Hip osteoarthritis and the active patient: will I run again?

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Abstract The popularity of running as a recreational sport for health gains has steadily increased. Runners may acquire several types of injuries including hip osteoarthritis (OA). Running is possible with mild forms of OA if proper joint mechanics, neuromuscular control, and technique are present. Recent literature will be discussed that builds upon previously published articles regarding forces encountered at the hip joint during running. This article will outline the biomechanics and necessary muscle forces during running, and theories regarding strengthening and neuromuscular control. Perspectives on treatment, based on known evidence and our clinical reasoning are presented.

Keywords Running · Osteoarthritis · Femoroacetabular impingement · Rehabilitation

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Introduction

Running is a recreational exercise of choice for many people due to the ease of access. According to a systematic review on running injuries [1], incidence of lower extremity injury varied between studies from 19.4% to 79.3%. Although the hip/pelvis was identified as the least common site, hip injury incidence ranged from 3.3% to 11.5%. Based on these estimations and the number of runners in America, more than 1 million runners experience hip injuries yearly.

Osteoarthritis (OA) affects a majority of people aged 65 years and older and roughly 80% of individuals over age 75 [2]. Appropriate evaluation of the runner with hip osteoarthritis (OA) is of the utmost importance. The examination process may begin with relevant radiographic imaging to determine whether the runner’s bony anatomy is suitable for the activity. This may be followed by a comprehensive musculoskeletal evaluation to determine neuromuscular deficits. As appropriate, running mechanics may be assessed to highlight technical faults that may increase joint loading. Once it has been determined that the bony structure may withstand the impact, muscular stability and neuromuscular timing has been optimized, and technique has been corrected to decrease joint loading, the runner may return to their activity in a graded fashion.

Running participation in the United States

Due to its inexpensive nature, accessibility, and perceived health benefits, running is one of the most popular recreational and competitive exercise activities in the United States, and its popularity is growing. In 2010, 35.5

million Americans aged 7 years or older ran more than once, a 10.3% increase from 2009 [3]. The Bureau of Labor reported in 2008 that 7.1% of Americans aged 15 years or older engaged in running on a daily basis over the years from 2003 to 2006, averaging 0.9 h per day [4].

Hip osteoarthritis: prevalence, etiology, and theory

By clinical standards of pain and physical examination, roughly 27 million American adults suffer from OA of at least one joint [5].

Radiographically, OA is defined by presence of osteophytes, sclerosis of subchondral tissue, formation of cysts, joint space narrowing, and bone contour abnormalities [2]. With advances in MRI, cartilage injury can be found earlier than with classic plain film radiographs. Current literature has been investigating MRI reliability and ability to predict patient outcomes [6].

The severity of OA can be objectified by using the Tönnis Grading Scale [7] (Table 1). Joint space narrowing and the bone reaction are assessed, rather than presence of osteophytes, as the radiographic feature most associated with pain [2].

Accepted models of OA progression posit that joint degeneration often begins with a compromise in the biomechanical properties of the joint. Clohisy [8] examined radiographic abnormalities of the hip and introduced the concept of femoral acetabular impingement (FAI) as a condition associated with labral tears and early hip joint failure. Once the joint has been compromised, a change in range of motion, shape, and/or force distribution causes excessive joint loading, which leads to further degeneration of the articular surface. Mechanical factors, such as obesity and increased loads across the hip, accelerate the rate of OA progression [2]. Early joint compromise, categorized as a Tönnis I grade on X-ray, still maintain the joint space and demonstrate bony sclerosis on radiographs. Once the

patient has reached a grade of Tönnis II, there are subchondral cysts of the bone and cartilage narrowing. For these reasons, it is felt that patients with evidence of early OA that have adequate joint space and uncompromised bony surfaces can run with appropriate knowledge of their condition and mechanics of running.

Previous research suggests that acetabular labral tears mark an early phase in the degeneration of the hip joint [9–14]. The labrum increases dynamic hip stability by deepening the hip joint, providing a suction-seal effect holding the femoral head in the acetabulum [9], and increasing the area of the articular surface [10]. The association between labral lesions and degeneration of hip joint articular surfaces is strong. 73% of 273 patients with labral lesions studied by McCarthy et al. [11] had evidence of chondral damage in the acetabulum, usually located below the lesion on the anterior acetabulum and/or labral-acetabular margin.

The mechanism of acetabular labrum injuries in the normal hip is still unknown, but it has been postulated that osseous morphological changes are one cause of labral injury [12]. In this study all hips with labral tears were found to have some morphological changes on imaging. Labral tears are also believed to be a part of the normal degenerative process in elderly people, as at least one tear was seen in 93% (50 out of 54) of cadavers in a study by McCarthy et al. [11] and 30 out of 30 elderly cadavers seen by Leunig et al. [13].

Along with other forms of OA, hip OA has traditionally been classified as primary (idiopathic) or secondary to preexisting conditions. Evidence is mounting that many cases of OA assumed to be primary are actually secondary to subtle joint abnormalities such as femoroacetabular impingement (FAI) [11, 15–19], and hip dysplasia [2, 7].

Ganz et al. [17] defined femoroacetabular impingement (FAI) as certain bony abnormalities causing a collision between the femur and the acetabulum at end range of motion, which then lead to damage of the acetabular labrum and acetabular cartilage. There may be femoral sided deformity (cam impingement caused by aspherical head) or acetabular-sided (pincer impingement caused by increased bone on the acetabular rim) or a combination of the two [20]. FAI is proposed to lead to OA by causing abnormal contact between the femoral head and the acetabular rim, which then leads to labral tears and avulsion of the cartilage. The repeated collision causes further degradation of the cartilage, which then progresses to arthritis [20]. Bardakos [21] examined 43 patients with radiographs taken at least 10 years apart. He found that coxa vara, increased size of the greater trochanter, and a positive posterior wall sign (indicating acetabular over-coverage) correlated with osteoarthritis progression. Leunig [22] advocates for early surgical intervention to correct these bony irregularities to preserve the joint and prevent premature arthritis caused by abnormal contact.

Table 1 Tönnis Grading. Tönnis [7] grading for hip OA (Adapted from Tönnis and Heinecke [7])

Grade	Radiographic features
0	No indication of OA
1	“Slight narrowing of [hip] joint space, slight lipping at joint margin, slight sclerosis of femoral head or acetabulum”
2	“Small cysts in femoral head or acetabulum, increasing narrowing of joint space, moderate loss of sphericity of femoral head”
3	“Large cysts, severe narrowing or obliteration of joint space, severe deformity of femoral head, avascular necrosis”

Drs. Bedi and Kelly [23] have proposed a concept of hip pain being either static or dynamic. They propose that four factors are associated with static overload in the prearthritic patient: lateral acetabular undercoverage, anterior acetabular undercoverage (dysplasia), increased femoral anteversion, and increased femoral valgus. A static problem exists without an inciting movement or position and can occur during running, walking and sitting. It is our opinion that patients with static structural issues and hip pain should not run recreationally.

Precursors for evaluation and hip mechanics

When evaluating the runner, an understanding of the mechanics of the hip are integral in the assessment and prognostic process. A physical exam and radiographs may reveal femoral anteversion, dysplasia and joint space loss; all could be contra-indications to running. Leunig [24] examined the use of radiographs and the reliability between observers.

The human hip, the weight-bearing diarthrodial joint containing the acetabulum and the femoral head, is a highly congruent joint built for stability. The femoral head is contained in the acetabulum and articulates with a “horse shoe” shaped acetabular cartilage. The acetabulum provides approximately 165° of circumferential bony coverage around the femoral head in the sagittal plane. In addition to its inherent stability, the human hip is also highly mobile with six degrees of freedom [25]. When evaluating the runner, it is important to consider normal values of range of motion (ROM) in the hip joint.

Internal and external rotation of the hip are influenced by femoral torsion. Excessive internal rotation correlates with femoral anteversion while an increase in external rotation is related to femoral retroversion [26]. The normal hip has approximately 15° of femoral anteversion and 15–18° of acetabular anteversion [7, 23].

Impact and hip joint forces during running

Raw force plate data can be used to calculate the vertical component of the ground reaction force (GRF) on impact (impact force). Although smaller in magnitude than GRF during late stance phase, impact forces, a term usually referring to the peak vertical GRF(s) occurring shortly (within 50 ms) after initial contact [27], are believed to be associated with running-related injuries. The gluteus maximus (GMax) works to decelerate the thigh into extension [28] prior to initial contact, to buffer the leg against the contact force. Although such forces are not

explicitly connected to hip OA, they demonstrate some of the forces involved in running.

Keller and colleagues [29] measured the relationship between impact forces and speed during walking, slow jogging, and running in a group of 10 male and 13 female recreational athletes. Impact forces increased in a linear manner only up to 3.5 m/s, after which point they did not significantly increase. The greatest increases in impact forces were seen between walking and slow jogging, increasing from $1.15 \times \text{bodyweight (BW)}$ to $2.36 \times \text{BW}$ for females and $1.23 \times \text{BW}$ to $2.45 \times \text{BW}$ for males. Noting that impact forces are a function of the change in center of gravity velocity, the authors attribute this increase to the rise-and-fall nature of slow jogging. The stability of impact forces after 3.5 m/s were attributed to the “forward leaning during running” style evident at higher speeds, which minimized the acceleration of the body’s center of gravity [29]. The vertical forces associated with rear-foot striking (RFS), the strike pattern of 75%–80% of modern endurance runners, are of particular interest [30]. Hip and knee joint loading may be significantly reduced with a slight increase in step rate. Heiderscheit [31•] found an inverse relationship between step length, vertical excursion, braking impulse and step rate. There were significant changes with a 5% or 10% increase in step rate, which was most likely due to the decreased step length.

A consideration of hip joint forces and moments is also important in the context of hip OA. Clarke [32] used accelerometry to investigate the effect of altered running stride rate on loading response peak trunk/shank deceleration (PSD). It was assumed that PSD correlated to joint reaction forces so long as joint angles remained constant at different stride rates, which they did for the hip, knee, and ankle. As stride rate increased at a 3.8 m/s run, PSD and peak joint forces decreased during impact loading [32]. The researchers note that decreases in float phase duration accompanied stride rate decreases, while stance phase duration remained nearly constant. Clarke recommends that the runner should increase stride rate to decrease joint forces at impact loading [32].

According to one mechanical model of OA derived from animal studies, clinical observation, and an extension of Wolff’s Law [33], repetitive impulse loading at the joint is the most important mechanistic factor in joint degeneration. Marti et al. [34] conducted a retrospective radiographic analysis of hip OA in long distance runners, bobsleigh riders, and non-athlete male controls. Runners demonstrated a significantly higher incidence of all radiographic variables (subchondral sclerosis, osteophyte formation, and decreased joint space) and reporting of pain compared to the other groups. However, sample size was too small to conclusively demonstrate increased risk of OA in runners. This study also lacked baseline radiographs, making OA progression an assumption.

Joint mechanics superior and inferior to the hip

When assessing joint mechanics in the runner, there are two important regions of the body to observe: the ankle joint and the lumbo-pelvic complex. This parallels Bramble and Lieberman's [35] belief that the gluteus maximus (GMax) and Achilles tendon are two structures that have evolved to allow bipedal running in modern humans.

The ankle joint of the runner should be examined. In the mid-stance phase of the running motion the ankle joint must dorsiflex adequately to allow eccentric loading of the Achilles tendon. The runner may effectively compensate for lack of ankle dorsiflexion by everting the calcaneus or over-pronating the mid-foot. This may lead to increased internal rotation and/or adduction of the femur.

The movement of the acetabulum on a fixed femur during the brief stance phase of running is dependent on pelvic tilt and rotation. During initial contact, the ipsilateral pelvis is posteriorly tilted, relative to the fixed femur. As the stance phase progresses, the ipsilateral pelvis must anteriorly rotate as the hip goes into extension and the upper body progresses over the lower extremity, leading into toe-off. The amount of hip extension required for running is only slightly more than that for walking, with the increased stride length being accounted for by anterior pelvic tilt [36].

Lumbar paraspinals are responsible for resisting the anterior shift of the trunk. Paraspinal muscle fatigue has been shown to decrease knee flexion, knee adduction, and hip external rotation moments during running. A 50.5% increase in the knee extension moment from baseline to post paraspinal fatigue was found during running [37]. This highlights the importance of trunk control for maximizing efficiency and reducing risk of injury. The pelvis must posteriorly tilt during the late swing phase of the lower extremity; uncontrolled posterior tilt may lead to premature and/or heavy landing on the ipsilateral foot. It may be surmised that flawed rotation of the pelvis, whether in the sagittal or transverse planes, may cause improper coping strategies in other regions of the kinetic chain. According to electromyographic (EMG) data, there is an eccentric contraction of the abdominals at the end of hip extension, which is followed by a concentric contraction prior to the initiation of hip flexion [38]. Schache [39] produced data reporting that an increase in hip extension is directly related to a decrease in anterior tilt during the running motion in 14 asymptomatic subjects. Inadequate anterior tilt or rotation of the pelvis during late stance phase may cause the runner to excessively extend the femur; this would reasonably stress the iliofemoral ligament and anterior joint capsule [40]. In the setting of instability, disproportionate forces may be placed on the tendon of the iliopsoas, which may act as a dynamic anterior stabilizer to the hip joint [41], possibly leading to tendinopathy.

During the running motion, the femur should remain fixed during the stance phase, avoiding excessive internal rotation or adduction. Increased femoral adduction has been linked to various injuries in the knee and hip joints [42–44].

Novacheck [28] stated that knee flexion amounts normally reach upwards of 90° during the mid-swing phase during the running motion, and 105° when sprinting. This knee flexion combined with mild hip extension and anterior tilt during the “heel-kick” portion of the swing phase in running may cause an eccentric load of the hip flexor muscles, allowing the femur to quickly advance to a flexed state, preparing for initial contact.

Hip motion in the sagittal plane during the running motion has the largest amplitude, relative to the other planes. The hip acts in flexion and extension, with maximum extension occurring at toe-off. The hip then flexes during swing phase, with maximum flexion occurring during mid swing. The degree of flexion increases with velocity, as maximum flexion increases step length. The hip reverses towards extension prior to initial contact, to buffer the leg against the contact force. The major muscles involved are the rectus femoris, hamstrings, and hip extensors. Both the hip flexors and extensors function concentrically and eccentrically at various points in the gait cycle [28].

Neuromuscular strength and timing

In the sagittal plane, it has been found that the Gluteus Maximus (GMax) fires at a higher frequency during running than walking [45] and produces considerably larger forces [46]. The GMax controls hip flexion during late swing phase as the foot prepares to come in contact with the ground. The GMax is also active during initial contact and weight acceptance to assist in proper dissipation of GRF. Forward trunk lean has been shown to increase when running speed or surface gradient increases [47]. The GMax and the erector spinae must counteract anterior trunk flexion. In addition, the trunk is subjected to forces in the coronal plane that tend to flex the trunk medially relative to the stance-side hip. These forces require contraction of the stance-side hip abductors to maintain stability [48].

Lewis [49] proposed that those with anterior hip pain, subtle hip instability, or an anterior acetabular tear may be able to decrease anterior hip joint forces by increasing gluteal muscle firing during hip extension, increasing iliopsoas strength for hip flexion, and avoiding greater than neutral hip extension. Distance runners with anterior hip pain may benefit from decreasing the amount of hip extension. However, this theory is based on models performing hip extension in a non-weight-bearing position.

Peak hip extension can be witnessed in terminal stance or early swing phase, with the knee flexed.

Muscle tendon unit (MTU) length of the rectus femoris and contralateral GMax increases significantly during running, and biceps femoris minimum length is significantly less (as compared to walking). The MTU length changes are contributed to most by shortening of the iliacus and lengthening of the GMax during hip flexion. Hip flexor length may influence the range of hip extension during running. The greatest iliacus and contralateral biceps femoris MTU lengths occur simultaneously during running [50]. This may be addressed with specific stretching that combines both motions to prevent injury. An example of this would be a side-lying stretch with superior leg in hip extension and knee flexion, and hip flexion and knee extension on the inferior leg to simulate the running motion.

The gluteus medius (GMed) is also important when assessing and treating the runner with hip OA. During initial contact, the gluteus medius is eccentrically loaded while the femur is slightly adducted in relation to the pelvis. This is done to maintain center of gravity and decrease excursion in the transverse and frontal planes. As the stance phase progresses and the femur moves into relative neutral abduction, the GMed transitions from an eccentric to concentric role, stabilizing the pelvis and maintaining the center of gravity.

Management

The treatment of the runner with hip OA begins with a thorough musculoskeletal examination. Joint ROM, muscle length, muscle strength and endurance should be examined. The evaluation is the most important component because it will reveal the impairments that will be addressed during rehabilitation.

The running motion is comparable to an elastic spring: either gaining potential energy through weight acceptance by way of eccentric contractions of muscle groups, or releasing the energy via concentric contractions in order to propel forward [51]. When considering the running motion in this manner, the clinician must evaluate each involved joint for the necessary motions at each phase of running and each muscle for the ability to adequate strength and the ability to eccentrically contract, either in an open-chain (e. g. hip flexors during swing phase) or closed-chain (e.g. hip abductors in stance phase) situation.

Most runners may exhibit strong musculature with typical testing positions, as defined by Kendall [52]. Each muscle should be tested five to ten times successively to assess endurance and strength. The runner should be observed performing several functional tasks: squats, lunges, single-leg standing, single-leg step-ups and step-

downs on an eight inch step and single-leg squats. The clinician should note each portion of the kinetic chain during each task to judge the runner's ability to transfer load through the limb. Often, failure to properly accept and dissipate forces at one joint leads to compensatory patterns elsewhere.

Injured runners may lack sufficient lumbar and pelvic control to maintain correct form during running. Fatigue of the transversus abdominis and other deep core musculature may allow increased lumbar flexion and posterior tilt of the pelvis throughout the running motion. However, the runner will continue to achieve extension of the hip by way of excessive extension at terminal stance, perhaps stressing the anterior stabilizers of the hip. Neuromuscular re-education of the lumbar and pelvic stabilizers is a foundation of treatment for most runners. This is begun in unweighted positions and gradually progressed to seated, standing, and squatting or lunging positions, all the while focusing on form and the ability to maintain a neutral spine.

Chance-Larsen [53] showed that 'abdominal hollowing' exercises improve the timing of the GMax in relation to the biceps femoris in healthy subjects. Takasaki [54] asked asymptomatic males to perform a prone hip extension exercise. With clinician-induced compression applied to the proximal portion of the bilateral ilia, GMax excitation was markedly improved in relation to the semitendinosus muscle. Based on these findings, it would seem that addressing lumbopelvic control, and specifically the transversus abdominis [55], may positively affect the timing of the GMax in relation to the hamstring muscles.

The GMed is important when assessing and treating the runner with hip OA. Gottschalk [56] proposed that the primary function of the GMed is to stabilize the femoral head in the acetabulum based upon dissection and EMG data. Acetabular contact pressures have been found to correspond with GMed activity more so than peak ground reaction forces [57]. It has also been demonstrated that there are changes in pelvic-femoral alignment during gait in those with hip osteoarthritis [58]. Those with advanced degenerative hip joint disease have shown significantly less muscle volume of the GMed and piriformis [58]. Specific retraining of the abductors with a focus on maintaining appropriate alignment is important in rehabilitating those with osteoarthritis and preventing pain.

GMed and GMax strengthening should begin in non-weight-bearing positions, focusing on form and endurance of the muscle. The runner should be able to elicit contraction of the deep lumbopelvic stabilizers when performing open-chain exercises, such as straight leg raises, in supine, side-lying or prone positions. As control and endurance improve, the runner can begin to strengthen the gluteal musculature in weight-bearing positions, beginning

with sagittal plane motions and progressing to transverse and multi-planar movements. Snyder's [59•] functional strengthening program for runners is based on the theory that the hip abductors and external rotators play a vital role in controlling lower extremity positioning during single leg stance. Single-leg stance closed-chain hip rotation challenges lower extremity stability in the frontal and transverse planes. The training program significantly increased the hip's abduction and external rotation strength. Knee abduction moment decreased following increases in hip strength. These strength gains change lower extremity joint loading by decreasing rearfoot eversion, hip internal rotation, knee abduction, and rearfoot inversion joint moments [59•].

When addressing the abilities of the GMed, the clinician must consider that this muscle mainly functions eccentrically in 0–10° of adduction during the stance phase [60]. Ward and colleagues [61] stated that, based on architectural design, the GMed has limited ability to abduct the hip beyond 20°. We advocate early non-weight-bearing exercises that stress activation of the GMed in small ranges of femoral abduction/adduction. Closed kinetic chain theory states that proximal control (hip and core stability) precedes distal segment control. Niemuth and colleagues [62], after exploring injury patterns in runners, concluded that hip muscle strength is critical in maintaining forces throughout the body. The authors found that the hip abductors and adductors on the injured leg were significantly weaker than those on the uninjured leg. The adductors often act to increase stability of a seemingly unstable limb.

The adductor muscle group may frequently compensate for hip flexion and hip extension deficiencies and may become painful and hypertonic. This improper firing pattern can be overridden by decreasing muscle tightness through soft tissue mobilization and muscle re-education. Muscle imbalances may not be exposed until duration or intensity of running is increased and injury results [62].

Those with hip OA may respond well to gentle hip mobilization as indicated by a capsular pattern or restricted motion with a capsular end feel. Hoeksma [63] showed significant short-term improvement in ROM and pain levels with specific manipulations and mobilizations directed at the hip in 109 patients with hip OA. When satisfactory muscular control is regained (as seen on functional testing) and running form is believed to be correct, they may attempt to return to running. First, the runner should begin on softer surfaces—such as grass, a track or the treadmill. Form must be maintained, with no emphasis on speed. As form and confidence improves, and pain is not present, speed may be increased gradually. Plyometric exercises may be indicated, if tolerated, to improve propulsion power and speed.

Conclusion

When considering running with a compromised hip joint, the clinician and the runner must work together to determine whether it is safe to continue the sport with or without modifications. The mechanical model of arthritis would support that these increased loads would lead to arthritis. If such a model is valid, the increased impact forces associated with running should correlate with an increased frequency of OA in runners. However, evidence of association between long-distance running and OA is unclear [64].

Our current belief is that individuals who are classified as Tonnis I may still run, provided they do not present with the following static mechanical hip abnormalities: increased femoral anteversion, lateral and anterior femoral undercoverage, and/or increased femoral valgus. Bony abnormalities can be compensated with better mechanics, proper strengthening and endurance of the local hip musculature and surrounding joints must be achieved. Increasing cadence and promotion of a mid- to forefoot strike may alleviate stresses on compromised joints. Re-education of form may also be necessary to allow the body to properly dissipate and distribute impact forces. With careful treatment and re-training by the clinician, those runners diagnosed with mild forms of OA may be able to run effectively.

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