, hip or pelvic pain, and restriction in hip flexion.

Findings show the ASIS is superior, the pubic bone is superior, and the PSIS is inferior on the affected side. There is a positive flexion test on the affected side; the pelvic rock test will also be positive (restricted) on the affected side.

The patient, while supine on the table, moves the affected side to the edge of the table and drops the involved leg off the side of the table. The clinician places one hand on the opposite ASIS to stabilize the pelvis and the other hand on the thigh just above the knee. The clinician extends the affected hip to the barrier (ligamentous tension) and has the patient push up against the counterforce for 3–5 s, pause, and take up the slack to reengage the barrier. This is repeated 3–5 times or until the ASIS/pubes are equal. The pressure needed from the patient is 8–10 pounds of force, so caution them not to throw you across the room.

11.8.2.3 SI Joint Dysfunction/Pubic Shear

Indications are osteitis pubis, groin strain, low back pain, hip/pelvic pain, and SI joint pain from sacral dysfunction.

Findings include a positive flexion test on the side with the dysfunction. The pelvic rock test is positive. The pubic symphysis or the SI joint is tender to palpation at the sacral base or the lumbosacral junction.

To treat, the patient is asked to lie supine on the examination table. The knees and the hips are flexed to 90°. The clinician wraps his/her arm around the patient's knees and asks the patient to separate the knees with a strong effort. This will stretch the abductors and cause a loosening of the SI joint. Repeat this 3–5 times. With the patient in the same position, the clinician separates the patient's knees and places his/her forearm between the patient's knees with the hand on the knee and the elbow on the opposite knee. Then the patient is asked to push the knees together with a strong effort. This is an isometric contraction that should be held for 3–5 s and repeated 3–5 times. Then reassess for ASIS/pubes equality.

11.8.2.4 Restricted Hip Abduction

Indications are osteitis pubis, greater trochanteric bursitis, groin strain, hip pointer (contusion), and snapping hip syndrome (iliotibial band [ITB] tightness).

Findings show decreased range of motion in abduction of the hip compared to the uninvolved side.

For treatment, the patient is asked to lie supine on the examination table with the involved hip near the edge of the table. The provider stands between the patient's legs, lifts the involved leg with the caudal hand, and stabilizes the opposite ASIS with the cephalad hand. The patient adducts the leg against the provider's counterforce for 3–5 s. When the patient relaxes, the provider pauses and then reengages the barrier. Repeat 3–5 times, then reassess for increased symmetrical motion.

11.8.2.5 Restricted Hip Adduction

Indications are osteitis pubis, greater trochanteric bursitis, hip pointer, and snapping hip syndrome.



Fig. 11.5 Treatment for restricted hip adduction

Findings demonstrate a decreased range of motion in adduction of the hip compared to the uninvolved side.

For treatment, the patient is asked to lie supine on the examination table with the feet at the end of the table (Fig. 11.5). The provider lifts the involved leg at the proximal tibia and adducts it to the restrictive barrier. The patient abducts the leg against the provider's counterforce for 3–5 s. When the patient relaxes, the provider pauses then takes up the slack to reengage the barrier. Repeat the process 3–5 times and reassess for improved range of motion in adduction.

11.8.2.6 Restricted Hip Extension

See anterior rotated inominate. The treatment techniques are the same as that for the anterior rotated inominate.

11.8.2.7 Restricted Hip Flexion

See posterior rotated inominate. The treatment techniques are the same as that for the posterior rotated inominate.

11.8.2.8 Restriction in Hip External Rotation

Indications include osteitis pubis, hip pointer, snapping hip syndrome, and tight internal rotators.

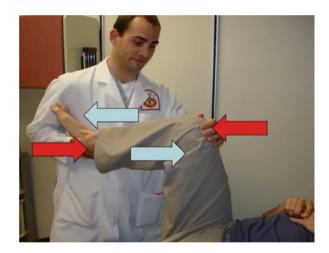
Findings show a decreased range of motion in external rotation of the hip.

To treat, the patient is asked to lie supine on the examination table with the affected hip flexed to 90° and the knee flexed to 90° (Fig. 11.6). The provider places his/her cephalad hand on the patent's knee and uses the caudad hand to apply force



Fig. 11.6 Treatment for restricted hip external rotation

Fig. 11.7 Treatment for restricted hip internal rotation



to the ankle, externally rotating the hip to the restrictive barrier. The patient is asked to push against the provider's hand to internally rotate the hip for 3-5 s and then asked to relax. The provider pauses and takes up the slack to reengage the barrier. Repeat 3-5 times then reassess for improved range of motion.

11.8.2.9 Restricted in Internal Rotation

Indications are osteitis pubis, hip pointer, snapping hip syndrome, and tight external rotators (piriformis).

Findings show a decreased range of motion in internal rotation and tight external rotators of the hip.

For treatment, the patient is asked to lie supine on the examination table with the affected hip and knee flexed to 90° (Fig. 11.7). The provider places his/her cephalad hand on the patient's knee and uses the caudad hand to apply force to the ankle, internally rotating the hip to the restrictive barrier. The patient is asked to push against the provider's hand to externally rotate the hip for 3–5 s., and then asked to relax. The provider will pause and then take up the slack to reengage the barrier. Repeat 3–5 times then reassess for improved range of motion.

11.8.2.10 Sacral Base Anterior

Indications include low back pain, pelvis pain, and hip pain.

On examination, sacral sulci are deep bilaterally, ILAs are level bilaterally, the sacral base will move anteriorly bilaterally, and the sacrotuberous ligaments are tight bilaterally.

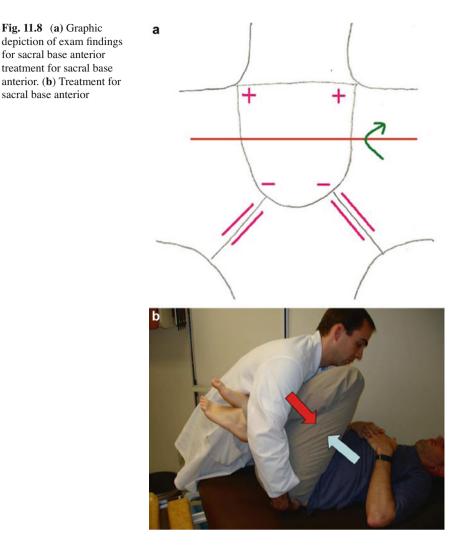
For treatment, the patient is asked to lie supine on the table with both hips and knees flexed to the chest (Fig. 11.8). The provider places his/her hands on the sacral base bilaterally to monitor motion and asks the patient to push against the force for 3–5 s, then asked to relax. The provider applies pressure to take up the slack and reengage the barrier. Repeat 3–5 times and then reassess for improved sacral positioning and motion.

11.8.2.11 Sacral Torsion About the Same Axis

Indications are low back pain, pelvic pain, hip pain, hip pointer, and snapping hip syndrome.

Findings reveal left rotation about a left oblique axis. They will be the opposite for a right-sided rotation on a right axis. Left ILA is posterior and inferior compared to the right. The left PSIS will be inferior; the right sacral sulcus will have motion and may be deeper than the left. The segmental examination of the lumbar spine will have an L5 sidebent left rotated right (SlRr) and the left sacrotuberous ligament will be tense with the right being loose.

To treat, the patient is placed prone on the examination table (Fig. 11.9). With the patient relaxed, have the patient move the hips so that the left hip is on the examination table and the knees and hips are flexed to 90° . The provider sits on a stool next to the table, supporting the patient's legs on the provider's thigh. The provider holds the patient's ankles and asks the patient to push them toward the ceiling for 3-5 s and then asked to relax. The provider applies a counterforce, then pauses and takes up the slack to reengage the barrier. The provider's other hand is monitoring the motion of the sacral base and inducing a left rotation of the spinous processes of L4–L5 vertebra. Repeat this process 3-5 times then reevaluate the sacrum for improved motion. This may be done on the right side for a right sacral torsion about a right oblique axis. This technique, when done with the patient supine to start, will treat a right rotation about a left oblique axis (Fig. 11.10).



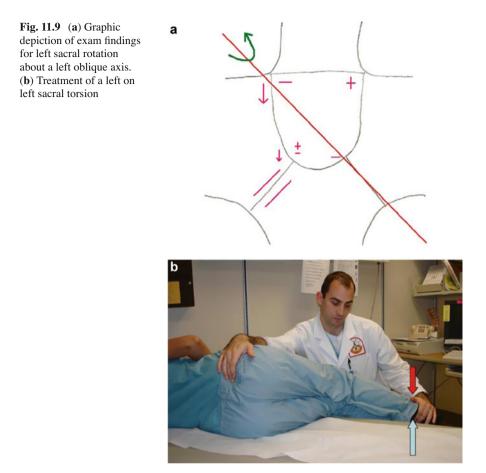
11.8.3 High Velocity Low Amplitude Techniques [15, 20, 22, 23]

11.8.3.1 Unilateral Sacral Shear, Superior Inominate Shear

Indications include low back pain, pelvic pain, SI joint pain, hip pain, and superior iliac shear.

On examination, one ILA is markedly inferior and posterior compared to the other. PSIS are usually equal, but may be superior on the affected side. The sacral base on the affected side may be anterior. Flexion and pelvic rock tests will be positive on the affected side.

To treat, the patient is placed supine with a small, rolled-up towel under the affected ILA (Fig. 11.11). From the end of the table, the provider grasps the affected lower



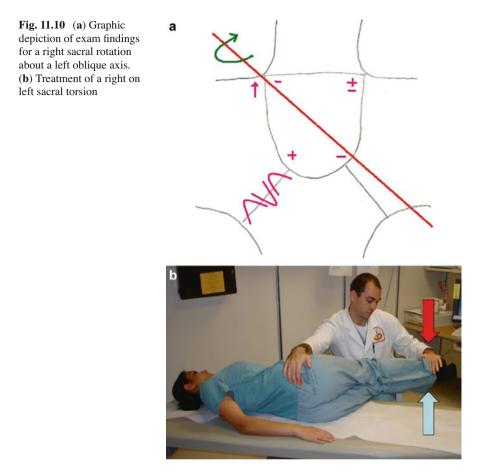
extremity with both hands and abducts and internally rotates the hip to a point of the closed packed position. Gentle traction is applied along the long axis of the leg. The patient is asked to take a few deep breaths and exhale fully; at the end of the full exhalation, the provider applies a short quick tug on the leg, taking care not to pull on the ankle itself. The leg is then returned to midline on the table and the patient is reexamined.

11.8.3.2 Right Posterior Innominate

Indications are low back pain and SI dysfunction.

Findings: PSIS inferior on the affected side, ASIS superior on the affected side, and positive seated and/or standing flexion test on the affected side.

Using a muscle energy technique to treat this condition, have the patient lay supine on the table with the affected side on the edge. Then have the patient drop the affected leg off the table, then with the provider holding the leg down off the table, have the patient push up on the providers hand. This contraction is sustained for 8-10 s, then the provider pauses before taking up the ligamentous slack. This can be repeated until the rotation of the innominate is corrected.



In an alternate technique, the patient is asked to lie on his/her side; the lower thigh and leg are straight and the upper thigh and leg are flexed (Fig. 11.12). The pelvis is brought toward edge of the table. The provider is anterior to the patient with the caudad hand making pisiform contact medial and inferior to the PSIS and the cephalad hand on the front of the shoulder. The caudad hand drives the PSIS anterior accompanied with a body drop.

11.8.3.3 Sacral Base Posterior

Indications include low back pain and SI joint pain. This condition is very common in the postpartum period.

On examination, the sacral sulci will be shallow bilaterally; there will be some motion at the ILAs bilaterally but not the sacral base. The PSISs and ILAs are equal and the sacrotuberous ligaments are relaxed bilaterally.

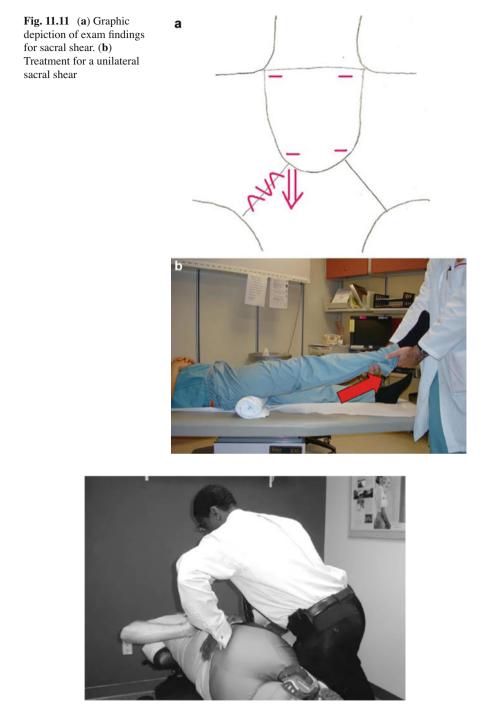
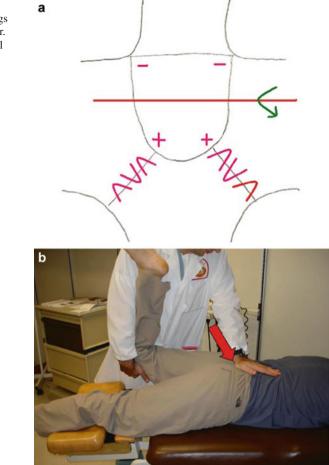


Fig. 11.12 Treatment for right posterior innominate shear using a high velocity low amplitude (HVLA) technique



To treat, the patient is placed prone on the examination table and is asked to rise up on their elbows (Fig. 11.13). The provider places the heel of one hand on the sacral base and the other hand on the lower extremity to stabilize the patient. Pressure is applied to the sacral base to engage the barrier. As the patient exhales a short quick thrust is applied to the sacral base. The sacrum is then rechecked.

11.8.4 Table Assisted Technique

11.8.4.1 Right Posterior Innominate

Patient is placed prone with the provider standing on either side of the table (Fig. 11.14). The provider's contact hand is on medial inferior aspect of the involved PSIS. The stabilization hand is on the inferior aspect of the ischial tuberosity on the

Fig. 11.13 (a) Graphic depiction of exam findings for a sacral base posterior. (b) Treatment for a sacral base posterior

Fig. 11.14 Treatment for right posterior innominate shear using a table assisted technique



uninvolved side. The thrust or line of correction is posterior to anterior with an inferior-to-superior aspect.

11.8.5 Articulatory Technique for the Lumbar Spine and the SI Joint [15]

Indications are low back pain, SI joint pain, and somatic dysfunction of the lumbar spine or pelvis.

Findings show lumbar segmental dysfunction, sacral rotation about a vertical axis, and a positive pelvic rock test on the involved side.

With the patient supine on the table, the provider stands opposite to the side with the dysfunction (Fig. 11.15). The patient is asked to interlock their fingers behind their neck. The provider then will place the cephalad hand through the patient's opposite arm and rest the dorsum of their hand on the patient's sternum. (This technique is best for males; with female patients, place the cephalad hand through the patient's opposite arm and grasp the inferior angle of the scapula.) Place the heel of the caudal hand on the opposite ASIS to stabilize the pelvis. Have the patient take a deep breath. As the patient exhales the provider stands, inducing a stretch of the entire spine from the thoracic region to the SI joint. Popping sounds are commonly heard but should cause no alarm.

11.8.6 Stretching Techniques

Stretching of the hip and pelvis to maintain the motion that is restored via manipulation is imperative for long-term success. Below are the stretches that this author routinely teaches to the patients seen in his clinic. Patients must understand that stretching must become a part of their daily activities. These stretches are to be done in sets of three for 15 s each [5, 6, 8, 23].



Fig. 11.15 Supine direct articulatory technique

11.8.6.1 Hamstrings (Semitendinosus, Biceps Femoris, Semimembranosus)

The patient stands with feet together. Keeping both knees fully extended, the patient slowly bends forward at the waist trying to place the palms on the ground. Hold this stretch for 15 s. and repeat three times. Then the patient crosses the right foot over the left foot and repeats, then the left foot over the right foot and repeats (Fig. 11.16).

11.8.6.2 Iliopsoas

The patient kneels on the right knee with the left foot forward on the floor, turning the right foot out to turn the right hip in and leaning forward from the waist while keeping the back straight. The stretch should be felt in the front of the right hip (Fig. 11.17).



Fig. 11.16 (a) Hamstring stretch (straight leg hang). (b) Hamstring stretch (left over right). (c) Hamstring stretch (right over left)

11.8.6.3 Quadriceps Femoris Stretch

The patient stands with the left hand supported on a stationary object to maintain balance. The right hand holds the right ankle. The patient pulls the right leg toward the buttocks keeping both knees together. The stretch is felt in the front of the right thigh. Repeat for the left leg (Fig. 11.18).

Fig. 11.17 Iliopsoas stretch





Fig. 11.18 Quadriceps stretch

Fig. 11.19 Iliotibial band stretch



11.8.6.4 Iliotibial Band Stretch

The patient stands perpendicular to and 2-3 feet from a door or wall with the right hand on the wall for support and the right leg behind the left leg. As the provider pushes down on the left hip, a stretch is felt in the lateral right thigh as the patient pushes the right hip toward the junction of the wall and the floor (Fig. 11.19).

11.8.6.5 Gluteal Stretch (Figure 4 Stretch)

The patient lies supine on the table. The ipsilateral lower extremity is flexed, abducted, and externally rotated to place the ankle on the opposite thigh just above the knee. The opposite hip is flexed with the knee bent by the patient reaching up and grasping the leg at mid-thigh. The stretch is felt with the patient pulling the leg to their chest (Fig. 11.20).

Fig. 11.20 Gluteal stretch



Fig. 11.21 Doctor-assisted resisted piriformis stretch



11.8.6.6 Piriformis Stretch

This stretch is similar to the gluteal stretch, but instead of placing the ankle just above the knee, when the legs are crossed, place the knee on the opposite knee, and then pull the leg toward the chest.

11.8.6.7 Doctor-Assisted Resisted Piriformis Stretch

The patient lies supine on the table. The involved lower extremity is flexed and crossed over the contralateral extremity. The involved side is internally rotated with the doctor resisting the patient's external rotation by standing on the ipsilateral side with his hand resisting external rotation. This stretch should be held for 20 s. and is usually done 2–3 times per leg (Fig. 11.21).

Fig. 11.22 Groin stretch



11.8.6.8 Groin Stretch

With the patient sitting up with feet together and knees bent, separate the knees to feel the stretch in the groin and inner thighs (Fig. 11.22).

11.9 Summary

The patient was found to have iliotibial band syndrome, as her pain was exacerbated shortly into her run, then would dissipate shortly after stopping. She had a positive Ober's test and tenderness over the greater trochanter on the right; she also has a right on right sacral torsion. X-rays of the hip and pelvis are negative for evidence of fracture, arthritis, and other bony abnormalities. She was treated with manipulation to include the ones listed above and did very well. She was given a stretching program and to follow up in 3 weeks. Upon returning in 3 weeks she was able to run without difficulty and was able to complete her goal; running the Portland Marathon.

These are but a few of the large variety of manual medicine techniques that have been described in both the chiropractic and osteopathic literature. Manual medicine or manipulative medicine or adjustments are all plays on a tool that is found in several physicians' bags. It is but another method of evaluating and treating the athlete not only in one's clinic but also on the field, court, or track, or anywhere else that athletes find themselves in competition. Manual medicine techniques have been found to be effective in the treatment of a variety of musculoskeletal as well as visceral ailments. So regardless of one's training, be it allopathic, osteopathic, or chiropractic, the sports minded provider who practices manual medicine has the ability to lay on hands, help restore motion to an area of the body, improve alignment for optimal performance, and above all do no harm.

References

- 1. Tettambel MA. An osteopathic approach to treating women with chronic pelvic pain. J Am Osteopath Assoc. 2005;105:S20–2.
- 2. Pedowitz RN. Use of osteopathic manipulative treatment for iliotibial band friction syndrome. J Am Osteopath Assoc. 2005;105:563–7.
- 3. Patriquin DA. Pain in the lateral hip, inguinal, and anterior thigh regions; differential diagnosis. J Am Osteopath Assoc. 1972;71:729.
- Anderson K, Strickland SM, Warren R. Current concepts: hip and groin injuries in athletes. Am J Sports Med. 2001;29:521–33.
- 5. Adkins SB, Figler RA. Hip pain in athletes. Am Fam Physician. 2000;61:2109–18.
- 6. Morelli V, Smith V. Groin injuries in athletes. Am Fam Physician. 2001;64:1405–13.
- 7. O'Kane JW. Anterior hip pain. Am Fam Physician. 1999;60(6):1687-96.
- Retzlaff EW, Berry AH, Haight AS, et al. The piriformis muscle syndrome. J Am Osteopath Assoc. 1974;73:799–807.
- 9. Ward RC, editor. Foundations for osteopathic medicine. Baltimore: Williams & Wilkins; 1997.
- Beal MC. The sacroiliac problem: review of anatomy, mechanics, and diagnosis. J Am Osteopath Assoc. 1982;81:667.
- Torry MR, Schenker ML, Martin HD, et al. Neuromuscular hip biomechanics and pathology in the athlete. Clin Sports Med. 2006;25(2):177–97.
- 12. Braly BA, Beall DP, Martin HD. Clinical examination of the athletic hip. Clin Sports Med. 2006;25(2):199–210.
- 13. TePoorten BA. The piriformis muscle. J Am Osteopath Assoc. 1969;69:150.
- 14. Kuchera WA, Kuchera ML. Osteopathic principles in practice. Kirksville: KCOM Press; 1994.
- Greenman PE, editor. Principles of manual medicine. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2003.
- Fuller DB. Osteopathic medical component missed in treating anterior hip pain. J Am Osteopath Assoc. 1997;97:514.
- 17. Gardner S, Mosby JS. Chiropractic secrets. Philadelphia: Hanley & Belfus; 2000: 231.
- Shekelle PG, Adams AH, Chassin MR, et al. The appropriateness of spinal manipulation for low back pain. Monograph no. R-4025/2-CCR/FCER. Santa Monica: RAND/UCLA; 1991.
- 19. Rumney IC. Techniques for determining, and ultimately modifying, areas of restricted motion in the lumbar spine and pelvis. J Am Osteopath Assoc. 1971;70:1203.
- 20. Kimberly PE. Outline of osteopathic manipulative procedures; the Kimberly manual. Millenniumth ed. Marceline: Walsworth Publishing; 2000.
- 21. DiGiovanna EL, Schiowitz S, editors. An osteopathic approach to diagnosis and treatment. Philadelphia: JB Lippincott; 1991.
- 22. Howard WH. Easy OMT. Ashville: C-4 Publishing; 1998.
- Karageanes SJ, editor. Principles of manual sports medicine. Philadelphia: Lippincott Williams & Wilkins; 2005.

Chapter 12 Taping and Bracing for Pelvic and Hip Injuries

Alfred Castillo, Lance Ringhausen, and Peter H. Seidenberg

Clinical Pearls

- There is little research available regarding the use of athletic taping and bracing for the hip and pelvis.
- The disadvantages of using athletic tape are the expense, the difficulty of application, and possible skin irritation.
- Tape's effectiveness at decreasing joint motion lessens after 30 min.
- Further research is needed to determine if compression shorts are able to prevent injury or enhance athletic performance.

12.1 Case Presentation

Taping has been utilized since ancient Roman times and was first introduced to organized sports in the 1890s in college athletics. Although the ankle is the most common joint taped and the most often studied, athletic taping techniques are used for nearly all body parts, including the shoulder, elbow, wrist, hand, fingers, knee, foot, and hip.

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To date, research has been focused mainly on taping for the ankle, wrist, hand, and knee joints. Unfortunately, there are very little data regarding the hip and pelvis to guide clinicians in its potential use in these areas.

12.2 Indications for Taping

Athletic taping techniques can be utilized after acute injury, for rehabilitation from injury, for injury prophylaxis, or for functional purposes. In general, taping is performed with the involved joint in a neutral position to limit the range of motion. Compressive taping techniques are used for an acute injury to limit joint motion and control edema. Tape may be applied during return to activity to protect the injured joint from further damage.

12.3 Efficacy of Taping

Originally, it was postulated that taping techniques could hold bones rigidly in approximation and, consequently, prevent injury to the associated ligament. However, research has proven that the effect of taping is mostly proprioceptive [1–7]. Radiographic studies showed that the affected bones do not maintain the same relationship in a weight-bearing dynamic position as they do in the non-weight-bearing static position after taping [8], disproving this theory. Contrary to most athletes' beliefs, tape's effectiveness at restricting joint motion is decreased within 30 min of the onset of physical activity [9, 10]. These undesirable changes were attributed to perspiration and loosening or stretching of the tape during weight-bearing activities.

12.4 Drawbacks

Although widely utilized, athletic taping has several obvious drawbacks [6]. For example, a trained individual is required to correctly apply the tape to the affected body part. Also, since the tape cannot be recycled after application, costs may be high. Frequent reapplication of tape can lead to skin irritation.

12.5 Types of Tape

Adhesive athletic tape is the most commonly used tape in sports. The tape width can vary from $\frac{1}{2}$ in. to 2 in. This tape is often used in taping ankles, it can also be used to add support to a hip wrap or secure closures once the wrap is complete. Adhesive

stretch tape can be used as a secondary support to a hip wrap or bracing effort. Tape width of 2 in. or more is the most effective due to the larger muscles, which are to be supported. Non-adhesive stretch tape is similar to the regular stretch tape only in that it has no adherent or sticky side, and adheres to itself. This tape can be applied directly to the skin and serve as a base layer for most tape jobs. Heavy adhesive stretch tape is a more robust version of regular stretch tape. It is firmer and has a stronger tension point than regular stretch tape. This style of tape comes in various sizes (1–3 in. widths) and is commonly used to provide ligamentous support of the elbow, knee, and ankle. The stronger tension strength also makes it useful to restrict extension at the elbow and knee.

There are many different brands of athletic tape. The most commonly used is Zonus. Other commonly used tape types include Elasticon, Elastoplast, Blister Tape, Leukotape, Cover Roll, and Kinesiotex (Kinesiotape). Except Kinesiotex, the tape is generally applied over a foam-like material called underwrap, or prewrap. The purpose of the prewrap is to protect the skin from the adhesive backing of the tape. In order to provide better adherence, a tacky substance (e.g., Tuf-Skin spray adherent) is often applied to the skin prior to the application of the underwrap.

Kinesiotape was invented by Kenzo Kase, D.C. in Japan. Kase attended chiropractic school in the USA, but practiced in Japan. He invented Kinesiotape in an effort to decrease pain and edema, assist in muscle function, and improve joint function. Kinesiotape was first utilized on Japanese Olympic volleyball players in the 1980s and now is commonly utilized in all professional sports in Japan. It is also very commonly utilized in the nonathletic population in that country. It was first introduced in the USA in 1995.

The theory behind the mechanism of action of Kinesiotape is based on augmenting the body's natural healing processes. The tape is flexible and has physical properties that affect the neurologic, proprioceptive, musculoskeletal, and lymphatic systems. Unlike traditional taping methods that employ compression to control edema, Kinesiotape is applied to the surface of the skin to facilitate lymphatic drainage. It is purported that the elasticity of the tape allows the skin to be gently lifted directly under the tape to facilitate extracellular fluid drainage. Kase and Hashimoto utilized Doppler ultrasound to measure fluid flow in patients with and without circulatory insufficiency. They found that there is an increase in flow after application of Kinesiotape and were unable to demonstrate any adverse effects from its use [11, 12].

Kinesiotape has also been utilized successfully in the treatment of muscle injuries. Its mechanism of action is purported to be much different from that of traditional athletic taping techniques. The tape has an elastic component that will attempt to recoil, once applied. Therefore, application of tape from the origin of the muscle to its distal insertion helps facilitate muscle function. When placed in the opposite direction, Kinesiotape has a tendency to inhibit or decrease muscle activity. This distinction is extremely important clinically, when utilizing Kinesiotape for rehabilitation of any musculoskeletal injury. Studies have been conducted measuring electromyographic muscle activity with the tape applied in each direction confirming that there is more electromyographic activity when the tape is applied in the facilitatory role and less electromyographic activity when it is applied for muscle inhibition [11, 12]. Kinesiotape is manufactured to mimic the thickness, weight, and elasticity of human skin. Kinesiotape has an elastic component that allows it to stretch 30–40% beyond its original length [13]. The adhesive is heat activated and allows the tape to function for several days without reapplication. It is also water resistant, such that it can be worn in a pool, shower, or bath without coming off. Kinesiotape is designed to "breathe" and also functions to remove sweat from the surface of the skin. It causes fewer skin reactions than conventional tape. The tape contains no latex and is hypoallergenic. It is available in different colors including neutral, blue, and pink. As per Eastern medicine theory, pink absorbs more light and generates a warming effect while blue reflects light and produces a cooling effect [6]. Unlike conventional tape, Kinesiotape can be cut lengthwise without losing any of its unique properties.

12.6 Bracing

There are different manners in which to support the hip and pelvis. One way would be to wrap or brace the area in a specific pattern to best restrict and or aid movement. Elastic support bandage is a commonly used means of support. These wraps are made of a combination cotton and stretch fabric material. They can range from being one inch in width to as much as 6 in. or more. The length can also vary and be as long as 12 feet of unstretched fabric. Pre-fabricated neoprene hip and groin wraps are also available are similar to the elastic support bandage. These wraps are not quite as long and have a stronger tension point. The wraps are used in a specific wrap technique and usually can be secured onto itself at some Velcro point along the wrap.

Bracing has several advantages over taping. Its use does not require any special expertise and there is no repeated expense as with each reapplication of tape (Fig. 12.1). The effectiveness of bracing compared to taping has been researched by several authors and no significant differences were found in the injured joint. Yet bracing and taping both increased the effectiveness of proprioceptive feedback and when studied were found to have a reduction in muscle action while landing [2, 7, 10, 14].

12.7 Taping Techniques

Numerous taping techniques have been purported to be efficacious in the treatment and prevention of injury about the hips and pelvis (Fig. 12.2). Some examples of injuries/conditions in which taping/bracing have been utilized include greater trochanteric bursitis, iliac crest contusion (hip pointer), and osteoarthritis of the hip.

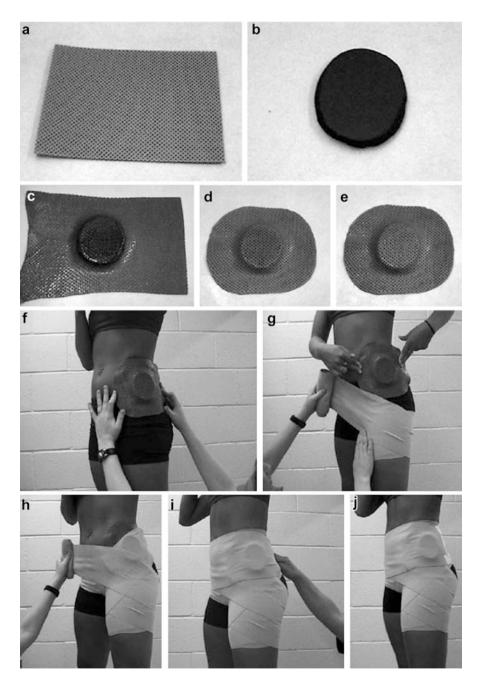


Fig. 12.1 Molded thermoplastic protection of the hip. (a) Select an appropriate thermoplastic material. (b) Half-inch high density foam. (c) Place the heated thermoplastic material over the foam. (d) Cut the thermoplastic material to appropriate size. (e) Remove the foam from the thermoplastic material. (f) Place the thermoplastic material over the involved side. (g-i) Using a hip spica technique, wrap the mold to the patient. (j) Secure the elastic bandage with white athletic tape

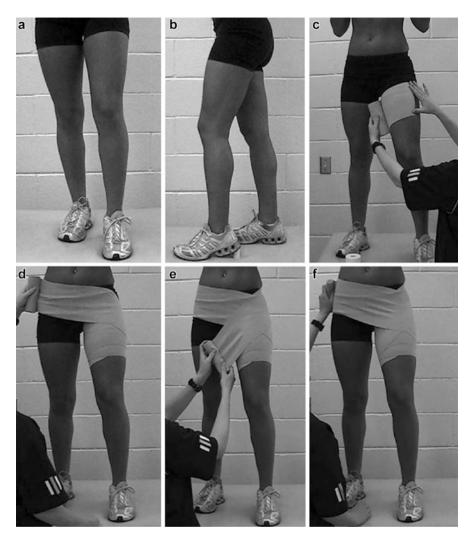


Fig. 12.2 Hip spica technique. (a) Place a 1-2 in. object under the heel to slightly flex the hip. (b) Lateral view of 12.1a. (c) Using a 6-in., double-length elastic bandage, wrap lateral to medial on the involved side at a slight downward angle. (d) Wrap the bandage around the involved side and cross the anterior hip. (e) Wrap the bandage around the back of the patient and around the anterior aspect of the involved side. (f) Continue across the anterior aspect of the hip

12.7.1 Compression Shorts

One common brace utilized in sports for injury prevention and rehabilitation about the hip and pelvis is compression shorts. It has become increasingly popular during the last decade despite little research to support its efficacy. Light compression materials (i.e., Spandex) have not been found to hinder performance [1, 15]. While no increase in single maximal jump power was noted, compression shorts did help maintain higher jumping power during repeated vertical jumping exercise [15]. Performance and proprioception at the hip with the use of elasticized compression shorts that offer considerably more compression and resistance to movement than conventional compression shorts have been studied. Their use did not limit performance on any measures except active range of motion during hip flexion [16, 17]. There is scientific evidence that movement of skin overlying activated muscle triggers cutaneous receptors to send proprioceptive information to the brain; [2, 9] however, there was no increased proprioception at the hip noted with the use of the compression shorts [17]. Subjective data revealed over 93% of subjects felt the shorts were supportive, although its proper fit was an issue [17]. Doan et al. [16] found countermovement vertical jump height and 60-m. sprint times to be enhanced with the use of neoprene and rubber compression shorts which provided compression similar to those used in the previous study. Further research is needed to delineate if compression shorts offer any enhancement to athletic performance or efficacy in prevention of injury.

12.8 Case Study 1

A 20-year-old female collegiate goalie presents herself to the athletic training clinic for the evaluation of right lateral hip pain. The pain started a day earlier when she dove and landed on the lateral aspect of her right hip in order to block a shot. She complains of pain in that area exacerbated by hip abduction.

On examination, there is edema overlying the greater trochanter but no ecchymosis is present. She has full passive range of motion of the hip and resisted abduction increases pain in the area of the greater trochanter. Log roll is negative. Ober test is negative but produces pain in the area of the greater trochanter. There is no pelvic obliquity noted. She is neurovascularly intact.

12.9 Case 1 Conclusion

The patient was diagnosed with traumatic greater trochanteric bursitis. Ice and analgesics were used for pain control. The athlete was advised to wear compression shorts and hip spica taping was performed during practice and competition. This treatment provided her with more confidence in hip movement but did not decrease her pain. Her symptoms completely resolved after 2 weeks.

12.10 Case Study 2

The athlete involved in this case study is a 6 feet 2 in., 260 lb. male Collegiate American Football player. The athlete is a defensive lineman. The injury sustained occurred during an NCAA sanctioned football game after the play was blown dead

and determined by the officials to be over. The athlete was slowly jogging in pursuit to the point of tackle, where the opposing running back had been stopped. The whistle had blown and the athlete did not anticipate the contact since the play was over. The athlete was clipped in the back of the right shoulder by an offensive lineman. His left foot was planted and right foot was elevated in mid-stride. The force received from his right shoulder caused his right leg to whip across the front side of his lower body, resulting in his right forefoot making hard contact with the ground. The whipping motion created a stretching force to the gluteal region. It also created an over contraction force to the hip flexors and adductor muscles. The athlete was able to walk over to the sideline on his power, but was badly limping. The initial evaluation by the team physician was performed on the sideline. Due to the athlete's level of discomfort, it was thought that he might have a hip fracture. X-rays performed at the stadium were negative, but the athlete was unable to return to the game and was placed on crutches to aid ambulation. The next day an MRI was ordered. The results showed no bony involvement. It did show injury to the gluteus medius, adductor brevis, and rectus femorus tendon origin.

Conservative therapy was conducted over the next few days including cryotherapy, light ROM work, light strengthening, along with interferential EMS to aid with general soreness. A stretch bandage was used initially to aid in muscular support and help minimize swelling to the hip region. Kinesiotape or K-tape was applied to aid movement on the upper thigh to assist with hip flexion, leg adduction, and abduction.

Thermal therapy was introduced 72 h post-injury to loosen the soft tissue and assist with increasing range of motion. Passive range of motion was increased and was performed more aggressively through isolated stretch therapy to aid in overall functional improvement. Aquatic therapy in a Hydroworx therapy pool was performed and allowed the athlete to perform more movements that would be more challenging on dry land. The buoyancy of the water lessened the affects of gravity over the injured area through all planes of movement.

On day five, the athlete was allowed to do modified position skill movements, along with some light jogging. A stretch wrap was applied to help and minimize hip flexion, hip abduction, and leg abduction. The wrap was restricting, but tolerable to the athlete. The wrap was reinforced with 2 in. adhesive stretch tape and secured with adhesive non-stretch 1.5 athletic tape. The athlete indicated that the support did make his injury feel better.

No practice was conducted on day 6. Continued ROM work was conducted and K-tape was again applied to aid movement on the upper thigh to assist with hip flexion, leg adduction, and abduction. The athlete's participation would be a game time decision.

On game day, the athlete was allowed to dress and warm up with the team. A stretch wrap was applied to help and minimize hip flexion, hip abduction, and leg abduction. The wrap was restricting, but tolerable to the athlete. The wrap was reinforced with 2 in. adhesive stretch tape and secured with adhesive non-stretch 1.5 athletic tape. The athlete was able to move through drills with minimal discomfort. The team physician allowed the athlete to play. His play reps were limited to 20 for this game. The athlete felt he was moving at 80% capacity, but did not further aggrevate the injury during the game.

12.11 Case Study 2 Conclusion

The athlete in this case was diagnosed with having a grade one muscle strain to the gluteus medius, adductor brevis, and rectus femorus tendon origin. The athlete in this case study benefited from the use of hip bracing and K-tape application. The injury fully resolved itself 14 days later following continued therapy.

References

- Kraemer WJ, Bush JA, Triplett-McBride NT, et al. Compression garments: influence on muscle fatigue. J Strength Cond Res. 1998;12:211–5.
- Simoneau GG, Denger RM, Cramper CA, Kittleson KH. Changes in ankle joint proprioception resulting from strips of athletic tape applied over the skin. J Athl Train. 1997;32:141–7.
- Carmines DV, Nunley JA, McElhaney JH. Effects of ankle taping on the motion and loading pattern of the foot for walking subjects. J Orthop Res. 1988;6:223–9.
- 4. Cordova ML, Ingersoll CD, LeBlanc MJ. Influence of ankle support on joint range of motion before and after exercise: a meta-analysis. J Orthop Sports Phys Ther. 2000;30:170–82.
- 5. Refshauge KM, Kilbreath SL, Raymond J. The effect of recurrent ankle inversion sprain and taping on prorioception at the ankle. Med Sci Sports Exerc. 2000;32:10–5.
- Kase K. Illustrated kinesiotaping. 3rd ed. Albuquerque: Universal Printing and Publishing; 1994.
- 7. Hopper DM, McNair P, Elliott BC. Landing in netball: effects of taping and bracing the ankle. Br J Sports Med. 1999;33:109–13.
- Cameron MH. Patellar taping in the treatment of patellofemoral pain. A prospective randomized study. Am J Sports Med. 1997;25:417.
- 9. Moberg E. The role of cutaneous afferents in position sense, kinaesthesia, and motor function of the hand. Brain. 1983;106:1–19.
- MacKean LC, Bell G, Burnham RS. Prophylactic ankle bracing versus taping: effects on functional performance in female basketball players. J Orthop Sports Phys Ther. 1995;22:77–81.
- 11. Association K-t. Kinesio-taping perfect manual. Albuquerque: Universal Printing and Publishing; 1998.
- Karlsson J, Sward L, Adreason GO. The affect of taping on ankle stability. Practical implications. Sports Med. 1993;16:210–5.
- 13. Burke WS, Bailey C. Believe the hype. Physical therapy products. July/August 2002.
- McGaw ST, Cerullo JF. Prophylactic ankle stabilizers affect ankle joint kinematics during drop landings. Med Sci Sports Exerc. 1999;31:702–7.
- Kraemer WJ, Bush JA, Bauer JA, et al. Influence of compression garments on vertical jump performance in NCAA Division I volleyball players. J Strength Cond Res. 1996;10:180–3.
- Doan BK, Kwon Y-H, Newton RU, et al. Evaluation of a lower-body compression garment. J Sports Sci. 2003;21:601–10.
- Bernhardt T, Anderson GS. Influence of moderate prophylactic compression on sports performance. J Strength Cond Res. 2005;19(2):292–7.

Chapter 13 Nonsurgical Interventions

Michael D. Osborne, Tariq M. Awan, and Mark Friedrich B. Hurdle

Clinical Pearls

- Given their relative safety, the ease of use in trained hands, and cost-effectiveness, injection therapies can be beneficial when more conservative treatment measures have failed.
- Injection therapies containing local anesthetics may help confirm a diagnosis, particularly when performed with the precision of image guidance.
- Informed consent should be obtained for all procedures and should include a discussion regarding the indications, anticipated outcome, potential risks and complications, possible side effects, and alternatives to the procedure.
- It is incumbent on the proceduralist to have a thorough understanding of the relevant anatomy, procedural technique, potential risks, procedural contraindications, and to be prepared to manage any unforeseen complications prior to attempting injection therapies.
- Injection therapies are very rarely indicated as first-line treatment.

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13.1 Case Presentation

A 24-year-old male recreational basketball player presented with right hip and groin pain of 2 months duration. The patient was playing basketball and landed awkwardly on his right leg. He subsequently developed progressive pain with activity, sharp in quality, and predominantly over the anterior hip with radiation to the groin. He denied any mechanical catching or locking. He was unable to run or play basketball without discomfort. Pain could be relieved by laying supine with his hip and knee flexed. Mild relief was obtained with nonsteroidal anti-inflammatory drugs (NSAIDs) and physical therapy.

13.1.1 Physical Examination

- Normal appearance, gait, and station.
- Normal strength, reflexes, and full hip range of motion.
- Tenderness over iliopsoas at the pelvic brim and hip adductors.
- FABER test negative; Stinchfield test positive.
- Passive hip extension, internal rotation, and adduction caused discomfort but did not reproduce the typical pain.
- Modified Thomas test was positive for anterior hip pain.

13.1.2 Differential Diagnosis

- Hip adductor strain
- Iliopsoas bursitis/tendonitis
- Hip labral tear
- Snapping hip syndrome
- Osteitis pubis
- Sports hernia/athletic pubalgia
- Femoral/pelvic stress fracture

13.1.3 Imaging

- Plain radiographs were unremarkable.
- Magnetic resonance imaging (MRI) arthrogram demonstrated a small linear extension of contrast beneath the superior acetabular labrum compatible with labral detachment (Fig. 13.1).



Fig. 13.1 MRI arthrogram of the hip demonstrating a small superior labral tear

13.2 Introduction

The aim of this chapter is to provide an overview of some of the more common injection therapies for sports-related disorders of the hip and pelvis. Additionally, we provide technical instruction that will allow the interested clinician an opportunity to learn basic office-based procedures.

The principal form of injection therapies involves the use of a combined corticosteroid and anesthetic injection into or around a symptomatic musculoskeletal structure. The use of cortisone was first reported in 1949 by a team of physicians at the Mayo Clinic [1] and resulted in a Nobel Prize in 1950. Since 1950, the injection of corticosteroid has been applied to a plethora of musculoskeletal conditions with varied efficacy.

Although corticosteroid injections are an exceedingly common form of treatment applied today, their role in symptomatic management of sports injuries remains a topic of some controversy. Prospective, randomized, controlled studies support their use in disorders such as osteoarthritis of the hip and knee [2, 3]. However, one should not automatically conclude that these results can be generalized to all musculoskeletal conditions.

In addition to corticosteroids and local anesthetics, proliferative therapy (prolotherapy) and viscosupplementation (injection of hyaluronic acid compounds) have been investigated as treatments for injured athletes. Preliminary studies have shown effectiveness for select musculoskeletal conditions and provide promise for further randomized, clinical controlled studies [4–7]. Lastly, regenerative medicine approaches with platelet rich plasma and mesenchymal stem cell injections are emerging therapies under study for their restorative and healing effects.

13.3 Rationale for Use

Given their relative safety, ease of use in trained hands, and cost-effectiveness, injection therapies can be a very useful modality when more conservative treatment measures have failed. Prior to performing any type of injection, the clinician must have a thorough understanding of the regional anatomy as well as procedural contraindications and precautions. The proceduralist must also have a thorough understanding of the various injection constituents and their potential side effects.

13.4 Diagnostic Injections

Injection therapies containing local anesthetics can be helpful in establishing a diagnosis when performed with precision. The rationale is that one can identify a symptomatic nociceptive structure by infiltrating it with a local anesthetic. Examples include intra-articular injections (hip and sacroiliac joint), soft tissue injections (bursa and peritendon infiltration), and peripheral nerve blocks (such as lateral femoral cutaneous nerve block).

To reduce the incidence of false positive responses one must use a small enough volume of injectate, such that it will only anesthetize the targeted nociceptive structure. Otherwise, the anesthetic may diffuse to nearby tissues and cause pain relief by inadvertent effect on structures not targeted in the procedure.

Image guidance can be used to significantly increase the accuracy of reaching the desired target tissue and thereby enhance diagnostic accuracy. Image guidance is highly recommended when performing diagnostic injections. Increasingly, ultrasound is being used in office-based sports medicine practices to help guide injection therapies [8, 9]. Ultrasound can be particularly helpful when trying to localize peripheral nerves and musculature for diagnostic block [10]. One advantage of ultrasound is that during the injection, additional relevant pathology such as hip joint effusion, bursitis, or tendinopathy may be visualized which may impact what structures are ultimately targeted during the procedure. One potential limitation, however, is the lack of sound wave penetration in large patients, which may limit visualization of deeper tissues. Additionally, it takes considerable training and experience with diagnostic ultrasound to use this method of image guidance effectively.

Fluoroscopy is the standard method of image guidance used in pain clinics and interventional radiology suites. Accurate placement of the procedural needle can be directly visualized under fluoroscopic guidance. With the injection of a radiopaque contrast media, one can confirm the injection has reached the target tissue. Furthermore, the use of contrast can identify vascular uptake when injections are being performed near blood vessels such as during nerve blocks. This reduces false negative responses (through inadvertent vascular injection) and guards against systemic toxicity when performing large-volume field blocks.

Diagnostic blocks can also be performed with great accuracy using computed tomography (CT) and MRI guidance [11]; however, this is rarely necessary if ultrasound or fluoroscopy is available to the experienced proceduralist.

An additional diagnostic application of injection therapies is aspiration and analysis of joint effusions. Joint fluid analysis can differentiate among various pathophysiologies such as infection, gout, pseudo gout, inflammation, and hemorrhage.

13.5 Therapeutic Injections

The purpose of therapeutic injections is principally to improve pain and allow for restoration of function. The precise effect of each procedure depends on the structure injected and the pharmacologic / biologic agent utilized.

13.6 Pharmacological Agents

13.6.1 Corticosteroids

Corticosteroid preparations are the most commonly utilized injectate because of their effects as potent inhibitors of inflammation. They modify the local inflammatory response through stabilization of lysosomal membranes, inhibition of cellular metabolism (e.g., neutrophil chemotaxis and function), inhibition of polymorphonuclear leukocyte membrane microtubular function, and establishment of decreased local synovial permeability. Corticosteroids can also increase the viscosity of synovial fluid, alter production of hyaluronic acid synthesis, and change synovial fluid leukocyte activity [12], all of which may improve symptoms secondary to degenerative, inflammatory, and overuse syndromes.

Though many different preparations are available for joint and soft tissue injections, corticosteroids differ with respect to potency, solubility, and relative duration of action. The relative potency of individual corticosteroids is compared in Table 13.1 [13].

Few studies have investigated the duration of action of corticosteroid agents in joints or soft tissues. In general, the duration of effect is inversely related to the solubility of the therapeutic agent. The less soluble agents remain in the joint or soft tissue longer and provide more prolonged effect. Nevertheless, shorter acting solutions are less irritating to the joint space and less likely to produce a post-injection pain flare. Agents with low solubility should be used primarily for intra-articular therapy and should be avoided in soft tissues due to the increased risk of soft tissue atrophy from prolonged local corticosteroid action.

	Relative anti-inflammatory	Equipotent doses
Corticosteroid	potencies	(mg)
Cortisone	0.8	25
Hydrocortisone	1.0	20
Prednisone	4	5
Methylprednisolone acetate	5	4
Dexamethasone sodium phosphate	25	0.6
Betamethasone	25	0.6

Table 13.1 Relative potency of corticosteroid preparations

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13.6.2 Hyaluronic Acid

Intra-articular injection of hyaluronic acid is used to treat the pain associated with osteoarthritis of the knee with several randomized controlled studies showing reasonable efficacy [14]. The rationale for the use of hyalurons therapeutically is based on observations that hyaluronic acid is an important component of synovial fluid that acts as a cushion and lubricant for the joint. It serves as a major component of the extracellular matrix of the cartilage, helping to enhance the ability of cartilage to resist shear forces and maintain a resiliency to compression [15]. A systematic review published in 2006 suggests that injections with hyaluronic acid may also benefit people with osteoarthritis of the hip [4]. Intra-articular injection of hyaluronic acid into the hip joint appears to be safe and well tolerated [16], however only a small number of randomized clinical trials in humans have been published [17–19].

13.6.3 Anesthetics

Local anesthetics can assist in identification of a symptomatic nociceptive structure by producing a rapid reduction in pain following injection/infiltration. Typically however, when performing intra-articular and soft tissue injections an anesthetic is mixed with a corticosteroid. Not only does this provide temporary analgesia and confirm delivery of the medication to the symptomatic structure, it dilutes the crystalline suspension of the corticosteroid and thus provides better diffusion of medication throughout the injected region. An allergic reaction to the amide local anesthetics such as lidocaine and bupivacaine is very rare.

13.6.3.1 Lidocaine

For most procedures 1 % lidocaine is used due to its rapid onset of action. However, because of its short half-life, lidocaine's duration of therapeutic effect is short (1–2 h) [20]. At high concentrations lidocaine (5 %) is neurotoxic to local peripheral nerves

and thus it can be used as a form of peripheral neurolysis [21]. Systemic toxicity would be rare in a standard sports medicine practice. The toxic effects of local anesthetics are highly dependent on the route of injection and the rapidity of absorption or uptake into the local vasculature [20]. Intra-articular and most soft tissues are not heavily vascularized, thus reducing the chance of central toxicity.

13.6.3.2 Bupivacaine

When longer acting local analgesia is desired, the use of an agent such as bupivacaine is preferable because of its duration of effect of 3-6 h [22]. However, bupivacaine has a longer time to the onset than lidocaine (2–10 min) and thus will not help attenuate the pain of the injection procedure itself. Bupivacaine is typically used in a strength of 0.25% for musculoskeletal injections.

13.6.4 Proliferants

Prolotherapy is the injection of a substance that activates the inflammatory cascade and thus induces fibroblast proliferation. One objective of proliferative therapy is to strengthen incompetent ligaments that exhibit laxity [23]. For example, a gymnast who has low back pain from a hypermobile sacroiliac joint, a series of prolotherapy treatments over the dorsal sacral ligaments may strengthen them and thereby reduce motion and pain. A second application of prolotherapy is to stimulate the repair of tendons that have undergone chronic degeneration (tendinosis), once again by inciting an inflammatory response which then reactivates the healing process [23]. A number of substances can be used for prolotherapy such as compounds containing phenol, glucose, and glycerine. Another commonly used substance is dextrose (10–12% concentration) which is potentially less neurotoxic than phenol preparations. However, part of the pain-relieving effect of compounds containing dilute phenol might also be due to its toxic action on nociceptors [24].

13.7 Regenerative Medicine

13.7.1 Platelet Rich Plasma

The application of biologic treatments for musculoskeletal disorders is growing significantly. Platelet rich plasma (PRP) is an autologous biologic treatment utilizing the patients' own blood plasma. The process involves injection of platelet derived growth factors that are obtained via density gradient centrifugation to remove plasma and red blood cells and to increase platelet concentration [25]. Platelets are well-known mediators of the coagulation cascade, however they also have hundreds of bioactive cytokines and growth factors that act via autocrine and paracrine mechanisms to enhance cell interaction and healing [26]. The rationale for the use of PRP is to stimulate tissue regeneration and the natural healing cascade by releasing these growth factors directly into the area of tissue damage [27].

It is important to mention that not all PRP preparations are the same and formulations vary in the concentration of platelets and leukocytes. This variability has made it difficult to compare PRP treatments between investigational studies. A classification system has been proposed that incorporates white blood cell concentration (increased vs. not increased over baseline), platelet concentration (greater or less than 5 times baseline), and platelet activation status [28]. Such classification allows researchers to standardize formulations of PRP for various treatment populations.

More substantiated clinical data is needed to improve our understanding of the best use of this treatment modality. Further research efforts are aimed at determining appropriate indications, type of PRP formulation, and timing/number of injections. Currently accepted indications for PRP include the treatment of chronic tendinopathies, muscle strains, and ligament injuries that have been resistant to standard medical and rehabilitative therapies [29–33]. More research is also needed to determine if there is a role for PRP to treat acute sports injuries in order to speed recovery and decrease the time missed from athletic competition [34, 35].

PRP is also being investigated as a tool for the management of knee osteoarthritis. An increasing number of clinical trials show increased function and decreased pain in the treatment of the arthritic knee [36–39]. With regard to osteoarthritis treatment, PRP is being looked at for its potential to increase anabolic effect on chondrocytes and a decrease catabolic effect in the inflammatory environment [40]. A meta-analysis on the use of PRP for the treatment of knee arthritis demonstrated better pain relief and functional improvement when compared to hyaluronic acid and placebo [41]. While the use of PRP in treatment of knee osteoarthritis seems promising, randomized controlled studies need to be initiated to explore the efficacy in other joints such as the hip.

13.7.2 Mesenchymal Stem Cells

Mesenchymal stem cells (MSCs) are another therapy which has received increased attention for not only musculoskeletal care but also in many areas of regenerative medicine. MSCs are being considered as a potential treatment for osteoarthritis because of their healing potential and anti-inflammatory effects [42]. Animal studies with MSCs used in the treatment of osteoarthritis show potential to slow down the progression of cartilage degeneration [43–46]. MSCs can modulate the inflammatory response, inhibit apoptosis, stimulate cell repair, and improve blood flow to joints [47]. By secreting paracrine factors, including cytokines and growth factors, MSCs can target injured tissues leading to a trophic effect that can initiate endogenous tissue repair [48].

MSCs are easily found in various tissue sources including bone marrow, adipose cells, periosteum, umbilical cord tissue, and synovial tissue [49]. MSCs have the capacity to differentiate into a variety of cell types. Bone marrow derived MSCs can differentiate into cells of chondrogenic lineage [46].

FDA guidelines explicitly dictate the extent to which stem cell-based therapies may be administered in clinical practice in the USA. FDA Tissue Regulation, 21 CFR Part 1271, outlines the guidelines for cell-based therapies that may be used by clinicians. These guidelines require that cells "be minimally manipulated," "used within a short period of time," and "be used only at the point of care." Minimal manipulation of cells at present does not allow for extended ex-vivo culturing of cells and treatment with growth factors [49, 50].

MSCs are in their infancy with respect to their role in modulating pain and the potential for treating articular cartilage degeneration. Well-designed clinical studies are needed to not only determine efficacy, but also to determine variables such as optimal concentration of MSCs, best source to harvest them, and the safety of both autologous and allogenic sources [51, 52].

13.8 Safety Considerations

The procedures described in this chapter are by-and-large considered minimally invasive. However, this does not mean that they are totally without risk. Informed consent should always be obtained for any procedure irrespective of the relative risks. Discussion with the patient should include the indications, anticipated outcome, potential risks and complications, possible side effects, and alternatives to the procedure. Patients should sign documentation that informed consent was given and understood. The documentation should be kept as part of the patient's record.

13.9 Contraindications

Contraindications to injections include an active infection or allergy to the products used. Anticoagulated state/coagulopathy is a relative contraindication. The procedures described in this chapter are generally considered low risk and can be performed while a patient is on blood thinning products. However, if a patient is on warfarin an INR should be checked to exclude the possibility of a supratherapeutic level. An INR of less than 3.0 is reasonable cutoff for low risk, superficial, soft tissue injections. It is also important to assess for any underlying medical contraindications (such as uncontrolled diabetes or adrenal insufficiency) prior to performing corticosteroid injections.

13.10 Potential Complications

Intra-articular and periarticular steroid injections have been found to be safe and to have low complication rates if performed while taking adequate precautions [53, 54]. Potential complications that could result from joint and soft tissue procedures include:

post procedural pain flair, subcutaneous fat atrophy, soft tissue calcification, tendon rupture, bleeding, infection, and allergic reaction [53]. These potential complications can be minimized with proper exclusion of patients with known contraindications as well as meticulous attention to injection site preparation and procedural technique.

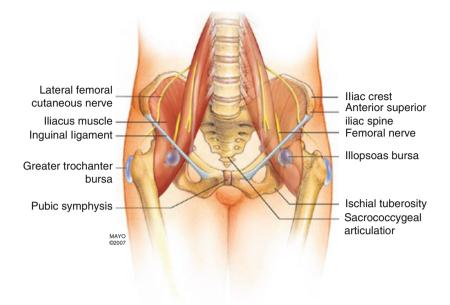
Injection site preparation is arguably the most important part of the procedure. Skin preparation can be performed with a variety of microbicides including alcohol, chlorhexidine and alcohol solutions, or povidone-iodine. One must allow the selected microbicide, time enough to kill the bacteria after application (1–2 min. is typically satisfactory, though for optimal bacteriocidal effects povidone-iodine products should be dry). Post-injection infection rates of 1:16,000 to 1–2:150,000 have been cited [12].

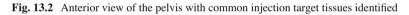
Local reactions at the injection site may include swelling, tenderness, and warmth, all of which can develop a few hours after the injection and may last up to 2 days. A post-injection steroid flare, thought to be a crystal-induced inflammatory response caused by preservatives in the injectate, may occur within the first 24–36 h after injection [55]. This reaction is self-limited and symptomatic patients are instructed to apply ice packs for temporary amelioration. Also, failure to remove residual skin preparation may cause local skin irritation.

Soft tissue (adipose) atrophy and local skin depigmentation are possible with any steroid injection into soft tissue, particularly at superficial sites and bony prominences where the subcutaneous adipose is less thick. Rarely, periarticular and soft tissue calcifications may occur, seemingly most preferentially at sites of multiple injections. The risk of tendon rupture can be reduced by taking great care to avoid intrasubstance injection of steroid into the tendon itself. The peritenon is the target tissue for treatment of tendonitis/tendinosis. To avoid direct needle injury to articular cartilage or local nerves, strict attention should be paid to anatomic landmarks and depth of the injection.

Systemic effects are uncommon but may arise, particularly following injection into highly vascularized tissue (such as a site of prior surgery) or with inadvertent direct vascular injection. The proceduralist must be vigilant to reduce the potential for systemic side effects. Patients should remain in the office for an appropriate period of time following their procedure to monitor for adverse reactions. Typically 10 min will suffice with minor procedures. If large volumes of long-acting anesthetics are used, a lengthier period of observation should be instituted. Symptoms of vascular uptake of local anesthetic include lightheadedness, tinnitus, a metallic taste in the mouth, and perioral tingling. Patients who exhibit these symptoms should not be released home and should be moved to a setting where additional monitoring can be instituted to observe for signs of central nervous system and cardiotoxicity.

Exogenous corticosteroids can have an effect on the endocrine system. Hyperglycemia can certainly occur following corticosteroid injection in patients with diabetes [23], particularly if they typically exhibit poor glycemic control or require high doses of insulin for management. All diabetics should be counseled regarding this possibility and given instructions for frequent glucose monitoring for the first several days following the injection. Other rare, but reported, complications include adrenal suppression and abnormal uterine bleeding [13, 56]. A benign facial flush can occur for 1–3 days following corticosteroid injections and is not considered an allergic reaction in the absence of other symptoms such as hives, shortness of breath, or pruritus.





13.11 Anatomy

Surface anatomy landmarks that are palpable and help guide injections into the hip and pelvic region include the ilium with its large anterior superior iliac spine (ASIS) and posterior superior iliac spine (PSIS), the greater trochanter, ischial tuberosity, and the coccyx. Palpable joints include the hip joint, sacroiliac joint, pubic symphysis, and the sacrococcygeal articulation. The sciatic notch is formed by the ilium and the lateral border of the sacrum. The proceduralist should have a detailed understanding of musculoskeletal anatomy, including muscle attachments, bursa, nerves, and blood vessels. Hip and pelvic anatomy with common injection target tissues are depicted in Fig. 13.2.

13.12 Procedures

13.12.1 Documentation

As previously discussed, the physician should review the rationale for the procedure and obtain written informed consent. In July 2004, the Joint Commission on the Accreditation of Healthcare Organizations (JCAHO) began requiring providers to follow a universal protocol for preventing wrong site, wrong procedure, and wrong person surgery. The protocol's three major elements include: (1) initial verification of the intended patient, procedure, and the site of the procedure; (2) marking the intended site with a sterile pen, where applicable; and (3) a final "time-out" immediately before beginning the procedure [57].

13.12.2 Injection Technique Fundamentals

13.12.2.1 Image Guidance

Determine whether image guidance (if available) will be necessary to appropriately perform the procedure. For injections such as the hip joint, iliopsoas bursa, sacroiliac joint, piriformis muscle and pubic symphysis, image guidance is recommended.

13.12.2.2 Positioning

Position the patient in a comfortable manner that will allow easy access to the target anatomy.

13.12.2.3 Target Selection

Superficial anatomy is palpated and a needle entry point is marked.

13.12.2.4 Sterile Preparation

The area is generously prepped with a microbiocidal agent.

13.12.2.5 Drapes

Drapes can be used if necessary to maintain a sterile field, however, are not always necessary for routine soft tissue and joint injections that have been prepped in a wide fashion around the target anatomy.

13.12.2.6 Universal Precautions

Universal precautions should always be observed.

13.12.2.7 Gloves

Sterile gloves must be worn if the physician needs to palpate the needle entry site after it has been prepped or to touch the needle. This is often the case with novice proceduralists. For the experienced proceduralist, once the needle entry point is marked and prepped, the injection can typically be performed without touching (contaminating) the needle entry point or needle. Thus non-sterile gloves may be worn.

13.12.2.8 Skin Wheal

Injections are much more tolerable for the patient if the proceduralist takes time to perform a separate skin wheal with 1 % lidocaine at the needle entry point using a 30 gauge needle, and infiltrates a little lidocaine along the initial needle trajectory. When performing a skin wheal, infiltrate very slowly; this will minimize the initial burning pain associated with subcutaneous lidocaine. To further reduce the sting of local anesthetics, 1 ml of sodium bicarbonate (8.4%) can be combined with 9 ml of 1% lidocaine. Buffering lidocaine in this manner will also speed the onset of analgesia for deeper subcutaneous and intramuscular/intra-articular injections. Patients will return for further procedures if they know their physician has excellent technique and causes them minimal pain.

13.12.2.9 Negative Aspiration

When the needle has reached the target site one should perform a 5 s. aspiration by applying negative pressure on the syringe. This will reduce the risk of inadvertent intravascular injection. The authors recommend using a 10 ml control syringe for this purpose with finger loops that allow a one-handed aspiration.

13.12.2.10 Injectate Volume

Typically when performing a therapeutic injection, an ample amount of injectate should be used to ensure adequate coverage of the target structure. If the diagnosis is in question and the patient's response to the procedure will influence further treatment, then the smallest possible injectate volume should be used. The volumes indicated in the forthcoming procedure descriptions reflect a standard therapeutic volume rather than a diagnostic volume.

13.12.2.11 Steroid Dosing

There is little data identifying the "necessary" amount of corticosteroid to administer with various procedures. The precise dose selected may be influenced by a number of factors, including the presence of systemic diseases like diabetes and osteoporosis or the amount of exogenous corticosteroid already administered in the preceding months. The doses listed reflect the authors' preferences and will be listed in milligrams of methylprednisolone. For equal-potency conversion to other corticosteroids, please consult Table 13.1.

13.12.2.12 Injection Resistance

With intra-articular and soft tissue injections there should be only slight resistance to the flow of medication while compressing the syringe. If resistance is considerable or there is significant pain induced, the needle tip should be repositioned. Usually this signifies injection into the bone, sub-periosteal, or within the substance of a tendon, all of which one wants to avoid! Withdrawing the needle by a millimeter or two will often alleviate the problem.

13.12.2.13 Microbicide Removal

All residual skin preparation solution should be washed off following the procedure to minimize skin irritation.

13.12.2.14 Dressing

Hold pressure on the punctured site until all the bleeding has stopped. A simple adhesive bandage will suffice as a dressing in most cases.

13.12.2.15 Sharps

Dispose of all sharps in properly labeled, puncture-proof containers.

13.13 Soft Tissue Injections

13.13.1 Greater Trochanteric Bursa (Fig. 13.3)

Positioning: Lying on one's side with the bottom leg straight and the top leg in partial hip and knee flexion.

Injection landmarks: The greater trochanter is palpable on the lateral aspect of the hip. Select the site of maximal tenderness over the trochanter for the target site.

Injectate composition: 5 ml of 1 % lidocaine and 30 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 5 cm, 25 gauge needle is advanced to the bone. Once the bone is contacted, the needle is withdrawn 3–5 mm (to the level of the bursa) and, following negative aspiration, the injection is performed.

Pearls: If the patient has more diffuse pain on palpation of the trochanter, consider placing one-half of the injectate at the site of maximal tenderness and then perform a four quadrant infiltration about the greater trochanter with the remainder of the solution.

13.13.2 Ischial Bursa (Fig. 13.4)

Positioning: Prone.

Injection landmarks: The ischium is palpable on the inferior aspect of the buttocks. Select the site of maximal tenderness over the ischium for the target site.

Injectate composition: 4 ml of 1 % lidocaine and 20-30 mg of methylprednisolone.

Fig. 13.3 Greater trochanteric bursa injection





Fig. 13.4 Ischial bursa injection

Injection technique: Following an appropriate skin wheal, a 6 cm, 22 gauge needle is advanced to the bone. Once ischium is contacted, the needle is withdrawn 3–5 mm (to the level of the bursa) and, following negative aspiration, the injection is performed.

Pearls: If the patient has more diffuse pain on palpation of the ischium, consider placing one-half of the injectate at the site of maximal pain, and then perform a four quadrant infiltration about the remainder of the ischium. For obese patients a longer (9 cm) needle may be required.

13.13.3 Hamstring Origin (Fig. 13.5)

Positioning: Prone

Injection landmarks: The ischium is palpable on the inferior aspect of the buttocks. Have the patient activate the hamstring muscles to aid palpation of the attachments to the ischium. Select the site of maximal tenderness for the target site.

Injectate composition: 4 ml of 1 % lidocaine and 20–30 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 6 cm, 22 gauge needle is advanced to the hamstring tendon origins at their attachments to the ischium. Initially aim for the proximal tendon and "walk" the needle cephalad until the inferior aspect of the ischium is contacted. Once the bone/tendon junction is contacted, the needle is withdrawn 1–2 mm and, following negative aspiration, the injection is performed.

Pearls: There should be minimal resistance to the flow of the injectate. If resistance is high, this may signify that the needle tip is within the substance of the tendon. In this case, the needle should be slowly withdrawn until minimal resistance is encountered. For obese patients, a longer (9 cm) needle may be required.



Fig. 13.5 Hamstring origin injection

13 Nonsurgical Interventions

Fig. 13.6 Iliopsoas bursa injection



13.13.4 Iliopsoas Bursa (Fig. 13.6)

Positioning: Supine.

Injection landmarks: Identify and mark the femoral pulse and neurovascular bundle at the level of the inguinal ligament. The needle entry point is 2.5 cm lateral and 2.5 cm inferior.

Injectate composition: 5 ml of 1 % lidocaine and 20-30 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 9 cm, 22 gauge needle is advanced via a slightly superior and medial trajectory from the entry point until the bone is contacted. Once the bone is contacted, the needle is withdrawn 3–5 mm (to the level of the bursa) and, following careful negative aspiration, the injection is performed.

Pearls: The accuracy of this injection is significantly enhanced by using image guidance such as ultrasound where the iliopsoas tendon and bursa can be visualized and targeted, or fluoroscopy where the lip of the acetabulum or inferomedial femoral neck can be directly targeted.

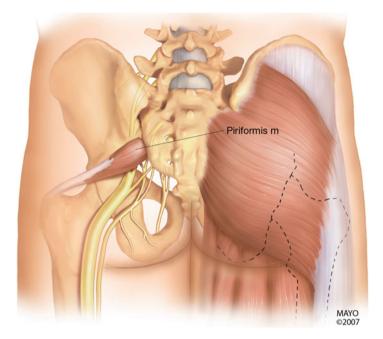


Fig. 13.7 Piriformis muscle located deep to the gluteal muscles in the buttock

13.13.5 Piriformis Injection (Ultrasound Guided) (Figs. 13.7 and 13.8)

Positioning: Prone with the symptomatic side closest to the proceduralist.

Injection landmarks: The PSIS should be palpated and a curvilinear probe should be placed over it in the axial plan. Moving the probe caudally and slightly lateral the SIJ should be visualized distally. Eventually, the ilium falls out of view as the probe scans over the sciatic notch. The first muscle observed deep to the gluteus maximus, lateral to the sacrum is the piriformis muscle. (Fig. 13.8) The lateral aspect of the probe can be slid distally to give a better view of the piriformis fibers. The sciatic nerve can be visualized deep to the piriformis frequently [10].

Injectate composition: 5 ml of ½ % lidocaine and 40 mg of methylprednisolone *Injection technique*: Following an appropriate skin wheal, a 9 cm, 22 gauge needle is advanced from distal lateral to proximal medial in plane with the probe until the sheath or the muscle belly has been reached. Following negative aspiration the injection is performed.

Pearls: The femur on the side of interest can be internally and externally rotated by flexing the knee and swinging the foot medially and laterally. This movement helps distinguish the piriformis from the gluteus maximus.



Fig. 13.8 Piriformis muscle identified using ultrasound guidance

13.13.6 Hip Adductor Origin (Fig. 13.9)

Positioning: Supine with hip slightly flexed, abducted, and externally rotated.

Injection landmarks: The pubis is easily palpable at the medial aspect of the inguinal crease. Have the patient activate the adductor muscles to aid palpation of the attachments to the inferior pubic ramus. Select the site of maximal tenderness for the target site.

Injectate composition: 4 ml of 1 % lidocaine and 20-30 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 6 cm, 22 gauge needle is advanced to the adductor tendon origins at their attachments to the inferior pubic ramus. Once the bone/tendon junction is contacted, the needle is withdrawn 1-2 mm and, following negative aspiration, the injection is performed.

Pearls: There should be minimal resistance to the flow of the injectate. If resistance is high this may signify that the needle tip is within the substance of the tendon. In this case the needle should be slowly withdrawn until minimal resistance is encountered.

13.14 Joint Injections

13.14.1 Sacroiliac (Non-image Guided) (Fig. 13.10)

Positioning: Prone.

Injection landmarks: Identify and mark the PSIS. The needle point is 1 cm medial to the midpoint of the PSIS.

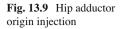




Fig. 13.10 Sacroiliac joint injection (non-image guided technique)



Injectate composition: 5 ml of 1 % lidocaine and 30-40 mg of methylprednisolone.

Injection technique (non-image guided): Following an appropriate skin wheal, a 6 cm or 9 cm, 22 gauge needle is advanced in an oblique fashion laterally under the PSIS until the bone is contacted. The injectate should be infiltrated in a fan-type distribution along the dorsal aspect of the posterior sacroiliac ligaments. This technique accomplishes a periarticular injection.

Pearls: The accuracy of sacroiliac injections is significantly enhanced by using fluoroscopic guidance where the posterior inferior (synovial) aspect of the sacroiliac joint is targeted and the needle can be placed for a true intra-articular injection.

13.14.2 Hip (Non-image Guided) (Fig. 13.11)

Positioning: Lateral—symptomatic side up with bottom leg flexed at the hip and knee. The midpoint of the ilium, greater trochanter, and femoral shaft should all line up in the same coronal plane.

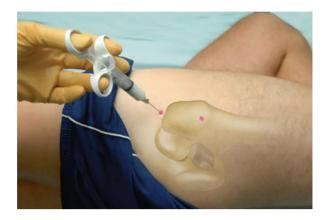


Fig. 13.11 Hip joint injection (non-image guided technique)

Injection landmarks: Palpate and mark the top of the ilium, greater trochanter, and proximal femoral shaft. The needle entry point is 1 cm cephalad to the most proximal portion of the greater trochanter (which may be as much as 5 cm cephalad to the most easily palpable lateral portion of the greater trochanter).

Injectate composition: 5–8 ml of 1 % lidocaine and 40 mg of methylprednisolone. *Injection technique*: Following an appropriate skin wheal, a 9 cm, 22 gauge needle is inserted in the coronal plane of the femoral neck at a 30° (downward) angle. The needle is advanced until the bone is contacted. The injection is made through the capsular attachment to the femoral neck.

Pearls: The accuracy of this injection is significantly enhanced by using image guidance such as ultrasound fluoroscopy. Under fluoroscopic guidance a more anterior approach is used to directly guide the needle through the hip capsule and into the joint space.

13.14.3 Hip (Ultrasound Guided) (Fig. 13.12)

Positioning: Supine- symptomatic side closest to the proceduralist with the hip and knee extended.

Injection landmarks: The anterior superior iliac spine can be palpated and the ultrasound probe can be placed over the bone. The probe can then slowly be moved medially and inferiorly in a sagittal oblique position (parallel with the femoral neck) until the junction of the femoral head and femoral neck can be visualized.

Injectate composition: 5-8 ml of 1 % lidocaine and 40 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 9 cm 22 gauge needle is inserted at the distal, lateral end of the probe and the needle is advanced under direct observation until the tip penetrates the joint capsule and is resting on the femur at the head–neck junction [58].

Pearls: One can use color Doppler to avoid the anterior circumflex artery.

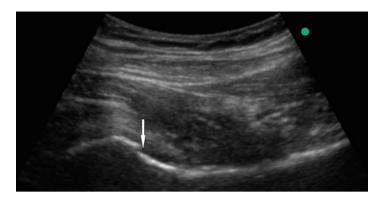
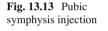
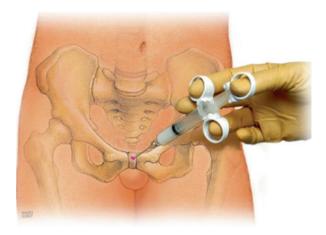


Fig. 13.12 Ultrasound image of the hip. The target for injection is at the proximal femoral neck





13.15 Other Bony Articulation Injections

13.15.1 Pubic Symphysis (Fig. 13.13)

Positioning: Supine.

Injection landmarks: Identify the pubic tubercles bilaterally and mark the cleft midline between.

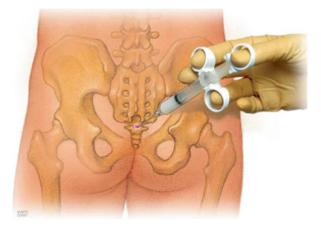
Injectate composition: 3 ml of 1 % lidocaine and 20 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 3.75–5 cm, 25 gauge needle is advanced to the symphysis. Once the fibrocartilaginous disc is contacted, advance the needle an additional 5 mm into the cleft.

Pearls: The accuracy of this injection is significantly enhanced by using image guidance. Prior to performing a corticosteroid injection into the pubic symphysis, infectious osteitis pubis must be definitively excluded.

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Fig. 13.14 Sacrococcygeal injection



13.15.2 Sacrococcygeal (Fig. 13.14)

Positioning: Prone.

Injection landmarks: Identify the sacral cornu bilaterally and mark 1 cm inferior, midline. The articulation of the sacrum and coccyx should be palpable near this position. Adjust the needle entry point accordingly.

Injectate composition: 3 ml of 1 % lidocaine and 20 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 3.75 cm, 25 gauge needle is advanced until the bone is contacted. The needle is withdrawn 1-2 mm and, following negative aspiration, the injection is performed.

Pearls: Caution should be exercised upon advancing the needle to guard against inadvertent puncture of the rectum by missing the coccyx.

13.16 Nerve Blocks

13.16.1 Lateral Femoral Cutaneous Nerve (Non-image Guided) (Fig. 13.15)

Positioning: Supine.

Injection landmarks: Identify the ASIS and mark 1 cm medial and inferior as the needle entry point.

Injectate composition: 6 ml of 1 % lidocaine and 20 mg of methylprednisolone.

Injection technique: Following an appropriate skin wheal, a 5 cm, 25 gauge needle is advanced slightly superior and laterally, underneath the ASIS until the ilium is contacted. The injection is then performed by infiltrating in a fan-type distribution perpendicular to the course of the traversing lateral femoral cutaneous nerve.

Pearls: The ilioinguinal and iliohypogastric nerves run in close proximity to this target site and the patient should be forewarned about the possibility of inadvertently anesthetizing these structures.

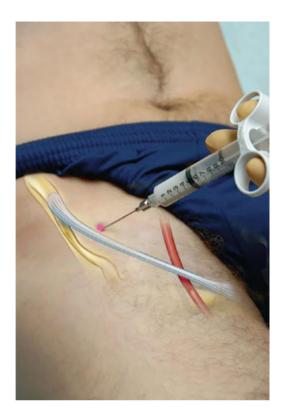


Fig. 13.15 Lateral femoral cutaneous nerve block

13.16.2 Lateral Femoral Cutaneous Nerve (Ultrasound Guided) (Fig. 13.16)

Positioning: Supine

Injections landmarks: Identify the ASIS and the lateral patella. Mark the skin 10 cm distal to the ASIS on the line between the ASIS and lateral patella. Place a linear ultrasound probe on the patient at the marked area in a transverse plane. Visualize the nerve in the hypoechoic perineural fat and trace it as far proximally to the inguinal ligament [59].

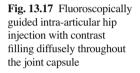
Injectate composition: 3 ml of 1 % lidocaine and 20 mg of methylprednisolone.

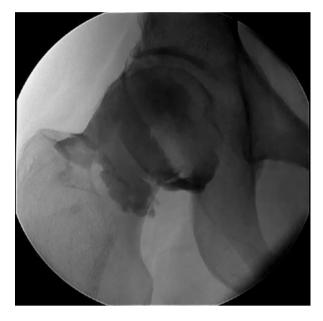
Injection technique: Visualize the nerve on the medial side of the probe. Following an appropriate skin wheal just lateral to the probe, a 5 cm, 25 gauge needle is advanced lateral to medial just deep to the nerve. Once the needle tip is next to the nerve inject around the nerve [59].

Pearls: The ilioinguinal and iliohypogastric nerves run in close proximity to this target site and the patient should be forewarned about the possibility of inadvertently anesthetizing these structures.



Fig. 13.16 Ultrasound visualization of the lateral femoral cutaneous nerve





13.17 Case Presentation Wrap-up

Fluoroscopically guided intra-articular local anesthetic/corticosteroid injection of the hip did not alleviate his pain (Fig. 13.17). Fluoroscopically, guided local anesthetic/corticosteroid injection of the iliopsoas bursa did completely relieve his pain (Fig. 13.18). The diagnosis was iliopsoas bursitis.

Treatment consisted of temporary activity modification, scheduled NSAID administration, physical therapy directed at gentle stretching of the iliopsoas muscle, and iliopsoas and hip girdle strengthening.

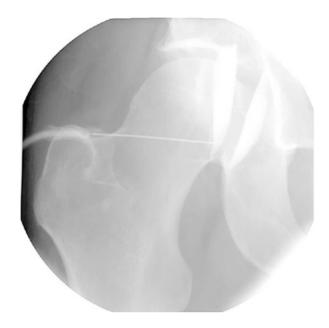


Fig. 13.18 Fluoroscopically guided injection of the iliopsoas bursa with the iliopsoas tendon traversing the center of the bursa

On follow-up, the patient was able to gradually return to running and basketball. No subsequent discomfort or dysfunction developed from the labral tear identified on MR arthrogram.

Various hip and pelvis disorders may often present with similar symptoms and examination findings. Diagnostic injections can be an effective tool to help identify the pain generator. In the case presented the iliopsoas bursa injection confirmed the diagnosis, distinguishing it from an incidental non-symptomatic hip labral tear.

References

- Hench PS, Kendall EC, et al. The effect of a hormone of the adrenal cortex (17-hydroxy-11dehydrocorticosterone; compound E) and of pituitary adrenocorticotropic hormone on rheumatoid arthritis. Proc Staff Meet Mayo Clin. 1949;24(8):181–97.
- Qvistgaard E, Christensen R, Torp-Pedersen S, Bliddal H. Intra-articular treatment of hip osteoarthritis: a randomized trial of hyaluronic acid, corticosteroid, and isotonic saline. Osteoarthritis Cartilage. 2006;14(2):163–70.
- 3. Raynauld JP, Buckland-Wright C, Ward R, Choquette D, Haraoui B, Martel-Pelletier J, et al. Safety and efficacy of long-term intraarticular steroid injections in osteoarthritis of the knee: a randomized, double-blind, placebo-controlled trial. Arthritis Rheum. 2003;48(2):370–7.
- Fernandez Lopez JC, Ruano-Ravina A. Efficacy and safety of intraarticular hyaluronic acid in the treatment of hip osteoarthritis: a systematic review. Osteoarthritis Cartilage. 2006;14(12):1306–11.
- Kim SR, Stitik TP, Foye PM, Greenwald BD, Campagnolo DI. Critical review of prolotherapy for osteoarthritis, low back pain, and other musculoskeletal conditions: a physiatric perspective. Am J Phys Med Rehabil. 2004;83(5):379–89.
- Rabago D, Best TM, Beamsley M, Patterson J. A systematic review of prolotherapy for chronic musculoskeletal pain. Clin J Sport Med. 2005;15(5):376–80.

13 Nonsurgical Interventions

- Topol GA, Reeves KD, Hassanein KM. Efficacy of dextrose prolotherapy in elite male kickingsport athletes with chronic groin pain. Arch Phys Med Rehabil. 2005;86(4):697–702.
- Naredo E, Cabero F, Beneyto P, Cruz A, Mondejar B, Uson J, et al. A randomized comparative study of short term response to blind injection versus sonographic-guided injection of local corticosteroids in patients with painful shoulder. J Rheumatol. 2004;31(2):308–14.
- 9. Sofka CM, Adler RS. Ultrasound-guided interventions in the foot and ankle. Semin Musculoskelet Radiol. 2002;6(2):163–8.
- Smith J, Hurdle MF, Locketz AJ, Wisniewski SJ. Ultrasound-guided piriformis injection: technique description and verification. Arch Phys Med Rehabil. 2006;87(12):1664–7.
- 11. Pulisetti D, Ebraheim NA. CT-guided sacroiliac joint injections. J Spinal Disord. 1999;12(4):310–2.
- 12. Carek PJ. Joint and soft tissue injections in primary care. Clin Fam Pract. 2005:359-78.
- Mader R, Lavi I, Luboshitzky R. Evaluation of the pituitary-adrenal axis function following single intraarticular injection of methylprednisolone. Arthritis Rheum. 2005;52(3):924–8.
- 14. Bellamy N, Campbell J, Robinson V, Gee T, Bourne R, Wells G. Viscosupplementation for the treatment of osteoarthritis of the knee. Cochrane Database Syst Rev. 2006;2, CD005321.
- Kelly MA, Kurzweil PR, Moskowitz RW. Intra-articular hyaluronans in knee osteoarthritis: rationale and practical considerations. Am J Orthop (Belle Mead NJ). 2004;33(2 Suppl):15–22.
- van den Bekerom MPJ, Lamme B, Sermon A, Mulier M. What is the evidence for viscosupplementation in the treatment of patients with hip osteoarthritis? Systematic review of the literature. Arch Orthop Trauma Surg. 2008;128(8):815–23.
- Atchia I, Kane D, Reed MR, Isaacs JD, Birrell F. Efficacy of a single ultrasound-guided injection for the treatment of hip osteoarthritis. Ann Rheum Dis. 2011;70(1):110–6.
- Migliore A, Massafra U, Bizzi E, Vacca F, Martin-Martin S, Granata M, et al. Comparative, double-blind, controlled study of intra-articular hyaluronic acid (Hyalubrix (R)) injections versus local anesthetic in osteoarthritis of the hip. Arthritis Res Ther. 2009;11(6):R183.
- Richette P, Ravaud P, Conrozier T, Euller-Ziegler L, Mazieres B, Maugars Y, et al. Effect of hyaluronic acid in symptomatic hip osteoarthritis: a multicenter, randomized placebocontrolled trial. Arthritis Rheum. 2009;60(3):824–30.
- 20. AstraZeneca Pharmaceuticals. Xylocaine injection, lidocaine HCl and epinephrine injection. Product Information, 2001.
- Choi YK, Liu J. The use of 5% lidocaine for prolonged analgesia in chronic pain patients: a new technique. Reg Anesth Pain Med. 1998;23(1):96–100.
- 22. Hospira Inc. Marcaine injection, bupivicaine HCl injection. Product Information; 2004.
- Hackett G. Ligament and tendon relaxation (skeletal disability) treated by prolotherapy (fibroosseous proliferation). 3rd ed. Springfield: Charles C Thomas; 1958.
- 24. Saunders S, Longworth S. Injection techniques in orthopedics and sports medicine. In: Elsevier, editor. 3rd ed. 2006.
- 25. Creaney L, Hamilton B. Growth factor delivery methods in the management of sports injuries: the state of play. Br J Sports Med. 2008;42(5):314–20.
- 26. Coppinger JA, Cagney G, Toomey S, Kislinger T, Belton O, McRedmond JP, et al. Characterization of the proteins released from activated platelets leads to localization of novel platelet proteins in human atherosclerotic lesions. Blood. 2004;103(6):2096–104.
- 27. Geaney LE, Arciero RA, DeBerardino TM, Mazzocca AD. The effects of platelet-rich plasma on tendon and ligament: basic science and clinical application. Oper Tech Sports Med. 2011;19(3):160–4.
- Mishra A, Harmon K, Woodall J, Vieira A. Sports medicine applications of platelet rich plasma. Curr Pharm Biotechnol. 2012;13(7):1185–95.
- Finnoff JT, Fowler SP, Lai JK, Santrach PJ, Willis EA, Sayeed YA, et al. Treatment of chronic tendinopathy with ultrasound-guided needle tenotomy and platelet-rich plasma injection. PM R. 2011;3(10):900–11.
- Hammond JW, Hinton RY, Curl LA, Muriel JM, Lovering RM. Use of autologous platelet-rich plasma to treat muscle strain injuries. Am J Sports Med. 2009;37(6):1135–42.
- Mejia HA, Bradley JP. The effects of platelet-rich plasma on muscle: basic science and clinical application. Oper Tech Sports Med. 2011;19(3):149–53.

- Podesta L, Crow SA, Volkmer D, Bert T, Yocum LA. Treatment of partial ulnar collateral ligament tears in the elbow with platelet-rich plasma. Am J Sports Med. 2013;41(7):1689–94.
- 33. Terada S, Ota S, Kobayashi M, Kobayashi T, Mifune Y, Takayama K, et al. Use of an antifibrotic agent improves the effect of platelet-rich plasma on muscle healing after injury. J Bone Joint Surg Am. 2013;95A(11):980–8.
- 34. Hamid MSA, Mohamed MR, Yusof A, George J, Lee LPC. Platelet-rich plasma injections for the treatment of hamstring injuries: a randomized controlled trial. Am J Sports Med. 2014;42(10):2410–8.
- 35. Laver L, Carmont MR, McConkey MO, Palmanovich E, Yaacobi E, Mann G, et al. Plasma rich in growth factors (PRGF) as a treatment for high ankle sprain in elite athletes: a randomized control trial. Knee Surg Sports Traumatol Arthrosc. 2015;23(11):3383–92.
- Akeda K, An HS, Okuma M, Attawia M, Miyamoto K, Thonar EJ, et al. Platelet-rich plasma stimulates porcine articular chondrocyte proliferation and matrix biosynthesis. Osteoarthritis Cartilage. 2006;14(12):1272–80.
- 37. Gobbi A, Karnatzikos G, Mahajan V, Malchira S. Platelet-rich plasma treatment in symptomatic patients with knee osteoarthritis: preliminary results in a group of active patients. Sports Health. 2012;4(2):162–72.
- Kon E, Mandelbaum B, Buda R, Filardo G, Delcogliano M, Timoncini A, et al. Platelet-rich plasma intra-articular injection versus hyaluronic acid viscosupplementation as treatments for cartilage pathology: from early degeneration to osteoarthritis. Arthroscopy. 2011;27(11):1490–501.
- 39. Rayegani SM, Raeissadat SA, Taheri MS, Babaee M, Bahrami MH, Eliaspour D, et al. Does intra articular platelet rich plasma injection improve function, pain and quality of life in patients with osteoarthritis of the knee? A randomized clinical trial. Orthop Rev (Pavia). 2014;6(3):5405.
- 40. Dold AP, Zywiel MG, Taylor DW, Dwyer T, Theodoropoulos J. Platelet-rich plasma in the management of articular cartilage pathology: a systematic review. Clin J Sport Med. 2014;24(1):31–43.
- 41. Laudy AB, Bakker EW, Rekers M, Moen MH. Efficacy of platelet-rich plasma injections in osteoarthritis of the knee: a systematic review and meta-analysis. Br J Sports Med. 2015;49(10):657–72.
- 42. Pittenger MF, Mackay AM, Beck SC, Jaiswal RK, Douglas R, Mosca JD, et al. Multilineage potential of adult human mesenchymal stem cells. Science. 1999;284(5411):143–7.
- 43. Anderson JA, Little D, Toth AP, Moorman 3rd CT, Tucker BS, Ciccotti MG, et al. Stem cell therapies for knee cartilage repair: the current status of preclinical and clinical studies. Am J Sports Med. 2014;42(9):2253–61.
- 44. Counsel PD, Bates D, Boyd R, Connell DA. Cell therapy in joint disorders. Sports Health. 2015;7(1):27–37.
- 45. Gobbi A, Karnatzikos G, Scotti C, Mahajan V, Mazzucco L, Grigolo B. One-step cartilage repair with bone marrow aspirate concentrated cells and collagen matrix in full-thickness knee cartilage lesions: results at 2-year follow-up. Cartilage. 2011;2(3):286–99.
- Wolfstadt JI, Cole BJ, Ogilvie-Harris DJ, Viswanathan S, Chahal J. Current concepts: the role of mesenchymal stem cells in the management of knee osteoarthritis. Sports Health. 2015;7(1):38–44.
- Veronesi F, Giavaresi G, Tschon M, Borsari V, Aldini NN, Fini M. Clinical use of bone marrow, bone marrow concentrate, and expanded bone marrow mesenchymal stem cells in cartilage disease. Stem Cells Dev. 2013;22(2):181–92.
- 48. Filardo G, Madry H, Jelic M, Roffi A, Cucchiarini M, Kon E. Mesenchymal stem cells for the treatment of cartilage lesions: from preclinical findings to clinical application in orthopedics. Knee Surg Sports Traumatol Arthrosc. 2013;21(8):1717–29.
- Anz AW, Hackel JG, Nilssen EC, Andrews JR. Application of biologics in the treatment of the rotator cuff, meniscus, cartilage, and osteoarthritis. J Am Acad Orthop Surg. 2014;22(2):68–79.
- 50. Administration USFaD. CFR-Code of Federal Regulations, Title 21. April 2015.

13 Nonsurgical Interventions

- Hernigou P, Homma Y, Flouzat Lachaniette CH, Poignard A, Allain J, Chevallier N, et al. Benefits of small volume and small syringe for bone marrow aspirations of mesenchymal stem cells. Int Orthop. 2013;37(11):2279–87.
- 52. Vangsness CT, Farr J, Boyd J, Dellaero DT, Mills CR, LeRoux-Williams M. Adult human mesenchymal stem cells delivered via intra-articular injection to the knee following partial medial meniscectomy a randomized, double-blind, controlled study. J Bone Joint Surg Am. 2014;96A(2):90–8.
- Gray RG, Gottlieb NL. Intra-articular corticosteroids. An updated assessment. Clin Orthop Relat Res. 1983;177:235–63.
- Kumar N, Newman RJ. Complications of intra- and peri-articular steroid injections. Br J Gen Pract. 1999;49(443):465–6.
- 55. Cardone DA, Tallia AF. Joint and soft tissue injection. Am Fam Physician. 2002;66(2):283-8.
- Mens JM, Nico de Wolf A, Berkhout BJ, Stam HJ. Disturbance of the menstrual pattern after local injection with triamcinolone acetonide. Ann Rheum Dis. 1998;57(11):700.
- Saufl NM. Universal protocol for preventing wrong site, wrong procedure, wrong person surgery. J Perianesth Nurs. 2004;19(5):348–51.
- Smith J, Hurdle MF. Office-based ultrasound-guided intra-articular hip injection: technique for physiatric practice. Arch Phys Med Rehabil. 2006;87(2):296–8.
- Hurdle MF, Weingarten TN, Crisostomo RA, Psimos C, Smith J. Ultrasound-guided blockade of the lateral femoral cutaneous nerve: technical description and review of 10 cases. Arch Phys Med Rehabil. 2007;88(10):1362–4.

Chapter 14 Treatment Options for Degenerative Joint Disease of the Hip

Adam T. Liegner, Heather M. Gillespie, and William W. Dexter

Clinical Pearls

- Strength training and aerobic exercise can reduce pain and improve function and health status in patients with hip degenerative joint disease and should be recommended for all patients.
- Current pharmacologic treatment is focused on symptom control and should be seen as adjunctive to nonpharmacologic therapies.
- Although widely considered as first line pharmacologic therapy for hip degenerative joint disease, recent evidence challenges the efficacy of acetaminophen.
- Intra-articular corticosteroid injections are an effective, low-risk therapy for pain associated with degenerative joint disease of the hip.
- After all conservative measures are exhausted, pain and function are the primary determinants for surgery.

14.1 Case Presentation

The patient is a 40-year-old nurse and aerobics instructor with a history of depression and fibromyalgia who presents with hip pain, increasing over a period of 2 years. She describes a dull, achy pain on the lateral hip that radiates to the groin.

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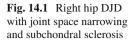
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The pain is worse with activity and limits her range of motion; however, she has been able to remain active with bicycling. On examination, she has a body mass index (BMI) of 24, no tenderness to palpation over the hip, but pain is reproduced with hip internal rotation. She has decreased range of motion in internal rotation of the right hip compared to the left. Radiographs show a mild amount of joint space narrowing and subchondral sclerosis (Fig. 14.1).

14.2 Epidemiology

Degenerative joint disease (DJD) is the most common cause of musculoskeletal pain and disability. A progressive and debilitating disease, DJD affects over 15% of the world's population [1] and is a major cause of morbidity and health care expenditures. One longitudinal population-based study evaluating over 3000 subjects has estimated the lifetime risk of hip DJD as 25% [2]. Likewise, data from the Center for Disease Control and Prevention's Behavioral Risk Factor Surveillance System shows that risk to increase to 35% for those over the age of 50 and further to more than 55% for those over the age of 70 [3]. One study ranked hip and knee DJD as the 11^{th} highest contributor to global disability of 291 conditions studied [4]. As the population continues to age and prevalence of obesity continues to rise, the cost and consequences from DJD will continue to grow.

DJD, characterized by joint pain and dysfunction, is associated with defective integrity of the articular cartilage and related changes in the underlying bone and

joint margins. Currently, there is no known cure for DJD. New treatments and disease-modifying therapies are currently under investigation, but the etiology of DJD is still not completely understood. Therapy is aimed at decreasing pain and dysfunction and increasing mobility and overall quality of life. The hip is the second most common large joint to be affected by DJD [5, 6]. The prevalence of hip DJD ranges from 3% to 11% in Western populations aged over 35 years [6], with reported prevalence variation due to differences in radiographic case definitions [7].

14.3 Risk Factors

Risk factors associated with DJD include systemic factors, such as genetics and bone density, as well as biomechanical factors that affect the joints, such as reduced muscle strength [8]. Age, obesity, high bone mass, joint location, joint malalignment, trauma, gender, comorbidities, biochemical changes, and lifestyle (participation in weight-bearing sports and occupations that require prolonged standing, lifting or moving of heavy objects) have all been associated with the development of DJD [1, 9, 10]. Age-related changes in cartilage alter the biomechanical characteristics of collagen and proteoglycans. Mechanical wear, chondrocytes, and cytokines, principally interleukin (IL)-1 β (beta) and transforming growth factor (TGF)- β (beta), all play roles in the pathogenesis of the disease [11].

In males, trauma and age are associated with DJD. A positive association with hip trauma is also found in unilateral but not bilateral hip DJD. Obesity is associated with bilateral but not unilateral hip DJD [12]. Several studies have found an association between increasing BMI and an increased incidence of DJD [10, 13, 14].

Other secondary risk factors associated with DJD include hemochromatosis, hyperparathyroidism, hypothyroidism, acromegaly, hyperlaxity syndromes, Paget's disease, gout, and chondrocalcinosis [9].

14.4 Diagnosis

The accurate diagnosis of hip DJD relies on a combination of both clinical and radiographic findings. Radiographic evidence of joint degeneration and characteristic subjective symptoms of pain and disability have been found to be superior to clinical criteria alone.

Physical symptoms include generalized hip pain, pain in the lateral or anterior thigh and groin, and pain with prolonged ambulation. Physical signs include antalgic gait, decreased range of motion, and pain with internal rotation. The pain is often described as deep and achy. In early disease, the pain may be intermittent and mostly with joint use, but as the pain becomes more chronic, patients may also experience pain at night. The joint is often described as "stiff" and patients have difficulty with initiating movements. In advanced disease, crepitus may develop and



Fig. 14.2 Hip DJD with joint space narrowing

range of motion may become limited [15]. Radiographic findings in patients with DJD include cartilage space narrowing, osteophytosis, subchondral cysts, subchondral sclerosis, femoral neck buttressing, and femoral head remodeling (Figs. 14.2, 14.3, 14.4, and 14.5) [11, 16]. Historically, radiographic evidence of osteophytes, cysts, and subchondral sclerosis has been used to diagnose DJD; however, more recently research has focused on joint space width (JSW) as the primary determinant and radiographic criterion for hip DJD.

Jacobsen et al. found that minimum JSW less than or equal to 2 mm had the closest association with self-reported hip pain in 3807 subjects whose mean age was 61 years old [7]. Gupta et al. found that cartilage space narrowing was the most sensitive predictor of hip DJD [11]. Croft et al. studied 1315 men aged 60–75 and found minimal joint space to be the best radiographic criterion of hip DJD to use in epidemiologic studies, at least for men [17]. In 1991, Altman et al. proposed a classification tree of (1) hip pain and osteophytosis or (2) hip pain and cartilage space narrowing with a sedimentation rate less than 20 mm/h. The study demonstrated a sensitivity of 89% and a specificity of 91% for hip DJD [18].

Once the diagnosis is made, severity can be quantitatively tracked using the Western Onterio and McMaster Universities DJD Index (WOMAC) scores, which is composed of 24 items assessing pain, stiffness, and physical function. The Visual Analog Scale (VAS) for pain is an alternative, more general measure of disease-associated pain. However, many providers choose to track disease severity by historical symptoms alone.

Fig. 14.3 Hip DJD with osteophytes and subchondral sclerosis



Fig. 14.4 Severe hip DJD with loss of joint space and subchondral sclerosis



14.5 Nonoperative Management

Overall, the goals of nonoperative management in DJD are to reduce pain and functional impairment, to improve mobility, and perhaps to delay or prevent the need for surgery. Treatments should be chosen to limit the side effects of therapy. Expert opinion supports the statement that optimal management of hip DJD requires a combination of nonpharmacologic and pharmacologic treatment modalities (Table 14.1) [6].



Fig. 14.5 Severe hip DJD with subchondral cysts, osteophytes, subchondral sclerosis, and joint space loss

Nonpharmacologic	Pharmacologic
Exercise	Intra-articular corticosteroid
Weight loss	Intra-articular viscosupplementation ^a
Patient education and self- management programs	Acetaminophen
Assistive devices	NSAIDs
Acupuncture	Tramadol
	Opioids
	Glucosamine and chondroitin
	Diacerein
	Other supplements

Table 14.1 Nonoperative management of hip DJD

Nonoperative treatments listed by nonpharmacologic and pharmacologic ^aNot FDA approved for treatment of hip DJD as of 2015

While several well-recognized organizations have produced evidence-based nonoperative treatment guidelines for DJD, three have created specific recommendations for hip and knee DJD: the American College of Rheumatology (ACR), the DJD Research Society International (OARSI), and the European League Against Rheumatism (EULAR) [19–21]. All three include review of high-level evidence including meta-analyses, systematic reviews, and randomized controlled trials.

Treatment of hip DJD should be tailored to the individual patient taking into account patient comorbidities, current medications, level of pain, disability and handicap, degree of structural damage, and baseline physical activity and functional status.

14.5.1 Nonpharmacologic Therapy

Nonpharmacologic treatment of hip DJD should include patient education, self-management programs, aerobic and resistance exercise, lifestyle changes, weight reduction if obese or overweight, and acupuncture. The evidence weakly supports the use of assistive devices such as walking canes and wheeled walkers to reduce pain and increase exercise participation [21]. Treatments previously in use that evidence suggests are ineffective include electromagnetic therapy [19].

14.5.1.1 Exercise: General Benefit

Exercise and physical activity have clearly been shown to benefit those with largejoint DJD [5, 15]. Decreased lower extremity strength is associated with increased disability in people with DJD. Disease-related factors such as impaired muscle function and reduced fitness are amenable to therapeutic exercise. Expert opinion from a 2005 systematic review states that improvement in muscle strength and proprioception gained from exercise programs may reduce the progression of hip DJD. Notably, the effectiveness of exercise is thought to be independent of the presence or severity of radiographic findings [5].

Svege et al. found that an exercise program specifically designed for hip DJD consisting of strengthening, flexibility, and functional exercises significantly delayed the time until total hip arthroplasty (THR) [22]. Interestingly, in this study the exercise therapy group had better self-reported hip function, but no significant differences were found for pain or stiffness.

Perhaps unsurprisingly, there is evidence to show that the beneficial posttreatment effects of exercise are not sustained in the long-term after routine exercise is no longer maintained [23].

14.5.1.2 Exercise: Type

Land-based exercise programs and water-based exercise programs, with components of strength training, aerobic exercise, and flexibility training have been shown to reduce pain and improve function and health status in patients with hip DJD [24, 25]. Likewise, a 2014 Cochrane Review of 10 RCTs concluded that land-based

physical exercise programs were effective in reducing pain and improving physical function associated with DJD of the hip [26].

If the resources and facilities are available, water-based therapy (hydrotherapy, aquatic physical therapy) can be an ideal setting for people with DJD. The buoyancy of the water reduces the load across the joints affected by pain and allows for performance of functional closed-chain exercises that would otherwise be too difficult. The warmth and pressure of the water may aid in pain relief, swelling reduction, and ease of movement. Warm water encourages muscle relaxation and reduces guarding around the joints, which leads to increased range of motion and ultimate functional gains [27].

In a 2007 RCT of 71 patients comparing 6 weeks of hydrotherapy versus no treatment, the hydrotherapy group had significantly less pain and improved physical function, strength, and quality of life compared to controls, with benefit sustained 6 weeks after cessation of the program [28]. Foley et al. studied 105 patients randomized to three water- or land-based exercise sessions a week for 6 weeks. Both water and gym exercises were found to improve function, with land-based exercises being better for strength and water-based superior for aerobic conditioning [29]. Similarly, a 2007 Cochrane Review reported some positive short-term effects, but a lack of evidence supporting a long-term effect, leading the authors to suggest that water-based therapy be used as a gateway to land-based exercise therapy [27].

14.5.1.3 Exercise: Adherence and Risk

One of the leading indicators of success and benefit from exercise therapy is patient adherence to the program. The bottom line is that performing exercise is more important than the type performed. Strategies to improve adherence such as longterm monitoring and review, setting specific exercise-related goals that are easy to achieve, frequent encouragement, and inclusion of a spouse or other family member in the exercise should be considered in the exercise prescription.

Supervised classes appear to be as beneficial as treatments on a one-to-one basis [15]. Group exercise and home exercise were found to be equally effective while a supervised group format potentially provides a more cost-effective alternative. Also, social contact with peers may help to increase adherence [5, 15].

Furthermore, while there is no data regarding intensity of exercise in hip DJD, a Cochrane review examining the effectiveness of therapeutic exercise at different intensities in people with DJD of the knee found both high-intensity and low-intensity aerobic exercise to be equally effective in improving patient's functional status, gait, pain, and aerobic capacity [30]. Studies have also shown that dropout rates are related to the intensity of the exercise, with higher intensity having a higher rate of withdrawal [30]. It is important to carefully tailor the exercise type and intensity to the individual patient, erring on the side of a low-intensity exercise program with a slow progression to achieve maximum short and long-term effects.

There are few contraindications to the prescription of strengthening or aerobic exercise in patients with hip DJD, however, age, comorbidities, resources, and overall mobility should all be taken into account to ultimately increase patient adherence and overall success [5]. Exercise prescription for DJD should include aerobic, strength, and flexibility training. These programs should be individualized.

14.5.1.4 Weight Loss

Obesity is a modifiable risk factor for developing hip DJD and weight loss has been shown to reduce the pain and disability associated with hip DJD [10, 31]. Expert guidelines universally consider weight loss a cornerstone of management for overweight patients with hip DJD [3].

Obesity is thought to contribute to DJD via two general mechanisms. The historically accepted mechanism attributes increased DJD risk to the increased load across weight-bearing joints. However, more recently, a second mechanism has been theorized in which increased levels of adipose-associated systemic inflammatory mediators (i.e., adipokines, free fatty acids, and reactive oxygen species) play a role [32–34]. This biochemical mechanism is based on the observation that the risk of DJD of non-weight-bearing joints is also increased in obese patients compared to non-obese counterparts. Subsequently, some now consider metabolic DJD a subtype of DJD and believe that DJD should be considered a fifth component of metabolic syndrome [35, 36].

Lastly, it is worth noting that some studies have demonstrated increased intraand post-operative adverse events in THR for obese patients [37–39]. These findings should motivate providers to recommend weight loss as an important treatment modality in the management of hip DJD regardless of the possible progression to THR.

14.5.1.5 Patient Education and Self-Management Programs

The evidence surrounding arthritis self help groups that teach patients how to manage their disease is mixed. A 2014 Cochrane Review of 29 studies by Kroon et al. concluded that review of the generally low quality evidence available regarding self help groups for DJD reveals either no or small benefit to pain, function, and quality of life, but they also concede that self-management education programs are unlikely to cause harm [40]. For those interested in pursuing self help groups, community organizations as well as the Arthritis Foundation can serve as a resource for education materials. Physicians may also organize group visits designed to address DJD alone or in combination with other common comorbidities such as obesity, diabetes, and hypertension.

Self-management programs have been shown to reduce anxiety and improve participants' perceived self-efficacy to manage their symptoms of DJD. Education that the disease is not relentlessly progressive and self-management tools, such as techniques to deal with problems such as pain, fatigue, frustration, and isolation, can decrease doctor visits. In addition, treating depression in patients with DJD may help reduce the amount of pain and improve their functional status and quality of life [41, 42].

14.5.1.6 Assistive Devices

While no research evidence exists regarding the use of specific shoes for hip DJD, both the ACR and EULAR recommend their use as low-risk interventions. EULAR specifies that appropriate shoes do not have a raised heel, have thick shock-absorbing soles, support for the arches of the foot, and a size big enough to allow comfortable space for the toes [21]. There is likewise no research evidence for appliances such as canes and walkers [6] in the treatment of hip DJD, but theoretically they may help to alter joint forces [6, 21]. In general, there are few contraindications if the device is found to be effective for individual patients.

14.5.1.7 Acupuncture

Evidence exists supporting the use of acupuncture in the management of chronic pain [43] and has specifically been studied in the treatment of DJD. A 2006 RCT of acupuncture and DJD found patients treated with acupuncture in addition to routine care to have significant improvement in symptoms and quality of life compared with patients who received routine care alone [44]. A 2014 systematic review and meta-analysis reaffirmed these conclusions [45]. Acupuncture is a safe intervention when administered by physicians, has a small potential for adverse events, and should be considered as adjuvant treatment for hip DJD [44].

14.5.2 Pharmacologic Therapy

Pharmacologic therapy in DJD should be undertaken as a supplement, not a replacement, for nonpharmacologic therapy. Drug therapy has been found to be most potent when combined with nonpharmacologic treatment. The majority of current pharmacologic options in the treatment of DJD are symptom-modifying therapies; however, structure- and disease-modifying therapies are currently in development.

Current symptom-modifying medications include analgesics (acetaminophen, opioids, and tramadol), nonsteroidal anti-inflammatory drugs (NSAIDs), COX-2 inhibitors, and intra-articular corticosteroid and viscosupplementation.

14.5.2.1 Acetaminophen

Several organizations consider acetaminophen, up to a maximum daily dose of 4000 mg, first line pharmacologic therapy for the management of hip DJD [19, 20], however, recent studies raise concerns about efficacy and safety. A 2006 Cochrane systematic review concluded that NSAIDs are superior to acetaminophen for pain control in DJD of the hip [46] and in 2009, an advisory committee of the US Food and Drug Administration recommended, but did not mandate, lowering the maximum total daily dose from 4000 mg to 3250 mg due to concerns about liver damage [47].

A 2015 meta-analysis and systematic review of studies evaluating a total of 3541 subjects over a period of up to 3 months concluded only minimal benefit (3.7 point improvement on a 0–100 point symptom and function scale) when used for management of hip DJD [48]. This analysis did not find any statistically significant difference between acetaminophen and placebo groups for participants reporting any adverse event or serious adverse events, but did find that the acetaminophen group was nearly four times as likely to have abnormal liver function tests (defined as 1.5 times the upper limit of normal) than the placebo group [48]. It is worth noting that a 2006 systematic review evaluating hepatotoxicity in acetaminophen-treated patients with baseline normal liver function found that low level, transient ALT elevations were shown to usually resolve or decrease with continued therapy and were not accompanied by signs of liver injury [49].

14.5.2.2 NSAIDs and COX-2 inhibitors

NSAIDs are widely used to control the pain of DJD. Expert opinion recommends that NSAIDs, at the lowest effective dose, should be added or substituted for patients who respond inadequately to acetaminophen. There is strong evidence that NSAIDs provide significant pain relief for DJD; however, they are also associated with significant side effects and risks, particularly adverse gastrointestinal (GI) events [6]. The practitioner should also be aware that these drugs now have a "Black Box Warning" due to the risk for both cardiovascular and gastrointestinal serious side effects.

GI side effects associated with NSAID use are reported to lead to over \$500 million annually in health care costs [50]. There is a 2–4% annual incidence of serious GI ulcer and complications in NSAID users which is four times higher than in nonusers [51]. NSAIDs result in at least 7000 deaths annually in the USA attributed to GI side effects [52]. Therefore, NSAIDs should be prescribed with caution in patients over the age of 65 or with a history of peptic ulcer disease, upper GI bleed, oral glucocorticoid therapy, and anticoagulation. All patients should be counseled regarding the risks of NSAIDs. In patients with increased GI risk, nonselective NSAIDs plus a gastroprotective agent, or a selective COX-2 inhibitor should be used. These strategies are more expensive and only cost-effective in patients with greater GI risk [6]. Both NSAIDs and COX-2 inhibitors should be prescribed with caution for those with renal and cardiovascular disease. A healthy kidney is able to compensate from prostaglandin inhibition, but if baseline renal function is impaired, there is a high risk of acute kidney injury [53]. More recent studies assessing the cardiovascular risk of NSAIDs and COX-2 inhibitors have shown significantly increased risk of cardiovascular events such as myocardial infarction and stroke [54, 55]. It is worth noting that there is some evidence to suggest that naproxen offers the lowest risk of cardiovascular side effects [54, 55].

14.5.2.3 Opioids

Opioids offer a modest benefit over placebo in the treatment of DJD, however, the shortcomings of opioid therapy are several fold. The primary risks include dependence, addiction, and overdose. Furthermore, pain control associated with opioid use typically diminishes within 4 weeks of use [56].

Opioids may be appropriately prescribed in hip DJD as a bridging therapy to impending THR [56]. Extended release forms are preferable to immediate-release formulations at providing consistent stable analgesia. It may be necessary to switch opioid medications, one or more times to achieve an acceptable balance, between adverse events and analgesia as patients have variable responses to different opioids. Side effects with a dose–response relationship include nausea, vomiting, constipation, dizziness, somnolence, and pruritus.

14.5.2.4 Tramadol

Tramadol is an opioid agonist and centrally acting analgesic not chemically related to opioids. When taken up to 3 months for DJD, a Cochrane Review gave Tramadol gold level evidence for decreasing pain, and improving stiffness, function, and overall well-being [57]. A 2006 RCT demonstrated that Tramadol ER was effective for patients with knee or hip DJD with limited side effects [58].

Tramadol, in contrast to NSAIDs, does not cause GI bleeding, renal problems or aggravate hypertension and CHF. Compared with narcotics, tramadol does not have significant abuse potential. Common side effects from tramadol include nausea, vomiting, dizziness, sweating, constipation, tiredness, and headache.

14.5.2.5 Intra-Articular Injectable Therapy

Glucocorticoids and viscosupplementation are the most common intra-articular therapies used for hip DJD. Three primary techniques exist for intra-articular injections of the hip: landmark-guided injections (LMGIs), fluoroscopically guided injections (FGIs), and ultrasound-guided injections (USGIs). Due to the deep location of the hip joint, injections should be performed under fluoroscopic or ultrasound guidance

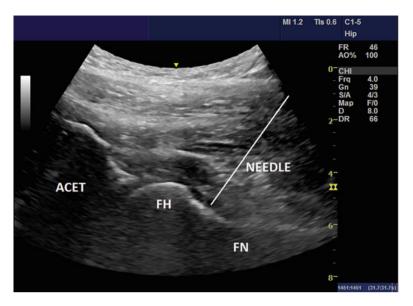


Fig. 14.6 Ultrasound image of intra-articular hip injection (needle enhanced for clarity). Legend: *ACET* acetabulum, *FH* femoral head, *FN* femoral neck

(Fig. 14.6). While both fluoroscopy and ultrasound are well tolerated, only ultrasound permits the rapid identification of soft tissue structures, notably the femoral neurovascular bundle, and is a radiation-free technique. Overall, the procedure is regarded as innocuous and safe [59]. Contraindications to joint injections include bacteremia, inaccessible joints, joint prosthesis, adjacent osteomyelitis, and overlying infection of the soft tissue.

In 2015, the American Medical Society for Sports Medicine evaluated the overall accuracy, efficacy, and cost effectiveness of USGIs versus LMGIs in the treatment of musculoskeletal disease. The group concluded that, in general across all types of injections, there was strong evidence that accuracy was superior in USGIs, moderate evidence that efficacy was superior in USGIs, and preliminary evidence that USGIs were more cost-effective than LMGIs. Within the subcategory of USGIs of the hip, strong evidence showed that accuracy of USGIs (91–100%) were much greater than LMGIs (64–81%). Regarding efficacy for injections involving the hip, study results again supported the use of ultrasound, however, the body of evidence was much less substantial. Unfortunately no studies assessing cost effectiveness of USGIs of the hip were identified [60].

Corticosteroids. Corticosteroids inhibit the inflammatory and immune cascade at several levels and may be most useful in patients with local inflammation and joint effusion. Intra-articular corticosteroids have an overall anti-inflammatory effect and have been shown to be effective in managing DJD. However, their long-term benefits and safety have not been established definitively [1]. There are no long-term studies examining the risks of multiple injections, but, in general, no more then 3–4 injections per year are recommended.

Several high quality studies have shown that intra-articular corticosteroid injections provide measureable benefit on pain, ROM, and function for DJD of the hip. Although the effect of corticosteroid was short lived, with regression of benefit by 2–4 months, these results support corticosteroid use in the treatment of hip DJD for acute pain relief. The presence of an effusion in the hip joint immediately postinjection was associated with good clinical response, suggesting that localization of the injection to within the joint capsule leads to improved outcomes [59, 61, 62].

If total hip replacement (THR) is expected in the near future, caution should be taken in proceeding with intra-articular steroid injection. In a 2006 retrospective review of 224 patients with primary THR implanted within 1 year of intra-articular steroid injection compared with 224 who had not received an injection, there was overall no statistically significant effect on postoperative rates of infection. However, the mean time from injection to THR in patients with deep postoperative infection was 44 days compared to a mean of 112 days for the group as a whole [63]. Subsequent studies have concurred with these findings [64–66]. It is recommended that intra-articular steroids injected into the hip should be avoided for a minimum of 3 months before THR.

Viscosupplementation. Hyaluronic acid (HA) is a long polysaccharide chain synthesized by type B synoviocytes and fibroblasts in the synovium and secreted into the joint space. Due to its HA content, synovial fluid has both viscous (lubricating) and elastic (shock-absorbing) properties [50]. A normal knee contains approximately 2 mL of synovial fluid with a HA concentration of 2.5–4.0 mg/mL. In an osteoarthritic knee, HA concentration is reduced by a factor of 2–3 due to degradation and dilution, and the molecular weight of HA is reduced as well [50].

Viscosupplementation is the intra-articular injection of exogenous HA. The dwelling time of exogenous HA in the joint is short lived. However, it exerts its effect over a longer period of time than corticosteroids and may also take longer to achieve this effect. The mechanism is not completely understood, but there are several theories including the restoration of rheologic (viscoelastic) properties of synovial fluid, an anti-inflammatory effect, an anti-nociceptive effect, normalization of endogenous HA synthesis, and chondroprotection.

In a prospective trial of 56 patients with primary hip DJD, aged 40 and older, over 50 % had a decrease in pain and an increase in function after a single 2 mL dose of HA under fluoroscopic guidance. The outcomes were better in less severe DJD and an inverse correlation was seen between the reduction in pain and the joint space narrowing score [67]. One double blind controlled study comparing low molecular weight HA, corticosteroid, and saline (placebo) showed overall no difference at 3 months, but hyaluronic acid did appear superior to placebo in moderate disease [59].

In the USA as of 2015, viscosupplementation is off label for use in hip DJD as it is currently only approved for knee DJD by the FDA. However, in several studies, intra-articular viscosupplementation has proven to be safe with ultrasound guidance in the hip [59, 68]. Overall incidence of side effects is 1–4% per injection with the most common being a severe, though self limited, local reaction. Precaution should be taken in patients with avian allergies for many formulations.

In summary, data suggest that viscosupplementation may reduce symptoms in hip DJD, especially in moderate disease, and is safe and well tolerated. It may be indicated for patients who have not responded to nonpharmacologic or oral drug therapy, but lack of FDA approval limits access to this treatment.

14.5.2.6 Diacerein

Diacerein is a drug with interleukin-1 β (beta) (IL-1 β (beta)) inhibitory properties. Not currently FDA approved in the USA, oral diacerein is available in Europe for the treatment of DJD. In a meta-analysis of 19 RCTs (2637 patients), Rintelen et al. found diacerein to be superior to placebo and similar to NSAIDs in active treatment of DJD. There was no difference in tolerability compared to NSAIDs and diacerein had an additional carryover and prolonged residual effect after the treatment phase [69].

Diacerein has, however, come under scrutiny due to the side effects of diarrhea and liver toxicity with the European Medicines Agency recommending against its use in those over 65 or those with a history of liver disease as of 2014.

14.5.2.7 Glucosamine and Chondroitin

Glucosamine sulfate participates in the synthesis of proteoglycans and glycosaminoglycans found in hyaline cartilage. Chondroitin sulfate is a glycosaminoglycan found in cartilage and other connective tissues. They are considered dietary supplements and are, therefore, not regulated by the FDA. Commercial preparations are readily available over the counter, but caution should be used as safety and efficacy may vary from preparation to preparation [1]. Typical doses of glucosamine and chondroitin are 1500 mg/day and 800–1200 mg/day respectively. Onset of effect is variable and results may not be seen for 2 months.

High quality studies evaluating glucosamine and chondroitin in the treatment of hip DJD have shown no benefit beyond placebo, however, risks of the treatment appear to be minimal. The Glucosamine/Chondroitin Arthritis Intervention Trial (GAIT) published in 2006 evaluated the efficacy of glucosamine and chondroitin in the treatment of knee arthritis in 1583 patients. There was a high placebo response in the study and results showed the supplements were not significantly better than placebo at reducing arthritis pain by 20 %. Nevertheless, subgroup analysis revealed the combination to be effective in moderate-to-severe knee pain [70]. A 2008 RCT evaluating the efficacy of glucosamine sulfate in 222 subjects with hip DJD with 2 years of daily therapy showed no significant effect of either placebo or glucosamine on pain, function, or joint space narrowing [71]. Similarly, a 2010 meta-analysis evaluating the effect of glucosamine and chondroitin individually and in combination compared to placebo found no effect on knee or hip DJD disease parameters [72].

Finally, it is worth noting that the American College of Rheumatology, as of 2012, conditionally recommends against the use of chondroitin sulfate and glucosamine in the management of hip DJD [20].

14.5.2.8 Other Supplements

While a 2009 Cochrane review concluded that insufficient evidence existed to recommend the use of S-adenosyl methionine (SAMe) in the treatment of knee or hip DKD, the authors of a 2011 review article found SAMe at a dose of 1200 mg/day was more effective than placebo and as effective as NSAIDs in the management of hip DJD when considering the outcomes of pain and function [73, 74]. The review also identified a meta-analysis in which patients taking SAMe were much less likely to report adverse events compared to those taking NSAIDs [74].

Avocado/soybean unsaponifiables are nutraceuticals shown in vitro to inhibit proinflammatory cytokines and stimulate chondrocyte collagen synthesis. Trials to date, including a 2014 Cochrane review, have resulted in mixed results with some suggesting improvements in both symptomatic and joint space width parameters and others showing no effect [1, 75–77].

Rose hip may be beneficial in both early and late stages of DJD. In a 2005 RCT of 94 patients, 5 g of a herbal remedy made from one subspecies of rose-hip (*Rosa canina*) taken for 3 months was found to alleviate symptoms of DJD and reduce consumption of rescue medication [78].

14.5.3 Future Directions

Disease-modifying therapies including gene therapy, matrix metalloproteinase inhibitors, and bisphosphonates as well as substances such as green tea and ginger are all potential therapies in need of further study in the treatment of DJD.

14.6 Indications for Surgery

Before any major reconstruction of the hip is recommended, conservative measures should be exhausted. These should include weight loss, exercise, and both systemic and intra-articular pharmacologic therapy. These measures may delay or obviate the need for surgery, and many insurance companies require documentation of conservative treatment failure prior to authorization of surgical treatment.

The most important and agreed upon measures qualifying a patient for THR are pain and function [6]. Radiographic change is important to confirm diagnosis, but

at this time the importance of the degree of change and indication for THR remains unclear [6]. Vinciguerra et al. found factors associated with the risk of THR to be of the age older than 54 at diagnosis, BMI greater than 27, and severe radiologic evidence of joint space narrowing at diagnosis [79]. Likewise, Hawker et al. have identified 6 key criteria with which to judge the appropriateness of THR: (1) evidence of arthritis on joint examination, (2) patient-reported symptoms negatively impacting quality of life, (3) an adequate trial of appropriate nonsurgical treatment, (4) realistic patient expectations of surgery, (5) mental and physical readiness of patient for surgery, and (6) patient–surgeon agreement that potential benefits exceed risks [80].

In young adults with symptomatic hip DJD, osteotomy and joint preserving surgical procedures should be considered, especially in the presence of dysplasia or varus/valgus deformity [6].

Just as shared decision making has been shown to lead to improved patient and physician satisfaction in other realms of health care, studies have also revealed its importance within the realm of THR. A 2013 RCT by Bozic et al. showed a higher rate of patients making an informed decision during the first visit, greater patient confidence in asking meaningful questions, and greater provider satisfaction with the quality and efficiency of office visits addressing the need for THR for patients receiving a shared decision-making intervention compared to those who did not. These findings were in spite of the fact that rates of surgery were not significantly different between groups [81]. In a follow-up secondary analysis using the same study population, Youm et al. found that those of higher socioeconomic status and education levels were more likely to come to a decision regarding surgery after the initial visit and were less likely to choose surgical treatment compared to those of lower socioeconomic status and education [82]. These findings highlight the importance of shared decision making in THR, especially for patients of lower socioeconomic status and educations levels.

14.7 Case Conclusion

Diagnosed with DJD, the patient began taking glucosamine and chondroitin and daily acetaminophen. With minimal results, she attempted a trial of daily NSAIDs without significant results. The pain continued to limit her functional capacity and quality of life. Several cortisone injections under ultrasound guidance were done, the effects never lasting more than a month. Given her young age and desire to remain moderately active, she elected to try viscosupplementation. Three weekly injections given under ultrasound guidance unfortunately provided very short-term relief. After exhausting all of her nonsurgical options, the patient underwent a THR. She subsequently has been able to return to many activities and overall reports an increased quality of life.

References

- 1. Fajardo M, Di Cesare PE. Disease-modifying therapies for osteoarthritis : current status. Drugs Aging. 2005;22(2):141–61.
- Murphy LB, Helmick CG, Schwartz TA, Renner JB, Tudor G, Koch GG, et al. One in four people may develop symptomatic hip osteoarthritis in his or her lifetime. Osteoarthritis Cartilage. 2010;18(11):1372–9.
- Nelson AE, Allen KD, Golightly YM, Goode AP, Jordan JM. A systematic review of recommendations and guidelines for the management of osteoarthritis: the chronic osteoarthritis management initiative of the U.S. bone and joint initiative. Semin Arthritis Rheum. 2014;43(6):701–12.
- Cross M, Smith E, Hoy D, Nolte S, Ackerman I, Fransen M, et al. The global burden of hip and knee osteoarthritis: estimates from the global burden of disease 2010 study. Ann Rheum Dis. 2014;73(7):1323–30.
- Roddy E, Zhang W, Doherty M, Arden NK, Barlow J, Birrell F, et al. Evidence-based recommendations for the role of exercise in the management of osteoarthritis of the hip or knee—the MOVE consensus. Rheumatology (Oxford). 2005;44(1):67–73.
- 6. Zhang W, Doherty M, Arden N, Bannwarth B, Bijlsma J, Gunther KP, et al. EULAR evidence based recommendations for the management of hip osteoarthritis: report of a task force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT). Ann Rheum Dis. 2005;64(5):669–81.
- Jacobsen S, Sonne-Holm S, Soballe K, Gebuhr P, Lund B. Radiographic case definitions and prevalence of osteoarthrosis of the hip: a survey of 4 151 subjects in the Osteoarthritis Substudy of the Copenhagen City Heart Study. Acta Orthop Scand. 2004;75(6):713–20.
- Zeggini E, Panoutsopoulou K, Southam L, Rayner NW, Day-Williams AG, Lopes MC, et al. Identification of new susceptibility loci for osteoarthritis (arcOGEN): a genome-wide association study. Lancet. 2012;380(9844):815–23.
- 9. Lane NE. Clinical practice. Osteoarthritis of the hip. N Engl J Med. 2007;357(14):1413–21.
- Richmond SA, Fukuchi RK, Ezzat A, Schneider K, Schneider G, Emery CA. Are joint injury, sport activity, physical activity, obesity, or occupational activities predictors for osteoarthritis? A systematic review. J Orthop Sports Phys Ther. 2013;43(8):515–B19.
- Gupta KB, Duryea J, Weissman BN. Radiographic evaluation of osteoarthritis. Radiol Clin North Am. 2004;42(1):11–41. v.
- Tepper S, Hochberg MC. Factors associated with hip osteoarthritis: data from the First National Health and Nutrition Examination Survey (NHANES-I). Am J Epidemiol. 1993;137(10):1081–8.
- Lohmander LS, Gerhardsson de Verdier M, Rollof J, Nilsson PM, Engstrom G. Incidence of severe knee and hip osteoarthritis in relation to different measures of body mass: a populationbased prospective cohort study. Ann Rheum Dis. 2009;68(4):490–6.
- Williams PT. Effects of running and walking on osteoarthritis and hip replacement risk. Med Sci Sports Exerc. 2013;45(7):1292–7.
- Fransen M, McConnell S, Bell M. Exercise for osteoarthritis of the hip or knee. Cochrane Database Syst Rev. 2003(3):Cd004286.
- Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16(4):494–502.
- Croft P, Cooper C, Wickham C, Coggon D. Defining osteoarthritis of the hip for epidemiologic studies. Am J Epidemiol. 1990;132(3):514–22.
- Altman R, Alarcon G, Appelrouth D, Bloch D, Borenstein D, Brandt K, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hip. Arthritis Rheum. 1991;34(5):505–14.
- Zhang W, Nuki G, Moskowitz RW, Abramson S, Altman RD, Arden NK, et al. OARSI recommendations for the management of hip and knee osteoarthritis: part III: changes in evidence following systematic cumulative update of research published through January 2009. Osteoarthritis Cartilage. 2010;18(4):476–99.

- Hochberg MC, Altman RD, April KT, Benkhalti M, Guyatt G, McGowan J, et al. American College of Rheumatology 2012 recommendations for the use of nonpharmacologic and pharmacologic therapies in osteoarthritis of the hand, hip, and knee. Arthritis Care Res. 2012;64(4):465–74.
- Fernandes L, Hagen KB, Bijlsma JW, Andreassen O, Christensen P, Conaghan PG, et al. EULAR recommendations for the non-pharmacological core management of hip and knee osteoarthritis. Ann Rheum Dis. 2013;72(7):1125–35.
- 22. Svege I, Nordsletten L, Fernandes L, Risberg MA. Exercise therapy may postpone total hip replacement surgery in patients with hip osteoarthritis: a long-term follow-up of a randomised trial. Ann Rheum Dis. 2015;74(1):164–9.
- 23. Pisters MF, Veenhof C, van Meeteren NL, Ostelo RW, de Bakker DH, Schellevis FG, et al. Long-term effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: a systematic review. Arthritis Rheum. 2007;57(7):1245–53.
- Tak E, Staats P, Van Hespen A, Hopman-Rock M. The effects of an exercise program for older adults with osteoarthritis of the hip. J Rheumatol. 2005;32(6):1106–13.
- Uthman OA, van der Windt DA, Jordan JL, Dziedzic KS, Healey EL, Peat GM, et al. Exercise for lower limb osteoarthritis: systematic review incorporating trial sequential analysis and network meta-analysis. BMJ (Clinical Research Ed). 2013;347:f5555.
- Fransen M, McConnell S, Hernandez-Molina G, Reichenbach S. Exercise for osteoarthritis of the hip. Cochrane Database Syst. Rev. 2014;4:Cd007912.
- Bartels EM, Lund H, Hagen KB, Dagfinrud H, Christensen R, Danneskiold-Samsoe B. Aquatic exercise for the treatment of knee and hip osteoarthritis. Cochrane Database Syst Rev. 2007(4):Cd005523.
- 28. Hinman RS, Heywood SE, Day AR. Aquatic physical therapy for hip and knee osteoarthritis: results of a single-blind randomized controlled trial. Phys Ther. 2007;87(1):32–43.
- 29. Foley A, Halbert J, Hewitt T, Crotty M. Does hydrotherapy improve strength and physical function in patients with osteoarthritis—a randomised controlled trial comparing a gym based and a hydrotherapy based strengthening programme. Ann Rheum Dis. 2003;62(12):1162–7.
- Brosseau L, MacLeay L, Robinson V, Wells G, Tugwell P. Intensity of exercise for the treatment of osteoarthritis. Cochrane Database Syst Rev. 2003(2):Cd004259.
- Maly MR, Robbins SM. Osteoarthritis year in review 2014: rehabilitation and outcomes. Osteoarthritis Cartilage. 2014;22(12):1958–88.
- 32. Abella V, Scotece M, Conde J, Lopez V, Lazzaro V, Pino J, et al. Adipokines, metabolic syndrome and rheumatic diseases. J Immunol Res. 2014;2014:343746.
- Kluzek S, Newton JL, Arden NK. Is osteoarthritis a metabolic disorder? Br Med Bull. 2015;115(1):111–21.
- Courties A, Gualillo O, Berenbaum F, Sellam J. Metabolic stress-induced joint inflammation and osteoarthritis. Osteoarthritis Cartilage. 2015;23(11):1955–65.
- 35. Sellam J, Berenbaum F. Is osteoarthritis a metabolic disease? Joint Bone Spine. 2013;80(6):568–73.
- Zhuo Q, Yang W, Chen J, Wang Y. Metabolic syndrome meets osteoarthritis. Nat Rev Rheumatol. 2012;8(12):729–37.
- 37. Elson LC, Barr CJ, Chandran SE, Hansen VJ, Malchau H, Kwon YM. Are morbidly obese patients undergoing total hip arthroplasty at an increased risk for component malpositioning? J Arthroplasty. 2013;28(8 Suppl):41–4.
- Rajgopal R, Martin R, Howard JL, Somerville L, MacDonald SJ, Bourne R. Outcomes and complications of total hip replacement in super-obese patients. Bone Joint J. 2013;95-b(6):758–63.
- 39. Wallace G, Judge A, Prieto-Alhambra D, de Vries F, Arden NK, Cooper C. The effect of body mass index on the risk of post-operative complications during the 6 months following total hip replacement or total knee replacement surgery. Osteoarthritis Cartilage. 2014;22(7):918–27.
- 40. Kroon FP, van der Burg LR, Buchbinder R, Osborne RH, Johnston RV, Pitt V. Self-management education programmes for osteoarthritis. Cochrane Database Syst Rev. 2014;1:Cd008963.
- Buszewicz M, Rait G, Griffin M, Nazareth I, Patel A, Atkinson A, et al. Self management of arthritis in primary care: randomised controlled trial. BMJ (Clinical Research Ed). 2006;333(7574):879.

- Baird CL, Murawski MM, Wu J. Efficacy of guided imagery with relaxation for osteoarthritis symptoms and medication intake. Pain Manag Nurs. 2010;11(1):56–65.
- Vickers AJ, Cronin AM, Maschino AC, Lewith G, MacPherson H, Foster NE, et al. Acupuncture for chronic pain: individual patient data meta-analysis. Arch Intern Med. 2012;172(19):1444–53.
- 44. Witt CM, Jena S, Brinkhaus B, Liecker B, Wegscheider K, Willich SN. Acupuncture in patients with osteoarthritis of the knee or hip: a randomized, controlled trial with an additional nonrandomized arm. Arthritis Rheum. 2006;54(11):3485–93.
- 45. Manyanga T, Froese M, Zarychanski R, Abou-Setta A, Friesen C, Tennenhouse M, et al. Pain management with acupuncture in osteoarthritis: a systematic review and meta-analysis. BMC Complement Altern Med. 2014;14:312.
- Towheed TE, Maxwell L, Judd MG, Catton M, Hochberg MC, Wells G. Acetaminophen for osteoarthritis. Cochrane Database Syst Rev. 2006(1):Cd004257.
- Krenzelok EP, Royal MA. Confusion: acetaminophen dosing changes based on NO evidence in adults. Drugs R&D. 2012;12(2):45–8.
- 48. Machado GC, Maher CG, Ferreira PH, Pinheiro MB, Lin CW, Day RO, et al. Efficacy and safety of paracetamol for spinal pain and osteoarthritis: systematic review and meta-analysis of randomised placebo controlled trials. BMJ (Clinical Research Ed). 2015;350:h1225.
- 49. Kuffner EK, Temple AR, Cooper KM, Baggish JS, Parenti DL. Retrospective analysis of transient elevations in alanine aminotransferase during long-term treatment with acetaminophen in osteoarthritis clinical trials. Curr Med Res Opin. 2006;22(11):2137–48.
- 50. Brockmeier SF, Shaffer BS. Viscosupplementation therapy for osteoarthritis. Sports Med Arthrosc. 2006;14(3):155–62.
- Lin J, Zhang W, Jones A, Doherty M. Efficacy of topical non-steroidal anti-inflammatory drugs in the treatment of osteoarthritis: meta-analysis of randomised controlled trials. BMJ (Clinical Research Ed). 2004;329(7461):324.
- 52. Schnitzer TJ, Burmester GR, Mysler E, Hochberg MC, Doherty M, Ehrsam E, et al. Comparison of lumiracoxib with naproxen and ibuprofen in the Therapeutic Arthritis Research and Gastrointestinal Event Trial (TARGET), reduction in ulcer complications: randomised controlled trial. Lancet. 2004;364(9435):665–74.
- 53. Gorsline RT, Kaeding CC. The use of NSAIDs and nutritional supplements in athletes with osteoarthritis: prevalence, benefits, and consequences. Clin Sports Med. 2005;24(1):71–82.
- 54. Solomon DH, Glynn RJ, Rothman KJ, Schneeweiss S, Setoguchi S, Mogun H, et al. Subgroup analyses to determine cardiovascular risk associated with nonsteroidal antiinflammatory drugs and coxibs in specific patient groups. Arthritis Rheum. 2008;59(8):1097–104.
- 55. Scarpignato C, Lanas A, Blandizzi C, Lems WF, Hermann M, Hunt RH. Safe prescribing of non-steroidal anti-inflammatory drugs in patients with osteoarthritis—an expert consensus addressing benefits as well as gastrointestinal and cardiovascular risks. BMC Med. 2015;13:55.
- 56. da Costa BR, Nuesch E, Kasteler R, Husni E, Welch V, Rutjes AW, et al. Oral or transdermal opioids for osteoarthritis of the knee or hip. Cochrane Database Syst Rev. 2014;9:Cd003115.
- 57. Cepeda MS, Camargo F, Zea C, Valencia L. Tramadol for osteoarthritis. Cochrane Database Syst Rev. 2006(3):Cd005522.
- Gana TJ, Pascual ML, Fleming RR, Schein JR, Janagap CC, Xiang J, et al. Extended-release tramadol in the treatment of osteoarthritis: a multicenter, randomized, double-blind, placebocontrolled clinical trial. Curr Med Res Opin. 2006;22(7):1391–401.
- Qvistgaard E, Christensen R, Torp-Pedersen S, Bliddal H. Intra-articular treatment of hip osteoarthritis: a randomized trial of hyaluronic acid, corticosteroid, and isotonic saline. Osteoarthritis Cartilage. 2006;14(2):163–70.
- Finnoff JT, Hall MM, Adams E, Berkoff D, Concoff AL, Dexter W, et al. American Medical Society for Sports Medicine position statement: interventional musculoskeletal ultrasound in sports medicine. Clin J Sport Med. 2015;25(1):6–22.
- 61. Atchia I, Kane D, Reed MR, Isaacs JD, Birrell F. Efficacy of a single ultrasound-guided injection for the treatment of hip osteoarthritis. Ann Rheum Dis. 2011;70(1):110–6.
- Kullenberg B, Runesson R, Tuvhag R, Olsson C, Resch S. Intraarticular corticosteroid injection: pain relief in osteoarthritis of the hip? J Rheumatol. 2004;31(11):2265–8.

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- McIntosh AL, Hanssen AD, Wenger DE, Osmon DR. Recent intraarticular steroid injection may increase infection rates in primary THA. Clin Orthop Relat Res. 2006;451:50–4.
- 64. Charalambous CP, Prodromidis AD, Kwaees TA. Do intra-articular steroid injections increase infection rates in subsequent arthroplasty? A systematic review and meta-analysis of comparative studies. J Arthroplasty. 2014;29(11):2175–80.
- McMahon SE, LeRoux JA, Smith TO, Hing CB. Total joint arthroplasty following intraarticular steroid injection: a literature review. Acta Orthop Belg. 2013;79(6):672–9.
- Meermans G, Corten K, Simon JP. Is the infection rate in primary THA increased after steroid injection? Clin Orthop Relat Res. 2012;470(11):3213–9.
- 67. Conrozier T, Bertin P, Bailleul F, Mathieu P, Charlot J, Vignon E, et al. Clinical response to intra-articular injections of hylan G-F 20 in symptomatic hip osteoarthritis: the OMERACT-OARSI criteria applied to the results of a pilot study. Joint Bone Spine. 2006;73(6):705–9.
- 68. Migliore A, Massafra U, Bizzi E, Vacca F, Martin-Martin S, Granata M, et al. Comparative, double-blind, controlled study of intra-articular hyaluronic acid (Hyalubrix) injections versus local anesthetic in osteoarthritis of the hip. Arthritis Res Ther. 2009;11(6):R183.
- Rintelen B, Neumann K, Leeb BF. A meta-analysis of controlled clinical studies with diacerein in the treatment of osteoarthritis. Arch Intern Med. 2006;166(17):1899–906.
- Clegg DO, Reda DJ, Harris CL, Klein MA, O'Dell JR, Hooper MM, et al. Glucosamine, chondroitin sulfate, and the two in combination for painful knee osteoarthritis. N Engl J Med. 2006;354(8):795–808.
- Rozendaal RM, Koes BW, van Osch GJ, Uitterlinden EJ, Garling EH, Willemsen SP, et al. Effect of glucosamine sulfate on hip osteoarthritis: a randomized trial. Ann Intern Med. 2008;148(4):268–77.
- Wandel S, Juni P, Tendal B, Nuesch E, Villiger PM, Welton NJ, et al. Effects of glucosamine, chondroitin, or placebo in patients with osteoarthritis of hip or knee: network meta-analysis. BMJ (Clinical Research Ed). 2010;341:c4675.
- Rutjes AW, Nuesch E, Reichenbach S, Juni P. S-Adenosylmethionine for osteoarthritis of the knee or hip. Cochrane Database Syst Rev. 2009(4):Cd007321.
- 74. De Silva V, El-Metwally A, Ernst E, Lewith G, Macfarlane GJ. Evidence for the efficacy of complementary and alternative medicines in the management of osteoarthritis: a systematic review. Rheumatology (Oxford). 2011;50(5):911–20.
- 75. Ameye LG, Chee WS. Osteoarthritis and nutrition. From nutraceuticals to functional foods: a systematic review of the scientific evidence. Arthritis Res Ther. 2006;8(4):R127.
- 76. Maheu E, Cadet C, Marty M, Moyse D, Kerloch I, Coste P, et al. Randomised, controlled trial of avocado-soybean unsaponifiable (Piascledine) effect on structure modification in hip osteoarthritis: the ERADIAS study. Ann Rheum Dis. 2014;73(2):376–84.
- Cameron M, Chrubasik S. Oral herbal therapies for treating osteoarthritis. Cochrane Database Syst Rev. 2014;5:Cd002947.
- 78. Winther K, Apel K, Thamsborg G. A powder made from seeds and shells of a rose-hip subspecies (Rosa canina) reduces symptoms of knee and hip osteoarthritis: a randomized, double-blind, placebo-controlled clinical trial. Scand J Rheumatol. 2005;34(4):302–8.
- Vinciguerra C, Gueguen A, Revel M, Heuleu JN, Amor B, Dougados M. Predictors of the need for total hip replacement in patients with osteoarthritis of the hip. Rev Rhum Engl Ed. 1995;62(9):563–70.
- 80. Hawker G, Bohm ER, Conner-Spady B, De Coster C, Dunbar M, Hennigar A, et al. Perspectives of Canadian stakeholders on criteria for appropriateness for total joint arthroplasty in patients with hip and knee osteoarthritis. Arthritis Res Ther. 2015;67(7):1806–15.
- Bozic KJ, Belkora J, Chan V, Youm J, Zhou T, Dupaix J, et al. Shared decision making in patients with osteoarthritis of the hip and knee: results of a randomized controlled trial. J Bone Joint Surg Am. 2013;95(18):1633–9.
- Youm J, Chan V, Belkora J, Bozic KJ. Impact of socioeconomic factors on informed decision making and treatment choice in patients with hip and knee OA. J Arthroplasty. 2015;30(2):171–5.

Chapter 15 Surgical Interventions in Hip and Pelvis Injuries

Matthew C. Bessette and Brian D. Giordano

Clinical Pearls

- The diagnosis of hip and pelvis pathology can be difficult, and a complete understanding of anatomy and pathology combined with a thorough history and physical examination is essential. Diagnostic injections can be invaluable for pain mapping and clarification of primary versus secondary pain generators.
- Most hip and pelvic pathologies respond favorably to an initial trial of nonoperative treatment.
- Femoral neck fractures and traumatic hip dislocations in an athlete require emergent treatment.
- The treating clinician should be comfortable with all available imaging modalities, including radiographs, MRI, and CT, and should be aware of the high rate of asymptomatic hip and pelvic pathology that may be seen in athletic individuals.
- Adolescents with hip pathology may present with knee pain, and there should be a high index of suspicion for hip or pelvic pathology in this setting.

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Fig. 15.1 Anteroposterior (AP) pelvic radiograph demonstrating preserved joint space in the right hip. There is cephalad acetabular retroversion, as demonstrated by a positive "crossover sign" (*arrow*) when comparing the anterior and posterior acetabular walls. The walls of the left hip, which also demonstrates cephalad retroversion, are outlined

15.1 Case Presentation

15.1.1 Chief Complaint

Left hip and anterior pelvic pain.

15.1.2 Patient History

A 20-year-old male hurdler presents with the insidious onset of left hip and anterior pelvic discomfort that worsens with activity, especially when hurdling and lunging. He is minimally symptomatic at rest. The pain has gradually worsened and is unresponsive to conservative treatments including activity modifications.

15.1.3 Physical Examination

The patient is a healthy-appearing young adult. He has tenderness around his pubic symphysis and the left parasymphyseal musculature. Passive hip flexion is 120° and at 90° of flexion, his hip can be internally rotated to 20° . He has pain with passive flexion, adduction, and internal rotation and has mild weakness with active hip flexion.

15.1.4 Imaging

Plain radiographs of the left hip and pelvis (Fig. 15.1) show well-maintained joint space with mild cephalad acetabular retroversion. Magnetic resonance arthrogram (MRA) and computerized tomography (CT) of the hip confirm focal acetabular retroversion as

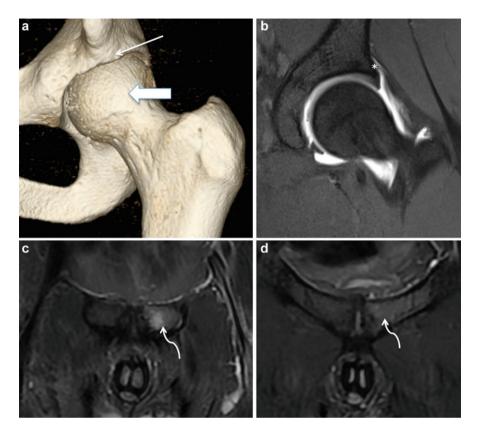


Fig. 15.2 (a) 3D CT reconstructions of the left hip demonstrate focal overcoverage from acetabular retroversion (*thin arrow*) at the anterosuperior aspect of the acetabulum as well as decreased femoral head–neck offset (*thick arrow*). (b) T2 MRA of the left hip demonstrating an intact chondrolabral junction (*). (c and d) T2 MRI sequences of the anterior pelvic ring demonstrating increased signal in the left parasymphyseal bone (*curved arrows*)

well as reduced femoral head–neck offset, but no definitive evidence of a discrete labral tear. MRI of the pelvis shows bony edema in the left parasymphyseal bone (Fig. 15.2).

15.2 Introduction

In athletes, hip and pelvic injuries occur less frequently than other lower extremity injuries, but can be a source of considerable impairment and compromised athletic performance. Forces exceeding several times the body's weight are transferred through structures of the hip and pelvis even during routine activities, rendering this area susceptible to injury [1]. The diagnosis and treatment of these disorders can present the treating physician with many challenges. Interrelated body regions and pathologic arthrokinematics can confuse the primary and secondary pain generators. Clarification of primary hip joint pathology from compensatory changes in the adjacent segments of the kinetic chain is vitally important to formulating a strategic treatment plan.

While few surgical emergencies do exist, non-operative modalities with extensive rehabilitation protocols have classically been the mainstay of treatment for most athletic hip and pelvic injuries. Progress in our understanding of the complex pathomechanics and advances in diagnostic imaging have led to increased recognition of these injuries. Technological advances, coupled with a parallel expansion in the application of hip arthroscopy, have enabled physicians to address a wide variety of intra- and extra-articular pathologies through less invasive means [2, 3]. Indications for arthroscopic hip surgery include, but are not limited to symptomatic

Indications for arthroscopic hip surgery include, but are not limited to, symptomatic femoroacetabular impingement (FAI), loose bodies, labral tears, chondral injuries, synovial pathology, joint sepsis, ligamentum teres injuries, psoas or extra-articular impingement patterns, pathology of the peritrochanteric space, instability, and coxa saltans [3]. Complications are uncommon, and generally stem from traction-related nerve injuries and fluid extravasations [4]. Dysplastic hips may actually undergo accelerated degeneration after arthroscopy due to iatrogenic instability [5]. It is important for the treating clinician to acknowledge the high rate of asymptomatic hip joint and periarticular pathology in athletic individuals when formulating a treatment plan. With advances in the sensitivity of diagnostic imaging, in particular magnetic resonance imaging (MRI), the clinician must use thoughtful clinical judgment and correlate radiographic findings with a focused clinical exam before pursuing surgical management of hip and pelvic pathology [6].

15.3 Skeletal Injuries

15.3.1 Hip Dislocation

Femoroacetabular dislocations are typically associated with high-energy trauma, such as motor vehicle accidents, and are often accompanied by fractures of the acetabulum, femoral head, and femoral neck. While rare, these injuries can occur during routine sporting activity. The majority of hip dislocations occur in a posterior direction (Fig. 15.3). With or without associated fractures, rapid reduction of the dislocation is paramount in order to decrease the risk of osteonecrosis. Mehlman's longitudinal study of pediatric dislocations found a 20-fold increase in the rate of osteonecrosis when reduction was delayed more than 6 h [7]. Osteonecrosis of the femoral head is one of the most feared complications of a hip dislocation as it is difficult to treat, and rates vary widely in the literature [8].

Reduction may first be attempted with appropriate sedation and analgesia through a variety of traction maneuvers. Failure may necessitate reduction under general anesthesia or an open reduction. Dislocations with associated fractures, such as those to the acetabulum or femoral head, should be treated urgently; most commonly, this involves open reduction and internal fixation. If appropriate resources are not available at the initial treating facility, strong consideration should

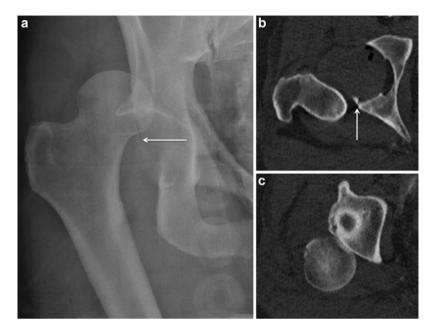


Fig. 15.3 (a) AP radiograph of the right hip demonstrating a traumatic hip dislocation in the characteristic posterior and superior location. An *arrow* highlights the associated posterior acetabular wall fracture. (b) and (c) demonstrate axial CT images of the same hip demonstrating the dislocation and the associated posterior acetabular wall fracture. Because of the small size of the fracture fragment, the fracture was able to be treated non-operatively after concentric reduction of the hip

be given to referral to a facility with experience and personnel to definitively treat both the dislocation and any associated injuries. After reduction, advanced imaging, including magnetic resonance imaging (MRI) and/or CT, should be used to verify a concentric reduction, assess for occult fractures, evaluate for any loose bodies incarcerated within the joint, and define associated chondral or labral injuries. A high rate of intra-articular pathology has been documented after traumatic hip dislocations. Fractures are typically treated with open procedures, while loose bodies and chondral or labral injuries are amenable to arthroscopic treatment [9, 10]. Follow-up MRI at 3 months is recommended by some to detect early signs of osteonecrosis [8].

Reduced hips without associated fractures have typically been treated with immobilization in young children for 3–4 weeks and weight-bearing restrictions in older patients for 6–12 weeks. Good outcomes have been observed in children, however older athletes may have more difficulty returning to high-level sports. Patients with traumatic hip dislocations are more likely to have pre-existing impingement morphology and increased acetabular retroversion, which may predispose them to a posterior dislocation event and could make recovery after a dislocation more difficult [11].

15.3.2 Hip Instability

Recognition of hip instability as a cause of hip pain and dysfunction has grown over the last several years. Instability of the hip can be classified as either traumatic or atraumatic. Traumatic hip instability results from an acute event that damages the osseous or soft-tissue structures of the hip leading to a transient loss of concentric femoroacetabular reduction. An initial traumatic instability event may lead to persistent subacute subluxation or micro-instability episodes due to capsular attenuation or compromise of the dynamic periarticular stabilizers. If a trial of conservative treatment does not resolve instability symptoms, surgical intervention may be necessary to address the offending pathology.

Surgical treatment may be directed at contributions to instability from bony architecture, the hip joint capsule, or other soft-tissue structures in any combination. As arthroscopic hip surgery continues to grow in popularity, it is important to recognize that instability can also be the result of an overzealous capsulotomy performed during prior hip arthroscopy. While some postulate that strategic capsulotomy or capsulectomy may be a therapeutic treatment for hip stiffness, intra-operative capsulotomies disrupt important capsuloligamentous stabilizers. In recent years, a more judicious approach towards capsular management has been adopted and capsular repair has gained support. Various capsular repair methods have been described, but no consensus exists regarding their efficacy or necessity [12].

Atraumatic instability is believed to result from repetitive microtrauma, which eventually leads to capsular attenuation and chondrolabral pathology [12]. Posterior instability in an athlete has also been shown to demonstrate a strong relationship with FAI morphology. According to this proposed mechanism, anatomic conflict between the femoral head/neck junction and the acetabulum may cause the femoral head to lever posteriorly out of the acetabulum [13, 14]. The joint may also be susceptible to atraumatic instability due to acetabular dysplasia (DDH), Legg-Calvé-Perthes disease, or systemic conditions that result in ligamentous laxity such as Marfans or Ehlers-Danlos syndromes, as well as patients who have undergone bariatric surgery [15]. Treatment is highly dependent on the specific pathology, though it is important to understand the limitations and dangers of arthroscopic treatment for patients with dysplastic morphology. Surgical options to address capsular laxity include thermal capsulorraphy or suture plication [16]. Bony abnormalities, such as FAI or DDH, are treated with targeted decompression of the offending structures or rotational osteotomy to provide further structural support. Hip arthroscopy alone is contraindicated in patients with significant DDH.

15.3.3 Hip Fractures

Proximal femoral fractures are rare in sports and usually are associated with highenergy trauma. Femoral neck fractures represent a surgical emergency and may have catastrophic long-term consequences if not treated in a timely manner. Concomitant injury to the tenuous vascular supply increases the risk of femoral head osteonecrosis. The causal relationship between time to reduction and osteonecrosis is less established than what is seen with traumatic hip dislocations. Furthermore, the type of fracture and quality of reduction may also have important prognostic implications for an athlete's long-term outcome. Capsular decompression after reduction and fixation has been advocated to decrease intra-articular pressure on the femoral head vascular supply [17]. Surgical stabilization is the mainstay of treatment for traumatic hip fractures, and a variety of fixation methods can be employed, depending on the specific fracture pattern and associated injuries. Typically, femoral neck fractures are stabilized with either multiple cannulated screws placed from lateral to medial across the fracture site or with a sliding hip screw and side-plate construct [18]. More distally, fractures of the intertrochanteric and subtrochanteric region can be treated with a variety of implants including sliding hip screws, cephalomedullary nails, and plate and screw constructs depending on fracture characteristics and surgeon preference.

15.3.4 Stress Fractures

In contrast to traumatic fractures, stress or fatigue fractures are overuse injuries. These are typically encountered among athletes who increase their training regimen, especially runners. Military personnel are also at risk due to the intensity of their conditioning. Appropriate imaging can be obtained to accurately confirm a suspected diagnosis. While many lesions may not be apparent on plain radiographs, MRI offers high sensitivity for stress fractures or stress reactions in addition to anatomic specificity [19]. MRI findings may include edema within the bone or a distinct fracture line. Certain stress fractures, including tension-sided fractures of the femoral neck, are at a higher risk for progression or displacement and may be treated more aggressively [20]. Lower risk stress fractures, such as those of the pubic ramus, can be treated with activity modifications. The clinician should be aware of and address contributing factors such as leg-length discrepancies or malalignment, femoroacetabular impingement, or acetabular dysplasia, which may alter hip joint arthrokinematics and place excess stress on adjacent structures. It is important to explore and treat any underlying metabolic bone disorders, patterns of nutritional insufficiency, and systemic medical disorders, as these may predispose athletes to stress fractures [21]. The female athlete triad represents a common spectrum of metabolic and nutritional compromise that can occur in the presence of overtraining, placing an athlete at an increased risk for injuries of attrition.

Missed femoral neck fractures can lead to disastrous consequences if not treated appropriately and in a timely fashion. Thus, the index for suspicion for these injuries should be high and the threshold for obtaining advanced diagnostic imaging should be low [22]. Diagnosis typically includes the use of radiographs and MRI. Multiple classification systems have been described, most of which discriminate between compression and tension-sided lesions and displaced fractures. Displaced fractures are generally a surgical emergency. These are treated with anatomic reduction followed by fixation with either cannulated lag screws or a sliding hip screw [23]. Non-displaced compression-sided fractures (found on the medial or inferior aspect of the femoral neck) are more inherently stable and may be treated with limited weight-bearing for 6-12 weeks if they are less than 50% of the width of the neck. Fracture line extension beyond 50% of the width of the neck warrants consideration for internal fixation. Most authors advocate fixation of tension-sided injuries, however successful conservative treatment with close follow-up has been reported [24]. Failure of non-operative treatment is an indication for operative intervention (Fig. 15.4). For non-displaced fractures, surgical stabilization often includes percutaneous placement of two or three cannulated lag screws across the femoral neck or a sliding hip screw and side-plate construct followed by a period of restricted weight-bearing. Patients with compression-sided injuries treated operatively may be able to progress weight-bearing earlier or even immediately, depending on their relative stability [25]. Complications after surgical stabilization include those related to hardware irritation and periarticular soft-tissue disruption during fixation, therefore, an attempt at non-operative treatment is preferable when possible.

15.4 Soft-Tissue Injuries

15.4.1 Muscle Strain and Avulsion

Muscle strains of the hip and pelvis are among the most common injuries an athlete can sustain in this anatomic region. They often occur during eccentric contractions and are more common in muscles that cross two joints. They occur most frequently at the myotendinous junction, but are also seen in the muscle belly [1]. In addition to clinical examination, advanced imaging may be helpful in both determination of the diagnosis and the prognosis. Plain radiographs may detect avulsion fractures. MRI can offer further detail regarding the nature of the soft-tissue injury, and the degree of injury seen on MRI has been shown to correlate with time until return to activities [26].

Treatment initially consists of non-steroidal analgesics and local measures to reduce hemorrhage and edema, including ice, elevation, and compression. NSAIDs are also considered to prevent heterotopic ossification or myositis ossificans. Range of motion and then strengthening are progressively introduced as pain improves [1]. Occasionally, however, surgical intervention is required. Following failure of functional rehabilitation, patients with chronic pain from tears of the proximal rectus

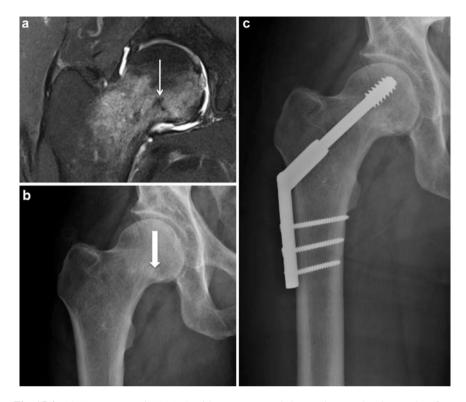


Fig. 15.4 (a) Fat-suppressed T1 MRA with contrast reveals bony edema and an incomplete fracture line in the compression side of the right femoral neck (*thin arrow*). (b) AP radiograph of the right hip from the same time point demonstrates subtle sclerosis in the femoral neck (*thick arrow*). (c) After a course of conservative treatment for the stress fracture failed, surgical fixation was undertaken utilizing a sliding hip screw and side-plate device

femoris musculotendinous junction have been shown to respond favorably to excision of their scar tissue [27]. Others have reported success after unilateral or bilateral adductor tenotomies for chronic groin pain from adductor tendinopathy in athletic populations [28–30]. Recently introduced non-surgical modalities such as instrumented or manual myofascial treatments or Active Release Technique (ART) have gained popularity for the treatment of periarticular myofascial scarring, connective tissue adhesions, or soft-tissue contractures that may develop as sequelae of muscular sprains or strains [31].

Avulsion fractures are less common in skeletally mature athletes. These occur in the same manner as their corresponding soft-tissue injury, and could occur at any muscular attachment with sufficient force. Displacement is limited by periosteum and surrounding fascia. These have been reported to occur with low energy trauma in patients with underlying malignancy, notably around the lesser trochanter [32]. Surgical treatment may be indicated when there is greater than 2 cm of displacement and the fragment is large enough for fixation, or if conservative management fails to allow healing [1].

Injuries to the proximal hamstring tendon complex have gained attention in recent years. These injuries commonly occur suddenly with the leg in a position of hip flexion and knee extension. Partial and full thickness injuries to any one of the three tendons of the proximal hamstring complex have been described. Surgical intervention is generally recommended for patients with an avulsion of at least two tendons, retraction greater than 2 cm, and for those with the desire to resume highdemand physical activity. Failure of conservative treatment may also constitute an appropriate indication for operative repair. A growing body of evidence now supports surgical repair for partial thickness, full thickness, and chronic full thickness tears [33]. The anatomic footprint of the proximal hamstring has been extensively cataloged in recent reports [34]. Advanced imaging, such as MRI, can be an invaluable tool to confirm the diagnosis of a proximal hamstring injury and grade the extent of the pathology. Surgical repair is conducted through an open longitudinal incision for a large retracted tear or chronic tear with nerve entrapment. When the tear is small or partial thickness, surgical repair may be accomplished through a transverse subgluteal incision, which is more cosmetic. Arthroscopic repair techniques have also been described and early reports reflect good and excellent outcomes with minimal soft-tissue morbidity [35]. Limited results reported in the literature have been generally good with regard to return to sport and strength for both full and partial thickness tears [36, 37].

15.5 Structural Pathology

15.5.1 Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is an increasingly recognized source of pain and functional impairment in active individuals. It is believed to result from morphologic alterations of the acetabulum and/or proximal femur (Fig. 15.5), which can lead to pathologic anatomic conflict, labral and chondral injuries, and synovitis. FAI has been causally linked to the progressive development of early onset osteoarthritis (OA) of the hip, though this is somewhat controversial [38].

Imaging is helpful for the diagnosis of FAI and for surgical planning, but it is important to note that FAI is a dynamic phenomenon and may occur in the absence of significant radiographic deformity in positions of extreme hip motions, such as those encountered during martial arts and dancing [39]. The presence of significant OA on weight-bearing radiographs is a poor prognostic indicator of clinical outcomes following arthroscopic hip surgery. While diagnoses can be made provisionally with plain radiographs, MRI and CT are more sensitive for uncovering more subtle abnormalities, gaining a more sound understanding of the global pathomorphology of the joint, and diagnosing associated pathologies prior to surgery [40].

Surgical intervention is indicated when symptomatic FAI does not respond to an appropriate period of rest, activity modifications, and non-steroidal anti-inflammatory medications. Thorough pre-operative workup should be conducted to fully

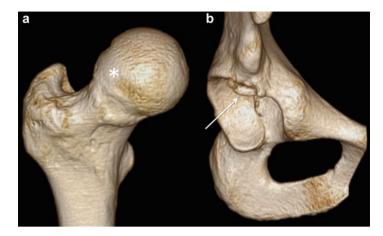


Fig. 15.5 (a) 3D CT reconstruction of the proximal femur showing anterosuperior "cam-type deformity" (*) with reduced femoral head–neck offset. (b) 3D CT reconstruction of the corresponding acetabulum demonstrating focal retroversion and overcoverage secondary to an acetabular rim fracture (*arrow*)

characterize the bony morphology of the joint as well as the degree of labral and chondral pathology that is present. Initially, open surgical hip dislocation was described to safely access symptomatic pathology within the central and peripheral aspects of the hip joint [41]. More recently, arthroscopic techniques have been increasingly utilized to address symptomatic FAI as well as synovial, labral, and chondral disease [3]. Bozic et al. reported a 600 % increase in the incidence of hip arthroscopies performed by orthopedic surgeons undergoing board certification between 2006 and 2010 [42], while Colvin et al. found an 18-fold increase in the number of hip arthroscopies performed during a study period of 1999 to 2009 [43]. Though preferable to many surgeons, it is important to recognize the limitations of arthroscopy. Cam deformities are typically most severe at the anterosuperior headneck junction of the proximal femur, which is readily accessible by arthroscopic means. Lesions that extend posteriorly approach the lateral retinacular vessels of the posterolateral neck, and thus they are less accessible and the dissection is more dangerous. Complex lesions, such as deformities from pre-existing Legg-Calvé-Perthes or slipped capital femoral epiphysis (SCFE), may also be difficult to adequately treat arthroscopically. Often these more global pathologies, rotational deformities, or abnormalities of version need to be treated concomitantly and require a more aggressive open approach [44].

Regardless of whether an open or arthroscopic approach is used to access the joint, goals for thorough FAI treatment involve bony decompression and stabilization of associated labral and chondral injuries. Treatment typically involves some combination of a femoral head–neck osteochondroplasty, acetabular rim osteoplasty, labral repair or debridement, and acetabular chondroplasty. Less frequently, an acetabular osteotomy may be indicated for more significant global acetabular retroversion or volume deficiency. Dynamic examination under fluoroscopic or arthroscopic

visualization can be used to confirm appropriate resection of bony deformities prior to the conclusion of the procedure [3, 45].

Outcomes following both open and arthroscopic hip surgery are generally favorable [46, 47]. Factors associated with improved outcomes include the absence of significant OA [48], labral repair versus debridement [49], and proper recognition and complete treatment of associated bony pathology [50]. In the absence of significant OA, age should not be a deterrent to arthroscopy [51]. Arthroscopy should be avoided in patients with evidence of dysplasia, as surgery may lead to iatrogenic instability and accelerate degenerative progression in certain cases [52].

15.5.2 Acetabular Labral Injuries

The acetabular labrum is a fibrocartilaginous ring that surrounds the bony acetabulum. It deepens the hip socket, maintains the intra-articular fluid-film layer, and creates a negative pressure seal within the joint. Free nerve endings have been found throughout the labrum, but are most densely located at the anterosuperior aspect [53].

Acetabular labral injuries can occur with or without underlying bony abnormalities, such as FAI. Labral pathology rarely results from acute injuries, including major trauma as well as minor twisting events or falls. More commonly, labral damage occurs as part of a global attritional process, such as OA. Labral injuries tend to result from a shearing mechanism and are typically oriented perpendicular to the articular surface at the chondrolabral junction or within the substance of the labral tissue in cases of degenerative pathology. They are often associated with adjacent acetabular chondral lesions [3], and most pathology is found in the anterosuperior quadrant [54]. When a labral tear is suspected, confirmatory MRI or MRA can be used to verify the diagnosis (Fig. 15.6). The addition of arthrography may enhance visualization of labral or chondral pathology and aid in pre-operative planning [55]. Ultrasonography can be used to detect labral pathology, paralabral cysts, and adjacent injuries within the periarticular soft tissues and may present an alternative imaging modality to aid in formulating an accurate diagnosis.

The natural history of labral pathology is poorly defined, and a course of nonoperative treatment is usually initiated before considering more invasive options. A high rate of chondral and labral pathology has been demonstrated in populations of asymptomatic volunteer subjects, strengthening the importance of an appropriate course of non-operative treatment prior to considering surgical intervention [56]. Conservative treatment includes some combination of activity modification, physical therapy, and anti-inflammatory medications. Intra-articular anesthetic or antiinflammatory injections are commonly used for pain mapping and to improve diagnostic accuracy. A positive response to an intra-articular injection may have prognostic importance when used to predict outcomes following arthroscopic hip surgery [57, 58]. Intra-articular cortisone injections may be limited long-term benefit, however, when employed in a therapeutic role [59].

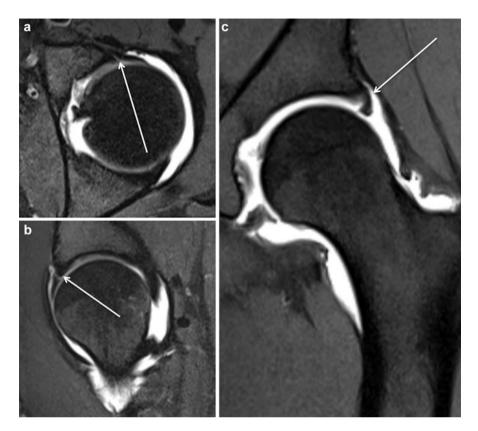


Fig. 15.6 Axial (**a**), sagittal (**b**), and coronal (**c**) T1 fat-suppressed MR arthrograms of the right hip demonstrate an anterosuperior labral tear with separation of the chondrolabral junction, highlighted by *arrows*

Hip arthroscopy is becoming a popular tool to access the joint and treat labral, chondral, or synovial pathology (Fig. 15.7). After appropriate arthroscopic visualization, the labrum is debrided of any nonviable tissue using arthroscopic shavers, biters, or radiofrequency ablation. Any remaining tissue that can be repaired is reattached to the underlying bone using suture anchors. Prior to suture repair, the acetabular rim is often freshened or trimmed to remove any areas of focal overcoverage and expose a bleeding bone surface, which may optimize the biologic healing response. Sutures may be passed through the labrum in a mattress fashion or around the damaged tissue in a looped configuration [3]. The arthroscopic treatment of labral pathology has been shown to yield favorable and durable results, however the presence of significant OA has been shown to be a poor prognostic factor and increases the chance of conversion to arthroplasty [60].

Labral reconstruction is utilized when severe injury or previous debridement has left the acetabular rim devoid of a functioning labrum. Various graft choices,

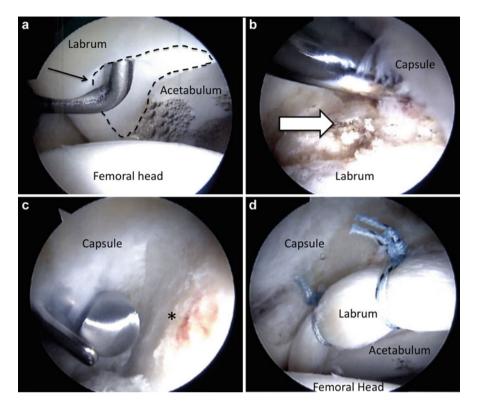


Fig. 15.7 Intra-operative arthroscopic imaging of the left hip demonstrating (a) a prominent "wave sign," (*dashed line*) where chondral delamination has occurred, as well as chondral-labral separation of the anterosuperior aspect of the acetabulum (*arrow*). (b) Acetabular rim prominence (*thick arrow*), leading to synovitis and labral injury (c) Arthroscopic image depicting decompression of the acetabular sided deformity (*) using an arthroscopic burr. (d) Decompression is followed by arthroscopic repair of the labral tear using suture anchors. Completed acetabuloplasty is shown superior to the labral repair

including allograft and autograft, have been reported in the literature [61]. Both open and arthroscopic methods are described [62]. Biomechanical [63]. and short-term functional results [61, 64]. show promise, but long-term follow-up has yet to be reported.

15.5.3 Extra-articular Hip Impingement

The concept of femoroacetabular impingement has evolved to include patterns of bony impingement that occur between anatomic regions outside the intra-articular space. The so-called extra-articular impingement can cause pain and loss of function in athletes. In contrast to intra-articular impingement, extra-articular pathology encompasses a number of much less common conditions, and treatment strategies are evolving. Abnormal bony contact and soft-tissue compression are responsible for symptoms. Various etiologies include psoas impingement, subspine impingement, ischiofemoral impingement, and greater trochanter/pelvic impingement [65].

Psoas impingement (PI) involves atypical labral wear in the direct anterior position secondary to compression or traction from the adjacent iliopsoas tendon. These distinct labral tears are found anterior and inferior (3 o'clock or 9 o'clock) to the typical location caused by intra-articular impingement. This pattern of impingement may cause anterior hip pain over the iliopsoas at the level of the anterior joint line and pain with hip flexion. Initial treatment may include activity modifications and focused rehabilitation. Refractory symptoms can be treated with image guided iliopsoas injections or arthroscopic fractional lengthening of the iliopsoas tendon along with appropriate labral and chondral treatment [66].

Subspine impingement (SSI) occurs when there is abnormal contact between the distal femoral neck and the undersurface of the anterior inferior iliac spine (AIIS). Prominence of the AIIS can be secondary to prior apophyseal or rectus avulsions, periacetabular osteotomies, acetabular retroversion, or developmental variants. SSI typically manifests as pain with hip flexion, limited hip flexion range of motion, and pain directly over the AIIS. Failure of conservative management, including rest and injections, can be treated with arthroscopic subspine decompression, which has been shown to improve both pain and functional symptoms [67].

Ischiofemoral impingement (IFI) is a less commonly reported form of extraarticular impingement and is caused by static or dynamic narrowing of the area between the ischial tuberosity or posterior acetabular rim laterally and the lesser trochanter or posterior greater trochanter medially. It can be associated with the sequelae of Perthes disease or otherwise morphologically abnormal hips. Surgical treatment involves open resection of the often-prominent lesser trochanter [68].

Greater trochanteric/pelvic impingement (GTPI) results from abnormal contact between the greater trochanter and the ilium, and is also often associated with abnormal hip morphology. Pain occurs with the hip in abduction and extension. Numerous open surgical treatments including osteotomies of the greater trochanter and acetabulum, rotational osteotomy, or arthroscopic decompression have been described [65].

15.5.4 Coxa Saltans

Coxa saltans, or "snapping hip," is an audible and potentially painful snapping about the hip during motion. It has multiple distinct etiologies. External snapping hip is caused by pathologic thickening or contracture of the posterior one-third of the iliotibial (IT) band or from pathologic translation of the gluteus maximus over the posterolateral border of the greater trochanter. Internal snapping hip results from translation of the psoas tendon over the femoral head, iliopectineal eminence, or the AIIS. Intra-articular snapping can be caused by loose bodies or labral pathology [69]. Treatment for coxa saltans depends on the severity and duration of symptoms. Most cases are minimally symptomatic, especially in athletic populations who can develop snapping due to the demands of repetitive training. Activity modification, anti-inflammatory medications, local injections, and focused hip and pelvic rehabilitation are effective strategies for most patients. When these measures fail, surgical intervention is indicated. Mechanical symptoms originating from an intra-articular source are typically due to an unstable labral tear or chondral flap. Arthroscopy is an effective tool used to address the offending pathology, which is typically due to an unstable labral tear or chondral flap [70].

Internal snapping hip can be treated by lengthening or release of the iliopsoas tendon. Traditionally, an open approach was described to lengthen the tendinous portion using multiple transverse incisions. Lengthening can be made at the level of the femoral head or the pelvic brim [69]. Arthroscopic techniques have become more popular with technical evolutions in hip arthroscopy [71]. Arthroscopic fractional lengthening can be performed at the level of the lesser trochanter using fluoroscopic assistance, or through a transcapsular window under direct arthroscopic visualization inside the joint. Arthroscopic lengthening offers the potential advantages of a less invasive approach and fewer complications compared with open techniques [72].

External snapping is typically treated with open or endoscopic trochanteric bursectomy with fractional lengthening of the thickened portion of the IT band or gluteus maximus. Multiple lengthening procedures have been described, including open Z-plasties, transverse releases, cruciate incisions, and tenodesis procedures [73, 74]. Attention should be paid to abnormal morphology or malalignment, such as coxa vara, which may predispose patients to this condition [75]. More recently, endoscopic techniques have emerged in the literature with promising short-term results. Endoscopic options utilize various strategies to relax or release the contracted portion of the IT band or gluteus maximus tendon [76, 77].

15.6 Inflammatory and Compression Syndromes

15.6.1 Greater Trochanteric Pain Syndrome

Reproducible tenderness over the greater trochanter can be due to numerous pathologies of the peritrochanteric space including trochanteric bursitis, abductor tearing, external coxa saltans, or even referred intra-articular pain [78]. The gluteus medius and minimus, which comprise the majority of the hip abductor complex, have been described as the "rotator cuff of the hip." Inflammation or scarring of the overlying trochanteric bursa can also lead to chronic pain and dysfunction. Much like the shoulder, there is a high prevalence of asymptomatic tendinopathy and tearing that advance with age [3, 79]. Both MRI and musculoskeletal ultrasonography (MSUS) are highly accurate imaging modalities to characterize the extent of abductor disease [80, 81].

15 Surgical Interventions in Hip and Pelvis Injuries

Conservative treatment is successful in most cases, but when non-operative measures fail to provide durable symptomatic relief, recalcitrant lateral hip pain may be amenable to operative intervention. Multiple treatment options for trochanteric bursitis exist. Most involve debridement of the bursa with or without IT band lengthening [82]. Arthroscopic bursectomy appears to be effective in the short term as well, though comparative studies are lacking [83]. Refractory lateral hip pain that fails to improve with conservative care or targeted peritrochanteric injections should also raise suspicion for occult intra-articular hip pathology. Because of the variable anatomic presentation of intra-articular pathology, a thorough clinical and diagnostic workup should be conducted before considering surgical intervention in order to clarify any potential contribution of the intra-articular joint space to the overall pain spectrum [84]. The treating clinician should be aware of the potential contribution of structural malalignment of the proximal femur or acetabulum in cases of refractory peritrochanteric pain. Fatigue overload of the abductor complex due to edge loading or abnormal arthrokinematics may manifest as dynamic lateral hip pain that does not respond to traditional conservative interventions.

Both partial and full thickness tears of the abductor complex have been described. Traditionally, an open approach utilizing bone tunnels was favored to achieve a successful repair [79]. More recently, endoscopic suture anchor repair techniques have been adopted and popularized. Good to excellent functional results have been reported for both techniques, but endoscopic repair may offer the advantage of fewer complications [85, 86].

15.6.2 Athletic Pubalgia

Athletic pubalgia, or "sports hernia," has recently been characterized as "central core dysfunction." This pathologic entity is a poorly understood spectrum of conditions affecting the posterior inguinal wall, central pelvic and soft tissues structures, and surrounding sensory nerves. It typically affects athletes who require repetitive overuse of the proximal thigh and lower abdominal musculature, including those who participate in soccer and ice hockey. Injuries to various structures, including the transversalis fascia, conjoined tendon, rectus abdominus insertion, and internal and external oblique musculature result in dilation and weakening of the internal inguinal ring. An insidious onset of deep groin pain with activity and prolonged course are common. There is no clinically detectable hernia, though local tenderness is commonly found. Imaging is nonspecific, but is helpful for ruling out other pathology [87].

If symptomatic athletic pubalgia does not respond favorably to a conservative treatment strategy, surgical intervention may be indicated [88]. After ruling out other associated pathologies, such as abdominal visceral disorders, surgical repair with either a conventional open or laparoscopic approach may be considered. Various open repair techniques have been described with a combination of procedures. Most involve repair of the posterior inguinal canal, often with mesh [89].

Concomitant procedures include repair of the external oblique aponeurosis, Bassini repair, plication of the transversalis fascia [90], pelvic floor repair [91], open external ring repair, and conjoined tendon repair [92]. Successful return to play has been reported in between 77 % and 97 % of athletes, with mostly excellent outcomes [93, 94]. Adductor tenotomy or repair has also been advocated as an adjunctive option and similar positive results have been documented in the literature [93, 95, 96]. When compared with open techniques, laparoscopic groin repair has been shown to demonstrate equivalent functional outcomes with more rapid return to play and durable results [97–99].

Gilmore's groin is a variant of a sports hernia that includes tearing of the external oblique aponeurosis, tearing of the conjoined tendon, dehiscence between the conjoined tendon and inguinal ligament, and a dilated superficial inguinal ring. It is commonly encountered in soccer players. A 97% success rate has been reported for surgical repair in high-level athletes [100].

Recently, clinicians have begun to recognize a causal link between athletic pubalgia and FAI. Mechanical conflict between the proximal femur and acetabulum is thought to impart mechanical stress on both the hip joint and the surrounding pelvic ring. The demands of functional activity then transfer these stresses to adjacent regions of the pelvis, including the pelvic brim, pubic symphysis, and sacroiliac joint [101]. Treatment of both conditions has been associated with superior outcomes and a higher rate of return to play compared with isolated treatment of either pathology [102, 103].

15.6.3 Osteitis Pubis

Osteitis pubis is a painful condition of the pubic symphysis and parasymphyseal bone. Anterior and medial groin pain is exacerbated by activity, particularly kicking, jumping, twisting, or impact loading. Mechanical overload of the symphysis from the abdominal and adductor muscles is believed to cause a local stress reaction at the pubic symphysis with surrounding soft-tissue inflammation [104]. Osteitis pubis is exacerbated by pregnancy and other conditions that lead to ligamentous laxity, including post-traumatic, post-bariatric surgery, or rheumatologic afflictions [1]. Plain radiographs characteristically demonstrate sclerosis, resorption, and symphyseal widening, while a bone scan often reveals increased activity at the symphysis. MRI may offer more detail or rule out other associated pathology, but has been shown to demonstrate a high rate of incidental parasymphyseal pathology in asymptomatic athletes [105].

Treatment of osteitis pubis includes a period of relative rest, anti-inflammatory medications, and rehabilitation focused on muscles acting across the pelvis. Corticosteroid injections can be helpful, especially if given within several weeks of symptom onset [106]. Several surgical options exist, including curettage of the symphysis [107], preperitoneal polypropylene mesh placement [105], and symphyseal stabilization and fusion [108]. No comparative studies exist, and overall return to

play is 80% in an average of 4–5 months after surgery [109]. Planning treatment for this condition should account for other local factors that may be contributing to increased mechanical stress across the symphysis, such as adjacent FAI.

15.6.4 Nerve Compression

Peripheral nerve entrapment or compression is an uncommon source of pain in athletic populations, but can occur after trauma or local surgical procedures. Obturator neuropathy has been reported as a fascial entrapment of the obturator nerve as it enters the thigh in athletes with over-developed adductors. This exercise-induced medial thigh pain diagnosed on electromyography (EMG) is treated with neurolysis, which results in a return to play within several weeks [110]. Genitofemoral neuropathy causes groin pain and paresthesias in the lower abdomen to medial thigh, including the genitals. Radiofrequency ablation and ultrasound guided cryoablation can be tried before surgical neurectomy [111]. Meralgia paresthetica is characterized by numbness, paresthesias, and pain in the anterolateral thigh from entrapment or a neuroma of the lateral femoral cutaneous nerve. Iatrogenic injury from local procedures or surgical positioning is a common cause. The diagnosis can be made with EMG or diagnostic local anesthetic injections. Treatment options include medial transposition or transection, which is more definitive but does result in permanent anesthesia [112].

Piriformis syndrome is caused by compression of the sciatic nerve as it courses around or through the piriformis muscle, which can lead to hip and buttock pain as well as radiculopathy. No consensus diagnostic criteria or methods exist, and some question its existence despite its presence in the literature. Therapy directed at stretching and injections are the mainstays of treatment. Myofascial techniques can also be effective for deep gluteal contractures and muscular imbalance. Rare refractory cases are treated with surgical exploration of the nerve and release of the piriformis with mixed results [113].

15.7 Pediatrics

15.7.1 Apophyseal Avulsions

Apophyseal avulsions occur in skeletally immature athletes after the appearance of the apophysis but before physeal closure, typically between the ages of 14 and 25. They present in a similar fashion to adult muscle strains or avulsions as a result of forceful muscular contractions. The ischium, anterior inferior iliac spine, and the anterior superior iliac spine are the most frequent sites of injury; less commonly, athletes may sustain avulsion injuries at the adductor tubercle, greater or lesser trochanter, or iliac crest [114]. Plain radiographs are diagnostic in most cases, however

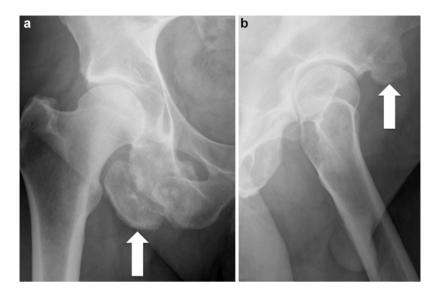


Fig. 15.8 (a) AP radiograph of the right hip demonstrating extensive heterotopic ossification at the origin of the hamstring tendon complex (*arrow*), likely from a chronic avulsion injury. (b) Lateral false-profile radiograph of the left hip demonstrating prominence of the left anterior inferior iliac spine (AIIS *- arrow*), likely from a remote avulsion injury. This bony prominence may lead to subspine impingement between the proximal femur and the AIIS

CT demonstrates optimal bony detail for more subtle injuries at the cost of increased radiation exposure. MRI may aid in the diagnosis for ossification centers that have yet to ossify [115]. Musculoskeletal ultrasound (MSUS) has also shown promise as a diagnostic option [116].

Treatment generally involves protected weight-bearing followed by functional rehabilitation and resumption of normal activity in 6–8 weeks [117]. Surgery is generally reserved for cases of symptomatic nonunions, painful exostoses, and when displacement is greater than 2 cm in athletes [115]. Fixation of bony ischial avulsions may prevent long-term sequelae of nonunion with painful heterotopic bone formation and "hamstring syndrome" (Fig. 15.8). Some authors advocate fixation of greater trochanteric avulsions, given the importance of the abductor complex [118]. Osteonecrosis of the femoral head has been reported with and without fixation of these injuries [119]. Fixation is generally carried out with screws, and data on rapid to return to play is limited but promising [115].

15.7.2 Slipped Capital Femoral Epiphysis

Slipped capital femoral epiphysis (SCFE) is one of the most severe hip disorders of adolescence. It is characterized by anterosuperior displacement of the metaphysis in relation to the femoral head through the zone of hypertrophy of the proximal femoral

Fig. 15.9 Severe right hip SCFE treated with *in situ* fixation as well as prophylactic fixation on the left side. A residual deformity leads to symptomatic intra and extra-articular impingement in this case



physis. It has been related to obesity, endocrinopathies, and African American race among other factors. It typically presents with groin, thigh, or knee pain, and is diagnosed on plain radiographs. A majority of cases are chronic, though some will present acutely with less than several weeks of symptoms or significant pain. Slips are considered stable if the patient is able to ambulate, and unstable if they are not [120].

Treatment of stable SCFE has traditionally consisted of surgical fixation with a single screw placed percutaneously through the metaphysis perpendicular to the epiphysis (Fig. 15.9). Though there is potential for remodeling after fixation of mild slips, there is increased concern for the resultant deformity from larger slips, which can cause symptomatic impingement [121]. Severe deformities can be corrected after the slip has healed with an intertrochanteric osteotomy that flexes, internally rotates, and abducts the distal fragment to correct for impingement and prevent OA [122]. Open surgical dislocation with femoral neck osteoplasty has also been reported with good results, but is technically demanding [48]. Mild deformities may be amenable to arthroscopic treatment [123].

Recently the modified Dunn procedure has been described as a surgical option for acute SCFE. This procedure involves a careful dissection of the femoral head vascular supply and an acute reduction of an unstable SCFE via an open surgical dislocation. Some studies have shown superior clinical results and more anatomic healing compared with *in situ* pinning [124]. Other series have demonstrated an unacceptably high rate of complications, such as osteonecrosis, using this approach [125]. In experienced hands, this procedure may minimize the long-term sequelae of high-grade SCFE. Further studies, with an emphasis on long-term outcomes and complications, will be necessary prior to widespread endorsement.

Patients with an unstable SCFE are at a much higher risk for developing osteonecrosis compared with patients with stable slips. In a survey of pediatric orthopedists, the majority favored emergent or urgent treatment with an "incidental reduction," conducted while positioning the patient under anesthesia, followed by fixation with one or two cannulated screws. There is no clear consensus regarding the need for capsular decompression to protect blood flow to the femoral head [126]. Osteonecrosis is a rare but catastrophic complication of SCFE, and has been associated with an unstable SCFE, over-reduction of unstable SCFE, reduction of stable SCFE, and pin placement in the posterosuperior quadrant of the epiphysis [120].

Patients who suffer a unilateral unstable SCFE have a higher incidence of subsequently suffering a contralateral SCFE. The odds are increased in patients who are younger and have endocrinopathies and other risk factors. In the case of highrisk patients, some advocate contralateral prophylactic fixation with a single screw [120].

15.8 Tumor/Other

Bone and soft-tissue tumors that mimic athletic injuries are relatively rare. Pain that occurs at night, is unrelated to physical activity, and is unresponsive to conservative measures should raise suspicion. The presence of a large, fixed mass on exam warrants further workup. Radiographic and advanced imaging modalities are invaluable in detection and classification of neoplasms. Appropriate treatment is highly variable and dependent on treatment, and the ability to make a prompt diagnosis may be life-saving [127].

Autoimmune conditions, rheumatologic disease, and connective tissue disorders also present with an uncharacteristic spectrum of clinical symptoms. Diffuse myalgias or arthralgias, visceral organ involvement, dermatologic manifestations, and/or a positive family history may reflect an underlying systemic pain source. An unusual history or clinical examination should alert the clinician to the possibility of these conditions and a thorough workup should be initiated when indicated.

15.9 Conclusion

Hip and pelvic pathology in the athlete can pose a substantial diagnostic challenge and require a thoughtful approach to optimize treatment. Fortunately, many injuries about the hip involve routine soft-tissue trauma and conservative treatment strategies should be applied when indicated. The treating clinician should exercise caution when interpreting advanced imaging studies in competitive athletes. Clinical suspicion for symptomatic hip and pelvic pathologies should be balanced with caution, as abnormal imaging findings in athletes are common and may not always necessitate intervention. While symptomatic hip and pelvic pathology traditionally required invasive open surgery, technical innovations have led to advances in minimally invasive hip surgery that offer the potential for faster recovery and less softtissue morbidity.

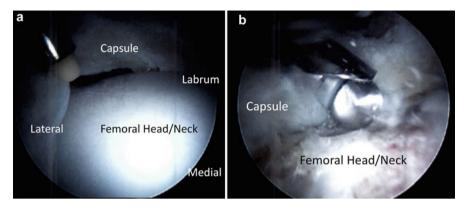


Fig. 15.10 Intra-operative arthroscopic photographs demonstrating a cam deformity of the femoral head/neck junction before (a) and during (b) decompression using an arthroscopic burr

15.10 Case Presentation (Continued)

15.10.1 Intervention

The patient underwent left hip arthroscopy. Intra-operative findings included mixed-type FAI with a complex intra-substance labral tear in the anterosuperior quadrant. He was treated with acetabuloplasty, femoroplasty, and labral repair (Fig. 15.10).

15.10.2 Follow-up

The patient experienced symptomatic improvement and was able to advance his functional activities under the guidance of a skilled physical therapist. A follow-up MRI completed 5 months post-operatively showed a resolution of his parasymphyseal edema and the patient was able to successfully return to full unrestricted athletic activity.

15.10.3 Decision Making

The patient presented with symptoms of early osteitis publes. He had failed conservative management consisting of rest and activity modifications. Advanced imaging, including MRI, MRA, and CT demonstrated mixed-type impingement morphology. By addressing his FAI, mechanical stresses on the pelvic ring were rebalanced and the stress reaction in the bone was able to finally heal.

References

- Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521–33.
- Frank JS, Gambacorta PL, Eisner EA. Hip pathology in the adolescent athlete. J Am Acad Orthop Surg. 2013;21(11):665–74.
- 3. Lynch TS, Terry MA, Bedi A, et al. Hip arthroscopic surgery: patient evaluation, current indications, and outcomes. Am J Sports Med. 2013;41(5):1174–89.
- 4. Flierl MA, Stahel PF, Hak DJ, et al. Traction table–related complications in orthopedic surgery. J Am Acad Orthop Surg. 2010;18(11):668–75.
- 5. Ross JR, Clohisy JC, Baca G, et al. Patient and disease characteristics associated with hip arthroscopy failure in acetabular dysplasia. J Arthroplasty. 2014;29(9 Suppl):160–3.
- Silvis ML, Mosher TJ, Smetana BS, et al. High prevalence of pelvic and Hip magnetic resonance imaging findings in asymptomatic collegiate and professional hockey players. Am J Sports Med. 2011;39(4):715–21.
- Mehlman CT, Hubbard GW, Crawford AH, et al. Traumatic hip dislocation in children. Long-term followup of 42 patients. Clin Orthop Relat Res. 2000;376:68–79.
- Herrera-Soto JA, Price CT. Traumatic Hip dislocations in children and adolescents: pitfalls and complications. J Am Acad Orthop Surg. 2009;17(1):15–21.
- 9. Philippon MJ, Kuppersmith DA, Wolff AB, et al. Arthroscopic findings following traumatic hip dislocation in 14 professional athletes. Arthroscopy. 2009;25(2):169–74.
- Ilizaliturri VM, Gonzalez-Gutierrez B, Gonzalez-Ugalde H, et al. Hip arthroscopy after traumatic hip dislocation. Am J Sports Med. 2011;39(1 suppl):50S–7.
- Steppacher SD, Albers CE, Siebenrock KA, et al. Femoroacetabular impingement predisposes to traumatic posterior hip dislocation. Clin Orthop Relat Res. 2013;471(6):1937–43.
- Domb BG, Philippon MJ, Giordano BD. Arthroscopic capsulotomy, capsular repair, and capsular plication of the hip: relation to atraumatic instability. Arthroscopy. 2013;29(1):162–73.
- 13. Krych AJ, Thompson M, Larson CM, et al. Is posterior hip instability associated with cam and pincer deformity? Clin Orthop Relat Res. 2012;470(12):3390–7.
- Berkes MB, Cross MB, Shindle MK, et al. Traumatic posterior hip instability and femoroacetabular impingement in athletes. Am J Orthop (Belle Mead NJ). 2012;41(4):166–71.
- Bjorkengren AG, Resnick D, Sartoris DJ. Enteropathic arthropathies. Radiol Clin North Am. 1987;25(1):189–98.
- 16. Boykin RE, Anz AW, Bushnell BD, et al. Hip instability. J Am Acad Orthop Surg. 2011;19(6):340-9.
- Boardman MJ, Herman MJ, Buck B, et al. Hip fractures in children. J Am Acad Orthop Surg. 2009;17(3):162–73.
- 18. Slobogean GP, Sprague SA, Scott T, et al. Management of young femoral neck fractures: is there a consensus? Injury. 2015;46(3):435–40.
- 19. Spitz DJ, Newberg AH. Imaging of stress fractures in the athlete. Radiol Clin North Am. 2002;40(2):313–31.
- Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. J Am Acad Orthop Surg. 2000;8(6):344–53.
- Teitz C, Hu S, Arendt E. The female athlete: evaluation and treatment of sports-related problems. J Am Acad Orthop Surg. 1997;5(2):87–96.
- 22. Visuri T, Vara A, Meurman KO. Displaced stress fractures of the femoral neck in young male adults: a report of twelve operative cases. J Trauma. 1988;28(11):1562–9.
- Lee CH, Huang GS, Chao KH, et al. Surgical treatment of displaced stress fractures of the femoral neck in military recruits: a report of 42 cases. Arch Orthop Trauma Surg. 2003;123(10):527–33.
- Aro H, Dahlstrom S. Conservative management of distraction-type stress fractures of the femoral neck. J Bone Joint Surg Br. 1986;68(1):65–7.
- Shin A, Gillingham B. Fatigue fractures of the femoral neck in athletes. J Am Acad Orthop Surg. 1997;5(6):293–302.

- Pomeranz SJ, Heidt Jr RS. MR imaging in the prognostication of hamstring injury. Work in progress. Radiology. 1993;189(3):897–900.
- Hughes C, Hasselman CT, Best TM, et al. Incomplete, intrasubstance strain injuries of the rectus femoris muscle. Am J Sports Med. 1995;23(4):500–6.
- Martens MA, Hansen L, Mulier JC. Adductor tendinitis and musculus rectus abdominis tendopathy. Am J Sports Med. 1987;15(4):353–6.
- Maffulli N, Loppini M, Longo UG, et al. Bilateral mini-invasive adductor tenotomy for the management of chronic unilateral adductor longus tendinopathy in athletes. Am J Sports Med. 2012;40(8):1880–6.
- 30. Robertson IJ, Curran C, McCaffrey N, et al. Adductor tenotomy in the management of groin pain in athletes. Int J Sports Med. 2011;32(1):45–8.
- Robb A, Pajaczkowski J. Immediate effect on pain thresholds using active release technique on adductor strains: Pilot study. J Bodyw Mov Ther. 2011;15(1):57–62.
- 32. Afra R, Boardman DL, Kabo JM, et al. Avulsion fracture of the lesser trochanter as a result of a preliminary malignant tumor of bone. A report of four cases. J Bone Joint Surg Am. 1999;81(9):1299–304.
- Rust DA, Giveans MR, Stone RM, et al. Functional outcomes and return to sports after acute repair, chronic repair, and allograft reconstruction for proximal hamstring ruptures. Am J Sports Med. 2014;42(6):1377–83.
- Neuschwander TB, Benke MT, Gerhardt MB. Anatomic description of the origin of the proximal hamstring. Arthroscopy. 2015;31(8):1518–21.
- 35. Domb BG, Linder D, Sharp KG, et al. Endoscopic repair of proximal hamstring avulsion. Arthrosc Tech. 2013;2(1):e35–9.
- Cohen S, Bradley J. Acute proximal hamstring rupture. J Am Acad Orthop Surg. 2007;15(6):350–5.
- Bowman Jr KF, Cohen SB, Bradley JP. Operative management of partial-thickness tears of the proximal hamstring muscles in athletes. Am J Sports Med. 2013;41(6):1363–71.
- Sankar WN, Nevitt M, Parvizi J, et al. Femoroacetabular impingement: defining the condition andits role in the pathophysiology of osteoarthritis. J Am Acad Orthop Surg. 2013;21(suppl):S7–15.
- Matsuda DK. The case for cam surveillance: the arthroscopic detection of cam femoroacetabular impingement missed on preoperative imaging and its significance. Arthroscopy. 2011;27(6):870–6.
- Nepple JJ, Prather H, Trousdale RT, et al. Diagnostic imaging of femoroacetabular impingement. J Am Acad Orthop Surg. 2013;21(suppl):S20–6.
- 41. Ganz R, Gill TJ, Gautier E, et al. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
- Bozic KJ, Chan V, Valone 3rd FH, et al. Trends in hip arthroscopy utilization in the United States. J Arthroplasty. 2013;28(8 Suppl):140–3.
- 43. Colvin AC, Harrast J, Harner C. Trends in hip arthroscopy. J Bone Joint Surg Am. 2012;94(4), e23.
- Nepple JJ, Byrd JWT, Siebenrock KA, et al. Overview of treatment options, clinical results, and controversies in the management of femoroacetabular impingement. J Am Acad Orthop Surg. 2013;21(suppl):S53–8.
- 45. Clohisy JC, Baca G, Beaule PE, et al. Descriptive epidemiology of femoroacetabular impingement: a North American cohort of patients undergoing surgery. Am J Sports Med. 2013;41(6):1348–56.
- 46. Stevens MS, Legay DA, Glazebrook MA, et al. The evidence for hip arthroscopy: grading the current indications. Arthroscopy. 2010;26(10):1370–83.
- 47. Steppacher SD, Huemmer C, Schwab JM, et al. Surgical hip dislocation for treatment of femoroacetabular impingement: factors predicting 5-year survivorship. Clin Orthop Relat Res. 2014;472(1):337–48.
- Beck M, Leunig M, Parvizi J, et al. Anterior femoroacetabular impingement: part II. Midterm results of surgical treatment. Clin Orthop Relat Res. 2004;418:67–73.

- Larson CM, Giveans MR, Stone RM. Arthroscopic debridement versus refixation of the acetabular labrum associated with femoroacetabular impingement: mean 3.5-year follow-up. Am J Sports Med. 2012;40(5):1015–21.
- Bardakos NV, Vasconcelos JC, Villar RN. Early outcome of hip arthroscopy for femoroacetabular impingement: the role of femoral osteoplasty in symptomatic improvement. J Bone Joint Surg Br. 2008;90(12):1570–5.
- Domb BG, Linder D, Finley Z, et al. Outcomes of hip arthroscopy in patients aged 50 years or older compared with a matched-pair control of patients aged 30 years or younger. Arthroscopy. 2015;31(2):231–8.
- 52. Parvizi J, Bican O, Bender B, et al. Arthroscopy for labral tears in patients with developmental dysplasia of the hip: a cautionary note. J Arthroplasty. 2009;24(6 Suppl):110–3.
- 53. Narvani AA, Tsiridis E, Tai CC, et al. Acetabular labrum and its tears. Br J Sports Med. 2003;37(3):207–11.
- Seldes RM, Tan V, Hunt J, et al. Anatomy, histologic features, and vascularity of the adult acetabular labrum. Clin Orthop Relat Res. 2001;382:232–40.
- 55. Chan YS, Lien LC, Hsu HL, et al. Evaluating hip labral tears using magnetic resonance arthrography: a prospective study comparing hip arthroscopy and magnetic resonance arthrography diagnosis. Arthroscopy. 2005;21(10):1250.
- 56. Lee AJ, Armour P, Thind D, et al. The prevalence of acetabular labral tears and associated pathology in a young asymptomatic population. Bone Joint J. 2015;97-B(5):623–7.
- Kivlan BR, Martin RL, Sekiya JK. Response to diagnostic injection in patients with femoroacetabular impingement, labral tears, chondral lesions, and extra-articular pathology. Arthroscopy. 2011;27(5):619–27.
- Martin RL, Irrgang JJ, Sekiya JK. The diagnostic accuracy of a clinical examination in determining intra-articular hip pain for potential hip arthroscopy candidates. Arthroscopy. 2008;24(9):1013–8.
- 59. Krych AJ, Griffith TB, Hudgens JL, et al. Limited therapeutic benefits of intra-articular cortisone injection for patients with femoro-acetabular impingement and labral tear. Knee Surg Sports Traumatol Arthrosc. 2014;22(4):750–5.
- 60. Byrd JW, Jones KS. Hip arthroscopy for labral pathology: prospective analysis with 10-year follow-up. Arthroscopy. 2009;25(4):365–8.
- Ayeni OR, Alradwan H, de Sa D, et al. The hip labrum reconstruction: indications and outcomes--a systematic review. Knee Surg Sports Traumatol Arthrosc. 2014;22(4):737–43.
- Costa Rocha P, Klingenstein G, Ganz R, et al. Circumferential reconstruction of severe acetabular labral damage using hamstring allograft: surgical technique and case series. Hip Int. 2013;23 Suppl 9:S42–53.
- 63. Lee S, Wuerz TH, Shewman E, et al. Labral reconstruction with iliotibial band autografts and semitendinosus allografts improves hip joint contact area and contact pressure: an in vitro analysis. Am J Sports Med. 2015;43(1):98–104.
- 64. Boykin RE, Patterson D, Briggs KK, et al. Results of arthroscopic labral reconstruction of the hip in elite athletes. Am J Sports Med. 2013;41(10):2296–301.
- 65. de Sa D, Alradwan H, Cargnelli S, et al. Extra-articular hip impingement: a systematic review examining operative treatment of psoas, subspine, ischiofemoral, and greater trochanteric/ pelvic impingement. Arthroscopy. 2014;30(8):1026–41.
- 66. Domb BG, Shindle MK, McArthur B, et al. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. HSS J. 2011;7(2):145–50.
- 67. Hapa O, Bedi A, Gursan O, et al. Anatomic footprint of the direct head of the rectus femoris origin: cadaveric study and clinical series of hips after arthroscopic anterior inferior iliac spine/subspine decompression. Arthroscopy. 2013;29(12):1932–40.
- 68. Ganz R, Slongo T, Turchetto L, et al. The lesser trochanter as a cause of hip impingement: pathophysiology and treatment options. Hip Int. 2013;23 Suppl 9:S35–41.
- 69. Allen WC, Cope R. Coxa saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3(5):303–8.

- 15 Surgical Interventions in Hip and Pelvis Injuries
- Yamamoto Y, Hamada Y, Ide T, et al. Arthroscopic surgery to treat intra-articular type snapping hip. Arthroscopy. 2005;21(9):1120–5.
- Ilizaliturri Jr VM, Villalobos Jr FE, Chaidez PA, et al. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. Arthroscopy. 2005;21(11):1375–80.
- Khan M, Adamich J, Simunovic N, et al. Surgical management of internal snapping hip syndrome: a systematic review evaluating open and arthroscopic approaches. Arthroscopy. 2013;29(5):942–8.
- Provencher MT, Hofmeister EP, Muldoon MP. The surgical treatment of external coxa saltans (the snapping hip) by Z-plasty of the iliotibial band. Am J Sports Med. 2004;32(2):470–6.
- 74. Bessette MC, Kenney RJ, Geary MB, et al. A novel iliotibial band and gluteus maximus tenodesis for the treatment of external coxa saltas in a patient with Ehlers–Danlos syndrome. J Hip Preserv Surg. 2015;2(3):316–7. doi:10.1093/jhps/hnv050.
- 75. Larsen E, Johansen J. Snapping hip. Acta Orthop Scand. 1986;57(2):168-70.
- Ilizaliturri Jr VM, Martinez-Escalante FA, Chaidez PA, et al. Endoscopic iliotibial band release for external snapping hip syndrome. Arthroscopy. 2006;22(5):505–10.
- Polesello GC, Queiroz MC, Domb BG, et al. Surgical technique: endoscopic gluteus maximus tendon release for external snapping hip syndrome. Clin Orthop Relat Res. 2013;471(8):2471–6.
- Strauss EJ, Nho SJ, Kelly BT. Greater trochanteric pain syndrome. Sports Med Arthrosc. 2010;18(2):113–9.
- 79. Kagan A, 2nd. Rotator cuff tears of the hip. Clin Orthop Relat Res. 1999;(368):135-40.
- Lachiewicz PF. Abductor tendon tears of the hip: evaluation and management. J Am Acad Orthop Surg. 2011;19(7):385–91.
- Dawes AR, Seidenberg PH. Sonography of sports injuries of the hip. Sports Health. 2014;6(6):531–8.
- Lustenberger DP, Ng VY, Best TM, et al. Efficacy of treatment of trochanteric bursitis: a systematic review. Clin J Sport Med. 2011;21(5):447–53.
- Baker Jr CL, Massie RV, Hurt WG, et al. Arthroscopic bursectomy for recalcitrant trochanteric bursitis. Arthroscopy. 2007;23(8):827–32.
- Bessette MC, Olsen JR, Mann TR, et al. Intra-articular hip injections for lateral hip pain. J Hip Preserv Surg. 2014;1(2):71–6.
- Alpaugh K, Chilelli BJ, Xu S, et al. Outcomes after primary open or endoscopic abductor tendon repair in the hip: a systematic review of the literature. Arthroscopy. 2015;31(3):530–40.
- Domb BG, Botser I, Giordano BD. Outcomes of endoscopic gluteus medius repair with minimum 2-year follow-up. Am J Sports Med. 2013;41(5):988–97.
- 87. Farber AJ, Wilckens JH. Sports hernia: diagnosis and therapeutic approach. J Am Acad Orthop Surg. 2007;15(8):507–14.
- 88. LeBlanc KE, LeBlanc KA. Groin pain in athletes. Hernia. 2003;7(2):68-71.
- Kumar A, Doran J, Batt ME, et al. Results of inguinal canal repair in athletes with sports hernia. J R Coll Surg Edinb. 2002;47(3):561–5.
- Polglase AL, Frydman GM, Farmer KC. Inguinal surgery for debilitating chronic groin pain in athletes. Med J Aust. 1991;155(10):674–7.
- Meyers WC, Foley DP, Garrett WE, et al. Management of severe lower abdominal or inguinal pain in high-performance athletes. PAIN (Performing Athletes with Abdominal or Inguinal Neuromuscular Pain Study Group). Am J Sports Med. 2000;28(1):2–8.
- Hackney RG. The sports hernia: a cause of chronic groin pain. Br J Sports Med. 1993;27(1):58–62.
- Ahumada LA, Ashruf S, Espinosa-de-los-Monteros A, et al. Athletic pubalgia: definition and surgical treatment. Ann Plast Surg. 2005;55(4):393–6.
- 94. Jakoi A, O'Neill C, Damsgaard C, et al. Sports hernia in National Hockey League players: does surgery affect performance? Am J Sports Med. 2013;41(1):107–10.
- Van Der Donckt K, Steenbrugge F, Van Den Abbeele K, et al. Bassini's hernial repair and adductor longus tenotomy in the treatment of chronic groin pain in athletes. Acta Orthop Belg. 2003;69(1):35–41.

- 96. Mei-Dan O, Lopez V, Carmont MR, et al. Adductor tenotomy as a treatment for groin pain in professional soccer players. Orthopedics. 2013;36(9):e1189–97.
- Ingoldby CJ. Laparoscopic and conventional repair of groin disruption in sportsmen. Br J Surg. 1997;84(2):213–5.
- 98. Kluin J, den Hoed PT, van Linschoten R, et al. Endoscopic evaluation and treatment of groin pain in the athlete. Am J Sports Med. 2004;32(4):944–9.
- 99. Susmallian S, Ezri T, Elis M, et al. Laparoscopic repair of "sportsman's hernia" in soccer players as treatment of chronic inguinal pain. Med Sci Monit. 2004;10(2):CR52–4.
- Gilmore J. Groin pain in the soccer athlete: fact, fiction, and treatment. Clin Sports Med. 1998;17(4):787–93. vii.
- 101. Larson CM. Sports hernia/athletic pubalgia: evaluation and management. Sports Health. 2014;6(2):139-44.
- Larson CM, Pierce BR, Giveans MR. Treatment of athletes with symptomatic intra-articular hip pathology and athletic pubalgia/sports hernia: a case series. Arthroscopy. 2011;27(6):768–75.
- 103. Hammoud S, Bedi A, Magennis E, et al. High incidence of athletic pubalgia symptoms in professional athletes with symptomatic femoroacetabular impingement. Arthroscopy. 2012;28(10):1388–95.
- 104. Hiti CJ, Stevens KJ, Jamati MK, et al. Athletic osteitis pubis. Sports Med. 2011;41(5):361-76.
- 105. Paajanen H, Hermunen H, Karonen J. Pubic magnetic resonance imaging findings in surgically and conservatively treated athletes with osteitis pubis compared to asymptomatic athletes during heavy training. Am J Sports Med. 2008;36(1):117–21.
- Holt MA, Keene JS, Graf BK, et al. Treatment of osteitis pubis in athletes. Results of corticosteroid injections. Am J Sports Med. 1995;23(5):601–6.
- 107. Radic R, Annear P. Use of pubic symphysis curettage for treatment-resistant osteitis pubis in athletes. Am J Sports Med. 2008;36(1):122–8.
- 108. Williams PR, Thomas DP, Downes EM. Osteitis pubis and instability of the pubic symphysis: when nonoperative measures fail. Am J Sports Med. 2000;28(3):350–5.
- 109. Choi H, McCartney M, Best TM. Treatment of osteitis pubis and osteomyelitis of the pubic symphysis in athletes: a systematic review. Br J Sports Med. 2011;45(1):57–64.
- 110. Bradshaw C, McCrory P, Bell S, et al. Obturator nerve entrapment. A cause of groin pain in athletes. Am J Sports Med. 1997;25(3):402–8.
- 111. Cesmebasi A, Yadav A, Gielecki J, et al. Genitofemoral neuralgia: a review. Clin Anat. 2015;28(1):128–35.
- 112. Grossman MG, Ducey SA, Nadler SS, et al. Meralgia paresthetica: diagnosis and treatment. J Am Acad Orthop Surg. 2001;9(5):336–44.
- Halpin RJ, Ganju A. Piriformis syndrome: a real pain in the buttock? Neurosurgery. 2009;65(4 Suppl):A197–202.
- 114. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skeletal Radiol. 2001;30(3):127–31.
- 115. McKinney BI, Nelson C, Carrion W. Apophyseal avulsion fractures of the hip and pelvis. Orthopedics. 2009;32(1):42.
- 116. Pisacano RM, Miller TT. Comparing sonography with MR imaging of apophyseal injuries of the pelvis in four boys. AJR Am J Roentgenol. 2003;181(1):223–30.
- 117. Holden CP, Holman J, Herman MJ. Pediatric pelvic fractures. J Am Acad Orthop Surg. 2007;15(3):172–7.
- 118. Mbubaegbu CE, O'Doherty D, Shenolikar A. Traumatic apophyseal avulsion of the greater trochanter: case report and review of the literature. Injury. 1998;29(8):647–9.
- O'Rourke MR, Weinstein SL. Osteonecrosis following isolated avulsion fracture of the greater trochanter in children. A report of two cases. J Bone Joint Surg Am. 2003;85-A(10):2000–5.
- Aronsson DD, Loder RT, Breur GJ, et al. Slipped capital femoral epiphysis: current concepts. J Am Acad Orthop Surg. 2006;14(12):666–79.

- 15 Surgical Interventions in Hip and Pelvis Injuries
- 121. Novais EN, Millis MB. Slipped capital femoral epiphysis: prevalence, pathogenesis, and natural history. Clin Orthop Relat Res. 2012;470(12):3432–8.
- 122. Kartenbender K, Cordier W, Katthagen BD. Long-term follow-up study after corrective Imhauser osteotomy for severe slipped capital femoral epiphysis. J Pediatr Orthop. 2000;20(6):749–56.
- 123. Kuzyk PRT, Kim Y-J, Millis MB. Surgical management of healed slipped capital femoral epiphysis. J Am Acad Orthop Surg. 2011;19(11):667–77.
- 124. Novais EN, Hill MK, Carry PM, et al. Modified Dunn procedure is superior to in situ pinning for short-term clinical and radiographic improvement in severe stable SCFE. Clin Orthop Relat Res. 2015;473(6):2108–17.
- 125. Peck K, Herrera-Soto J. Slipped capital femoral epiphysis: what's new? Orthop Clin North Am. 2014;45(1):77–86.
- 126. Mooney 3rd JF, Sanders JO, Browne RH, et al. Management of unstable/acute slipped capital femoral epiphysis: results of a survey of the POSNA membership. J Pediatr Orthop. 2005;25(2):162–6.
- 127. Krych A, Odland A, Rose P, et al. Oncologic conditions that simulate common sports injuries. J Am Acad Orthop Surg. 2014;22(4):223–34.

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