

Slipped Capital Femoral Epiphysis: Prevalence, Pathogenesis, and Natural History

Eduardo N. Novais MD, Michael B. Millis MD

Published online: 9 October 2012
© The Association of Bone and Joint Surgeons® 2012

Abstract

Background Obesity is a risk factor for developing slipped capital femoral epiphysis (SCFE). The long-term outcome after SCFE treatment depends on the severity of residual hip deformity and the occurrence of complications, mainly avascular necrosis (AVN). Femoroacetabular impingement (FAI) is associated with SCFE-related deformity and dysfunction in both short and long term.

Questions/Purposes We examined obesity prevention, early diagnosis, reducing AVN and hip deformity as strategies to reduce SCFE prevalence, and the long-term outcomes after treatment.

Methods A search of the literature using the PubMed database for the key concepts SCFE and treatment, natural history, obesity, and prevalence identified 218, 15, 26, and 49 abstracts, respectively.

Where Are We Now? A correlation between rising childhood obesity and increasing incidence of SCFE has been recently reported. Residual abnormal morphology of

the proximal femur is currently believed to be the mechanical cause of FAI and early articular cartilage damage in SCFE.

Where Do We Need to Go? Reducing the increasing prevalence rate of SCFE is important. Treatment of SCFE should aim to reduce AVN rates and residual deformities that lead to FAI to improve the long-term functional and clinical outcomes.

How Do We Get There? Implementing public health policies to reduce childhood obesity should allow for SCFE prevalence to drop. Clinical trials will evaluate whether restoring the femoral head-neck offset to avoid FAI along with SCFE fixation allows for cartilage damage prevention and lower rates of osteoarthritis. The recently described surgical hip dislocation approach is a promising technique that allows anatomic reduction with potential lower AVN rates in the treatment of SCFE.

Introduction

Slipped capital femoral epiphysis (SCFE) is a disorder of the immature hip in which anatomic disruption occurs through the proximal femoral physis. SCFE is associated with a highly variable degree of posterior translation of the epiphysis and simultaneous anterior displacement of the metaphysis. In SCFE, there is a spectrum of each of the following elements: temporal acuity [18]; physical stability of the slipping physis [50]; degree of displacement between the proximal femoral neck and the epiphysis [10, 78]; and the amount of deformity that the protruding anterior metaphyseal prominence presents to the anterior acetabular rim with hip flexion [69].

Prevalence of SCFE varies widely among ethnic groups [46], geographic locations, and different seasons [47, 48]. Pathogenesis includes factors that either reduce the

Each author certifies that he or she, or a member of their immediate family, has no commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc) that might pose a conflict of interest in connection with the submitted article

All ICMJE Conflict of Interest Forms for authors and *Clinical Orthopaedics and Related Research* editors and board members are on file with the publication and can be viewed on request.

E. N. Novais
Department of Orthopaedic Surgery, Children's
Hospital of Colorado, Aurora, CO, USA

M. B. Millis (✉)
Department of Orthopaedic Surgery, Children's Hospital,
300 Longwood Avenue, Boston, MA 02115, USA
e-mail: michael.millis@tch.harvard.edu;
michael.millis@childrens.harvard.edu

resistance to shear or that increase the stresses across the proximal femoral physis [20, 45, 68]; for example, endocrine disorders [52, 83], obesity [9, 14, 34, 61, 63, 67, 71, 82], femoral [28] or acetabular retroversion [17], and coxa profunda [72]. The natural history is largely dependent on both the degree of deformity [11] and the occurrence of complications of treatment, mainly avascular necrosis of the femoral head (AVN) and chondrolysis [37, 39, 50, 53]. Most long-term studies demonstrate some loss of function over time with all degrees of deformity [16, 31, 62, 74, 78]. Contemporary analysis suggests femoroacetabular impingement (FAI) as the major pathomechanical element in SCFE-related dysfunction in both short and long term [21, 22, 41, 42, 58, 69].

In this article, we review the current knowledge on SCFE prevalence, pathophysiology, and natural history (“Where are we now?”). We explore the increase in the prevalence of SCFE over the last decades and its direct pathophysiological association with the childhood obesity pandemic. We further discuss the goals of reducing the occurrence of SCFE and improving its long-term results (“Where do we need to go?”). Finally, we examine obesity prevention, early diagnosis, reducing the rate of AVN, articular cartilage damage, and hip deformity as potential strategies to achieve the aforementioned goals (“How do we get there?”).

Search Strategies and Criteria

We searched the PubMed database with terms including “SCFE” combined with “treatment”, “natural history”, “obesity” and “prevalence”. A total of 218, 15, 26, and 49 abstracts were respectively identified for each search. After review of the abstracts by one of the authors (EN), we included peer-reviewed studies in English. For the treatment and natural history search we included studies with a minimum of 2 years followup and radiographic and clinical outcomes. We excluded abstracts of case reports and if they did not specifically investigate one of the searched terms. References of included articles were further searched for additional studies. A total of 80 articles were included in this analysis.

Where Are We Now?

Slipped capital femoral epiphysis is the most common hip disorder affecting adolescents. The overall prevalence varies from 0.71 to 10.8 per 100,000 children [33, 40, 46]. It is known to affect boys more often than girls with a male-to-female ratio of approximately 1.5 [40, 46], although unstable slips seem to be at least as common in females as

in males. The prevalence of bilateral SCFE has been reported from 20% to 80% and in bilateral cases, the second SCFE usually occurs during the first year after the first slip [49]. There is a racial variability with a higher prevalence rate in blacks, Hispanics, Polynesians, and Native Americans when compared with whites [8, 40, 46]. There is also seasonal and geographic variability with higher rates in the north and western parts of the United States [40]. The age of onset of SCFE is approximately 12.7 to 13.5 years for boys and 11.2 to 12 years for girls [40, 46]. There is a trend toward onset of SCFE at a younger age compared with previous reports [30, 40, 61]. It has been postulated that this phenomenon correlates with earlier children maturation [64]. Of concern is the fact that the incidence of SCFE has been rising over the past decades [8, 61, 63, 77].

Although different theories [12, 15, 70] have been proposed in the past, the pathogenesis of SCFE remains unclear. Ultrastructural analysis shows slippage of the physis occurs secondary to weakness of the supporting fibrous network caused by collagen disturbance [2, 19, 57]. Mechanical [4, 20, 45, 68], endocrine [52, 85, 86], and metabolic disorders of puberty [60] have been postulated to cause the pathological disturbance in the growth plate that ultimately fail mechanically and slip. Despite previous reports on the association of human leukocyte antigen in identical twins with SCFE [3], a genetic basis has not been established.

The pathogenesis of SCFE is most likely multifactorial. However, mechanical factors, mainly obesity and abnormal morphology of the proximal femur and acetabulum, seem to play a determinant role [85].

The high prevalence of obesity among patients with SCFE is widely recognized [9, 14, 34, 61, 63, 67, 71, 82]. More than 80% of the children diagnosed with SCFE are reportedly obese (body mass index greater than the 95th percentile) [55]. Obesity may increase the risk of SCFE as a result of both higher mechanical loads across the femoral physis and a metabolic disorder. Obese children have decreased femoral anteversion [24] and a more vertical-oriented proximal femoral physis [59]. Increased prevalence of childhood obesity is a worrisome phenomenon. Obesity should be seen as a chronic disease with deleterious health consequences that can potentially constitute a metabolic disorder. Previous reports in the United States have shown the prevalence of obesity has doubled in the past two decades [38]. Three recent reports on American [8], Scottish [61, 63], and Australian children [63] have revealed a close correlation between rising childhood obesity and an increase in the incidence of SCFE worldwide.

Certain abnormal morphologies of the proximal femur have been associated with SCFE. Reduced femoral

anteversion and absolute femoral retroversion may predispose the proximal femoral physis to slip, particularly if the AP shear forces are increased secondary to abnormally high patient weight [28, 81]. The increased obliquity of the proximal femoral physis is reportedly a mechanical factor in the development of SCFE [59, 68]. In the past, axial (version) orientation of the acetabulum was reportedly not found to play a role in the pathogenesis of SCFE [80]. However, recently both acetabular retroversion and increased coverage of the femoral head (coxa profunda) were described in the involved and the uninvolved contralateral hip in patients with SCFE [35, 72].

Traditionally, the natural history of SCFE has been directly related to the degree of the slip and the complications of treatment. The main predictive factors for degree of stable SCFE are age at diagnosis and symptom duration [36, 51]. The most worrisome complication of treatment is AVN of the femoral head, which is strongly associated with SCFE instability [50, 65, 66, 84]. AVN is recognized as a risk factor for early development of severe osteoarthritis of the hip [6, 37, 39]. In the past, two classic studies defined the role of in situ fixation in the treatment of SCFE as a safe and reliable method [10, 11]. The Iowa hip rating score after in situ fixation of moderate and severe chronic SCFE was reportedly 85 out of a possible 100 points at an average of 41 years of followup [11]. However, residual deformities associated with mild SCFE have been reported to play a role in development of hip osteoarthritis (OA) [83]. In addition, an investigation in cadaveric human femora reported approximately 70% incidence of a severe degree of OA in hips with minimum postslip morphology [29].

Recent studies investigating clinical and radiographic evidence of FAI during the first decade after SCFE treatment revealed a persistent femoral deformity in patients undergoing in situ fixation [13, 21]. Dodds et al. [13] reported pain in 31% of 49 patients with a mean followup of 6.1 years after in situ pinning. Fraitzl et al. [21] reported on 16 patients who underwent in situ pinning for mild SCFE with an average of 14 years of followup. None of the 16 patients had normal proximal femoral morphology assessed by the head-neck offset ratio, although only six of 16 patients had a positive impingement provocation test. The authors however reported a lower level of physical activity in the patients studied. They hypothesized that remodeling of the head-neck junction and following a less physically demanding lifestyle may have allowed the patients to remain asymptomatic.

In the past, a high (75%) probability of remodeling of the femoral head-neck junction an average of 7.1 years after in situ pinning of SCFE has been reported [32]. The remodeling process has been associated with 39% excellent and 50% good results assessed by the Heyman and

Herndon criteria in a series of 44 patients followed for an average of 11.4 years [7]. Current investigations however have challenged remodeling as a benign process [42, 54, 69]. Using a three-dimensional modeling study, Rab [69] described two types of mechanical conflict between the femoral metaphysis and the acetabulum in the production of abnormal motion after SCFE. Impaction occurs when the proximal femoral metaphysis comes in contact with the acetabular rim, which limits the ROM of the hip, resulting in damage to the anterior part of the acetabular labrum. Inclusion impingement occurs when the remodeled proximal femoral metaphysis enters the acetabulum, which can lead to articular cartilage damage. More recently, Ganz et al. [27] described the pathomechanics of FAI. FAI is a dynamic phenomenon in which there is abnormal contact between the proximal femoral head-neck junction and the acetabular rim. FAI has been associated with the development of OA of the hip [27]. The abnormal morphology of the proximal femur is currently believed to be the mechanical cause of FAI and early articular cartilage damage in SCFE [41, 42, 54, 69, 75].

Different investigators have (recently) reported on early acetabular cartilage damage even after mild SCFE [23, 41, 75]. Labral tear and anterosuperior acetabular cartilage damage was reported on hip arthroscopy in four patients with acute to chronic hip pain with SCFE [23]. Leunig and colleagues [41] reported on early mechanical damage to the acetabular cartilage by the prominent metaphysis in mild, moderate, and severe SCFE. The authors postulated that mechanical jamming was the main factor causing direct and early mechanical acetabular rim and cartilage damage that may lead to hip OA [41]. In a retrospective study, Sink et al. [75] reported acetabular cartilage injury in 33 and labral injury in 34 of 39 hips at the time of surgical dislocation of the hip for the treatment of symptomatic stable SCFE.

Where Do We Need to Go?

The increasing incidence of SCFE over the past few decades [8, 61, 63, 77] suggests interventions should be considered both to reduce SCFE prevalence and to promote early diagnosis and treatment. There is an increasing need for community awareness of the symptoms associated with SCFE, thigh and groin pain, limp, and referred knee pain to allow early diagnosis and implement treatment because delayed diagnosis is associated with increased slip severity that ultimately leads to poorer long-term outcome. The correct radiographic technique is important to identify the slip and to avoid abrupt maneuvers that potentially could cause further displacement of the epiphysis.

The correlation between childhood and adolescent SCFE and hip OA needs to be explored further. Treatment

complications, mainly AVN, have a direct impact on the outcome of disease. Therefore, there is a need to develop a treatment strategy that takes into consideration lower risks of both residual hip deformity and AVN. Ideally this would imply two lines of action: (1) improve the understanding of cartilage damage and implement surgical techniques that would avoid early articular cartilage injury in cases of stable SCFE; and (2) improve the knowledge about the pathogenesis of damage to the femoral head blood supply and implementing safe surgical techniques with low risk of further damaging the blood supply in cases of unstable SCFE.

How Do We Get There?

The goal of reducing SCFE incidence might be achieved with interventions on both patient-level and population-level bases. Once SCFE is diagnosed in one hip, prophylactic treatment of the contralateral hip should be considered strongly because of the high prevalence of bilateral SCFE. Although prophylactic pinning of the contralateral hip is still controversial [18, 36, 49], young age at diagnosis, unstable SCFE, endocrine disorders, and unreliable patient followup are relative indications for prophylactic treatment [49]. The increasing incidence of SCFE and childhood obesity [8, 61, 63] reinforces the necessity for promoting population-level health policies to support childhood obesity prevention [56]. Further studies will be required to determine whether reducing the incidence of obesity would have a positive impact on SCFE incidence.

The current recognition of frequent early articular cartilage damage, even in mild SCFE [23, 41, 73], needs to be considered when establishing goals and a strategy of treatment. Traditionally the goal of primary treatment of SCFE has been to stabilize the epiphysis and prevent additional displacement and complications (AVN), thereby restoring reasonable function and delaying or preventing OA [11]. The widely accepted in situ fixation might not be adequate to accomplish these goals [58]. In many hips, in situ fixation serves the purpose of stabilizing the slip and at best allows early symptomatic pain relief. In situ fixation in any but the mildest of slips requires metaphyseal remodeling to allow near-normal ROM of the hip [54, 69]. We now understand that proximal femur remodeling is not necessarily a benign process. Currently, remodeling is believed to be a major factor in articular cartilage damage that occurs as the remodeling metaphysis enters the acetabulum [42, 54, 69]. However, further investigations are essential to identify patients at risk to develop symptomatic FAI after SCFE and to determine whether the abnormal morphology after in situ pinning requires treatment.

Concomitant treatment of the slip by screw fixation across the physis and of the abnormal FAI-predisposing morphology of the proximal femur, by a femoral head-neck osteochondroplasty, may be the appropriate treatment for mild SCFE [43]. A prospective randomized clinical trial will be necessary to establish the efficacy of this approach compared with in situ pinning alone to avoid cartilage damage, pain, and development of hip OA after mild SCFE. In moderate to severe SCFE, intertrochanteric osteotomies (ITO) have been indicated to realign the proximal femur [1, 5, 31, 74, 79]. In a previous study, ITO allowed for 90% excellent or good results according to the Southwick criteria after an average of 9 years (range, 2–24 years) [1]. In another series with an average of 24 years of follow-up (range, 20–29 years), 55% of the patients showed no radiographic evidence of OA or clinical pain after an ITO for severe SCFE [74]. However, the role of proximal femur osteotomies to correct healed SCFE deformity and avoid FAI-related cartilage damage and development of OA should also be determined by further studies.

In a recent systematic review of the literature, instability independently predicted AVN: patients with unstable slips had a 9.4-fold greater risk of developing AVN [84]. The rates of AVN of the femoral head after unstable SCFE treatment vary from 5% to 47% [50, 65, 66, 82]. Open reduction seems to play a role in reducing the rates of AVN in unstable SCFE [65, 76, 87]. The recently described surgical hip dislocation (SHD) approach [25] and development of a retinacular flap [26] that protects the blood supply to the femoral head has allowed treatment of unstable SCFE with lower rates of AVN [44, 73, 76, 87]. The SHD approach offers the advantage of providing an anatomic reduction while preserving the soft tissue retinaculum containing the deep branch of the medial circumflex artery, the most important source of blood supply to the femoral head. SHD and subcapital realignment have been reported to have a low complication rate in moderate to severe stable SCFE with wide open physes [76, 87]. However, it is in the unstable SCFE that the SHD approach may theoretically decrease the complication of AVN [87]. Further prospective comparison studies are necessary to determine the role of this cutting-edge technique.

In summary, SCFE is an increasingly common adolescent hip problem with frequent lifelong sequelae. Many opportunities exist for improved understanding of factors determining outcomes and for better decision-making in multiple subgroups. Contemporary treatment strategies that allow anatomic reduction are promising. However, they are technically demanding and should be proven to be safe (low rates of AVN) to become widely implemented. The use of contemporary analytic methods, study designs, and

outcome measures will be essential to determine the role of different surgical strategies in preserving the native hip and avoiding development of hip arthritis requiring future joint arthroplasty.

References

- Abraham E, Garst J, Barmada R. Treatment of moderate to severe slipped capital femoral epiphysis with extracapsular base-of-neck osteotomy. *J Pediatr Orthop.* 1993;13:294–302.
- Agamanolis DP, Weiner DS, Lloyd JK. Slipped capital femoral epiphysis: a pathological study. I. A light microscopic and histochemical study of 21 cases. *J Pediatr Orthop.* 1985;5:40–46.
- Allen CP, Calvert PT. Simultaneous slipped upper femoral epiphysis in identical twins. *J Bone Joint Surg Br.* 1990;72:928–929.
- Aronson J, Tursky EA. The torsional basis for slipped capital femoral epiphysis. *Clin Orthop Relat Res.* 1996;322:37–42.
- Barmada R, Bruch RF, Gimbel JS, Ray RD. Base of the neck extracapsular osteotomy for correction of deformity in slipped capital femoral epiphysis. *Clin Orthop Relat Res.* 1978;132:98–101.
- Bartonicek J, Vavra J, Bartoska R. Operative treatment of avascular necrosis of the femoral head after slipped capital femoral epiphysis. *Arch Orthop Trauma Surg.* 2011;131:497–502.
- Bellemans J, Fabry G, Molenaers G, Lammens J, Moens P. Slipped capital femoral epiphysis: a long-term follow-up, with special emphasis on the capacities for remodeling. *J Pediatr Orthop B.* 1996;5:151–157.
- Benson EC, Miller M, Bosch P, Szalay EA. A new look at the incidence of slipped capital femoral epiphysis in new Mexico. *J Pediatr Orthop.* 2008;28:529–533.
- Bhatia NN, Pirpiris M, Otsuka NY. Body mass index in patients with slipped capital femoral epiphysis. *J Pediatr Orthop.* 2006;26:197–199.
- Boyer DW, Mickelson MR, Ponseti IV. Slipped capital femoral epiphysis. Long-term follow-up study of one hundred and twenty-one patients. *J Bone Joint Surg Am.* 1981;63:85–95.
- Carney BT, Weinstein SL, Noble J. Long-term follow-up of slipped capital femoral epiphysis. *J Bone Joint Surg Am.* 1991;73:667–674.
- Chung SM, Batterman SC, Brighton CT. Shear strength of the human femoral capital epiphyseal plate. *J Bone Joint Surg Am.* 1976;58:94–103.
- Dodds MK, McCormack D, Mulhall KJ. Femoroacetabular impingement after slipped capital femoral epiphysis: does slip severity predict clinical symptoms? *J Pediatr Orthop.* 2009;29:535–539.
- Dunbar J, Goulding A. Slipped capital femoral epiphysis: more New Zealand cases likely as obesity rises in children and adolescents? *N Z Med J.* 2001;114:559–560.
- Eisenstein A, Rothschild S. Biochemical abnormalities in patients with slipped capital femoral epiphysis and chondrolysis. *J Bone Joint Surg Am.* 1976;58:459–467.
- Engelhardt P. [Natural course of epiphysiolysis of the femur head] [in German]. *Orthopade.* 1994;23:195–199.
- Ezoe M, Naito M, Inoue T. The prevalence of acetabular retroversion among various disorders of the hip. *J Bone Joint Surg Am.* 2006;88:372–379.
- Fahey JJ, O'Brien ET. Acute slipped capital femoral epiphysis: review of the literature and report of ten cases. *J Bone Joint Surg Am.* 1965;47:1105–1127.
- Falciglia F, Aulisa AG, Giordano M, Boldrini R, Guzzanti V. Slipped capital femoral epiphysis: an ultrastructural study before and after osteosynthesis. *Acta Orthop.* 2010;81:331–336.
- Fishkin Z, Armstrong DG, Shah H, Patra A, Mihalko WM. Proximal femoral physis shear in slipped capital femoral epiphysis—a finite element study. *J Pediatr Orthop.* 2006;26:291–294.
- Fraitzl CR, Kafer W, Nelitz M, Reichel H. Radiological evidence of femoroacetabular impingement in mild slipped capital femoral epiphysis: a mean follow-up of 14.4 years after pinning in situ. *J Bone Joint Surg Br.* 2007;89:1592–1596.
- Fraitzl CR, Nelitz M, Cakir B, Kafer W, Reichel H. [Transfixation in slipped capital femoral epiphysis: long-term evidence for femoro-acetabular impingement] [in German]. *Z Orthop Unfall.* 2009;147:334–340.
- Futami T, Kasahara Y, Suzuki S, Seto Y, Ushikubo S. Arthroscopy for slipped capital femoral epiphysis. *J Pediatr Orthop.* 1992;12:592–597.
- Galbraith RT, Gelberman RH, Hajek PC, Baker LA, Sartoris DJ, Rab GT, Cohen MS, Griffin PP. Obesity and decreased femoral anteversion in adolescence. *J Orthop Res.* 1987;5:523–528.
- Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. 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;83:1119–1124.
- Ganz R, Huff TW, Leunig M. Extended retinacular soft-tissue flap for intra-articular hip surgery: surgical technique, indications, and results of application. *Instr Course Lect.* 2009;58:241–255.
- Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Orthop Relat Res.* 2003;417:112–120.
- Gelberman RH, Cohen MS, Shaw BA, Kasser JR, Griffin PP, Wilkinson RH. The association of femoral retroversion with slipped capital femoral epiphysis. *J Bone Joint Surg Am.* 1986;68:1000–1007.
- Goodman DA, Feighan JE, Smith AD, Latimer B, Buly RL, Cooperman DR. Subclinical slipped capital femoral epiphysis. Relationship to osteoarthritis of the hip. *J Bone Joint Surg Am.* 1997;79:1489–1497.
- Hagglund G, Hansson LI, Ordeberg G. Epidemiology of slipped capital femoral epiphysis in southern Sweden. *Clin Orthop Relat Res.* 1984;191:82–94.
- Imhauser G. [Late results of Imhauser's osteotomy for slipped capital femoral epiphysis (author's transl)] [in German]. *Z Orthop Ihre Grenzgeb.* 1977;115:716–725.
- Jones JR, Paterson DC, Hillier TM, Foster BK. Remodelling after pinning for slipped capital femoral epiphysis. *J Bone Joint Surg Br.* 1990;72:568–573.
- Kelsey JL. Epidemiology of slipped capital femoral epiphysis: a review of the literature. *Pediatrics.* 1973;51:1042–1050.
- Kelsey JL, Acheson RM, Keggi KJ. The body build of patients with slipped capital femoral epiphysis. *Am J Dis Child.* 1972;124:276–281.
- Kitadai HK, Milani C, Nery CA, Filho JL. Wiberg's center-edge angle in patients with slipped capital femoral epiphysis. *J Pediatr Orthop.* 1999;19:97–105.
- Kocher MS, Bishop JA, Weed B, Hresko MT, Millis MB, Kim YJ, Kasser JR. Delay in diagnosis of slipped capital femoral epiphysis. *Pediatrics.* 2004;113:e322–325.
- Krahn TH, Canale ST, Beaty JH, Warner WC, Lourenco P. Long-term follow-up of patients with avascular necrosis after treatment of slipped capital femoral epiphysis. *J Pediatr Orthop.* 1993;13:154–158.
- Krebs NF, Jacobson MS. Prevention of pediatric overweight and obesity. *Pediatrics.* 2003;112:424–430.
- Larson AN, McIntosh AL, Trousdale RT, Lewallen DG. Avascular necrosis most common indication for hip arthroplasty in patients with slipped capital femoral epiphysis. *J Pediatr Orthop.* 2010;30:767–773.

40. Lehmann CL, Arons RR, Loder RT, Vitale MG. The epidemiology of slipped capital femoral epiphysis: an update. *J Pediatr Orthop*. 2006;26:286–290.
41. Leunig M, Casillas MM, Hamlet M, Hersche O, Notzli H, Slongo T, Ganz R. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. *Acta Orthop Scand*. 2000;71:370–375.
42. Leunig M, Fraitzl CR, Ganz R. [Early damage to the acetabular cartilage in slipped capital femoral epiphysis. Therapeutic consequences] [in German]. *Orthopade*. 2002;31:894–899.
43. Leunig M, Horowitz K, Manner H, Ganz R. In situ pinning with arthroscopic osteoplasty for mild SCFE: a preliminary technical report. *Clin Orthop Relat Res*. 2010;468:3160–3167.
44. Leunig M, Slongo T, Kleinschmidt M, Ganz R. Subcapital correction osteotomy in slipped capital femoral epiphysis by means of surgical hip dislocation. *Oper Orthop Traumatol*. 2007;19:389–410.
45. Litchman HM, Duffy J. Slipped capital femoral epiphysis: factors affecting shear forces on the epiphyseal plate. *J Pediatr Orthop*. 1984;4:745–748.
46. Loder RT. The demographics of slipped capital femoral epiphysis. An international multicenter study. *Clin Orthop Relat Res*. 1996;322:8–27.
47. Loder RT. A worldwide study on the seasonal variation of slipped capital femoral epiphysis. *Clin Orthop Relat Res*. 1996;322:28–36.
48. Loder RT, Aronson DD, Bollinger RO. Seasonal variation of slipped capital femoral epiphysis. *J Bone Joint Surg Am*. 1990;72:378–381.
49. Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. A study of children in Michigan. *J Bone Joint Surg Am*. 1993;75:1141–1147.
50. Loder RT, Richards BS, Shapiro PS, Reznick LR, Aronson DD. Acute slipped capital femoral epiphysis: the importance of physeal stability. *J Bone Joint Surg Am*. 1993;75:1134–1140.
51. Loder RT, Starnes T, Dikos G, Aronsson DD. Demographic predictors of severity of stable slipped capital femoral epiphyses. *J Bone Joint Surg Am*. 2006;88:97–105.
52. Loder RT, Wittenberg B, DeSilva G. Slipped capital femoral epiphysis associated with endocrine disorders. *J Pediatr Orthop*. 1995;15:349–356.
53. Lubicky JP. Chondrolysis and avascular necrosis: complications of slipped capital femoral epiphysis. *J Pediatr Orthop B*. 1996;5:162–167.
54. Mamisch TC, Kim YJ, Richolt JA, Millis MB, Kordelle J. Femoral morphology due to impingement influences the range of motion in slipped capital femoral epiphysis. *Clin Orthop Relat Res*. 2009;467:692–698.
55. Manoff EM, Banffy MB, Winell JJ. Relationship between body mass index and slipped capital femoral epiphysis. *J Pediatr Orthop*. 2005;25:744–746.
56. McPherson ME, Homer CJ. Policies to support obesity prevention for children: a focus on of early childhood policies. *Pediatr Clin North Am*. 2011;58:1521–1541.
57. Mickelson MR, Ponseti IV, Cooper RR, Maynard JA. The ultrastructure of the growth plate in slipped capital femoral epiphysis. *J Bone Joint Surg Am*. 1977;59:1076–1081.
58. 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.
59. Mirkopoulos N, Weiner DS, Askew M. The evolving slope of the proximal femoral growth plate relationship to slipped capital femoral epiphysis. *J Pediatr Orthop*. 1988;8:268–273.
60. Morscher E. Strength and morphology of growth cartilage under hormonal influence of puberty. Animal experiments and clinical study on the etiology of local growth disorders during puberty. *Reconstr Surg Traumatol*. 1968;10:3–104.
61. Murray AW, Wilson NI. Changing incidence of slipped capital femoral epiphysis: a relationship with obesity? *J Bone Joint Surg Br*. 2008;90:92–94.
62. Murray RO. The aetiology of primary osteoarthritis of the hip. *Br J Radiol*. 1965;38:810–824.
63. Nguyen AR, Ling J, Gomes B, Antoniou G, Sutherland LM, Cundy PJ. Slipped capital femoral epiphysis: rising rates with obesity and aboriginality in South Australia. *J Bone Joint Surg Br*. 2011;93:1416–1423.
64. Parent AS, Teilmann G, Juul A, Skakkebaek NE, Toppari J, Bourguignon JP. The timing of normal puberty and the age limits of sexual precocity: variations around the world, secular trends, and changes after migration. *Endocr Rev*. 2003;24:668–693.
65. 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.
66. Peterson MD, Weiner DS, Green NE, Terry CL. Acute slipped capital femoral epiphysis: the value and safety of urgent manipulative reduction. *J Pediatr Orthop*. 1997;17:648–654.
67. Poussa M, Schlenzka D, Yrjonen T. Body mass index and slipped capital femoral epiphysis. *J Pediatr Orthop B*. 2003;12:369–371.
68. Pritchett JW, Perdue KD. Mechanical factors in slipped capital femoral epiphysis. *J Pediatr Orthop*. 1988;8:385–388.
69. Rab GT. The geometry of slipped capital femoral epiphysis: implications for movement, impingement, and corrective osteotomy. *J Pediatr Orthop*. 1999;19:419–424.
70. Rennie AM. The pathology of slipped upper femoral epiphysis. A new concept. *J Bone Joint Surg Br*. 1960;42:273–279.
71. Restrepo R, Reed MH. Impact of obesity in the diagnosis of SCFE and knee problems in obese children. *Pediatr Radiol*. 2009;39(Suppl 2):S220–225.
72. Sankar WN, Brighton BK, Kim YJ, Millis MB. Acetabular morphology in slipped capital femoral epiphysis. *J Pediatr Orthop*. 2011;31:254–258.
73. Sankar WN, McPartland TG, Millis MB, Kim YJ. The unstable slipped capital femoral epiphysis: risk factors for osteonecrosis. *J Pediatr Orthop*. 2010;30:544–548.
74. Schai PA, Exner GU, Hansch O. Prevention of secondary coxarthrosis in slipped capital femoral epiphysis: a long-term follow-up study after corrective intertrochanteric osteotomy. *J Pediatr Orthop B*. 1996;5:135–143.
75. Sink EL, Zaltz I, Heare T, Dayton M. Acetabular cartilage and labral damage observed during surgical hip dislocation for stable slipped capital femoral epiphysis. *J Pediatr Orthop*. 2010;30:26–30.
76. Slongo T, Kakaty D, Krause F, Ziebarth K. Treatment of slipped capital femoral epiphysis with a modified Dunn procedure. *J Bone Joint Surg Am*. 2010;92:2898–2908.
77. Song KS, Oh CW, Lee HJ, Kim SD. Epidemiology and demographics of slipped capital femoral epiphysis in Korea: a multicenter study by the Korean Pediatric Orthopedic Society. *J Pediatr Orthop*. 2009;29:683–686.
78. Southwick WO. Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. *J Bone Joint Surg Am*. 1967;49:807–835.
79. Southwick WO. Compression fixation after biplane intertrochanteric osteotomy for slipped capital femoral epiphysis. A technical improvement. *J Bone Joint Surg Am*. 1973;55:1218–1224.
80. Stanitski CL, Woo R, Stanitski DF. Acetabular version in slipped capital femoral epiphysis: a prospective study. *J Pediatr Orthop B*. 1996;5:77–79.
81. Stanitski CL, Woo R, Stanitski DF. Femoral version in acute slipped capital femoral epiphysis. *J Pediatr Orthop B*. 1996;5:74–76.
82. Steiner MM. Obesity with slipped femoral epiphysis. *Case Rep Child Meml Hosp Chic*. 1947;6:1017.

83. Stulberg SD, Cordell LD, Harris WH, Ramsey PL, MacEwen GD. Unrecognized childhood hip disease: a major cause of idiopathic osteoarthritis of the hip. In: *Third Open Scientific Meeting of the Hip Society*, St Louis, MO, USA: CV Mosby; 1975:212–228.
84. Tosounidis T, Stengel D, Kontakis G, Scott B, Templeton P, Giannoudis PV. Prognostic significance of stability in slipped upper femoral epiphysis: a systematic review and meta-analysis. *J Pediatr*. 2010;157:674–680, 680–681.
85. Weiner D. Pathogenesis of slipped capital femoral epiphysis: current concepts. *J Pediatr Orthop B*. 1996;5:67–73.
86. Wells D, King JD, Roe TF, Kaufman FR. Review of slipped capital femoral epiphysis associated with endocrine disease. *J Pediatr Orthop*. 1993;13:610–614.
87. Ziebarth K, Zilkens C, Spencer S, Leunig M, Ganz R, Kim YJ. Capital realignment for moderate and severe SCFE using a modified Dunn procedure. *Clin Orthop Relat Res*. 2009;467:704–716.