



Femoroacetabular Impingement: Fact, Fiction, or Fantasy?

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OBJECTIVE. Proponents of femoroacetabular impingement (FAI) now claim that FAI is an important risk factor for hip osteoarthritis and argue that early, aggressive treatment is indicated to stave off long-term complications. The result is more young patients undergoing corrective surgery; does the literature support these claims or has hype trumped reality? This article critically reviews these assertions together with the current scientific evidence that defends (or refutes) them.

CONCLUSION. Each reader will need to weigh the evidence carefully when interpreting images or planning management for patients with possible FAI.

I come to bury Caesar, not to praise him [1].

When Mark Antony eulogized the slain Caesar to his friends, Romans, and countrymen, he faced an uphill battle. Brutus, speaking on behalf of Caesar's murderers, had already explained his interpretation of Caesar's acts to the crowd and convinced them that the conspirators' cause was just. Antony had to subsequently show the plebeians, through a more considered analysis of the same facts that Brutus used, that in fact Caesar had been wronged. But Antony needed to tread carefully, using subtle irony as in the quotation above, so that he would not immediately be shouted down by the throng who already thought that they knew the truth. Unlike Antony, who slyly supported Caesar by insincerely "burying" him, I come to take femoroacetabular impingement (FAI) down a notch by sarcastically singing some of its praises—my apologies to the Bard.

FAI is a recently recognized syndrome with characteristic imaging and clinical findings and evolving management strategies. I will assume that the reader is already familiar with the salient features of the condition or can refer to one of the many recently published reviews [2–4]. Briefly, certain configurations of the bones in the hip joint predispose the labrum and articular cartilage to damage when the anterosuperior femoral head and neck abut against the acetabular rim during hip flexion and rotation. The primary femoral

lesion is asphericity: For simplicity, I will regard all the different forms of proximal femur distortion—including the pistol-grip deformity, decreased head-neck offset, increased alpha angle, overgrowth of the femoral head epiphysis, subclinical slipped epiphysis, and so on—as being encompassed by this concept. The main morphologic feature on the acetabular socket side is anterior overcoverage (including coxa profunda, acetabular retroversion, and lateral rim lesions). Most symptomatic hips have both femoral (cam) and acetabular (pincer) factors, which I will refer to as "FAI morphology." Other anatomic features, such as decreased femoral torsion, may contribute as well [5]. There are good data that FAI syndrome is a source of symptoms (pain and limited motion) in some patients and that treatment (surgical removal of the impinging bone structures together with treatment of associated intraarticular soft-tissue lesions) may relieve these symptoms.

Because the initial descriptions of FAI syndrome are barely 15 years old and are still being refined, little longitudinal data are available about the natural history of the disease. Although the morphologic bone changes, labral lesions, and articular cartilage infractions of the FAI syndrome are frequently observed in hips with clinical and radiographic osteoarthritis, the data proving causality are less certain. Nevertheless, some researchers believe that the majority of cases of "primary" hip os-

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teoarthritis are caused by FAI [6, 7]. Additionally some surgeons now claim that treatment of FAI not only relieves symptoms but also prevents or delays progression of osteoarthritis [7–9]. On the other hand, most radiologists who routinely interpret hip imaging studies should realize that reality does not jibe with this overly broad definition of FAI and its links to premature hip osteoarthritis. First, as more sensitive means are devised to show the characteristic morphologic changes (from new oblique radiographic projections to radially oriented MR planes to 3D surface-rendered CT), the number of hips identified with FAI morphology continues to soar. Some days it seems that 25–50% of the entire population—whether they have symptoms or not—show some finding associated with FAI. However, until 10 years ago, we rarely if ever made this diagnosis or treated these patients. If we were overlooking so many patients with FAI for the whole of the 20th century and if untreated FAI leads to premature hip osteoarthritis, why aren't there countless (relatively young) people walking around now with crippling hip osteoarthritis? If FAI syndrome really is that common and has been underdiagnosed and untreated in the past, shouldn't almost every adult have had their hip replaced by now? Or is this condition a new one that only appeared in the population in the past few years? Even the popular press now has an opinion: A recent *New York Times* article [10] on the increase in FAI procedures notes the lack of solid evidence and asks, "Might the bumps or irregular shapes they call impingement be just normal variations?" Are cam and pincer just new monikers describing normal differences in the population?

This article grew out of a debate at the 2011 International Skeletal Society meeting

that I was invited to present with an orthopedic surgeon and my accompanying syllabus entry. This article reviews the current scientific data on FAI in an effort to support the true assertions and to debunk some of the myths and dogma currently espoused on the subject. Obviously as research progresses and the FAI concept is further refined, the line between fact and fantasy will likely shift. This article is only meant to reflect our (my) current level of understanding. It is possible (albeit unlikely in my opinion) that all of the claims attributed to FAI will eventually prove true once the dust settles.

Assertion I: Femoroacetabular Impingement Exists as a Distinct Syndrome—Fact

There is ample evidence supporting the existence of FAI as a syndrome as long as FAI is defined by specific symptoms (groin pain exacerbated by hip flexion-rotation with decreased range of motion), signs (positive clinical impingement test [i.e., pain reproduced by hip flexion, internal rotation, and adduction]), imaging findings (FAI morphology on the femoral or socket side), and demographics. A positive impingement test alone, however, is not a specific indication of the FAI syndrome [3]. Results of several studies indicate that the impingement test actually detects acetabular labral tears [11–13]. As I will show, these tears occur both in the FAI syndrome and in other conditions, so the predictive value of a positive impingement test will depend on the patient population to which it is applied. For example, patients with labral tears due to iliopsoas impingement—a condition distinct from FAI—also have positive impingement tests [14]. Thus the reader

should cautiously interpret studies in the literature that evaluate treatment responses in FAI patients if only a positive impingement test is used to define the FAI population. Second, some investigators use pain relief after an intraarticular anesthetic injection as a sign of FAI. This finding is also nonspecific and can be seen in patients with labral tears and chondral abnormalities independent of the presence of FAI [14, 15].

Corollary IA: Impingement Between the Femur and Acetabulum Is Abnormal—Fiction, Probably

At surgery in patients with FAI syndrome, the anterolateral proximal femur is seen to impact on the rim of the acetabular socket when the hip is flexed and rotated, which several investigators use to argue for the mechanism of FAI [6]. Missing from the literature are observations of hips without FAI put through the same maneuvers: Do asymptomatic hips or those with "normal" morphology also show this abutment between the bones? If the answer is yes, then that fact would argue impingement alone is not pathologic. Although asymptomatic hips are not typically subject to surgical procedures, *in vivo* studies have been accomplished using imaging. For example, Yamamura et al. [16] performed open MR examinations of five asymptomatic Japanese women, none of whom had FAI morphology, while the subjects reproduced several postures common in daily activities. MR studies of all five showed impingement of the femur against the acetabulum (to the point where the femoral head was actually levered from the socket by the anterior acetabular rim) while they were sitting with the legs in a W position, but none developed symp-

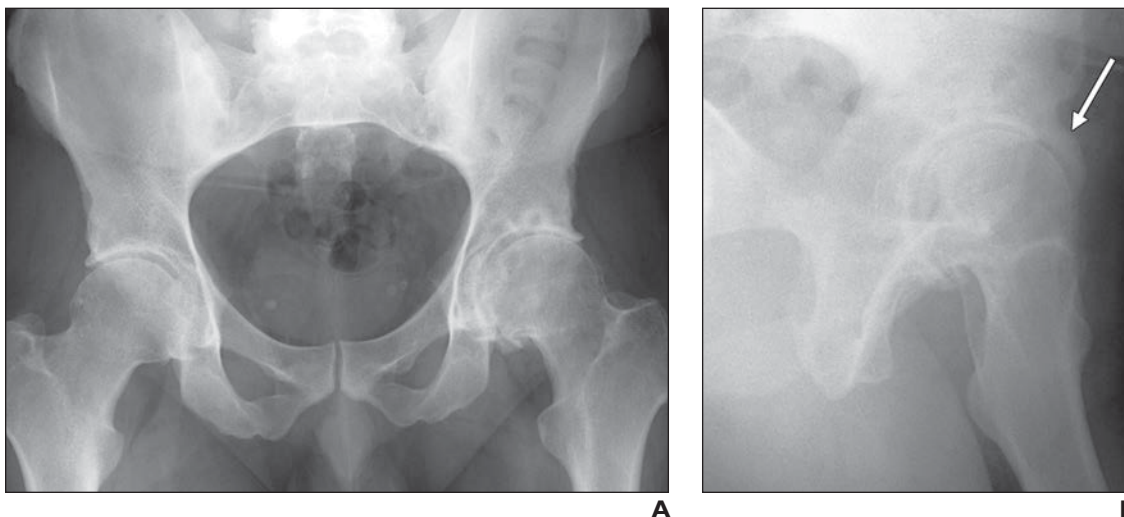


Fig. 1—Chronic hip pain in 42-year-old man. **A** and **B**, Anteroposterior pelvic radiograph (**A**) and false profile view of hip (**B**). Patient had been labeled as having femoroacetabular impingement in part because of acetabular "overcoverage" (arrow, **B**) and early-onset hip arthritis. Hip disease was actually due to long-standing ankylosing spondylitis. Note symmetrically fused sacroiliac joints in **A**.

toms in this position and all used this pose in daily activities. Interestingly, although Japanese life entails a lot of squatting and “W sitting” where impingement should be common, hip osteoarthritis is relatively rare (other than cases caused by dysplasia) in Japan and FAI accounts for fewer than 1% of the cases of hip osteoarthritis seen there [17].

Corollary 1B: Treating Femoroacetabular Impingement Relieves the Symptoms—Fact, But...

Multiple studies have investigated short-term clinical response to FAI surgery. Altering the FAI morphology in symptomatic hips with the FAI syndrome at arthroscopy or open surgery reduces pain, increases motion, and improves function [9, 18–20]. Overall quality of life also improves 1 year after arthroscopic FAI treatment [21]. Outcomes are poorer in patients who have coexistent osteoarthritis or moderate chondrosis [18]. The reported results are better, however, when labral repair or fixation, instead of simple labral débridement, is combined with bone reshaping [22, 23]. The confounder here is that research has not been done to investigate whether soft-tissue procedures (for the labrum and articular cartilage) done without bone débridement might result in similar symptomatic relief. For example, in dysplastic hips evidence indicates that treating labral and chondral lesions without a concomitant bone operation can result in clinical improvement [24].

Assertion 2: Having Femoroacetabular Impingement Morphology Automatically Means That a Patient Has Femoroacetabular Impingement Syndrome—Fiction

As is the case for all medical conditions, although radiologic findings may be necessary for a diagnosis of FAI syndrome, they are not sufficient for diagnosis without clinical signs or symptoms [25, 26]. Both radiologists and surgeons commonly overlook this fact with newly described entities, becoming enamored with the new measurements and criteria published nearly monthly. Just like every patient with positive ulnar variance of the wrist does not have ulnocarpal impaction and not every light-skinned individual has (or will ever develop) melanoma, not everyone with FAI morphology has (or will develop) the FAI syndrome. Additionally, even in patients with hip pain (with or without arthritis), the shape of the bones cannot be assumed to be the cause of FAI syndrome (Fig. 1). Nev-

ertheless, especially in the orthopedics community, once FAI became an explanation for hip pain in young patients and the tools for treating it became commonplace, the definition and scope of the diagnosis expanded. As Bill Palmer [26] aptly stated recently, “Optimism and opportunity have converted many [orthopedic surgeons] into FAI believers.” Or, put another way, once you own a hammer, everything begins to look like a nail.

Consider a (slightly absurd) analogy: Imagine a 7-ft-tall man. His measured height would certainly put him well out of the “normal” range; I suspect that less than 0.1% of the population reaches that height and that 7 ft is many SDs away from the mean. So, would we diagnose his height as “abnormal”? Some might. I’m sure that very tall people frequently bump their heads on low overhangs. Would he warrant the label “head–airplane ceiling impaction syndrome”? More importantly, would we suggest “fixing” his abnormal morphology (I guess by doing reduction osteotomies of his lower extremities) to manage this “syndrome” (and maybe prevent a long-term complication, such as premature dementia from repeated concussions)? If he makes his living playing basketball, would he see his “abnormal” height as a disease to treat or as a fortunate variation in human anatomy?

Corollary 2A: Femoroacetabular Impingement Morphology Represents an “Abnormality”—Fiction

All quantifiable measures in the body lie on a continuum. Within the population, there is a range for the roundness of the proximal femur (determined by any offset or angle measurement) and for the amount of anterior acetabular coverage (again, specified using any of the described measurements). Setting an arbitrary cutoff to separate “normal” from “abnormal” will always include certain outliers while excluding true patients. The recent trend in the literature has been to describe newer techniques (e.g., radially oriented cross-sectional studies or new radiographic projections) to depict the anatomy and then to apply previously established numeric cutoffs to these new methods. Researchers will then conclude that a newer imaging procedure is more sensitive for depicting FAI morphology if it captures a larger number of subjects than previously described methods [27–30]. In reality, we need to know that a given projection—say, the “Dunn” view—best separates normal from abnormal, not simply that it is the view that shows more deficiencies in head-neck offset than others

[28]. Without a reference standard for defining the FAI syndrome that is independent of anatomic measurements, assigning clinical importance to measured values becomes a self-fulfilling prophecy. For example, a study of 157 young, military personnel presenting with hip pain found that the majority (87%) had at least one imaging feature of FAI morphology using relatively loose definitions of femoral asphericity (alpha angle > 50°) and abnormal acetabular depth (center edge angle > 38°) [31]. Although FAI devotees would use this result to support the idea that FAI morphology is common and is clinically important, in reality the study patients had a variety of diagnoses including bursitis, osteonecrosis, sacroiliitis, sciatica, and muscle strains, which shows how nonspecific the presence of FAI morphology becomes when it is defined more broadly. Other authors have concentrated on finding optimal cutoffs to apply to each measurement. For instance, Sutter et al. [32] showed that the range of anterosuperior alpha angles in symptomatic patients and asymptomatic control subjects largely overlapped but that choosing 60° instead of 55° as a threshold identified fewer asymptomatic control subjects (fewer false-positives). The problem with this analysis is that it misses the point: The alpha angle is a number, not a symptom. We would never base a diagnosis of appendicitis (and subsequent surgery) on a research study showing that a fever of 39°C rather than 38°C better separated appendicitis patients from others. A patient’s temperature would be meaningless without the historical, physical examination, and laboratory findings.

Even more important is testing these anatomic measures in the general population to determine whether the measures are specific. A prospective study of a healthy, population-based cohort (2081 individuals) found that more than one third of men and 10% of women had radiographic FAI morphology in at least one hip [33]. Similar results have been reported in recent investigations of asymptomatic subjects using CT and radially oriented MR images [34–36]. Studies of specific target groups (such as young former soccer players) have found that more than half the asymptomatic men had an alpha angle greater than 55° [37]. If the majority of a population meets a certain criterion, can that measure ever be considered “abnormal”? Even when the upper limit of the alpha angle is defined as 62° on a cross-table lateral view (which was found to be the 95% CI in the tested population and is higher than the cut-

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off advocated in the literature), a prospective study of 166 strictly screened, asymptomatic hips still found that 10% of the healthy subjects' hips exceed this measurement [38]. Investigations of some acetabulum-sided FAI morphologies produced similar conclusions when a variety of symptomatic and asymptomatic subjects are included. For example, a recent study reported that coxa profunda was present in more than half of all hips and was actually seen more frequently in asymptomatic hips than in those with clinical FAI syndrome, especially in women [39]. Based on these data (and everyday experience), it should be obvious then that not everyone with FAI morphology has symptoms.

The logical extension of this idea is that there must be patients who have hip pain due to a cause other than FAI syndrome who nonetheless have FAI morphology. An easily understood analogy would be patients with chronic back pain and radiographic features of degenerative disk disease: Lumbar discogenic disease is so common in the general population (with or without symptoms) that no one would automatically conclude that disk degeneration is the cause of back pain whenever it is found radiographically (Fig. 2).

Assertion 3: Acetabular Labral Tears Characteristically Occur in the Femoroacetabular Impingement Syndrome—Fact, But...

Labral tears are common in patients with FAI syndrome, especially in those with the pincer variety [7]. Even when the symptoms of FAI syndrome are absent, just the presence of FAI morphology is a risk factor for labral lesions [40]. However, acetabular labral tears will occur in any hip with osteoarthritis, just like meniscal tears are frequent in osteoarthritic knees. And, as I will discuss, FAI morphology is common in hips with osteoarthritis. Thus, coexistent labral tears and osteoarthritis will occur by association. For example, Wenger et al. [41] reported that 89% of 40 patients who had labral tears were also found to have abnormal bone morphology, but only 21 of these patients had FAI morphology: The remaining "abnormalities" were osteophytes or coxa valga. Similarly, a study of 125 patients undergoing total hip arthroplasty for idiopathic osteoarthritis showed that 100% had a pistol-grip deformity of the femur [42]. The same study, however, found that the pistol-grip deformity (using their definition) was clearly age-related: It was present in only 14–15% of patients in their third decade of life but was present in 68% of pa-



Fig. 2—Back pain in 72-year-old man. Lateral projection of lumbar spine shows severe degenerative disk disease and retrolisthesis at L1–L2. Because discogenic disease is so common (and is frequently asymptomatic) and back pain has myriad potential causes, it would be incorrect to assume that diseased disk is this patient's pain generator. His symptoms resolved next day after passing urinary calculus.

tients 50–70 years old and in 100% of patients more than 80 years old. Therefore, the anatomic finding in this study does not represent an underlying abnormal femoral shape that leads to later hip disease; rather, the deformity is acquired in middle age. It is likely that these researchers are simply identifying osteophytes, which mimic cam deformities when viewed tangentially in hips with osteoarthritis from any cause (Fig. 3). Most importantly, both these studies suffer from the same biases: They were retrospective, were without control groups, and were composed of patient groups that are not representative of the general population. As an analogy, imagine doing a retrospective study of patients who presented with distal fibular stress fractures without a control group. You would probably find that 100% of those patients were young female runners (virtually the only group that tends to develop stress fractures in this location). Would it be right to conclude that all female runners will develop these injuries (which are relatively rare)? Or, worse, would it be right to use those

results to "intervene" and recommend that all young women stop jogging (which would probably hurt the health of many more people than it would help)?

Corollary 3A: All Anterosuperior Acetabular Labral Tears Are Caused by Femoroacetabular Impingement—Fiction

This falsehood originates partly from studies that make the mistake of incorrectly generalizing findings from a specific patient population (often patients referred to a given surgeon who specializes in one entity) to the whole population. Acetabular labral tears occur for multiple reasons, including aging, developmental dysplasia, trauma, iliopsoas impingement, and osteoarthritis that is due to disorders other than FAI. Like the case for the shoulder labrum [43], degeneration, fragmentation, and absence of the acetabular labrum increases with age even in asymptomatic subjects [44, 45]. A study of 54 cadavers with a mean age of 78 years at death found that 93% of hips had abnormal labra and that most of these abnormalities were located anteriorly or anterosuperiorly [46]. Now that imaging studies are robust enough to evaluate the labrum noninvasively, there is evidence that even young, active subjects who are completely asymptomatic also frequently harbor torn labra in their hips with or without FAI morphology [47, 48].

A second flawed argument used to support this theory is the observation that most of the symptomatic patients with acetabular labral tears also have chondral abnormalities, often in the same location [6, 46]. However, like labral tears, chondral defects also increase with age [46], so it is not possible to tell whether one causes the other, whether they are simply associated because they occur in the same patient population, or whether they coexist because each is the result of the same underlying cause. The same would apply for meniscal tears and knee chondral defects: They are frequently found in the same knee compartment but that does not necessarily prove that one causes the other.

Last, the assertion that traumatic labral tears are rare [7] is also just a consequence of a given surgeon's patient population. In practices that primarily see athletes as opposed to arthritic patients, labral tears are frequently caused by injuries and they frequently occur in hips with normal bone morphology [13, 49] (Fig. 4). Interestingly, most of the traumatic labral tears also have adjacent chondral injuries [13], just like the case for labral tears associated with osteoarthritis or FAI.

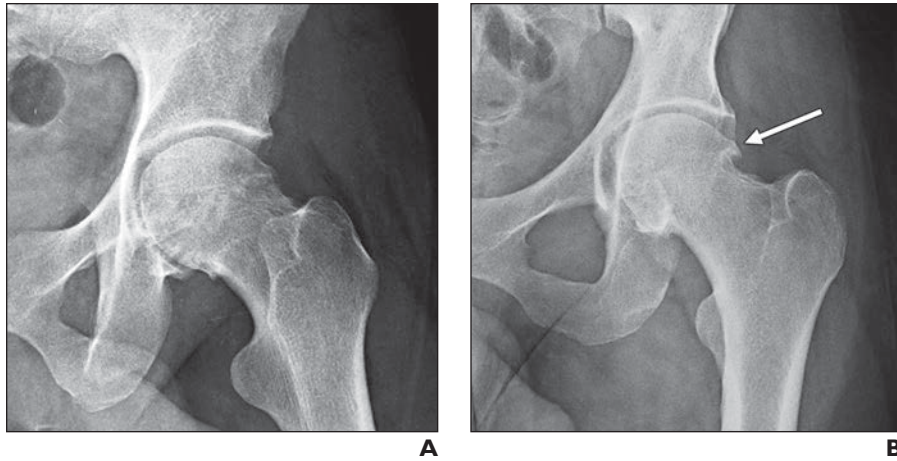


Fig. 3—Femoral head osteophytes mimicking cam deformity in 35-year-old man. **A**, Frogleg lateral radiographic projection appears to show asphericity of femoral head (alpha angle = 74°). **B**, Anteroposterior radiographic projection shows collar of overhanging osteophytes in profile (*arrow*) that produced apparently decreased head-neck offset in **A**. Osteoarthritis in this patient was caused by hemophilia and repeated hemarthroses, not femoroacetabular impingement.

Assertion 4: Femoroacetabular Impingement Morphology (or Femoroacetabular Impingement Syndrome) Is a Risk Factor for Premature Hip Osteoarthritis—Unknown

This concept, of course, is the most important issue. The currently accepted theory of secondary osteoarthritis is that conditions that produce gross deformities resulting in joint incongruity (developmental dysplasia, slipped capital femoral epiphysis, Legg-Perthes disease, healed intraarticular malunions, osteonecrotic femoral heads that have collapsed) result in abnormal contact stresses between the femoral head and acetabular socket and that these altered stresses lead to osteoarthritis. With more subtle abnormalities such as FAI morphology, however, the articular surfaces remain largely congruent, so this explanation does not apply. Rather, the mechanical impaction itself is proposed as the cause of both chondral and labral lesions [7]. Now if one defines osteoarthritis to include lesions that are limited to focal chondral defects, then FAI does indeed cause osteoarthritis (early in the cam type and later in the pincer type). But it is still unknown if these focal chondral defects necessarily progress to generalized joint failure; certainly in other joints, such as the knee, this is not always the case.

If FAI does predispose the hip to premature osteoarthritis, it would open the door to interventions not only to relieve symptoms but also perhaps to slow the progression of osteoarthritis or to prevent it completely. And if FAI is truly a risk factor for osteoarthritis, it would be useful to know which patients will be afflicted and when. Unfortunately, the jury is

still out on this issue, mainly because FAI was described relatively recently and osteoarthritis may take years or decades to develop so there has not been enough time to investigate the natural history of the syndrome. Studies that look at patients who have already developed osteoarthritis suffer from the same retrospective bias (and, often, the same lack of control groups) that hamper the studies investigating the relationship between FAI and labral tears. For example, an unblinded retrospective study using only historical control subjects found FAI morphology in 89% of hips that developed osteoarthritis before the age of 50 years without a definable reason [50]. In the same study, 73% of the contralateral hips progressed to osteoarthritis, with an increased alpha angle and acetabular inclination being the strongest risk factors. A second study in patients with one hip replaced for primary osteoarthritis found that the contralateral hips that already had mild osteoarthritis showed a higher prevalence of cam and pincer morphology than those without radiographic osteoarthritis [51]. Recall, however, that once osteoarthritis due to any cause has developed, osteophytes emanating from the femoral head may give the false impression of an underlying deformity; see Figure 3, for example.

To date, retrospective studies of patients with hip osteoarthritis show that many of these hips have abnormal bone shapes (including FAI morphology); studies based on stronger research designs have produced mixed results. One large cohort of women found that those with higher alpha angles at year 2 of the study had significantly more hips replaced by year 20 [52]. A cross-sectional population

study of 3620 subjects with a mean age of 60 years found that hips with FAI morphology on an anteroposterior radiograph were associated with a 2.2- to 2.4-fold increase in hip osteoarthritis (15–19% of those with a deep socket and 5–20% of those with a pistol-grip deformity had osteoarthritis, which was defined as a joint space < 2 mm) [53]. However, in that study, no association was found between these morphologic abnormalities and groin pain; thus, although these patients had FAI morphology, whether they had the FAI syndrome is unclear. And, conversely, 29% of the men and 63% of the women with hip osteoarthritis had no imaging findings of FAI, indicating that even if FAI is a contributing factor, FAI alone cannot fully account for the development of hip osteoarthritis. Not surprisingly, the biggest risk factor for hip osteoarthritis was simply older age [53]. Conversely, a study of 121 patients with FAI morphology found that there were no features that could predict the age of onset of osteoarthritis and that not every hip with FAI morphology developed osteoarthritis even in patients who were highly physically active [54]. Interestingly, that study also found no association between the severity of femoral head asphericity and the development of osteoarthritis: The mean alpha angle was 89° in hips that suffered progressive osteoarthritis and also in those that did not [54].

Corollary 4A: Femoroacetabular Impingement Morphology Always Leads to Hip Osteoarthritis—Fiction

Longitudinal and prospective studies are now beginning to appear in the literature to address this issue. In a group of 43 hips with cam morphology who had already developed mild osteoarthritis, one third of the hips showed no progression of osteoarthritis after 10 years [55]. More impressive is a recently published study [56] of 96 hips with FAI morphology on anteroposterior pelvis radiographs but no symptoms; in that study, sequential radiographs over many years were available because the patients were being followed for contralateral hip disease, most cases of which were neither osteoarthritis nor FAI. Here, the majority of hips (82%) did not develop osteoarthritis or symptoms after 10–40 years (mean follow-up, 18.5 years), and the alpha angles in the 17% of hips that did develop osteoarthritis were statistically no different (the mean was actually slightly smaller) than the alpha angles in the hips that did not develop osteoarthritis (57° vs 59° , respectively) [56]. In both studies, acetabular retroversion seemed to confer a higher risk of subsequent osteoarthritis com-

pared with femoral head asphericity [55, 56]. Nevertheless, it is clear that some hips with even severe FAI morphology will not develop osteoarthritis (Fig. 5), making rigorous evaluation of treatments designed to stave off osteoarthritis problematic.

Corollary 4B: Treatment of Femoroacetabular Impingement Delays or Prevents Premature Hip Osteoarthritis—Fantasy, for Now

At least based on the data currently available, it is not possible to find any support for this statement. First, we now know that FAI morphology does not always lead to hip osteoarthritis, which makes empiric sense going back to one of my original queries: If FAI is so prevalent but was unrecognized and untreated until recently, why are there not an overwhelming number of patients walking around with hip osteoarthritis and arthroplasties? Because of this fact, we must carefully interpret studies that report short-term outcomes after surgical interventions for FAI. When these studies show that most of the patients who undergo these operations do not develop osteoarthritis and the authors conclude that somehow their treatment was responsible, they may actually just be reporting the natural history of the condition. A recent meta-analysis of eight studies looking at outcomes after surgery for FAI found that osteoarthritis progressed in “only” 0–33%, with a follow-up time of 14–58 months [18]. In fact, there were only two studies that followed patients for more than 4 years, and both of those did find patients whose osteoarthritis progressed. Recall the longitudinal studies showing that even after 10–40 years, osteoarthritis does not necessarily progress even in the absence of treatment. But the bias in the orthopedics literature is to interpret outcomes after surgical interventions as resulting from the surgery itself while ignoring the natural history of the condition. For example, a retrospective, uncontrolled, nonblinded study following 19 FAI patients who underwent open surgery found that five patients progressed to requiring a hip arthroplasty after 4–5.2 years. However, the authors concluded that the surgery was “successful” because the other 14 patients had no progression in their joint space narrowing during this short follow-up period. It is telling that the authors calculated that the “expected” progression would have had to be 0.4–0.5 mm in 4–5 years and claimed success because they were not able to measure that amount of change [8].

Perhaps future studies will be able to identify other risk factors—phenotypes driven by

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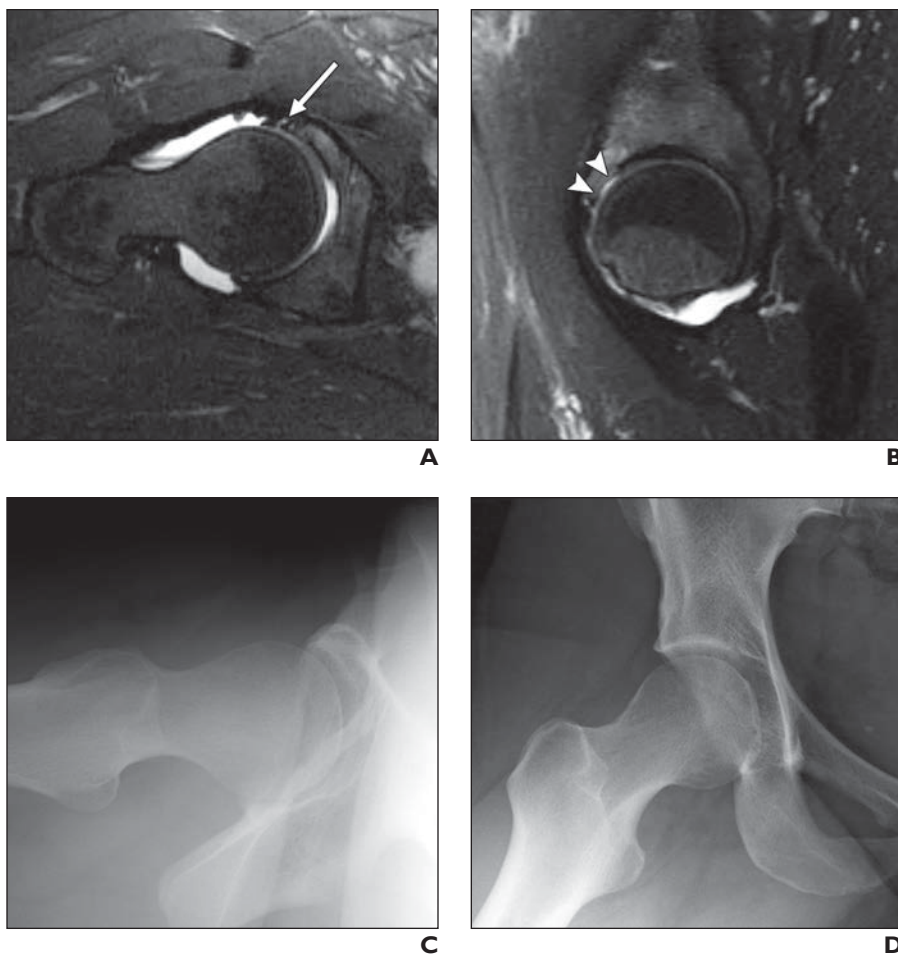


Fig. 4—Traumatic labral tear and chondral defect in 35-year-old woman. **A and B**, Oblique transverse (**A**) and sagittal T2-weighted (**B**) images obtained as part of hip MR arthrography. Note tear of anterosuperior labrum (*arrow*, **A**) and full-thickness acetabular chondral defect (*arrowheads*, **B**); both findings were confirmed at subsequent arthroscopy. **C and D**, Cross-table lateral (**C**) and “Dunn” (**D**) radiographic projections show normal bone morphology (alpha angle < 50° on all projections and MR arthrography). Patient had no symptoms until recent twisting injury suffered while playing softball.

genetic differences, for example—that will allow us to better stratify patients (regardless of the shapes of their bones) as to their future risk of hip osteoarthritis, at which time it may be possible to design a study to see whether an intervention can change long-term patient outcomes. Hopefully prospective, controlled trials that include prolonged follow-up and multiple management limbs will be conducted that can address these issues. But, for now, it is premature to conclude that FAI—especially with the broader definitions of the syndrome now being applied—is the dominant mechanism responsible for most hip symptoms. We also cannot predict with certainty which patients with FAI morphology will develop symptoms or osteoarthritis or when that might occur. Maybe after more data are collected and analyzed, it will indeed be the

case that FAI is primarily responsible for hip osteoarthritis. However, until then, it seems wise to proceed cautiously both when counseling and treating patients whose hip bones seem a little bumpy or twisted.

Practical Advice for Practicing Radiologists

What should radiologists do, based on the evidence available, when interpreting hip examinations? I am constantly asked this question. Are we helping patients if we identify a morphologic finding that may be associated with a clinical syndrome to suggest FAI as a diagnosis or to direct further workup (perhaps with a statement such as, “Please correlate for symptoms of impingement” or “This finding may be associated with FAI” in our reports)? Or is that approach contributing to overdiagno-

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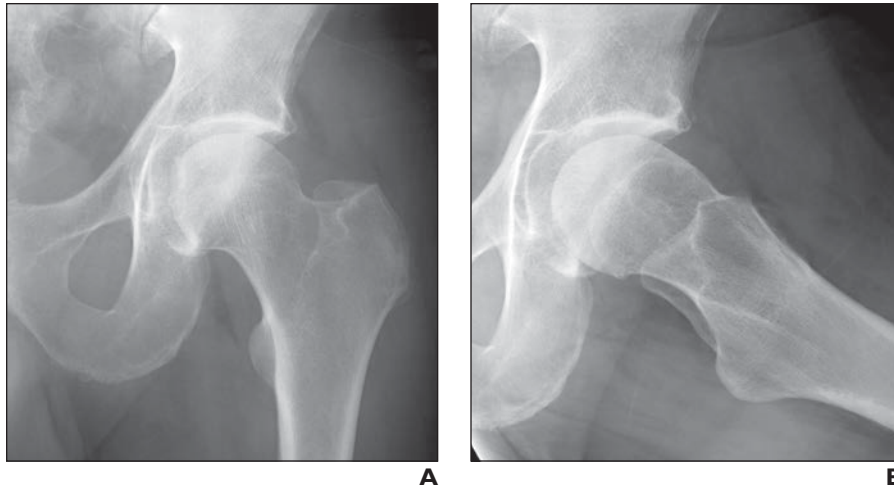





Fig. 5—Severe cam deformity without osteoarthritis in 75-year-old man.

A and B, Anteroposterior (**A**) and frogleg lateral (**B**) radiographic projections of hip. Frogleg view shows virtually complete loss of anterior head-neck offset (alpha angle = 88°). However, there is no joint narrowing, and there are few, if any, osteophytes. Clearly femoroacetabular impingement morphology did not result in “premature” osteoarthritis in this older man.

TABLE 1: Practical Suggestions for Radiologists Interpreting Hip Imaging Studies

Patient		Representative Radiograph	Findings in Body of Report	Report Impression	Notes
Age (y)	Sex				
31	M		Deficient head-neck offset (bump), normal joint width	Aspherical proximal femur without hip osteoarthritis	Patient has cam morphology, but there is no way to know whether he has FAI syndrome
71	F		Deep acetabular socket with femoral head overcoverage, normal joint width	Coxa profunda or normal hip	No reason to institute a workup for a condition such as FAI that is highly unlikely to affect this patient’s future
38	M		Nonuniform joint narrowing, osteophytes, subchondral cysts	Moderate-to-severe hip osteoarthritis	Once osteoarthritis is this advanced, the cause doesn’t matter; this hip was replaced 1 year later because of rapidly progressive hip disease, not FAI

Note—The radiographic report can be complete and accurate without specifically using the terms “cam,” “pincer,” or “femoroacetabular impingement.” FAI = femoroacetabular impingement.

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TABLE 2: My Assessment of Several Common Assertions Pertaining to Femoroacetabular Impingement (FAI)

No.	Description	Assessment	Notes
Assertion 1	FAI syndrome exists as a distinct syndrome	Fact	Defined by signs, symptoms, and imaging (FAI morphology)
Corollary 1A	Impingement between the femur and acetabulum is abnormal	Fiction	Imaging studies in asymptomatic subjects show physiologic impingement between the femoral head and acetabular rim
Corollary 1B	Treating FAI relieves the symptoms	Fact	Clinical improvement occurs with bone reshaping and labral treatment
Assertion 2	FAI morphology = FAI syndrome	Fiction	Imaging findings are necessary but are not sufficient for the diagnosis
Corollary 2A	FAI morphology is abnormal	Fiction	Studies of asymptomatic hips have found large numbers of subjects with the same morphologic features
Assertion 3	Acetabular labral tears occur in FAI syndrome	Fact	Most patients with FAI syndrome have labral tears without controlling for confounders such as coexistent osteoarthritis
Corollary 3A	All anterosuperior acetabular labral tears are caused by FAI	Fiction	Labral tears occur because of trauma and other hip conditions and even occur in asymptomatic patients with or without FAI morphology
Assertion 4	FAI morphology is a risk factor for osteoarthritis	Unknown	Retrospective studies show an association between FAI morphology and hip osteoarthritis; prospective studies show conflicting results
Corollary 4A	FAI always leads to osteoarthritis	Fiction	Many (most) hips with FAI morphology do not develop hip osteoarthritis when followed over many years
Corollary 4B	Treatment of FAI delays or prevents premature hip osteoarthritis	Fantasy	Because of corollary 4A, it is impossible to attribute a lack of osteoarthritis after treatment to the intervention as opposed to the natural history of the condition

sis or unnecessary physician and patient anxiety? Should the imaging findings be interpreted differently if a patient is being referred from an orthopedic surgeon for hip pain as opposed from an internist for a recent fall? My approach is very straightforward and applies to all imaging modalities, anatomic regions, and referring scenarios: When there are findings present that constitute a diagnosis (say nonuniform joint space narrowing and osteophyte formation for osteoarthritis), I describe the findings and give the diagnosis. When I can offer a reasonable differential diagnosis for a finding (maybe metastasis or myeloma for a lytic bone lesion in an older adult), I do. But when I can make a valid observation for which associations with symptoms, syndromes, and outcomes are loose, I simply list the observations in the radiology report, but I do not use them to influence a diagnosis or differential diagnosis (which may be normal or abnormal, depending on the other findings on the study). So for wrist radiographs that show positive ulnar variance and no other salient findings, the body of my report describes the ulnar variance, but my impression remains “normal” (that’s what “variance” indicates, after all). If a shoulder study shows os acromiale, which may or may not be relevant to that patient’s symptoms and management, I include the finding in the report’s impression, but I don’t specifically explain that a mobile os may be asymptomatic, or may be a cause of impingement, or may make rotator cuff repair more likely to fail. Table 1 shows a few examples of hip radiographs that might be encoun-

tered in a typical radiologist’s day, together with the way I would structure my report. The same principles would apply to reports for other imaging modalities. As shown, a radiology report can be complete, useful, and accurate without having to invoke statements such as “correlate clinically for symptoms of FAI.”

Conclusion

Table 2 summarizes the main assertions about FAI that I have addressed. Each reader will need to weigh the evidence in the literature carefully and decide whether Caesar (FAI) should be praised or buried. “Hail FAI! Long live FAI!” or “Et tu, Brute?”

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