

## Femoroacetabular Impingement: A Natural History

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### Abstract

This review article is devoted to the natural course of hip femoroacetabular impingement (FAI). The evolution of the understanding and views of surgeons on primary (idiopathic) hip osteoarthritis (HO) are considered. It is noted that nowadays, the primary hip osteoarthritis in fact almost always arises from anatomical variations. Among those are cam- and pincer-type deformities, leading to impingement. In most of the major works, a reliable causal relationship of cam-type FAI with hip osteoarthritis has been proven. Moreover, cam-type FAI also carries the risk of impending arthroplasty. The association of pincer-type FAI with hip osteoarthritis has been controversial. The course of FAI hip osteoarthritis, most likely, is not as aggressive as, for example, dysplastic hip osteoarthritis, and less often requires arthroplasties. Since cam-type FAI is a reliable risk factor for hip osteoarthritis and arthroplasty, it could be assumed that timely surgical correction of FAI would stop or slow down hip osteoarthritis and avoid arthroplasty. However, at present, this assumption has not been proven, therefore, there is no reason to assert that surgery for FAI in any way affects its natural history in the form of severe hip osteoarthritis following by arthroplasty.

**Keywords:** femoroacetabular impingement, cam-FAI, pincer-FAI, hip osteoarthritis, hip arthroplasty.

### Evolution of the views

Debate and research on the etiology of hip osteoarthritis, despite the long history of study, continue. Usually the researchers identify:


- primary (idiopathic) hip osteoarthritis, without cause;
- secondary hip osteoarthritis that develops for any known cause. It would be more accurate to say, that this type of hip osteoarthritis developed in connection with the proven cause.

For the first time, hip joint impingement and its connection with hip osteoarthritis (called *malum coxae senilis* at that time) were described by M.N. Smith-Peterson in 1936. It was assumed at that time that hyaline cartilage and subcortical bone did not have nociceptors. So the impingement pain of the femoral neck and acetabular anterior edge were attributed to the interposition of the synovium. M.N. Smith-Peterson successfully performed periacetabular osteotomy for painful impingement in the patients not only with hip osteoarthritis (*malum coxae senilis*), but also with the consequences of epiphyseolysis. Besides, he

performed the surgery in the case of “acetabular intrapelvic protrusion”. He did not call it “impingement” [1], although, from a modern point of view, this could be designated as pincer-type impingement.

As early as in 1933, R. Elmslie noted that although some reasons for the development of hip osteoarthritis were well known, “but in the majority of cases it is impossible to ascribe the condition to any particular accident. ... Many patients who develop osteoarthritis at a comparatively early age – for example, at from 40 to 50 – will be found to have a pre-existing deformity of the joint” [2]. R. Elmslie paid special attention to *coxa plana*, which, in his opinion, at a young age triggered a chain of biomechanical disorders and leads to early hip osteoarthritis. It is logical to assume that any biogeometric deviations will contribute to the development of hip osteoarthritis, and not only *coxa plana* dysplasia.

The authors of 4 works, published from 1947 to 1961, failed to find the radiologically manifested causes for the development of hip osteo-

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arthritis in 24.3 to 65.0% of the cases [2, 3, 4, 5]. However, in 1965 R. Murray once again questioned the concept of primary hip osteoarthritis, suggesting that primary hip osteoarthritis was actually secondary against the background of minimal anatomical variations, "so small that even their radiographic presence can be interpreted as normal." R. Murray also introduced the term "tilt deformity of the head", describing it as a varus tilt of the head in relation to the shortened neck and remodeling of the lateral sectors of the head. After the detailed re-examination of 200 X-rays, he argued that 65% of primary hip osteoarthritis cases were in fact the result of existing asymptomatic anatomical abnormalities (25.5% – acetabular dysplasia and 39.5% – tilt deformity). R. Murray believed that early closure of growth zones, minor trauma, transient synovitis, and minor epiphyseolysis were possible causes of tilt deformity of the head (Table 1) [6].

R. Murray's assumptions were supported in 3 papers of the 1970s. In 1974, S. Stulberg and W. Harris found small forms of acetabular dysplasia in more than 40% of the patients with "idiopathic" hip osteoarthritis [7]. In 1975 S. Stulberg et al. described "pistol grip deformity" (in fact, an analogue of the term "tilt deformity of the head" introduced by R. Murray [6]) [8]. The re-examining of 75% of X-rays with primary hip osteoarthritis, dysplasia was found in 39% of cases, and a pistol grip deformity – in 40%. And finally, in 1976, L. Solomon argued that hip osteoarthritis was always secondary to some cause (he did not find it in only 27 out of 327 patients, while he still believed that even in these cases there were some deviations, but they just not yet known) [9].

The theory of mechanical conflict (due to deformation of the acetabulum and/or the femoral head), as the main pathological mechanism of most pain in the hip joint with or without hip osteoarthritis dominated until the mid-1970s. However, the relationship between hip osteoarthritis itself and biogeometric disorders described in the fundamental works of S. Stulberg [7], L. Solomon [9] and R. Murray [6] was not sufficiently studied. After all, the authors only described the deformities that were present in hip osteoarthritis, but this did not mean that these deformities preceded hip osteoarthritis, and even more so became its cause. In this regard, in 1976 D. Resnick suggested that the "tilt deformity of

the head" described earlier by R. Murray [6] may be a consequence of hip osteoarthritis, and not its cause [10].

Many works devoted to the rupture of the acetabular labrum, appeared in the 1980-1990s. But the role of the acetabular labrum in normal or arthritic joint was not studied. In most cases, the cause of labrum rupture was considered trauma [11], while ruptures without obvious trauma were regarded as prerequisites for hip osteoarthritis [12, 13, 14]. With the development of arthroscopy, the diagnosis and attention to labrum rupture strengthened [15]. Works recorded the relationship between labrum rupture and cartilage damage began to appear. N. Santori, R.N. Villar [16] and L. Farjo et al. [17] reported that up to 95% of labrum rupture cases were accompanied by cartilage damage. However, as before, the causal relationship between these two phenomena remained unclear.

In 2001, J. McCarthy et al. [18], as a result of cadaver studies, suggested that rupture of the labrum altered the joint biomechanics, led to cartilage degeneration and hip osteoarthritis. However, this statement was also speculative.

In the late 1990s and early 2000s, the era of widespread use of high-quality MRI that allowed hip visualization, including a detailed assessment of the hyaline cartilage, labra, and pathological changes in the area of the head-to-neck transition, which was especially important in femoroacetabular impingement (FAI) [19]. However, advances in MRI did not lead to understanding the etiopathogenesis [20]. Only with the advent of the technique of the safe femoral head dislocation at the same period of time, it became possible to fully assess the biomechanics and visualize the relevant changes. As a result, the etiopathogenesis was finally formulated, linking minor biogeometric deformations with pain, decreased range of motion and damage to the hyaline cartilage and acetabular labrum [21] and proving a causal relationship between FAI and hip osteoarthritis [22, 23].

Particularly important and noteworthy in this series was the work of R. Ganz et al., which they titled: "Femoroacetabular impingement: a cause for osteoarthritis of the hip". The work was based on authors' experience of studying more than 600 patients undergone surgical dislocation for impingement in a nondysplastic joint [24].

Table 1

**Evolution of terminology and understanding of femoroacetabular impingement and hip arthritis etiopathogenesis**

Year	Author	Idea/term
1935–1936	M.N. Smith-Peterson [1]	Hip joint impingement (M.N. Smith-Peterson first coined this term. His work is undeservedly practically not mentioned in reviews).
1965	R.O. Murray [6]	Tilt deformity of the femofal head. Biogeometry is the cause of arthritis. Primary arthritis in 65% of cases is actually secondary to small changes in biogeometry.
1975	S.D. Stulberg et al. [8]	Pistol grip deformity.
1976	L. Solomon [9]	Arthritis is always secondary and occurs only in the joints with preexisted other problems.
1976	D. Resnick [10]	Collisions of head, neck and acetabulum osteophytes during movements were described. The role of impingement as a cause of arthritis is denied.
1991	K. Klaue et al. [25]	The acetabular rim syndrome. There is always an avulsion of the acetabular labrum, maybe <i>os acetabuli</i> . The authors identified two types of the syndrome: type 1 – incongruent shallow acetabulum, type 2 – the acetabulum is congruent, but the coverage of the femoral head is deficient
1991	R. Ganz et al. [26]	Cervicoacetabular impingement.
1999	S.R. Myers et al. [25]	Postoperative femoroacetabular impingement. For the first time, the femoroacetabular impingement was described as an independent nosology on the example of the patients undergone periacetabular osteotomy.
2001	K. Ito et al. [27]	Cam-type of femoroacetabular impingement.
2003	J. Parvizi & R. Ganz [28]	Femoroacetabular impingement is one of the causes of hip arthritis.
2005	M. Leunig & R. Ganz [29]	Femoroacetabular impingement can be the pincer- and cam-type.

Since the mid-2000s, the number of publications on the FAI problem has significantly increased [30, 31, 32, 33, 34, 35, 36]. C.L. Peters and J.A. Erickson [36] among their 30 patients, undergone surgical dislocation of the hip, in 26 cases found damage to the labrum or nearby hyaline cartilage in the anterior-superior quadrant of the acetabulum. M. Tannast et al. showed [37] that the intraoperative size and degree of cartilage damage were larger than in preoperative planning, which emphasized the underestimation of the problem [20].

The concept of impingement damage to hyaline cartilage is not unique to the hip joint. For example, as far back as 1957, D. O'Donoghue [38] reported about the impingement of exostosis of the talus and the tibia. And although this impingement was located outside the joint zone, it led to the cartilage damage due to changes in biomechanics and the increase in intra-articular pressure. The similar thought in relation to the hip joint was expressed by G.T. Rab [39]. The computer simulation of the hip movements in case of

epiphysiolysis consequences (with the existing pistol grip deformity), he found a significant increase in intra-articular pressure.

Nowadays, it is customary to distinguish 3 etiopathogenetic types of FAI: cam-type, pincer-type, and mixed type (Fig. 1). It is believed that the preclinical cam-type develops in childhood and progresses with bone growth. After the growth zones are closed, adaptive deformity of the head and neck progresses [40]. The pincer-type is characterized by a spherical hypercoated head.

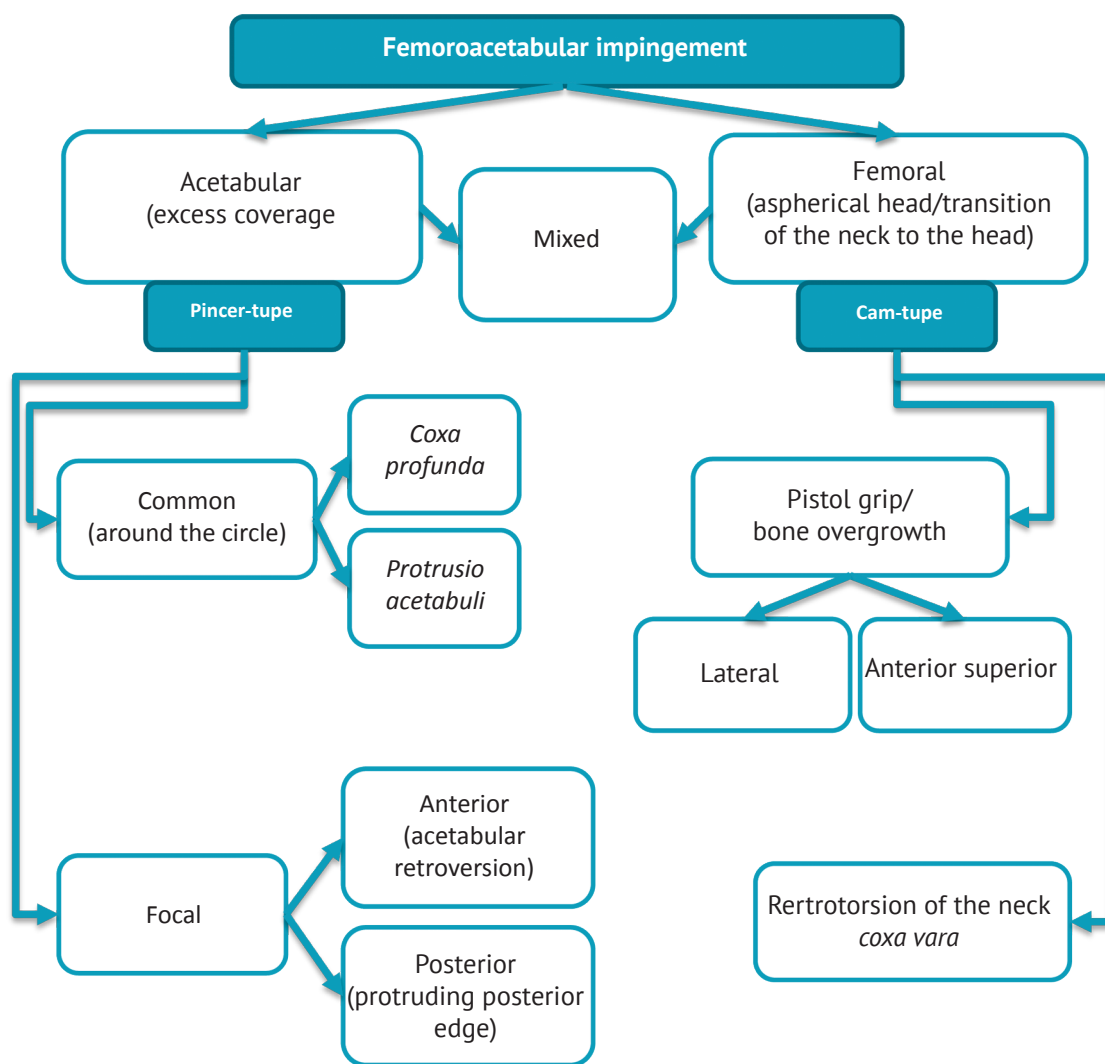
At the present stage, the assumption of 1933 by R. Elmslie [2] that any "idiopathic" hip osteoarthritis still had a cause, and if we did not find it, then only because we did not yet know about it, is illustrated by the work of J.C. Clohisy et al. [42]. At the initial assessment of the patients' X-rays before arthroplasty, in 48% of cases there was acetabular dysplasia, in 10% – the consequences of Perthes disease, in 6% – the consequences of epiphysiolysis, in 36% the reason for arthroplasty was "idiopathic" hip osteoarthritis. However, af-

ter the detailed assessment of these "idiopathic" cases, it turned out that 63% of them had the cam-type FAI, 6% had the pincer-type FAI, and 29% had the mixed type FAI. The authors failed to establish the cause of hip osteoarthritis only in less than 1% of the total number of the patients [42]. It is important to note that this study was performed in the patients under 50. The percentage of truly idiopathic hip osteoarthritis in older patients is likely to be higher.

Detailed coverage of X-ray indices, criteria, features and differences of lesions for various types of FAI was not the topic of our work. So we

will focus on its natural history. In any case, the causes and pathogenesis of the cam- and pincer-type of FAI are different, and their natural course is probably different. Therefore, they must be considered separately.

The exploration of the natural history of the FAI course is possible in two types of observational studies: in cohort studies and in case-control studies [43]. The former ones are very long in time, but they give a greater understanding of the causal relationship between the initial anatomical variations and the final outcome (arthroplasty).



**Figure 1.** FAI classification by Banerjee P. and McLean C.R. [41], modified by the author.

### Natural history of the cam-type FAI

We were able to find 2 cohort studies investigated the relationship between cam-type FAI and the development of hip osteoarthritis.

The Chingford-1000 study, which lasted 20 years in the UK, included 1003 women undergone X-rays at the 2nd year of enrollment and at the 20th [44]. The cam-deformity was assessed by the alpha angle and triangular index. We draw the readers' attention that this is not the same alpha angle that is measured in children to assess dysplasia. The method for assessing these FAI criteria by X-rays is described in the works of K.K. Gosvig [45] and H.P. Notzli [46]. It turned out that both the alpha angle and the triangular index were strongly associated with the development of X-ray hip osteoarthritis signs and with arthroplasty. An increase in the alpha angle for each additional degree over 65° led to an increase in the likelihood of X-ray hip osteoarthritis signs by 5% and an increase in the risk of arthroplasty by 4% after 20 years.

In addition to cam-type FAI, the Chingford-1000 also studied dysplasia [44]. It is interesting to note that cam-type FAI is a less powerful predictor of hip osteoarthritis and arthroplasty than dysplasia. Thus, a decrease in the lateral center edge angle in dysplasia by one degree (at the values less than 28°) led to an increase in the risk of hip osteoarthritis by 13%, and arthroplasty – by 18%.

The results of the Chingford-1000 study [44] also gave the idea that hip osteoarthritis of various origins was accompanied by pain of varying intensity. The dysplastic hip osteoarthritis is more "painful" and more often leads to arthroplasty against the background of the corresponding X-ray picture. The cam-type impingement is less painful.

The CHECK study included 865 patients aged 45 to 65 years with initial clinical hip osteoarthritis and cam-type FAI [47]. On the baseline X-rays, the diagnosis of FAI and hip osteoarthritis was established in 75% of the patients. In the remaining 25%, the diagnosis was doubtful. The X-rays were repeated in 2 and 5 years, each time measuring the alpha angle. In 5 years, the end stages of hip osteoarthritis by Kellgren and Lawrence developed in 2.76% of the patients. The moderate (> 60°) and severe (> 83°) increases of the alpha angle led to a significant

increase in the risk of arthroplasty (relative risk 3.67 and 9.66, respectively). The combination of large alpha-angle and restriction of internal rotation led to a colossal increase in the relative risk (25.2). In 5 years, 52.6% of such "combined" patients had end-stage hip osteoarthritis.

In 2017, C.C. Wyles et al. published the results of an interesting study that was both the cohort and case-control by design [48]. By the screening of 226 asymptomatic athletes aged 12 to 18 years, the authors found that internal rotation was limited at the 90° flexion position in at least one of the two hips in 13 athletes (21 out of 26 joints had limited internal rotation). The control group included 13 athletes, matched by gender and age. X-rays and MRI were performed at the start and in 5 years. It turned out that certain signs of cam-type FAI (bone, cartilaginous, joint labrum lesion) on baseline X-rays and MRI scans were present in 62% of participants in the case group and in 31% – in the control group. The average alpha angle in the case group was 58°; in the control-group – 44°. In 5 years, some MRI abnormalities were noted in 95% of the participants in the case group and in 54% – in the control group. The new MRI abnormalities or the progression of previously existing occurred in 58% of the case group and only in 30% of the control.

Other current case-control studies also show a link between cam-type FAI and hip osteoarthritis. For example, K.K. Gosvig et al., studying the X-rays of 3620 patients (symptomatic and asymptomatic) with an average age of 60 years, found the signs of cam-type FAI (pistol grip deformity) in 24.2% of men and 5.4% of women (the criterion was the triangular index greater than 0 mm) [49]. The hip osteoarthritis signs (narrowing of the joint space ≤ 2 mm) among the patients with pistol grip deformity were found in 15.2% of men and 12.5% of women. The correlation was statistically significant with both X-ray and clinical criteria for hip osteoarthritis.

Another work by C.C. Wyles et al. studied 162 patients younger than 55 years old (mean age 47 years) undergone arthroplasty of one hip in the absence of degenerative changes in the other joint [50]. For an average of 20 years (min 10 years, max 35), the authors studied the dynamics of the contralateral, non-operated joint. There were signs of dysplasia in 48 patients, FAI – in 74 patients, no anatomical abnormalities – in 40. By



the end of the follow-up, the contralateral joint arthroplasty was required in 33.3% of the patients with dysplasia (16 of 48), in 17.6% – with FAI (13 of 74), and in 15% – with a joint that initially was normal (6 of 40).

We analyzed in detail the initial data of C.C. Wyles et al. [50]. And we can assume that the course of FAI hip osteoarthritis is less aggressive and requires arthroplasty almost twice less often than dysplasia. Moreover, the rate of arthroplasty in the patients with FAI ultimately turned out to be the same as in the patients with a normal joint ( $p = 0.7979$  according to our calculations). Thus, it could be assumed that although FAI is a reliable risk factor for hip osteoarthritis, it is not a reliable risk factor for arthroplasty.

The authors of the study themselves made an almost similar conclusion that the natural course of FAI hip osteoarthritis did not differ from the natural course of hip osteoarthritis of the joint without initial anatomical variations, in contrast to dysplasia, in which the progression of degeneration occurred most rapidly and aggressively [50].

Thus, it can be assumed that the surgical correction of cam-type FAI and return of the joint to the normal anatomy will not affect the final outcome in the form of arthroplasty, since the natural course of hip osteoarthritis in cam-type FAI and in an initially normal joint is the same. It should also be borne in mind that the relevance of this hypothesis in relation to frequently occurring cam-type FAI patients with high physical activity is unknown, because the study of C.C. Wyles et al. included the patients with a contralateral joint arthroplasty [50].

If there is a combination of dysplasia and cam-type FAI, then the progression of hip osteoarthritis becomes even more rapid than in isolated dysplasia [50].

In other works, the connection between cam-type FAI and hip osteoarthritis is generally denied. For example, L.A. Anderson et al. found no statistically significant association in 547 patients [51].

### Natural history of the pincer-type FAI

The data on the natural course of the pincer-type FAI are scarce and less unambiguous than those on the cam-type. Some works proved the connection between pincer-type FAI and hip os-

teoarthritis, while other works, on the contrary, denied any connection or even argued about the "protective" effect of the pincer on the joint.

The reason for these disagreements, perhaps, is the fact that we still do not have a clear understanding and criteria of what can be considered the pincer-type FAI. In fact, this collective diagnosis can now include very heterogeneous patients with variations from total head hypercoverage to isolated acetabular retroversion [52].

In the already mentioned 20-year study of Chingford-1000 [44], the authors also investigated the pincer-type FAI. To assess it, they measured the Wyberg angle [53] and the extrusion index of the head [54, 55]. The larger the Wiberg angle and, conversely, the lower the extrusion index, the more pronounced was the pincer-type FAI. It turned out that there was no connection between the high values of the Wiberg angle ( $> 33.7^\circ$ ), low values of the extrusion index and the hip osteoarthritis development. The X-ray evaluation at the 2nd year of the study with an increase in the Wiberg angle by one degree above  $33.7^\circ$  resulted in the hip osteoarthritis odds ratio of 0.98;  $p = 0.746$  by the 20th year of the follow-up, and a decrease in the extrusion index by 1SD – of 1.15;  $p = 0.553$  [44].

In the cohort study CHECK, the connection between the high values of the Wiberg angle ( $> 40^\circ$ ), anterior center-edge angle ( $> 40^\circ$ ) and the risk of developing hip osteoarthritis not only was absent, but even, on the contrary, it was found that the simultaneously high values of the Wiberg angle and anterior center-edge were 3 times reduce the risk of developing the end stage of hip osteoarthritis (RR 0.34;  $p = 0.025$ ) in five years [56].

In other works, the connection between pincer-type FAI and hip osteoarthritis was proved. K.K. Gosvick et al. found that the Wiberg angle  $> 45^\circ$  increased the risk of hip osteoarthritis by almost 2.5 times (RR 2.4) [49]. Retroversion of the acetabulum is another condition that is referred to as pincer-type FAI. W.Y. Kim et al. by the retroversion CT measurements found that it was correlated with hip osteoarthritis [57]. N.J. Giori et al. reported that among the patients admitted for arthroplasty, retroversion occurred 4 times more often compared with the control group of the patients without hip osteoarthritis [58].

In the already mentioned study by C.C. Wyles et al., with a mean follow-up of 20 years, retro-

version, low head extrusion index ( $\leq 0.25$ ), *coxa profunda* or *protrusio acetabuli* were not significantly associated with the progression of hip osteoarthritis (for each of the parameters separately) [50].

In another recent review, 2019 C.C. Wylie et al. [52] stated that the increased Wiberg angle reduced the risk of hip osteoarthritis and arthroplasty, citing the work of 2011 J.C. Clohisy et al. [42]. But we cannot agree with their interpretation, since upon detailed acquaintance with the latter work one could conclude that, according to their data, the risk of arthroplasty was higher in the group of the patients with an average Wiberg angle of  $21.1 \pm 7.3^\circ$  compared to the Wiberg angle of  $28.3 \pm 8.7^\circ$  in the group of the patients who did not need arthroplasty ( $p < 0.001$ ). It is difficult to say that the Wiberg angle of  $28.3 \pm 8.7^\circ$  is pointed to the pincer-type FAI.

### Can we influence the course of hip osteoarthritis in FAI?

The cam-type FAI is more often operated than the pincer-type for obvious reasons. It is easier to return to the normal joint anatomy in cam. There are a lot of publications on the study of the closest results of cam-type FAI correction. We will not dwell on them, since this is beyond the scope of this work.

In many works that studied the causal effect of cam-type FAI on the hip osteoarthritis development, the authors, in their conclusions, expressed the idea that cam-type FAI was a modifiable factor. If such patients were subjected to the surgery, it could slow down the hip osteoarthritis development [24, 47, 49].

However, such an assumption, despite all its logical evidence, needs proof. Despite the fact that in most studies the connection between cam-type FAI and hip osteoarthritis was significant, we did not have any convincing evidence that surgical correction of the cam-type FAI could somehow positively affect the course of hip osteoarthritis and the likelihood of arthroplasty.

There are few studies on the effect of cam-type FAI correction on the course of hip osteoarthritis. P.E. Beaulé et al. conduct an ongoing study of 10 patients undergone the cam-type FAI correction. The preliminary results of 2017 [59] and 2018 [60] showed the normalization of a number of

biochemical and instrumental parameters of the cartilage and subchondral bone after the surgery.

In 2019 D.I. Rhon et al. reported the results of the surgery of 1870 patients with FAI without hip osteoarthritis (mean age at the time of the surgery was 32.2 years) [61]. The 2 year follow-up results in the context of the specificity of the surgery were obtained in 1269 patients. It turned out that the risk of hip osteoarthritis development was not affected by femoroplasty for the cam-type FAI ( $p = 0.153$  for the rate of femoroplasty in the group with and without hip osteoarthritis outcome). Acetabuloplasty and labrum reconstruction had no effect as well ( $p = 0.412$  and  $p = 0.228$ , resp.). And in general, hip osteoarthritis in 2 years after FAI correction developed in 22% of all the patients. The disadvantage of this study, in our opinion, was its retrospective nature, the recruitment of the patients by codes of operations, probable hip osteoarthritis underdiagnosis at the time of the surgery, and the lack of comparison with the group of non-operated patients with FAI. So we still do not know if the surgery makes it possible to influence the rate of hip osteoarthritis development.

In another 2019 study, E. Honda et al. reported the results of arthroscopic treatment of 84 patients with cam-type FAI [62]. On average, in 32 months after the surgery, arthroplasty was required in 0% of the patients in the groups under 50 and over 70 years old. 17% of the patients aged 50 to 69 years underwent arthroplasty and in 33% of them hip osteoarthritis continued to progress. The authors concluded that arthroscopic correction of cam-type FAI in the patients over 70 years of age did not have a risk of hip osteoarthritis progression.

However, the results of E. Honda et al. [62] should also be viewed through the prism of the fact that if a particular patient has a tendency to develop hip osteoarthritis requiring arthroplasty, then he or she is operated before the age of 70. The patients with clinically unexpressed hip osteoarthritis to 70 years are often may be limited to conservative measures. In general, their hip osteoarthritis is non-aggressive and with little progression.

Therefore, this work does not give us an answer to the question of the effect of correction of the cam-type FAI on the course of hip osteoarthritis in general. And thanks to the surgery, we only have an improvement in short-term and medium-term clinical results.

There is much less papers on the correction of the pincer-type FAI than on cam. We were able to find only publications by S.Y. Poh et al. (arthroscopic technique of the surgery was described) [63], J.W. Byrd and K.S. Jones (19 patients) [64], C.M. Larson et al. (18 patients) [65], A.A. Krych et al. (5 patients) [66] and T.J. Jackson et al. (19 patients) [67]. All these works are characterized by a short follow-up period (1 to 4 years) and are not focused on the assessment of the final outcomes (end stages of hip osteoarthritis or arthroplasty).

Thus, the question of whether the surgical correction of the pincer-type FAI affects the course of hip osteoarthritis also remains open. And we hope that future studies will provide us with an answer.

The concept of idiopathic hip osteoarthritis is gradually becoming a thing of the past. In fact, in the overwhelming majority of cases in "idiopathic" hip osteoarthritis, one can always find some anatomical variations that can be considered a deviation from the norm. Indeed, in most studies, the relationship between the biogeometric characteristics of the cam- and pincer-type FAI and the development of hip osteoarthritis was proved.

Among the anatomical variations, the issue of underdiagnosis of small forms of epiphysiolysis in children, which subsequently lead to pistol grip deformity and to the cam-type FAI, has recently become more and more urgent.

In cohort studies, the correlation of X-ray indices and angles characteristic of the cam-type FAI with the risk of arthroplasty development was proven. In this regard, many authors suggest that cam-type FAI correction is not just a surgery for relieving pain and improving function, but indeed an operation that makes it possible to preserve the joint and reduce the risk of arthroplasty. However, the latter assumption is still only theoretical. In our opinion, many surgeons, especially those involved in joint-preserving hip surgery, tend to assess FAI too dramatically as an inevitable predictor of subsequent arthroplasty.

It can be assumed that the cam-type FAI hip osteoarthritis is less aggressive than dysplastic and less often requires arthroplasty, even with X-ray indications for the surgery. This moment is very important in the context of sometimes forced decision-making about arthroplasty remotely.

It is no secret that a patient with the cam-type FAI and hip osteoarthritis complains of pain at a

local outpatient clinic or a regional small hospital. Hip osteoarthritis is detected on the X-rays and the patient is referred for arthroplasty on a quota. Then the risk of making a positive decision without an in-person assessment of the pain intensity, real reducing the quality of life and the effectiveness of conservative measures is possible. At least in our clinical practice, we have repeatedly met such patients with the cam-type FAI hip osteoarthritis with really unexpressed pain, who were referred for arthroplasty based on the X-ray picture.

In this regard, we see the prospect of studying the quality of life of the patients before arthroplasty according to relevant scales depending on the hip osteoarthritis genesis (dysplastic, *coxa profunda*, etc.). It is likely that this will help to make some correlation between the "X-ray indications" and the actual clinical indications.

The surgical correction of FAI (cam- or pincer-type) allows improving the quality of patient's life, especially young and older age groups and without or with initial hip osteoarthritis. However, there is no convincing evidence that the surgical correction of the FAI somehow affects the course of hip osteoarthritis in general in terms of the risk of developing the hip osteoarthritis end stages and the need for arthroplasty.

The existing studies on this issue have a short follow-up period and a small number of patients. But even in them it is still not possible to see unambiguous optimism that the FAI surgery could stop the development of hip osteoarthritis.

More well-designed research is needed to answer these questions.

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