

# Slipped capital femoral epiphysis

## Early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis

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**ABSTRACT** – On the basis of intraoperative observations in 13 consecutive adolescents (14 hips) with slipped capital femoral epiphysis (SCFE), we found that when the anterior femoral metaphysis was level with or extended past the epiphysis, it caused labrum and cartilage damage. As a result of an impingement between the metaphysis and the superomedial acetabular rim, the labrum revealed erosions, scars or tears. Further jamming of the metaphysis into the joint damaged the adjacent acetabular cartilage, varying from a partial- to a full-thickness cartilage loss. In all patients, the femoral head cartilage was intact; no avascular necrosis was present. Our findings suggest that arthrosis in SCFE can be triggered by early mechanical damage of the acetabular cartilage. ■

Sequelae of slipped capital femoral epiphysis (SCFE) include chondrolysis (Waldenström 1930), avascular necrosis (AVN) (Herman et al. 1996) and compromised load-distribution function of a malpositioned femoral head (Dunn 1964), leading to an overall arthrosis rate in 25–41% of patients (Imhäuser 1957, Murray 1965, Southwick 1967, Kaufmann 1968, Engelhardt 1984, Schai et al. 1996). Recent theoretical considerations have suggested anterior femoro-acetabular impingement as a potential cause of failure of the SCFE hip (Rab 1999). We report operative findings which support this hypothesis.

### Patients and methods

Since April 1996, 2 male and 11 female adolescents, ranging between 11 and 15 years of age, were referred to our department with untreated SCFE (13 hips) or after in situ screw fixation for SCFE performed recently elsewhere (1 hip). The patients had complained of groin and/or knee pain ranging between 1 week and more than 2 years, which had been exacerbated by vigorous activity such as skiing or playing volleyball. Clinical assessment revealed a limited range of flexion and internal rotation; all showed forced external rotation with increasing flexion of the hip joint. The diagnosis was confirmed by radiographs (Figure 1; Imhäuser 1957). 1 patient had a symptomatic SCFE of the contralateral side. No patient had acute SCFE, 6 early chronic SCFE, 3 acute-on-chronic SCFE and 5 chronic SCFE, classified according to Dunn and Angel (1978). The severity of SCFE was mild ( $< 30^\circ$ ) in 3, moderate ( $30^\circ - 60^\circ$ ) in 10 and severe ( $> 60^\circ$ ) in 1 hip, when graded on the basis of their posterior slip-angle, measured from lateral radiographs (Ballmer et al. 1990).

In all patients, surgery was performed in a lateral position, using a straight lateral or a Kocher-Langenbeck-type approach, routinely combined with an osteotomy of the greater trochanter, not extending posteriorly into the trochanteric fossa so as to protect the nutrient blood vessels (Gautier et al. 2000). To approach the capsule, the gluteus

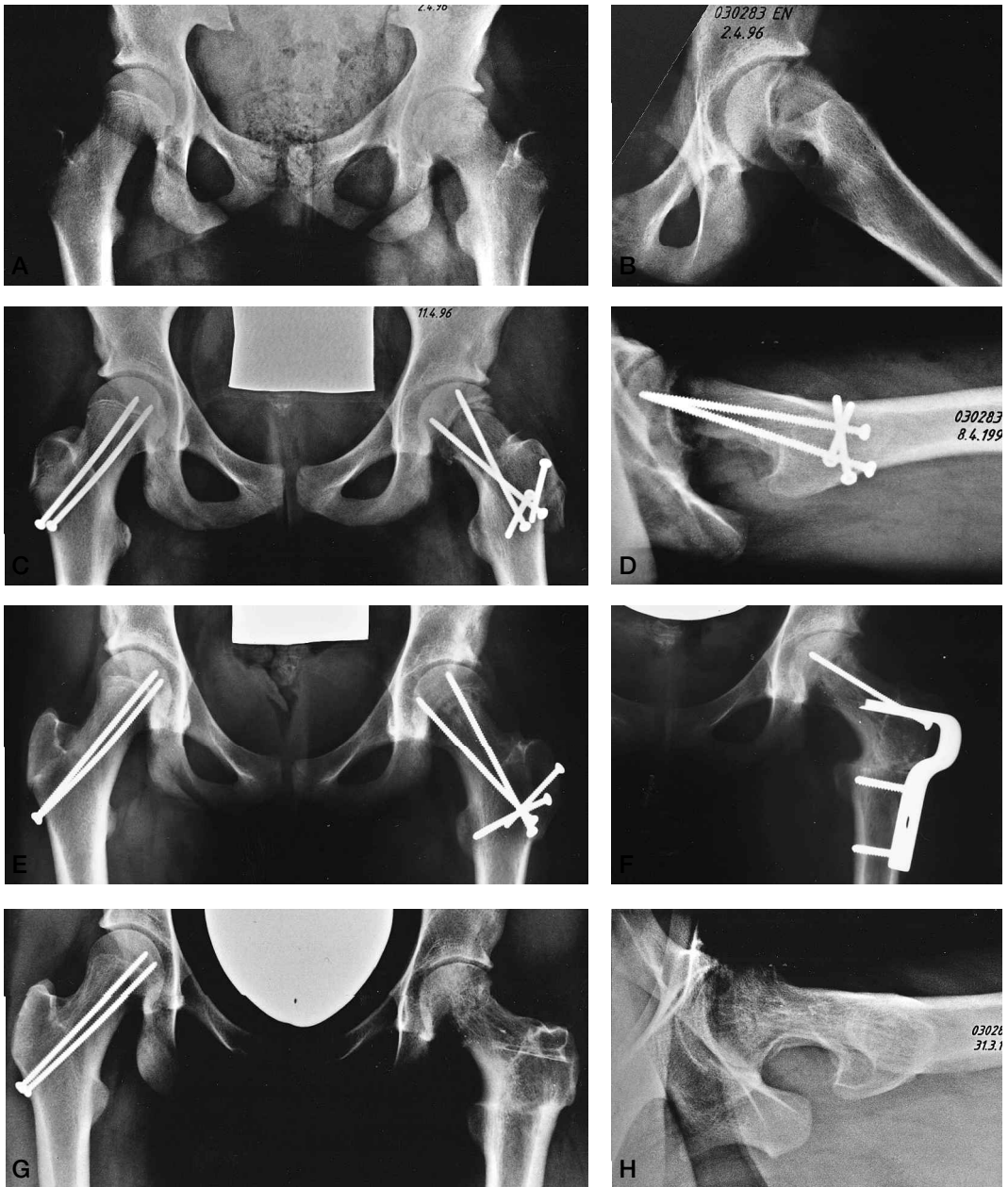


Figure 1. (A) Anteroposterior and (B) lateral radiographs 6 months after the initial development of pain in the left hip. An acute-on-chronic SCFE was present with a posterior slip of  $60^\circ$ . Postoperative (C) anteroposterior and (D) lateral radiographs after a subcapital osteotomy and callus resection. The joint remained non-concentric (even after institution of spica treatment in slight abduction and flexion) (E). At 4 months, a varus-extension intertrochanteric osteotomy was performed. Anteroposterior (G) and lateral radiographs (H) 3 years after surgery show an almost normal joint space, but signs of prearthrosis.

minimus was mobilized through the gap with the piriformis muscle. The capsulotomy was z-shaped (right side) with the anterior extension along the femoral capsular insertion and the superior and

posterior branch along the acetabular insertion. The joint was inspected by sublaxating or luxating of the femoral head. Before performing any further manipulation, a 2-mm drill hole was placed in

Preoperative morphological findings in 13 patients (14 hips)

| Severity of SCFE | Posterior slip angle | No. of hips | Damage             |                   |                      |
|------------------|----------------------|-------------|--------------------|-------------------|----------------------|
|                  |                      |             | Femoral metaphysis | Acetabular labrum | Acetabular cartilage |
| Mild             | < 30°                | 3           | 3                  | 2                 | 3                    |
| Moderate         | 30°–60°              | 10          | 10                 | 10                | 8                    |
| Severe           | > 60°                | 1           | 1                  | 1                 | 1                    |
| Total            |                      | 14          | 14                 | 13                | 12                   |

the epiphysis, where bleeding indicated viability of the femoral head (Gill et al. 1998).

Depending on the type and severity of SCFE, in situ screw fixation plus the creation of a femoral neck offset (trimming of the prominent anterolateral femoral neck metaphysis) (Poland 1898), an intertrochanteric flexion osteotomy (Imhäuser 1957) plus the creation of a femoral neck offset or a subcapital osteotomy and callus resection (Dunn 1964) were performed. In all hips, the acetabular rim and the joint cartilage were assessed during surgery. Due to the 40%–80% risk of a SCFE on the contralateral side (Hägglund 1996), the unaffected hip was prophylactically treated by in situ screw fixation when the physeal plate was still open.

## Results

The first patient in this series was a 12-year-old white girl with an acute-on-chronic SCFE. She presented with hip pain for months, mostly caused by playing volleyball. An anteroposterior radiograph revealed mild chronic SCFE with a concentric hip. 6 months later, the girl had sudden onset of severe groin pain after skiing. On physical examination, the left hip was extremely painful and showed a concomitant external rotation with flexion. Repeated radiographs revealed progression of the SCFE to a posterior slip of 60° (Figure 1). A diagnosis of acute-on-chronic SCFE was made and, at surgery on the same day, the femoral head was unstable. The metaphysis was trimmed and the posterior callus resected. The remaining growth plate of the epiphysis was removed. The epiphysis was anatomically reduced and fixed

with 2 screws. Bleeding from drill holes before and after this procedure indicated that the femoral head was viable.

The postoperative radiographs showed a correct position of the head on the neck, a narrowed superior-lateral joint space (1.0 mm), and an increased distance of 2 mm between the teardrop and the femoral head. Since lateral migration disappeared with slight abduction and flexion, this position was maintained with a plaster spica for 5 weeks. Lateral migration of the head and narrowing of the joint space (1.5 mm) recurred after removal of the spica and the girl reported discomfort and walked with a limp. Physical examination revealed decreased abduction. Anteroposterior radiographs at 3.5 months showed a new subchondral bone reaction at the superior acetabular rim and that the hip was still not concentric with persistent narrowing of the joint space. Because of the radiographic abnormalities and the unsatisfactory clinical course, the hip was revised 4 months after index surgery. During surgery, using the same approach, the hip joint was inspected. The femoral head was vital and correctly positioned on the femoral neck; the cartilage was intact. Subluxation of the femoral head showed a 1.5 cm × 3.0 cm, full-thickness cartilage defect in the superomedial acetabular region. The cartilage of the remaining acetabulum was intact and appeared healthy. The defect was covered by a thin organizing membrane, the labrum was scarred. To recenter the femoral head and reduce the load-transfer in the affected zone, a varus-extension (15°/15°) intertrochanteric femoral osteotomy was performed (Müller 1979). After 3 years, the hip shows an acceptable radiographic result with grade 1 arthrosis, according to the classification of Tönnis (Tönnis 1978), and a good

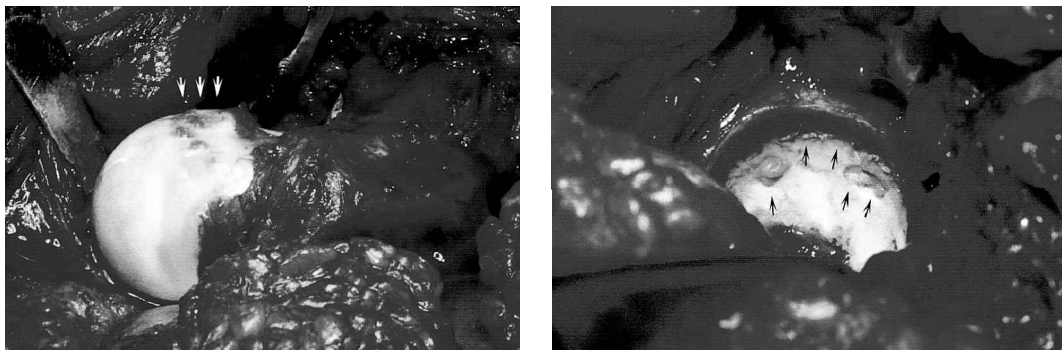


Figure 2. Intraoperative photographs of a moderate SCFE. Note (left) rough edges (arrows) at the prominent femoral metaphysis which caused (right) severe cartilage damage at the superomedial aspect of the acetabulum (arrows).

clinical result without pain or restricted motion.

In the 12 patients treated thereafter, similar pathomorphologies were present (Table and Figure 2). Cartilage abrasion was observed in hips, which had been symptomatic for a few weeks only. In all patients, the metaphysis was level or extended more anteriorly than the femoral head, revealing rough edges and even bleeding ulcerations of the metaphyseal surface in 7 hips. Independently of the degree of SCFE, flexion of the hip joint and more pronounced additional internal rotation caused impingement of this metaphyseal area against the superomedial acetabulum and labrum. In mild-to-moderate SCFE during further flexion, the rough metaphysis even entered the acetabulum, after displacing the labrum, while in severe SCFE, the metaphyseal impingement against the acetabular rim limited further flexion. The labrum revealed deformation and marks of contusion, such as erosions (5), scars (3) or tears of the inner surface (5). The acetabular cartilage showed semilunar-shaped damage ranging from chondromalacia, cleavage of the cartilage from the subchondral bone to a full-thickness loss of cartilage. The extension of the cartilage damage from the acetabular rim into the joint ranged from 1.0 cm to 1.5 cm and the length was up to 3 cm.

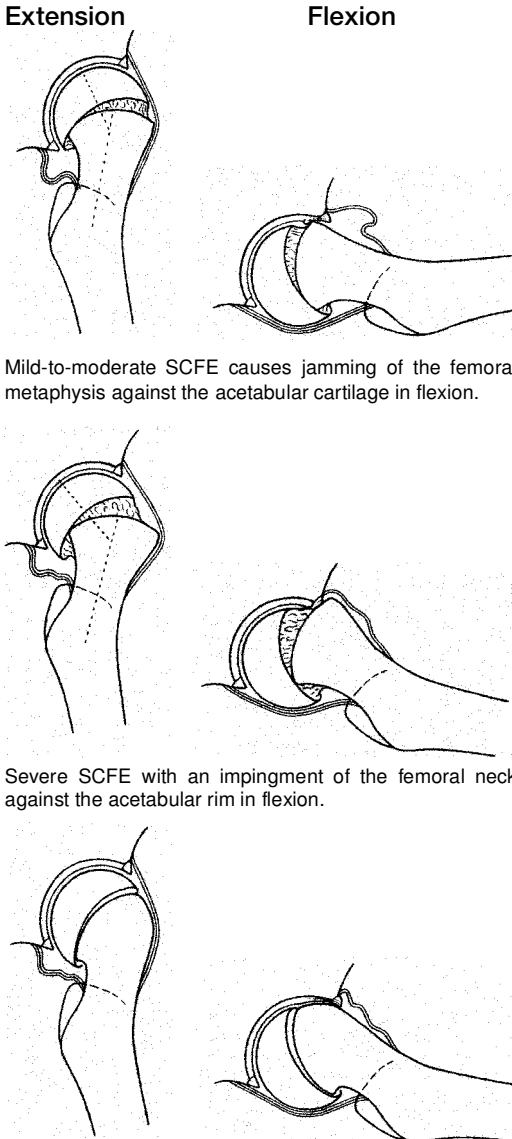
## Discussion

On the basis of our findings, we propose a new hypothesis about the etiology and pathogenesis of arthrosis in SCFE. The hip joint as a socket joint allows a predefined range of motion governed by

the joint's geometry and soft tissue components. In SCFE, the range of motion of the hip is limited for flexion (and internal rotation). This is caused by the discrepancy between the prominent femoral metaphysis and the acetabulum whereby the amount of joint damage is governed by the severity of SCFE. In milder degrees of SCFE, the well-constrained joint becomes jammed, causing direct abrasion at the labrum and adjacent cartilage by the metaphysis during flexion, a common causative factor (Figure 3). This effect is increased by the rough surface of the anterior metaphyseal edge (Figure 2). In moderate SCFE, the damage to the acetabulum increases, if the step-off of the metaphysis is still squeezed into the acetabulum during flexion. In severe SCFE, the even more prominent metaphysis extends beyond the epiphysis and impinges against the acetabular rim, but it may be unable to enter the joint. Therefore, pathologic loading may develop at the labrum (Figure 3). The presence of intraarticular damage in severe SCFE may be due to the fact that these hips have passed through a period where the metaphysis was able to enter and damage the acetabulum. In chronic SCFE with flattening and rounding of the remodeled metaphysis, jamming of this area against the acetabular cartilage is likely to occur again (Figure 3).

Mechanical jamming seems to be the main factor causing direct and early mechanical acetabular rim and cartilage damage, triggering arthrosis in SCFE. Intraarticular damage ranged from partial-to-full thickness cartilage defects that did not penetrate the subchondral bone. Cartilage damage with no involvement of subchondral bone, blood

Figure 3. Schematic lateral drawings of varying types and degrees of SCFE in extension and flexion.



Mild-to-moderate SCFE causes jamming of the femoral metaphysis against the acetabular cartilage in flexion.

Severe SCFE with an impingement of the femoral neck against the acetabular rim in flexion.

Severe SCFE after remodeling showing reappearance of jamming in the constrained hip joint.

vessels and bone marrow is incapable of healing (Landells 1957, Kim et al. 1991). Although long-term follow-ups are not yet available and only a few patients have been included, we suggest that the observed cartilage damage is irreversible. This view is supported by the finding that even asymptomatic slight degrees of SCFE have been shown to represent a major risk factor predis-

ing to arthrosis (Goodman et al. 1997). Secondary mechanical phenomena such as a compromised load-distribution but also biological phenomena such as synovitis and compromised chondrocyte nutrition may contribute to the development of arthrosis.

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