# Chapter 5
# Gait Pathomechanics in Hip Disease

**Kharma C. Foucher**

## Overview

Mobility is an important aspect of our daily lives that is often taken for granted. Everyone, from those of us who are sedentary and those of us who are elite athletes, must be able to walk, climb stairs, and sit at some point in our daily routines. For some of us, kneeling and squatting are also important. Ideally, we can do all of things without thinking about it. Hip disease, however, can prevent this "thoughtless" mobility. A research participant in our laboratory remarked that she knew it was time to seek care for her hip because she noticed that she was thinking about her hip all the time; in her words "a 30 year old should not be thinking about her hip!" Arguably, 50 year olds and 90 year olds should not be thinking about their hips either. Neither the young ballet dancer with symptoms of femoroacetabular impingement (FAI) nor the grandmother who wants to stay active after hip arthroplasty should have their mobility compromised or be annoyed by thoughts about their hips.

The way that pathology affects mobility is central to the patient experience of disease. It is therefore critical for surgeons and care providers to understand how important mobility is to the individual patient experience. Take, for example, total hip arthroplasty (THA), which can be considered the end of the spectrum of degenerative hip disease. Most of criteria for surgical success are implant-oriented, e.g. quality of fixation, signs of loosening, revision rates [1–3]. However, as the use of patient-oriented or patient-reported outcome measures (PROMs) increases, and the concepts of "value" and appropriateness for surgery are expanding [4, 5], it is

K.C. Foucher, MD, PhD (✉)
Department of Kinesiology and Nutrition, University of Illinois at Chicago,
1919 W. Taylor St., 650 AHSB, Chicago, IL 60612, USA
e-mail: kfouch1@uic.edu



R.K. Aaron (ed.), *Diagnosis and Management of Hip Disease*,
DOI 10.1007/978-3-319-19905-4_5

increasingly important for surgeons to consider how much THA improves patient well-being and quality of life as well. New research from large patient cohorts in the US and abroad is showing that up to 50 % of patients have some self-reported functional limitations 2–5 years after THA [6–8]. Severe limitations or a failure to have a meaningful clinical response based on change in PROMs may affect 4–15 % of unilateral THA patients in these studies. This is a problem. Recent studies also show that the most important desires or expectations that people undergoing THA report involve regaining the ability to be physically active [9–11]. In fact, participation in valued activities may be even more important to people than pain relief itself is per se [11]. This bears repeating. People seeking THA want to be able to move, work, and play even more than they want their hip to stop hurting. Considering that over 300,000 THA procedures are performed each year, and the prevalence of THA is ever-increasing [12, 13], the fact that tens of thousands of patients may be having inadequate functional recovery after THA should raise alarms for practitioners working in a value-driven environment. Understanding motion and how disease or trauma affects motion is an important part of providing good care.

Understanding how gait mechanics change with common hip pathologies is important because doing so can give insight into how to diagnose and treat, and how to assess a treatment's effectiveness. The goal of this chapter will be to explain how, and possibly why, hip pathology affects gait mechanics. The chapter will be organized around two (related) ways to think about gait pathomechanics:

1. Gait pathology as disruption of the normal relationship between structure and function.
2. Gait pathology as a behavioral response to disease.

After a brief discussion of terminology, these concepts will be presented with examples taken from morphological disorders (hip dysplasia, FAI), hip osteoarthritis (OA), and THA. The bulk of the chapter will be spent considering the structure–function framework, followed by a brief discussion of behavioral aspects of gait mechanics.

## *Terminology*

This section will define basic terminology needed to describe key events of walking. At the same time, we will discuss the events that characterize normal gait and hint at gait alterations that can occur during hip pathology. At this time, it should be noted that for the most part the terms "gait" and "walking" will be used interchangeably in this chapter. However, hip pathology can affect other activities like stair climbing, sit-to-stand, running, etc., and the terms and methods discussed in this chapter can be and have been applied to other activities.

## Gait Cycle and Gait Variables

Walking is a cyclic activity that can be demarcated by several key events. Hip pathology can change the timing of any these events, with reverberations throughout the cycle. The most basic way to divide the gait cycle is into two main phases—stance and swing. The stance phase of gait, intuitively, refers to the time when the foot is on the ground. It typically lasts approximately 60 % of the gait cycle. The swing phase of gait, again intuitively, refers to the time when the foot is off the ground. It typically lasts approximately 40 % of the gait cycle. Stance and swing can further be subdivided. Typically this is done based on the actions of a *lead limb*, compared to a *trailing limb* (Fig. 5.1). Alternatively, the stance phase can be considered in terms of periods of double support or *double limb stance*, when both feet are on the ground during loading response and preswing, and a period of *single limb stance* when only one foot is on the ground during midstance and terminal stance. This concept is useful when attempting to understand the effects of hip pathology because, as we will discuss later, hip musculature plays a key role in frontal plane

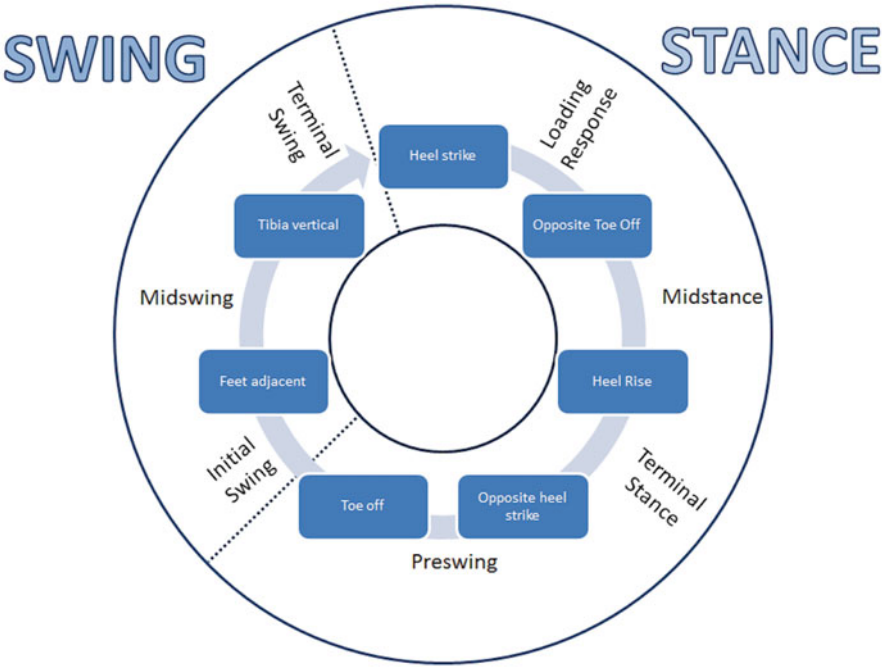


**Fig. 5.1** Terminology defining the phases and subphases of the gait cycle. The key events that delineate each phase are shown. Stance can also be considered as two periods of double limb support—loading response and preswing, and a period of single limb support—midstance and terminal stance

control of the upper body, and the ability to smoothly accomplish weight transfer from one limb to the other.

There are several types of variables that can be measured during gait analysis. We'll discuss them here in order of increasing complexity with respect to equipment and computations required.

## Spatiotemporal Gait Variables

Spatiotemporal gait variables describe the timing of the events of gait. Walking speed is perhaps the simplest and most intuitive gait variable. It is also the easiest to measure, requiring no specialized equipment, and the one with perhaps the broadest relevance. Walking speed has been proposed as a "6$^{th}$ vital sign" because of its relevance to so many aspects of health and the ease and reliability of its measurement [14, 15]. *Speed* is simply distance traveled per unit of time. (Some authors prefer to use the term velocity, which refers to speed combined with an indication of direction.) To increase (or decrease) your walking speed, you can either take more (or fewer) steps, take longer (or shorter) strides, or both. In other words you can alter your *cadence*—steps per unit time, *stride length*—distance per stride, or both. The relationship between speed, cadence, and stride length can be described by the equation: speed = cadence x stride length. Be aware that some authors may refer to *step length* rather than stride length. A *step* is demarcated by heel strike of one foot to heel strike of the other foot; a *stride* is demarcated by heel strike of one foot to heel strike of the *same* foot. A stride is therefore composed of two consecutive steps.

## Kinematic Gait Variables

Kinematics refers to joint angles and motions. Motions of the hip can be described in sagittal, frontal, and transverse planes. In the sagittal plane, the hip typically passes through an arc of 30–40°. It is maximally flexed—around 15–20°—at heel strike. The hip typically passes through a smooth arc of extension, and reaches up to 20° of extension by toe off. Next, during the swing phase, the hip flexes to about 15–20° and the cycle repeats. In the frontal plane, the hip passes through a small arc of approximately 15°. The hip is typically neutral at heel strike, then adducts to approximately 10° during loading response. The hip then gradually abducts reaching approximately 5° of abduction during the swing phase. Motions in the transverse plane are very small. The hip is typically neutral at heel strike. During stance, a small amount of internal and external rotation of the femur with respect to the pelvis may occur, but the total range of motion is typically less than 10°.

Although it may seem obvious, at this point it is important to note that the hip and pelvis function together. It is both conceptually and methodologically difficult to isolate the hip and pelvis. This is particularly true when describing the frontal and transverse plane motion of the hip. For example, much of the internal rotation of the thigh on the leading limb during stance is perhaps more accurately thought of as

transverse plane rotation of the pelvis as the trailing limb enters the swing phase of its gait cycle. Even in the sagittal plane, the small amount of pelvic tilt that occurs during walking can be difficult to distinguish from hip flexion. There are two methodological challenges in separating pelvis motion from hip motion. First, some commonly used marker sets use the anterior superior iliac spine to define the proximal end of the thigh segment, because this pelvic landmark can be easily palpated. This means that the measurements of thigh motion being taken are quite literally a measurement of coupled thigh and pelvic motion. Secondly, whatever the marker set, soft tissue movement can introduce measurement error that is larger around the hip. The reader is also cautioned that hip angles are occasionally reported as the position of the thigh relative to the vertical, instead of relative to the pelvis. Range of motion should be comparable in either case, but the absolute angles would differ.

## Kinetic Gait Variables

Kinetic variables can refer to power, work, and external moments. The discussion here will be limited to external moments. Other sources can provide more information on other kinetic variables. (A classic text by Jacquelin Perry, MD—recently updated with Judith Burnfeld, PhD, PT is an excellent supplemental source for all of the basic gait terminology discussed here [16]).

Why measure external moments? The goal of quantitative gait analysis is to learn information about how muscles may be functioning to accomplish the task at hand. As yet, there is no way to measure muscle forces in vivo. Electromyography (EMG) can be used to detect the electrical activity of the muscles, which can then be used to infer the on-off timing of muscle firing and the relative intensity of the contractions. The actual amount of force being produced *internally* by the muscles cannot be measured or approximated, even with EMG. Forces *external* to the body, however, can be easily measured. We can measure the forces between the foot and the ground, the ground reaction force during walking, and calculate the external moments and forces that act at each joint using *inverse dynamics.* Based on Newton's second law—the principle that for every action there is an equal and opposite reaction—we can infer the functional activity of agonist muscle groups in each plane during walking.

External moments arise by the action of the ground reaction force acting at a certain distance from the joint (hip) center. This distance is the *lever arm* or *moment arm*. The torque created by this force is called an external moment. A schematic depicting measurement and interpretation of the hip moments in the sagittal plane at three instances during stance is shown in Fig. 5.2. At heel strike and during loading response, the GRF is passing anterior to the hip center and the moment arm is quite large. We would measure an external moment that tends to flex the hip. We know that there must be an equal and opposite moment that tends to extend the hip. The hip muscles are primarily responsible for creating this internal moment. Thus we can infer that there must be net activity of the hip extensors. During midstance, the GRF is large but it passes very near to the hip center. The moment arm is

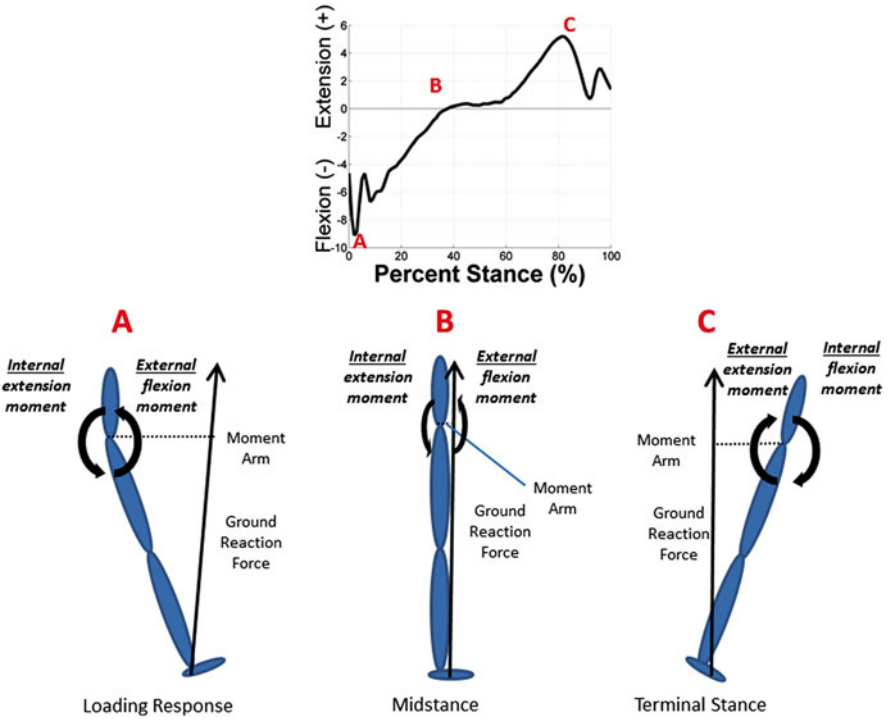


**Fig. 5.2** Cartoon depiction of the moments about the hip in the sagittal plane. During loading response (**a**), the ground reaction force (*black arrows*) passes in front of the hip center. This *external* force will cause a moment tending to flex the hip. It must be balanced by an equal and opposite internal moment. Similar reasoning can be applied during midstance (**b**) and terminal stance (**c**) to interpret the pattern of external and internal moments. The magnitude of the ground reaction force and the size of the moment arm determine the size of the external moment measured

therefore very small and the corresponding external flexion moment is near zero. Finally, during terminal stance and preswing, the GRF passes posterior to the hip center and the moment arm is large again. An external extension moment would be measured; this moment must be balanced by the hip flexors.

Similar reasoning can be used to understand the pattern of moments seen in the frontal and transverse planes. In the frontal plane, the GRF passes medial to the hip center during most of stance. This means that there is an external adduction moment for most of stance that must be balanced by the hip abductors. Sometimes, during loading response or preswing, the GRF passes lateral to the hip center and an external abduction moment is measured. Finally in the transverse plane, an internal rotation moment, which must be balanced by the muscles that externally rotate the hip, is typically seen in the first half of stance. An external rotation moment, which must be balanced by muscles that internally rotate the hip, is typically seen in the second half of stance.

There are some additional caveats about interpreting external moments. Note that measuring an external flexion moment, for example, and using this information

to infer net hip extensor activity does NOT mean that the hip flexors are not active. In fact, one of the main limitations of gait analysis is that these measures tell nothing about antagonistic muscle activity. Electromyography can be a useful adjunct to measuring external moments, to give additional information about muscle firing patterns. Given that the rationale, stated above, for conducting quantitative gait analysis was to understand the forces within the muscles, it should also be noted that muscle forces per se cannot directly be measured using gait analysis. The external moments measured can be used with or without electromyographic information as input into computer models to calculate potential muscle and joint forces [17, 18].

Next, it is often only the peak external moments about the hip that are reported and analyzed in research studies. While this common approach neglects some potentially useful information, it does provide a useful snapshot of dynamic muscle function in each plane and is used in research routinely to study hip pathology. Finally, readers should be aware that while it is *external* moments that are measured during gait analysis, some authors prefer to report them as their corresponding *internal* moments. This may or may not be explicitly stated. To determine which convention the author is using, look for clues such as indications about the timing of the peak moments. For example, if an author refers to a peak extensor moment at heel strike, the reader should be alert that internal moments are being reported. This distinction is critical for accurate interpretation of data presented.

Newcomers to the gait analysis literature should be cautioned against conflating moments and motion. When interpreting external moments, one must think about the position of the GRF relative to the position of the hip center. While these relative positions are certainly related to the action of the hip at that time, the motion and moments are not the same. For example, when the peak flexion moment is measured, the action that the hip is undergoing is *extension.* Likewise, an external adduction moment can be measured both while the hip is ADducting, as during midstance, and while the hip is ABducting, as during terminal stance. Also note a related methodological point—one does not need to be able to measure motion in a given plane in order to measure external moments in that plane. For example, although transverse plane *motion* cannot be accurately measured with some common marker sets, the coordinates of the proximal and distal ends of each limb segment and the joint centers can be accurately localized in 3D space. Thus all the necessary information for calculating transverse plane *moments* is available. A knowledge of relative joint motion could be helpful to enhance the overall interpretation of the findings, but is not necessary for accurate calculation of external moments.

## *Summary of This Section*

At this point we have introduced the most common variables used to describe gait mechanics in health and disease. Spatiotemporal (speed, stride, cadence), kinematics (motions), and kinetics (moments). Normal hip kinematics and kinetics have been outlined briefly. In the next two sections we will discuss how gait mechanics change with pathology, gait *patho*mechanics.

## Gait Pathomechanics as Disruption of Structure–Function Relationship

### *Overview of This Section*

Structure and function are intimately related. Structural changes in the hip joint, due to pathology, change hip function. These changes can be reflected as changes in one or more of the gait variables discussed above. To explore this concept, we will consider how hip joint structure influences function throughout the spectrum of hip degenerative disorders. We will first consider two disorders of hip morphology, dysplasia and FAI. This will be followed by hip osteoarthritis (OA) and THA. In each case, the common gait anomalies seen before and, where applicable, after surgical reconstruction will be described. Next the connection between abnormal structure and gait function will be discussed.

### *Pathological Disorders of Hip Morphology (Hip Dysplasia and FAI)*

Hip dysplasia and FAI are considered to be disorders of hip morphology that are believed to be precursors to hip OA [19]. Their pathophysiology is covered in detail elsewhere in this volume, but most simply, in either case the relative coverage of the femoral head by the acetabulum is either less (dysplasia) or more (FAI) that what is considered normal. This structural abnormality has three interrelated biomechanical consequences. First, the way that the joint surfaces move against each other—the *arthrokinematics*—will be abnormal. This is a problem because it puts parts of the joint in contact that aren't designed to be in contact, and changes the pattern of stress distribution at the joint [20, 21]. When areas of cartilage encounter stresses to which they are not adapted, damage can occur; this is a proposed mechanism for OA initiation [22, 23]. Secondly, changing the shape of the femoral head or acetabulum can change the location of the hip center. This will in turn alter the moment arms for the muscles that cross the hip. This could have consequences for the ability of the muscles to balance the loads required by normal gait—adaptations may arise that are reflected in the external moments measured. Finally, these disorders may physically reduce the available range of joint motion at the hip. This will also lead to gait adaptations that will be manifested in the gait variables measured.

There are surprisingly few quantitative gait analysis studies in the literature on hip dysplasia and FAI. In the case of hip dysplasia, this may be because our awareness of this disorder emerged well before the advent of clinical gait analysis, and because it is typically diagnosed and treated in pre-ambulatory children. FAI, on the other hand, is a recently recognized and still controversial disease entity. There are

only a few studies on gait analysis in people with FAI because the knowledge is still emerging. This is currently a very active research area, however, and new studies appear in the literature regularly.

## Gait Alterations in Hip Dysplasia

A review of the literature identified four fairly recent studies that report some of the spatiotemporal, kinematic, or kinetic gait variables discussed above in patients with hip dysplasia (Table 5.1) [24–27]. Unfortunately the literature is relatively sparse and the study populations are very different so it is difficult to identify common trends. Compared to control subjects, subjects with hip dysplasia may have less hip extension during walking [25, 27]. This restriction may be compensated for with increased pelvic excursion [25]. Reduced peak hip extension moments have also been seen [25, 27]. This indicates reduced net activity of the hip flexors in terminal stance or preswing. Two studies that evaluated subjects before and after a surgical intervention found that surgery did not significantly alter spatiotemporal or

**Table 5.1** Summary of recent gait analysis studies involving subjects with hip dysplasia

| Source | Study population | Select gait variables (of those discussed in this chapter) | Significant findings |
|---|---|---|---|
| Pedersen et al. [24] | 9 adult women, 18 months pre/post periacetabular osteotomy | • Max hip extension<br>• Peak flexion moment<br>• Peak extension moment | Pre-to-post:<br>• No change in hip extension<br>• Peak flexion moment decreased |
| Omeroglu et al. [25] | 10 children with previously treated DDH undergoing soft tissue release<br>20 healthy children | • Speed<br>• Step length<br>• Pelvic and hip kinematics<br>Sagittal and frontal plane hip moments | Vs. control:<br>• Increased frontal and sagittal plane pelvic excursion<br>• Slightly reduced peak extension moment<br>• Delayed transition from flexion moment to extension moment during midstance |
| Sucato et al. [26] | 21 adolescents and young adults evaluated before and after Ganz periacetabular osteotomy | • Speed<br>Hip Abductor Impulse (time integral of hip adduction moment) | Vs. control:<br>• Slower speed both before and after surgery<br>Pre-to-post:<br>No differences by 1 year |
| Jacobsen et al. [27] | 32 adults with untreated hip dysplasia<br>32 control subjects | • Sagittal plane hip kinematics<br>• Sagittal plane hip moments | Vs. control:<br>• Less hip extension<br>• Lower peak extension moment |

kinematic measures taken [24, 26]. One study did find that the peak flexion moment, which peak during loading response and indicates net activity of hip extensors, decreased after surgery [24].

A methodological aside: Two studies [24, 26] employed an interesting technique of analyzing gait variables that was not discussed above. In both studies, the angular impulse—the time integral of the moment—was calculated. This technique takes advantage of more of the available information. In knee OA, the angular impulse of the adduction moment has been shown to be a more sensitive marker of disease than the peak adduction moment [28]. The significance of the angular impulses of moments at the hip has not been fully established but this use of this new variable is an interesting emerging trend. Similarly, two studies analyzed the temporal properties of the sagittal plane moments. Omeroglu et al. noted that the transition between having an external flexion moment and an external extension moment, which usually occurs in the middle of the stance phase of gait (see center of Fig. 5.2) was delayed in subjects with hip dysplasia [25]. We have noticed this trend in subjects with hip OA (unpublished additional finding from a previously published study [29]). Again, the significance of this is as yet unknown, but it may indicate a subtle deficit in postural control during single limb stance. Finally, the paper by Pedersen et al. offers a good example of the need to ascertain whether or not the terminology being used matches the terminology with which one is familiar. When describing the sagittal plane moments that were analyzed, Pedersen refers to "maximal extensor dominance in the first half of the stance phase (H1) and maximal flexor dominance in the second half of the stance phase (H2)." [24] The reference to the timing of these peaks tells us that the authors are referring to what we have called, respectively, the peak external flexion moment—balanced by net hip extensor activity, and the peak external extension moment—balanced by net hip flexor activity.

## Gait Alterations in FAI

Gait alterations associated with FAI both before and after surgical intervention have been nicely summarized in two very recent review articles [30, 31]. Only five studies, so far, have reported results of walking gait analysis studies (Table 5.2) [32–36]. Across these studies, limitations in range of motion in all planes have been found in people with FAI compared to self-reported healthy subjects or, where available, subjects with verified radiographically normal hips. Hip kinetics have been less frequently reported, and findings have been less consistent so far. While Hunt and Brisson have found reduced hip moments either before [35] or after [36] surgery for FAI, others have not. It should be noted that so far, gait studies of FAI subjects have had small sample sizes (typically fewer than 20 subjects) and have not been heterogeneous with respect to type of FAI (cam vs. pincer vs. mixed) or surgical approach and management. Thus it is so far difficult to draw detailed conclusions about the effect of FAI on gait.

**Table 5.2** Summary of recent gait analysis studies involving subjects with femoroacetabular impingement

| Source | Study population | Select gait variables (of those discussed in this chapter) | Significant findings |
|---|---|---|---|
| Kennedy et al. [32] | 17 subjects with cam FAI<br>14 controls | • Speed and step length<br>• 3D pelvis and hip kinematics<br>• 3D hip moments | Vs. control:<br>• Reduced sagittal and frontal plane hip and pelvis range of motion (ROM) |
| Rylander et al. [33] | 11 subjects tested before and 1 year after arthroscopic reconstruction | • Speed<br>• Sagittal and frontal plane hip kinematics and kinetics | Pre to post changes:<br>• ROM increased<br>Max flexion increased |
| Hunt et al. [35] | 30 subjects with FAI<br>30 control subjects | • Speed, step length, cadence<br>• 3D hip kinematics<br>3D hip kinetics | Vs. control:<br>• Slower speed, cadence<br>• Lower ROM all planes<br>• Lower peak flexion, external rotation moments |
| Brisson et al. [36] | 10 subjects with cam FAI tested before and after open hip reconstruction<br>13 control subjects | • Speed, stride length, cadence<br>• 3D pelvis and hip kinematics<br>3D hip kinetics | Vs. control:<br>• Reduced sagittal and frontal plane hip ROM<br>• Reduced peak adduction, internal rotation moments after surgery |
| Rylander et al. [34] | 17 patients with FAI tested before and after arthroscopic surgery<br>17 healthy controls | • 3D pelvis and hip kinematics | Vs. control<br>• ROM reduced in all planes before surgery<br>• Sagittal and transverse plane ROM improved to within normal |

An additional methodological note: Some studies have observed a reversal of sagittal plane hip motion during walking (Fig. 5.3) [33–35]. We have also identified this kinematic pattern in patients with mild to severe hip OA [29], and others have seen it in endstage hip OA [37]. Brisson and Kennedy specifically noted that they did not observe this motion pattern [32, 36], but this could be because their studies were restricted to cam-type FAI, or because of slightly different gait methodologies. (See Michaud 2014 for a discussion of different methods of identifying relative joint motions [38].)

## Gait Alterations in Hip Dysplasia or FAI as Alterations of the Structure–Function Relationship

Today, both hip dysplasia and FAI can best be understood as heterogeneous families of hip morphologic abnormalities. It is clear that altering the shape of the femoral head and its articulation with the pelvis results in some gait changes. The fact that

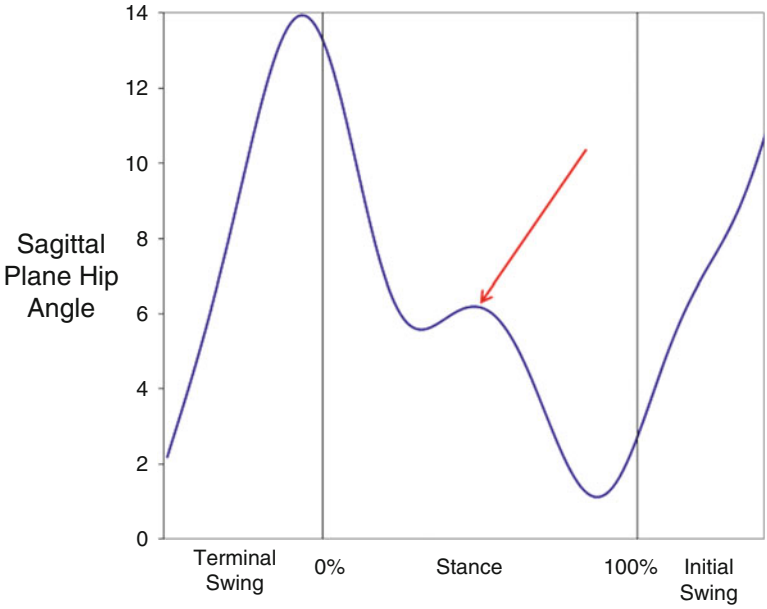


**Fig. 5.3** Sagittal plane hip angle in degrees for a subject with severe hip osteoarthritis. *Arrow* indicates a reversal of hip motion in midstance. This kinematic pattern has been observed in patients with femoroacetabular impingement [33] and hip osteoarthritis [29]

restoring normal morphology only partially normalizes gait (with even more residual abnormalities observed during more demanding activities like squatting and stair climbing [34, 39]), however, demonstrates that abnormal bony morphology is not solely responsible for gait changes. Weakness of the gluteus medius and other muscles, as well as alterations in the anatomy of the hip abductors has been observed in both hip dysplasia and FAI [40, 41]. These and other structural changes in the soft-tissue could certainly contribute to the gait alterations seen before surgery in both disorders. Liu in particular notes that the surgeon must be mindful of muscular abnormalities when planning treatment and postoperative physical therapy so that a fuller recovery can be achieved.

## *Gait Alterations in Mild to Moderate Hip OA*

Most studies of gait in hip OA have focused on patients with endstage disease. A few articles—most notably a 2012 study by Eitzen et al.—have either focused specifically on subjects with mild to moderate disease [42] or included subjects with less severe disease [29]. In addition, a recent review article summarized spatiotemporal characteristics of gait in hip OA [43].

Almost universally, people with mild to moderate hip OA walk with reduced speeds [29, 42, 43], Constantinou's review suggests that this speed deficit is attributable to reduced stride lengths [43]. However, even after statistically accounting for the effect of walking speed, kinematic and kinetic differences are found in people with OA compared to healthy controls [29, 42]. Eitzen reported that the hip range of motion in the sagittal plane is reduced in subjects with mild to moderate hip OA compared to controls, most notably in extension (Fig. 5.4) [42]. We have also observed reduced hip range of motion in the sagittal plane in subjects with mild to severe hip OA [29]. Furthermore, we have also reported an increased prevalence of the hip motion discontinuity gait pattern discussed above (Fig. 5.3) [29]. This sagittal plane motion pattern was associated with presence of hip OA, having more radiographically severe OA, and having more severe gait abnormalities overall.

Kinetic gait abnormalities have also been reported. Eitzen demonstrated that the sagittal plane hip moments are reduced compared to control subjects, with the greatest deficits again seen in the second half of stance (Fig. 5.4) [42]. In Eitzen's figure we can also appreciate the delay in the timing of the sagittal plane hip moment's switch from an external flexion moment to an external extension moment, similar to

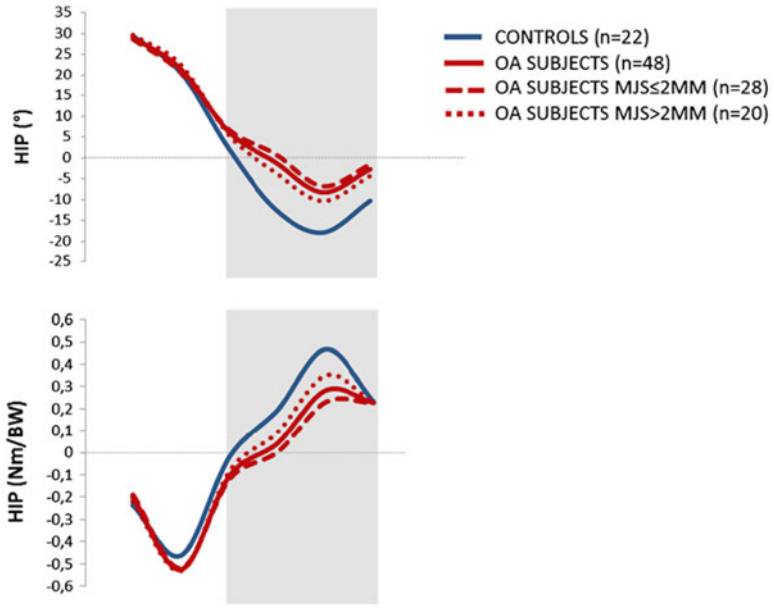


**Fig. 5.4** This figure shows sagittal plane hip motion (*top*) and moments (*bottom*) for subjects with and without mild to moderate symptomatic hip OA. Subjects were subdivided based on radiographic OA severity based on Minimum Joint Space (MJS). Deficits in hip extension angles and the peak extension moments are seen in late stance. [Modified from Eitzen I, Fernandes L, Nordsletten L, et al. Sagittal plane gait characteristics in hip osteoarthritis patients with mild to moderate symptoms compared to healthy controls: a cross-sectional study. BMC Musculoskeletal Disorders 2012; 13: 258. With permission from BioMed Central, Ltd.]

the shift seen in subjects with hip dysplasia reported by Omeroglu et al. [25], as discussed above. We have also reported abnormalities in the other planes. With the exception of the peak hip abduction moment, all peak external moments were reduced in subjects with hip OA compared to control groups [29].

### Gait Alterations in Mild to Moderate Hip OA as Alterations of the Structure–Function Relationship

As was hinted at in the discussion of hip dysplasia and FAI, there is a rapidly emerging body of evidence suggesting that femoral head shape is an important contributor to hip OA etiopathogenesis [44–47]. Modeling studies reveal that cartilage stresses are sensitive to the shape of the femoral head [21]. It would not be a stretch to consider that gait differences associated with early OA may be associated with subtle alterations of hip articular structure. There is evidence of a relationship between radiographic hip OA severity, as determined by the modified Kellgren–Lawrence (KL) grading system [48], and peak external moments during gait (Fig. 5.5). KL grading is arguably a relatively crude metric of hip structure, as it is based on a visual inspection of the joint space, and does not account for the morphologic changes that have recently been associated with OA. Nevertheless, an overall unloading pattern can be seen as hip OA severity increases. We have also demonstrated that having the sagittal plane motion discontinuity described above is associated with having reduced sagittal plane range of motion and peak flexion, extension, and internal rotation moments. As discussed above, several authors have speculated that there

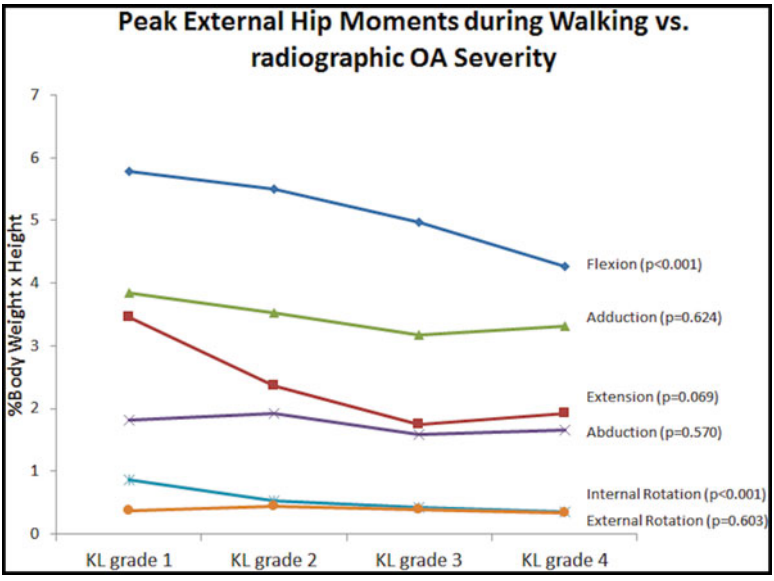


**Fig. 5.5** The peak external moments during walking plotted against radiographic hip OA severity. In general an unloading pattern is seen. Correlations were statistically significant for the peak flexion and internal rotation moments

is an association between this sagittal plane motion discontinuity and abnormal hip morphology. Together these findings provide at least circumstantial evidence for a link between abnormal hip structure and abnormal gait function in hip OA.

## *Gait Alterations, Structure, and Function Links, Before and After THA*

### Gait in Endstage Hip OA

Endstage hip OA, which is the indication for more than 80 % of all THAs [49], is associated with markedly abnormal gait. Most of the gait anomalies seen in endstage OA can be viewed as more severe forms of the adaptations discussed above. Slower gait speeds, reduced stride lengths, reduced range of motion, and markedly reduced peak external moments have all been reported. Figure 5.5 illustrates how subjects with moderate to severe hip OA (KL Grade 3 and 4) have markedly reduced external moments compared to their counterparts with less severe disease. Sagittal plane gait mechanics have been discussed in detail above; reductions in hip range of motion, peak flexion and peak extension moments are even more dramatic in endstage hip OA. Arguably the most vulnerable muscle group in hip OA and THA, however, is the hip abductors. The frontal and transverse plane gait moments reflect the role of these muscles so they are important to consider in a bit more detail. The peak hip adduction moment in endstage hip OA is markedly reduced compared to healthy subjects [50, 51]. As we have previously discussed, an external adduction moment must be balanced by an internal hip abduction moment. The hip abductors (i.e., gluteus medius and gluteus minimus) are primarily responsible for this balance, so this gait deficit is usually interpreted as a sign of dynamic abductor dysfunction. During walking, the hip abductors are also well positioned to balance transverse plane loads [52, 53]. Thus the reduced internal rotation and external rotation moments that are also seen before surgery [50, 51] may reflect abnormal function of these muscles as well.

### Structure Function Link in Endstage OA

Spatiotemporal and sagittal plane gait deficits in endstage OA may be associated with the sagittal plane motion restrictions associated with endstage OA and apparent on clinical exam. Loss of passive hip extension and hip flexion contractures are common in people with severe hip OA. Loss of passive extension is associated with restricted dynamic range of motion in the sagittal plane in people with hip OA as well as healthy elderly [37, 54, 55]. Hip extension is needed in the second half of stance to achieve "normal" gait patterns. Restricted hip extension will necessarily reduce stride length, which will in turn reduce speed. (Recall that speed = stride length × cadence). Flexion contractures can also influence the sagittal plane moments by affecting the position of the ground reaction force with respect to the hip center. If a patient has a flexed hip for most or all of stance, as

in the example shown in Fig. 5.3, even in late stance she may not be able to move the ground reaction force posterior to the hip center. This means it will not be possible to produce the large hip extension moment typically seen in late stance (refer back to Fig. 5.2c).

The frontal and transverse plane gait abnormalities could be partly explained by structural changes that happen within the abductor muscles. Hip OA is associated with atrophy and fatty infiltration of the hip abductors, as well as weakness [56–58]. While strength and external moments are not always directly correlated [59], these structural changes can still affect external moments. Maintaining a level pelvis during single limb stance is conventionally thought of as the primary role of the abductors during gait. If the abductors cannot function normally to achieve this, the body must compensate. This compensation is commonly done with exaggerated trunk lean. Even a small amount of lateral trunk lean moves the ground reaction force closer to the hip center in the frontal plane. This action thus reduces the effective moment arm and reduces the external adduction moment and thus the demand on the hip abductors. To understand the transverse plane moment deficits, consider both about a permanently flexed hip and weakened abductors. With persistent hip flexion, the anterior fibers of the gluteus medius and minimus, which contribute most to internal rotation, are being shortened and the posterior fibers, which contribute most to external rotation are being stretched. Neither position will result in an optimal fiber length for contraction, and the transverse plane moment generating capacity of the abductors will be diminished.

## *Gait After THA*

Along with dramatic improvements in pain and quality of life, THA improves most aspects of gait. In one study by our group in which subjects who underwent primary unilateral THA with one of two minimally invasive surgical approaches, after a decline in ROM and external moments 3 week after surgery, there was a dramatic improvement in most gait measures, with gait stabilizing by 6 months after surgery (Fig. 5.6) [51]. There are, however, significant gait abnormalities after surgery [60], that may in part reflect a persistence of abnormal preoperative gait patterns [50]. A review and meta-analysis by Ewen et al. summarizes several studies in which the gait of patients after THA has been compared to that of healthy controls and identifies several consistent deficits [60]. These are walking speed, stride length, sagittal plane hip range of motion and the peak adduction moment. The most dramatic gait abnormality is again the significant reduction seen in the hip adduction moment compared to healthy subjects. Not only is it well below normal both before and after surgery, from Fig. 5.6 we can appreciate that not much improvement takes place on average.

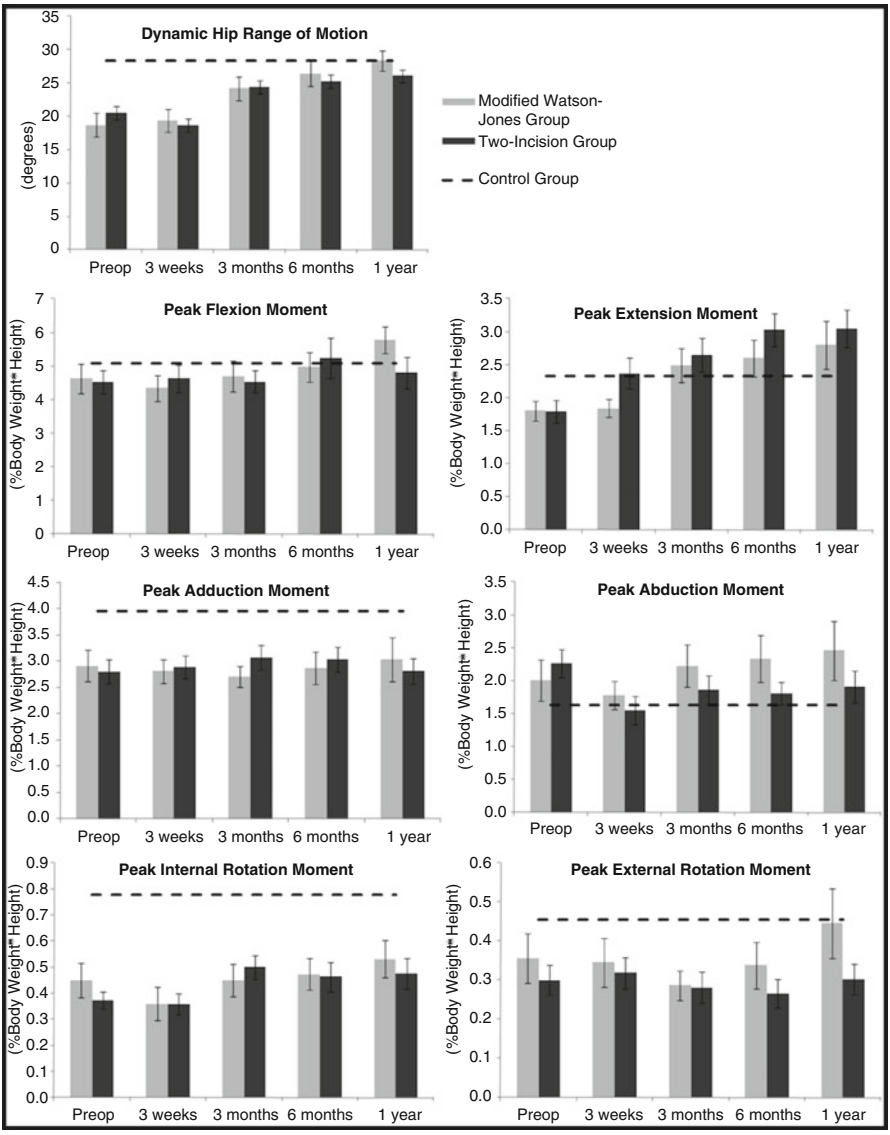


**Fig. 5.6** Peak external moments before and during the first year after primary unilateral total hip arthroplasty conducted with two minimally invasive approaches. There were no differences in rate of recovery between the two approach groups. *Horizontal lines* show mean values for a group of age-matched control subjects. [Reprinted from Foucher KC, Wimmer MA, Moisio KC, et al. Time course and extent of functional recovery during the first postoperative year after minimally invasive total hip arthroplasty with two different surgical approaches—a randomized controlled trial. Journal of Biomechanics 2011; 44(3): 372–378. With permission from Elsevier]

### Postoperative THA Gait as Alteration of the Structure–Function Relationship

While some gait abnormalities after THA may be attributable to the same factors that contribute to abnormal preoperative gait, postoperative joint geometry may also play a role. Neck length, femoral offset, cup inclination angle, and other aspects of hip joint geometry can all be directly or indirectly manipulated by the surgery. Although the relationships between joint geometry and implant longevity have been investigated, there have been few in vivo studies on the relationships between joint geometry and gait. We can consider these relationships indirectly, however, but reviewing what is known about the influence of hip joint geometry on muscle strength or function and on hip joint forces.

The joint geometry measures linked to adverse events, including poorer clinical outcomes, include shorter femoral neck, reduced femoral offset, or a more superior or lateral position of the hip center [61–67]. In theory, these positions may all reduce the moment generating capacity of the hip abductors [62, 68–70]. The result will be either that the muscles must produce more force to generate the same moment or that there must be a compensation to reduce the demand on these muscles [71].

These theoretical findings seem to hold true in recent and older studies. Reduced femoral offset relative to the healthy contralateral hip is associated with a reduced hip range of motion in the frontal plane [72, 73]. Lower offset is also associated with reduced abductor muscle strength [72, 74–76], and greater abductor muscle activation [76]. Higher hip centers are also linked to reduced abductor strength [77–80]. These findings suggest that indeed more force is needed from the muscles to accomplish the task at hand when joint geometry is different from what is optimal.

What does this mean in practice? Careful preoperative planning and templating is done to optimize joint geometry. However there has arisen recent concern in the literature that templating based on X-rays may underestimate femoral offset, particularly when hip or pelvic rotation is present [78–80]. Reduced offset can adversely impact hip abductor function—which is already compromised due to the effects of the underlying disease. Clinicians should be watchful for subtle deficits in abductor function. While joint geometry cannot be changed after surgery, rehabilitation interventions can help patients regain lost strength needed to overcome the residual disease effects or the effects of slightly suboptimal implant positioning.

## *Summary of This Section*

This section described how gait pathomechanics can be viewed as a disruption of the normal structure–function relationship of the hip joint and its surrounding structures. This is true throughout the spectrum of hip degenerative diseases from prearthritic conditions through recovery from THA. Considering this spectrum, it is noteworthy that dynamic hip extension, which is intimately linked to hip joint

structure, may be one of the first manifestations of functional impairment and one of the last to recover. The evolution of abductor muscle impairment in hip OA and its persistence after THA also raises interesting questions about the relationship between intramuscular structure and gait function, joint geometry, body position and gait function, and the role of implant positioning. Future work in developing patient-specific biomechanical models in hip OA and prearthritic conditions will lead to new advances in the coming years that promote optimal functional recovery.

## Gait Pathomechanics as Behavioral Response to Disease

### *Overview*

Although structure and function are intimately connected, the patient's experience of hip pathology revolves around the connection between pain and function. Pain, or more accurately, the inability to adequately modify activities to reduce pain, sends patients to a clinician. So it is also useful to consider pathomechanics in terms of people's conscious or unconscious strategies to alleviate pain. To do this, we will briefly revisit some of the gait adaptations seen in hip OA and THA.

### *Slowing Down and Other Spatiotemporal Gait Adjustments*

Walking speed has been proposed as a sixth vital sign [14, 15]. It can be easily measured clinically with little to no specialized equipment. Walking speed is associated with fall risk [81], incident disability, cognitive decline, and even mortality [15, 82, 83]. Thus, although all of the disorders discussed above have reduced walking speed as one of their main signs, the fact that walking speed does not return to normal after THA is particularly concerning.

In one study we measured self-selected normal walking speeds for subjects who had undergone primary unilateral THA, and a group of health controls, a gait analysis lab, then assessed habitual speed with ankle-worn activity monitors for 24 h in subjects' home environments [84]. We found that both subject groups walked faster in the gait lab than they did in external settings. This was perhaps due to a desire to perform well for observers. However the gap between lab-based speed and habitual speed was larger in the control group. Our interpretation was that people who had THAs might have wished to walk faster, but may have been unable to do so because of the residual gait abnormalities discussed above. This "speed-gap" may have important implications for subjects' overall health and well-being. Evidence of a functional speed-gap also comes from Mauffiuletti et al., who showed that gait characteristics were comparable between subjects after THA and controls when walking at a self-selected normal speed, but that differences emerged when subjects walked a fast speeds [85].

In another recent study of 163 subjects tested before and after primary unilateral THA [86], we examined the relationships among walking speed, kinematic and kinetic gait variables, and Harris hip scores (HHS) before and after THA. Notably, there were no statistically significant relationships between pain and walking speed either before or after THA, or between change in pain and change in walking speed ($R = 0.120$, $p = 0.154$). This suggests that slower walking speeds are not actually a response to pain, but may be more strongly associated with other aspects of abnormal hip biomechanics. Indeed, speed was significantly correlated with all of the other kinematic and kinetic gait variables measured about the hip ($R = 0.178$–$0.614$, $p < 0.001$ to $p = 0.018$). In a way, this is good news because it means that speed deficits could be amenable to rehabilitation or other interventions.

## *Gait Kinematics and Kinetics*

Gait kinematics and kinetics are not volitionally adjusted in response to pain and other behaviorally experienced aspects of hip disease. Lab-measured hip gait kinematics and kinetics, however, directly influenced by walking speed [87], and related to self-reported pain and other aspects of clinical function. In patients with hip dysplasia, Jacobsen et al. found that self-reported pain and sports/recreation function scores were significantly correlated with the peak hip extension angle and the peak external extension moment [27]. We have had similar findings at the other end of the disease spectrum.

In our study of THA subjects discussed above [86], we identified several associations between pain or self-reported function and gait kinematic and kinetic variables. More preoperative pain was associated with lower peak extension moments and more having a greater postoperative improvement in pain was associated with greater increases in dynamic sagittal plane range of motion and the peak external rotation moment. In contrast to pain, both preoperative and postoperative hip function scores were strongly associated with walking speeds. After statistically controlling for these relationships, we found that improvement in self-reported gait function was associated with improvement in the hip range of motion, peak adduction moment, and peak external rotation moment. Note that these are the same gait variables in which recovery has been found to be lacking [50, 51, 60].

## *Summary of This Section*

Briefly, this section should have convinced you that, while structure and function are intimately related, function is also influenced by the patient's symptoms and experience of disease. It is not truly possible to separate these two concepts. Clinicians are asked to treat pain by restoring structure, with the sometimes indirect goal of restoring function. The assumption that restoring structure will restore

function is not always accurate. So far the consensus in both the surgical treatment of hip dysplasia and FAI is that even when structure is normalized, functional deficits can persist. The problem of incomplete functional recovery after THA is also gaining recognition. With a more thorough understanding of the connections among gait mechanics, structure, function, and symptoms, clinicians of the future will have the opportunity to further improve recognition and treatment of hip pathology.

## Final Summary and Recommendations for Use of Clinical Gait Analysis with Hip Pathology

This chapter has presented variables that describe the spatiotemporal characteristics of walking, hip motions, and, indirectly, hip muscle activity and function. By way of a summary, this final section will touch on some ways that clinicians may choose to apply these concepts, and briefly mention some methods for doing so.

Most of this chapter has dealt with quantitative gait analysis as conducted in a fully equipped gait analysis laboratory. However, one of the most important gait measures can be assessed in the clinic with only a stopwatch. The 10 or 4 m walk tests can be used to assess speed in a clinical setting [15]. Walking speed is reduced in all of the hip conditions described here and does not return to normal after THA. Walking speed may also be directly linked to the hip structural changes associated with hip pathology. We have argued that limitations in hip extension, which again is seen in each of the conditions discussed, can lead to reduced walking speeds via reduced stride length. Walking speed is associated with self-reported function in people after THA, and as such is a good objective reflection of the patient's experience of disease that has good clinimetric properties compared to PROMs. Finally, in older adults, walking speed is linked to incident disability, other mobility restrictions, and a host of other general health problems. It is important to know whether or not hip pathology is the source of reduced walking speed in your older patients. An assessment of walking speed is an excellent complement to other clinical tests that adds little time and no cost to the clinical encounter.

Walking speed, along with ever more complicated gait measures, can now be measured outside of the traditional lab setting as well. Instrumented mats and walkways can be used to measure spatiotemporal gait characteristics. Body worn sensors and activity monitors are also rapidly evolving [88]. These technologies, often based on accelerometers, can be used to quantify speed and other spatiotemporal gait variables as we have discussed [84]. Activity levels can also be quantified using these devices [51, 89, 90]. Some technologies can also be used to actually obtain joint angles as well [88]. An important feature of these types of devices is that feedback can be given to the wearer in real time.

Detailed kinematic and kinetic analysis can be performed most accurately in a gait laboratory. A basic gait lab will have at least four cameras (eight to ten are most common now), at least one multi-directional forceplate embedded in a walkway at least

8–10 m long. Reflective markers are placed in defined locations on the lower extremities or the entire body. At least two markers are necessary to define the proximal and distal ends of each segment; a third marker is necessary if rotations will be measured. The cameras typically emit pulses of infrared light and record the reflection of the light from the markers. Before testing, the system is calibrated so that the 3D coordinates of each point in the volume space is known. Then as long as at least two cameras can "see" a marker, its 3D position can be determined. From marker locations, ground reaction forces, measured by the force plates, and joint centers either determined by marker positions or by other methods, *inverse dynamics* can be used to calculated external moments about the hip and other joints. There are many vendors that produce and distribute camera systems, forceplates, and analysis software.

Today it would be unusual to find an academic medical center or major research hospital without at least one gait analysis lab. Labs are typically housed within orthopedic surgery, physical therapy, kinesiology, or bioengineering departments. Many institutions may have more than one. There is strong support for the efficacy of clinical gait analysis [91]; however, its use is still currently primarily in children. Gait analysis has been investigated as a potential outcome tool for FAI [30, 34] and hip OA [92, 93]. In addition, Bhave et al. published a case series in which gait analysis and complementary assessments were used to customize rehabilitation intervention for THA patients who failed "conventional" therapy [94]. The potential is there. With additional work in this area, there is great potential for gait analysis to be a useful, and reimbursable, clinical tool for many type of hip pathology in various patient populations. Clinicians should consider ordering gait analysis studies where available and reimbursable.

There are several important concepts that were beyond the scope of this chapter but are relevant to this material. First, activities other than walking were not considered here. More intense activities such as squatting, stair climbing, and running may be needed to fully assess the capabilities and limitations of younger patients with the prearthritic conditions [27, 34, 39]. Second, we have neglected the effects of hip pathology on joints other than the affected hip. For example, Eitzen et al. nicely describe the sagittal plane kinematics and kinetic changes at the knee and ankle associated with mild to moderate hip OA [42]. Shakoor et al. have established that the pattern of joint-to-joint progression of OA is "nonrandom" and can be linked to the abnormal hip joint loading patterns [95, 96]. We have also investigated the longitudinal effects of THA on the contralateral hip and knee [97, 98]. Finally, numerical modeling was only hinted at here. Musculoskeletal models, including finite element analysis, discrete element analysis, and simulations, have great potential to be invaluable clinical tools in the future [21, 99]. Predicting and personalizing interventions will no doubt be essential in the years to come.

After reading this chapter, you should be convinced of the importance of understanding gait, and have gained a foundation for how to approach gait pathomechanics associated with hip disease. When approaching the patient with hip pain or known hip disease, gait should always be assessed at least observationally. Whether gait analysis is conducted just using visual observation, or in a gait lab, watch for

events at key points in the gait cycle. For example, the transitions from double limb stance to single limb stance, and the midpoint of the stance phase of gait have been shown to be important in multiple pathological conditions. Try to make connections between walking and other activities that may be meaningful to the patient. When available, consider utilizing quantitative gait analysis, and documenting how it guides your clinical practice. Understand the importance of gait function in your patients' daily lives—doing so is essential to providing good patient care.

## References


1. Malchau H, Herberts P, Eisler T, et al. The Swedish Total Hip Replacement Register. J Bone Joint Surg Am. 2002;84-A Suppl 2:2–20.
2. Hallan G, Lie S, Furnes O, et al. Medium-and long-term performance of 11 516 uncemented primary femoral stems from the Norwegian arthroplasty register. J Bone Joint Surg Br. 2007;89:1574–80.
3. Katz JN, Wright EA, Wright J, et al. Twelve-year risk of revision after primary total hip replacement in the US Medicare population. J Bone Joint Surg Am. 2012;94:1825–32.
4. Ghomrawi HMK, Schackman BR, Mushlin AI. Appropriateness criteria and elective procedures — total joint arthroplasty. N Engl J Med. 2012;367:2467–9.
5. Andrawis JP, Chenok KE, Bozic KJ. Health policy implications of outcomes measurement in orthopaedics. Clin Orthop Relat Res. 2013;471(11):3475–81.
6. Judge A. Patient-reported outcomes one year after primary hip replacement in a European collaborative cohort. Arthritis Rheum. 2010;62:480–8.
7. Singh JA, Lewallen DG. Patient-level clinically meaningful improvements in activities of daily living and pain after total hip arthroplasty: data from a large US institutional registry. Rheumatology (Oxford). 2013;52:1108–18.
8. Hawker GA, Badley EM, Borkhoff CM, et al. Which patients are most likely to benefit from total joint arthroplasty? Arthritis Rheum. 2013;65:1243–52.
9. Mancuso CA, Jout J, Salvati EA, et al. Fulfillment of patients' expectations for total hip arthroplasty. J Bone Joint Surg Am. 2009;91:2073–89.
10. Hobbs N, Dixon D, Rasmussen S, et al. Patient preoperative expectations of total hip replacement in European orthopedic centers. Arthritis Care Res (Hoboken). 2011;63:1521–7.
11. Heiberg KE, Ekeland A, Mengshoel AM. Functional improvements desired by patients before and in the first year after total hip arthroplasty. BMC Musculoskelet Disord. 2013;14:243.
12. Kurtz S, Ong K, Lau E, et al. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am. 2007;89:780–5.
13. Kurtz SM, Lau E, Ong K, et al. Future young patient demand for primary and revision joint replacement: national projections from 2010 to 2030. Clin Orthop Relat Res. 2009;467:2606–12.
14. Fritz S, Lusardi M. White paper: "walking speed: the sixth vital sign". J Geriatr Phys Ther. 2009;32:46–9.
15. Middleton A, Fritz SL, Lusardi M. Walking speed: the functional vital sign. J Aging Phys Act. 2015;23:314–22.
16. Perry J, Burnfield JM. Gait analysis: normal and pathological function. 2nd ed. Thorofare, NJ: Slack; 2010. p. 551.
17. Hurwitz DE, Foucher KC, Andriacchi TP. A new parametric approach for modeling hip forces during gait. J Biomech. 2003;36:113–9.
18. Heller MO, Bergmann G, Kassi JP, et al. Determination of muscle loading at the hip joint for use in pre-clinical testing. J Biomech. 2005;38:1155–63.
19. Harris-Hayes M, Royer NK. Relationship of acetabular dysplasia and femoroacetabular impingement to hip osteoarthritis: a focused review. PM R. 2011;3:1055–67.e1.

20. Russell ME, Shivanna KH, Grosland NM, et al. Cartilage contact pressure elevations in dysplastic hips: a chronic overload model. J Orthop Surg Res. 2006;1:6.
21. Henak CR, Anderson AE, Weiss JA. Subject-specific analysis of joint contact mechanics: application to the study of osteoarthritis and surgical planning. J Biomech Eng. 2013;135:021003.
22. Andriacchi T, Mndermann A, Smith RL, et al. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann Biomed Eng. 2004;32:447–57.
23. Felson DT. Osteoarthritis as a disease of mechanics. Osteoarthritis Cartilage. 2013;21:10–5.
24. Pedersen EN, Alkjaer T, Soballe K, et al. Walking pattern in 9 women with hip dysplasia 18 months after periacetabular osteotomy. Acta Orthop. 2006;77:203–8.
25. Omeroglu H, Yavuzer G, Bicimoglu A, et al. No detectable major changes in gait analysis after soft tissue release in DDH. Clin Orthop Relat Res. 2008;466:856–61. doi:10.1007/s11999-008-0146-2.
26. Sucato DJ, Tulchin K, Shrader MW, et al. Gait, hip strength and functional outcomes after a Ganz periacetabular osteotomy for adolescent hip dysplasia. J Pediatr Orthop. 2010;30:344–50. doi:10.1097/BPO.0b013e3181d9bfa2.
27. Jacobsen JS, Nielsen DB, Sorensen H, et al. Changes in walking and running in patients with hip dysplasia. Acta Orthop. 2013;84:265–70. doi:10.3109/17453674.2013.792030.
28. Thorp LE, Sumner DR, Block JA, et al. Knee joint loading differs in individuals with mild compared with moderate medial knee osteoarthritis. Arthritis Rheum. 2006;54:3842–9.
29. Foucher KC, Schlink BR, Shakoor N, et al. Sagittal plane hip motion reversals during walking are associated with disease severity and poorer function in subjects with hip osteoarthritis. J Biomech. 2012;45:1365.
30. Alradwan H, Khan M, Hamel-Smith Grassby M, et al. Gait and lower extremity kinematic analysis as an outcome measure after femoroacetabular impingement surgery. Arthroscopy. 2015;31:339–44. pii: S0749-8063(14)00537-4.
31. Diamond LE, Dobson FL, Bennell KL, et al. Physical impairments and activity limitations in people with femoroacetabular impingement: a systematic review. Br J Sports Med. 2015;49:230–42.
32. Kennedy MJ, Lamontagne M, Beaule PE. Femoroacetabular impingement alters hip and pelvic biomechanics during gait walking biomechanics of FAI. Gait Posture. 2009;30:41.
33. Rylander JH, Shu B, Andriacchi TP, et al. Preoperative and postoperative sagittal plane hip kinematics in patients with femoroacetabular impingement during level walking. Am J Sports Med. 2011;39(Suppl):36S–42.
34. Rylander J, Shu B, Favre J, et al. Functional testing provides unique insights into the pathomechanics of femoroacetabular impingement and an objective basis for evaluating treatment outcome. J Orthop Res. 2013;31:1461–8. doi:10.1002/jor.22375.
35. Hunt MA, Guenther JR, Gilbart MK. Kinematic and kinetic differences during walking in patients with and without symptomatic femoroacetabular impingement. Clin Biomech (Bristol, Avon). 2013;28:519–23. doi:10.1016/j.clinbiomech.2013.05.002.
36. Brisson N, Lamontagne M, Kennedy MJ, et al. The effects of cam femoroacetabular impingement corrective surgery on lower-extremity gait biomechanics. Gait Posture. 2013;37:258–63. doi:10.1016/j.gaitpost.2012.07.016.
37. Hurwitz DE, Hulet CH, Andriacchi TP, et al. Gait compensations in patients with osteoarthritis of the hip and their relationship to pain and passive hip motion. J Orthop Res. 1997;15:629–35.
38. Michaud B, Jackson MI, Prince F, et al. Can one angle be simply subtracted from another to determine range of motion in three-dimensional motion analysis? Comput Methods Biomech Biomed Engin. 2014;17:507–15.
39. Lamontagne M, Brisson N, Kennedy MJ, et al. Preoperative and postoperative lower-extremity joint and pelvic kinematics during maximal squatting of patients with cam femoro-acetabular impingement. J Bone Joint Surg Am. 2011;93 Suppl 2:40–5.
40. Liu R, Wen X, Tong Z, et al. Changes of gluteus medius muscle in the adult patients with unilateral developmental dysplasia of the hip. BMC Musculoskelet Disord. 2012;13:101.

41. Casartelli NC, Maffiuletti NA, Item-Glatthorn JF, et al. Hip muscle weakness in patients with symptomatic femoroacetabular impingement. Osteoarthritis Cartilage. 2011;19:816–21.
42. Eitzen I, Fernandes L, Nordsletten L, et al. Sagittal plane gait characteristics in hip osteoarthritis patients with mild to moderate symptoms compared to healthy controls: a cross-sectional study. BMC Musculoskelet Disord. 2012;13:258.
43. Constantinou M, Barrett R, Brown M, et al. Spatial-temporal gait characteristics in individuals with hip osteoarthritis: a systematic literature review and meta-analysis. J Orthop Sports Phys Ther. 2014;44:291-B7. doi:10.2519/jospt.2014.4634.
44. Nicholls AS, Kiran A, Pollard TC, et al. The association between hip morphology parameters and 19-year risk of end-stage osteoarthritis in the hip: a nested case–control study. Arthritis Rheum. 2011;63:3392–400.
45. Agricola R, Heijboer M, Bierma-Zeinstra SMA, et al. Cam impingement causes osteoarthritis of the hip: a nationwide prospective cohort study (CHECK). Ann Rheum Dis. 2013;72: 918–23.
46. Agricola R, Heijboer MP, Roze RH, et al. Pincer deformity does not lead to osteoarthritis of the hip whereas acetabular dysplasia does: acetabular coverage and development of osteoarthritis in a nationwide prospective cohort study (CHECK). Osteoarthritis Cartilage. 2013;21:1514–21. doi:10.1016/j.joca.2013.07.004.
47. Pollard TC, Batra RN, Judge A, et al. The hereditary predisposition to hip osteoarthritis and its association with abnormal joint morphology. Osteoarthritis Cartilage. 2013;21:314–21. doi:10.1016/j.joca.2012.10.015.
48. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16:494–502.
49. Bone and Joint Decade. The burden of musculoskeletal diseases in the United States. Prevalence, societal and economic cost, 2008.
50. Foucher KC, Hurwitz DE, Wimmer MA. Preoperative gait adaptations persist one year after surgery in clinically well-functioning total hip replacement patients. J Biomech. 2007;40:3432–7.
51. Foucher KC, Wimmer MA, Moisio KC, et al. Time course and extent of functional recovery during the first postoperative year after minimally invasive total hip arthroplasty with two different surgical approaches—a randomized controlled trial. J Biomech. 2011;44:372–8.
52. Flack NA, Nicholson HD, Woodley SJ. A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus minimus, and tensor fascia lata. Clin Anat. 2012;25:697–708.
53. Flack N, Nicholson H, Woodley S. The anatomy of the hip abductor muscles. Clin Anat. 2014;27:241–53.
54. Kerrigan DC, Lee LW, Collins JJ, et al. Reduced hip extension during walking: healthy elderly and fallers versus young adults. Arch Phys Med Rehabil. 2001;82:26–30.
55. Hulet C, Hurwitz DE, Andriacchi TP, et al. Functional gait adaptations in patients with painful hip. Rev Chir Orthop Reparatrice Appar Mot. 2000;86:581–9.
56. Arokoski MH, Arokoski JP, Haara M, et al. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. J Rheumatol. 2002;29:2185–95.
57. Rasch A, Bystrom AH, Dalen N, et al. Reduced muscle radiological density, cross-sectional area, and strength of major hip and knee muscles in 22 patients with hip osteoarthritis. Acta Orthop. 2007;78:505–10.
58. Rasch A, Dalen N, Berg HE. Muscle strength, gait, and balance in 20 patients with hip osteoarthritis followed for 2 years after THA. Acta Orthop. 2010;81:183–8.
59. Rutherford DJ, Hubley-Kozey C. Explaining the hip adduction moment variability during gait: implications for hip abductor strengthening. Clin Biomech (Bristol, Avon). 2009;24:267–73.
60. Ewen A, Stewart S, St Clair Gibson A, et al. Post-operative gait analysis in total hip replacement patients—a review of current literature and meta-analysis. Gait Posture. 2012;36:1–6.
61. Yoder SA, Brand RA, Pedersen DR, et al. Total hip acetabular component position affects component loosening rates. Clin Orthop Relat Res. 1988;228:79–87.

62. Doehring TC, Rubash HE, Shelley FJ, et al. Effect of superior and superolateral relocations of the hip center on hip joint forces. An experimental and analytical analysis. J Arthroplasty. 1996;11:693–703.
63. Doehring TC, Rubash HE, Dore DE. Micromotion measurements with hip center and modular neck length alterations. Clin Orthop Relat Res. 1999;362:230–9.
64. Zahiri CA, Schmalzried TP, Ebramzadeh E, et al. Lessons learned from loosening of the McKee-Farrar metal-on-metal total hip replacement. J Arthroplasty. 1999;14:326–32.
65. Wan Z, Boutary M, Dorr LD. The influence of acetabular component position on wear in total hip arthroplasty. J Arthroplasty. 2008;23:51–6.
66. Cassidy KA, Noticewala MS, Macaulay W, et al. Effect of femoral offset on pain and function after total hip arthroplasty. J Arthroplasty. 2012;27:1863–9.
67. Judge A, Arden NK, Batra RN, et al. The association of patient characteristics and surgical variables on symptoms of pain and function over 5 years following primary hip-replacement surgery: a prospective cohort study. BMJ Open. 2013;3.
68. Delp SL, Maloney W. Effects of hip center location on the moment-generating capacity of the muscles. J Biomech. 1993;26:485–99.
69. Delp SL, Komattu AV, Wixson RL. Superior displacement of the hip in total joint replacement: effects of prosthetic neck length, neck-stem angle, and anteversion angle on the moment-generating capacity of the muscles. J Orthop Res. 1994;12:860–70.
70. Delp SL, Wixson RL, Komattu AV, et al. How superior placement of the joint center in hip arthroplasty affects the abductor muscles. Clin Orthop Relat Res. 1996;328:137–46.
71. Lengsfeld M, Bassaly A, Boudriot U, et al. Size and direction of hip joint forces associated with various positions of the acetabulum. J Arthroplasty. 2000;15:314–20.
72. McGrory BJ, Morrey BF, Cahalan TD, et al. Effect of femoral offset on range of motion and abductor muscle strength after total hip arthroplasty. J Bone Joint Surg Br. 1995;77:865–9.
73. Sariali E, Klouche S, Mouttet A, et al. The effect of femoral offset modification on gait after total hip arthroplasty. Acta Orthop. 2014;85:123–7.
74. Asayama I, Chamnongkich S, Simpson KJ, et al. Reconstructed hip joint position and abductor muscle strength after total hip arthroplasty. J Arthroplasty. 2005;20:414–20.
75. Yamaguchi T, Naito M, Asayama I, et al. Total hip arthroplasty: the relationship between posterolateral reconstruction, abductor muscle strength, and femoral offset. J Orthop Surg (Hong Kong). 2004;12:164–7.
76. Chamnongkich S, Asayama I, Kinsey TL, et al. Difference in hip prosthesis femoral offset affects hip abductor strength and gait characteristics during obstacle crossing. Orthop Clin North Am. 2012;43:e48–58. doi:10.1016/j.ocl.2012.07.008.
77. Kiyama T, Naito M, Shitama H, et al. Effect of superior placement of the hip center on abductor muscle strength in total hip arthroplasty. J Arthroplasty. 2009;24:240–5. doi:10.1016/j.arth.2008.08.012.
78. Merle C, Waldstein W, Pegg E, et al. Femoral offset is underestimated on anteroposterior radiographs of the pelvis but accurately assessed on anteroposterior radiographs of the hip. J Bone Joint Surg Br. 2012;94:477–82. doi:10.1302/0301-620X.94B4.28067.
79. Lechler P, Frink M, Gulati A, et al. The influence of hip rotation on femoral offset in plain radiographs. Acta Orthop. 2014;85:389–95.
80. Hassani H, Cherix S, Ek ET, et al. Comparisons of preoperative three-dimensional planning and surgical reconstruction in primary cementless total hip arthroplasty. J Arthroplasty. 2014;29:1273–7. doi:10.1016/j.arth.2013.12.033.
81. Marques NR, LaRoche DP, Hallal CZ, et al. Association between energy cost of walking, muscle activation, and biomechanical parameters in older female fallers and non-fallers. Clin Biomech (Bristol, Avon). 2013;28:330–6. doi:10.1016/j.clinbiomech.2013.01.004.
82. Kuo HK, Leveille SG, Yen CJ, et al. Exploring how peak leg power and usual gait speed are linked to late-life disability: data from the National Health and Nutrition Examination Survey (NHANES), 1999–2002. Am J Phys Med Rehabil. 2006;85:650–8.
83. Kuo HK, Leveille SG, Yu YH, et al. Cognitive function, habitual gait speed, and late-life disability in the National Health and Nutrition Examination Survey (NHANES) 1999–2002. Gerontology. 2007;53:102–10.

84. Foucher KC, Thorp LE, Hildebrand M, et al. Differences in preferred walking speeds in a gait lab compared to the "real world" after total hip replacement. Arch Phys Med Rehabil. 2010;91:1390–5.
85. Maffiuletti NA, Impellizzeri FM, Widler K, et al. Spatiotemporal parameters of gait after total hip replacement: anterior versus posterior approach. Orthop Clin North Am. 2009;40:407–15.
86. Behery OA, Foucher KC. Are Harris hip scores and gait mechanics related before and after THA? Clin Orthop Relat Res. 2014;472:3452–61.
87. Moisio KC, Sumner DR, Shott S, et al. Normalization of joint moments during gait: a comparison of two techniques. J Biomech. 2003;36:599–603.
88. Shull PB, Jirattigalachote W, Hunt MA, et al. Quantified self and human movement: a review on the clinical impact of wearable sensing and feedback for gait analysis and intervention. Gait Posture. 2014;40:11–9. doi:10.1016/j.gaitpost.2014.03.189.
89. Harding P, Holland AE, Delany C, et al. Do activity levels increase after total hip and knee arthroplasty? Clin Orthop Relat Res. 2013;472:1502–11. doi:10.1007/s11999-013-3427-3.
90. Kuhn M, Harris-Hayes M, Steger-May K, et al. Total hip arthroplasty in patients 50 years or less: do we improve activity profiles? J Arthroplasty. 2013;28:872–6. doi:10.1016/j.arth.2012.10.009.
91. Wren TA, Gorton 3rd GE, Ounpuu S, et al. Efficacy of clinical gait analysis: a systematic review. Gait Posture. 2011;34:149–53. doi:10.1016/j.gaitpost.2011.03.027.
92. Ornetti P, Maillefert JF, Laroche D, et al. Gait analysis as a quantifiable outcome measure in hip or knee osteoarthritis: a systematic review. Joint Bone Spine. 2010;77:421–5.
93. Laroche D, Duval A, Morisset C, et al. Test-retest reliability of 3D kinematic gait variables in hip osteoarthritis patients. Osteoarthritis Cartilage. 2011;19:194–9.
94. Bhave A, Marker DR, Seyler TM, et al. Functional problems and treatment solutions after total hip arthroplasty. J Arthroplasty. 2007;22:116–24.
95. Shakoor N, Block JA, Shott S, et al. Nonrandom evolution of end-stage osteoarthritis of the lower limbs. Arthritis Rheum. 2002;46:3185–9.
96. Shakoor N, Hurwitz DE, Block JA, et al. Asymmetric knee loading in advanced unilateral hip osteoarthritis. Arthritis Rheum. 2003;48:1556–61.
97. Foucher KC, Wimmer MA. Contralateral hip and knee gait biomechanics are unchanged by total hip replacement for unilateral hip osteoarthritis. Gait Posture. 2012;35:61–5.
98. Foucher KC, Wimmer MA. Does hip implant positioning affect the peak external adduction moments of the healthy knees of subjects with total hip replacements? J Orthop Res. 2013;31:1187–94. doi:10.1002/jor.22350.
99. Anderson DD, Iyer KS, Segal NA, et al. Implementation of discrete element analysis for subject-specific, population-wide investigations of habitual contact stress exposure. J Appl Biomech. 2010;26:215–23.