VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

Slipped Capital Femoral Epiphysis is a leading cause of femoroacetabular impingement and early onset hip osteoarthritis. A review

Panagiotis V. Samelis

Orthopaedic Surgeon, "P&A Kyriakou" Children's General Hospital of Athens

ABSTRACT

Slipped Capital Femoral Epiphysis is the leading cause of limp in the adolescent population. Obesity and endocrine disorders are major predisposing factors that very often lead to bilateral hip involvement. Not infrequently, a delayed or a missed diagnosis results in progression of the slip towards stages of higher severity, implying less favorable long term results after treatment. *In situ* stabilization of the slipped physis is the globally preferred treatment, yet it does not prevent the affected hip to develop early onset hip osteoarthritis compared to the general population. Femoroacetabular impingement due to a deformed femoral neck seems to be the intermediate pathologic process that causes labral and acetabular cartilage damage and inevitably leads to early hip arthrosis. In fact the vast majority of the slips, especially the moderate and severe slips, will present signs of femoroacetabular impingement. The severity of the slip correlates with the magnitude of the labral and acetabular cartilage lesions. In moderate and severe slips, residual growth and remodelling after *in situ* stabilization are unable to compensate for the remaining femoral neck deformity. In these slips, an arthroscopic femoral neck osteochondroplasty or a modified Dunn procedure might be more effective in order to reduce the risk of early onset hip osteoarthritis.

KEY WORDS: SCFE; femoroacetabular impingement; early onset osteoarthritis; hip

1. Introduction

Slipped Capital Femoral Epiphysis (SCFE) is the leading cause of a nontraumatic painful hip of the adolescence [1,2]. Pathologically, it is a separation of the proximal femoral growth cartilage at the level of the hypertrophic cell zone, which is the most vulnerable area of the growth cartilage.

Historically, the first report of SCFE is attributed to Ambroise Pare' (1554), but the clearest, detailed and impressive description of the disease in the pre-X-ray era belongs to Ernst Mueller (Tuebingen, 1888): "A specific hip disease seen in 14-18 year old children", "the femoral neck gives the impression of being molded by pressure from above downward", etc [3].

CORRESPONDING AUTHOR, GUARANTOR

Panagiotis V. Samelis
Orthopaedic Surgeon
E-mail: samelis_takis@yahoo.com

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

Mechanical and hormonal factors underlie SCFE pathogenesis. Endocrine and developmental processes - normal or not - of puberty, render the proximal femoral physis susceptible to shear stresses [4,5], especially in case of concomitant obesity of the patient. The often mild clinical presentation of SCFE, combined with the low incidence of the disease, result in delayed diagnosis and treatment, even by healthcare professionals, thus triggering a cascade of events that result in the progressive destruction of the hip.

Mild slips have excellent prognosis, as stated since more than 50 years ago [6,7]. On the contrary, large slips (moderate and severe) impair hip function and lead to early onset hip osteoarthritis [8]. The basic guidelines for the treatment of SCFE haven't changed for decades: *In situ* stabilization of mild slips, corrective or compensatory osteotomy in severe slips. Treatment of moderate slips sways between these two options, Wilson stating that the best choice for a moderate slip is to accept deformation after *in situ* stabilization [6]. Open femoral neck osteoplasty, first described in 1898, may be performed simultaneously with *in situ* stabilization of the epiphysis, or later as a separate procedure, in order to diminish the deformity of the femoral neck [6].

Even today, there is no consensus about the ideal treatment for SCFE. *In situ* stabilization of the upper femoral epiphysis until physeal closure remains the universally accepted primary treatment for all slips. However, there has been some progress in understanding and treating SCFE. Recent studies suggest that even mild slips may impinge on the acetabulum and predispose to early hip osteoarthritis. Femoral neck growth and remodelling may only partially restore the post-slip femoral neck deformity and may eventually not protect from femoroacetabular impingement. Modern implants for slip fixation are available, which spare the remaining growth and remodelling potential of the proximal femoral physis. Innovative surgical techniques, such as hip arthroscopy and surgical hip dislocation provide new surgical options for SCFE treatment.

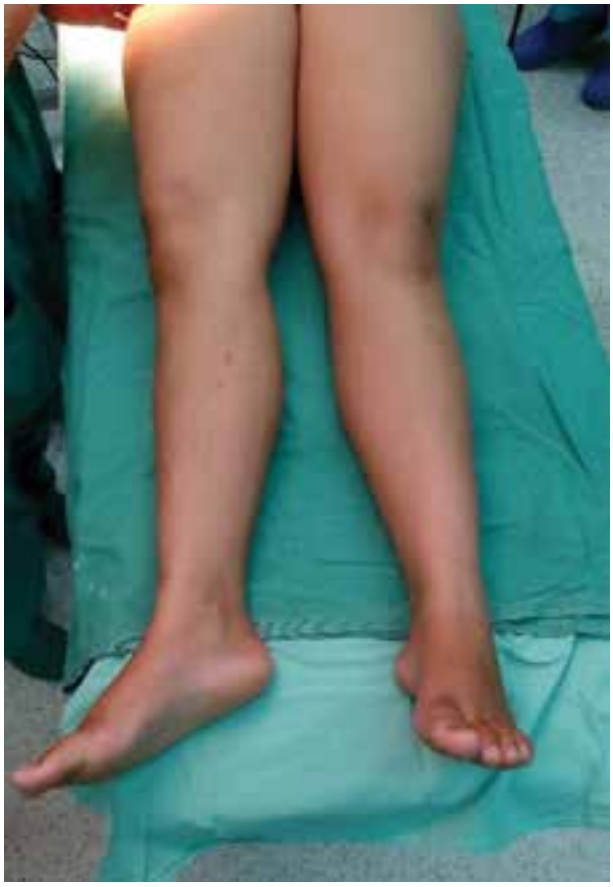

Fig. 1. *Shortening and external rotation of the right lower limb due to a Slipped Capital Femoral Epiphysis of the right hip*

2. Definition - Pathology

Hip deformity of the typical SCFE consists of the primary deformity (slip), followed by the secondary deformities of the femoral neck (anterosuperior bone resorption, posteroinferior bone apposition). The latter are the result of femoral neck growth and remodelling and continue to progress until physeal closure.

The primary deformation starts with external rotation and lateral (varus) tilt of the femoral neck relative to the upper femoral epiphysis, which is stable seated in the acetabulum. At the early stages of the disease, the proximal femoral epiphysis moves posteriorly and medially in the acetabulum. If not diagnosed, the progressively increasing external rotation and varization of the femoral neck leads to central migration of the femur, while the proximal

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

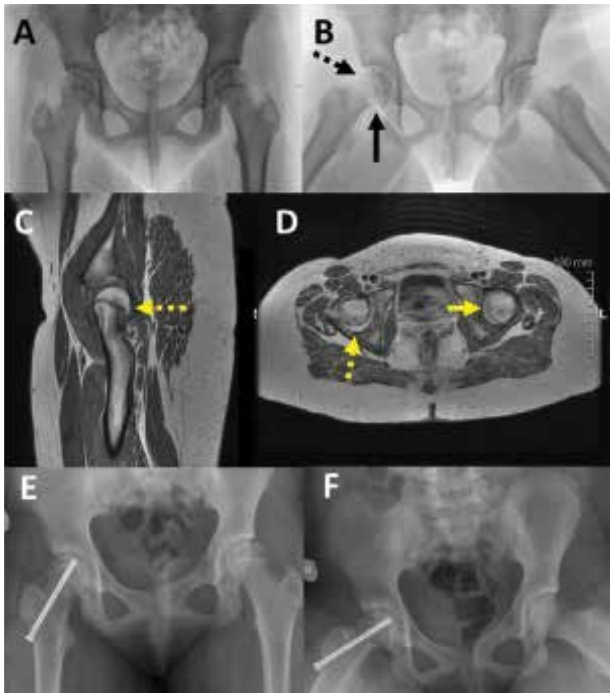

Fig. 2. *Valgus SCFE in a girl complaining of a painful right hip. Her sister presented a typical (varus) SCFE, which was addressed with in situ pinning a few weeks before. A. The femoral neck is tilted medially (valgus) and the femoral head posteriorly and laterally. B. The diagnosis is clear on the frog lateral pelvis projection, showing the small femoral head-neck offset (dashed arrow) with respect to the left hip and the apposition of new bone in the posteroinferior aspect of the neck metaphysis of the R hip (continuous arrow). Hip MRI (C, D) confirms the posterior version of the epiphysis (dashed arrow) of the R hip compared to the epiphysis of the asymptomatic contralateral hip (continuous arrow). The slip is stabilized in situ by means of one cannulated 6,5 mm screw (E,F). The patient is asymptomatic*

femoral epiphysis moves posteriorly and distally in the acetabulum. The final clinical outcome is a short and externally rotated lower limb (**Fig. 1**).

Secondary deformities emerge at about three weeks after slip onset and exacerbate if there is any delay in diagnosis and treatment: callus formation at the posterior-inferior aspect of the femoral neck [9,10] bridges the step between the epiphysis and the metaphysis. The formation of this callus marks

Fig. 3. *The epiphyseal tubercle is an eccentric projection of the physeal surface of the capital femoral epiphysis that fits in a respective impression of the metaphysis and stabilizes the growth plate against shear forces, however it allows pivoting of the epiphysis on the metaphysis*

the transition of the acute to the chronic form of SCFE. The callus is gradually transformed into bone that resembles to an acute shaped osteophyte, described by Klein with the term "crow's beak" [11]. Opposite processes deploy on the anterior-superior femoral neck. Progressive bone resorption results in smoothening of the femoral neck-femoral head junction and in the formation of a prominent neck. The final result of the primary and secondary deformities (slip, remodelling) is shortening and varization of the femoral neck, a prominent anterosuperior aspect of the femoral neck and a retroverted femoral head. On X-rays, these deformities resemble to a pistol grip and are described by the term "pistol grip deformity".

A rare form of SCFE (4.7-9.6%), in which the epiphysis of the femoral head slips posteriorly and laterally (valgus SCFE) has also been described (**Fig. 2**). This slip pattern is more common in girls (76%), affects preferably the right hip and may easily go undiagnosed on the classic anteroposterior pelvis projection if not suspected [2,12,13,14], however the diagnosis is obvious in the frog-lateral pelvis radiograph [15].

3. Epidemiology

The prevalence of SCFE ranges from 0.71 to 10.8 per 100,000 children, but there is seasonal [16] (increased incidence in summer and autumn in north-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

TABLE 1. *Predisposing factors for Slipped Capital Femoral Epiphysis:*

1. Anatomic factors:
- Oblique orientation of the growth plate
- Acetabular retroversion, coxa profunda
- Retroverted femoral neck, varus femoral neck

2. Mechanical causes:
- Chronic: obesity
- Acute: trauma (minimal)

3. Hormonal causes:
- Obesity
- Hypogonadism
- Hypothyreoidism
- Hypoparathyreoidism
- Pseudo-hypoparathyroidism
- Renal osteodystrophy, rickets
- Hypopituitarism

4. Radiation therapy for cancer

5. Chronic disease

ern countries), geographical (only 1-2: 100,000 in Japan and Singapore) and racial (more frequent in Hispanic, African-American, Indian) variation [2]. Boys are more frequent affected compared to girls (boys to girls ratio 1.5: 1). In 20-80%, the disease involves the contralateral hip within one year of the first (primary hip, index hip) hip involvement. Children with bilateral hip disease (simultaneously or not with the primary hip) are usually younger than those with unilateral disease. Unilateral disease preferably (60%) affects the left hip [6,17]. The reason is unknown, but it could be due to the fact that the left lower limb is the center of rotation of the body for the right-handed individuals [6].

Female patients are relatively younger than males. The incidence of SCFE is globally increasing and affects children of younger ages compared to the past, possibly due to the rising incidence of childhood obesity and early puberty [18]. However, the true incidence of SCFE may be even higher, because not infrequently the disease may go undiagnosed, either because of a mild clinical or a subclinical presentation, or due to lack of suspicion by the medical professional, who first examines the limping child.

4. Etiology

The etiology of SCFE is multifactorial [16]. Factors that may predispose to SCFE are listed in **Table 1**.

Mechanical factors increase the shear forces on the growth cartilage and lead to a rupture at the level of the hypertrophic cell zone, which is the weakest area of the growth cartilage (l*ocus minoris resistentiae*)[4]. Such mechanical factors are: The body weight, the relatively vertical slope of the proximal femur [18], a deep acetabulum (coxa profunda: Wiberg's angle $>35^0$, the inner wall of the acetabulum in contact or medial to the ilioischial line in the AP pelvis view) [19] and the relatively retroverted femoral neck (hip anteversion $<10^0$) [20].

The progressive transformation of the growth plate from a relatively wavy and irregular to a flat configuration as the child enters adolescence has also been implicated as a factor that reduces the resistance of the growth cartilage to shear forces. The decrease in height and extent of the epiphyseal tubercle (a peg-like projection of the physeal surface of the capital femoral epiphysis that adapts to a respective impression on the metaphyseal side of the growth plate, **Fig. 3**) may also contribute to the increased susceptibility of the growth plate to shear forces [21,22,23]. The epiphyseal tubercle might function as an axis of rotation, which allows the epiphysis to pivot on the metaphysis without slipping, at least at the early stages of stable slips. This pivoting mechanism refers only to the stable SCFE and probably explains the absence of aseptic necrosis in stable slips [22,23].

Various biochemical factors lead to inherent weakening of the growth cartilage [24]. These factors are not necessarily pathological, since they are related to the adolescence, which *per se* is a hormonal storm from which the child exits as an adult. SCFE is related to the onset of the growth spurt [4,16], that is observed in boys two years later than in girls and lasts longer [24]. This explains the younger age of female patients (12 years) compared of that of males (13.5 years) [24]. The growth spurt of the skeleton is related to the widening of the growth cartilage, commonly observed in SCFE. Endocrine disorders in SCFE patients are often subclinical [16] and in-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

clude hypothyroidism, hypogonadism, and pseudo-hypoparathyroidism. Chronic disease may also predispose to SCFE, through deterioration of normal hormonal pathways [24].

Obesity is very common in SCFE patients. A Body Mass Index (BMI) above the 95th percentile is observed in 63-81.1% of children with SCFE [25,26]. A BMI greater than 35kg/m^2 is associated with a higher risk of bilateral disease [27]. Obesity affects physis stability not only mechanically, but also by means of hormonal interaction. The adipose tissue is a potential endocrine gland that interferes with steroid hormone metabolism and also secretes a variety of cytokines, such as leptin. Leptin levels are proportional to BMI and cause increased proliferation and differentiation of the chondrocytes of the growth cartilage. Leptin also interacts with the Growth Hormone - Insulin-like Growth Factor pathway that controls linear growth [24]. A higher body weight is associated with a younger age at slip onset and is probably the result of early onset of puberty in obese children compared with children with normal weight [16].

Patients with SCFE frequently present a characteristic phenotype consisting of obesity associated with small gonads, the so called adiposogenital phenotype (prepubertal obesity, hypogonadism combined with a high BMI). This phenotype suggests a disturbance of sex hormones that stimulate growth during puberty (**Fig. 4**).

Estrogens accelerate physeal closure. This explains the rare occurrence of SCFE in girls, especially after menarche. Recombinant human growth hormone replacement therapy (rhGH) does not seem to increase the risk of developing SCFE in patients with idiopathic short stature despite of opposite previous reports [28]. Therapeutic irradiation for cancer is also a predisposing factor for SCFE [29,30], frequently associated with the rare variant of valgus SCFE. Patients with SCFE often have a lower biological bone age relative to chronological bone age, implying skeletal immaturity for age. The frequent occurrence of SCFE cases within the same family strongly indicates a hereditary component for SCFE, but on the other hand, it might solely reflect a common exposure of the family to the same environmental conditions that ultimately predispose to SCFE, e.g nutritional factors combined with obesity [31,32].

Fig. 4. *The adiposogenital phenotype (obesity combined with very small genital organs) is frequently observed in SCFE patients*

5. Histologic – Ultrastructural changes in SCFE

Normal femoral neck growth is accomplished by endochondral ossification at the proximal femoral physis. This process seems to be disturbed in SCFE.

Several changes of the affected proximal femoral physis are observed on microscopy [33]: The resting zone of the SCFE physis represents only 20-40% of the physis width (normally 60-70%). On the contrary the proliferating and the hypertrophic zones expand up to 60-80% of the entire thickness of the physis. As a whole, the affected physis is wider (up to five times) than the normal. The normal columnar arrangement of the chondrocytes of the proliferating, hypertrophic, maturation and degenerating zones is disturbed: The chondrocytes of the slipped physis form large clusters that are separated by longitudinal septae.

Histochemical and ultrastructural changes are observed. The chondrocytes in the proliferating and hypertrophic zones present increased nuclear and cytoplasmic density and contain a high amount of cytoplasmic glycogen. Collagen and proteoglycan disorders are found in the extracellular matrix [33]. Microcysts containing calcium phosphate, hydroxyapatite and matrix metalloproteinases

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

TABLE 2. *The Anteroposterior and Frog Lateral Pelvis Views are sufficient to set the diagnosis in most cases of SCFE, to assess severity of SCFE and to monitor treatment outcome*

A) Diagnosis of SCFE

a) Pelvis X-rays: AP, FL

(i) Pre-slip: wide, irregular growth plate

(ii) Established SCFE

- Primary sign: The Klein's line does not intersect capital femoral epiphysis
- Secondary signs:

(i) Metaphyseal blanch sign (Steel-Bloomberg sign)

(ii) Narrow capital femoral epiphysis compared to contralateral

iii) Capener's sign

(iv) Acetabulotrochanteric - Articulotrochanteric distance (ATD) >2 mm)

(v) acetabulotrochanteric angle-ATA> 1^{o}

(vi) Shenton's line disruption

b) Frog lateral pelvis view (Flexion Abduction External Rotation of the hips, the soles of the feet opposed):

- The Klein's line does not intersect capital femoral epiphysis.

B) Slip severity assessment:

(i) Any projection: % displacement of the epiphysis on femoral neck (Wilson)

(ii) Frog Lateral: Slip angle (Southwick)

C) Signs indicative of chronic SCFE on the frog lateral pelvis view: Femoral neck remodelling

- Crow's beak
- Herndon's hump (pistol grip deformity)

D) Signs indicating SCFE complications (femoroacetabular impingement, avascular necrosis, chondrolysis, osteoarthritis)

(MMPs) are seen in the hypertrophic zone. The content of these vesicles is related to the ossification of the matrix [24]. Increased apoptosis is observed throughout the growth cartilage, normally confined only to the hypertrophic zone. Bone formation is impaired [33].

It is unclear whether these changes are the cause or the result of SCFE. In the preslip stage normal cartilage areas alternate with pathologic ones, while in established slips the whole physis is pathologic. Physeal architecture seems to restore to normal after slip stabilization [33].

6. History - Clinical Presentation

The clinical presentation of SCAFE depends on the extent and on the rate of progression of the slip. The latter is a function of how stable the proximal femoral epiphysis is seated on the metaphysis. Various conditions affect the proximal femoral physis and weaken its resistance to shear forces. A preslip stage precedes the actual slip. The child may complain of mild pain at the hip and/or the ipsilateral thigh and knee and presents a limp. These symptoms are very common (67%) [16] and may emerge weeks or months before slip initiation. Subsequently, and without a clear history of injury, frank slippage of the femoral epiphysis on the metaphysis ensues along with exacerbation of hip associated symptoms: Pain, stiffness, restriction of flexion, adduction and internal rotation, increased external rotation and shortening of the affected lower limb (**Fig. 1**). Forced external rotation with increasing hip flexion (Drehman's sign) is characteristic of the established slip and may be obvious even on simple daily activities like sitting on a couch. Hip abduction is restricted due to proximal migration of the femoral neck on the proximal femoral epiphysis, while restriction of hip flexion in neutral rotation is due to the anterior sliding of the femoral neck relative to the epiphysis. The amount of restricted hip

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

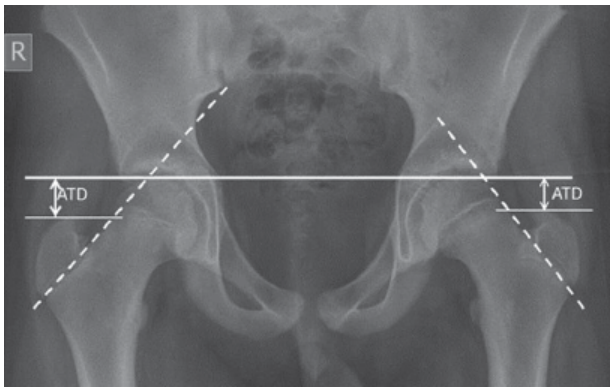

Fig. 5. *A slipped capital femoral epiphysis is strongly suspected in case of a non-traumatic limp of the adolescent. The Klein line (dotted line) on the AP pelvis view is not always diagnostic of a slip. Secondary radiologic signs on the AP pelvis view are helpful in order to set the diagnosis of a slipped epiphysis. The acetabulotrochanteric distance (ATD: the vertical distance between the superolateral edge of the acetabulum and the greater trochanter) should be equal for both hips. A difference ≥ 2 mm is indicative of a slip on the shorter side (left hip)*

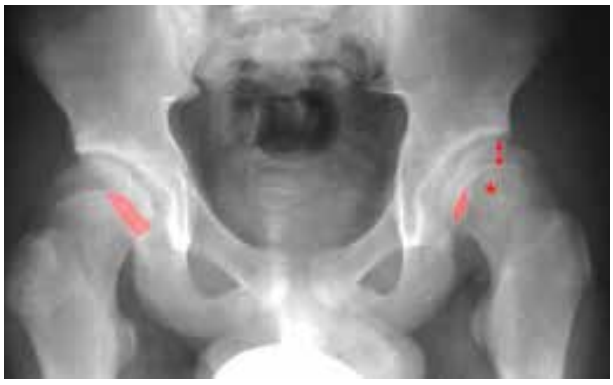

Fig. 6. *The secondary diagnostic signs of SCFE of the Left hip on the AP pelvis view. These signs are the result of posterior, downward and medial displacement of the capital femoral epiphysis relative to the femoral neck. A positive Capener's sign is the result of decreased overlapping of the medial part of the femoral neck on the posterior acetabular wall (less shaded area on the affected Left hip compared to the healthy Right hip). Increased density at the upper margin of the femoral neck is due to the overlapping of the retroverted capital femoral epiphysis and the femoral neck (Steel-Blomberg sign, asterisk). The height of the affected Left capital femoral epiphysis is smaller compared to the Right healthy hip (double arrow) because of the posterior version of the slipped epiphysis*

flexion and abduction are useful in the preoperative planning of a Southwick's subtrochanteric correctional osteotomy [7].

Mild slips do not cause significant gait alterations compared to healthy children of the same age and weight [34]. However, moderate and severe slips lead to the development of compensatory movements during gait cycle in order to avoid the impingement of the deformed femoral neck on the acetabulum: Reduced hip abduction leads to pelvic obliquity at the coronal level due to ipsilateral hip elevation. As a result, apparent limb length discrepancy with a shorter limb on the SCFE side may be observed, that is added to the true ipsilateral shortening due to the actual proximal migration of the femoral neck [35]. Compensatory ipsilateral knee extension and ankle flexion along with opposite changes in the contralateral healthy extremity follow. Out-toeing of the affected limb compensates for femoroacetabular impingement during hip flexion, thus exacerbating the external rotation attributed to the primary deformity of the slip. In order to maintain normal foot progression angle and to restrict out-toeing, the patient advances the pelvis on the SCFE side (a forward turn of the pelvis on the transverse plane). However, besides slip severity, other factors may also affect the gait cycle in SCFE patients, such as the inclination of the femoral neck and/or the acetabulum [35].

7. The Natural history of SCFE

The long-term results after *in situ* stabilization are reportedly quite good and most patients are satisfied for decades [8,36,37]. Depending on the residual deformity and the patient's activities, patients seem to be satisfied with their hip for an initial postoperative period of months or years [38,39,40], yet they report a permanent reduction of internal hip rotation. It seems that SCFE patients do not significantly restrict daily activities, or alter their social or working life [36].

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

However, despite the widespread view that if SCFE is not complicated by aseptic necrosis or chondrolysis patients are "doing well", it seems that at mean age of 35 years, patients cannot run due to hip pain and stiffness [41]. After this age, femoroacetabular impingement becomes painful, indicating permanent labral and acetabular cartilage lesions that ultimately result in secondary hip osteoarthritis [39]. Even small slips are not spared from this process [42,43,44,45].

The risk of deterioration of hip function and the development of secondary osteoarthritis increases with increasing severity and duration of the slip [16,36,37,42,46], with mild slips having favorable results compared to moderate and severe slips [42]. Overall, SCFE patients undergo a total hip replacement at a younger age, about ten years earlier compared with patients with primary osteoarthritis [1,41].

8. Radiology

Imaging tests are mandatory in order to set the diagnosis, to describe the severity of the slip and to monitor short and long term outcomes after slip stabilization. Pelvis X-rays, Anteroposterior (AP) and Frog Lateral (FL) views are easily obtained and are sufficient to set the diagnosis in almost all cases (**Table 2**).

The anteroposterior (AP) plain X-ray projection of the pelvis (**Fig. 5, Fig. 6**) is usually ordered first when examining a limping child. In this projection the following signs are diagnostic of SCFE [47]:

- The femoral neck line (Klein's line) does not intersect the capital femoral epiphysis (**Fig. 5**). This sign is occasionally referred as "the Trethovan's sign"[48,49]. In his original paper Klein states that "the continuation of the femoral neck line does not intersect the capital femoral epiphysis *compared* to the contralateral normal hip"[11]. Klein describes this sign only on the AP pelvis view, and reports that it is positive only in 68% of cases. On the frog lateral pelvis view Klein describes a relative flattening of the anterior surface of the femoral neck, compared to the concave configuration seen on the normal hip (Klein actually describes the loss of head-neck offset)[11].
- The proximal femoral growth plate is wide and irregular compared to the normal hip (**Fig. 6**).
- Decreased height of the capital femoral epiphysis compared to the healthy contralateral hip (**Fig. 6**), due to the retroversion of the capital epiphysis.
- The Metaphyseal Blanch sign (Steel-Bloomberg sign, **Fig. 6**): Increased density on plain x-ray caused by the overlapping of the retroverted capital femoral epiphysis on the anteverted femoral neck.
- The Capener's sign: Decreased overlapping of the femoral neck on the posterior acetabular wall, due to external rotation of the femoral neck and posteromedial version of the capital epiphysis [50], (**Fig. 6**).
- The acetabulotrochanteric distance (ATD, **Fig.5**) is the distance between a line that spans the superolateral edge of the acetabulum and a line parallel to the first line, that passes through the top of each greater trochanter [48]. A difference between the two sides >2 mm is indicative of SCFE. Normally, the ATD is >20 mm [51]. An ATD <5 mm is associated with a positive Trendelenburg's sign [52].
- The acetabulotrochanteric angle (ATA) is the angle formed between the line connecting the superolateral edge of the acetabulum and the line that connects the tip of the greater trochanter. An ATA $>1^0$ is indicative of SCFE [48].
- A disruption of the Shenton line due to proximal migration of the femoral neck.

The signs indicating SCFE on the AP pelvis view have high specificity but low sensitivity. Mild slips may not be apparent (false negative), thus increasing the risk to miss the diagnosis [49]. Ways to overcome this problem have been proposed. In mild slips, the Klein's line usually cuts the proximal capital femoral epiphysis, but not at the same extent as on the contralateral hip. The comparison of the width of the segment of the capital femoral epiphysis that is cut by the Klein's line between the suspected for SCFE and the contralateral hip may point out the diagno-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

TABLE 3. *The frequency of diagnostic signs for SCFE in the Frog Lateral and AP pelvis X-ray projection. The frog lateral pelvis view is superior to the AP pelvis view in establishing the diagnosis of SCFE* [15]

Pelvis X-Ray	**Frog Lateral**					**AP PELVIS VIEW**
	Frog Klein	Wide physis	AP Klein	Narrow epiphysis compared to healthy contralateral	Capener's sign	Metaphyseal blanch (Steel) sign
n	52	41	27	24	24	7
%	100	78.8	51.9	46.1	48.1	13.4

sis of SCFE. In fact, Klein originally described this difference between the SCFE hip and the healthy contralateral hip [11]. This sign was studied quantitatively: A difference > 2 mm between the suspected for SCFE and the normal contralateral hip is indicative of SCFE (79% sensitivity) [53].

The importance of the Frog Lateral (FL) pelvis projection must be emphasized (**Fig. 7**). The frog lateral view is an anteroposterior pelvis view with the hips and knees flexed about 45°, the hips are abducted and the soles of the feet are in contact and fully opposed [11,54]. The frog lateral view seems to be the examination of choice, because it is diagnostic in almost 100% of SCFE cases [15,55]. In the FL view, the x-ray beam is almost parallel to the plane of the deformity and thus the FL view is efficient to portray the anterior gliding of the femoral neck on the capital femoral epiphysis. In the FL view the Klein's line is always shown to intersect the capital femoral epiphysis (100% sensitivity and specificity). Therefore, it has been suggested that the FL projection should be the examination that should be ordered first, prior to the classic AP view, when investigating a nontraumatic limp of the adolescent [15]. It is important to obtain radiographs not only of the painful hip, but also of the contralateral hip, in order to rule out bilateral disease [49]. The sensitivity of diagnostic signs of SCFE on the AP and Frog Lateral pelvis view is shown in **Table 3** [15].

It has been postulated that the FL pelvis view may exacerbate slippage because it requires extreme external rotation and hip abduction. Moreover, especially in cases of unstable SCFE, the clinical presentation is too dramatic to allow any standard positioning of the patient in order to obtain the FL view. In such cases the Billing pelvis projection has been proposed: supine patient, knee flexed at 90°, lower limb elevated by 25° [56,57].

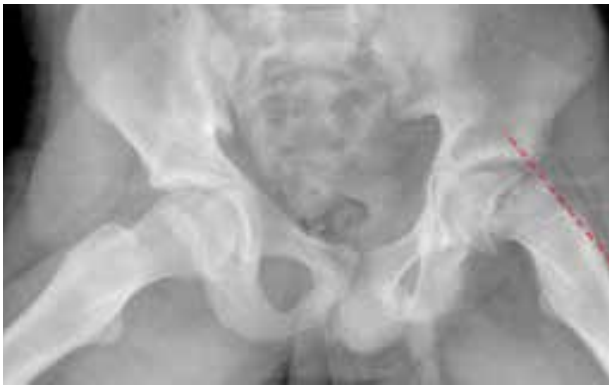

Fig. 7. *The frog lateral pelvis projection is the examination of choice for the diagnosis of SCFE. In this projection the Klein's line is almost always shown not to transect the capital femoral epiphysis*

Imaging modalities, such as Bone scan, Computerized Axial Tomography (CAT) and Magnetic Resonance Imaging (MRI) are not first-line examinations, because the diagnosis and treatment of SCFE is largely based on plain radiographs. CAT scan, bone scan and MRI are rarely useful, and only in case of inconclusive plain radiographs (e.g. bilateral pre-slips) or in order to early diagnose SCFE complications such as avascular necrosis of the femoral head or labral and acetabular cartilage pathology due to femoroacetabular impingement. Ultrasound examination of the affected hip may add some information in order to classify the slip: The detection of a haemarthrosis

TABLE 4. *SCFE classification*

1. based on clinical presentation:
- **a. according to the duration of symptoms**
  - i. acute (< 3/52)
  - ii. chronic (> 3/52)
  - iii. acute on chronic
- **b. according to the severity of the symptoms – ability to walk (Loder)**
  - i. stable (walks in, 95%)
  - ii. unstable (wheels in, 5%)

1. based on imaging of the slip: frog – lateral pelvis view
- **a. Southwick 1967 (head-shaft angle, compared with contralateral hip, if not both hips on the X-ray, 12° are subtracted from the head-shaft angle of the affected hip):**
  - i. mild < 29°
  - ii. moderate 30–50°
  - iii. severe > 50°
- **b. Wilson 1965 (% slippage of the epiphysis on the metaphysis)**
  - **i. mild < 33%**
  - **ii. moderate 33-50%**
  - **iii. severe > 50%**

increases the likelihood of an unstable SCFE, while remodelling and callus formation on the posteroinferior head-neck junction sets the diagnosis of a chronic slip [9,10].

9. Classification

SCFE is classified according to the duration of the symptoms as acute (<3/52), chronic (>3/52) or acute on chronic (>3/52 with episodes of exacerbation). Callus formation on the posteroinferior aspect of the femoral neck-head junction, discriminates acute from chronic SCFE. This callus is obvious three weeks after acute slip onset [10]. However, this classification is not helpful in predicting the risk for the most serious complication of SCFE, namely avascular necrosis (AVN) of the capital femoral epiphysis. On the contrary, a classification according to the ability of the patient to walk is more efficient to predict the risk of AVN [59]. If the patient is ambulating ("walks in"), SCFE is deemed "stable". A dramatic, extremely painful clinical presentation with the patient completely unable to walk, even with support, is characteristic of an "unstable" SCFE. In this case the patient is admitted to the emergency department lying on a stretcher ("wheels in"). Clinically stable cases make up the majority of SCFE (95%), the rest (5%) beeing unstable slips [16]. AVN is the dominating complication of unstable slips (50%) [16], but never complicates *in situ* fixed stable slips [60]. Currently accepted classifications of SCFE are summarized on **Table 4**.

Most cases of unstable SCFE are indeed an acute fracture (epiphysiolysis) through the growth plate. However, the different clinical presentation between a stable or an unstable SCFE does not always correspond to a distinct pathology. Not infrequently, the clinical presentation of instability can be observed without an acute separation of the growth cartilage, while there are clinically stable slips with a true epiphysiolysis but actual retention of the femoral head in place by an intact, firm perichondrium. This has been observed intraoperatively in cases treated with surgical hip dislocation and modified Dunn procedure. These cases revealed the entire spectrum of pathology between stable and unstable SCFE, from a stable growth cartilage to full epiphysiolysis. In fact, significant intraoperative instability (easy separation of the epiphysis) was noted in 50% of the clinically stable slips! The authors described the proximal growth cartilage of the hip of these patients to be "disconnected" or "disrupted" and classified the intraoperative stability of the growth cartilage as stable (intact growth cartilage, intact perichondri-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

um), easy surgical separation (intact perichondrium, easy cartilage separation), and gross instability (torn perichondrium and physis) [61,62].

If an intraarticular effusion detected by hip ultrasound is used as an index of physis instability, the frequency of unstable SCFE rises to 25-60%, which is much higher than that observed for clinically unstable SCFE [63]!

It has been supported that the vast majority of unstable slips present precursor symptoms, that emerge about six weeks before slip onset [64]. Consequently, most slips could receive treatment before they - spontaneous or after minor trauma - convert to the unstable type. In fact, the two classifications (chronicity *vs.* clinical presentation) are largely identical, because an intraoperatively stable physis may be clinically extremely painful, mimicking the clinical presentation of an unstable SCFE [62]. Therefore, a more accurate diagnosis of a stable or an unstable SCFE could be obtained with MRI [65] or ultrasound scan of the affected hip. The presence of hip effusion without sings of head-neck junction remodelling makes the diagnosis of an unstable SCFE highly possible. Absence of joint effusion and positive signs of remodelling indicate a stable slip [9,10,63].

Severity assessment of SCFE is based either on the amount of the capital femoral epiphysis displacement on the femoral neck metaphysis, according to Wilson (maximum displacement irrespective of the plane in which it occurs: Mild 0-33%, moderate 33-50%, severe > 50%) [6], or on the measurement of the slip angle, according to Southwick [7]. In chronic slips, femoral neck remodelling and deformation makes the measurement of the displacement of the epiphysis on the metaphysis impossible [66]. The Southwick method overcomes this problem because slip angle measurement is not affected by femoral neck remodelling. The slip angle is the difference of the head-shaft angle between the painful and the healthy contralateral hip. The head-shaft angle (**Fig. 8**) is measured on the FL projection and is deemed the angle formed between the axis of the diaphysis and the axis of the epiphysis (axis of the epiphysis: a line perpendicular to the line connecting both ends

Fig. 8. *The head-shaft angle (a,b) is measured on the frog lateral pelvis view. It is formed between the axis of the femoral shaft and the axis of the capital femoral epiphysis (a line vertical to the line that spans the epiphysis). The Southwick's slip angle is the difference between the head-shaft angle of the affected and the healthy hip: Slip angle = a-b*

of the epiphysis). According to the slip angle, SCFE is classified as mild (slip angle <30^0), moderate (30^0-50^0) and severe (> 50^0). In case of bilateral disease, or when both hips are not pictured on the FL view, the slip angle is the head-shaft angle of the affected hip minus 12^0, which is considered to be the maximum head-shaft angle of the normal hip [67].

The slip angle correlates with the outcome of the disease. Mild [7] and moderate [37,42] slips have better prognosis compared to the poor prognosis of severe slips [16]. Furthermore, the slip angle is useful in preoperative planning of a Southwick's intertrochanteric correctional osteotomy: It represents the amount of correction that is required in the sagittal plane [7].

10. Treatment

Whatever the clinical presentation, SCFE is a surgical emergency. Surgery for SCFE aims to stabilize the slip and to minimize post-slip sequellae, such as femoroacetabular impingement and secondary hip osteoarthritis [16,45]. Decision making includes the selection of the recommended for each case surgical technique (*in situ* stabilization, anatomic or compensatory osteotomy) and the selection of the proper implant (pin, screw) for slip stabilization, in order to ensure a successful surgical outcome. Important factors that should be considered

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

are the stability (stable - unstable) and the severity (mild, moderate, severe) of the slip, the remaining growth (open or closed triradiate cartilage), the risk for complications that may compromise long term outcome and the surgeon's experience on modern surgical techniques, such as hip arthroscopy and surgical hip dislocation. SCFE surgery deals with the following situations:

A) Treatment of stable SCFE (mild, moderate, severe)
B) Treatment of unstable SCFE
C) Treatment of the contralateral hip: Is prophylactic fixation of a painless contralateral hip justified?

10.a. The treatment of stable SCFE

The treatment of stable slips is dictated by the severity of the deformity, the child's remaining growth and the surgeon's preference and surgical experience. *In situ* stabilization is still the preferred treatment for stable slips. Novel surgical techniques, such as hip arthroscopy or surgical hip dislocation, have been recently implemented, while older techniques (compensatory subtrochanteric femoral osteotomies) are no longer favored as in the past.

10.a.1. Treatment of stable mild slips

In situ stabilization is the universally accepted treatment for stable mild slips (slip angle $<30^0$), preferably by a technique that does not restrict the remaining growth of the proximal femoral epiphysis [68]. If hip flexion is greater than 90^0 and there is no Drehman sign (mandatory external rotation with hip flexion), implant removal, if ever decided, will be the only future hip surgery related to SCFE. However, mild slips often present restricted internal rotation with hip flexion (indicating femoroacetabular impingement) and arthroscopic findings of labral and articular cartilage damage [43,69]. Subsequently, it has been supported that, in case of femoroacetabular impingement, arthroscopic osteochondroplasty should be done simultaneously or soon after *in situ* slip stabilization [43,45,68], especially if no substantial spontaneous improvement of the deformity is anticipated (a closed triradiate cartilage indicates poor remaining growth and remodelling of the deformed femoral neck).

10.a.2. Treatment of stable severe slips

Bone remodelling is ineffective to improve the deformity of the femoral neck after a severe slip, especially in older children with a closed triradiate cartilage [70]. Thus the basic question in the treatment of stable severe slips (slip angle $> 50^0$) is whether *in situ* stabilization is sufficient to restore function and long-term hip health or if some type of femoral osteotomy (anatomic-corrective, reorientation-compensatory) should be implemented [57] in order to avoid femoroacetabular impingement.

Therefore, in stable severe slips, many surgeons suggest that *in situ* stabilization should be combined either with a corrective (anatomic reduction of the slip, through the growth cartilage) osteotomy [43,68,71] or with a femoral head reorientation (compensatory, non-anatomic) osteotomy (at the middle or the base of the femoral neck, intertrochanteric or subtrochanteric). Femoral head reorientation osteotomies are by definition performed away from the plane of the deformity. According to the level of the osteotomy, femoral osteotomies for SCFE are either intra-articular (Dunn: Subcapital osteotomy - epiphysiodesis, Fish: Femoral neck osteotomy and anterior bone wedge removal, Kramer: Osteotomy of the base of the femoral neck) or extraarticular (Imhaeuser, Southwick). Intraarticular osteotomies have a serious risk of injury to the nutrient vessels of the femoral head and may cause avascular necrosis. Extraarticular osteotomies (Southwick: Sub-trochanteric flexion-valgus osteotomy, Imhauser: Subtrochanteric flexion-internal rotation osteotomy) improve hip movement and gait, but are rather salvage than reconstruction procedures [7] and do not have favorable long term results, especially in severe deformities. However, extraarticular osteotomies are useful even in late cases, after proximal femoral physis fusion [1].

Complications, such as femoroacetabular impingement after stable SCFE [38,45,69] and avascular necrosis after unstable SCFE [16] or after any attempt to reduce stable SCFE [4,6], urged for novel surgical techniques, in order to restrict these complications. Following the studies of the vascular anatomy of the hip [72] and the description of Surgical Hip Disloca-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

tion [73], the anatomic reduction of the capital femoral epiphysis on the femoral neck metaphysis has become an attractive goal of SCFE treatment.

Surgical hip dislocation (SHD) is a procedure that spares the blood vessels of the femoral head. These vessels pass through intra-articular synovial plicae, known as the retinaculi of Weitbrecht: lateral (upper/superolateral) retinaculum, anterior retinaculum and medial (inferior/posteroinferior) retinaculum [74]. After dividing the capsule, the retinaculi of Weitbrecht are exposed. The synovium is incised longitudinally, between the lateral and the anterior retinaculum. The incision of the synovium is extended distally and laterally, thus creating two giant flaps, the posterosuperior (referred also as posterolateral) and the anteroinferior (or anteromedial) flap. The posterosuperior flap contains the main terminal branches of the deep branch of the medial circumflex artery (deep femoral artery) that supply the weight bearing surface of the femoral head. These flaps extend quite distally until the level of the lesser trochanter. Essentially, the entire proximal femur is stripped off its soft tissue envelope (the fibrous capsule, the synovium and the anterior and posterior periosteum of the proximal femur). The insertion of the external rotators of the hip is part of the posterosuperior flap, following osteotomy of the posterior third of the greater trochanter, in order to protect the branches of the medial circumflex artery. The flaps attach centrally to the periphery of the femoral head and distally continue to the femoral periosteum. At this point the slip is fully visible. Subsequently, the epiphysis is detached from the metaphysis (if not already separated from the metaphysis as in the case of an unstable slip). The two retinacular flaps open like an envelope from which the central part of the femur (without soft tissue attachments and without the femoral head epiphysis) emerges. Removal of the posteroinferior callus and of the remaining physis is performed, in order to facilitate reduction of the epiphysis without tension on the nutrient vessels of the epiphysis (physis removal leads to a shorter femoral neck) and to promote epiphysiodesis. The ligamentum teres is divided and the femoral head epiphysis (with the retinaculi attached at its periphery) is anatomically reduced on the femoral neck and is stabilized with K-wires [61,75]. Intraoperative assessment of femoral head blood flow is possible at this point [61]. This method is known as the modified Dunn procedure, as opposed to the originally described Dunn subcapital osteotomy, in which the epiphysis was reduced on the metaphysis after removal of an anterior wedge (1-2 cm) of the femoral neck and of the posteroinferior callus, by means of a lateral transtrochanteric approach to the hip, but without hip dislocation [66]. Dunn reported that avascular necrosis was observed mainly when treating acute on chronic slips, because the nutrient vessels of the epiphysis were compressed on the newly formed neck callus. The removal of this callus and the protection of the retinacular vessels by careful elevation of the synovium off the femoral neck was part of the originally described Dunn procedure [66,76]. However, Dunn's osteotomy was abandoned due to increased rates of aseptic necrosis (21%) [77].

It is of historical interest to mention that Dunn used his own classification for SCFE (acute traumatic, early chronic, acute on chronic, severe chronic with open physis, severe chronic with closed physis) [66]. It is clear that Dunn was aware of the two main clinical presentations of SCFE according to the ability of the patient to walk or not to walk, for which Loder used the terms stable and unstable SCFE [59]. Dunn recommended *in situ* fixation (pin in the position of deformity) if the amount of the slip is less than one third of the width of the femoral neck and osteotomy (neck or trochanteric) in more severe slips. Furthermore, he considered that an osteotomy at the trochanteric level just "masks" the clinical deformity of the lower limb, and if an impingement of the femoral neck on the acetabulum still exists, it should be treated with removal of the excess bone of the anterior neck [66].

The modified Dunn osteotomy is an interesting alternative compared to *in situ* stabilization of the slip [78], but there are no clear indications for its widespread use. It is applicable in unstable and moderate and severe stable SCFE, either as primary treatment or later, after *in situ* stabilization, in order to avoid the late complications of femoroacetabular impingement

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

and osteoarthritis, provided that the growth cartilage is still open. The Ganz's team recommends the modified Dunn procedure for moderate and severe slips, while mild slips are candidates for anterosuperior neck osteochondroplasty after *in situ* fixation [43,62]. The Pediatric Orthopaedic Society of North America (POSNA) considers the modified Dunn procedure as the ideal treatment for moderate and severe stable slips [79]. Other surgeons prefer the modified Dunn procedure for the treatment of severe slips, due to better clinical outcome and less risk for additional future surgery [71]. It seems reasonable, that restoration of hip anatomy by means of SHD theoretically promotes long-term survival of the hip [45]. However, there are several arguments that do not justify routine use of SHD in the treatment of SCFE. First of all, SHD has not been validated by prospective randomized studies. Second, slip severity (Southwick's slip angle) does not clearly correlate with the risk of symptomatic femoroacetabular impingement [40]. Third, although the team of Ganz [61] reports excellent results with almost zero risk of avascular necrosis after surgical hip dislocation for SCFE, case series from other centers report a much higher risk of avascular necrosis with this technique (20-26%) [80,81]. Additionally, the minimal risk of avascular necrosis following *in situ* stabilization, as well as the extremely demanding technique of SHD, are strong arguments against the adoption of the modified Dunn's procedure instead of *in situ* stabilization, even for the treatment of severe stable slips [81]. Similarly, other studies suggest that the excellent long-term outcomes after *in situ* stabilization do not justify open surgical techniques in mild and moderate slips [82].

10.a.3. Treatment of stable moderate slips

Treatment of stable slips of moderate severity (Slip angle 30°-50°) is controversial, since these slips comprise an unclear entity between mild and severe SCFE. Wilson argued that patients would probably have to accept the residual deformation after *in situ* stabilization and not undergo compensatory osteotomy [6]. Other authors state that the *in situ* stabilization of stable moderate slips gives very good long-term results [83], especially if hip flexion is greater than 75° and there is no significant external rotation. In younger children with adequate residual growth and remodelling potential (open triradiate cartilate), stabilization of the slip should be achieved by a growth preserving surgical technique. If internal rotation is < 10^0 in 90° of hip flexion [44], or if there is symptomatic femoroacetabular impingement, some surgeons recommend arthroscopic treatment, while others prefer the modified Dunn osteotomy either as a primary procedure (if the triradiate cartilage is closed), or later, provided that the proximal femoral physis is still open [68]. In case of a closed proximal femoral physis, a compensatory osteotomy (Imhaeuser or Southwick), may be an option [68].

In brief, there are two extremes of SCFE: Mild slips and severe slips. The aim of treatment is to stabilize the slip and to prevent and/or to treat femoroacetabular impingement in order to avoid permanent labral and articular cartilage damage of the acetabulum. It seems that the cut-off point for femoroacetabular impingement is a slip angle of about 30^0. In such slips, *in situ* stabilization (along with primary or late arthroscopic osteochondroplasty) is probably the best option [84]. In moderate and severe slips, *in situ* stabilization will probably not suffice to prevent femoroacetabular impingement. These slips may present better long term results if treated primary with an anatomic osteotomy (modified Dunn procedure) or by a compensatory osteotomy (Imhaeuser, Southwick). However, until prospective randomized studies will point out the most appropriate method of treatment, *in situ* stabilization remains the treatment of choice for all stable slips.

10.a.4. Closed reduction for stable SCFE?

The extremely high risk of avascular necrosis of the femoral head if closed reduction of a stable slip is attempted has been described since almost a century ago: "Forcible manipulation of an old case ... is not justified" (Key, 1926) [4]. It is well known that the manipulations of closed anatomic reduction of a stable SCFE always result in direct pressure and occlusion of the femoral head vessels by the callus that is formed at the posteroinferior femoral neck-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

head junction [6,8,85]. On the contrary, aseptic necrosis almost never complicates *in situ* stabilized stable SCFE [60]. Therefore, a stable slip is an absolute contraindication for closed reduction.

10.b. The treatment of unstable SCFE

The following options are available for the treatment of unstable SCFE:

1. Incidental (postural) closed reduction of the epiphysis on the metaphysis after simple positioning of the patient on the surgical table, without any manipulation and any attempt to obtain anatomic reduction. *In situ* stabilization (with or without decompression of the hematoma within 8-24 hours) follows. The quality of the reduction and hence the long-term results of this treatment are a matter of luck.

2. Targeted closed reduction: An attempt of closed reduction with manipulation of the hip under general anesthesia, with or without decompression of the hematoma, is followed by pin or screw fixation. However, manipulations towards closed reduction may compress the retinacular vessels against the posteroinferior call us of the neck metaphysis and thus lead to avascular necrosis of the capital femoral epiphysis.

3. Partial anatomical reduction of the capital femoral epiphysis (Parsch method) is achieved by limited anterior arthrotomy of the hip and reduction of the slip by direct pressure on the neck metaphysis. Reduction stops when the epiphysis reaches the posteroinferior neck callus. Fixation at this point follows [86].

4. Anatomical reduction of the capital femoral epiphysis, open (Modified Dunn Procedure) [61] or arthroscopic [87], is accomplished after posteroinferior neck callus removal.

Since there are no prospective randomized trials to evaluate the efficacy of each method, the simplest and most accepted treatment is the emergent (within 8-24 hours) postural reduction of the slip after hematoma decompression, followed by *in situ* stabilization. The patient has to be informed about the high risk of complications, especially avascular necrosis, and the increased likelihood of secondary future surgery in order to address these complications [67,88].

10.b.1. The timing of unstable SCFE surgery

The timing of unstable SCFE surgery bears some controversy. Some authors suggest that the blood supply of the femoral head is at risk at the moment the acute epiphysiolysis occurs [16,58,63], while others support that this risk is higher during reduction and stabilization of the unstable slip [89]. It seems that the vascular compromise of the capital femoral epiphysis is multifactorial and may be the result of rupture, anatomic obstruction or functional occlusion of the retinacular vessels due to increased intraarticular pressure that very often accompanies an unstable slip [90].

The risk of AVN is in some way related to the timing of unstable SCFE surgery. The sooner the femoral head is reduced and stabilized, the greater the probability to restore the blood supply and to avoid AVN, probably because mechanical obstruction or kinking of the nutrient vessels is early reversed [90]. Treatment within 8-24h (urgent) is associated with a significantly lower incidence of AVN compared to the post 24-hour treatment [46]. If surgery is performed between 24 hours and 7 days after the acute slip, the risk of AVN seems to rise, and drops again if surgery is done after the first week. These observations suggest that there is an unsafe window that is related with a higher risk of AVN. This window extends from 24 hours to 7 days after the acute slip, and should be bypassed, in order not to increase the risk of AVN after an acute slip [57,91,92,93].

10.b.2. Hematoma decompression prior to reduction and stabilization of unstable SCFE

The unstable SCFE is an intraarticular acute epiphysiolysis and hence it is accompanied by hematoma formation. The intraarticular pressure rises and may subsequently compromise the vascular supply (veins, arteries) of the femoral head. In order to reduce intraarticular pressure and pain, the hip acquires the resting position, which is slight flexion, abduction and external rotation. Any reduction maneuvers, especially longitudinal traction, cause further increase of the hip joint pressure [45,90] that may even exceed the pressure of the compartment [89]. Hence, manipulations to reduce an unstable

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

SCFE may indeed be part of the etiology of avascular necrosis of the femoral head [89]. Therefore, hip joint hematoma decompression, either by hip arthrotomy or by a less invasive hip puncture, prior to any attempt to reduce the acute slip, is a reasonable option for the surgeon and is suggested to reduce the risk of avascular necrosis of the femoral head [88,94].

10.b.3. Incidental or anatomic reduction of unstable SCFE?

Symptoms that precede weeks before an unstable slip [64] as well as the intraoperative detection of newly formed callus at the posteroinferior neck metaphysis in cases treated by surgical hip dislocation [62], indicate that some unstable slips probably started as stable slips and after a period of time exacerbated and turned acutely to the unstable form of the disease. Any attempt of anatomic (targeted) reduction of the unstable slip by means of traction and manipulation of the lower limb results in compression of the femoral head vessels against the posteroinferior neck callus with subsequent obstruction of the blood flow into the femoral head and is therefore not recommended. Closed reduction of the unstable slip should be obtained without any manipulations of the lower limb (incidental, postural, random), as a result of careful positioning of the patient on the operating table. The ideal position of the lower limb in order to achieve incidental reduction is: stress free 15°-20° internal rotation and 20°-30° of abduction of the lower limb [95]. However, avascular necrosis after incidental unstable SCFE reduction is still extremely high and may complicate up to 47% of the unstable slips [59].

Non-anatomical reduction is achieved by the Parsch method that, reportedly, is accompanied by aseptic necrosis in only 4.7-10% of cases [86,90]. After a limited anterior approach without hip dislocation, the femoral neck is gently pushed back until the capital femoral epiphysis attains not the anatomical position, but the position it had prior to the unstable slip. Anatomic reduction is avoided, since it would compromise the femoral head blood flow either by increasing tension on the nutrient vessels or by compressing the nutrient vessels against the posteroinferior callus of the femoral neck. Arthroscopically assisted reduction of unstable SCFE may be an option to overcome this obstacle [96].

Anatomical reduction of the unstable SCFE without jeopardizing the femoral head perfusion is possible only after removal of the posteroinferior neck callus. This callus can be removed either by open surgical techniques (Dunn procedure, modified Dunn procedure) [73], or arthroscopically (without hip dislocation) [87]. The modified Dunn procedure has several advantages over the classic closed technique (incidental reduction and stabilization) in the treatment of unstable SCFE because anatomic reduction of the femoral head is accomplished only after careful exposure of the retinacular vessels, followed by removal of the posteroinferior callus and the remaining physis [45,46]. Intraoperative assessment of blood flow of the capital femoral epiphysis is also possible [61]. The easy separation of the epiphysis from metaphysis, as well as the smaller size of the posteroinferior neck callus of unstable slips compared to the callus observed in stable slips, make the modified Dunn procedure a quite attractive option in treating an unstable slip [97]. In addition, provided that the surgeon is familiar with surgical hip dislocation, the modified Dunn procedure seems to present a lower risk of avascular necrosis compared with the incidental reduction and stabilization technique (29% *vs.* 43%) [81]. It has been proposed that, if there is no experience or ability to perform a modified Dunn's procedure on an emergency basis (within 24 hours), the unstable slip should be treated provisionally by the classic incidental reduction and stabilization method and within a few weeks the patient should be referred to a center with experience on the surgical hip dislocation technique [45].

10.c. Post-operative treatment of SCFE

There are no specific postoperative follow up protocols for SCFE, stable or unstable. Follow up examinations depend on the patient's preoperative status (obesity, compliant patient), the type of the slip (stable, unstable), the type of fixation (pins, screw), the postoperative symptoms and the expected growth

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

of the patient. These factors correlate with the risk of further slipping, the risk of contralateral hip disease and the risk of early or late complications that are related to the implant (migration, loosening, pain) or the disease (avascular necrosis, femoroacetabular impingement, etc).

After *in situ* stabilization of a stable SCFE partial weight bearing until pain relief is recommended. Full weight bearing is usually allowed within a few weeks. After incidental reduction and stabilization of an unstable SCFE, the patient remains bedridden for 4-6 weeks, depending on the remaining hip pain and the patient's coexisting pathological conditions. Protected weight bearing is usually possible after that period, and the patient is allowed to walk with crutches until the acute epiphysiolysis is healed, as indicated by a painless hip.

The patient is observed every 4-6 months and is advised to carefully participate in competitive sports until the fusion of the growth cartilage of both hips is complete. After that point implant removal may be performed.

The treatment is completed with detection and reversal of any underlying endocrine disorder and with treatment of obesity as well. Regular follow up focuses on early detection and treatment of complications such as femoroacetabular impingement, avascular necrosis and chondrolysis, in order to provide long term hip survival. A

Conflict of interest

The authors declared no conflicts of interest.

REFERENCES

1. Wensaas A, Svenningsen S, Terjesen T. Long-term outcome of slipped capital femoral epiphysis: A 38-year follow-up of 66 patients. *J Child Orthop* 2011; 5(2): 75-82. Published online 2010 Dec 12. doi: 10.1007/s11832-010-0308-0. PMCID: PMC3058209.
2. Witbreuk Melinda Maria Eva Helena. Slipped Capital Femoral Epiphysis - Pathogenetic and Clinical aspects 2015; *Thesis*, ISBN: 978-90-6464-942-4.
3. Mercer Rang. *The Story of Orthopedics* 2000; W B Saunders ISBN 0-7216-7141-1, pp. 87-89.
4. Key JA. Epiphyseal coxa vara or displacement of the capital epiphysis of the femur in adolescence. 1926. *Clin Orthop Relat Res* 2013; 471(7): 2087-2117. doi: 10.1007/s11999-013-2913-y.
5. Murray RO. The aetiology of primary osteoarthritis of the hip. *Br J Radiol* 1965; 38(455): 810-24. PMID: 5842578, doi: .10.1259/0007-1285-38-455-810
6. Wilson PD, Jacobs B, Schechter L. Slipped Capital Femoral Epiphysis: An end result study. *J Bone Joint Surg Am* 1965; 47: 1128-1145.
7. Southwick WO. Osteotomy through the Lesser Trochanter for Slipped Capital Femoral Epiphysis. *J Bone Joint Surg Am* 1967; 49(5): 807-835.
8. Hägglund G, Hannson LI, Sandström S. Slipped capital femoral epiphysis in southern Sweden. Long-term results after nailing/pinning. *Clin Orthop Relat Res.* 1987; (217): 190-200.
9. Kallio PE, Lequesne GW, Paterson DC, et al. Ultrasonography in slipped capital femoral epiphysis. Diagnosis and assessment of severity. *J Bone Joint Surg Br* 1991; 73 (6): 884-889.
10. Kallio PE, Paterson DC, Foster BK, et al. Classification in slipped capital femoral epiphysis. Sonographic assessment of stability and remodeling. *Clin Orthop Relat Res* 1993; (294): 196-203.
11. Klein A, Joplin RJ, Reidy JA, et al. Slipped capital femoral epiphysis: Early diagnosis and treatment facilitated by "normal" roentgenograms. *J Bone Joint Surg Am* 1952; 34: 233-239.
12. Koczewski P: Valgus slipped capital femoral epiphysis: Subcapital growth plate orientation analysis. *J Pediatr Orthop B* 2013; 22(6): 548-52. doi: 10.1097/BPB.0b013e3283633136.
13. Shank CF, Thiel EJ, Klingele KE. Valgus slipped capital femoral epiphysis: Prevalence, presentation, and treatment options. *J Pediatr Orthop* 2010; 30(2): 140-146. doi: 10.1097/BPO.0b013e3181d076b6.
14. Loder RT, O'Donnell PW, Didelot WP, et al. Valgus

slipped capital femoral epiphysis. *J Pediatr Orthop* 2006; 26: 594-600.

15. Samelis P, Kantanoleon S, Meziridis I, et al. Missed Slipped Capital Femoral Epiphysis: The Frog lateral wiew is the examination of choice in the nontraumatic limping adolescent and should be ordered prior to the classic anteroposterior view. A retrospective analysis of 52 cases. Poster Presentation, 18th EFORT Congress, Vienna, 31 May - 02 June 2017.
16. Loder RT, Aronsson DD, Dobbs MB, et al. Slipped Capital Femoral Epiphysis. *J Bone Joint Surg Am* 2000; 82A(8): 1170-1188.
17. Loder RT. The demographics of slipped capital femoral epiphysis. An international multicenter study. *Clin. Orthop* 1996; 322: 8-27.
18. Novais EN, Millis MB. Slipped capital femoral epiphysis: Prevalence, pathogenesis, and natural history. *Clin Orthop Relat Res* 2012; 470(12): 3432-3438. doi: 10.1007/s11999-012-2452-y.
19. Podeszwa DA, Gurd D, Riccio A, et al. Increased acetabular depth may influence physeal stability in slipped capital femoral epiphysis. *Clin Orthop Relat Res* 2013; 471(7): 2151-2155. doi: 10.1007/s11999-013-2807-z. Epub 2013 Jan 26.
20. Gelberman RH, Cohen MS, Shaw BA, et al. The association of femoral retroversion with slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1986; 68: 1000-1007.
21. Tayton K. The epiphyseal tubercle in adolescent hips. *Acta Orthop* 2009; 80(4): 416-419. doi: 10.3109/17453670903153550.
22. Liu RW, Armstrong DG, Levine AD, et al. An anatomic study of the epiphyseal tubercle and its importance in the pathogenesis of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 2013 20; 95(6): e341-8. doi: 10.2106/JBJS.L.00474.
23. Liu RW, Fraley SM, Morris WZ, et al. Validity and Clinical Consequences of a Rotational Mechanism for Slipped Capital Femoral Epiphysis. *J Pediatr Orthop* 2016; 36(3): 239-246. doi: 10.1097/BPO.0000000000000438.
24. Witbreuk M, van Kemenade FJ, van der Sluijs JA, et al. Slipped capital femoral epiphysis and its association with endocrine, metabolic and chronic diseases: A systematic review of the literature. *J Child Orthop* 2013; 7(3): 213-223. doi: 10.1007/s11832-013-0493-8.
25. Manoff EM, Banffy MB, Winell JJ. Relationship between body mass index and slipped capital femoral epiphysis. *J Pediatr Orthop* 2005; 25: 744–746.
26. Macía-Villa CC, Sanchez-Lite I, Medina-Luezas J. Slipped capital femoral epiphysis in adults: Case report and review of literature. *Reumatismo* 2016; 68(1): 40-47. doi: 10.4081/reumatismo.2016.860.
27. Bhatia NN, Pirpiris M, Otsuka NY. Body mass index in patients with slipped capital femoral epiphysis. *J Pediatr Orthop* 2006; 26(2): 197-199. doi: 10.1097/01.bpo.0000218526.36362.3f
28. Allen DB. Safety of Growth Hormone Treatment of Children with Idiopathic Short Stature: The US Experience. *Horm Res Paediatr* 2011; 76 (suppl 3): 45-47, DOI: 10.1159/000330159.
29. Mostoufi-Moab S, Isaacoff EJ, Spiegel D, et al. Childhood cancer survivors exposed to total body irradiation are at significant risk for slipped capital femoral epiphysis during recombinant growth hormone therapy. *Pediatr Blood Cancer* 2013; 60(11): 1766-1771. doi: 10.1002/pbc.24667. Epub 2013 Jul 2.
30. Vedi A, Neville K, Johnston K, et al. Slipped capital femoral epiphyses after total body irradiation. *Pediatr Blood Cancer* 2014; 61(6): 1140. doi: 10.1002/pbc.24895. Epub 2014 Jan 10.
31. Hägglund G, Hansson LI. Slipped capital femoral epiphysis in three generations. *Acta Orthop Scand* 1986 Jun; 57(3): 240-242.
32. Loder RT, Nechleba J, Sanders JO, et al. Idiopathic slipped capital femoral epiphysis in Amish children. *J Bone Joint Surg Am* 2005; 87: 543-549.
33. Tresoldi I, Modesti A, Dragoni M, et al. Histological, histochemical and ultrastructural study of slipped capital femoral epiphysis. *J Child Orthop* 2017; 11(2): 87-92. doi: 10.1302/1863-2548-11-170023.
34. Song KM, Halliday S, Reilly C, et al. Gait abnormalities following slipped capital femoral epiphysis. *J. Pediatr Orthop* (2004); 24: 148-155.
35. Sangeux M, Passmore E, Gomez G, et al. Slipped capital femoral epiphysis, fixation by single screw *in situ*:

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

A kinematic and radiographic study. *Clin Biomech (Bristol, Avon)* 2014; 29(5): 523-350. doi: 10.1016/j.clinbiomech.2014.03.012. Epub 2014 Apr 12.

36. Ordeberg G, Hansson LI, Sandström S. Slipped capital femoral epiphysis in southern Sweden. Long-term result with no treatment or symptomatic primary treatment. *Clin Orthop Relat Res* 1984; (191): 95-104.
37. Carney BT, Weinstein SL, Noble J. Long-term follow-up of slipped capital femoral. Epiphysis. *J Bone Joint Surg* [Am] 1991; 73(A): 667-674.
38. Sink EL, Zaltz I, Heare T, et al. Acetabular cartilage and labral damage observed during surgical hip dislocation for stable slipped capital femoral epiphysis. *J Pediatr Orthop* 2010; 30: 26-30.
39. Ross PM, Lyne ED, Morawa LG. Slipped capital femoral epiphysis long-term results after 10-38 years. *Clin Orthop Relat Res* 1979; (141): 176-180.
40. Dodds MK, McCormack D, Mulhall KJ. Femoroacetabular impingement after slipped capital femoral epiphysis: Does slip severity predict clinical symptoms? *J Pediatr Orthop* 2009; 29(6): 535-539. doi: 10.1097/BPO.0b013e3181b2b3a3.
41. Larson AN, Sierra RJ, Yu EM, et al. Outcomes of slipped capital femoral epiphysis treated with *in situ* pinning. *J Pediatr Orthop* 2012; 32(2): 125-130. doi: 10.1097/BPO.0b013e318246efcb.
42. Carney BT, Weinstein SL. Natural history of untreated chronic slipped capital femoral epiphysis. *Clin. Orthop* 1996; 322: 43-47.
43. Leunig M, Horowitz K, Manner H, et al. *In situ* pinning with arthroscopic osteoplasty for mild SCFE: A preliminary technical report. *Clinical Orthopaedics and Related Research* 2010; 468 (12): 3160-3167.
44. Millis MB, Novais EN. *In situ* fixation for slipped capital femoral epiphysis: Perspectives in 2011. *J Bone Joint Surg Am* 2011; 93 Suppl 2: 46-51. doi: 10.2106/JBJS.K.00040.
45. Kim YJ, Sierra RJ. Report of Breakout Session: Slipped Capital Femoral Epiphysis management 2011. *Clin Orthop Relat Res*. 2012; 470(12): 3464-3466. Published online 2012 Oct 19. doi: 10.1007/s11999-012-2587-x.
46. Ziebarth K, Leunig M, Slongo T, et al. Slipped Capital Femoral Epiphysis: Relevant pathophysiological findings with open surgery. *Clin Orthop Relat Res* 2013; 471(7): 2156-2162.
47. Peck D. Slipped Capital Femoral Epiphysis: Diagnosis and management. *American family physician* 2010; 82(3): www.aafp.org/afp.
48. Song KS, Ramnani K, Min BW, et al. Acetabulotrochanteric distance in slipped capital femoral epiphysis. *J Pediatr Orthop*. 2011; 31(6): 644-7. doi: 10.1097/BPO.0b013e3182288ae3.
49. Sharma V, Oddy MJ. Slipped capital femoral epiphysis: A review. *Br J Hosp Med (Lond)* 2014; 75(3): 155-161.
50. Scham S. The triangular sign in the early diagnosis of slipped capital femoral epiphysis. *Clinical Orthopaedics & Related Research* 1974; 103: 16-17.
51. Ordeberg G, Hagglund G, Hansson L I, et al. Articulotrochanteric distance in slipped capital femoral epiphysis. *Arch Orthop Trauma Surg* 1990; 109 (4): 191-193.
52. Druschel C, Placzek R, Funk JF. Growth and deformity after *in situ* fixation of slipped capital femoral epiphysis. *Z Orthop Unfall* 2013; 151(4): 371-379.
53. Green DW, Mogekwu N, Scher DM, et al. A modification of Klein's Line to improve sensitivity of the anterior-posterior radiograph in slipped capital femoral epiphysis. *J Pediatr Orthop* 2009; 29(5): 449-53. doi: 10.1097/BPO.0b013e3181aa20cd.
54. Clohisy JC, Nunley RM, Otto RJ, et al. The frog-leg lateral radiograph accurately visualized hip cam impingement abnormalities. *Clin Orthop Relat Res*. 2007; 462: 115-121.
55. Pinkowsky GJ, Hennrikus WL. Klein line on the anteroposterior radiograph is not a sensitive diagnostic radiologic test for slipped capital femoral epiphysis. *J Pediatr* 2013; 162(4): 804–807.
56. Jerre R, Billing L, Hansson G, et al. Bilaterality in slipped capital femoral epiphysis: Importance of a reliable radiographic method. *J Pediatr Orthop B* 1996; Spring; 5(2): 80-84.
57. Vanhegan IS, Cashman JP, Buddhdev , et al. A.: Outcomes following subcapital osteotomy for severe slipped upper femoral epiphysis. *Bone Joint J* 2015; 97-B: 1718-1725.
58. Rhoad RC, Davidson RS, Heyman S, et al. Pretreat-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

ment bone scan in SCFE: A predictor of ischemia and avascular necrosis. *J. Pediat. Orthop* 19: 164-168, 1999.

59. Loder RT, Richards BS, Shapiro PS, et al. Acute slipped capital femoral epiphysis: The importance of physeal stability. *J Bone Joint Surg Am* 1993; 75(8): 1134-1140.
60. Kennedy JG, Hresko MT, Kasser JR, et al. Osteonecrosis of the femoral head associated with slipped capital femoral epiphysis. *J Pediatr Orthop* 2001; 21: 189-193.
61. Ziebarth K, Zilkens C, Spencer S, et al. Capital realignment for moderate andsevere SCFE using a modified dunn procedure. *Clin Orthop Relat Res* 2009; 467(3): 704–716. Published online 2009 Jan 14. doi: 10.1007/s11999-008-0687-4.
62. Ziebarth K, Domayer S, Slongo T, et al. Clinical stability of slipped capital femoral epiphysis does not correlate with intraoperative stability. *Clin Orthop Relat Res* 2012; 470(8): 2274-2279.
63. Kallio PE, Mah ET, Foster BK, et al. Slipped capital femoral epiphysis: Incidence and clinical assessment of physical instability. *J Bone Joint Surg* Br 1995; 77: 752-755.
64. McPartland TG, Sankar WN, Kim YJ, et al. Patients With Unstable Slipped Capital Femoral Epiphysis have antecedent symptoms. *Clin Orthop Relat Res* 2013; 471(7): 2132-2136. Published online 2013 May 9. doi: 10.1007/s11999-013-3042-3, PMCID: PMC3676584.
65. Tins B, Cassar-Pullicino V, McCall I. The role of pre-treatment MRI in established cases of slipped capital femoral epiphysis. *European Journal of Radiology* 70 2009; 570-578.
66. Dunn DM. The treatment of adolescent slipping of the upper femoral epiphysis. *J Bone Joint Surg* [Br] 1964; 46-B: 621-629.
67. Aronsson DD, Loder RT, Breur GJ, et al. Slipped capital femoral epiphysis: Current concepts. *J Am Acad Orthop Surg* 2006; 14(12): 666-679.
68. Schoenecker PL, Gordon JE, Luhmann SJ, et al. A treatment algorithm for stable slipped capital femoral epiphysis deformity. *J Pediatr Orthop* 2013 J; 33 Suppl 1: S103-11. doi: 10.1097/BPO.0b013e31829774d6.
69. Castañeda P, Ponce C, Villareal G, et al. The natural history of osteoarthritis after a slipped capital femoral epiphysis/the pistol grip deformity. *J Pediatr Orthop* 2013; 33 Suppl 1: S76-82. doi: 10.1097/BPO.0b013e318277174c.
70. Chotel F. Cannulated "pin-screw" fixation for slipped capital femoral epiphysis: An original concept to allow stabilisation and growth. EPOS 33rd Annual Meeting, 2014, Apr 2-5, Bruges, Belgium.
71. Novais EN, Hill MK, Carry PM, et al. Modified dunn procedure is superior to *in situ* pinning for short-term clinical and radiographic improvement in severe stable SCFE. *Clin Orthop Relat Res* 2015; 473(6): 2108-2117. doi: 10.1007/s11999-014-4100-1. Epub 2014 Dec 12.
72. Gautier E, Ganz K, Krügel N, et al. Anatomy of the medial femoral circumflex artery and its surgical implications. *J Bone Joint Surg* [Br] 2000; 82-B: 679-683.
73. Ganz R, Gill TJ, Gautier E, et al. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. *J Bone Joint Surg Br* 2001; doi: 83: 1119-1124.10.1302/0301-620X.83B8.11964.
74. Gojda J, Bartoníbek J. The retinacula of Weitbrecht in the adult hip. *Surg Radiol Anat* 2012; 34: 31-38. doi: 10.1007/s00276-011-0829-3.
75. Huber H, Dora C, Ramseier LE, et al. Adolescent slipped capital femoral epiphysis treated by a modified Dunn osteotomy with surgical hip dislocation. *J Bone Joint Surg* [Br] 2011; 93-B: 833-838.
76. Dunn DM, Angel JC. Replacement of the femoral head by open operation in severe adolescent slipping of the upper femoral epiphysis. *J Bone Joint Surg Br* 1978; 60-B(3): 394-403.
77. Abdelazeem AH, Beder FK, Abdel Karim MM, et al. The anatomical reduction of a moderate or severe stable slipped capital femoral epiphysis by modified Dunn subcapital osteotomy using the Ganz approach: Functional and radiological outcomes. *Bone Joint J* 2016; 98-B(9): 1283-1288. doi: 10.1302/0301-620X.98B9.37071.
78. Thawrani DP, Feldman DS, Sala DA. Current practice in the management of slipped capital femoral epiphysis. *J Pediatr Orthop* 2016; 36(3): e27-37. doi: 10.1097/BPO.0000000000000496.
79. Perry D. Management of slipped capital femoral epiphysis (SCFE) - A nationwide drive to develop evi-

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

dence based care. Volume 04 / Issue 02 / June 2016 boa.ac.uk, JTO Peer-Reviewed Articles.

80. Sankar N, Vanderhave KL, Matheney T, et al. The modified dunn procedure for unstable sipped capital femoral epiphysis. A multicenter perspective. *J Bone Joint Surg Am* 2013; 95 (7): 585-591. doi: 10.2106/JBJS.L.00203Abstract.
81. Souder CD, Bomar JD, Wenger DR. The role of capital realignment versus *in situ* stabilization for the treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 2014; 34: 791-798.
82. De Poorter JJ, Beunder TJ, Gareb B, et al. Long-term outcomes of slipped capital femoral epiphysis treated with *in situ* pinning. *J Child Orthop* 2016; 10(5): 371-9. doi: 10.1007/s11832-016-0759-z. Epub 2016 Jul 20.
83. Hansson G, Billing L, Högstedt B, et al. Long-term results after nailing *in situ* of slipped upper femoral epiphysis. A 30-year follow-up of 59 hips. *J Bone Joint Surg Br* 1998; 80(1): 70-77.
84. Murgier J, de Gauzy JS, Jabbour FC, et al. Long-term Evolution of Slipped Capital Femoral Epiphysis treated by *in situ* fixation: A 26 years follow-up of 11 hips. *Orthop Rev (Pavia)* 2014 3; 6(2): 5335. doi: 10.4081/or.2014.5335. eCollection 2014
85. Bellemans J, Fabry G, Molenaers G, et al. Slipped capital femoral epiphysis: A long-term follow-up, with special emphasis on the capacities for remodeling. *J Pediatr Orthop* 1996; B 5(3): 151-157.
86. Parsch K, Weller S, Parsch D. Open reduction and smooth Kirschner wire fixation for unstable slipped capital femoral epiphysis. *J Pediatr Orthop* 2009; 29: 1-8.
87. Roos BD, de Assis MC, Roos MV, et al. Arthroscopic subcapital realignment osteotomy in chronic and stable slipped capital femoral epiphysis: Early results. *Rev Bras Ortop*. 2017 J; 52(1): 87-94. Published online 2016 Dec 29. doi: 10.1016/j.rboe.2016.12.007.
88. Lykissas MG, McCarthy JJ. Should all unstable slipped capital femoral epiphysis be treated open? *J Pediatr Orthop* 2013; 33 Suppl 1: S92-98.
89. Herrera-Soto JA, Duffy MF, Birnbaum MA, et al. Increased intracapsular pressures after unstable slipped capital femoral epiphysis. *J Pediatr Orthop* 2008; 28: 723-728.
90. Loder R.T. What is the cause of avascular necrosis in unstable slipped capital femoral epiphysis and what can be done to lower the rate? *J Pediatr Orthop* 2013; 33 Suppl 1: S88-91.
91. Lowndes S, Khanna A, Emery D, et al. Management of unstable slipped upper femoral epiphysis: A meta-analysis. *Br Med Bull* 2009; 90: 133-146 [PMID: 19376800, doi: 10.1093/bmb/ldp012.
92. Otani T, Futami T, Kita A, et al. Treatment for unstable slipped capital femoral epiphysis: Current status and future challenge in Japan. *J Orthop Sci*. 2016; 21(6): 847-851. doi: 10.1016/j.jos.2016.07.024. Epub 2016 Sep 6.
93. Kohno Y, Nakashima Y, Kitano T, et al. Is the timing of surgery associated with avascular necrosis after unstable slipped capital femoral epiphysis? A multicenter study. *J Orthop Sci* 2016; 11. pii: S0949-2658(16)30159-2. doi: 10.1016/j.jos.2016.08.012.
94. Ibrahim T, Mahmoud S, Riaz M, et al. Hip decompression of unstable slipped capital femoral epiphysis: A systematic review and meta-analysis. *J Child Orthop*. 2015; 9(2): 113-120. doi: 10.1007/s11832-015-0648-x. Epub 2015 Mar 17.
95. Uglow MG, Clarke NMP. The management of slipped capital femoral epiphysis. *J Bone Joint Surg* [Br] 2004; 86-B: 631-635.
96. De Jong A, van Riet R, Melkebeek JV. Arthroscopically assisted reduction of an unstable severe slipped capital femoral epiphysis: A case report. *Acta Orthop*. Belg., 2012; 78: 271-274.
97. Rebello G, Spencer S, Millis MB, et al. Surgical dislocation in the management of pediatric and adolescent hip deformity. *Clin Orthop Relat Res* 2009; 467(3): 724-731. doi: 10.1007/s11999-008-0591-y. Epub 2008 Nov 12.

VOLUME 68 | ISSUE 4 | OCTOBER - DECEMBER 2017

READY - MADE CITATION

Samelis PV. Slipped Capital Femoral Epiphysis is a leading cause of femoroacetabular impingement and early onset hip osteoarthritis. A review. *Acta Orthop Trauma Hell* 2017; 68(4): 116-137.

ΠΕΡΙΛΗΨΗ

Η ολίσθηση της άνω μηριαίας επίφυσης είναι η κύρια αιτία χωλότητας στην εφηβική ηλικία. Η παχυσαρκία και οι διάφορες ενδοκρινοπάθειες προδιαθέτουν συχνά σε αμφίπλευρες προσβολές. Η καθυστέρηση, ή η αστοχία της διάγνωσης, προκαλεί συνήθως επιδείνωση της ολίσθησης, προδικάζοντας φτωχά μακροπρόθεσμα αποτελέσματα μετά την εφαρμογή της θεραπείας. Η συγκράτηση της επίφυσης *in situ* είναι η παγκοσμίως αποδεκτή μέθοδος, αν και δεν προλαμβάνει το πάσχον ισχίο να εμφανίσει πρώιμη αρθροπάθεια, συχνότερα απ' ό,τι συμβαίνει στο γενικό πληθυσμό. Η κοτυλομηριαία πρόσκρουση που οφείλεται στην παραμόρφωση του μηριαίου αυχένα, αντιπροσωπεύει ενδιάμεση παθολογική κατάσταση, καθώς οδηγεί σταδιακά σε βλάβες του επιχείλιου χόνδρου και του αρθρικού χόνδρου της κοτύλης, με αναπόφευκτη συνέπεια την εκδήλωση πρώιμης αρθροπάθειας του ισχίου. Οι περισσότερες ολισθήσεις, μέτριας ή μεγάλης βαρύτητας, θα εμφανίσουν κοτυλομηριαία πρόσκρουση. Η βαρύτητα της ολίσθησης είναι ευθέως ανάλογη με το μέγεθος της βλάβης των επιχείλιου και αρθρικού χόνδρων της κοτύλης. Στις μέτριας βαρύτητας και στις σοβαρές ολισθήσεις, η προσδόκιμη αύξηση και η δυναμική του ανασχηματισμού μετά την *in situ* συγκράτηση αδυνατούν να προλάβουν την παραμόρφωση του μηριαίου αυχένα. Σε αυτές τις περιπτώσεις, μπορεί να φανούν χρήσιμες η αρθροσκοπική οστεοχονδροπλαστική του αυχένα ή μία τροποποιημένη επέμβαση κατά Dunn, προκειμένου να μειωθεί ο κίνδυνος πρώιμης εμφάνισης αρθροπάθειας του ισχίου.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: ολίσθηση άνω μηριαίας επίφυσης, κοτυλομηριαία πρόσκρουση, πρώιμη οστεοαρθρίτιδα, ισχίο