CHAPTER 25

Robert M. Kay
Young-Jo Kim

Slipped Capital Femoral Epiphysis

SURGICAL PROCEDURE INDEX

Slipped *capital femoral epiphysis* (SCFE) is defined as the displacement of the femoral head relative to the femoral neck and shaft. The term *slipped capital femoral epiphysis* is actually a misnomer. The femoral head is stabilized in the acetabulum, whereas the femoral neck and shaft move relative to the femoral head and acetabulum. In almost all cases of SCFE, the proximal femoral neck and shaft move anteriorly and rotate externally relative to the femoral head (1). If progression occurs to the point at which the femoral neck is completely anterior to the femoral head, then proximal migration of the femoral neck occurs as well.

EPIDEMIOLOGY

The epidemiology of SCFE has been reported frequently in the last century. The male population with SCFE outnumbers the female population by 1.4 to 2.0 in most studies (2–10). The annual incidence is 2 to 13 per 100,000, and the cumulative risk is between 1 per 1000 and 1 per 2000 for the male population and is between 1 per 2000 and 1 per 3000 for the female population (11–14). Incidence of SCFE varies significantly among different populations, with higher incidences in those groups with higher mean body weights (15). Loder (15) has noted more than a 40-fold difference in the incidence among differing races, with the highest rate being found in Polynesian children and the lowest rate being found in children from the Indo-Mediterranean region. In a more recent survey of the epidemiology of SCFE, Lehmann et al. (16) reported that SCFE remains more common in boys (13/100,000) than girls (8/100,000) but that that the age of onset appears to be getting younger. In the same study, the incidence was almost 4 and 2.5 times the incidence in blacks and Hispanics compared to whites (16).

Most children with SCFE are peripubertal. Loder (15) reported an average age of 12 ± 1.5 years for girls and 13.5 ± 1.7 years for boys in an international study carried out with more than 1600 patients. At the time of presentation, approximately 80% of the boys are reported to be between 12 and 15 years and 80% of the girls between 10 and 13 years (17). Onset of SCFE is unusual for children of either sex <10 years old and for girls older than 14 and boys older than 16. Diagnosis of SCFE in such patients should raise the orthopaedist's suspicion that an underlying metabolic or systemic condition may have played a causative role. Furthermore, subclinical endocrine abnormalities may be common. In a prospective study, 14 patients with SCFE were screened for 154 endocrine abnormalities. Despite lack of clinical symptoms, 27% of the surveyed laboratory findings had some evidence of abnormality (18).

The range of skeletal ages of children with SCFE has been reported to be significantly narrower than the range of their chronologic age (10, 19, 20). Most of the children with SCFE have open triradiate cartilage and are Risser 1 (21).

Obesity has been reported in 51% to 77% of patients with SCFE (6, 15, 22–24). Approximately 50% of the patients are at or above the 90th percentile for weight (25), and approximately 70% are above the 80th percentile (26). Obese children with slow maturation appear to be at especially high risk for SCFE (27).

Unilateral involvement is noted in 80% of children with SCFE at the time of presentation, with left hip involvement

in most unilateral cases (12, 13, 15, 28, 29). In addition to the 20% who initially present with bilateral SCFE, 10% to 20% develop a symptomatic contralateral slip in adolescence (6, 13, 30–32). Long-term studies have reported radiographic evidence of a long-term bilateral involvement in as many as 80% of the patients (33), although most series report bilateral involvement at long-term follow-up in the 60% range in adulthood (13, 31). Attempts have been made to identify demographic and radiographic factors associated with the development of bilateral SCFE. However, the data remain controversial. Among the factors associated with increased risk include young chronologic age (girls <12 years and boys <14 years), open triradiate cartilage, and body mass index >35 (34–36).

Some authors have noted significant seasonal variation in the incidence of SCFE at latitudes above 40 degrees, but not in lower latitudes (37–39). Others have not noted any seasonal variation (13). Such data appear to have little impact on the diagnosis and treatment of children with SCFE.

In summary, SCFE is most commonly seen in overweight, peripubertal children. Although any child presenting with hip, groin, thigh, or knee pain must be evaluated for possible hip pathology, the orthopaedist should be particularly suspicious of the possibility of SCFE in overweight, peripubertal children.

ETIOLOGY

In most children with SCFE, the precise etiology is unknown. Regardless of the underlying etiology, the final common pathway appears to be a mechanical insufficiency of the proximal femoral physis to resist the load across it (40). SCFE may be thought of as occurring because of physiologic loads across an abnormally weak physis or abnormally high loads across a normal physis.

Conditions that weaken the physis include endocrine abnormalities, systemic diseases (such as renal osteodystrophy), and previous radiation therapy in the region of the proximal femur (41–46). Multiple mechanical factors have been postulated to account for abnormally high loads across the proximal femoral physis in children with SCFE, including obesity and anatomic variations in the proximal femoral and acetabular morphology.

Endocrine Factors. The endocrinologic basis of SCFE has been studied both *in vivo* and *in vitro*. For more than 50 years, laboratory studies have demonstrated that estrogen strengthens and testosterone weakens the physis (47–49). These effects appear to be secondary to the impact that these hormones have on physeal width since mechanical strength of the physis varies inversely with physeal width (47, 49, 50).

Endocrinopathies appear to account for 5% to 8% of the SCFE cases, and SCFE has been estimated to be six times more common in patients who have an endocrinopathy than in those who do not (41–46, 51–58). Although one recent study showed frequent endocrine abnormalities, most investigators have been unable to demonstrate consistent abnormalities in most children with SCFE (25, 26, 59, 60).

The most common endocrinopathies in children with SCFE are hypothyroidism, panhypopituitarism, growth hormone (GH) abnormalities, and hypogonadism (41–46, 51–58). Other endocrine causes of SCFE include hyperparathyroidism or hypoparathyroidism (41, 44, 61). The increased prevalence of hypothyroidism in children with Down syndrome is a likely explanation for the increased risk of SCFE in these children (62–64).

The relative risk of SCFE is increased in children with GH deficiency, both prior to and during GH treatment (65–67). Other children with short stature and normal GH levels do not appear to share the same increased risk of SCFE (65, 66). The initial diagnosis of hypothyroidism is often made after the diagnosis of SCFE; in most children with SCFE and GH deficiency, the endocrine abnormality is known prior to the diagnosis of SCFE (41).

SCFE has been noted to be most common in children around the time of puberty. It may be that the abnormalities in the complex interplay of hormones at puberty put their hips at risk for SCFE (26, 68). Laboratory studies in rats have also shown a decreased physeal strength at puberty (69).

Because the rate of endocrinopathy in children with SCFE is relatively low, previous authors have recommended against the routine screening of patients with SCFE without clinical evidence of an endocrinopathy (59). Burrow et al. (52) reported that a person's height below the 10th percentile was the only useful screening characteristic for endocrine abnormalities; the sensitivity and the negative predictive value of using height below the 10th percentile as a cutoff were each reported to be at least 90%.

On the basis of the aforementioned data, routine screening of all patients with SCFE for any potential endocrine disease is not warranted. For children with suspected endocrine disease (including those who are younger than 10 years or older than 15 years and those who are of short stature), thyroid function tests should be carried out. GH levels should be checked for children of short stature. It is important to remember that most children with SCFE and thyroid dysfunction have no known history of any thyroid dysfunction at the time of presentation with SCFE. Among other children with both endocrinopathies and SCFE, the underlying endocrine disorder is often known prior to the diagnosis of SCFE.

Other Systemic Diseases. Previous radiation therapy to the region of the femoral head also increases the risk of SCFE (70, 71). The absolute risk of SCFE in patients with previous radiation therapy is unknown, although a risk as high as 10% has been cited (70). Unlike the typical patient with SCFE, children with SCFE following previous radiation therapy have been reported to have a median weight at the 10th percentile (71).

Renal osteodystrophy is associated with a sixfold to eightfold increased risk of SCFE (66). The incidence of SCFE has been reported as 0.03 to 0.64 per 1000 person-years among patients with end-stage renal disease receiving GH, with the highest rates in those patients who were on dialysis and receiving GH (72). Patients with renal osteodystrophy and SCFE are noted to be small in both weight and height (73).

The increased rate of SCFE associated with renal osteodystrophy is due to secondary hyperparathyroidism in these children, and medical management of the secondary hyperparathyroidism is of primary importance (73). If the hyperparathyroidism is controlled, slip progression will become rare, and surgical stabilization may not be necessary (73). Unlike the situation in other causes of SCFE, the displacement in patients with renal osteodystrophy is often through the metaphysis (35% of reported SCFE in one series), and other epiphyses have also been known to displace (73–75). Bilateral involvement has been reported in 82% to 95% of the patients with SCFE and renal osteodystrophy in large series studies (73, 75). That many of these so-called SCFE cases do not occur through the physis may partly be the reason for the poorer results in the treatment of SCFE in children with renal osteodystrophy.

Immunology. Elevated levels of serum immunoglobulins and the C3 component of complement have previously been reported in patients with SCFE (76). In patients with chondrolysis, serum immunoglobulin M level was elevated as well (76). More recent studies have failed to show such abnormalities in serum levels, although synovial fluid abnormalities were noted in patients with SCFE (77, 78). One study reported that plasma cells were a significant component of the synovitis in SCFE (77). In the same study, two of three patients with IgG and C3 present on synovial immunofluorescence developed chondrolysis (77). A later study revealed the presence of immune complexes in the synovial fluid in 10 of the 11 hips with SCFE (91%), but not in 2 of the 21 joints without SCFE (10%) (77, 78). The role of these immune complexes in SCFE has not been defined.

Genetics. A genetic basis for SCFE has not been definitively established. Among the patients with SCFE, a second member of their family has been reported to be affected in 3% to 7% of the cases in most series of studies carried out (10, 24, 32, 79–86). SCFE has been reported in identical twins (79, 81, 87) and has been found to have autosomal dominant inheritance with variable penetrance in familial cases (85, 86). Whether this is due simply to a genetic predisposition for SCFE or due also to a tendency toward other risk features (such as obesity) remains unclear (85, 88).

Some authors have reported an association of human leukocyte antigen (HLA) B12 with SCFE (79, 81, 89, 90), whereas others have reported an association of HLA DR4 with SCFE (91). Other authors have noted that neither of these HLA phenotypes is a reliable marker of SCFE (92).

Mechanical Factors. A variety of mechanical factors appear to play a role in the etiology of SCFE. Anatomic risk factors in the proximal femoral and acetabular morphology have been described. The high incidence of obesity in this patient population also suggests a mechanical role in the etiology of SCFE.

An association of SCFE with a decreased femoral anteversion has been reported, and this has been attributed to increased shear force across the proximal femoral physis in such patients (93, 94). Anteversion values of the unaffected hips in the same patients were closer to normal (93).

Finally, reduced femoral anteversion has been noted in obese adolescents compared to adolescents of normal weight (95). This relative retroversion in obese adolescents may help explain the increased incidence of SCFE in this population group.

Decreased femoral neck–shaft angle in the hips of patients with SCFE compared to the hips of unaffected persons has also been reported (94). Such a decrease in the neck–shaft angle results in a more vertical physis, which may increase the shear force across the physis. Proximal femoral physeal inclination has previously been shown to change significantly between the ages of 9 and 12 years in humans, which is a potential contributing factor for SCFE (96). In the laboratory, the shear strength has also been shown to vary with physeal inclination (50).

Children with deeper acetabuli appear to be at greater risk for SCFE (97). The supposition is that with the capital femoral epiphysis anchored more deeply in the acetabulum, forces across the physis may be exaggerated, especially at the extremes of the range of motion. Variability in acetabular depth has been suggested as a potential cause for differences in the incidence of SCFE among different races. A recent study of acetabular morphology in patients with trauma calls this finding into question (98). It is possible that this study did not find such a correlation either because of limited sample size and/or because SCFE may simply be occurring in a small subset of the population who are outliers regarding such measures as acetabular depth.

Kordelle et al. (99) have not found any difference in acetabular morphology in the affected and the unaffected hips of children with SCFE. The lack of such acetabular differences is likely because SCFE generally occurs at an age at which little potential remains for acetabular remodeling, and this may help explain the high incidence of bilateral SCFE. Such bilateral acetabular symmetry in those with unilateral SCFE suggests that even if increased acetabular depth is a risk factor, there must be other etiologic factors involved as well.

Chung et al. (100) reported that the mechanical forces across the femoral head during gait can be 6.5 times body weight and that such forces may be enough to cause a SCFE in an obese patient with a normal physis. A finite element analysis by Fishkin et al. (101) showed that, in an "overweight" child, the combination of proximal femoral varus and retroversion could result in sufficient forces at the physis to cause SCFE. Other authors have confirmed that mechanical forces across the hip during normal activities such as running are great enough to potentially cause SCFE (102).

In summary, the etiology of SCFE appears to be complex and is likely to be multifactorial. Endocrinopathies, other systemic diseases, and local abnormalities (such as those caused by previous radiation exposure) have been noted to result in an increased risk of SCFE. Studies carried out on humans and animals indicate that such an increased risk of SCFE appears related to the impact that these maladies have on the strength of the growth plate. The association of hypothyroidism in children with Down syndrome and of secondary

hyperparathyroidism in those with renal osteodystrophy explains the sometimes unclear risk profile of SCFE in certain groups of patients. Subtle abnormalities of hormonal balance at the time of puberty may also be partially responsible for SCFE in children without any definite systemic or hormonal abnormalities.

Mechanical factors also appear to play an etiologic role in the development of SCFE. Clearly, systemic and local factors alone cannot explain all the cases of SCFE because many patients with the aforementioned abnormalities do not develop a SCFE. In addition, most patients with SCFE provide evidence of increased forces across the proximal femoral physis due to one or more potential causes, including obesity and variations in the proximal femoral and/or acetabular morphology.

CLINICAL FEATURES

Traditionally, classification of SCFE has been made on a temporal basis. Chronic slips are those causing symptoms for a period of at least 3 weeks, whereas acute slips are those that are symptomatic for <3 weeks. Acute-on-chronic slips are those with an acute exacerbation of the symptoms following a prodrome of symptoms of at least 3 weeks' duration. Chronic slips appear to account for 80% to 90% of all SCFE. Although not part of the preceding scheme, a "preslip" has been defined as a symptomatic hip with evidence of physiolysis prior to true movement of the femoral neck relative to the femoral head.

In 1993, Loder et al. (103) suggested a new classification of SCFE based on physeal stability. An unstable SCFE was defined as occurring in an extremity upon which the child had such severe pain that walking is not possible even with crutches. With a stable slip, the child can walk with or without crutches. Unstable SCFE account for 50% to 60% of acute SCFE and for 5% to 10% of all SCFE (103–106). This classification of SCFE based on stability has largely supplanted the aforementioned temporal classification scheme because of its improved ability to predict both osteonecrosis (ON) and poorer outcomes. Whereas ON is usually reported in 10% to 15% of acute SCFE, Loder et al. (103) reported ON in 47% of unstable SCFE and 0% stable SCFE in their landmark paper. Even in cases of acute SCFE, only the unstable subset appear to be at significant risk for ON and a poor outcome (103, 107).

The most common findings at presentation of SCFE include pain, limp, and decreased range of motion of the hip. Hip or groin pain in an obese, peripubertal child is highly suggestive of SCFE. However, hip pain is absent in as many as 50% of the children with SCFE, including up to 8% with a painless limp (108). Pain is localized to the knee and/or distal thigh in 23% to 46% of cases (4, 6, 108, 109). Previous studies have noted that distal thigh and/or knee pain often result in significant misdiagnosis of SCFE, delay in diagnosis, unnecessary radiographs, increased slip severity, and sometimes in unnecessary knee arthroscopy (4, 6, 23, 108–111). These findings indicate the importance of examining the hip in all children presenting with distal thigh and/or knee pain.

Symptoms of SCFE are generally present for weeks to several months prior to presentation to the orthopaedist (15, 112). Although patients report a specific inciting event as the cause of pain in approximately 50% of cases, severe trauma is rarely reported (108). Even when trauma is reported, further questioning often reveals a history of pain for weeks or months preceding the inciting event.

A significant proportion of the 5% to 10% of children with unstable SCFE present with an acute onset of severe hip pain in the absence of prodromal symptoms (15, 113, 114). Such SCFE often follow mild trauma.

As has been noted, most children with SCFE are obese. Short stature (height less than the 10th percentile) has been reported to be an indicator of increased risk for underlying systemic disease in children with SCFE (52). Loder and Greenfield (115) noted that SCFE due to an underlying cause (such as underlying systemic disease or previous radiation exposure) was much greater in children older than 16 years and/or those who were below the 50th percentile for weight at the time of presentation.

When a child presents with hip, groin, thigh, or knee pain, care must be taken to evaluate both hips. The physician needs to be persistent when asking about symptoms in both hips, because a child often initially complains of only the more symptomatic hip in cases of bilateral SCFE.

One of the most helpful tip-offs in these patients is the observational gait analysis when the child walks into the examining room. The limp in children with SCFE is due to several gait deviations. Hip abductor weakness commonly manifests as a trunk lean to the affected limb in stance (Trendelenburg gait). If there is marked pain, an antalgic gait (decreased stance phase on the affected limb) will be present as well. Finally, because of the external rotation of the femoral neck and shaft (relative to the femoral head), the foot and knee progression angles on the affected side are often markedly external. Children with unilateral involvement have significant asymmetry of foot and knee progression angles with a unilateral Trendelenburg gait, whereas children with bilateral SCFE present with a more "waddling" gait bilaterally, and bilateral external foot and knee progression.

On physical examination, range of motion of the hips—including the rotational profile of the hips—should be measured and compared. Hip flexion to 90 degrees is unusual, and hip flexion contractures are common. Because both hip flexion and extension are lost, there is significant diminution of the sagittal arc. Hip abduction is significantly limited both actively and passively, and the hip abductors are weak.

Hip rotation is abnormal because of both the abnormal anatomy and the synovitis that accompany SCFE. Loss of the hip internal rotation is combined with preservation of (or even an increase in) external rotation. With a SCFE, the hip will

automatically fall into external rotation (the so-called obligate external rotation) as it is progressively flexed. Obligate external rotation of the hip(s) is essentially pathognomonic for SCFE. In cases of unilateral SCFE, comparison with the rotation of the contralateral hip clearly demonstrates this change in the arc of motion. In bilateral SCFE, both hips will demonstrate this shift toward external rotation.

In summary, any patient between the ages of 10 and 16 years who presents with a limp and pain in the groin, hip, thigh, or knee should be considered to have a SCFE until proven otherwise. Diagnoses such as pulled groin muscles are rarely correct in children, although such misdiagnoses are commonly made in children with SCFE. The index of suspicion for the diagnosis of SCFE is markedly increased in obese, peripubertal children with a limp, external foot progression, and pain in the groin, hip, thigh, or knee. The index of suspicion is also very high in patients with a known history of endocrine abnormalities and in those with underlying diseases associated with endocrine abnormalities, such as Down syndrome and renal osteodystrophy.

RADIOGRAPHIC FEATURES

Radiographs. High-quality anteroposterior and lateral radiographs of each hip should be obtained to confirm the diagnosis of SCFE. Because of the high frequency of bilateral SCFE, bilateral imaging has been recommended for decades (23, 116, 117). In an unstable, acute SCFE, a frog lateral view is not obtained preoperatively in order to avoid causing pain and because of the potential for displacement of the SCFE. However, it is usually possible to obtain a crosstable lateral view of the affected hip without adversely affecting the unstable SCFE.

On the anteroposterior view, widening and irregularity of the physis may be the only radiographic findings prior to, or with minimal, displacement of the femoral neck and shaft relative to the femoral head. Cowell (108) noted that the displacement may not be evident in 14% of the anteroposterior views. Another common finding on the anteroposterior view is a decreased height of the capital femoral epiphysis when the epiphysis lies posterior to the femoral neck. As slipping progresses, the metaphysis appears progressively more lateral relative to the acetabular teardrop, and an increased radiodensity of the proximal metaphysis (the so-called metaphyseal blanch) may be noted (118). Osteopenia of the affected hip is common as well.

Lateral views are more sensitive for detecting mild degrees of slip. With increased magnitude of slipping, the SCFE becomes evident on the anteroposterior view as well. Normally, a portion of the femoral head lies lateral to Klein line (a line drawn along the lateral border of the femoral neck) (116) (Fig. 25-1A,B). A SCFE is present if the Klein line lies cephalad to the femoral head, or if the amount of femoral head cephalad to the Klein line is less than that is seen for the contralateral hip.

Crosstable lateral views are often cited as more reliable than frog lateral views in the assessment of SCFE, which may be due to difficulties with the positioning of these children (119, 120). However, using a femoral model, Loder (121) reported that an accurate representation of the SCFE was obtained with either crosstable or frog lateral views when the

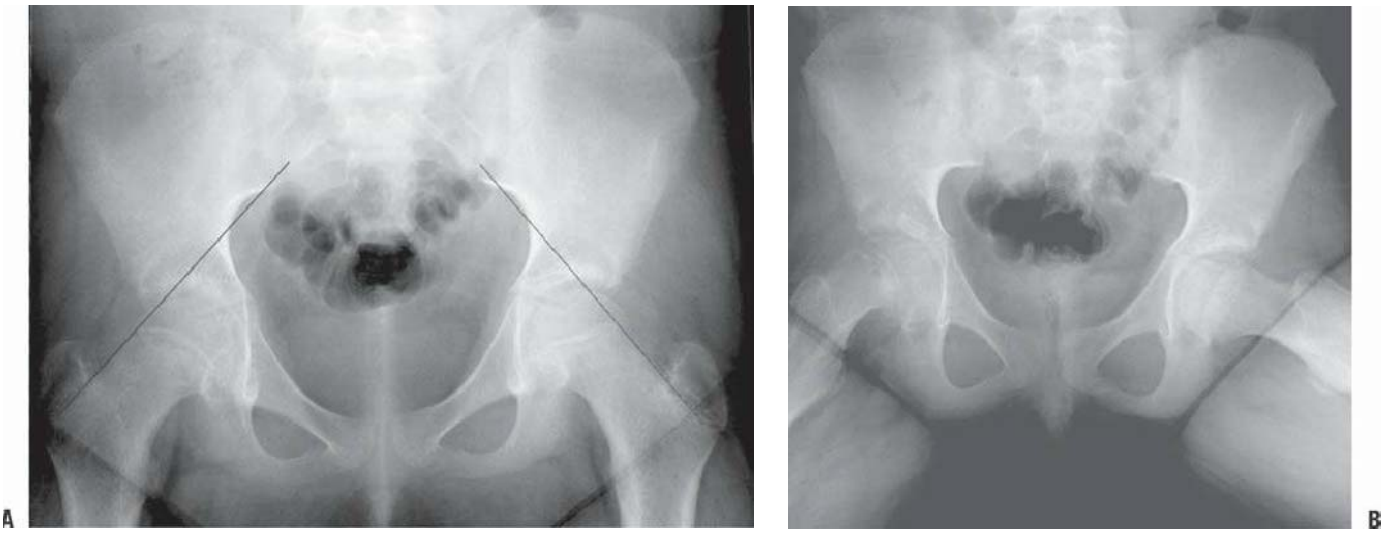


FIGURE 25-1. Radiographs of a 12-year-old boy with 3 months of hip pain show typical findings of a SCFE. **A:** Anteroposterior view demonstrates physeal widening, osteopenia, decreased epiphyseal height, increased metaphyseal-teardrop distance, and asymmetry of Klein line. **B:** Although many of these features are seen on the anteroposterior view, the most striking feature is how much more easily the displacement is seen on the frog lateral view. The importance of obtaining lateral views when evaluating for SCFE cannot be overemphasized.

femur is rotated externally by 30 degrees or less. The value of other specialized views, such as the Billing lateral, is still being debated (121, 122).

The degree of slip is commonly quantified as the amount of femoral head displacement as a percentage of the femoral neck diameter, and was first described by Wilson in 1938 (23). Slips have been categorized as mild (<33%), moderate (33% to 50%), and severe (more than 50%) (6, 24). Although frequently used, this measurement can be inconsistent because of variations in patient positioning and can change over the passage of time because of proximal femoral remodeling. This measurement should therefore be used only in the evaluation of SCFE prior to remodeling (123).

Southwick (124) recommended measuring the angles between the proximal femoral physis and the femoral shaft, the so-called head–shaft angles, on both anteroposterior and lateral radiographs. The difference between these two angles obtained at the affected and the unaffected sides determines the degree of abnormal alignment and is often referred to as *Southwick angles*. The lateral view gives an indication of posterior angulation. A difference of <30 degrees has been deemed mild, a difference of 30 to 50 degrees moderate, and more than 50 degrees is deemed as severe (125).

The angle between the proximal femoral physis and femoral neck, the so-called head–neck angle, may be measured but is less reliable because remodeling adjacent to the SCFE may artificially decrease this number in the absence of clinically significant changes in femoral version.

Other Imaging Studies. Radiographs are sufficient for the evaluation of most children with SCFE. However, additional imaging may be warranted in special circumstances, such as in the evaluation of a presumed "preslip" in a child with normal radiographs, or in the early evaluation of a patient with SCFE at risk for ON.

Computed tomography (CT) scans are rarely needed as a part of the initial assessment of children with SCFE (120). Some authors report that CT scan is more accurate than radiographs in evaluating the anatomy of SCFE (120), whereas others report comparable reliability between the two modalities (126, 127). If a child presents very late in the course of SCFE, a CT scan may be useful in determining whether sufficient physeal closure has already occurred, thereby potentially precluding the need for an *in situ* fixation. A CT scan may also be helpful postoperatively in determining whether any hardware used during surgery has accidentally penetrated the joint surface. This is particularly true in the case of femoral head collapse in association with ON of the femoral head.

Ultrasound has been championed by some authors, but currently appears to have little use in the routine evaluation of patients with SCFE (128–131). Previous studies using ultrasound images have indicated the presence of effusion in 42% to 60% of patients with SCFE (130, 131). In experienced hands, ultrasound may have a role in confirming a suspected case of SCFE in the absence of any radiographic findings, but magnetic resonance imaging (MRI) is more commonly used in such situations.

MRI often plays an important role in the evaluation of hips of patients who are presumed to have SCFE but have normal radiographs, and MRI may also be used for the early detection of ON. The MRI findings in SCFE have been well described (127, 132–134). Physeal widening, osseous edema adjacent to the physis, and the anatomic deformity associated with SCFE are typically seen, with the findings of physeal widening and irregularity as well as osseous edema adjacent to the physis seen in cases of "preslips" (134). In a child with suspected SCFE and normal radiographs, MRI is useful in determining whether a preslip is present (Fig. 25-2). Currently, MRI scanning is rarely used in evaluating patients with evident SCFE.

MRI may be used to assess femoral head circulation in order to evaluate for the presence of ON, as well as its extent and distribution if present. Unfortunately, metal artifact may significantly interfere with MRI signals. The findings of ON seen on MRI scans have not been correlated with subsequent radiographic findings and the clinical course of the affected hips.

Bone scans may be used to assess femoral head viability in potential cases of ON of the femoral head, with decreased uptake being evident in cases of ON. Multiple studies have reported the utility of bone scanning in the detection of ON in SCFE (130, 135, 136). Sensitivity in detecting ON has been 100% in several series, although a false-negative bone scan has been reported in a child who went on to develop mild ON (130, 135–137).

Although pretreatment bone scans are quite sensitive, they are also associated with false-positive results (i.e., an abnormal bone scan in a hip that does not develop ON). In two series, false-positive bone scans have been reported in one of the six (17%) (136) and two of three (67%) hips that were imaged (130).

Since pretreatment bone scans and MRI do not generally change treatment, they are not routinely obtained in children with SCFE at most centers.

PATHOANATOMY

Because the femoral head is relatively fixed inside the acetabulum, the slip is best thought of as a slip of the proximal femoral neck and shaft relative to the femoral head. In children younger than 3 years, the perichondral ring imparts significant physeal stability, whereas the mammillary processes of the physis are primarily responsible for increasing physeal shear strength thereafter (100).

In laboratory rats, physeal cracks are evident in the planes of shear stress used to create SCFE (69). The mechanical patterns of physeal fracture and the zone through which physeal shear causes fractures have been shown in rabbits to vary with increasing age and with the direction of loading (138, 139).

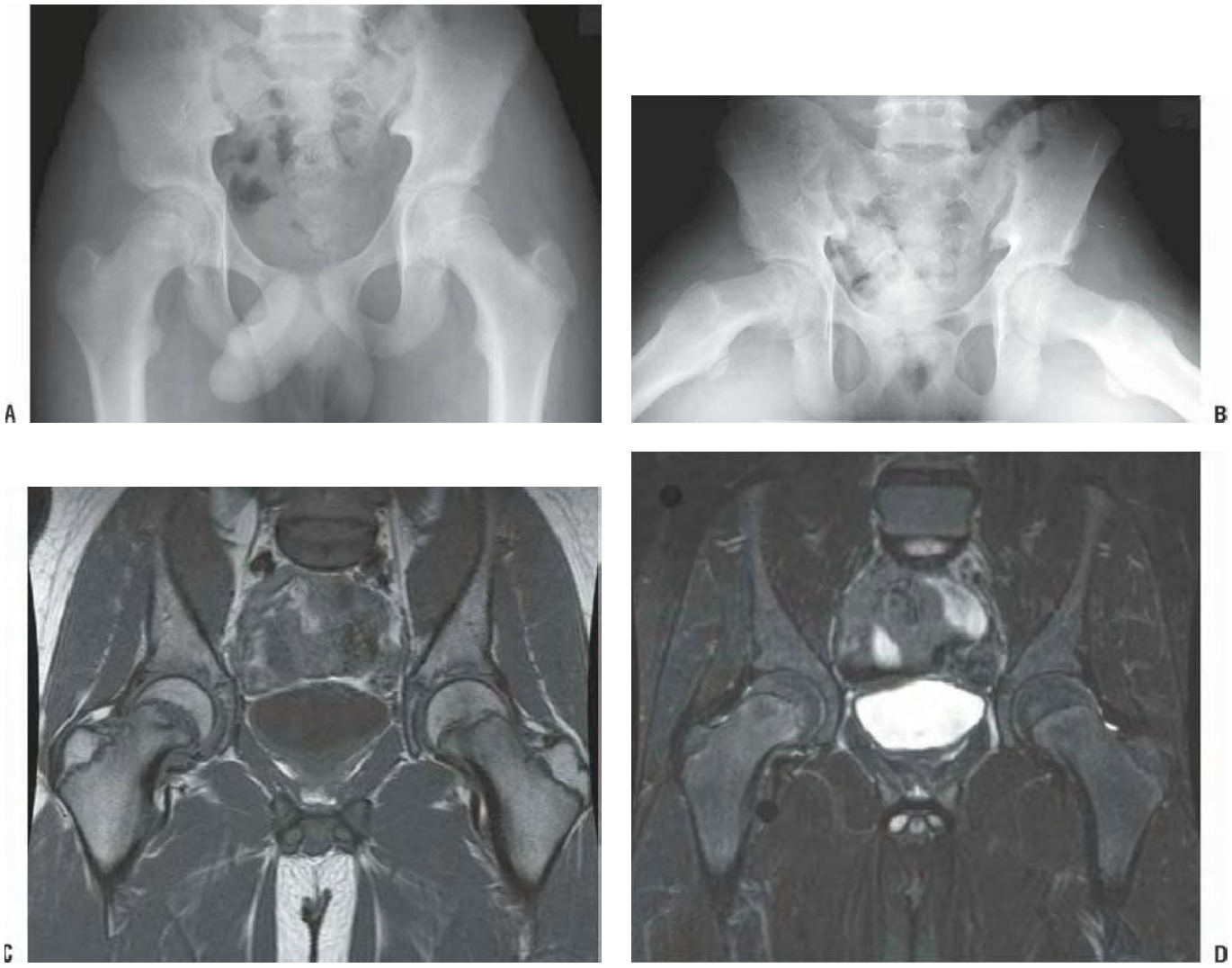


FIGURE 25-2. A 12-year-old boy presented with pain in the right hip for 2 months. On further questioning, he reported some vague, intermittent symptoms in the left hip. Physical examination revealed pain in the right hip and obligate external rotation, but no such findings on the left. **A,B:** Anteroposterior and frog pelvis views at the time of presentation. A right SCFE is evident, without definite plain radiographic changes on the left. **C,D:** Because of the vague left hip symptoms, MRI was done to rule out a left SCFE. MRI demonstrated physeal widening and irregularity (T1: flip angle 90 degrees, TR 700, TE 18) (seen best in C) and signal change on the right, mostly in the metaphysis in this case (fat saturation: flip angle 90 degrees, TR 4500, TE 75.37) (best seen in D), without any definite abnormalities on the left. Only the right hip underwent *in situ* fixation because of the normal physical examination and the lack of considerable MRI findings in the left hip. The patient denied ongoing pain in the left hip until 9 months following *in situ* pinning of the right hip. He then had progressive pain in the left hip and re-presented to the orthopaedist 1 month later, at which time a mild left SCFE was noted and *in situ* fixation of the left hip was performed.

In humans, the direction of slip has been known for decades (1). In most cases, the proximal femoral neck and shaft migrate anteriorly and rotate externally, although slips have been noted to occur in other directions (140, 141). Previous authors have confirmed this anatomy and suggested a torsional force as the cause of acute SCFE (142). With progression of the slip, the femoral neck may come to lie completely anterior to the femoral head. When this occurs, proximal migration of the proximal femur is possible (Fig. 25-3). However, most SCFE do not appear to progress to this point, and the apparent varus seen radiographically has been attributed to parallax (143, 144). Degenerative changes, including cyst formation, may be seen in the anterior femoral neck and/or acetabulum because of impingement of the anterior femoral neck against the acetabulum during hip flexion, and such changes may be evident within years of the diagnosis of SCFE.

On the basis of computer modeling, Rab has noted that metaphyseal impingement limits the motion in severe SCFE

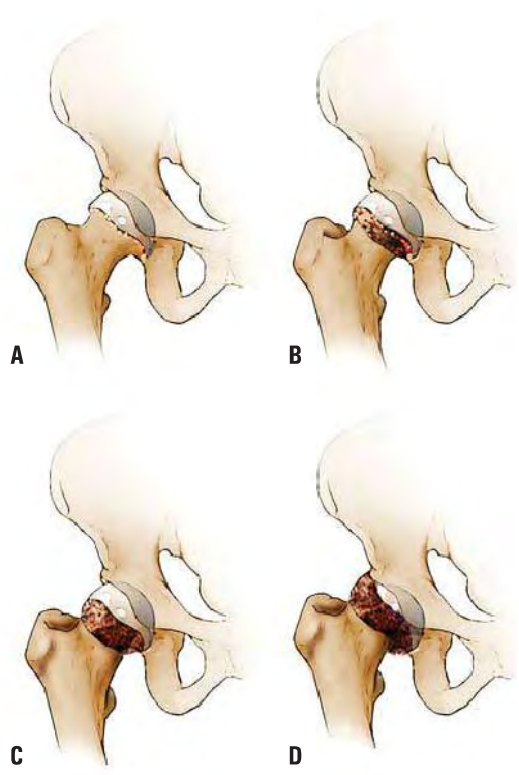


FIGURE 25-3. Pathoanatomy of SCFE is demonstrated. **A:** No displacement is seen. **B:** Rotation of the proximal femoral neck, with the femoral head (which is anchored in the acetabulum) posterior relative to the femoral neck. **C:** Progressive external rotation, with progressive posterior relation of the femoral head to the femoral neck. **D:** Proximal migration of the femoral neck due to the markedly posterior relation of the femoral head to the femoral neck. (From Morrissy RT. Principles of *in situ* fixation in chronic slipped capital femoral epiphysis. *Instr Course Lect* 1989;38:257–262, with permission.)

(145). He reported that as the slip angle increases, progressively greater external hip rotation is necessary to avoid anterior impingement of the proximal femoral metaphysis against the acetabulum during gait. Such levering can damage the anterosuperior acetabular cartilage and/or cause posterolateral labral injuries (145–148). Intraoperative evaluation by other authors has confirmed the mechanical impingement of the metaphysis against the superomedial acetabulum, with resulting cartilage and labral damage (149). Femoroacetabular impingement has been suggested as a cause of idiopathic arthritis as well (150). As noted by Rab (145), as the proximal femur remodels and motion returns toward normal, an increasing portion of the remodeled metaphysis becomes an intra-articular weight-bearing surface, potentially contributing to late osteoarthritis (OA).

Multiple studies have investigated the pathologic changes in SCFE (138, 139, 151–159). Multiple authors have noted the replacement of normal physis with abnormal cartilage, fibrocartilage, and fibrous tissue (156, 159). The physis is often hypocellular, with increased amounts of ground substance in lieu of the normal columnar architecture (151, 157). Others have noted a widening of the physis, with a loss of normal organization and the presence of clefts within the physis (158). Subsequent authors have confirmed the columnar disorganization with cartilage cell clumping in the physis, metaphysis, and epiphysis (157, 159). Groups of cartilage cells have been noted between metaphyseal trabeculae (155, 157, 159). Collagen fibrils are markedly diminished in the hypertrophic zone (157). The resting zone is essentially normal (155, 157). The proliferative zone has less densely packed collagen and increased disorganization, with ground substance replacing the normal chondrocytes. The hypertrophic zone is much larger than usual (up to 80% of the physeal width in comparison to 15% to 30% in normal physes) with marked disorganization, increased ground substance, and significant staining for glycoproteins (155, 157). Cell degeneration and death have been noted in the proliferative and hypertrophic zones (151–153). The slip occurs through the proliferative and hypertrophic zones of the physis in an irregular pattern (68, 155, 157, 159). Histologic sections of the physis in SCFE before and after *in situ* fixation demonstrate a return to a more normal architecture following fixation; such findings have been postulated to indicate that mechanical stabilization of the physis, with removal of the abnormal shear forces across the physis, allows at least a partial reversal of the pathology seen with SCFE (154).

BLOOD SUPPLY

ON is one of the few potentially devastating complications associated with SCFE, and understanding the proximal femoral blood supply is important in attempting to minimize the frequency of this complication. The blood supply of the proximal femur can be divided into the intraosseous and extraosseous components, as has been well documented by Crock and subsequently by Chung (160, 161) (Fig. 25-4). Chung (160) noted that these components are present in an individual at birth and persist without significant change into adulthood. In cases of SCFE, the blood supply can be disrupted because of the SCFE itself (especially in cases with unstable SCFE), and it may also be compromised at the time of surgery.

It appears that the cause of ON is likely to be the disruption of the blood supply, which may occur because of displacement at the time of injury or at any time prior to operative fixation. Angiography performed in 12 patients with SCFE preoperatively showed filling of the superior retinacular artery in all 7 stable slips and in only 2 of the 5 unstable slips (162). In one of the three unstable SCFE without preoperative filling of the superior retinacular artery, postoperative angiography demonstrated appropriate filling (162).

Extraosseous Blood Supply. The extraosseous blood supply to the proximal femur may be disrupted in acute SCFE and has been well described. An arterial ring at the base of the femoral neck gives rise to ascending cervical arteries that penetrate the hip capsule and provide circulation to the femoral head, neck, and greater trochanter (160, 161). The

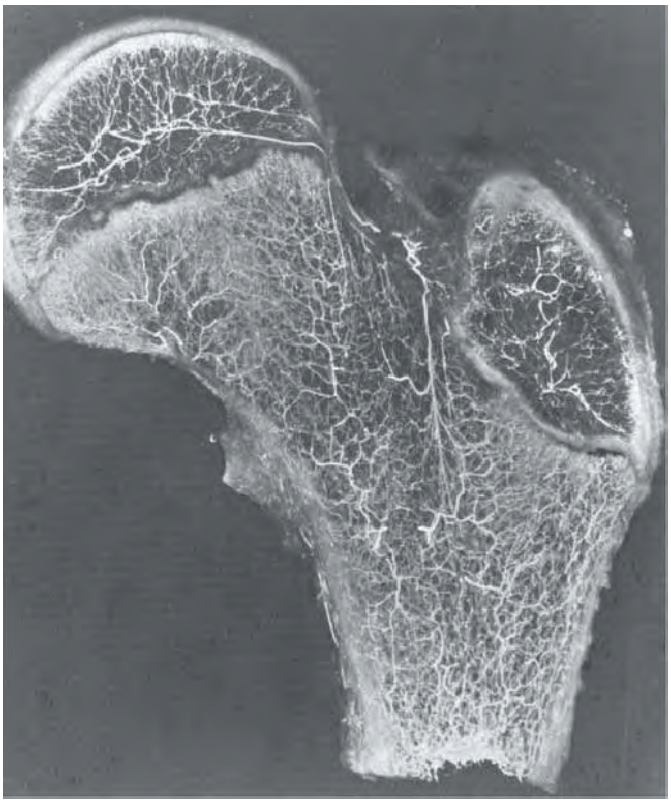


FIGURE 25-4. A coronal section demonstrating vascularity of the proximal femur in a 13-year-old boy. Part of the vascular ring is visible at the base of the femoral neck, giving rise to the ascending cervical arteries, which then enter the femoral head and supply blood to the superior head. (From Crock HV. A revision of the anatomy of the arteries supplying the upper end of the human femur. *J Anat* 1965;99:77–88, with permission.)

arterial ring at the base of the femoral neck consists of the lateral femoral circumflex artery, which runs anteriorly and constitutes the anterior portion of the arterial ring, and the medial femoral circumflex artery, which travels posteriorly and constitutes the medial, lateral, and posterior portions of the ring. The ring is most commonly incomplete, without communication between the branches from the medial and lateral circumflex arteries.

Ascending cervical arteries (also known as *retinacular vessels*) arise from each portion of this extracapsular arterial ring and penetrate the hip capsule to enter the hip joint. The numerous branches from the lateral ascending cervical artery (which branch from the medial femoral circumflex artery) provide circulation to the greatest portion of the femoral head and neck. After penetrating the hip capsule, the ascending cervical arteries form a second arterial ring that is also usually incomplete. This intra-articular, subsynovial ring is smaller than the extracapsular ring and is located at the border between the articular surface of the femoral head and the femoral neck. These subsynovial vessels are consistently present medially and laterally and less commonly present anteriorly and posteriorly. The epiphyseal branches of these vessels cross the physis on the surface of the femoral head, enter the perichondral ring, and then cross into the epiphysis.

Intraosseous Blood Supply. The intraosseous blood supply may be compromised by proximal femoral osteotomies or the internal fixation of SCFE. The ascending cervical arteries penetrate the intracapsular femoral neck, with different vessels supplying the metaphysis and epiphysis (160, 161, 163). The intraosseous blood supply of the femoral head is mainly located in its posterior and superior portions, with potential implications for the positioning of hardware (160). The extent of anastomoses between these vessels and the arterial branches of the ligamentum teres (which supply the medial third of the femoral head) appears to be quite limited (160, 161, 163).

NATURAL HISTORY

In the short term, the natural history of the affected hip is one of progressive displacement, followed ultimately by stabilization of the slip and physeal closure. Although all slips must eventually cease progressing, the timing of cessation and the degree of the slip prior to cessation and physeal closure are unpredictable. Most slips progress slowly, although some may have significant, acute progression. The hips with such acute progression are the ones at the highest risk for significant complications.

Bilateral SCFE at the time of initial presentation accounted for approximately 20% of the children with SCFE in recent series (15, 28, 29). It is probable that this frequency will further increase with the increased awareness of the frequency of bilateral involvement and with the ongoing improvements in the imaging of SCFE.

An additional 10% to 20% of patients with SCFE are diagnosed with a contralateral SCFE in adolescence (6, 13, 31, 32, 164). About 80% to 90% of symptomatic, contralateral SCFE cases are diagnosed within 18 months of the diagnosis of the first slip, with 66% to 81% being diagnosed in the first year (15, 28, 125, 165). The average duration between the diagnosis of the first and second slips in metachronous bilateral SCFE has been reported as 1.0 ± 0.8 years (15). Contralateral slips have been reported as late as 4 to 5 years following the initial SCFE (6, 15, 24).

The true frequency of bilateral SCFE at long-term follow-up appears to be approximately 60% (13, 31), although rates of up to 80% have been reported (33). Many of the late contralateral SCFE cases reported in long-term radiographic follow-up are mild, asymptomatic slips (13, 31, 33). These data suggest that if 20% of the patients present with bilateral SCFE, then half of the 80% who present with unilateral SCFE will ultimately have a contralateral SCFE.

In the short term, 61% to 100% of children with endocrinopathies and SCFE have bilateral slips, although metachronous involvement is common (41, 58). Because of this significant short-term risk, prophylactic pinning of the contralateral hip is recommended in patients with SCFE and endocrine disease (41, 58).

In the long term, SCFE puts the hip at significant risk of OA, poorer results being associated with an increasing degree of SCFE (8, 125, 166–169). Hagglund et al. (31) reported radiographic

evidence of OA in 27% (28 of 104) of hips with SCFE at long-term follow-up (mean follow-up: 33 years) compared with 9% of control hips (9 of 101). Carney and Weinstein (167) reported a long-term follow-up (mean follow-up: 41 years) of 28 patients with 31 untreated SCFEs (between 1915 and 1952) and correlated the degree of the slips with radiographic and clinical scores. Patients with mild slips fared better than did those with moderate and severe slips with regard to radiographic changes and Iowa hip scores. At long-term follow-up, Iowa hip scores were at least 80 in all 17 hips with mild slips and in 9 of the 14 hips (64%) with moderate or severe slips. There was radiographic evidence of OA in 64% (9 of 14) of the mild slips and in 100% (13 of 13) of the moderate and severe slips.

Ordeberg et al. (169) reported a 20- to 60-year follow-up (mean follow-up: 37 years) of 49 cases of SCFE who did not undergo primary treatment. They reported that only "a few" patients had restrictions regarding their work or social lives and that only 2 of 49 (4%) had required surgery for arthritis. Limb-length discrepancy (LLD) of at least 2 cm was noted in 31% of the cases. The authors also noted that these results were far superior to a comparable group of patients treated with closed reduction and casting. Jerre (32) noted superior results in untreated patients in Sweden as well.

Previous authors have noted that known cases of SCFE account for 2% to 9% of end-stage hip arthritis (170–174). A cadaveric study noted "postslip" morphology in 8% of the skeletons and showed that OA was associated with such morphology (175).

In older case series studies, a significant proportion of adults with "idiopathic" OA have been reported as having a stigma of pediatric hip disease, such as a "pistol grip" deformity. Murray (176) reported an apparent association with SCFE in 40% of the adult hips thought to have degenerative arthritis as evidenced by the so-called tilt deformity of the femoral head. Stulberg et al. (177) reported such deformity in 40% of patients with hip OA and no previously diagnosed hip disease. Stulberg et al. (177), however, noted that the "tilt deformity" did not appear to be unique to SCFE. Resnick (178) has suggested that the "tilt deformity" is not due to SCFE, but is due to the remodeling of the osteoarthritic hip; hence, the underlying etiology of most end-stage hip OA remains unclear.

In summary, 20% of patients with SCFE present with unilateral disease, an additional 10% to 20% develop a contralateral slip during adolescence, and 60% of the patients have bilateral SCFE, which is evident at long-term follow-up. In all the cases of SCFE, OA appears to result, with worse slips being associated with increased rates and severity of the OA. Although SCFE leads to late degenerative changes, most hips function well into their fifth decade or later.

TREATMENT

Once the diagnosis of SCFE is made, the child is admitted to the hospital and is confined to bed until surgery is performed, as has been recommended for decades (23). Under no circumstances should the child be allowed to bear weight once the diagnosis of an acute/unstable SCFE is made, as it may result in ON.

The goals of treatment in SCFE are early detection, prevention of further slipping, and avoidance of complications. Although attention is often focused on the affected hip, care of the unaffected hip (either through careful observation or through prophylactic treatment) cannot be forsaken.

Care of children with SCFE continues to advance along with our understanding of this disease. Increased vigilance and enhanced imaging allow the early detection of SCFE, and percutaneous fixation techniques allow for short hospital stays (or even outpatient surgery). With these enhancements in care, one recent study comparing treatment of children with SCFE at a pediatric hospital to the treatment given at a general hospital reported shorter hospital stays and lower hospital charges at the children's hospital (179).

As has been noted, SCFE puts the patient at long-term risk of OA, with the risk increasing along with the increase in the degree of slip. In some cases, the outcomes of SCFE (treated or untreated) are so poor that salvage treatment by arthrodesis or arthroplasty may be needed.

Historical Methods

Spica Casting. The goal of spica casting is to prevent the progression of a SCFE. Although used in the treatment of SCFE for much of the last century, spica casting is now rarely used in the treatment of SCFE. Because most children with SCFE are obese adolescents, use of a spica cast for these children holds little appeal for most patients, their families, and physicians.

Traditionally, spica casting has been associated with high rates of complications (180). Meier et al. (180) reported complications in 14 of 17 hips in which a SCFE (82%) had been treated with spica casting, including 9 cases of chondrolysis (53%), 3 cases of further slip after cast removal (18%), and 2 cases in which a total of 3 pressure sores developed (12%). Chondrolysis has been reported in 14% to 53% of the cases of SCFE treated with spica casting, and it has also been reported in the uninvolved hip following immobilization (32, 180–183). ON has commonly been reported with the use of spica casting as well, although most cases of ON appear to be due to the forceful manipulation of the SCFE rather than to the spica cast itself.

Progressive slip occurs in 5% to 18% of cases of SCFE treated with spica casting (180, 182). Although Betz et al. (182) cited only a 3% incidence (1 per 37 hips), the true rate is 5% in their study because they excluded the progression of one additional hip that had been followed up for <2 years.

The duration of casting has often been arbitrary. In the absence of any operative intervention, most proximal femoral physes do not close for a year or more following the diagnosis of SCFE. Most children treated with casting are immobilized for 3 to 4 months (180, 182). Betz et al. (182) noted that spica casts could safely be removed when the

juxtaphyseal metaphyseal radiolucency was no longer visible, and that this occurred by 16 weeks in their patients. Although all patients were immobilized in a cast for periods ranging from 117 to 124 days, Meier reported progressive slips in 18% (3 of 17) of the hips after cast removal (180).

With the advent of current fluoroscopic imaging techniques, cannulated screw systems, and the decrease in operative morbidity, there is little role for nonsurgical treatment in children with SCFE.

Bone Graft Epiphysiodesis. The goal of bone graft epiphysiodesis, as with *in situ* fixation, is the prevention of slip progression. However, the way in which this is achieved with the two methods differs. Slip progression is prevented with bone graft epiphysiodesis primarily by hastening physeal closure, whereas *in situ* fixation prevents slip progression primarily by stabilizing the physis. Indications for bone graft epiphysiodesis include acute/unstable or chronic/stable SCFE of any magnitude, although some authors have conceded that cases of mild SCFE are better treated with *in situ* fixation (184) (Fig. 25-5A–E).

Bone graft epiphysiodesis, which involves drilling across the physis into the epiphysis with placement of bone graft (most commonly autologous bone pegs), was first described in 1931 by Ferguson and Howorth (185). Although reported results have often been good (184, 186–194), this operation has been abandoned at many institutions because of potential for morbidity and technical difficulties (195–197).

The surgery may be performed through an anterior or an anterolateral approach and may be combined with osteoplasty of the anterior femoral neck (184, 194, 198). A 50-year experience with bone graft epiphysiodesis in 318 cases of SCFE presents this procedure as a "reasonable alternative" for the treatment of SCFE (187). Patients with acute SCFE are placed in a spica cast or brace postoperatively and kept without bearing weight for 6 to 8 weeks. Patients with chronic slips begin touch-down weight bearing 2 to 3 days postoperatively and bear weight progressively as the physeal closure progresses. Some authors have reported the time required until full weight bearing as averaging 10 weeks (193).

As reported in most series, surgical time (excluding casting, when necessary) averages 2 hours (186, 193, 196). Weiner et al. (193) have reported estimated blood loss (EBL) for autologous bone peg epiphysiodesis of at least 200 mL in 52% of patients (25 of 48), and other authors have reported mean EBL ranging from 426 to 800 mL (195, 196, 199). When allograft is used instead of autograft, mean EBL has been reported as 360 mL (186).

Physeal closure following an autograft bone peg epiphysiodesis is reported to occur at 4 to 6 months by most authors (186, 188, 189, 195, 196). In a series of bone peg epiphysiodesis with allograft, a partial physeal closure was noted radiographically after an average of 11 weeks and complete closure after an average of 28 weeks, with physeal closure occurring in the operated hip before it occurred in the unoperated hip in all of the 16 unilateral cases (186).

Complications of this procedure include graft failure, failure to achieve physeal closure, slip progression, heterotopic ossification, lateral femoral cutaneous nerve (LFCN) palsy, donor site morbidity, chondrolysis, and ON. Heterotopic ossification has been reported in up to 69% of patients (196). Despite intraoperative protection of the nerve, Ward and Wood (199) reported LFCN palsy in 10 out of 14 patients (71%) specifically examined for this finding postoperatively. Rao et al. (196) reported transient LFCN palsy in 11% of their patients.

Graft complications following bone peg epiphysiodesis are well described. In two large series with very good results, Adamczyk et al. (187) reported graft resorption with failure of epiphysiodesis in a period of 1 year in 4% of cases (12 of 318 hips), and Howorth reported graft resorption in 2% of cases (4 of 200 hips), with no cases of progressive slip (188). In a series of 17 cases of SCFE, Ward and Wood (199) reported "graft insufficiency," defined as graft movement, resorption, or fracture, in eight hips (47%). Protrusion of the graft into the hip joint has also been reported (195).

The rate of ON associated with bone peg epiphysiodesis has generally been low, with most reports in the range of 0% to 6%, with higher rates in acute/unstable SCFE (186–188, 196, 198, 199). Adamczyk et al. (187) reported an overall rate of 2%, with a risk of 7% in acute slips (3 of 45 cases) and 1.5% in chronic slips (4 of 273 cases). The low rate of ON in bone peg epiphysiodesis is likely due to placement of the grafts from the anterolateral neck and into the center of the epiphysis, thereby avoiding the intraosseous blood supply.

Chondrolysis is reported to occur in 0% to 6% of the cases of SCFE treated with bone peg epiphysiodesis (186–188, 196, 199). Most cases of chondrolysis occur in acute/unstable SCFE (187, 196).

Progressive slip has been reported in 0% to 19% of cases following bone peg epiphysiodesis, with the highest risk being in acute SCFE (186–188). Although Rao et al. (196) noted a change in the femoral head–shaft angle of at least 5 degrees in 42% of patients (27 of 64), the angle increased in 19% and decreased in 23% of the cases. One presumed reason for slip progression is that the bone graft does not stabilize (and may actually destabilize) the proximal femur as well as does a screw. Another potential cause of progressive slip in these patients is the delayed or incomplete physeal closure.

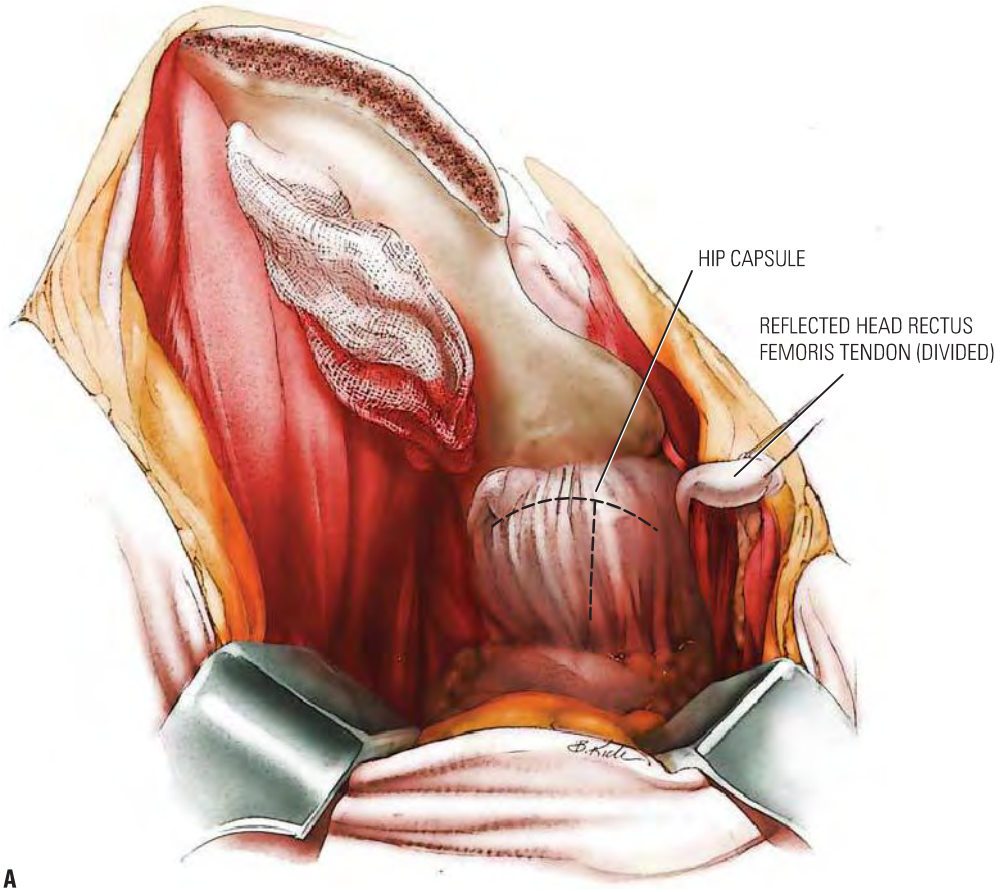
Femoral neck fracture has also been reported in 0% to 5% of cases following bone peg epiphysiodesis (186, 187, 195). Schmidt et al. (186) reported 2 proximal femoral fractures in a series of 40 bone peg epiphysiodeses (5%), in striking contrast to Adamczyk et al. (187), who reported no fractures in a series of 318 bone peg epiphysiodeses.

Bone graft epiphysiodesis does not have significant advantages relative to *in situ* fixation of SCFE, although there are significant drawbacks to its use. Children treated with bone graft epiphysiodesis have greater blood loss, increased donor site morbidity, increased risk of nerve palsy, increased risk of slip progression, and are not allowed to bear weight as early as do those

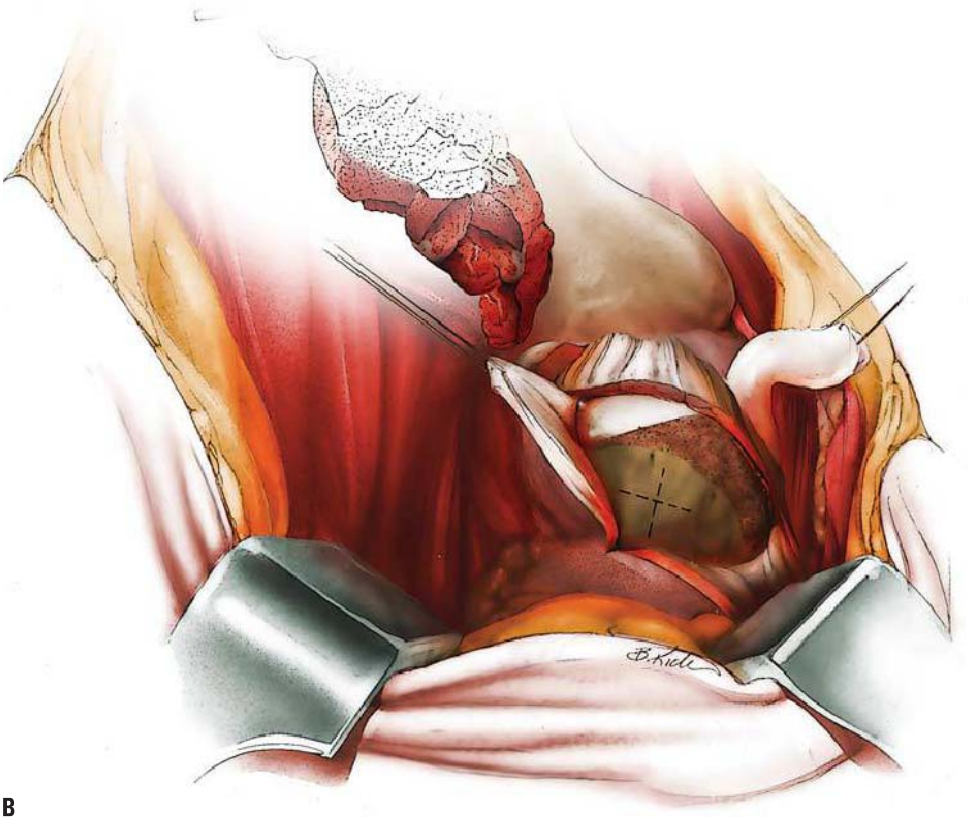
Text continued on page 1178

Technique for Bone Graft Epiphysiodesis for Treatment of SCFE (Fig. 25-5A–E)

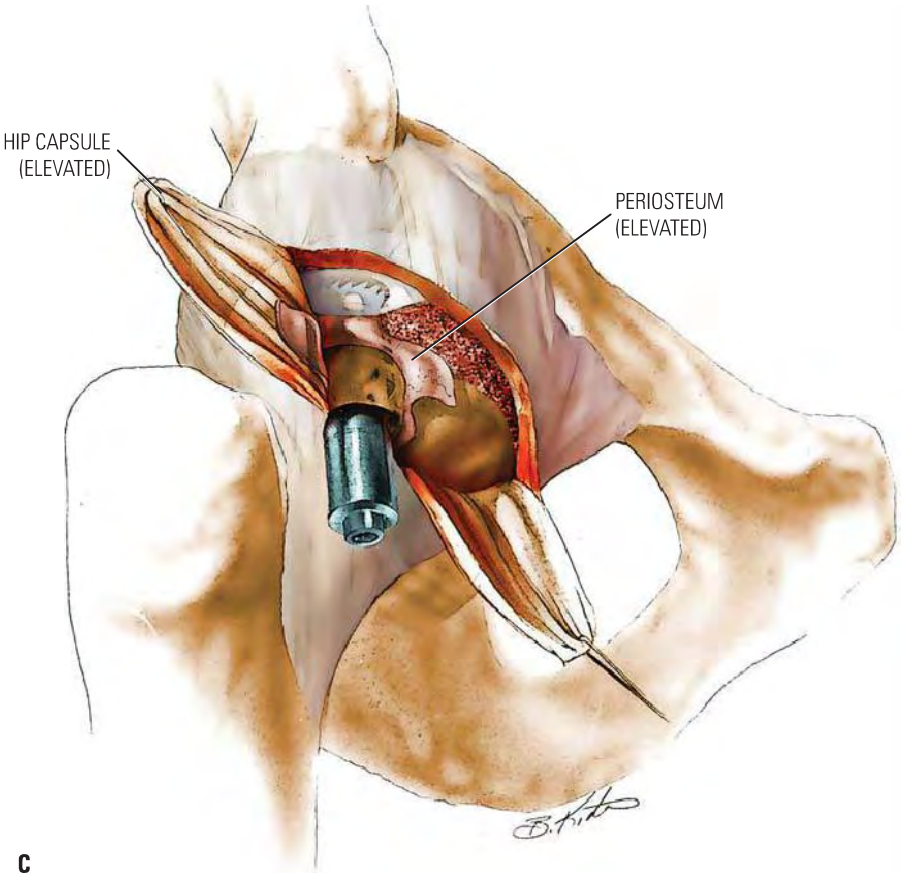
FIGURE 25-5. Technique for Bone Graft Epiphysiodesis for Treatment of SCFE. A: The capsule of the hip joint and the outer table of the ilium are exposed as for a Salter osteotomy or an anterior open reduction of the hip. It is not necessary to expose the inner table of the ilium, but it is important to obtain good exposure of the hip capsule. The anterior capsule is opened with an incision parallel to and 1 cm from the acetabular margin. The second incision extends at right angles from this incision over the anterior femoral neck. **B:** The hip joint is now exposed. Inspection shows the articular surface of the femoral head to be displaced posteriorly, with the amount of displacement depending on the severity of the slip. The capsule can be slightly adherent to the anterior femoral neck as a result of the healing callus and inflammation. The surgeon should see an adequate amount of the femoral neck and the articular surface of the femoral head to ensure that he or she is properly oriented to the anatomy. The periosteum over the anterior neck is incised in a cruciate fashion and elevated, exposing the bone. This should be placed in a location that allows the hollow mill drill to cross perpendicular to the epiphyseal plate.



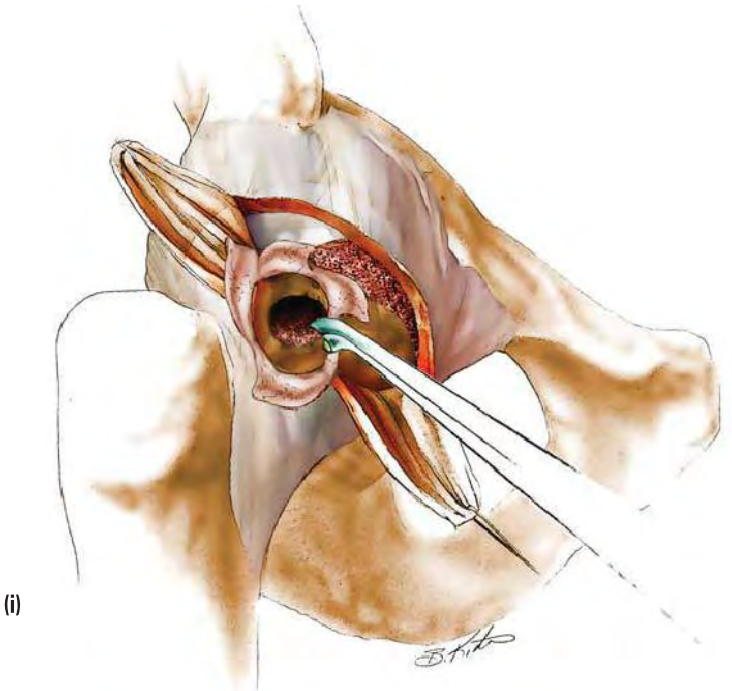
A



B



C

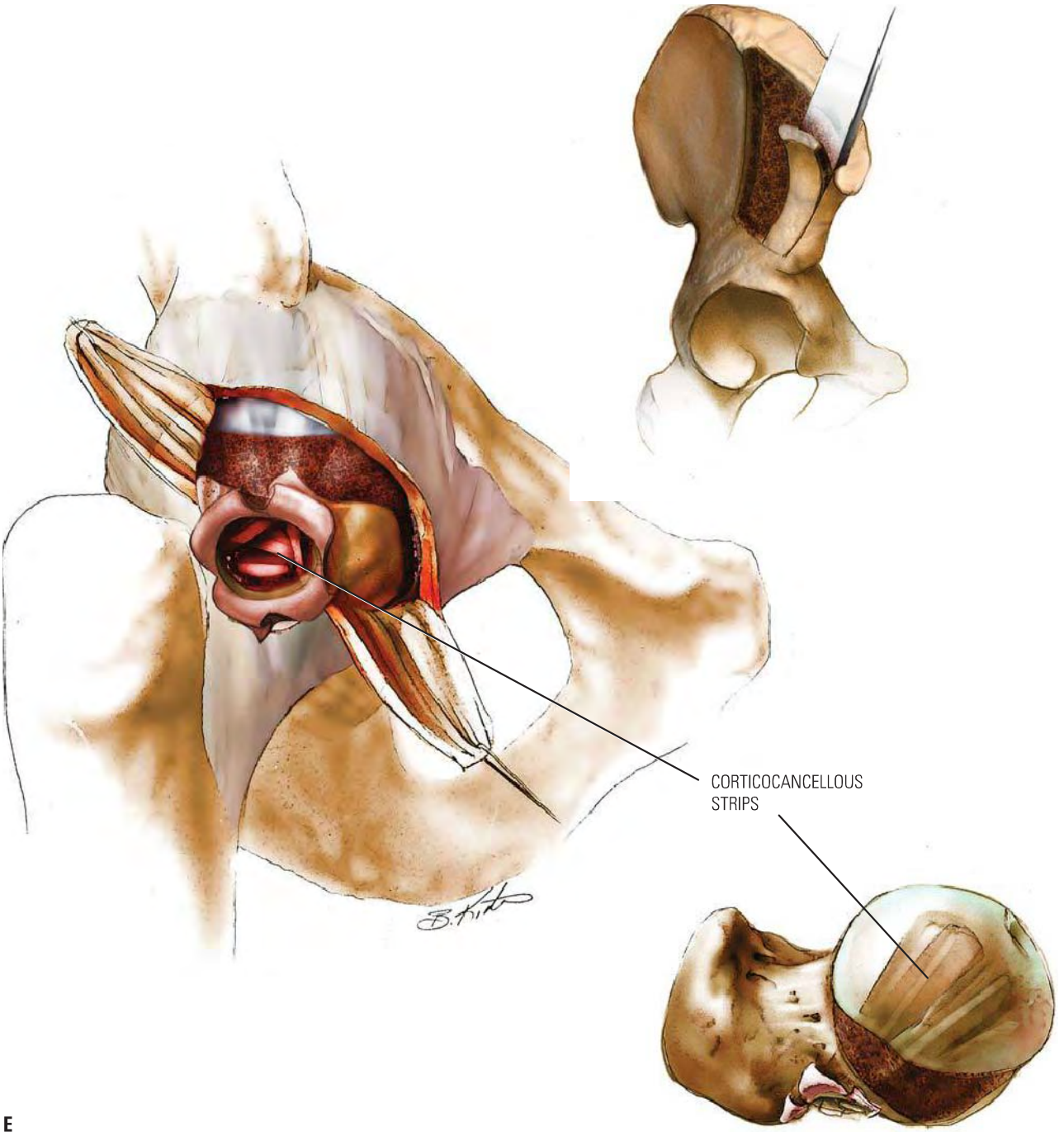


(i)

(ii) (iii)

D

FIGURE 25-5. (*continued*) **C:** A guide wire is inserted, and its proper placement in the center of the femoral head and at a safe distance from the articular surface is verified by an anteroposterior and frog lateral view on the image intensifier. When the proper direction is verified, the hollow mill is drilled through the anterior cortex of the femoral neck, across the physeal plate, and into the femoral head. **D:** The hole in the cortex can be enlarged with a curette, which also can be used to remove additional physeal plate (*i*). The hollow mill is angled in multiple directions to enlarge the hole (*ii,iii*). This allows the placement of grafts to provide sufficient strength for temporary stability to the epiphysis.



E

FIGURE 25-5. (*continued*) **E:** Three or four corticocancellous strips of bone are removed from the outer table of the ilium. This is preferable to several small matchstick-sized pieces because the larger grafts possess more strength. These grafts are driven into the hole, and their location is verified on the image intensifier. The periosteum, the capsule, and the wound are closed.

children undergoing *in situ* fixation. Bone graft epiphysiodesis does not appear to have a significant role in the treatment of SCFE at this time.

Postoperative Care. A drain can be used at the discretion of the surgeon, but there should be no dead space and little bleeding at the conclusion of the procedure. Those series that reported no further slipping after grafting used a spica cast for immobilization until healing was complete in 8 to 12 weeks. There have been reports of using only crutch protection, not a cast, with an incidence of further slipping in some patients (193).

Current Methods

***In Situ* Fixation.** The goal of *in situ* fixation of SCFE is to prevent slip progression. *In situ* fixation is currently the

preferred initial treatment for most cases of SCFE, both stable and unstable, although the outcome of such treatment differs depending on the slip stability and severity.

Over a period of more than 50 years of *in situ* fixation for SCFE, surgical techniques, implants, and imaging techniques have evolved significantly (200). Early fixation was with large nail-type devices, followed by pin fixation, which have since been replaced by cannulated screw systems in most centers. Because of the wide availability of fluoroscopic imaging, the ability to optimally position the fixation devices has improved as well. Cannulated screw systems now allow these procedures to be performed percutaneously.

The surgery may be performed on either a fracture table or a radiolucent table (200, 201). Use of a fracture table allows a true lateral radiograph to be obtained, although the quality of such images in obese patients is often suboptimal and this setup requires the presence of a technician to rotate the fluoroscope. In contrast, with the patient on a radiolucent table, a technician is not needed, as the fluoroscope may be left in one position and it is easy to obtain a higher quality frog lateral radiograph; however, a true lateral can only be obtained by moving the patient. In addition, the guide wire for percutaneous fixation may be bent as the hip is rotated. For an unstable SCFE, the radiolucent table may be preferable in order to limit traction that may forcefully reduce the femoral head.

Understanding the three-dimensional pathoanatomy of the SCFE is essential for understanding how to position the hardware optimally and minimize complications. As noted previously, the proximal femoral neck and shaft migrate anteriorly and rotate externally in most SCFE. As a result, a greater portion of the femoral head is located posterior to the femoral neck as the SCFE progresses. In very severe cases of SCFE, the entire femoral head is posterior to the femoral neck.

When placing the *in situ* pin, the goals are to stabilize the physis with minimal hardware, avoid ON by avoiding pin penetration of the posterior femoral neck and pin placement in the anterior-superior head, avoid chondrolysis by avoiding pin penetration into the joint, and finally avoid screw impingement by avoiding intra-articular placement of screw entry point. Because of the direction of the slip, fixation should be inserted from the anterior femoral neck in most cases in order to allow fixation perpendicular to the physis and to prevent hardware penetration through the posterior femoral neck (144, 202) (Fig. 25-6). However, in very severe cases, the hardware may need to be inserted in a directly anterior-to-posterior direction (Fig. 25-7), which may cause screw head impingement and articular damage. When possible, the screw insertion point should be lateral to the intertrochanteric line even though the screw may not be perpendicular to the physis (203). However, insertion of hardware from the far lateral cortex (as is done in the pinning of adult hip fractures) will generally result in one or more of the following problems: poor biomechanical alignment of the hardware (very oblique rather than perpendicular to the physis), purchase of the hardware in only a small portion of the femoral head, joint penetration, hardware exiting the posterior femoral neck before entering the femoral head, and creation of

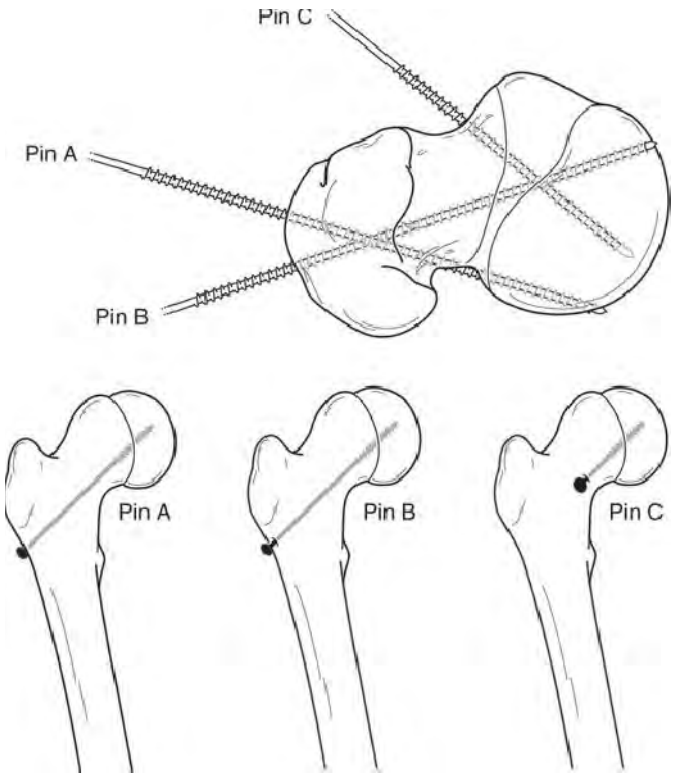


FIGURE 25-6. Two common problems associated with lateral-entry pins (pins *A* and *B*) in SCFE are contrasted with correct pin positioning (pin *C*) using an anterior entry point. **Top:** Because of their lateral starting points, both pins A and B are eccentric in the femoral head and oblique to the physis. In addition, pin *A* is shown exiting the posterior femoral neck before entering the epiphysis. **Bottom:** How pins *A, B,* and *C* will look on an anteroposterior radiograph, and how a potential blind spot exists in which a protruding screw may be missed radiographically. This reinforces the importance of imaging a pinned hip as the hip is rotated through a complete range of motion.

stress risers on the tension side of the proximal femur. Common sequelae with a lateral starting point are that the hardware either entirely misses or engages only a small portion of the anterior femoral head, and that such hardware also often penetrates the joint surface. If the hardware exits the posterior femoral neck before entering the femoral head, as has been reported in up to 6% of cases (103, 204), the extraosseous blood supply to the femoral head are at risk, thereby increasing the risk of ON.

Ideally, the fixation device should be located in the center of the proximal femoral epiphysis on both the anteroposterior and lateral views and should be perpendicular to the physis in both views as well (205, 206). This so-called center–center position minimizes pin penetration into the joint and provides optimal fixation of the physis (68, 207–209). Additionally, it minimizes superior or posterior placement of the screw, which may place at risk the intraosseous blood supply within the head. One of the significant difficulties in pinning SCFE is the three-dimensional interpretation of intraoperative radiographic images. Walters and Simon (207) alerted the orthopaedic community to the risk of unrecognized pin penetration in cases of SCFE treated with *in situ* fixation, and the associated risk of chondrolysis. They

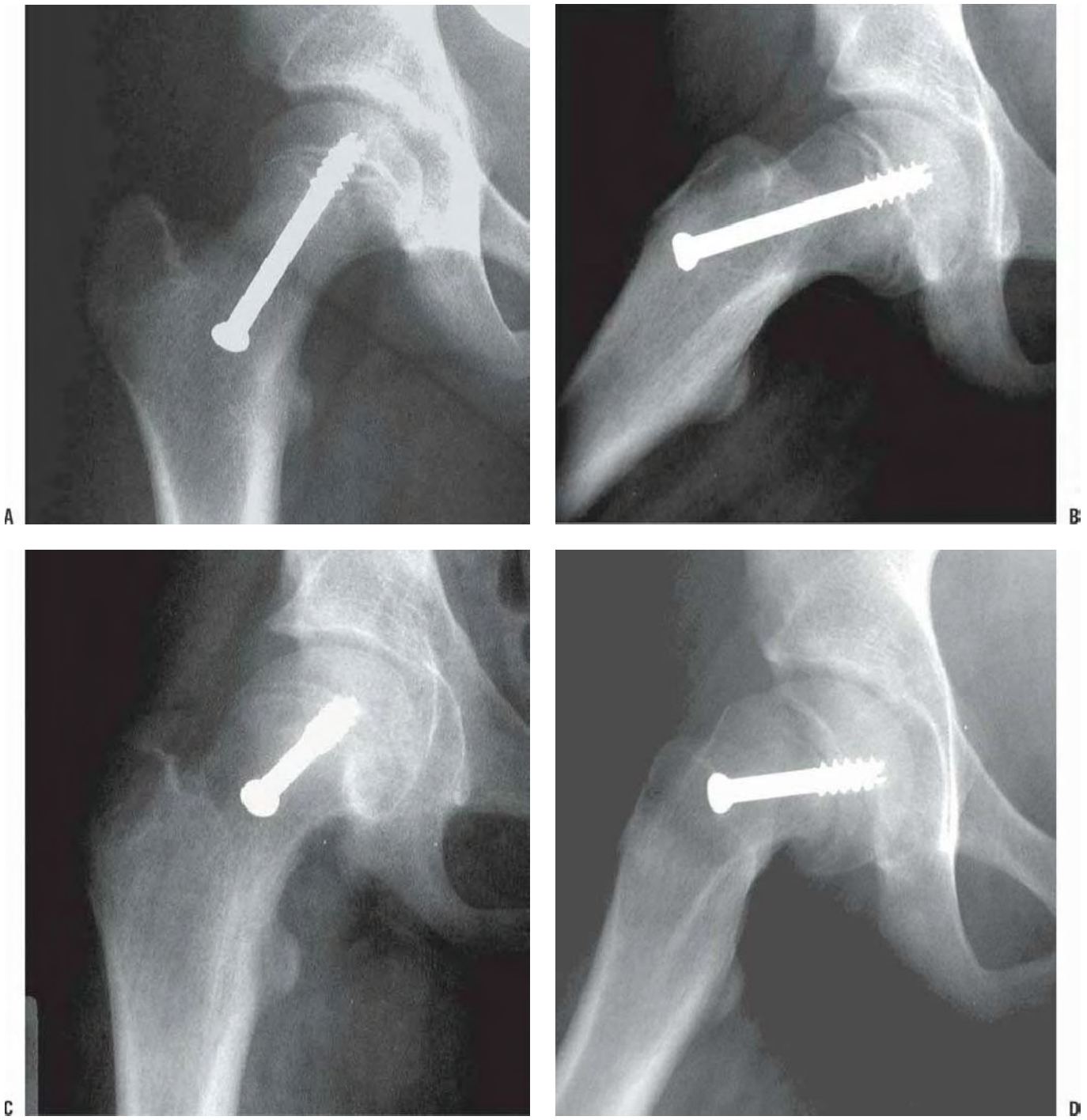


FIGURE 25-7. Proper screw locations in slips of varying severity in three different cases: **(A,B), (C,D)**, and **(E,F)**. In all three cases, the screws enter the anterior femoral neck, are perpendicular to the physis, and are located in the center of the femoral head. The starting point is more proximal and the screw is angled progressively more posteriorly as the magnitude of slip progresses from least (A,B) to most (E,F) severe.

demonstrated that a "blind spot" can exist radiographically, since a protruding pin may appear to be located within the femoral head on both anteroposterior and lateral views (207). Other authors have described a geometric analysis of the blind spot, although this technique is rarely used (210).

In practice, the operative hip is taken through a full range of motion while using fluoroscopy. This can be done throughout the procedure if a radiolucent table is used. If a fracture table is used, this can only be done following removal of traction on the operated leg. The "approach-withdraw phenomenon" described by Moseley (211) is when the fluoroscopic appearance of the implanted hardware approaches the subchondral bone and then moves away from it. When the hardware reaches the apex of this arc and then begins to recede,

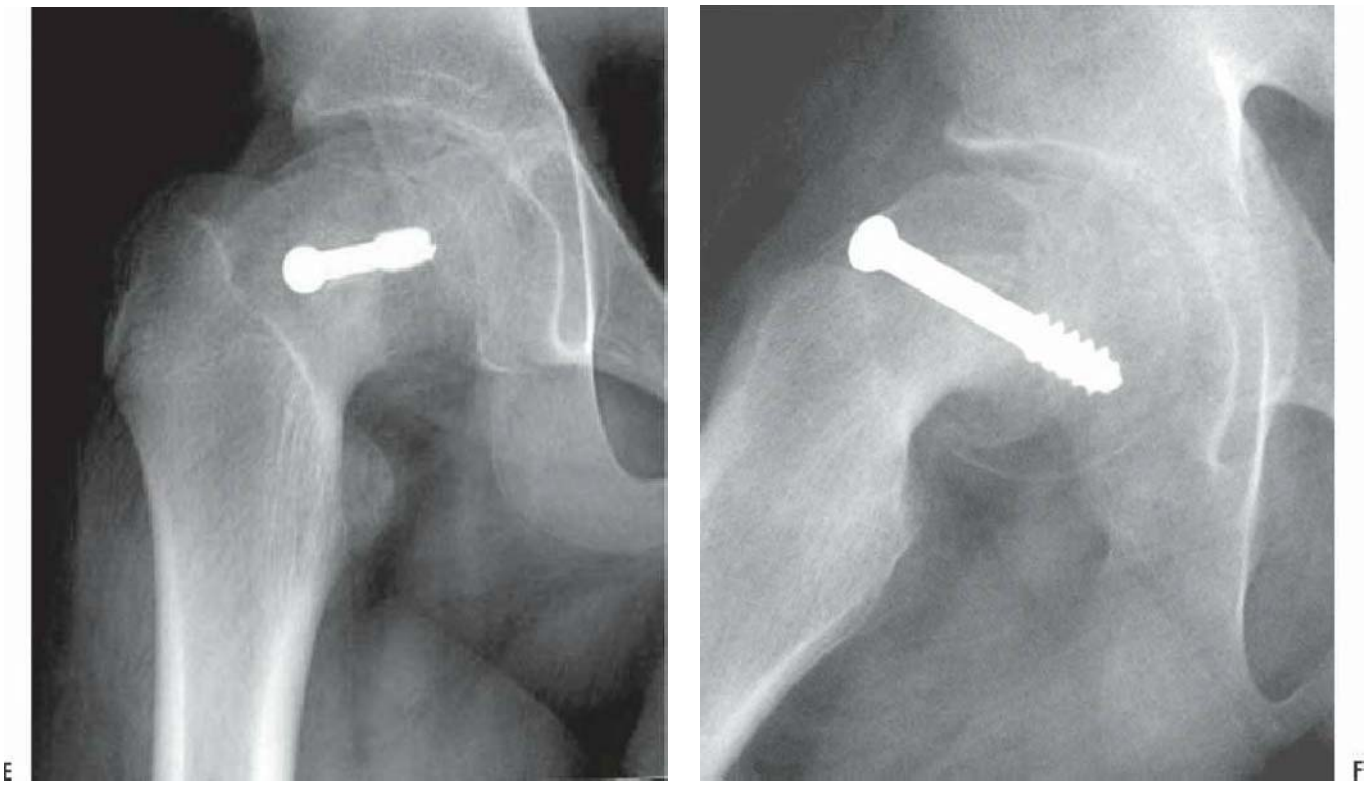


FIGURE 25-7. (*continued*)

the point of maximal proximity to the subchondral bone has been reached, and this distance should be measured. Center–center pins are left 5 to 6 mm from the subchondral bone (corrected for magnification), while other pins are left 10 mm from the subchondral bone (207). Poor hardware position has been noted to correlate with poor clinical outcomes (202, 212).

Injection of arthrographic dye through the hardware under fluoroscopic control and bone endoscopy are two ways that have been reported for checking for pin penetration when high-quality radiographic images cannot be obtained intraoperatively (213, 214). With current fluoroscopy machines, neither of these techniques are used on a regular basis. In addition, each of these techniques has the potential risk of flushing bone chips into the hip joint. If radiographic imaging is deemed insufficient intraoperatively, then a hip arthrogram through a standard anterior approach may be performed to better ascertain the relation of the hardware to the femoral head.

The pathoanatomy of SCFE markedly limits the amount of space in the femoral head and neck for appropriate hardware positioning. Multiple clinical studies have confirmed increasing rates of pin penetration and complications with an increasing number of implants (164, 204, 209, 212, 215–218). In 1984, Lehman et al. (219) reported a 37% incidence of unrecognized pin penetration in cases of SCFE undergoing treatment with implants and noted that some areas of the head may not be well visualized fluoroscopically. In a study of SCFE fixed with multiple pins or screws, Riley et al. (220) reported hardware-related complications in 26% of the treated hips, which included pin penetration in 14% (Fig. 25-8).

The biomechanical properties of various fixation techniques have been studied. Four previous biomechanical studies of acute physeal disruptions in animal femora stripped of soft-tissue attachments have demonstrated an increased rigidity for two-pin or two-screw constructs compared to those using only one comparable fixation device (221–224), and another found no statistically significant difference in resistance to creep in between single- and double-screw constructs in bovine femora (223). The authors of the two bovine studies stated that the biomechanical advantages of two-screw constructs were insufficient to justify the increased risk of pin penetration when two screws are used instead of one (221, 223). Snyder et al. (224) noted enhanced stability with two screws (compared to one screw) in a porcine model and suggested that fixation with two screws be considered in unstable SCFE. One additional study using bovine femora with acutely created physeal disruptions indicated that compression across the physis may be obtained if screw threads do not cross the physis, although there was no significant difference in the ultimate strength or the energy absorbed or in the degree of failure as compared to the results with a standard screw (225). In immature porcine femora stripped of soft tissues, partial and fully threaded screws have been noted to provide comparable physeal stability *in vitro* (226). Because all these studies involve acute physeal disruptions, their applicability to stable SCFE is limited. Their applicability to even acute SCFE in humans is unclear as well. Hence, for most SCFE, a single well-placed cannulated screw is recommended (Fig. 25-9)

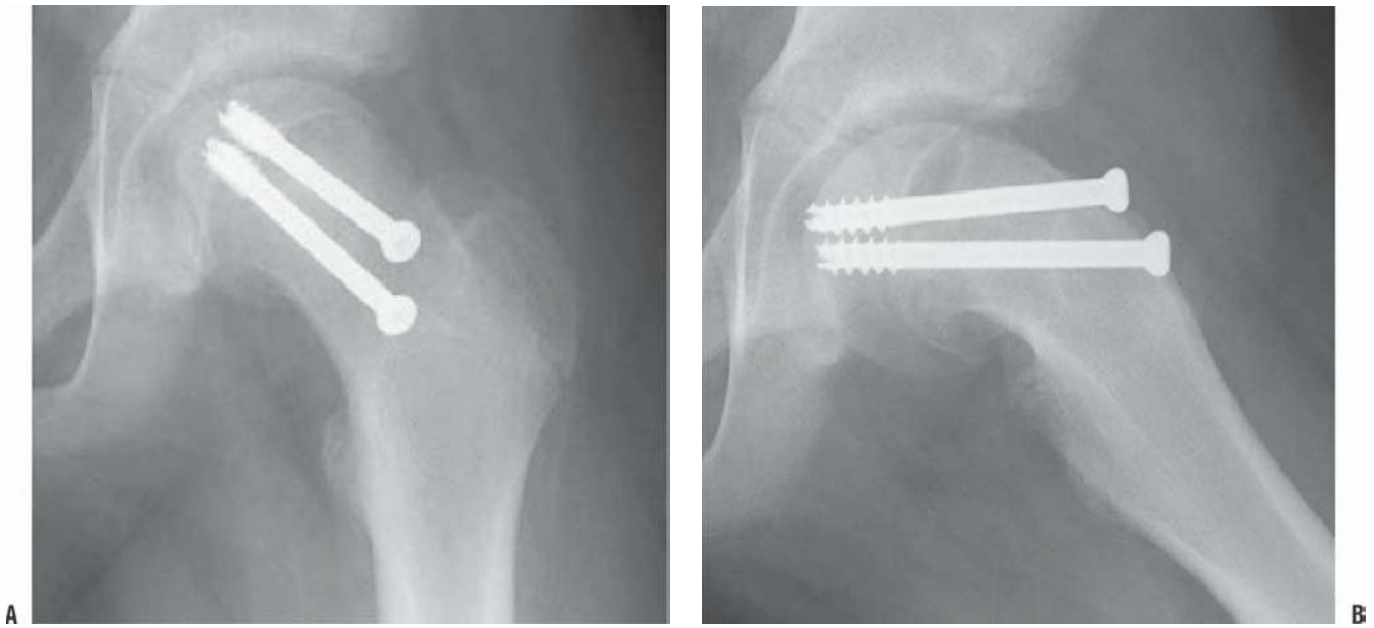


FIGURE 25-8. An 11.5-year-old boy presented with hip pain 1 month following *in situ* fixation of a stable SCFE. Anteroposterior radiograph **(A)** demonstrates what appears to be adequate alignment of the hardware, although the frog lateral view **(B)** is suggestive of pin penetration. The proximity of the hardware to the joint surface had not been recognized at the time of surgery, and demonstrates the importance of leaving the pin at least 5 mm from subchondral bone, even if the hip is imaged through a range of motion at the time of surgery. This case also illustrates that only one implant can be in both a center–center position and perpendicular to the physis.

Physeal closure generally occurs within 6 to 12 months following *in situ* fixation of a SCFE (116, 164, 204, 227–229). Physeal closure occurs in the operated hip first in most cases, and simultaneous closure occurs in fewer than 10% of cases (116, 164, 204, 227–229). Prolonged time for closure has been associated with eccentric screw placement and increasing severity of the SCFE (228). Whether the rapid physeal closure is due to the SCFE itself or to the fixation across the physis is not known. In a young child with unilateral SCFE, rapid unilateral physeal closure has the undesired effect of causing a potential LLD. Multiple studies carried out in Europe have touted the use of fixation devices without threads crossing the physis (including smooth wires, hook pins, and partially threaded screws) as a way to avoid physeal closure and to allow further growth of the proximal femur (230, 231). In young patients with underlying causes of SCFE, some authors have noted that epiphysiodesis may be needed in combination with *in situ* fixation (232).

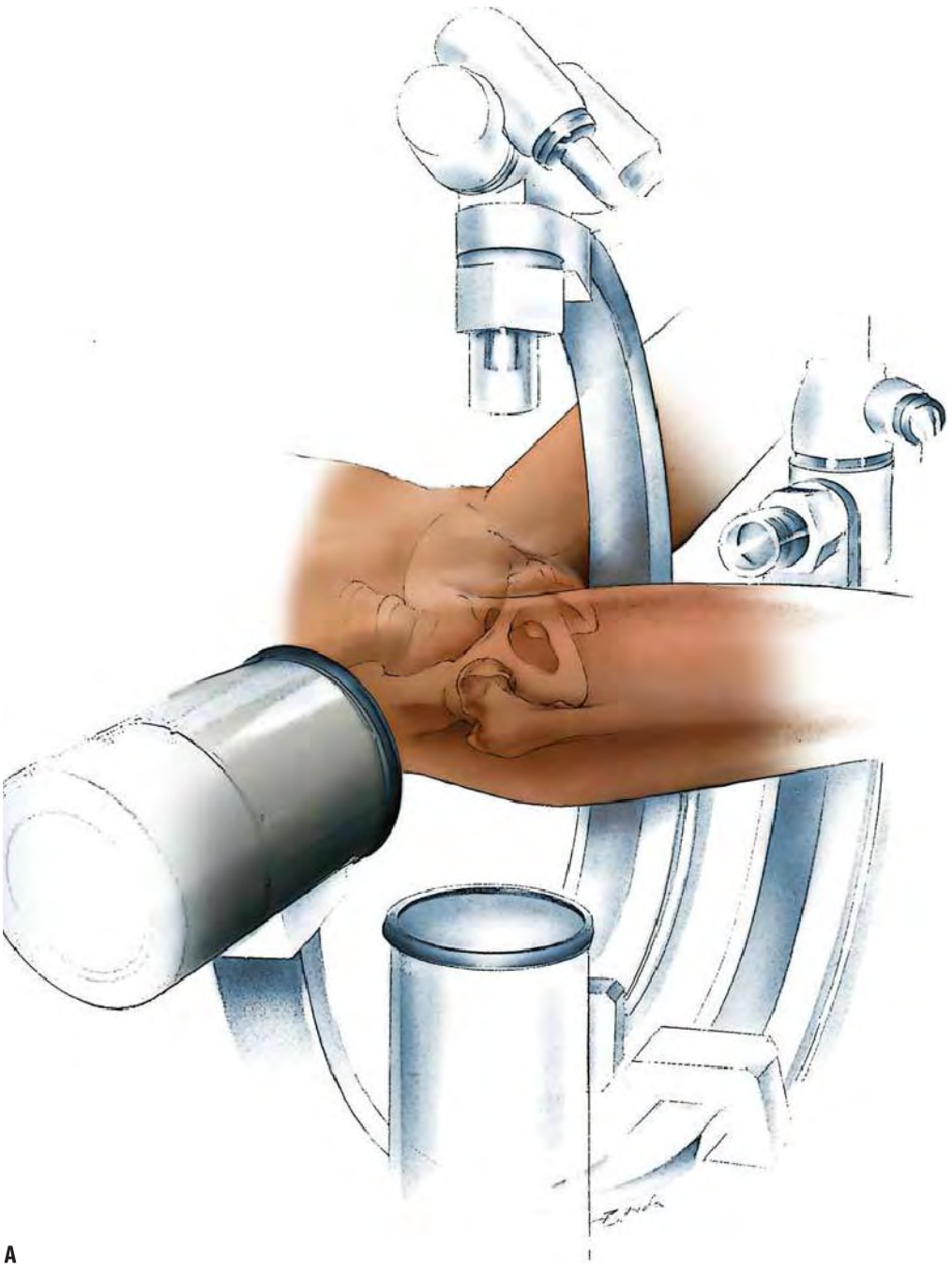
Recent studies of a combination of acute, acute-on-chronic, and chronic cases of SCFE (with 75% to 86% chronic cases of SCFE) reported good or excellent results in 90% to 95% of the patients (2, 233). Another recent series, limited to 21 hips with acute or acute-on-chronic SCFE treated with single screws, reported 95% good-to-excellent results, with no cases of ON or chondrolysis (208). In series of studies with worse results, it is seen that the results are better in milder slips than in the more severe slips (202). Aronson et al. (202) reported good or excellent results in 70% of the overall cases, with 86% good or excellent results in cases of mild SCFE, 55% in cases of moderate SCFE, and 24% in cases of severe SCFE. Poor results may be due to a variety of factors including range of motion limitation due to the residual deformity, OA, ON, or chondrolysis.

Radiographs often demonstrate a remodeling of the SCFE following *in situ* fixation (22, 29, 125, 229, 233, 234). This remodeling typically involves resorption of a portion of the prominent superior femoral neck, and has also been reported to result in changes in the proximal femoral head–neck and head–shaft angles. Studies that report proximal femoral remodeling typically report angular changes in the range of 7 to 14 degrees (29, 229, 233, 234). Remodeling is most commonly reported in more severe slips and has been reported in 68% to 83% of moderate-to-severe cases of SCFE at long-term follow-up (22, 125, 233, 234). An open triradiate cartilage has been reported to be an indicator of more potential for such remodeling (234, 235). However, some authors have even reported remodeling after proximal femoral physeal closure (209). More contemporary reports suggest some slight improvement in head-shaft angle and reduced but persistent metaphyseal prominence (236).

All of these studies on remodeling have significant limitations. One such limitation is the inherent error in radiographic measurements. Another limitation is the variability in patient positioning, especially when a painful hip with synovitis is imaged at the time of presentation and a painless hip is imaged on subsequent evaluations. Finally, significant remodeling in the slowly growing peripubertal proximal femur with a fixation device across the physis seems unlikely.

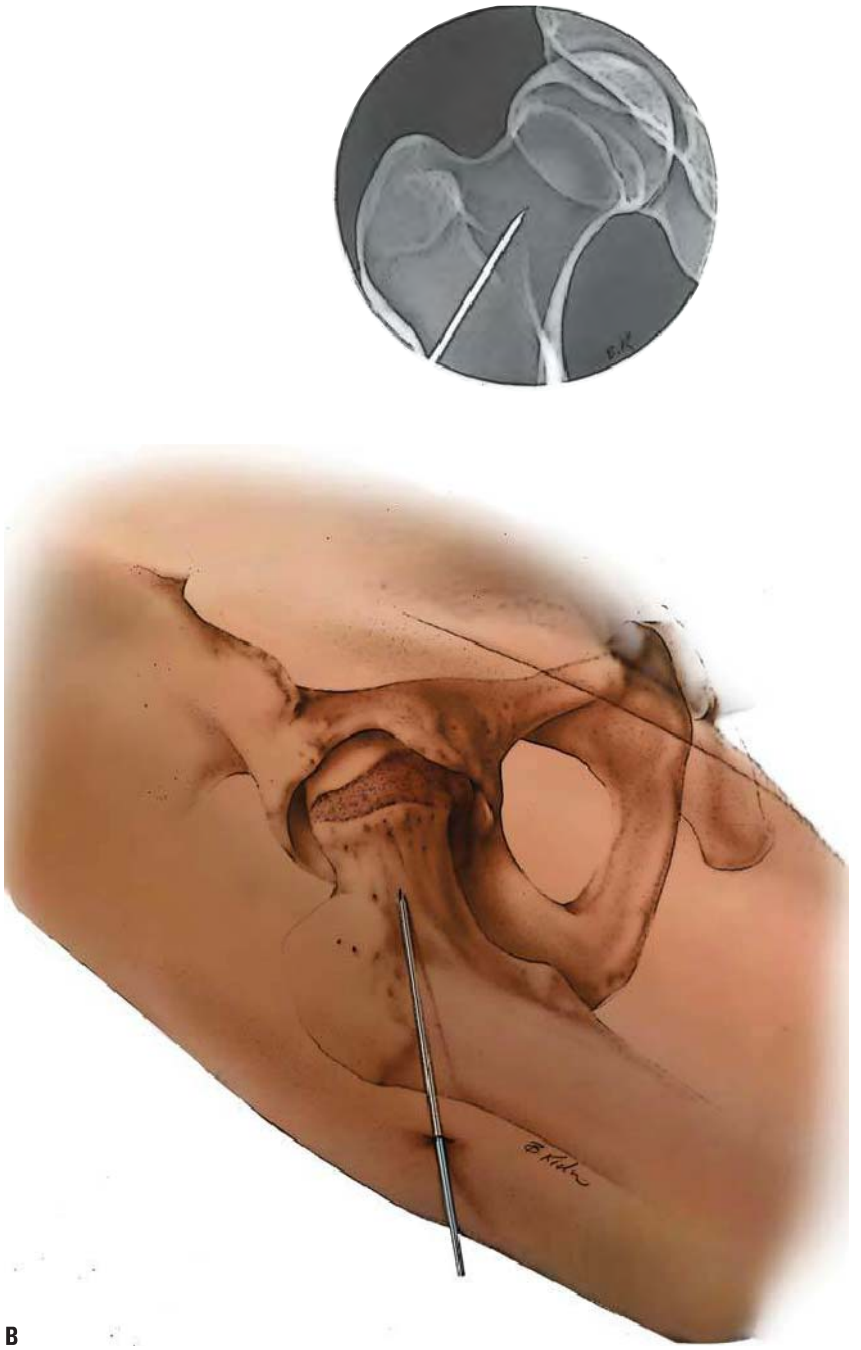
Text continued on page 1187

Technique for Percutaneous SCFE Pinning on a Fracture Table (Fig. 25-9A–F)



A

FIGURE 25-9. Technique for Percutaneous SCFE Pinning on a Fracture Table. A: The patient is placed on a fracture table with the image intensifier between the legs.



B

FIGURE 25-9. (*continued*) **B:** The exact location and angle of the femoral neck are identified by laying a guide pin over the femoral neck on the anterior thigh under radiographic control. This permits more accurate placement of the percutaneous guide wire. At this point, the percutaneous guide wire is inserted through a stab wound in the skin down to the bone of the femoral neck and is drilled into the bone for a short distance under radiographic control. Now the surgeon is certain that the guide pin and the screw are heading in the correct direction as they enter the femoral head in the anteroposterior projection. The surgeon does not know, however, whether the guide pin is headed too far anteriorly or posteriorly. This determination is best left until the next step because this guide wire is not fixed in the bone and tends to move. After a little experience and a better understanding of the anatomy of a SCFE, it becomes surprisingly easy to judge this posterior inclination of the percutaneous guide pin and the screw correctly.

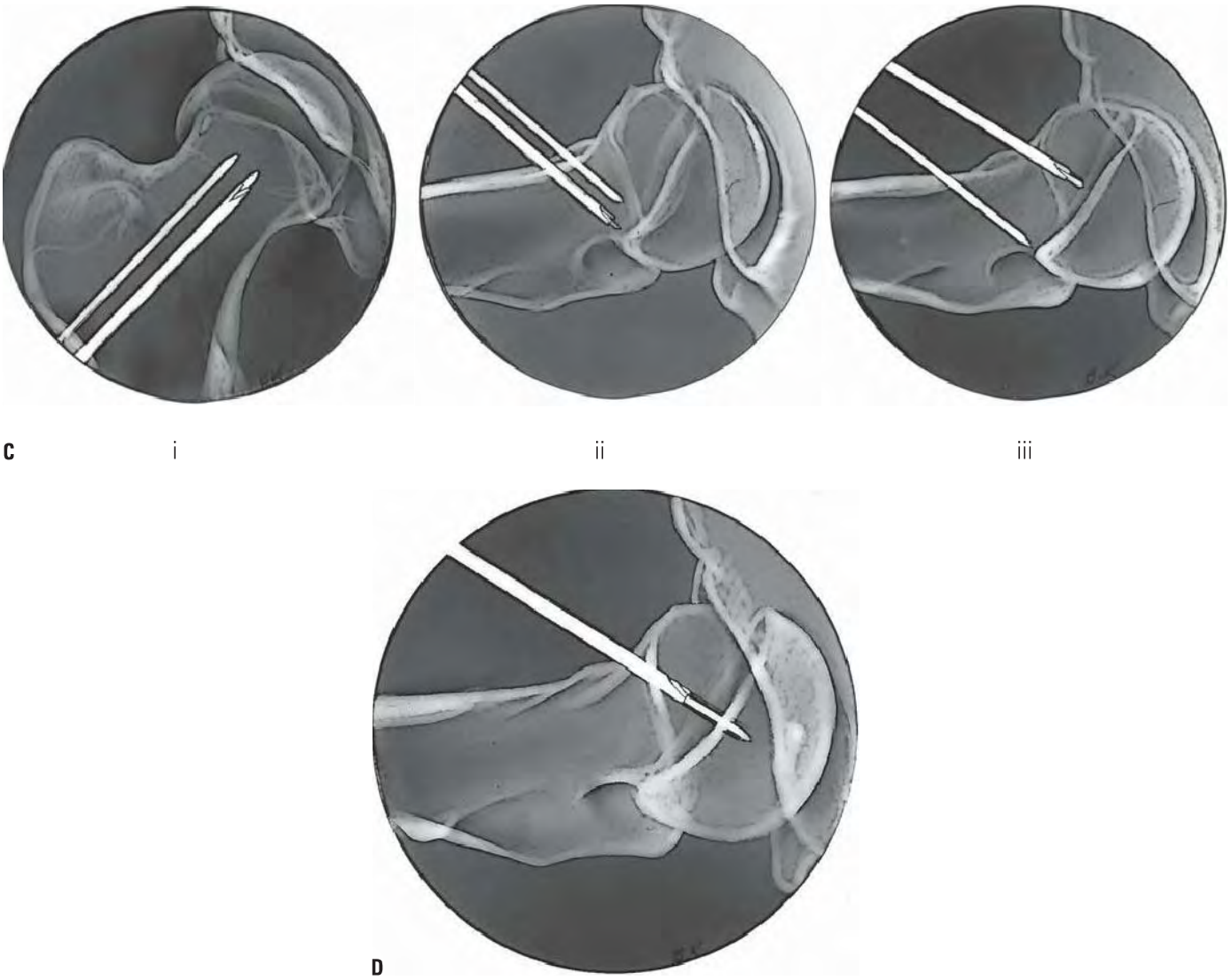
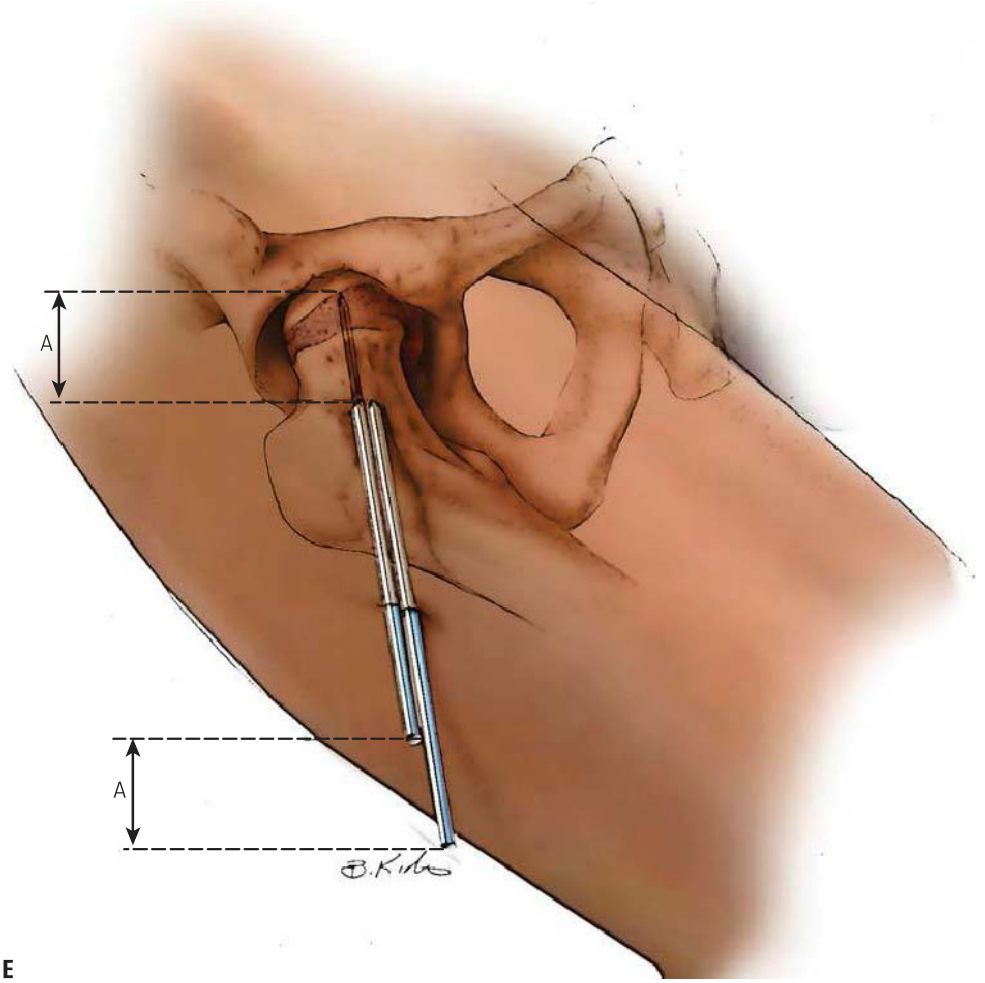
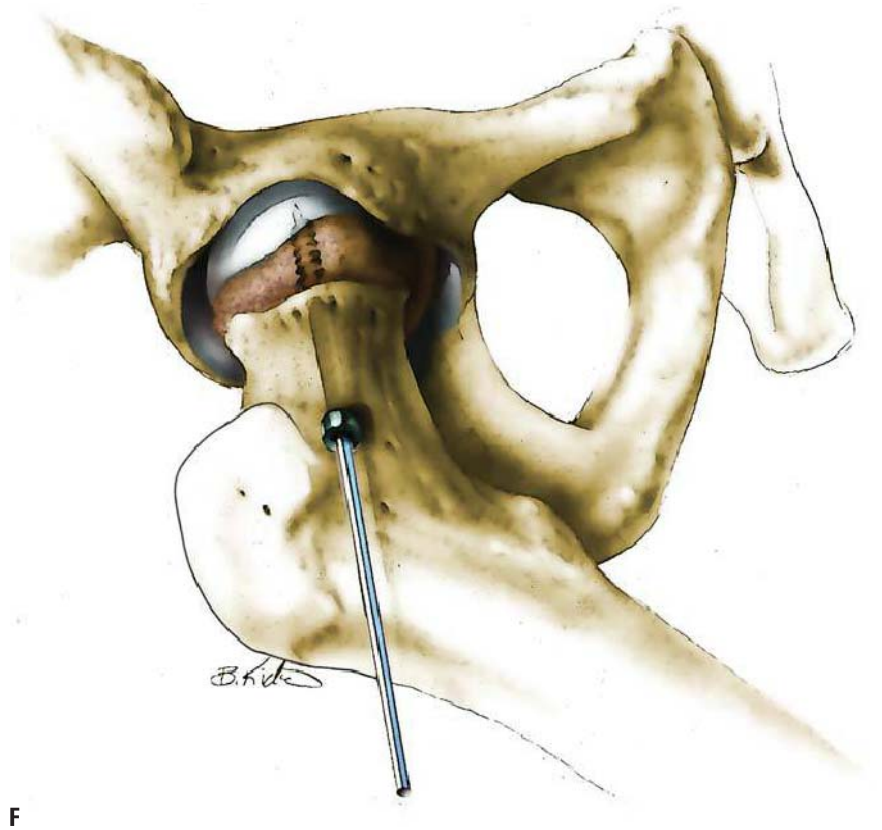


FIGURE 25-9. (*continued*) **C:** The guide pin is fixed in the bone and does not move. The image intensifier is switched to the lateral projection to see how the pin is directed in the anteroposterior plane. If the direction is incorrect, a second pin parallel to the first pin on the anteroposterior projection is inserted, this time with the correct anteroposterior inclination. It is imperative that the direction of this second pin be verified on both projections, and the surgeon must not assume that the pin has been placed perfectly parallel on the anteroposterior projection. A few degrees of change at the starting point results in the tip of the pin being in a different location by the time it is in the femoral head. (i) The guide pin is parallel and just inferior to the guide pin on the skin surface. It is well placed in this projection. (ii) Two different pins are shown that are not correct on the lateral projection. They are correct in that the inclination places them perpendicular to the surface of the physis of the femoral head, but they are starting too far laterally on the femoral neck. (iii) One of the pins is redirected in the correct direction. After the pin is in the bone, its progress should be monitored on the lateral view because this view shows the correct depth of penetration. The guide pin should advance easily until the cortical bone or the epiphyseal plate is encountered. As the guide pin reaches the physeal plate, drilling becomes more difficult. Before the surgeon drills the guide pin across the physeal plate and into the femoral head, he or she must be confident that the pin is headed for the center of the femoral neck to avoid damaging the lateral epiphyseal vessels in the superior quadrant of the femoral head. **D:** When the proper placement and direction of the guide pin are achieved, it is drilled across the physeal plate. This should be monitored on the lateral view. The anteroposterior view does not accurately portray the true depth of the guide pin and screw because they are not perpendicular to the x-ray beam. When the tip of the guide pin is in the desired location (the center of the femoral head and 5 mm from the subchondral bone), its length can be measured. Now the pin should be advanced about 5 mm so that the threads engage in the subchondral bone. The cannulated drill is then used to drill over the pin. The drill should stop short of the end of the pin so that it does not loosen in the bone and come out with the drill. As the drill is removed (still turning in the same direction used for its insertion), the image intensifier must be checked to ensure that the guide pin remains in place. If it does not, a second guide pin is inserted through the drill to push it back into the femoral head.

FIGURE 25-9. (*continued*) **E:** Although the length of the guide pin (and therefore the desired length of the screw) can be measured with the device that is provided, it is sometimes difficult with this percutaneous approach to ensure that this device is in close contact with the bone, a condition that is required for accurate measurement. If such a device is used, its contact with the bone should be verified radiographically. **F:** The correct length of screw is inserted over the guide wire, and its position is monitored on the lateral view. It is comforting to check for screw penetration before leaving the operating room. The leg is removed from traction and put through a range of motion to confirm under the image intensifier that the screw does not penetrate the joint. It is important to recognize that this method of seeking screw penetration has a significant limitation. Unless the screw can be aligned perpendicular to the plane of the x-ray beam, it will not be completely accurate. This can be difficult to achieve because of the lack of internal rotation in the hip with a SCFE.



E



F

Marked improvement in the dynamic and static measures of hip motion has been noted postoperatively, especially in the first 6 months (29, 237). Siegel et al. (29) reported such rapid improvement prior to significant remodeling, even in hips with severe deformity. At 2-year follow-up, by which time the average slip angle had decreased from 44 to 30 degrees, mean hip flexion had improved by 22 degrees (to 118 degrees), hip abduction by 11 degrees (to 40 degrees) and hip internal rotation in flexion by 19 degrees (to 11 degrees) (29). Other authors have noted similar improvement in the range of hip motion postoperatively, with improvements of 31 degrees for hip flexion, 25 degrees for internal rotation, 19 degrees for external rotation, and 21 degrees for abduction (212). However, a decreased range of motion was still noted relative to the unaffected hip in 40% of the patients, with flexion decreased by 15 degrees, internal rotation decreased by 17 degrees, and external rotation decreased by 10 degrees in this same study (212). O'Brien and Fahey noted painless hips in 83% (10 of 12) of moderate-to-severe cases of SCFE 2 to 17 years following *in situ* pinning, with 7 of these 10 hips having "essentially normal" motion except for a loss of 5 to 20 degrees of internal rotation (234).

In a long-term study (mean follow-up: 32.7 years) in cases of SCFE hips without OA, there were no significant differences between the range of motion of normal hips and those that had not been treated for SCFE or those treated with *in situ* fixation (238). The only significant loss of range was the loss of external rotation of hips treated previously with osteotomy. The hips without treatment (slip angle: 18.8 degrees) or treated with *in situ* fixation (slip angle: 25.4 degrees) had markedly lower slip angles than did those with osteotomy (slip angle: 73.7 degrees). Although this study has obvious selection bias, it does demonstrate that in cases of hips without OA, there is no inexorable loss of motion in mild SCFE.

Numerous studies have repeatedly reported lower rates of ON with stable SCFE than with unstable SCFE. Many series of studies have reported 0% ON in stable SCFE, with the rates of ON in unstable SCFE ranging from 12.5% to 58% (2, 103, 186, 215, 239). In a series of 55 acute cases of SCFE treated with internal fixation, Loder et al. (103) reported ON in 14 cases (25%) with a rate of 47% in unstable slips (14 of 30) and 0% in stable slips (n = 25). Dietz et al. (240) reported a 10% rate of ON in cases of acute SCFE, Dietz et al. reported a 21% incidence in unstable SCFE and 0% in stable SCFE.

Chondrolysis is another potentially severe complication. Aprin et al. (241) have demonstrated that pin penetration in rabbits can lead to chondrolysis, and that the severity of chondrolysis is related to the duration of pin penetration. In another rabbit study, Sternlicht et al. (242) demonstrated that pin protrusion caused mechanical destruction of the cartilage and loss of proteoglycans in the articular cartilage, but did not result in decreased joint space.

Chondrolysis following *in situ* pinning varies from 0% to 9% in most series and appears to be due to unrecognized intraoperative pin penetration (2, 24, 30, 125, 181, 202, 204, 208, 212, 216, 220, 228, 243). Multiple studies, each with more than 50 cases of SCFE treated using current fixation techniques with a single screw, have reported no cases of chondrolysis (2, 228, 244). Rates of chondrolysis appear to be higher when multiple fixation devices are used because of the increased risk of unrecognized pin penetration with the use of multiple fixation devices (204). Pin penetration with single cannulated screws appears to be quite low. Ward et al. (228) reported pin penetration in 1.7% (1 of 59 hips) fixed with one screw, and others (208) have reported a 0% rate. Several studies have reported that if pin penetration is recognized at the time of surgery and the protruding pin is removed, there does not appear to be an increased risk of chondrolysis or other complication (245, 246).

It is important to note that there are many cases of unrecognized pin penetration in the treatment of SCFE that does not result in chondrolysis (204, 243). Previous authors have reported chondrolysis to occur in 11% to 51% of the cases with unrecognized pin penetration. In one study with pin penetrations reported in 28 cases, chondrolysis resulted in only 3 of these 28 hips (11%) (204). The location of pin penetration is important (212), with less apparent risk if the penetration occurs in the inferior head or fovea.

Slip progression can occur following *in situ* fixation if the progressive growth of the proximal femur results in loss of fixation across the physis, or if a properly located screw loses fixation. Slip progression following *in situ* pinning has been reported in 0% to 3% of the cases in most series (2, 103, 181, 228, 247–249). Carney et al. (250) reported a rate of 20%, though this high rate is likely due to femoral neck resorption and changes in patient positioning rather than to true slip progression. In another series, the proximal femur was noted to grow off 29% of hips fixed with Steinmann pins, 18% of hips fixed with Knowles pins, and 0% of hips fixed with cannulated screws (251). Growing off a screw appears much less common than growing off wires (244, 252). Previous authors have also noted the risk of progressive slip if hardware is removed prior to physeal closure (253).

Jerre et al. (252) noted slip progression in 1.5% of hips (3 of 202) without any evident cause, and progression in an additional 5% of the hips after the fixation device(s) no longer engaged the epiphysis. By far the highest rate of progression following *in situ* fixation was recently reported by Carney et al. (250), who found progression of SCFE in 20% of hips following *in situ* fixation with a single screw. In a series of seven progressive slips with appropriate hardware positioning, fixation in the epiphysis remained good, but metaphyseal loosening with "windshield wipering" was noted in each case (249).

Proximal femoral fracture is a rare, although potentially disastrous, complication associated with *in situ* pinning of SCFE, occurring in 0% to 2% of the cases (28, 254–256). Such fractures often follow relatively minor trauma. Many reports have focused on subtrochanteric fractures following insertion of the hardware from the lateral aspect of the femur, the tension side of the bone (212, 220, 244, 257). Most such

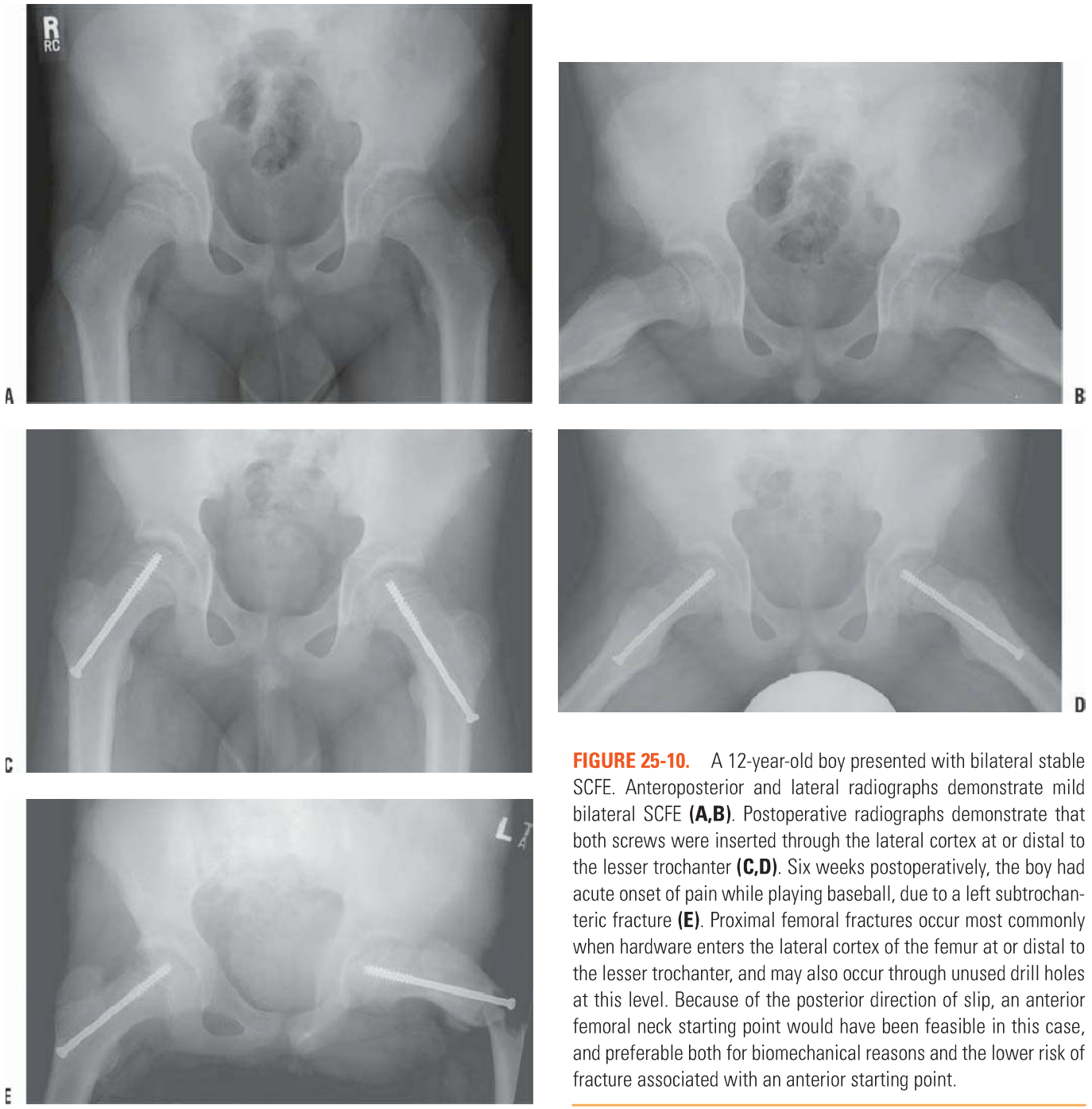


FIGURE 25-10. A 12-year-old boy presented with bilateral stable SCFE. Anteroposterior and lateral radiographs demonstrate mild bilateral SCFE **(A,B)**. Postoperative radiographs demonstrate that both screws were inserted through the lateral cortex at or distal to the lesser trochanter **(C,D)**. Six weeks postoperatively, the boy had acute onset of pain while playing baseball, due to a left subtrochanteric fracture **(E)**. Proximal femoral fractures occur most commonly when hardware enters the lateral cortex of the femur at or distal to the lesser trochanter, and may also occur through unused drill holes at this level. Because of the posterior direction of slip, an anterior femoral neck starting point would have been feasible in this case, and preferable both for biomechanical reasons and the lower risk of fracture associated with an anterior starting point.

fractures occur through the used or unused drill holes at or distal to the lesser trochanter (257, 258) (Fig. 25-10). Fracture has also been reported following hardware removal (2).

Femoral neck fractures have also been reported following appropriate placement of hardware through the anterior femoral neck (137, 259, 260). Previous reports have focused on the importance of minimizing the number of drill holes (and, therefore, stress risers) in the proximal femur. Local bone death due to the high temperatures associated with reaming through dense bone has been suggested as a possible etiology in some cases as well (259). Stress fracture of the femoral neck has also been reported (244). It appears that the way to minimize the risk of proximal femoral fractures is to use an anterior starting point in the femoral neck and to avoid drilling into the proximal femur until the precise insertion site is localized.

Stambough et al. (212) reported LLD of at least 1 cm in 14%, and of at least 2 cm in 5%, of patients treated with *in situ* pinning. Chen et al. (261) reported LLD of at least 1 cm in 6 of 10 patients (60%) treated with *in situ* pinning. To prevent significant LLD in children with unilateral SCFE, prophylactic pinning of the contralateral hip should be considered for children younger than 10 years at presentation. If a projected LLD is the only concern, then an alternative would be to perform a contralateral distal femoral epiphysiodesis at a later stage.

Complications of hardware removal have been reported in 19% to 53% of hips with implants (233, 262–264). Complications of hardware removal include hardware breakage, inability to retrieve the hardware, difficulties requiring extensive bone removal, and fracture. Bellemans et al. reported inability to remove the hardware in 30% of the cases and the need for major decortication to remove hardware in 20% of the cases in the same series (233, 265, 266). Greenough et al. (265) reported two subtrochanteric fractures in a study of 57 hips following hardware removal (4%), presumably due to significant bone removal at the time of hardware removal. Crandall et al. (267) reported lower complication rates with cannulated screw removal compared to pin removal, although the screws were noted to be buried and difficult to remove in 36% of the cases. Screw breakage during attempted removal has been reported in 6% of the cases in one series (2). Removal of titanium screws has been reported to be more difficult than removal of stainless steel screws, possibly due to the significant amount of osseous integration seen with titanium screws (268).

Because of the high rate of complications with hardware removal, routine hardware removal is not recommended following *in situ* fixation in cases of SCFE. If hardware removal is necessary (e.g., for later surgery), then removal of screws with reverse-cutting threads may prove easier.

Although late OA is commonly reported after SCFE, Hagglund (269) noted that no patient who had a hip with a mild or moderate slip in childhood or adolescence and who had been treated with *in situ* pinning developed arthritis before the age of 50 years. Hansson et al. (168) reported that at 30.9 years mean follow-up, OA was seen in 22% of mild slips (30 degrees or less) and in 50% of moderate slips (30 to 50 degrees) and that Harris hip scores were at least 90 in 93% of the cases with mild slips and in 78% of the cases with moderate slips. They also noted that radiographic findings correlated with Harris hip scores, with hips with mild OA having a mean score of 96.5 and hips with severe OA having a mean score of 74.3 (168).

Long-term follow-up studies have compared the results of various treatment modalities (125, 181). These studies noted that the best long-term results were obtained with *in situ* fixation, and that SCFE reduction or realignment resulted in higher rates of complications (including ON and chondrolysis). Carney et al. (181) also noted that Iowa hip scores decreased with the increase of every decade in follow-up studies. However, even those with late OA often function relatively well into their 50s in the absence of any significant complications of the initial treatment. Carney et al. (181) stated that *in situ* fixation is the procedure of choice, regardless of slip magnitude, because of its long-term functional and radiographic outcomes and low risk of complications. Patients with all severity of SCFE did better without surgical realignment of SCFE than did those with realignment osteotomies. One flaw in this study, however, is that the surgical techniques (including fixation options) available from 1915 to 1952 are not comparable to those available currently.

Other authors cite a more guarded long-term prognosis following *in situ* fixation. Ross et al. reported good or excellent results in patients without intraoperative complications at 10- to 20-year follow-up, but fair-to-poor results in 10 of 15 hips (67%) at more than 20-year follow-up (270). One potential reason for this difference, in addition to increased duration of follow-up and potential bias in selection, is that moderate and severe slips accounted for 40% of the hips followed up for <20 years and 53% of the hips followed up for more than 20 years. Ross et al. (270) also noted that this deterioration seemed related to bilateral SCFE.

With current radiographic and surgical techniques, the complication rates following *in situ* fixation of cases with SCFE have decreased considerably. Much of this decrease is due to a reduction in the rates of ON and chondrolysis because of the recognition of the importance of proper pin or screw placement. *In situ* fixation is considered the treatment of choice for cases of SCFE of all degrees in most centers because of the relative simplicity of this extensively studied and well-documented technique (166, 181, 271, 272).

Authors' Preferred Method. One of the authors (RMK) uses cannulated screw fixation for the initial treatment of all cases of SCFE, regardless of the slip stability and degree of displacement. The other (YJK) will perform realignment in some cases at the time of initial presentation. These are cases of severe slip severity with severe limitation of range of motion preventing comfortable sitting or walking.

We prefer to use a radiolucent table to pin a SCFE because the frog lateral image on a radiolucent table is of superior quality to the true lateral obtained on a fracture table. It is also easier and quicker to reposition the patient's leg (as is done with a radiolucent table) to obtain a lateral view than to move the fluoroscopy machine (as is necessary with a fracture table) (Fig. 25-11).

However, if a radiolucent table is used, care must be taken to gently rotate the affected hip internally until the patella is facing forward before obtaining an anteroposterior image of the hip when choosing a pin insertion point and directing the guide wire. Failure to do so will result in the pin being inserted with the hip in a degree of external rotation; as a result, when a true anteroposterior view is obtained, the screw will be seen to be located in the superior portion of the femoral head. Other potential disadvantages of using a radiolucent table are that obtaining a true lateral is more difficult, and that the guide wire may be bent as the hip is moved into the frog position. In order to obtain a true lateral radiograph on a radiolucent table, a blanket roll may be placed under the affected hip and the fluoroscopy machine frogged 50 degrees. This will provide a lateral view of the hip without the need to rotate the hip and hence is useful in unstable SCFE.

For a stable SCFE, one 7.3-mm cannulated screw with reverse-cutting threads is used. The starting point is on the anterior femoral neck, and the screw is inserted so that it is essentially perpendicular to the physis on all views, is in a center–center position in the femoral head, and is 5 to 6 mm from the subchondral bone at its closest location when the hip is taken through a full range of motion intraoperatively. However, care must be taken to have the entry point lateral to

Text continued on page 1192

Technique for Percutaneous SCFE Pinning on a Radiolucent Table (Fig. 25-11A–D)

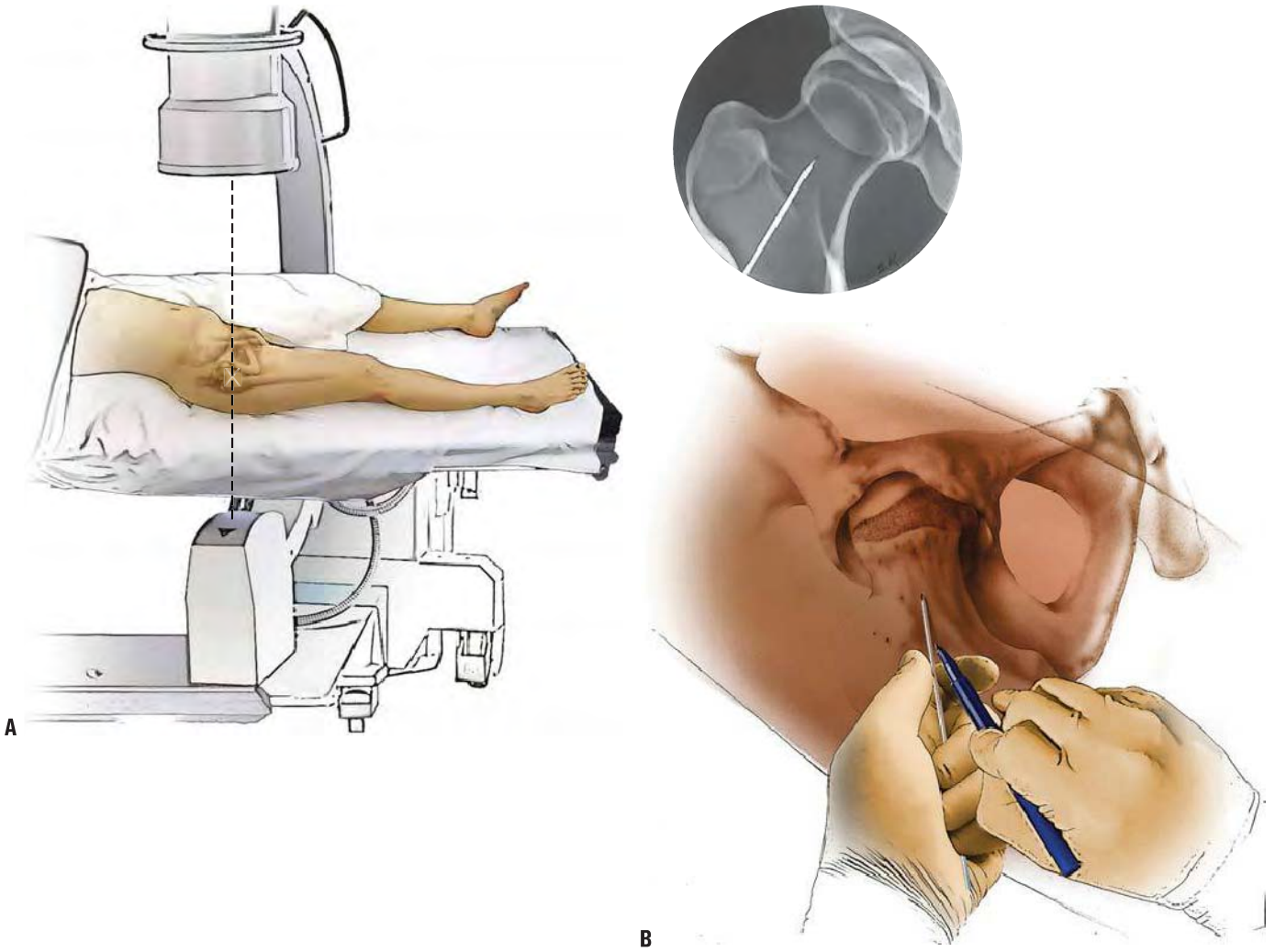
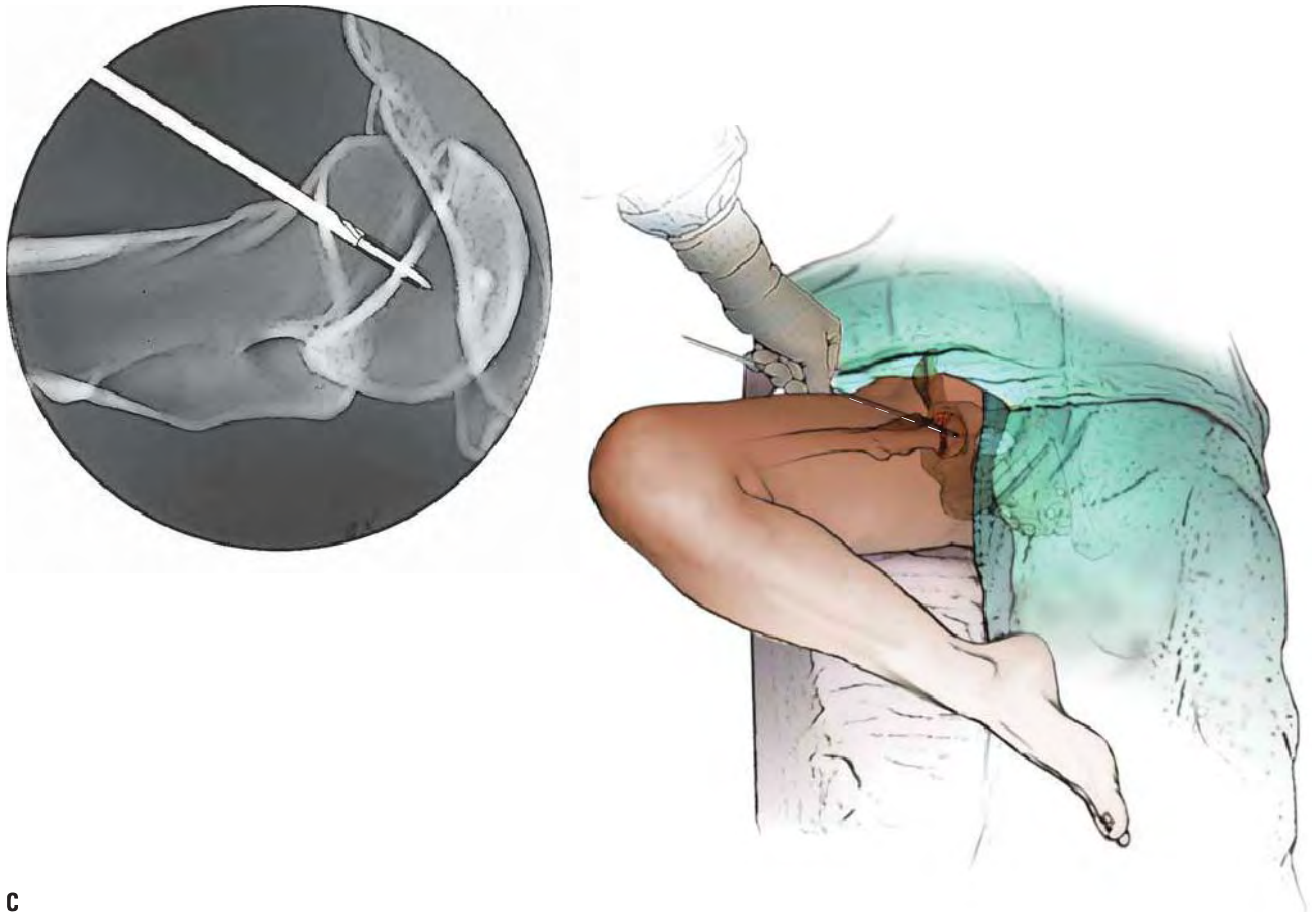
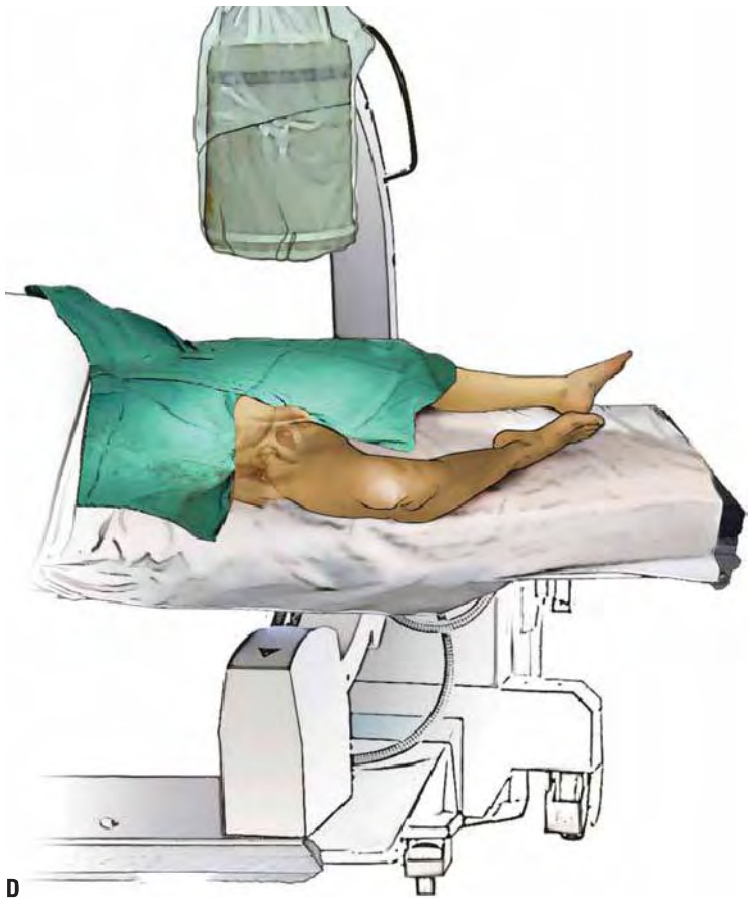


FIGURE 25-11. Technique for Percutaneous SCFE Pinning on a Radiolucent Table. A,B: The patient is placed supine on a radiolucent table. Care is taken to ensure that good visualization is obtained in both the AP and the frog lateral positions before starting this,



C



D

FIGURE 25-11. (*Continued*) **C:** A guide wire is placed anterior to the femoral neck to check the angle for pin insertion on the AP view. A line is marked on the skin along this line to facilitate later pin placement. **D:** On the lateral view, the guide pin starting point and angle are checked and adjusted to allow the pin (and cannulated screw) to pass perpendicular to the physis and end in the center of the femoral head.

the intertrochanteric line to avoid screw head impingement. In severe SCFE, the screw may need to enter oblique to the physis in order to avoid screw head impingement.

It is important to remember, however, that there may be some differences in pin position and results depending on the duration of symptoms and the magnitude of the slip. As a rule, stable slips will be only mildly displaced in patients with only a few days or weeks of symptoms, and will be more displaced in patients with many months of symptoms. For a mild slip, the starting point on the anterior femoral neck will be more distal and the screw will be inserted more horizontally than for a more displaced slip because of the posterior direction of the slip. Because the screw in a more severe slip has a more proximal starting point and often has to traverse a shorter distance in order to fix the physis, a more severe SCFE is often less difficult to pin than a very mild slip, if the correct starting point is used.

For an unstable SCFE (as classified in preceding text), two cannulated 7.3 mm screws are used. The decision to use two screws is arbitrary, although the added stability seems worthwhile in markedly unstable slips, despite the increased risk of pin penetration.

For an unstable slip, unlike the treatment of a stable SCFE, a temporary guide wire may be placed across the physis before checking a lateral x-ray film in order to assess the proximal femoral alignment before definitive fixation. Frequently, simply positioning the child on the radiolucent table with the patella directed anteriorly results in satisfactory alignment. However, if there is still marked displacement following this positioning, the hip is returned to an anteroposterior position on the radiolucent table, the temporary pin is backed out the epiphysis, and gentle traction and internal rotation are applied. Fluoroscopic images are checked following this repositioning. Following stabilization of the epiphysis with the guide wire, the frog view may be rechecked. Once the alignment is deemed appropriate, two screws are inserted with avoidance of the posterosuperior head.

Despite inconclusive evidence regarding arthrotomy or aspiration in the setting of unstable SCFE, the risk of such an intervention is low, and aspiration or arthrotomy of the joint should be considered in cases of acute SCFE. If aspiration is performed, an 18-gauge spinal needle is used. Usually, only a few milliliters of blood can be aspirated without any clear evidence of significant pressure. Alternatively, a small anterior arthrotomy can be performed along the anterior neck at the time of hip pinning.

Currently, an early bone scan or MRI is not generally indicated following unstable SCFE to evaluate for ON. Regardless of results, treatment is generally not changed based following such studies.

Postoperatively, children with stable SCFE may bear weight as tolerated. They are given crutches that are generally discarded by the time of the 1-week postoperative office visit. Sporting activities are allowed at 3 to 6 months postoperatively. Non–weight bearing is recommended for children with unstable SCFE for 3 to 4 weeks postoperatively. In the absence of any evidence of ON, they resume sporting activities at 4 to 6 months postoperatively.

Following the first 2 months (during which I obtain x-ray films at 1 week, 1 month, and 2 months postoperatively), radiographic and clinical follow-up is continued every 3 months for at least the first year, and every 3 to 6 months thereafter until both proximal femoral physes are closed.

Physeal Reduction. The goal of manipulation is to decrease the proximal femoral deformity. In the past, use of manipulation in the case of a SCFE has been described with a variety of treatments, including spica casting and internal fixation. Forceful manipulation should never be used in the treatment of SCFE (1, 137, 273). In a study of four patients with SCFE and treated with manipulation, Jerre et al. (273) noted poor results at long-term follow-up in all four; two had to undergo salvage surgery, and the other two had poor clinical hip scores.

Previous reports focused on the incidence of ON following the forceful manipulation/reduction of SCFE. Casey reported ON in 14% of acute cases of SCFE, with ON in 42% of those treated with only manipulation and casting and in none of those treated with traction and internal fixation, with or without supplemental reduction (104). Aadalen reported ON in 15% of the acute cases of SCFE, with a rate of 5% (1 of 19) among those treated with manipulation, epiphysiodesis, and casting; 19% (3 of 16) among those treated with manipulation and internal fixation; and 25% (3 of 12) among those treated with manipulation and epiphysiodesis (274). Hall (275) noted ON in 5% of the cases of SCFE treated with *in situ* fixation using a Smith-Peterson nail and a 37.5% incidence among those treated with fixation using a Smith-Peterson nail following manipulation, although these results may have been influenced by selection bias.

Multiple authors have reported that the degree of reduction does not appear to correlate with the risk of ON (103, 105, 106, 113, 130, 239, 274), although others have reported a correlation between the degree of reduction and the risk of ON (215). The timing of SCFE reduction has also been suggested as a causative factor for ON. Several series have reported ON in 0% to 9% of hips treated within 24 hours of symptom onset and in 18% to 20% of cases treated thereafter (106, 137, 274, 276). Loder et al. (103), however, did not demonstrate any benefit to early reduction in a series of 55 acute cases of SCFE, 30 of which were unstable.

Forceful manipulation in cases of SCFE is never indicated because of the increased risks of complications including ON. A serendipitous closed reduction, which may occur with patient positioning on the operating table, does not appear to negatively affect patient outcome. A recent report by Parsch et al. (277) demonstrated a 4.7% rate of ON after gentle open reduction of 66 unstable SCFE within 24 hours of presentation. Such results suggest some benefit of gentle urgent reduction in the treatment of these unstable SCFE.

Most cases of ON occur in unstable SCFE and appear to be due to the SCFE itself, although it is likely that the intraosseous blood supply to the femoral head may be disrupted if internal fixation devices are located in the superior or posterosuperior regions of the femoral head (160, 161, 163, 278, 279). Difficulty in avoiding these areas with any implants may be the reason that the rate of ON is greater when multiple implants are used to fix a SCFE (204, 215).

In one previous study, preoperative angiography showed a filling of the superior retinacular artery in only two of five unstable slips (162). One of the three hips without filling of the superior retinacular artery preoperatively was studied postoperatively, at which stage postoperative restoration of the filling of the artery was evident (162). Preoperative bone scans are quite sensitive in detecting ON, although both false positives and false negatives have been reported (137). Because almost all cases of ON were noted to have abnormal tracer uptake preoperatively, the surgery does not appear to be the main cause of ON in these patients.

The impact of capsulotomy on the rate of ON following the treatment of cases of unstable SCFE is undetermined. Clinical and laboratory studies have suggested a potential benefit, with capsulotomy reducing the rate of ON in adults and children with proximal femoral fractures; studies have also shown an increase in intracapsular pressure when the hip is maintained in internal rotation (280–285). In the laboratory, Woodhouse documented ON in dogs with intracapsular pressures of at least 50 mm of mercury for at least 12 hours (285). As seen from this and other studies (286, 287), the amount of pressure required to cause a significant decrease in the femoral head perfusion seems to greatly exceed the increased intracapsular pressure present in human hips with SCFE. Herrera-Soto et al. (288) noted that when intracapsular pressure was monitored in unstable SCFE, the affect hip had significantly increased pressure compared to the unaffected hip. Furthermore, the intracapsular pressure decreased with a capsulotomy. Additionally, they noted that even gentle manipulative reduction increased the intracapsular pressure in the affected hip.

In clinical practice, the issue of whether or not capsulotomy is beneficial remains unresolved. Some authors have recommended capsulotomy at the time of SCFE fixation in an attempt to decrease the rate of ON (137). Such recommendations are based on inconclusive data from a small number of cases. Gordon et al. (137) advocate the importance of performing a capsulotomy at the time of reduction and fixation of unstable SCFE, although examination of their data demonstrates that this recommendation is based on a single case of "mild" ON out of a total of five patients who underwent early reduction without capsulotomy, in comparison to no ON in six cases treated early with capsulotomy. Even in this case of "mild" ON, the authors reported that the child with mild ON was asymptomatic at 5-year follow-up. The supposition with recommending capsulotomy is that there is a significant hemarthrosis under pressure, which should be decompressed, although the pressures that appear necessary to cause vascular embarrassment to the proximal femur do not likely occur in most children with SCFE. At the current time, there is insufficient evidence to conclude whether capsulotomy is beneficial in reducing the rate of ON following acute/unstable SCFE, though attempts to decrease intracapsular pressure via arthrotomy or hip aspiration have become increasingly common.

Proximal Femoral Osteotomy. Osteotomy in the treatment of SCFE can be classified in both temporal and anatomic terms. Temporally, osteotomies can be thought of as either early or delayed. Early osteotomies are undertaken as part of the primary treatment of SCFE in an attempt to restore a more normal anatomy as well as to prevent further slipping. These osteotomies require fixation across the physis in order to prevent progression. Late osteotomies are generally undertaken to correct residual deformity after physeal closure. Usually, these are performed at least 1 year after the initial treatment if significant symptoms persist or if the anatomic derangement is felt to be severe enough to require treatment.

Anatomically, osteotomies may be classified as subcapital, femoral neck, or intertrochanteric (Fig. 25-12). The ability to

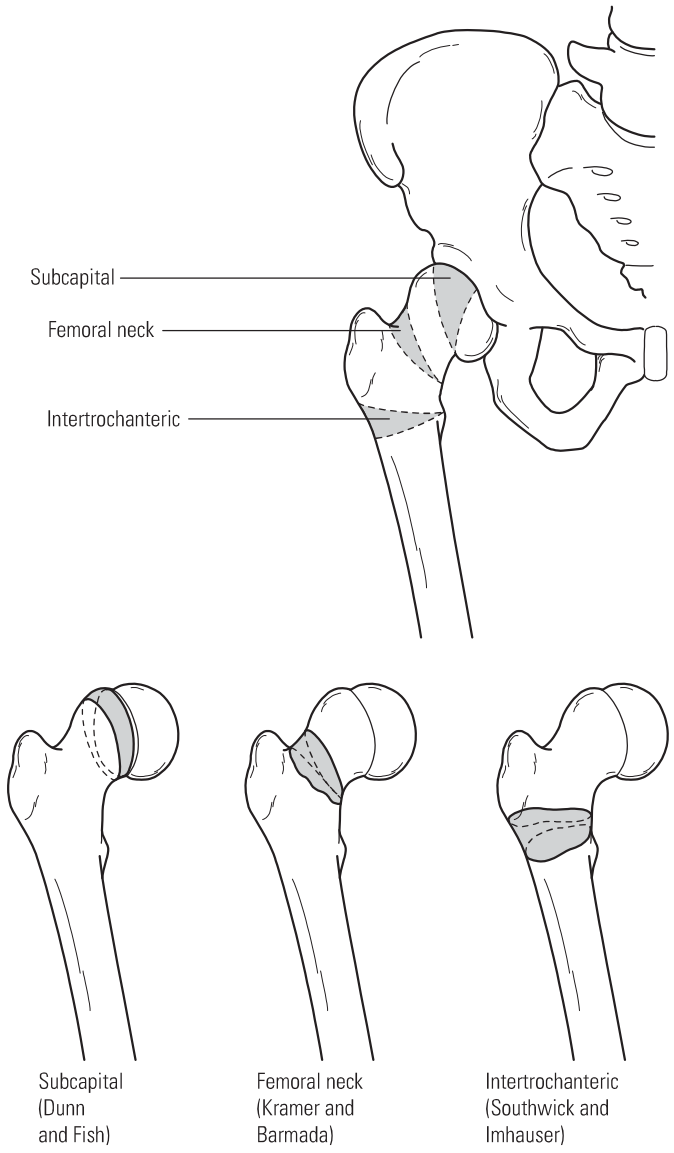


FIGURE 25-12. The three levels of osteotomy to correct the proximal deformity following SCFE. The ability to correct the deformity is greatest with a subcapital osteotomy, least with a femoral neck osteotomy, and intermediate with an intertrochanteric osteotomy. The risk of ON is inversely related to the distance from the physis to the osteotomy. Intertrochanteric osteotomies are currently the most commonly performed osteotomies because of the low rate of ON and the ability to obtain good correction.

correct the deformity is greatest with a subcapital osteotomy, least with a femoral neck osteotomy, and intermediate with an intertrochanteric osteotomy. The risk of ON is inversely related to the distance from the physis to the osteotomy, with subcapital osteotomies having the highest risk and intertrochanteric osteotomies having the lowest. Frymoyer (289) reported ON in 30% of femoral neck osteotomies and 0% of intertrochanteric osteotomies. Interestingly, Jerre et al. (273) reported lower short-term complications but poorer long-term outcomes with intertrochanteric osteotomies compared to subcapital osteotomies.

Using computer modeling, previous authors have reported lower intra-articular contact stress in femoral neck osteotomy compared to intertrochanteric osteotomy (290). Although still experimental, preoperative computer simulation of osteotomies has been suggested to optimize surgical planning for patients with SCFE (291).

Previous computer modeling (145) and clinical studies (145–149) have demonstrated the potential risks of femoroacetabular impingement, with resultant cartilage damage and/or labral tears, in hips with SCFE, especially in hips with more severe slips. After remodeling, as the range of motion increases, an increasing portion of the remodeled metaphysis becomes an intra-articular weight-bearing surface, potentially contributing to late OA (145). Although theoretical reasons and indications from some medium-term clinical studies argue that restoring a more normal osseous alignment may be beneficial to the hip in the long term (89, 292), there are no clinical data in the literature to prove that such realignment results in enhanced long-term hip function or durability.

Subcapital Osteotomy. Subcapital proximal femoral osteotomies have most commonly been described as a primary treatment of moderate-to-severe SCFE, with the goals of deformity correction and prevention of slip progression. Because they are performed at the level of deformity, subcapital osteotomies are the most powerful osteotomies for deformity correction. These are very technically demanding operations, are associated with high rates of ON, and are rarely performed. As early as 1948, Martin (293) noted the importance of avoiding tension on the posterior periosteal vessels in order to minimize the risk of ON. Subcapital osteotomies have been referred to as *orthopaedic roulette* because of their risky nature (294).

In slips exceeding 30 degrees, Fish (256, 295) reported good-to-excellent results in 92% of patients following cuneiform subcapital osteotomy at a mean follow-up of 13 years. He noted the importance of removing all callus and physis in order to avoid tension on the posterior periosteum as the epiphysis is reduced onto the femoral neck, and stated that this osteotomy should only be performed in hips with an open proximal femoral physis. He demonstrated excellent corrections with low rates of complications. Other reports of such excellent results following subcapital osteotomy are rare, although Nishiyama et al. (296) reported 93% excellent results at a mean follow-up of 10 years in the cases of 15 patients with 18 SCFEs treated with cuneiform osteotomies.

Dunn (297) and Dunn and Angel (298) also described a transtrochanteric subcapital osteotomy, which shortened the femoral neck and preserved the posterior blood supply to the femoral head. Even in Dunn's hands results were mixed, with good clinical results in only 55 of 73 hips (75%) and good radiographic results in only 41 (56%) of the hips at a mean follow-up of <9 years (298). Other authors following Dunn have reported mixed results and high rates of complications (197, 273, 299–302). Average EBL exceeding 500 mL has been reported (300). Recently, the Dunn procedure was combined with a transtrochanteric surgical hip dislocation approach (303–305). In a series of 40 patients, moderate-to-severe stable and unstable SCFE were fully corrected without development of ON or chondrolysis (305). The added surgical exposure and careful dissection of the femoral neck periosteum may facilitate this technically demanding procedure.

Complications including ON, chondrolysis, OA, and LLD are common after subcapital femoral osteotomies (197, 273, 299–302). One exception is slip progression, which does not appear to have been reported following subcapital femoral osteotomy (256, 295–298, 300, 306).

Chondrolysis has been reported in 3% to 42% of cases following subcapital osteotomies, with most authors reporting rates in the range of 3% to 18% (256, 296, 297, 299–302, 307). Dunn reported chondrolysis in 18% of his cases (13 of 73), with a rate of 17% in 24 in acute-on-chronic cases and 18% in 49 in chronic cases (297).

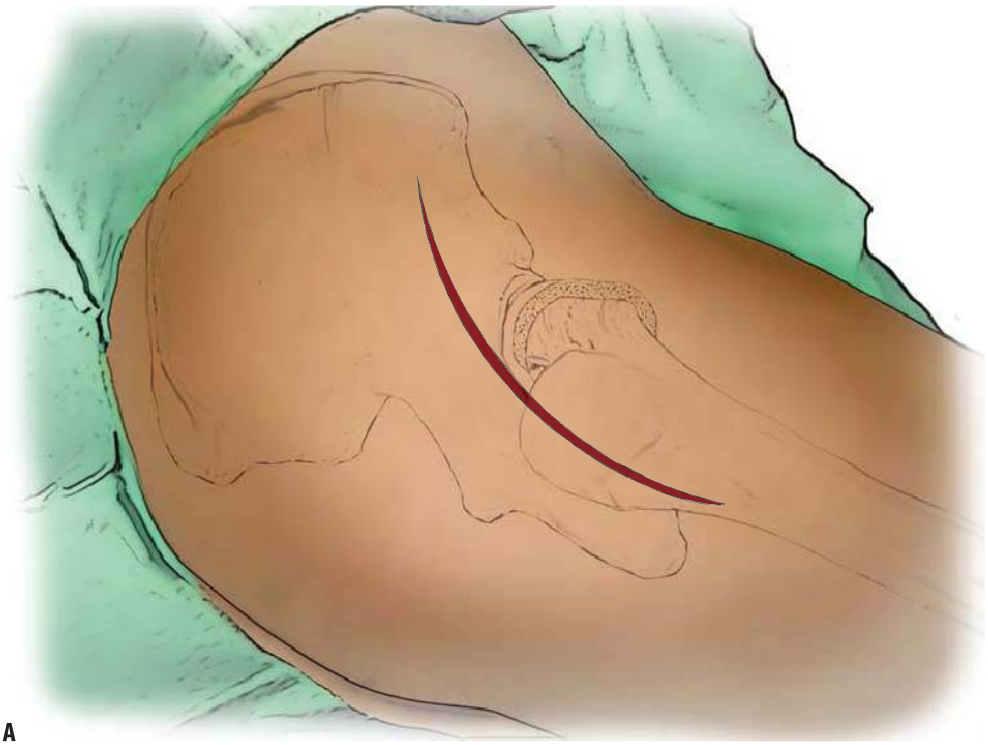
LLD is common following subcapital femoral osteotomy (256, 296, 302). Fish reported an LLD of at least 1 cm in 35% of patients (23 of 66) and of at least 2 cm in 6% (4 of 66) of patients treated with cuneiform subcapital osteotomy, with a maximum difference of 5 cm (256). Nishiyama et al. (296) reported an average LLD of 1.5 cm in their series of subcapital osteotomies, ranging from 1 to 2 cm. Velasco et al. (302) noted LLD of at least 1 cm in 6% and at least 2 cm in 3% of their patients.

Subcapital osteotomies are theoretically very appealing because of the powerful correction they afford. However, because the learning curve is steep, complications are frequent and severe, and experience is necessary for good results, subcapital osteotomies are rarely used currently. A possible role for subcapital osteotomy may be in the unstable moderate-to-severe SCFE where the rate of ON is inherently high, but the physis is mobile, facilitating the subcapital dissection and hence making the procedure less technically challenging.

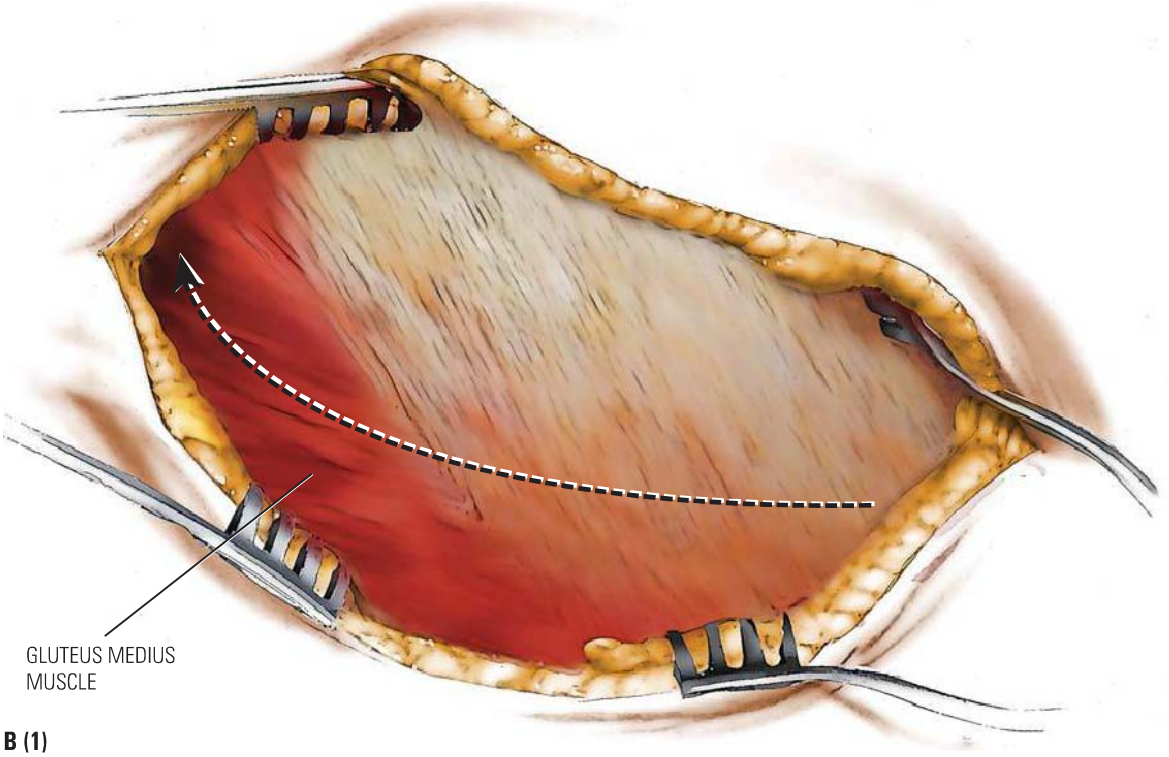
Femoral Neck Osteotomy. In comparison to subcapital osteotomies, femoral neck osteotomies have less power to correct deformity but are also associated with a somewhat lower risk of ON. These osteotomies can be performed in the middle of the neck or at the base of the neck and may be performed as either a primary or a secondary treatment of SCFE. As with other proximal femoral osteotomies, the goal of femoral neck osteotomy is to restore a more normal proximal femoral alignment (Fig. 25-13).

Text continued on page 1198

Technique for Base of Femoral Neck Osteotomy for SCFE (Fig. 25-13A–D)

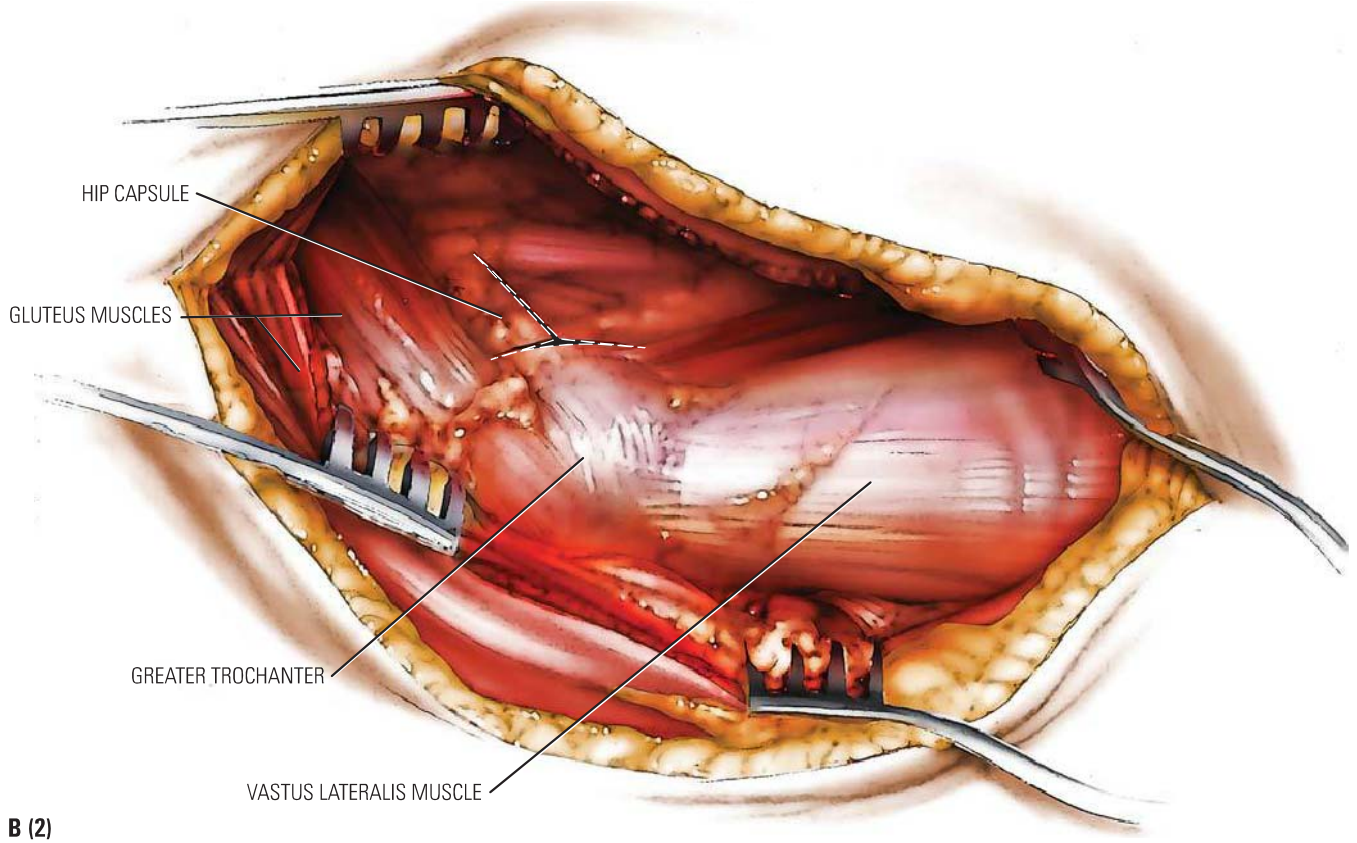


A

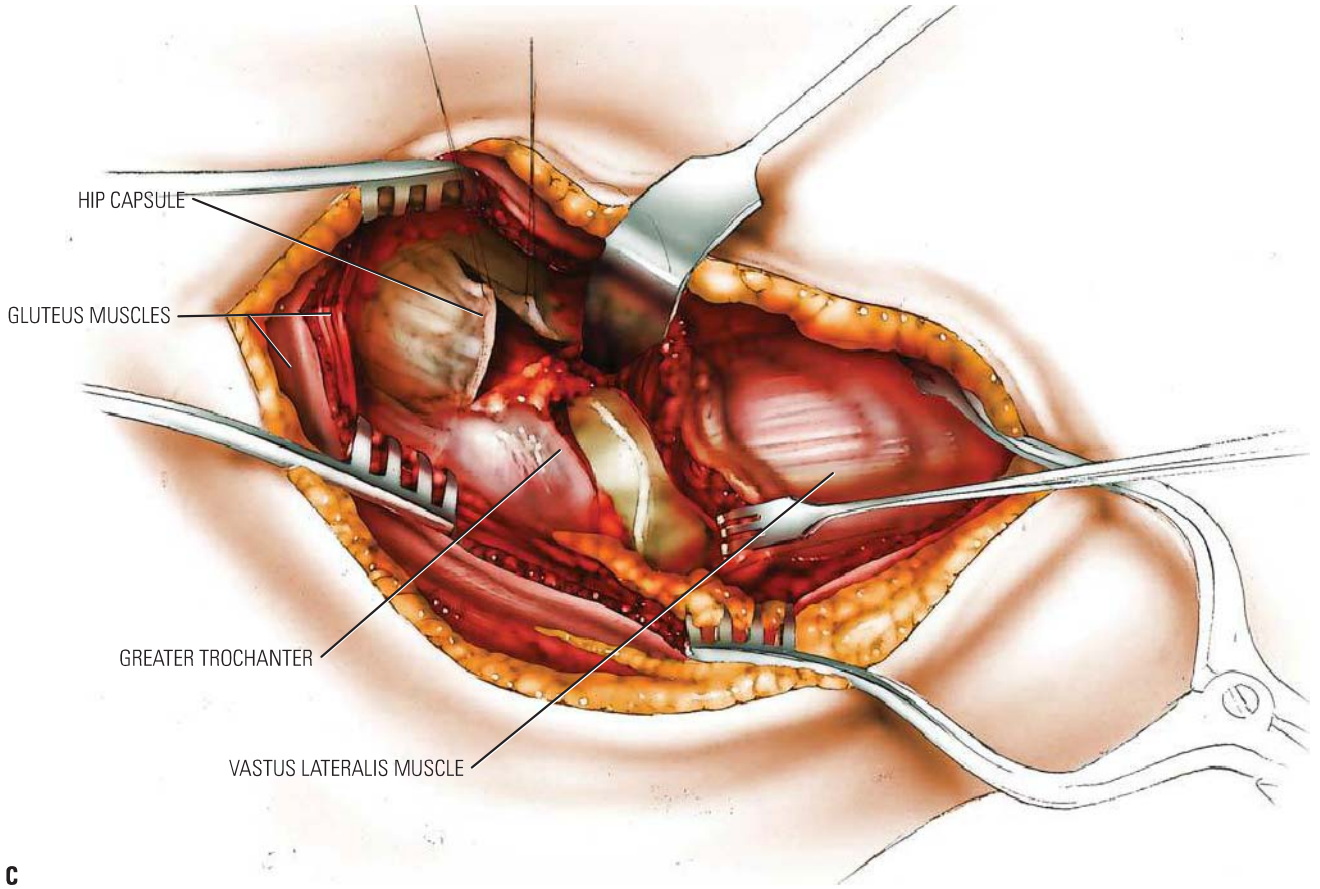


B (1)

FIGURE 25-13. Technique for Base of Femoral Neck Osteotomy for SCFE. A: The incision begins about 2 cm distal and lateral to the anterosuperior iliac spine. It curves over the tip of the trochanter, staying slightly posterior, and ends far enough distally to permit the insertion of the pins at the proper angle into the neck. **B:** The interval between the tensor fascia lata muscle and the gluteus medius muscle can be difficult to identify until experience is gained with this approach. To identify this interval, the fascia lata is incised distally and the incision is continued proximally, staying along the posterior border of the tensor fascia lata muscle. The interval is identified more easily distally, where the difference in insertion and direction of the fibers is more apparent (**1**). The tensor fascia lata muscle is retracted anteriorly, and with blunt dissection, the plane between this muscle and the gluteus medius muscle is found and developed. There are some vessels in this interval that will need to be divided, but the dissection should not continue so far anteriorly as to divide the branch of the superior gluteal nerve to the tensor fascia lata muscle (**2**).



B (2)



C

FIGURE 25-13. (*continued*)

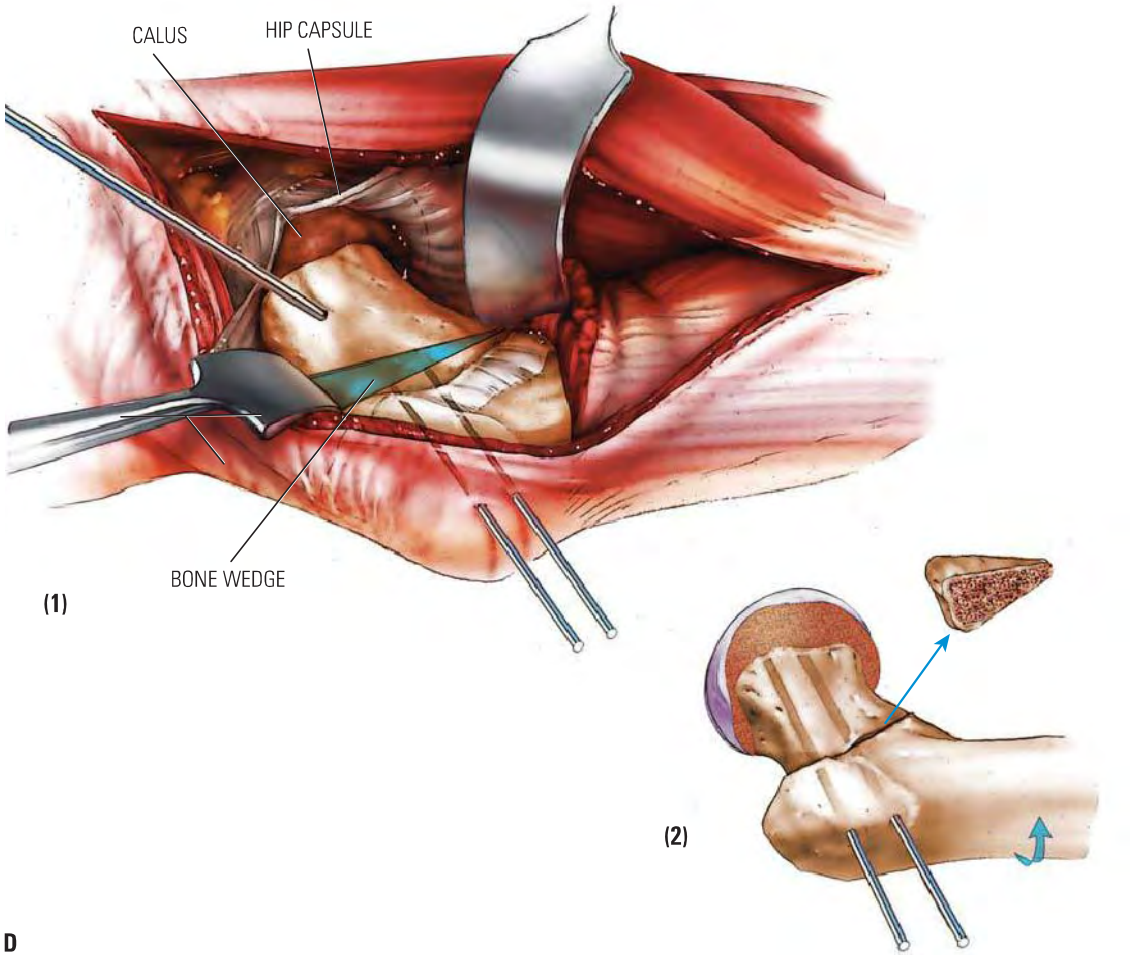


FIGURE 25-13. (*continued*) **C:** After the interval is developed, the tensor fascia lata muscle is retracted anteriorly and the gluteus medius posteriorly. The fibrofatty tissue seen is that covering the hip capsule. To expose the capsule externally, the hip is rotated. This tissue is incised and dissected off the anterior capsule, often along with the capsular origins of the iliacus muscle joining the psoas medially and capsular insertions of the gluteus muscle laterally. In addition, it is often necessary to incise the origin of the vastus lateralis muscle along the trochanteric line, reflecting it distally a short distance. The capsule of the hip joint is now opened with an H-shaped incision with the transverse arm of the *H* near the base of the neck. This permits adequate inspection of the callus at the site of the slip, which Kramer et al. (309) recommend to determine the size of the wedge, and adequate exposure at the base of the neck for the osteotomy. It is probably important that retractors are not placed around the neck of the femur, in order to avoid damage to the vessels in the retinaculum posteriorly. **D:** The amount of bone to be removed is difficult to calculate. Kramer et al. (309) describe measuring the callus at the site of the slip both superiorly and anteriorly to define the dimensions of the wedge. Only two-thirds of the measured amount is removed anteriorly to avoid overcorrection of the retroversion, whereas the full amount measured is removed superiorly. This can also be estimated preoperatively by measuring the head–neck angle on the anteroposterior and lateral radiograph. Kramer et al. (309) stated that a common error is to remove too much bone anteriorly and not enough superiorly. The idea is to make the distal cut perpendicular to the femoral neck and the proximal cut perpendicular to the physeal surface of the femoral head. The wedge can be removed with an osteotome, as described by Kramer et al. (309), or with a high-speed burr, as described by Gage et al. (307). It is easier to start with the osteotome but safer to finish with a burr. The burr is used to thin the posterior and inferior cortex but not break it, a point on which all surgeons seem to agree. A heavy Steinmann pin is now placed in the proximal fragment to control it. The Steinmann pins that will be used to fix the osteotomy site are drilled into the distal fragment from the lateral cortex, in line with the femoral neck. The posterior and inferior cortices are thinned until they can be fractured easily in a greenstick fashion by manipulating the Steinmann pins to close the osteotomy site (**1**). This can be aided by abducting and internally rotating the leg. When the osteotomy site is closed, the Steinmann pins are advanced from the lateral cortex across the osteotomy site, across the physeal plate, and into the femoral head (**2**). The pin in the proximal fragment is removed, and the pins are cut at the lateral cortex, leaving them long enough for removal after healing and closure of the physis. At this point, Kramer et al. (309) recommend performing an epiphysiodesis of the greater trochanter to prevent overgrowth. The shortening of the femoral neck leads to a decrease in the articulotrochanteric distance, but a greater trochanteric epiphysiodesis will not correct this in this age group because insufficient growth remains. This makes their second recommendation, a trochanteric transfer, more reasonable when the articulotrochanteric distance is decreased significantly. The capsule and the wound are closed.

Because these osteotomies are somewhat distant from the deformity, maximum correction may be incomplete in moderate-to-severe slips. Despite the incomplete correction of the underlying deformity, proponents note that sufficient correction can be obtained to significantly improve hip alignment and biomechanics (264, 308, 309).

Osteotomy at the base of the femoral neck may be intracapsular or extracapsular (264, 308, 309). Care must be taken to preserve the posterior blood supply to the femoral head. Extracapsular osteotomies have the theoretical benefit of a decreased risk of ON, although they are less able to correct the underlying deformity because of their more distal location (264, 308). In a series of 36 extracapsular osteotomies, Abraham et al. (264) reported 89% good-to-excellent results and no cases of ON at an average follow-up of 9 years.

Complications of femoral neck osteotomies include unrecognized pin penetration, chondrolysis, ON, hardware failure, LLD, joint space narrowing, and OA. Gage et al. (307) have noted decreasing rates of both ON and chondrolysis with more distal osteotomies. Even with attempts to preserve the blood supply, ON has been reported in up to 10% of cases following femoral neck osteotomy (307). Chondrolysis has been reported in 2% to 10% of the base-of-neck osteotomies for SCFE (264, 308, 309).

In a series of 56 intracapsular osteotomies, Kramer et al. (309) reported two cases of pin penetration and an 11% reoperation rate. Barmada et al. (308) reported one case each of loss of fixation and joint penetration by hardware in their study of a series of 20 hips. Joint space narrowing has been noted in 10 of 11 hips (91%) followed up for at least 13 years in one series (264).

Base-of-neck osteotomy has been reported to lead to LLD of at least 1 cm in 61% of patients and at least 2 cm in 42% (15 of 36 patients) (264). Three patients in the same series (8%) had LLD of at least 4 cm, and in male adolescents with at least 3 years of growth remaining, unilateral SCFEs were noted to lead to LLD of 3 to 5 cm (264).

Currently, the trend is away from femoral neck osteotomies and toward intertrochanteric osteotomies because of the technical difficulties of femoral neck osteotomies, their risk of complications, and the limited ability to completely correct the deformity.

Intertrochanteric Angular Osteotomy. Intertrochanteric osteotomies are currently the most commonly performed osteotomy for SCFE. Such osteotomies are generally performed after physeal closure in patients with significant limitations in the range of motion, significant pain, and/or marked proximal femoral deformity. The most common intertrochanteric osteotomies are angular osteotomies described by Southwick and by Imhauser (124, 310–314) (Figs. 25-14 and 25-15). These osteotomies are generally fixed with plates, but use of external fixation has also been reported (315, 316).

Angular osteotomies may be uniplanar, biplanar, or triplanar. The three common components of the osteotomy are valgus, flexion, and internal rotation through the osteotomy. The degree of correction is based on anatomic alignment, range of motion deficit, and patient complaints. The Southwick osteotomy is the most commonly performed intertrochanteric osteotomy in North America, and the Imhauser osteotomy is more popular in Europe. Southwick described a valgus and flexion osteotomy to which internal rotation of the distal fragment is generally added (124, 310–313). Therefore, Southwick osteotomies are generally triplanar osteotomies. Imhauser described a biplanar flexion and internal rotation osteotomy without valgus. A "reverse" Imhauser osteotomy has been reported for the uncommon valgus SCFE (317). With either type of intertrochanteric osteotomy, significant internal rotation of the distal fragment must usually be performed in order to restore both proximal femoral anatomy and a more normal rotational arc of motion.

Recommendation for intertrochanteric osteotomy can be made on the basis of clinical signs and symptoms or on a biomechanical basis in an attempt to normalize proximal femoral anatomy with the theoretical decrease in the long-term risk of OA. Clinical indications for intertrochanteric osteotomy may include hip and/or groin pain with prolonged sitting (owing to femoral neck impingement on the acetabulum) and difficulty in performing activities because of the abnormal arc of hip motion. Lack of hip flexion and internal rotation may make routine activities such as sitting in a chair, climbing stairs, riding a bicycle or scooter, donning and doffing socks, and cutting one's toenails difficult or impossible. A significant varus deformity of the proximal femur may result in significant abductor weakness, with a persistent Trendelenburg gait and fatigue pain with ambulation. If the recommendation for intertrochanteric osteotomy is based on clinical signs and symptoms, the physician should wait at least 1 year following *in situ* fixation to be certain that such signs and symptoms do not spontaneously improve.

Increasingly, a recommendation for proximal femoral osteotomy is being made on a biomechanical basis regardless of the patient's symptomatology. The argument for such surgery is the increasing body of knowledge that OA is more common and more severe in the more severe cases of SCFE, even in the absence of short-term complications (113, 125, 166, 168, 169, 181). In addition, biomechanical modeling studies have shown that the deformity associated with SCFE would place patients with SCFE at long-term risk of OA (145). A SCFE can result in the anterior femoral metaphysis articulating with acetabular cartilage and can also cause impingement of the femoral neck against the anterior acetabulum (145). Despite these theoretical benefits, prophylactic surgery is of disputed value. In a matched cohort study comparing hips treated with in situ pinning and those treated with intertrochanteric osteotomy, Diab et al. (318) showed increased range of motion in chronic, severe, stable SCFE, but were not able to show a significant clinical difference. In contrast, Spencer et al. (319) showed clinical benefit of the osteotomy. Two studies have reported an apparent decrease in the expected rate of OA following a realignment at a follow-up of more than 20 years (89, 292). Despite these results, there are currently no long-term clinical data that conclusively demonstrate advantages of routine proximal femoral osteotomy in patients followed into middle and old ages (94, 320, 321).

Text continued on page 1202

Technique for Southwick Intertrochanteric Osteotomy for SCFE (Fig. 25-14A–E)

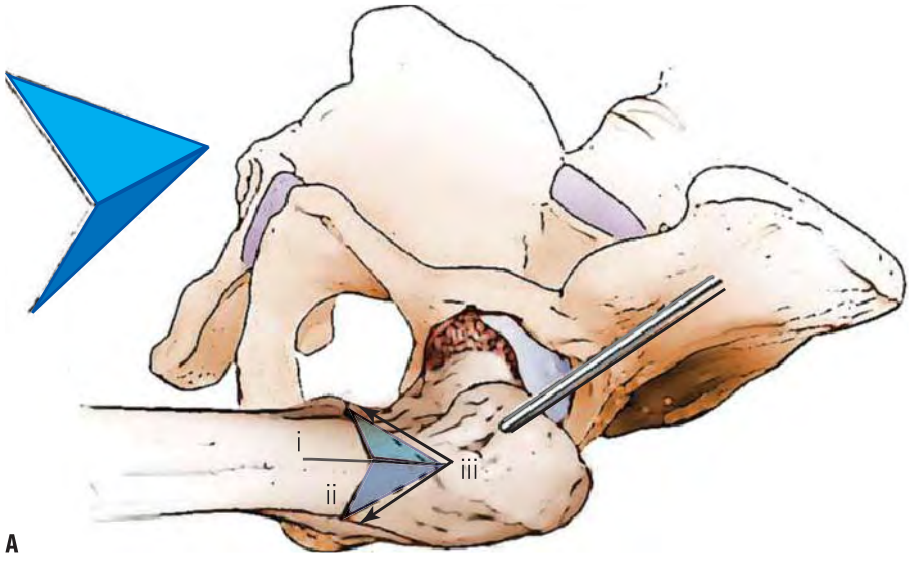
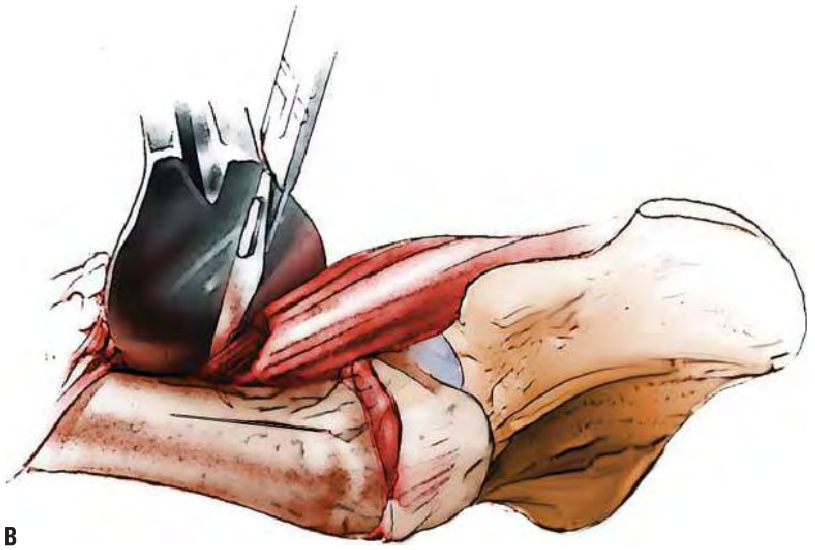
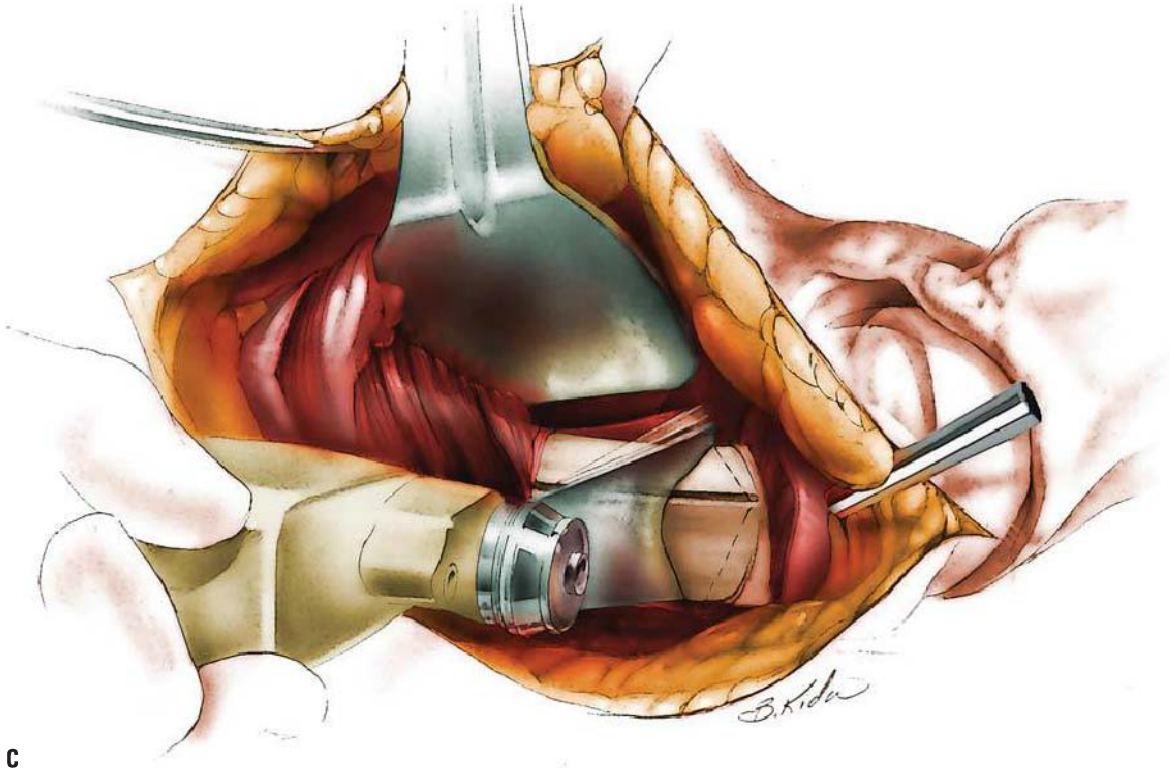


FIGURE 25-14. Technique for Southwick Intertrochanteric Osteotomy for SCFE. A: The intertrochanteric area of the proximal femur is exposed as described for a varus intertrochanteric osteotomy. Care must be taken to expose the lesser trochanter adequately if release of the iliopsoas tendon, as initially recommended by Southwick (124), is planned. Marks outlining the desired wedges are scored on the femoral shaft using an osteotome or saw. First, a vertical line is scored in the femoral shaft separating the anterior from the lateral femoral shaft (i). Next, the base of the wedge is marked. It is the same on the lateral and anterior femoral surfaces and corresponds to a line perpendicular to the femoral shaft just cephalad to the lesser trochanter. This is the same as the definitive osteotomy line (ii) used in planning an intertrochanteric osteotomy. The surgeon now must determine the wedge that will be removed anteriorly to produce flexion and the one that will be removed laterally to produce valgus. The maximal angles of the original Southwick templates are 45 degrees anteriorly and 60 degrees laterally. These are unlikely to be available. The angle guides from the AO set or any other templates that are available can be used. This represents the maximal wedges that can be removed. They are marked to describe a wedge that includes the entire diameter of the femoral shaft. The apex of both wedges is marked (iii).



B



C

FIGURE 25-14. (*continued*) **B:** Before the osteotomy is made, it is necessary to gain control of the proximal fragment. This can be done with the chisel for the blade plate. It is driven in with its flat surface parallel to the osteotomy surface and at least 1.5 cm above it (see Fig. 4-14). This is a difficult part of the operation, and the surgeon may not have confidence in doing this until the correction through the osteotomy is made. In that case, a large smooth pin is drilled into the superior aspect of the greater trochanter, aiming posteriorly. This provides good fixation and keeps the pin out of the path of the seating chisel, which can be inserted after the correction is achieved and after the surgeon sees a more normal orientation of the femur. The first cut is through the base of the osteotomy (see Fig. 4-14). This cut should be made to the medial cortex, which is best left intact until the other cuts are made. **C:** Confusion often arises when attempting to remove two separate wedges, one lateral and one medial. Rather, the cut that follows should remove one wedge: both the medial and lateral wedges as one piece. This is accomplished by starting the saw on the line separating the anterior and lateral aspects of the femoral shaft. It is angled so that it includes both wedges, aiming toward the first cut at the medial cortex.

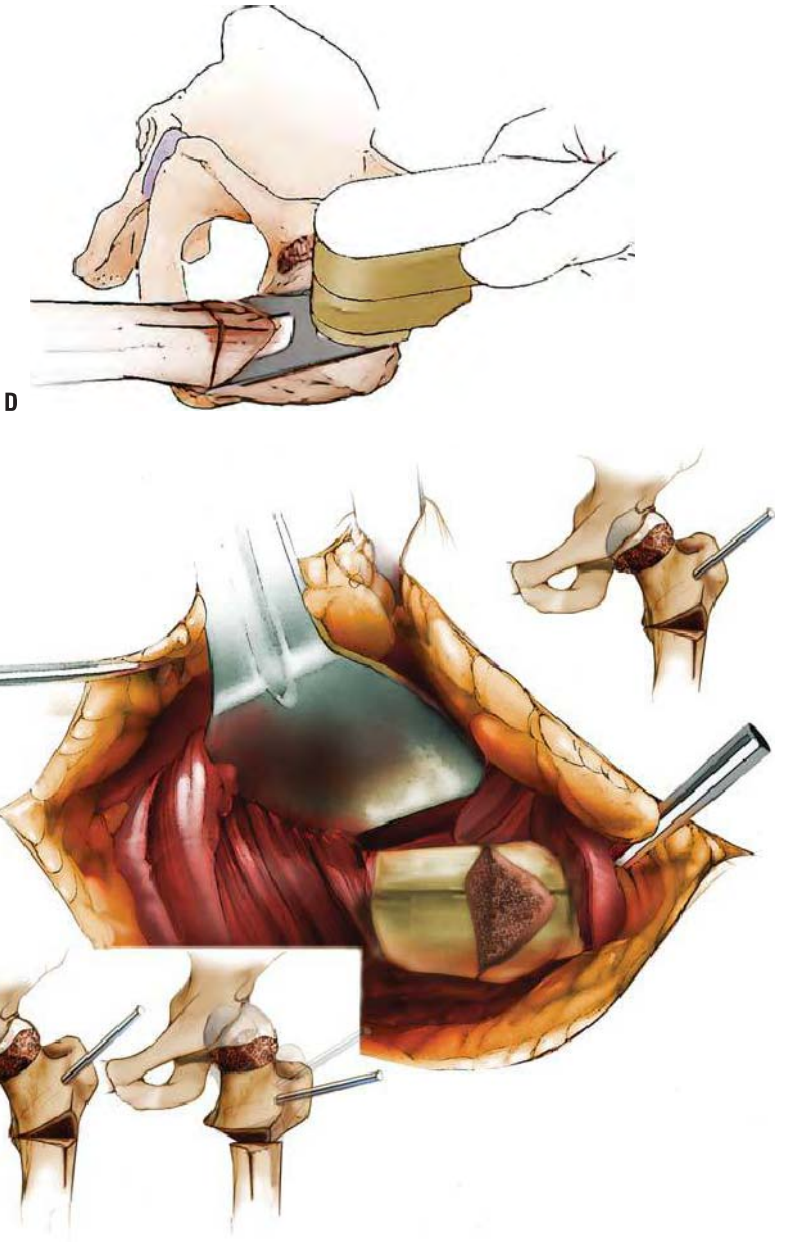


FIGURE 25-14. (*continued*) **D:** With the wedge of bone removed, the osteotomy is completed through the medial cortex and is ready for closure. This is best accomplished by a combination of maneuvers. First, the fracture table, which holds the leg, is raised to produce flexion and abducted to produce valgus. The pin in the greater trochanter is used at the same time to pull down the proximal fragment, thereby closing the osteotomy. Although correcting the deformity of the proximal femur should in theory correct the lack of internal rotation, it is usually necessary to add some internal rotation to the distal fragment in order to achieve this. In most severe slips, this is about 30 degrees and can be noted accurately by placing two pins on either side of the osteotomy, as described for rotational intertrochanteric osteotomy. **E:** With the osteotomy held closed by an assistant using the heavy pin, the steps used to insert the blade plate, as described in the technique of varus intertrochanteric osteotomy are performed. The angled plate used depends on the configuration of the bone at the completion of the osteotomy. It is possible to use either the 90-degree angled plate or the 120-degree repositioning plate. The 90-degree blade plate is easier to place accurately but will not provide as good fixation in the proximal fragment as the 120-degree blade plate does. However, the 120-degree blade plates, in the standard form supplied, often seem too long. For those with considerable experience in the use of the AO blade plates, there is an alternate method of inserting the blade and reducing the osteotomy. The 90-degree blade plate is designed so that the blade is inserted into the proximal fragment, 1.5 cm proximal to the osteotomy cut, and the blade is kept parallel to the plane of the osteotomy cut in the proximal fragment. Therefore, if the blade is inserted into the proximal fragment beginning 1.5 cm proximal to the osteotomy and the flat surface of the blade is kept parallel to the plane of the osteotomy cut in the proximal fragment, when the osteotomy is closed the plate will lie in correct apposition to the lateral aspect of the distal fragment and the osteotomy will be closed. Using this technique, the chisel is driven into the proximal fragment after the wedge of bone is removed, and the insertion of the Steinmann pin to control the proximal fragment is not necessary. At least four screws should attach the plate to the femoral shaft to provide sufficient fixation to overcome the need for a cast. The wound is closed and drained as in other intertrochanteric osteotomies.

Imhauser Osteotomy (Fig. 25-15)

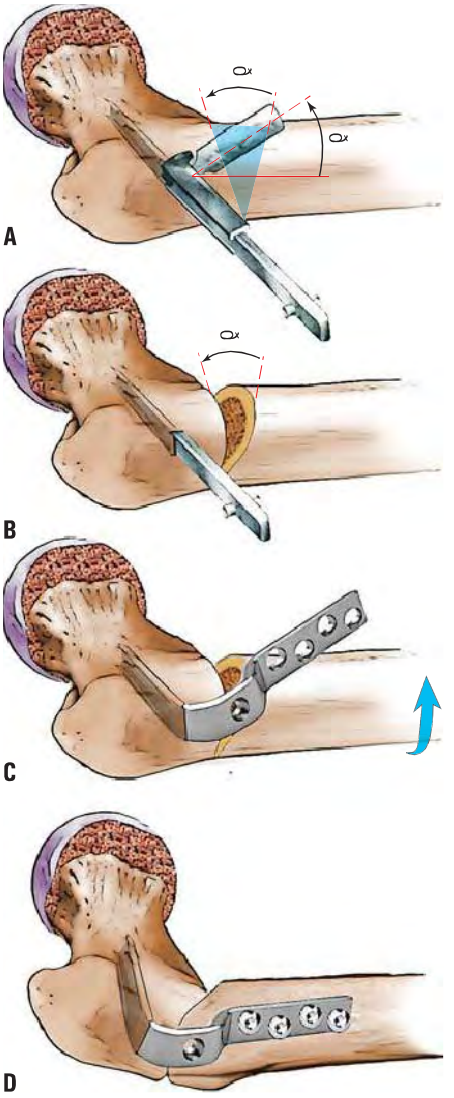


FIGURE 25-15. Imhauser Osteotomy. The exposure is as noted for a Southwick osteotomy. The Imhauser osteotomy is similar to a Southwick osteotomy but only includes two of the three planes (flexion and internal rotation), and does not include a varus component. **A:** The blade plate chisel is inserted in the proximal femur at an angle (alpha) that is the desired amount of flexion desired for the osteotomy. **B:** An anterior closing wedge osteotomy of the same angle (alpha) is performed to allow for optimal bone contact after blade plate insertion. **C:** The blade plate is inserted and the distal fragment internally rotated (to correct the external rotation deformity). **D:** The osteotomy is closed anteriorly and the plate is fixed to the femoral shaft.

The amount of correction necessary depends on the degree of deformity and the clinical arc of motion. As a result, the amount of correction noted at follow-up may vary with patient selection criteria. In one series of Southwick osteotomies, Salvati et al. (322) noted a mean increase in internal rotation of 33 degrees and an abduction of 17 degrees at follow-up. Computer simulation studies using CT scan data demonstrate the importance of the proximal metaphyseal prominence in limiting hip range of motion in SCFE (323). Furthermore, clinical outcome after in situ pinning appears to correlate with the persistence of the metaphyseal prominence (236). There may be some benefit to combining a proximal femoral osteoplasty with a proximal femoral osteotomy (319). The proximal femoral osteoplasty will decrease the amount of correction needed at the intertrochanteric level.

Overall, the results of intertrochanteric osteotomies have been good, with acceptable rates of complications. At a follow-up at least 5 years after the surgery, Southwick reported excellent or good results in 93% of the patients (124), and 13 years later the long-term follow-up results were reported as good or excellent in 87%, although the precise duration of the follow-up was not specified (310). Other authors have reported good-to-excellent clinical results in 80% to 85% of patients who were followed up for 5 to 10 years, with good radiographic results in about 60% (324, 325). Other authors who reported poor results noted that the poor results were seen in conjunction with insufficient surgical correction of the underlying deformity (326).

Similarly, good clinical results have been reported with Imhauser osteotomies, with Parsch et al. (327) reporting good or very good results in 92% of the hips operated on from 1975 to 1982, and an average Iowa hip score exceeding 90 in patients operated on subsequently.

Intertrochanteric osteotomies, despite their low risk of ON, are noted to have other significant complications including chondrolysis, delayed union, need for reoperation, late arthritis, LLD, and fracture. Because these osteotomies are typically performed after physeal closure, progressive slip does not occur.

Chondrolysis has typically been reported in 2% to 25% of hips following intertrochanteric osteotomies, although rates up to 59% have been reported in small series (124, 243, 289, 310, 322, 324, 327). Delayed unions have been reported in up to 3% or 4% of the hips following intertrochanteric osteotomy in several series (292, 322, 325). Loss of fixation has been reported in 4% to 6% of cases in some series (322, 326). Fractures are not reported in most series following osteotomy, but were reported in 6% of 130 cases of intertrochanteric osteotomies in one series (327).

LLD following intertrochanteric osteotomy is well described. However, because a Southwick intertrochanteric osteotomy includes a valgus component, it is believed to lead to less LLD than other osteotomies. LLD following Southwick osteotomy has been reported in 19% to 26% of the cases, with a maximum LLD of 2 cm (322, 325, 326). The operated leg was short in 15% to 19% of patients in these series and was long in 0% to 11% (322, 325, 326). Schai et al. (292) reported LLD in 81% of patients (38 of 47) following Imhauser osteotomy, with the affected leg being an average of 0.9 cm short in 35 patients and being 0.5 to 2.0 cm long in the other 3 patients.

At a mean of 24 years following Imhauser intertrochanteric osteotomy, Schai et al. (292) reported moderate OA in 28% of the hips and severe OA in 17%. Jerre et al. (272) reported 36% good-to-excellent results in 11 hips at long-term follow-up averaging 36.1 years, although radiographic and surgical techniques have advanced significantly in the interim. One of these 11 patients (9%) was found to have undergone salvage surgery by the time of long-term follow-up (273).

Intertrochanteric osteotomies are currently the most common osteotomies performed on children with SCFE. These osteotomies can be challenging in these very large, heavy patients with significant deformity. Intertrochanteric osteotomies are also somewhat limited in their ability to correct the deformity because of their considerable distance from the site of deformity. Despite incomplete correction, there is generally sufficient correction to allow for good clinical outcomes with an acceptably low rate of complications (Fig. 25-16).

In asymptomatic patients with severely affected slips, the role of osteotomy is unclear. The real question to consider in such patients is whether they are best served by an osteotomy in adolescence, with its attendant risks, in an attempt to delay or avoid total hip arthroplasty (THA). One of the confounding variables in such an evaluation is that it is impossible to be certain what impact the advances in THA or basic science (such as gene manipulation) may have on the long-term outcomes of SCFE and OA in the coming decades.

Transtrochanteric Rotational Osteotomy. Sugioka described a rotational intertrochanteric osteotomy for correction of significant residual proximal femoral deformity following SCFE (328, 329). Sugioka states that such an osteotomy

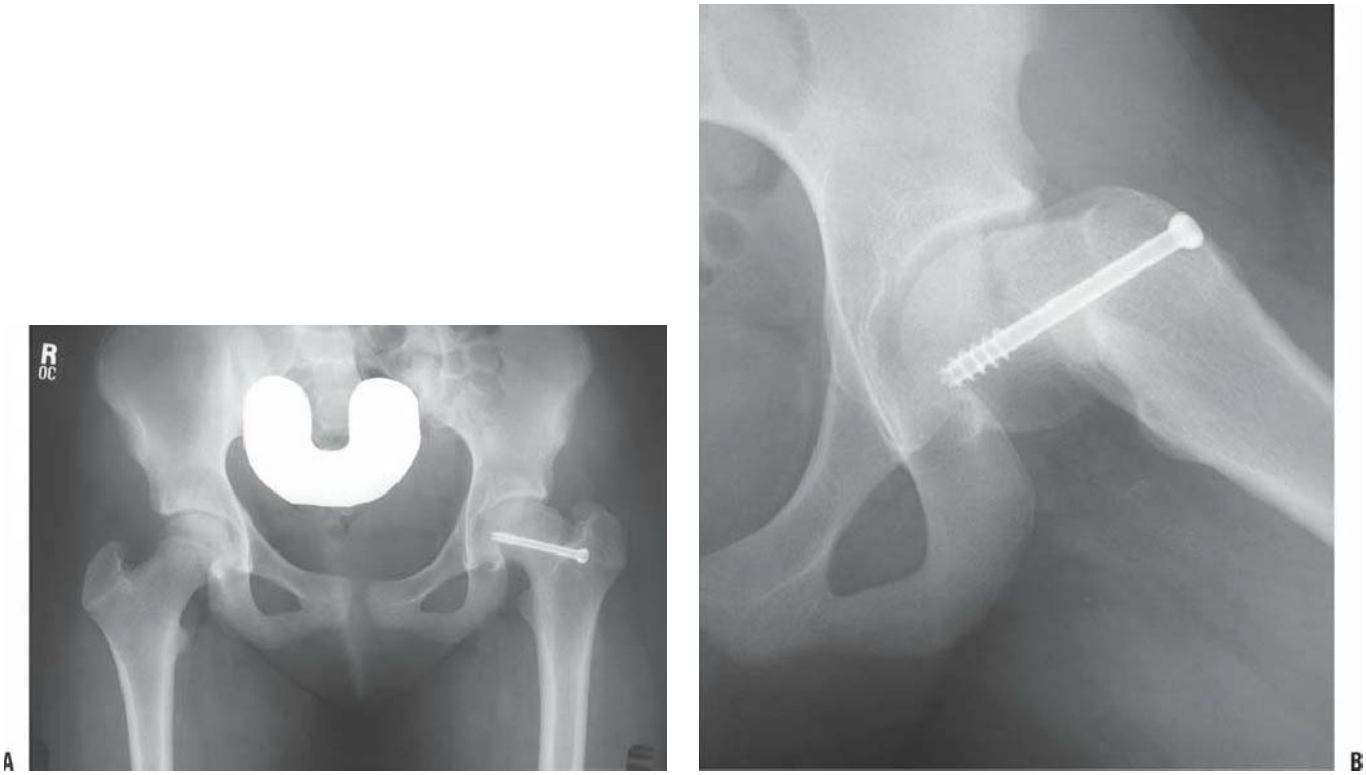


FIGURE 25-16. A 13-year-old girl presented with pain on sitting and difficulty riding a bike because of external rotation of the left hip 16 months following *in situ* fixation. Anteroposterior pelvis **(A)** and lateral **(B)** radiographs of the left hip show the residual deformity 16 months following pinning. Lateral x-ray film shows the proximal femoral metaphysis articulating with the acetabulum preoperatively. Anteroposterior

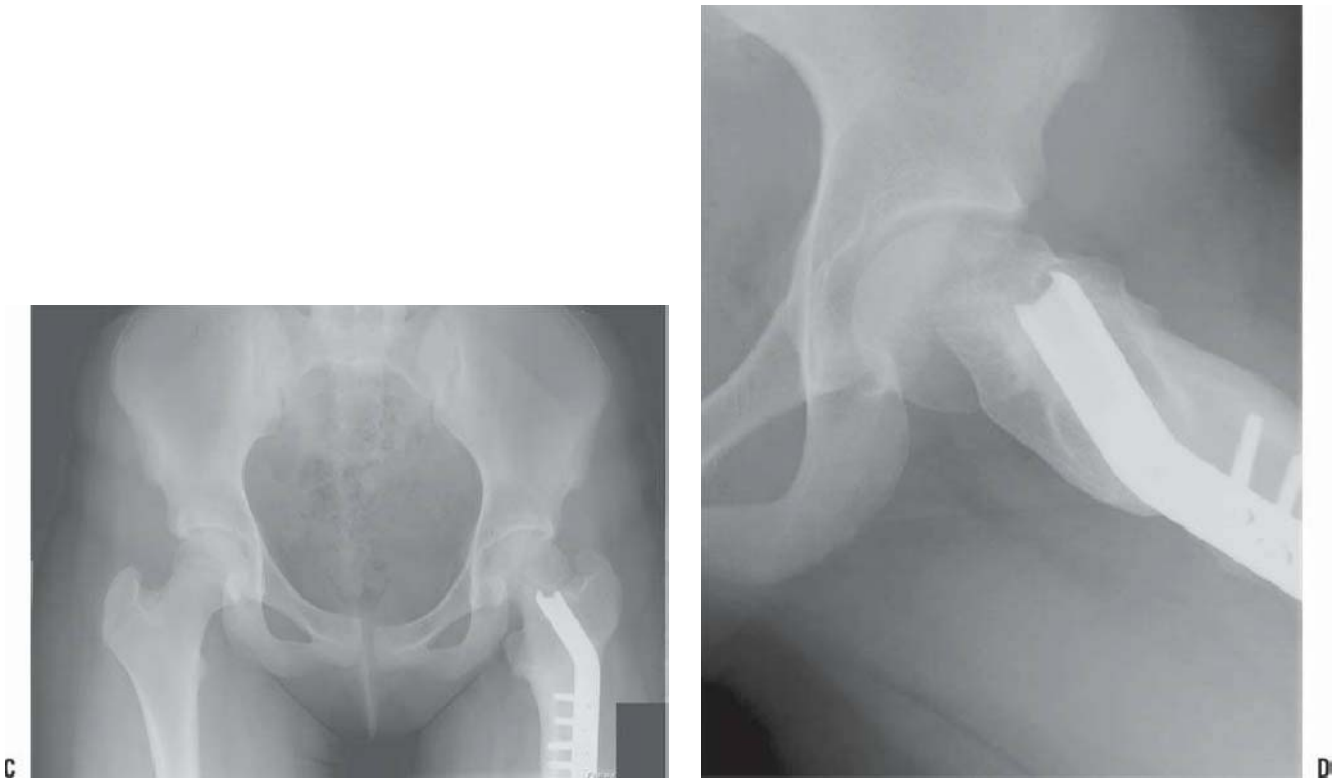


FIGURE 25-16. (*Continued*) **(C)** and lateral **(D)** radiographs show the alignment 1 year after triplanar (flexion–valgus–internal rotation) osteotomy. Following redirectional osteotomy, there is an increased neck–shaft angle, with distal and slightly lateral translation of the greater trochanter (with a resultant increase in the articulotrochanteric distance), and the relation of the femoral head and acetabulum has changed. The metaphysis is no longer intra-articular. A downside of the surgery is that if THA is necessary in the future, distortion of the proximal femoral anatomy will make such a replacement more difficult.

is indicated for SCFE with a displacement >45 degrees, based on the head–shaft angle measurement (329). Although this osteotomy has the potential to significantly enhance the anatomic alignment, it is quite demanding technically and is rarely used.

Sugioka reported 90% excellent results in 10 cases of SCFE treated with this method, with an improved range of motion of the hip in 90% of the patients (329). One case of ON was reported among these 10 hips. Sugioka noted postoperative valgus in three hips that had marked deformity preoperatively.

Another series of five hips has been described, with complications in two patients (40%) (330). One patient had ON and the other had loss of fixation. At <3 years mean follow-up, four hips were clinically asymptomatic. No cases of chondrolysis have been reported in the literature in the 2 small series (a total of 15 patients) of transtrochanteric rotational osteotomies in children with SCFE (329, 330).

Although the results with this technique appear promising, the technique is demanding, complications are common, and its use has been reported in only a few centers.

Authors' Preferred Method. One of the authors (RMK) reserves proximal femoral osteotomies for those patients with significant signs and/or symptoms that persist at least 1 year following *in situ* fixation. Most commonly, these include hip and/or groin pain with prolonged sitting (owing to femoral neck impingement on the acetabulum) and functional limitations due to loss of the hip range of motion. Although such signs and symptoms are common in the first few months following *in situ* fixation, they often improve within 1 year of pinning. If such limitations persist and affect a patient's quality of life, an osteotomy is performed. This is typically a Southwick osteotomy, with valgus, flexion, and internal rotation. The same author (RMK) does not perform prophylactic osteotomies in asymptomatic patients with moderate or severe slips. He thinks that the risks of such osteotomies in asymptomatic patients are too great relative to the uncertain potential for long-term gains, especially given the anticipated ongoing advances in the fields of orthopaedics and basic science during the lifetime of these children. In addition, asymptomatic patients who undergo proximal femoral osteotomy are generally made clinically worse for at least the first 6 months following a proximal femoral osteotomy. The other author (YJK) performs an intertrochanteric osteotomy through a surgical dislocation approach (319) (Fig. 25-17). The proximal femoral osteoplasty will decrease the need for an intertrochanteric correction and hence will decrease the shortening effect on the limb. The same author (YJK) generally performs an Imhauer-type flexion osteotomy with a

Text continued on page 1206

Performing an Intertrochanteric Osteotomy Through a Surgical Dislocation Approach (Fig. 25-17A–G)

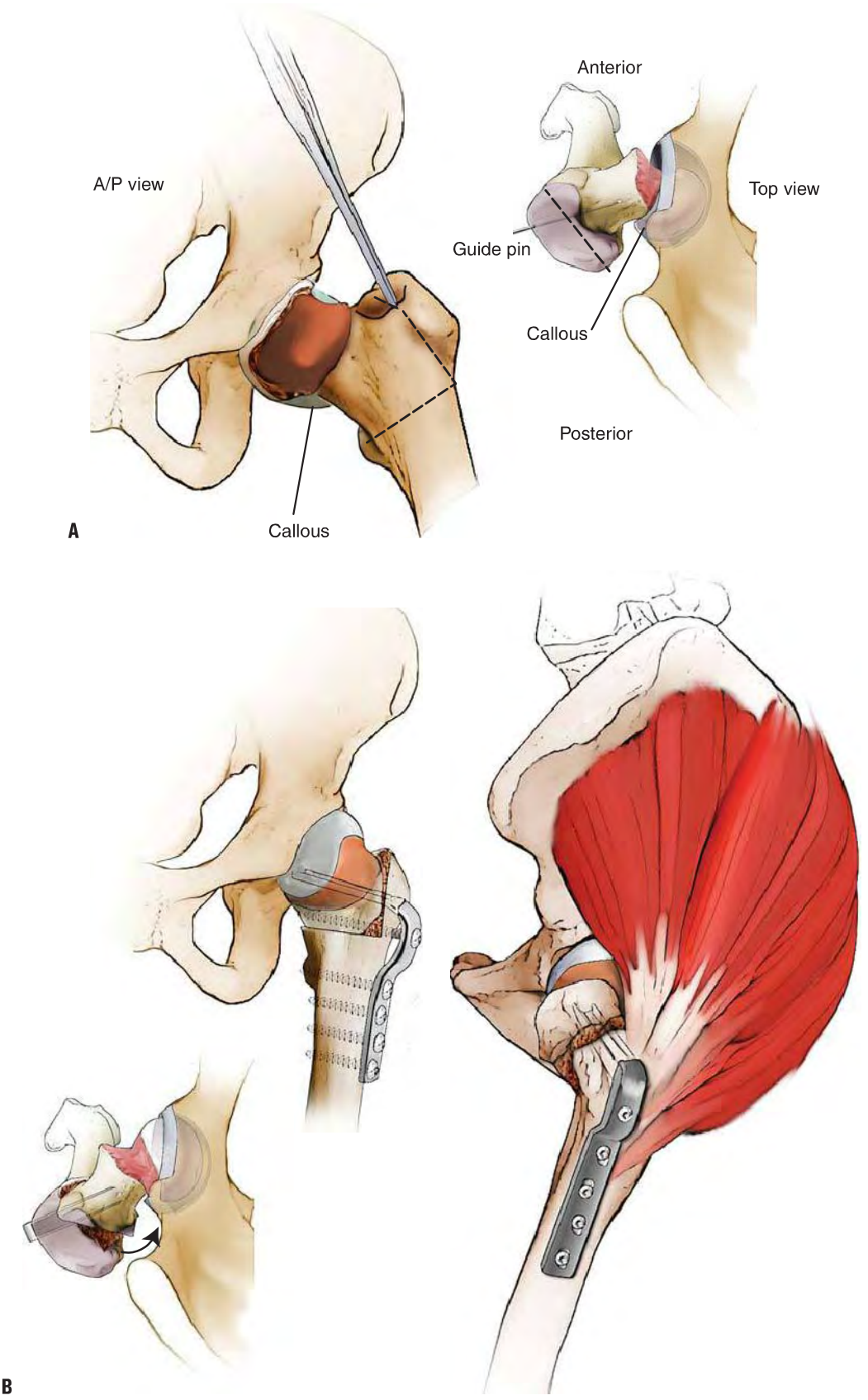


FIGURE 25-17. **Performing an Intertrochanteric Osteotomy Through a Surgical Dislocation Approach** allows the ability to inspect the joint and perform a proximal femoral osteoplasty **(A)**. A pure flexion osteotomy is performed, which will correct the apparent varus appearance of a SCFE **(B)**. The trochanter osteotomy and the femoral osteotomy are fixed simultaneously with a blade plate. The trochanter is placed back in an anatomic position **(C)**.

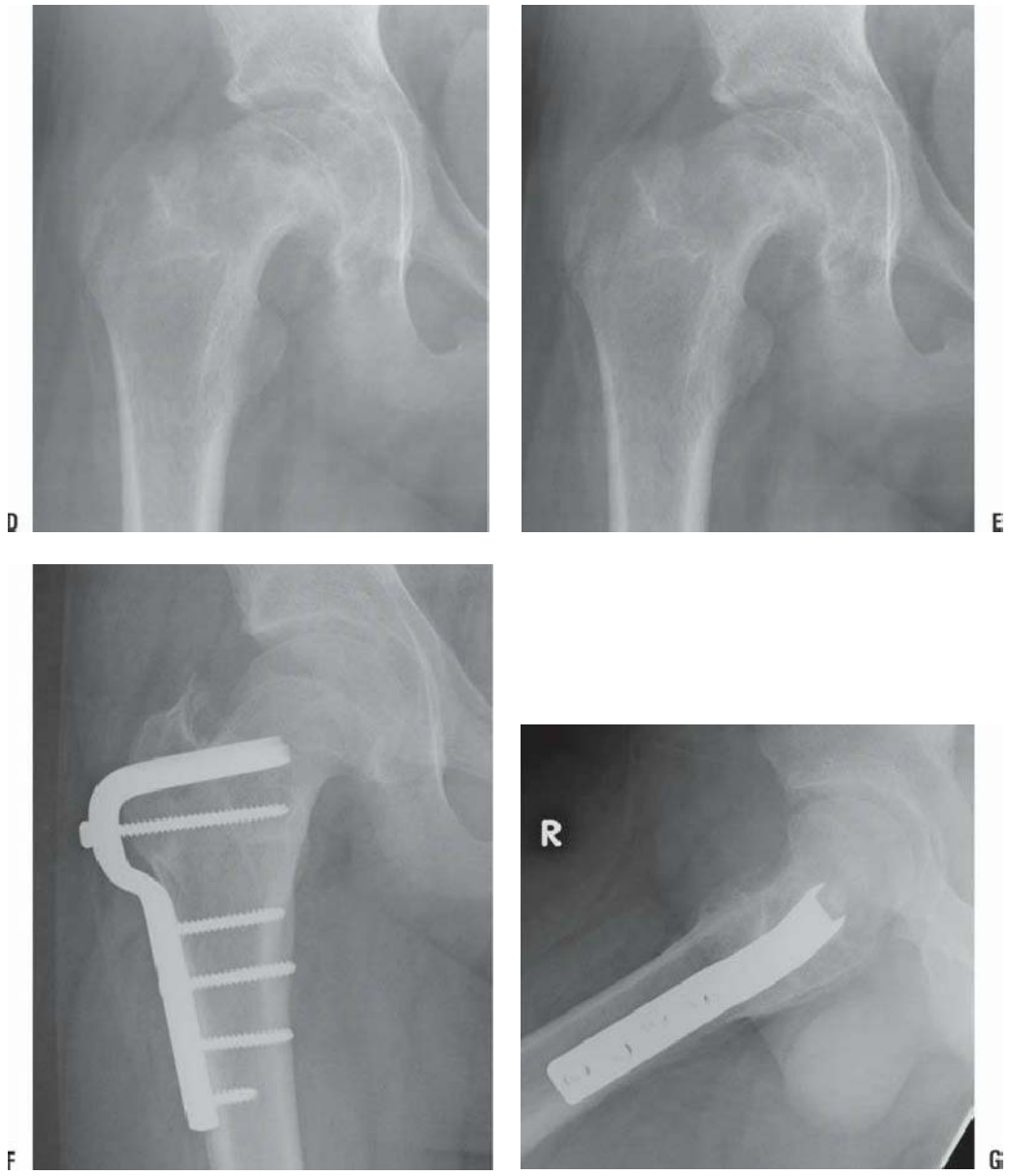


FIGURE 25-17. (*continued*) Panels **(D)** and **(E)** are preoperative radiographs of a 16-year-old with a chronic stable severe SCFE. Panels **(F)** and **(G)** are the postoperative radiographs after a flexion intertrochanteric osteotomy performed through a surgical dislocation approach.

derotation component for its simplicity. The author generally plans on fully correcting the slip angle of the affected hip but does not exceed 50 degrees of correction. Preoperatively, the patient is advised that preexisting articular cartilage damage is commonly found at time of surgery (305) and that OA may progress with time.

Prophylactic Pinning. Prophylactic pinning of the uninvolved hip remains an area of significant controversy in the management of children with SCFE. Recent authors have attempted to weigh the risks and benefits and their recommendations are conflicting. Proponents of prophylactic pinning cite the high rates of bilateral SCFE, the increased risk of OA in patients with SCFE at long-term follow-up, and the decreased risks of prophylactic pinning as technology and techniques improve (9, 269, 331–333). Schultz et al. (334) indicated that prophylactic pinning of the contralateral hip in cases with unilateral SCFE would appear to be beneficial in terms of long-term Iowa hip scores, but cautioned that clinical judgment and patient preferences should be used on a case-by-case basis.

Opponents of prophylactic pinning cite the complications of prophylactic treatment, noting the potential risks of pinning numerous hips that will never slip, and also pointing out that with appropriate patient counseling and close follow-up most subsequent slips will be detected while still mild. Previous authors have reported complication rates of up to 34% with prophylactic pinning of possible SCFEs, although the techniques

and results in some of these studies would not be considered acceptable by current standards (233, 265, 335, 336). In one study of 94 hips treated with prophylactic pinning, there were no complications (9).

When considering the risks and benefits of prophylactic pinning in unilateral SCFE, it is important to consider the data regarding the risk of contralateral SCFE, the anticipated severity and stability of such a slip, and the risks and benefits of observation and of prophylactic treatment. An important consideration in prophylactic pinning is that because the distance the screw must traverse in an unslipped hip is much greater than the distance in a moderate-to-severe SCFE, there is actually less room for error in selecting the starting point and the angle of screw insertion. This geometry dictates that for every few degrees of deviation from the optimal path, the tip of the screw will be more eccentric in the epiphysis in a mild slip (or in a hip that has not slipped) than it would be in a severe slip (Fig. 25-18).

As noted earlier, in children without any evident systemic causes of SCFE, bilateral SCFE is present in approximately 20% at the time of initial presentation, is identified in another 15% to 20% in adolescence, and is present at long-term follow-up in approximately 60% (8–10, 12, 13, 22, 28, 31–33, 104, 108, 110, 113, 117, 125, 164, 166, 181, 331, 337–339). These data indicate that in the 80% of the patients who present with unilateral SCFE, 20% to 25% will develop a contralateral SCFE in adolescence, and half of the 80% will develop a second SCFE by the time of long-term follow-up. It can be inferred from these data that the probability of a contralateral slip first being recognized after adolescence is 25% to 30%. It is likely that these represent minimal, asymptomatic slips that were not recognized during adolescence.

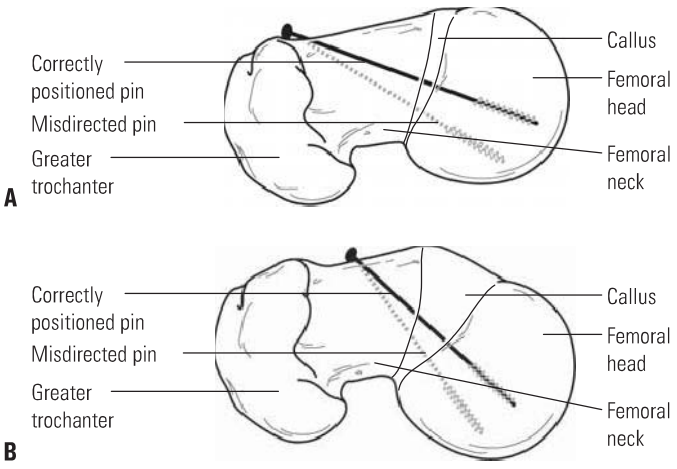


FIGURE 25-18. Despite an accurate starting point on the anterior femoral neck, if the angle of insertion varies from the optimal angle, the hardware will not be perpendicular to the physis and will be eccentric in the femoral head. For a given degree of misdirection, the biomechanical alignment and eccentricity in the femoral head will be worse for a mild slip **(A)** than it will be for a more severe slip **(B)** because of the longer distance the screw must traverse in a mild slip. These problems are most pronounced for a preslip, or a hip that is pinned prophylactically.

Because the risk of major complications is closely tied to the stability of the SCFE, it is also important to estimate the frequency of unstable contralateral metachronous slips. Because 20% to 25% of those presenting with unilateral SCFE will develop a contralateral slip during adolescence, and because 5% to 10% of the SCFEs are unstable, the risk of a contralateral, unstable SCFE occurring during adolescence is 1% to 2%. With appropriate counseling about the risk of contralateral SCFE and potential signs and symptoms thereof, the risk of unstable contralateral SCFE may be even less.

For stable contralateral SCFE, progression to a moderate or a severe contralateral SCFE during adolescence should also be low if there is no underlying systemic cause of SCFE and the child is compliant with follow-up (Fig. 25-19). Most contralateral SCFEs initially noted at the time of long-term radiographic follow-up are also quite mild.

Summarizing these data, it appears that prophylactic pinning of all hips will potentially prevent two complications: ON due to unstable SCFE and late OA. Given the data provided in preceding text, prophylactic pinning will only prevent ON in 1% or fewer of all contralateral hips even if the rate of ON in unstable SCFE is as high as 50%. Prophylactic pinning of contralateral hips in children with unilateral SCFE would potentially decrease OA by 9% in the contralateral hips of these patients, based on Hagglund et al. (31)report of radiographic evidence of OA in 27% of hips (28 of 104) with SCFE compared with 9% of control hips (9 of 101) at a mean follow-up of 33 years and based on his report that the risk of metachronous contralateral SCFE is approximately 50%. Further, Hagglund noted that no hip with a mild or moderate slip treated with *in situ* pinning developed arthritis before the age of 50 years. Because most patients with appropriate follow-up would likely be diagnosed before a severe contralateral slip develops, OA occurring before 50 years of age would be expected to be unusual in contralateral hips (269). Chondrolysis will not be prevented because chondrolysis occurs almost exclusively in treated hips.

These potential benefits of prophylactic fixation must be weighed against the frequency of its potential complications, including those of chondrolysis (2%), ON (1%), and proximal femoral fracture (1%). It is also important to recognize that slip progression occurs postoperatively in 0% to 3% of the cases of SCFE in most of the series of SCFE treated with *in situ* fixation (2, 103, 181, 228, 233, 249, 340).

Prophylactic pinning is commonly performed in certain patient groups. Prophylactic pinning should be performed for children with underlying endocrine disease because of their high rate of contralateral slip. Previous pelvic radiation, which included the contralateral hip in the field, is another indication for prophylactic pinning. Prophylactic pinning should be strongly considered in children younger than 10 years at the time of presentation because of potential LLD following unilateral pinning and the high risk of bilateral involvement in such young children (28, 341). In addition, children residing in more remote areas, who do not have easy access to medical care, should be considered for prophylactic fixation of the contralateral hip. In children with renal disease, medical management rather than prophylactic pinning is recommended.

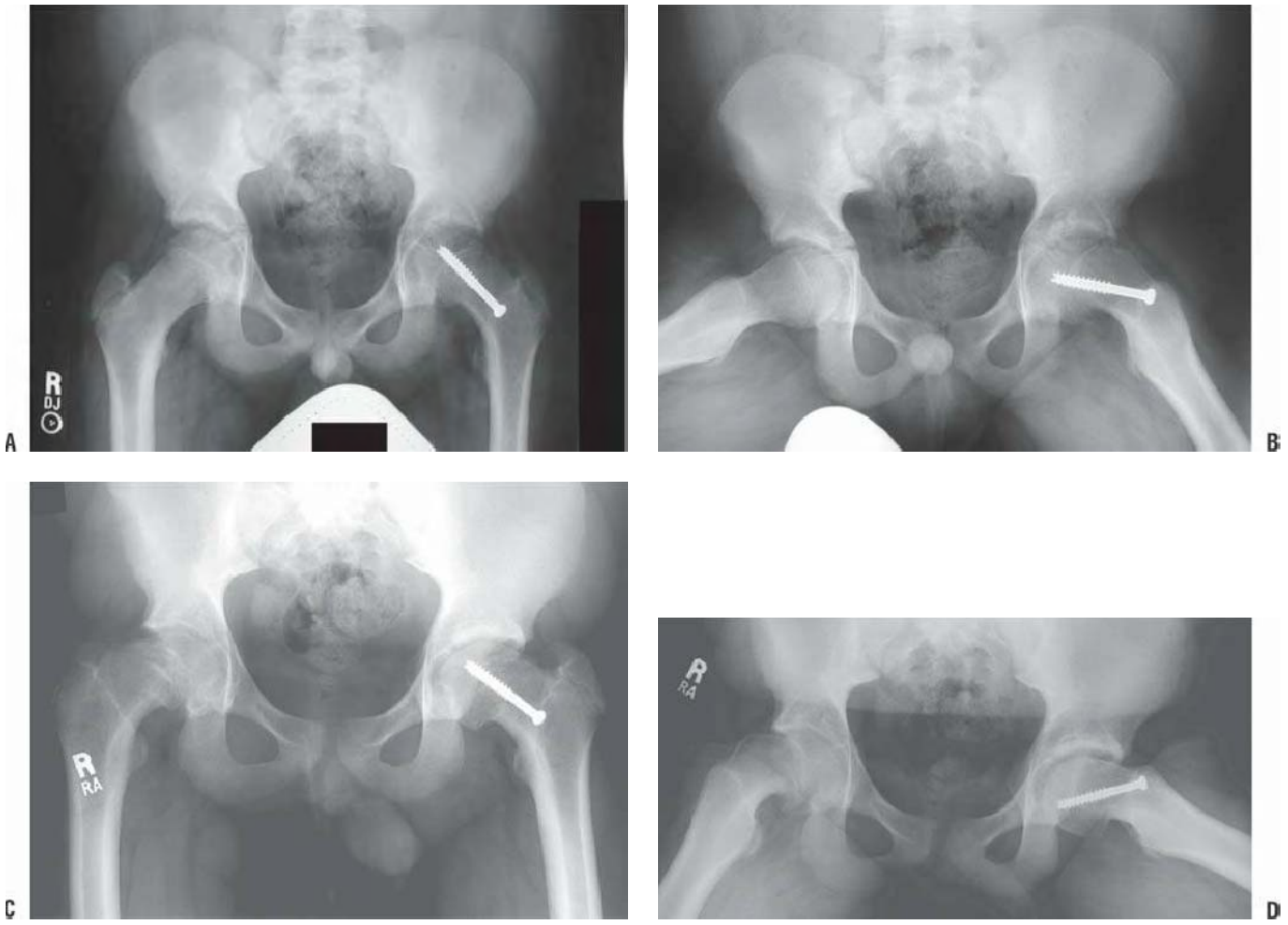


FIGURE 25-19. Anteroposterior **(A)** and frog lateral **(B)** radiographs show a left SCFE following *in situ* fixation with a single cannulated screw. The hardware is aligned adequately. Anterior metaphyseal prominence is evident. Right hip pain began several months later, although the child did not return to the orthopaedist for more than 1 year. At the time of re-presentation, anteroposterior **(C)** and frog lateral **(D)** radiographs demonstrate marked slip of the previously normal right hip. Significant remodeling of the left hip (including resorption of the metaphyseal prominence) is visible, although there is no significant change in the femoral head–neck angle. This case exemplifies the importance of expert clinical and radiographic follow-up of children with SCFE until closure of the proximal femoral physes.

Femoral Neck Osteoplasty. Prominence of the anterosuperior femoral neck following SCFE has been cited as a cause for decreased hip flexion, abduction, and internal rotation (342). Femoral neck osteoplasty involves removal of the prominent anterosuperior femoral neck and may be performed alone or in combination with other procedures, such as proximal femoral osteotomies (124, 342, 343). The goals of such treatment are to enhance hip range of motion and/or to potentially prevent anterior femoroacetabular impingement and OA (150).

A potential indication for osteoplasty is a prominent anterosuperior femoral neck that abuts the acetabulum in cases of chronic SCFE (198, 343). Symptoms that may suggest the potential benefit of osteoplasty include pain on sitting caused by the impingement with hip flexion.

If performed in isolation, osteoplasty leaves unchanged the abnormal relation between the femoral head, neck, and shaft, with relative retroversion, extension, and varus. Because the anatomic relation between the femoral head, neck, and shaft is not changed by osteoplasty, isolated osteoplasty may still result in impingement of the anterior femoral neck against the acetabulum and persistent range of motion deficits. For mild SCFE with symptomatic femoroacetabular impingement, proximal femoral osteoplasty may be beneficial (319). In general, proximal femoral osteoplasty in isolation is reserved for mild SCFE. In more severe SCFE, osteoplasty alone may not be sufficient to relieve the impingement, and an intertrochanteric osteotomy should be performed as the primary procedure. Previous authors have noted that osteoplasty may further enhance hip range of motion following intertrochanteric osteotomies (124, 342, 343).

COMPLICATIONS

Complications of SCFE may occur early or late. The late complications include OA, ON, and chondrolysis. OA is a nearly inevitable late sequela of SCFE whether treated or not, whereas

ON and chondrolysis are often devastating complications that occur almost exclusively following treatment of SCFE.

The most important factor in the long-term outcome in cases of SCFE is avoidance of complications. Hall (275) noted that complications were the only factor that seemed to lead to an early poor result. Previous authors have reported that the results of treated SCFE were often worse than those of untreated SCFE because of the lack of catastrophic complications in untreated hips (32, 169, 344).

Natural history studies confirm that most children who have an uncomplicated course following a SCFE function well until at least the fifth decade. At a mean follow-up of 41 years, Carney and Weinstein (167) reported Iowa hip scores of at least 80 in 26 of 31 hips (84%). At a mean follow-up of 37 years, Ordeberg et al. reported that, of 49 cases of SCFE, only "a few" patients had restrictions regarding their work or social lives, and that only 2 of the 49 (4%) had required surgery for arthritis (169, 345).

Osteoarthritis. OA appears to be an essentially universal sequela of both treated and untreated SCFE because any significant biomechanical derangement of the hip joint can lead to OA if the affected individual reaches old age. The prevalence and severity of OA increase with the increased time to follow-up and increased slip severity. The complications of chondrolysis and ON markedly accelerate the development of OA, with OA occurring in adolescents and young adults who have a history of these serious complications (113).

Biomechanical modeling studies have shown that the deformity associated with SCFE would place the patients with SCFE at long-term risk of OA (145). A SCFE can result in the anterior femoral metaphysis articulating with acetabular cartilage and can also cause impingement of the femoral neck against the anterior acetabulum (145). As noted earlier, in an attempt to potentially decrease this long-term risk of arthritis from such mechanical malalignment, some authors use such biomechanical studies to advocate early osteotomies in children with significant residual deformity following SCFE (89, 94, 292, 320, 321). With the advent of the surgical dislocation approach to hips with SCFE, the extent of articular cartilage damage can now be seen at time of surgery. Leunig et al. (149) have demonstrated that articular cartilage damage occurs very early in SCFE. Furthermore, the amount of damage appears to be related to the duration of symptoms of a SCFE (305). Although currently there are no clinical data to support prophylactic realignment for the purposes of preventing OA, the dilemma remains that when symptoms are present, the amount of articular cartilage damage may be too extensive (319) for the realignment procedure to be effective in salvaging the joint long term.

Long-term studies show significantly increased rates of OA in patients with a history of SCFE (31, 181, 238). Many authors have also reported increasing rates of OA with increasing degrees of SCFE at long-term follow-up (113, 125, 166, 168, 169, 181). Hagglund (269) noted that no hip with a mild or moderate slip treated with *in situ* pinning developed arthritis before 50 years of age.

Despite SCFE affecting between 1 in 1000 and 1 in 2000 persons, only 2% to 9% of those with end-stage OA have been reported to have a history of SCFE (170–174). Studies of adults undergoing THA have shown that up to 40% of these patients have evidence of pediatric hip disease, including SCFE, at the time of joint arthroplasty (177). Radiographic studies of adult hips also demonstrate the stigmata of pediatric hip disease in up to 40% of those with findings of OA (176). Not all authors agree with these studies, and some have noted that these radiographic findings are common in end-stage OA of various etiologies (178).

As noted previously, severe deformity following SCFE results in significant biomechanical changes in the hip because a portion of the proximal femoral metaphysis articulates with the acetabulum and leads to accelerated degenerative changes in the hip. Recent authors have sought to prevent late arthritis by restoring more normal proximal femoral anatomy by performing proximal femoral redirectional osteotomies (89, 292). Although such authors report an apparent decrease in OA following realignment at follow-up beyond 20 years in each study (89, 292), longer follow-up will be needed to know if these apparently superior results continue in the ensuing decades, and to decide whether such procedures are indicated in patients who are asymptomatic despite significant residual deformity.

In summary, OA appears to be an almost inevitable sequela of both treated and untreated SCFE, with earlier onset and more severe degeneration in high-degree slips. The complications of ON and chondrolysis greatly accelerate the development of OA and often lead to end-stage OA in adolescence.

Osteonecrosis. ON is, along with chondrolysis, one of the two most serious complications encountered in the treatment of children with SCFE. ON is reported to occur in 4% to 25% of the cases of SCFE in most series and is found almost exclusively in hips classified as acute on a temporal basis or unstable (as classified by Loder). The rate of ON is most commonly reported as 10% to 15% in acute or acute-on-chronic SCFE (104, 113, 142, 240).

There appear to be two main potential causes of ON in children with SCFE: disruption of the blood supply preoperatively and disruption of the blood supply due to the surgery itself. With current techniques, including recognition of the posterosuperior blood supply to the femoral head and the importance of accurate hardware placement, the risk of iatrogenic ON should decrease.

Whether or not the degree of slip influences the rate of ON in unstable SCFE is debated. In two series in which ON was noted to occur only in unstable SCFE, Kennedy et al. (239) reported that the degree of slip does not appear to be an independent predictor of ON, whereas Tokmakova et al. (215) reported that the degree of slip is a risk factor for developing ON. When considering this, however, the degree of displacement evident radiographically at the time of presentation may have no relation to the true amount of maximal displacement that has already occurred or will occur prior to operative stabilization.

In addition to the slip stability at presentation, the method of treatment affects the rate of ON. The incidence of ON following proximal femoral osteotomy is greatest with

subcapital osteotomies and progressively decreases with more distal osteotomies (24, 256, 293, 296, 298–301, 306, 307). Most authors report ON rates of 5% to 35% following subcapital osteotomy, with rates at times as high as 42% (24, 256, 275, 289, 293, 296, 298–301, 306, 307). Base-of-neck osteotomies result in ON in 0% to 5% of the cases (264, 308, 309). Most authors do not report any cases of ON following intertrochanteric osteotomy for SCFE (124, 289, 324, 352), although rates of up to 6% have been reported (292, 322, 326, 327).

ON occurs in 0% to 5% of patients treated with *in situ* pinning, with rates most commonly at 2% to 3% in recent series (2, 202, 204, 208, 212, 216, 220, 228, 244, 254). The rate of ON reported following bone peg epiphysiodesis is generally between 0% and 6% (186–188, 196, 198, 199).

Two recent studies report no cases of ON in a total of more than 50 cases of SCFE treated in spica casts (180, 182). In these two series, the overall breakdown of slips was 76% chronic, 21% acute-on-chronic, and 3% acute. Many cases of ON previously associated with spica casting were likely due to a manipulative reduction prior to cast application and/or positioning in the cast.

The prognosis of ON associated with SCFE is poor, although it is better than the prognosis attributed to ON from other causes (346). In a series of 22 patients with 24 cases of SCFE complicated by ON, who were followed an average of 31 years, 9 of the hips (38%) had required salvage treatment and the other 15 hips had osteoarthritic changes that were evident radiographically (346).

If ON is diagnosed prior to femoral head collapse, treatment is aimed at maintenance of the range of motion, prevention of progressive femoral head collapse, and joint preservation when possible. The combination of anti-inflammatory medications, physical therapy, and protected weight bearing may be helpful in maintaining the range of motion and preventing progressive femoral head collapse. When femoral head collapse occurs in the area of previously placed screws, the screws must often be backed out or removed in order to prevent joint penetration and chondrolysis.

Joint-preserving procedures, including redirectional osteotomies (317, 347–351), vascularized fibular grafting (352), and bone grafting procedures (353), have been reported following ON in children, although no large series has been reported specifically addressing ON following SCFE.

If ON is not diagnosed until after femoral head collapse, the long-term prognosis is significantly worse. With progressive collapse and joint degeneration, salvage procedures are often necessary.

In summary, ON is one of the devastating complications of SCFE. With the passage of time, hips with ON complicating SCFE will inexorably develop arthritic changes if left untreated. Even if ON is detected early, salvage procedures are often necessary for these hips. As a result, one of the prime goals in the treatment of SCFE should be the avoidance of ON.

Authors' Preferred Method. The authors do not routinely screen children for ON after SCFE, whether the SCFE is stable or unstable. However, if a child initially does well in the weeks or months immediately after surgery and then begins to have recurrent hip symptomatology, ON must be considered. Alternatively, ON may first be evident months after fixation on routine follow-up radiographs. In either suspected or documented ON, MRI is a critical part of a thorough evaluation.

It is important to remember that, although imperfect, treatment of ON carries a better prognosis when undertaken prior to femoral head collapse. Free vascularized fibular grafting, despite donor site morbidity, appears to be the most appropriate option for a hip with segmental ON involving the weight-bearing portion of the femoral head before femoral head collapse. If ON is not detected until after femoral head collapse occurs, redirectional femoral osteotomy may be considered if a sufficient pillar of viable bone can be moved into a weight-bearing position.

Whenever femoral head collapse occurs, it is important to remove any hardware that is protruding into the joint and to replace screws into another area of the head so long as the physis remains open.

If surgical intervention is not undertaken in children with ON, either because it is not indicated (as with severe collapse) or because such treatment is declined by the patient's family, conservative measures should be undertaken in an attempt to delay salvage treatment. Impact activities such as running, jumping, and ball sports should be avoided, whereas swimming and bicycling may be undertaken to maintain cardiovascular fitness, strength, and range of motion. Anti-inflammatory medications and ambulatory aids may be beneficial as well, although these are often rejected by otherwise healthy adolescents and young adults.

Chondrolysis. Although first described in conjunction with SCFE in 1930, chondrolysis remains poorly understood (354). Chondrolysis involves cartilage destruction of both the femoral head and the acetabulum, and is defined as the triad of pain, decreased hip range of motion, and radiographic joint space narrowing (Fig. 25-20). Normal cartilage thickness of the pediatric hip has been reported to decrease from a mean of 6 mm in children aged 1 to 7 years, to 5 mm in those aged 8 to 12 years, and to 4 mm in those aged 13 to 17 years (355). Chondrolysis has been reported to occur in 0% to 28% of patients with SCFE (4, 107, 181, 204, 208, 243, 247, 356–358).

Chondrolysis should be suspected if synovitis and hip range of motion are not improved in the first 2 to 3 weeks following surgery, and any child with decreasing range of motion postoperatively must be suspected of having chondrolysis. Unlike many other hip maladies, chondrolysis causes the hip to be held in abduction and ultimately results in a fixed abduction contracture.

Chondrolysis is more common in the female population than in the male population (243, 245, 359). Previously, chondrolysis was believed to be more common in black children (76, 180, 212, 243, 356, 360), although more recent studies have refuted this assertion (202, 216, 243, 360, 361). One series reported a higher incidence of chondrolysis in those of Hawaiian descent (357).

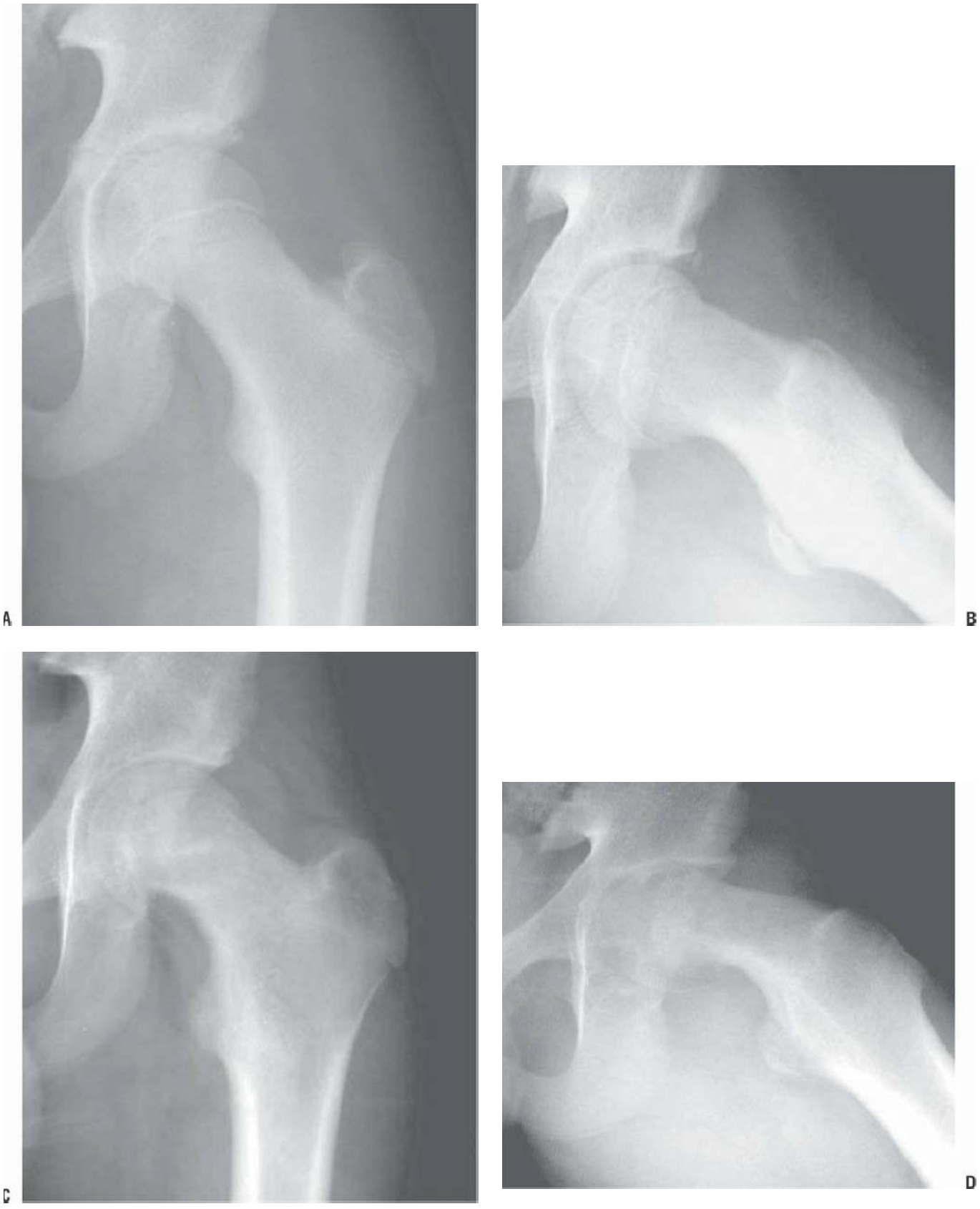


FIGURE 25-20. Left hip chondrolysis in a 13-year-old boy. **A,B:** Normal joint space of the left hip when the patient presented with a right SCFE. Ten months later, the patient presented to the office with a 1-month history of left hip pain. **C,D:** Radiographs at that time demonstrated a left SCFE and joint space narrowing. The left hip was pinned *in situ* with prompt symptom resolution. However, 2 months postoperatively the patient began to have increased hip pain, difficulty walking, and decreased hip range of motion.

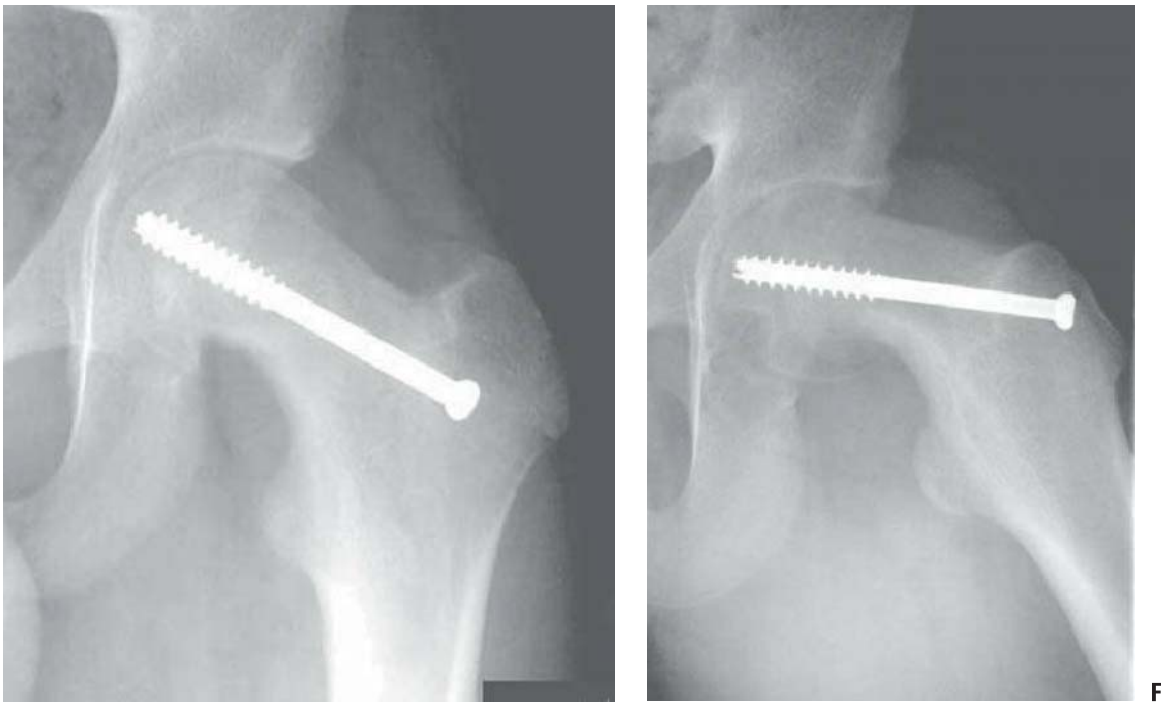


FIGURE 25-20. (*Continued*) **E,F:** Radiographs at that time revealed mild additional joint space narrowing. This case demonstrates that joint space narrowing can occur without treatment but that chondrolysis is not simply joint space narrowing. Rather, it is the triad of joint space narrowing, pain, and decreased.

Chondrolysis is seen following all forms of treatment and has also been reported to be present at the time of initial presentation in some patients (243, 245). Maurer and Larsen (357) suggested that chondrolysis was more common with severe slips and with spica casting, open reduction, or prolonged casting. Ingram et al. (243) noted that chondrolysis was more common in acute-on-chronic slips, and that the highest rates occurred with osteotomies and the lowest rates with *in situ* fixation. Chondrolysis in the unaffected hip has been reported following immobilization (32, 183).

Chondrolysis appears most common following treatment of SCFE with spica casting, with its incidence reported as 14% to 53% of the cases (180–182, 299, 300). Rates of chondrolysis are commonly reported as 3% to 18% following subcapital osteotomy (256, 296, 297, 301, 302, 307), 2% to 10% following base-of-neck osteotomy (264, 308), and 2% to 25% following intertrochanteric osteotomy (124, 243, 289, 310, 322, 324, 327). Reported rates of chondrolysis following *in situ* pinning and bone peg epiphysiodesis are most commonly <5% (2, 24, 30, 125, 181, 186–188, 196, 199, 202, 204, 208, 212, 216, 220, 228, 243).

In addition to narrowing of the joint space, radiographs may also reveal premature closure of the apophysis of the greater and lesser trochanters (243, 362). Bone scan has been noted to demonstrate decreased activity in the apophysis of the greater trochanter in 47% of hips affected with chondrolysis, a finding that may precede radiographic changes (363). In cases with an unusual presentation, workup for a septic hip including joint aspiration may be indicated.

Chondrolysis varies from a relatively minor, self-limited condition from which full recovery may occur to the rapid destruction of a joint necessitating salvage treatment in teenagers (245, 358, 364). Despite decades of experience in treating children with chondrolysis, the reasons for such disparate prognoses remain unclear. When an individual patient presents with chondrolysis, it is still impossible to accurately predict the child's prognosis.

Treatment of chondrolysis is generally conservative, with a combination of protected weight bearing, physical therapy (for range of motion and attempted strengthening of the hip musculature), and oral anti-inflammatory medications. Distraction of the hip joint with external fixation has been reported to be of value in selected cases (365). Failure of such conservative measures may require surgical intervention such as arthrodesis or arthroplasty.

Chondrolysis remains one of the most devastating complications of SCFE. Early recognition and treatment are indicated, but the prognosis following this complication is guarded. In patients unresponsive to conservative measures, salvage procedures may be necessary.

SALVAGE PROCEDURES

Most hips with SCFE will function well into the fifth to the seventh decades of life if the complications of chondrolysis and ON are avoided. Unfortunately, if treatment is complicated by chondrolysis and/or ON, rapid clinical deterioration may occur in adolescence or early adulthood. Significant symptomatology such as pain with sitting, with sleeping, and with activities of daily living may necessitate salvage

treatment. In the child with such significant symptomatology following SCFE that has been complicated by ON, joint-preserving procedures including osteotomies, vascularized fibular grafting, and bone grafting procedures are sometimes possible, although such procedures are not beneficial in children with chondrolysis following SCFE because of their diffuse joint destruction.

If such significant symptoms are present in hips that are not good candidates for joint-sparing procedures, then salvage treatment with hip fusion or hip arthroplasty should be considered. The extent of hip disease in both hips is important in making the decision regarding hip arthrodesis versus arthroplasty. Patients can be thought of as falling into one of three categories: a unilateral salvage hip with a normal contralateral hip, a unilateral salvage hip with a mild contralateral SCFE, or bilateral salvage hips. In an adolescent with a unilateral salvage hip and either a normal contralateral hip or a mild contralateral SCFE, either hip fusion or arthroplasty may be considered. Hip fusion should never be considered in a case with bilateral salvage hips.

Hip fusions have traditionally been the treatment of choice for a degenerated hip in adolescents with unilateral hip disease because of the poor long-term results of THA in heavy, young, active patients (366–369). Currently, many teenagers and their families are reluctant to accept the physical limitations associated with hip arthrodesis, despite the less than stellar results of hip arthroplasty in young patients.

An articulation between metal and ultrahigh-molecular-weight polyethylene has been the gold standard since the earliest total joint replacements. The long-term results in young patients have been inferior to those in older patients, with much of the loosening attributable to the generation of particulate debris and component loosening (171, 368–371). New bearing surfaces such as those including highly crosslinked polymers, metal-on-metal, and ceramics have given hope to a new generation of surgeons and patients about the potential for hip arthroplasty in the young and active population. However, the current reality is that THA is expected to have a limited lifespan in this patient population and that multiple revisions will likely be necessary throughout adulthood.

Even with these constraints, hip fusion should only be considered if the contralateral hip is normal or has only a mild SCFE due to the increased demand that would be placed on the contralateral hip following hip fusion of the affected hip. Many procedures have been described for hip fusion including intra-articular and extra-articular fusion using a variety of fixation devices including screws, plates, and external fixation (372–377). The most common hip fusion technique in children currently is an intra-articular fusion with subtrochanteric osteotomy, which has been reported by multiple authors since it was first reported by Farkas in 1939 (374, 378–380). This technique is felt to promote fusion because contact between the femoral head and acetabulum can be maximized and the long lever arm of the leg is avoided by performing the subtrochanteric osteotomy. With the typical deformity following SCFE and ON, optimizing the fit between femoral head and acetabulum would result in unacceptable positioning of the leg without subtrochanteric osteotomy. Further, the alignment of the leg can be readjusted postoperatively if needed.

The advantages of hip fusion include the durability of the fusion and the ability to return to full activity, including manual labor. Long-term results in studies with mean follow-up exceeding 35 years have been quite satisfying, although reported findings include back pain in 57% to 61%, ipsilateral knee pain in 45% to 57%, and contralateral hip pain in 17% to 27% (373, 377). Conversion to total joint arthroplasty was reported in 13% to 21% in the two studies (373, 377).

Other authors have reported results following the conversion of hip arthrodesis to THA (381–383). There is reliable relief of back, hip, or knee pain, although the results are not comparable to those with primary joint replacements. The results are also better in hips that have fused spontaneously than in those that have undergone surgical fusion. Technically, the conversion procedure is much easier and the results are better if the abductor musculature was not disturbed with the initial procedure.

In a child with bilateral salvage hips, arthrodesis is not an option and arthroplasty should be considered if symptoms are severe. The advantages of THA are the rapid restoration of motion and function without added stress across the contralateral hip, ipsilateral knee, and the spine. Because many of these patients are rather active, once the THA renders them essentially asymptomatic early failure is a frequent result. In a recent series of primary THA in patients 50 years and younger at implantation, THA survivorship was only 54% at 15-year follow-up (369). Others have reported actual or potential loosening in 57% of the prostheses at the 5-year follow-up in patients who had undergone THA prior to 30 years (367). One study of THA in patients aged <50 years reported more encouraging results, noting that the survivorship of the original prosthesis was 63% in patients living at least 25 years postoperatively (171).

If a significantly symptomatic joint that is not amenable to redirectional osteotomy needs salvage treatment, the two options that remain are THA and hip arthrodesis. Neither of these treatments has outstanding long-term results for the hip and the other joints of the lower extremity and spine. Currently, decisions continue to be made on a case-by-case basis to choose between THA (with better short-term hip function and less risk to other joints) and hip arthrodesis (with better long-term durability but more risk to the remainder of the lower extremities and the spine). Promising technologic advances in bearing surfaces hold hope for the future of THA in young, active patients.

Authors' Preferred Method. Obviously, both hips must be considered when weighing the most appropriate salvage treatment of a given hip, as well as the current and future demands that would be placed upon the hip. As noted, hip arthrodesis and arthroplasty are both reasonable options in an adolescent with a unilateral salvage hip and no or mild contralateral hip disease, and arthroplasty is the only reasonable choice in a child with bilateral salvage hips.

The decision between the two suboptimal options for treating a unilateral salvage hip in adolescents with SCFE remains difficult. In the short term, the range of motion and rehabilitation benefits clearly favor arthroplasty. Although the patients can walk extremely well following arthrodesis, within several years of an arthrodesis they may have back and ipsilateral knee pain at the end of the day. These adolescents also often struggle with routine activities including donning and doffing socks, cutting toenails, riding a bicycle, and climbing stairs.

In the long term, the trade-offs are more difficult to define precisely. The risks of arthroplasty involve the affected hip itself, and can be marked and repetitive because of wear, loosening, and dislocation. In contrast, the complications of arthrodesis most frequently are resultant degeneration in the lumbar spine, ipsilateral knee, and contralateral hip. An additional confounding variable is the uncertainty of the future changes in orthopaedics and basic science, which may dramatically alter the implications of these long-term risks.

We are very reluctant to recommend arthroplasty in adolescents exceeding a weight of 200 pounds, given the high demands placed on such a joint. For those weighing <200 pounds, reasonable options include both hip fusion and hip replacement, though impact-type sporting activities must be avoided following either surgery. Some young women are not interested in arthrodesis because of concerns regarding sexuality and childbearing. Only the patient and the family can make the ultimate decision between arthroplasty and arthrodesis.

REFERENCES

1. Key JA. Epiphyseal coxa vara or displacement of the capital epiphysis of the femur in adolescence. *J Bone Joint Surg Am* 1926;8:53–117.
2. Aronson DD, Carlson WE. Slipped capital femoral epiphysis. A prospective study of fixation with a single screw. *J Bone Joint Surg Am* 1992;74(6):810–819.
3. Bennet GC, Koreska J, Rang M. Pin placement in slipped capital femoral epiphysis. *J Pediatr Orthop* 1984;4(5):574–578.
4. Dreghorn CR, Knight D, Mainds CC, et al. Slipped upper femoral epiphysis—a review of 12 years of experience in Glasgow (1972–1983). *J Pediatr Orthop* 1987;7(3):283–287.
5. Henrikson B. The incidence of slipped capital femoral epiphysis. *Acta Orthop Scand* 1969;40(3):365–372.
6. Jacobs B. Diagnosis and natural history of slipped capital femoral epiphysis. *Instr Course Lect* 1972;21:167–173.
7. 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;5(2):80–84.
8. Oram V. Epiphysiolysis of the head of the femur; a follow-up examination with special reference to end results and the social prognosis. *Acta Orthop Scand* 1953;23(2):100–120.
9. Seller K, Raab P, Wild A, et al. Risk-benefit analysis of prophylactic pinning in slipped capital femoral epiphysis. *J Pediatr Orthop B* 2001;10(3):192–196.
10. Sorensen KH. Slipped upper femoral epiphysis. Clinical study on aetiology. *Acta Orthop Scand* 1968;39(4):499–517.
11. Kelsey JL. The incidence and distribution of slipped capital femoral epiphysis in Connecticut. *J Chronic Dis* 1971;23(8):567–578.
12. Hagglund G, Hansson LI, Ordeberg G. Epidemiology of slipped capital femoral epiphysis in southern Sweden. *Clin Orthop* 1984;191:82–94.
13. Jerre R, Karlsson J, Henrikson B. The incidence of physiolysis of the hip: a population-based study of 175 patients. *Acta Orthop Scand* 1996;67(1):53–56.
14. Kelsey JL, Keggi KJ, Southwick WO. The incidence and distribution of slipped capital femoral epiphysis in Connecticut and Southwestern United States. *J Bone Joint Surg Am* 1970;52(6):1203–1216.
15. Loder RT. The demographics of slipped capital femoral epiphysis. An international multicenter study. *Clin Orthop* 1996;322(322):8–27.
16. Lehmann CL, Arons RR, Loder RT, et al. The epidemiology of slipped capital femoral epiphysis: an update. *J Pediatr Orthop* 2006;26(3):286–290.
17. Kelsey JL, Keggi KJ. An epidemiological study of the effect of fluorides in drinking water on the frequency of slipped capital femoral epiphysis. *Yale J Biol Med* 1971;44(3):274–285.
18. Papavasiliou KA, Kirkos JM, Kapetanos GA, et al. Potential influence of hormones in the development of slipped capital femoral epiphysis: a preliminary study. *J Pediatr Orthop B* 2007;16(1):1–5.
19. Loder RT, Farley FA, Herzenberg JE, et al. Narrow window of bone age in children with slipped capital femoral epiphyses. *J Pediatr Orthop* 1993;13(3):290–293.
20. Morsher E. Strength and morphology of growth cartilage under the hormonal influence of puberty. *Reconstr Surg Traumatol* 1968;10:3.
21. Puylaert D, Dimeglio A, Bentahar T. Staging puberty in slipped capital femoral epiphysis: importance of the triradiate cartilage. *J Pediatr Orthop* 2004;24(2):144–147.
22. Moreau MJ. Remodelling in slipped capital femoral epiphysis. *Can J Surg* 1987;30(6):440–442.
23. Wilson PD. The treatment of slipping of the upper femoral epiphysis with minimal displacement. *J Bone Joint Surg Am* 1938;20(2):379–399.
24. Wilson PD, Jacobs B, Schecter L. Slipped capital femoral epiphysis: an end-result study. *J Bone Joint Surg Am* 1965;47:1128–1145.
25. Brenkel IJ, Dias JJ, Davies TG, et al. Hormone status in patients with slipped capital femoral epiphysis. *J Bone Joint Surg Br* 1989;71(1):33–38.
26. Wilcox PG, Weiner DS, Leighley B. Maturation factors in slipped capital femoral epiphysis. *J Pediatr Orthop* 1988;8(2):196–200.
27. Kelsey JL, Acheson RM, Keggi KJ. The body build of patients with slipped capital femoral epiphysis. *Am J Dis Child* 1972;124(2):276–281.
28. 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(8):1141–1147.
29. Siegel DB, Kasser JR, Sponseller P, et al. Slipped capital femoral epiphysis. A quantitative analysis of motion, gait, and femoral remodeling after in situ fixation. *J Bone Joint Surg Am* 1991;73(5):659–666.
30. Bianco AJ Jr. Treatment of slipping of the capital femoral epiphysis. *Clin Orthop* 1966;48:103–110.
31. Hagglund G, Hansson LI, Ordeberg G, et al. Bilaterality in slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1988;70(2):179–181.
32. Jerre T. A study in slipped upper femoral epiphysis. *Acta Orthop Scand* 1950;(Suppl 6):1–157.
33. Billing L, Severin E. Slipping epiphysis of the hip; a roentgenological and clinical study based on a new roentgen technique. *Acta Radiol* 1959;51(Suppl 174):1–76.
34. Bhatia NN, Pirpiris M, Otsuka NY. Body mass index in patients with slipped capital femoral epiphysis. *J Pediatr Orthop* 2006;26(2):197–199.
35. Koenig KM, Thomson JD, Anderson KL, et al. Does skeletal maturity predict sequential contralateral involvement after fixation of slipped capital femoral epiphysis? *J Pediatr Orthop* 2007;27(7):796–800.
36. Riad J, Bajelidze G, Gabos PG. Bilateral slipped capital femoral epiphysis: predictive factors for contralateral slip. *J Pediatr Orthop* 2007;27(4):411–414.
37. Brown D. Seasonal variation of slipped capital femoral epiphysis in the united states. *J Pediatr Orthop* 2004;24(2):139–143.
38. Loder RT. A worldwide study on the seasonal variation of slipped capital femoral epiphysis. *Clin Orthop* 1996;322:28–36.
39. Maffulli N, Douglas AS. Seasonal variation of slipped capital femoral epiphysis. *J Pediatr Orthop B* 2002;11(1):29–33.
40. Speer DP. The John Charnley Award Paper. Experimental epiphysiolysis: etiologic models slipped capital femoral epiphysis. *The hip: proceedings of the tenth open scientific meeting of the Hip Society*. St. Louis, MO: C.V. Mosby Company, 1982:68–88.
41. Loder RT, Wittenberg B, DeSilva G. Slipped capital femoral epiphysis associated with endocrine disorders. *J Pediatr Orthop* 1995;15(3):349–356.

42. McAfee PC, Cady RB. Endocrinologic and metabolic factors in atypical presentations of slipped capital femoral epiphysis. Report of four cases and review of the literature. *Clin Orthop* 1983;180:188–197.
43. Primiano GA, Hughston JC. Slipped capital femoral epiphysis in a true hypogonadal male (Klinefelter's mosaic XY-XXY). A case report. *J Bone Joint Surg Am* 1971;53(3):597–601.
44. Qadan L, Al-Quaimi M, Ahmad A. Slipped capital femoral epiphysis associated with primary hyperparathyroidism and severe hypercalcemia. *Clin Pediatr (Phila)* 2003;42(5):439–441.
45. Rennie W, Mitchell N. Slipped femoral capital epiphysis occurring during growth hormone therapy. Report of a case. *J Bone Joint Surg Br* 1974; 56-B(4):703–705.
46. Zubrow AB, Lane JM, Parks JS. Slipped capital femoral epiphysis occurring during treatment for hypothyroidism. *J Bone Joint Surg Am* 1978;60(2):256–258.
47. Harris WR. The endocrine basis for slipping of the upper femoral epiphysis: an experimental study. *J Bone Joint Surg Br* 1950;32(1):5–11.
48. Hillman JW, Hunter WA Jr, Barrow JA III. Experimental epiphysiolysis in rats. *Surg Forum* 1957;8:566–571.
49. Oka M, Miki T, Hama H, et al. The mechanical strength of the growth plate under the influence of sex hormones. *Clin Orthop* 1979;145: 264–272.
50. Williams JL, Vani JN, Eick JD, et al. Shear strength of the physis varies with anatomic location and is a function of modulus, inclination, and thickness. *J Orthop Res* 1999;17(2):214–222.
51. Benjamin B, Miller PR. Hypothyroidism as a cause of disease of the hip. *Am J Dis Child* 1938;55:1189–1211.
52. Burrow SR, Alman B, Wright JG. Short stature as a screening test for endocrinopathy in slipped capital femoral epiphysis. *J Bone Joint Surg Br* 2001;83(2):263–268.
53. Heatley FW, Greenwood RH, Boase DL. Slipping of the upper femoral epiphyses in patients with intracranial tumours causing hypopituitarism and chiasmal compression. *J Bone Joint Surg Br* 1976;58(2): 169–175.
54. Heyerman W, Weiner D. Slipped epiphysis associated with hypothyroidism. *J Pediatr Orthop* 1984;4(5):569–573.
55. Hirano T, Stamelos S, Harris V, et al. Association of primary hypothyroidism and slipped capital femoral epiphysis. *J Pediatr* 1978;93(2): 262–264.
56. Moorefield WG Jr, Urbaniak JR, Ogden WS, et al. Acquired hypothyroidism and slipped capital femoral epiphysis. Report of three cases. *J Bone Joint Surg Am* 1976;58(5):705–708.
57. Nicolai RD, Grasemann H, Oberste-Berghaus C, et al. Serum insulin-like growth factors IGF-I and IGFBP-3 in children with slipped capital femoral epiphysis. *J Pediatr Orthop B* 1999;8(2):103–106.
58. Wells D, King JD, Roe TF, et al. Review of slipped capital femoral epiphysis associated with endocrine disease. *J Pediatr Orthop* 1993;13(5): 610–614.
59. Mann DC, Weddington J, Richton S. Hormonal studies in patients with slipped capital femoral epiphysis without evidence of endocrinopathy. *J Pediatr Orthop* 1988;8(5):543–545.
60. Razzano CD, Nelson C, Eversman J. Growth hormone levels in slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1972;54(6):1224–1226.
61. Jingushi S, Hara T, Sugioka Y. Deficiency of a parathyroid hormone fragment containing the midportion and 1,25-dihydroxyvitamin D in serum of patients with slipped capital femoral epiphysis. *J Pediatr Orthop* 1997;17(2):216–219.
62. Hayes A, Batshaw ML. Down syndrome. *Pediatr Clin N Am* 1993; 40(3):523–535.
63. Karlsson B, Gustafsson J, Hedov G, et al. Thyroid dysfunction in Down's syndrome: relation to age and thyroid autoimmunity. *Arch Dis Child* 1998;79:242–245.
64. Tuyusz B, Beker DB. Thyroid dysfunction in children with Down's syndrome. *Acta Paediatr* 2001;90:1389–1393.
65. Blethen SL, Rundle AC. Slipped capital femoral epiphysis in children treated with growth hormone. A summary of the National Cooperative Growth Study experience. *Horm Res* 1996;46(3):113–116.
66. Clayton PE, Cowell CT. Safety issues in children and adolescents during growth hormone therapy—a review. *Growth Horm IGF Res* 2000;10(6):306–317.
67. Rappaport EB, Fife D. Slipped capital femoral epiphysis in growth hormone-deficient patients. *Am J Dis Child* 1985;139(4):396–399.
68. Weiner D. Pathogenesis of slipped capital femoral epiphysis: current concepts. *J Pediatr Orthop B* 1996;5(2):67–73.
69. Bright RW, Burstein AH, Elmore SM. Epiphyseal-plate cartilage. A biomechanical and histological analysis of failure modes. *J Bone Joint Surg Am* 1974;56(4):688–703.
70. Chapman JA, Deakin DP, Green JH. Slipped upper femoral epiphysis after radiotherapy. *J Bone Joint Surg Br* 1980;62(3):337–339.
71. Loder RT, Hensinger RN, Alburger PD, et al. Slipped capital femoral epiphysis associated with radiation therapy. *J Pediatr Orthop* 1998;18(5):630–636.
72. Fine RN, Ho M, Tejani A, et al. Adverse events with rhGH treatment of patients with chronic renal insufficiency and end-stage renal disease. *J Pediatr* 2003;142(5):539–545.
73. Loder RT, Hensinger RN. Slipped capital femoral epiphysis associated with renal failure osteodystrophy. *J Pediatr Orthop* 1997;17(2):205–211.
74. Mehls O, Ritz E, Krempien B, et al. Slipped epiphyses in renal osteodystrophy. *Arch Dis Child* 1975;50(7):545–554.
75. Oppenheim WL, Bowen RE, McDonough PW, et al. Outcome of slipped capital femoral epiphysis in renal osteodystrophy. *J Pediatr Orthop* 2003;23(2):169–174.
76. Eisenstein A, Rothschild S. Biochemical abnormalities in patients with slipped capital femoral epiphysis and chondrolysis. *J Bone Joint Surg Am* 1976;58(4):459–467.
77. Morrissy RT, Kalderon AE, Gerdes MH. Synovial immunofluorescence in patients with slipped capital femoral epiphysis. *J Pediatr Orthop* 1981;1(1):55–60.
78. Morrissy RT, Steele RW, Gerdes MH. Localised immune complexes and slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1983;65(5): 574–579.
79. Allen CP, Calvert PT. Simultaneous slipped upper femoral epiphysis in identical twins. *J Bone Joint Surg Br* 1990;72(5):928–929.
80. Burrows HJ. Slipped upper femoral epiphysis; characteristic of a hundred cases. *J Bone Joint Surg Br* 1957;39-B(4):641–658.
81. Gajraj HAR. Slipped capital femoral epiphysis in identical twins. *J Bone Joint Surg Br* 1986;68:653–654.
82. Grant IR. The treatment of slipped upper femoral epiphysis by fibular grafting. *Clin Orthop* 1976;144:270–275.
83. Hagglund G, Hansson LI, Sandstrom S. Familial slipped capital femoral epiphysis. *Acta Orthop Scand* 1986;57(6):510–512.
84. Montsko P, de Jonge T. Slipped capital femoral epiphysis in 6 of 8 first-degree relatives. *Acta Orthop Scand* 1995;66(6):511–512.
85. Moreira JF, Neves MC, Lopes G, et al. Slipped capital femoral epiphysis. A report of 4 cases occurring in one family. *Int Orthop* 1998;22(3):193–196.
86. Rennie AM. The inheritance of slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1982;64(2):180–184.
87. Bednarz PA, Stanitski CL. Slipped capital femoral epiphysis in identical twins: HLA predisposition. *Orthopedics* 1998;21(12):1291–1293.
88. Diwan A, Diamond T, Clarke R, et al. Familial slipped capital femoral epiphysis: a report and considerations in management. *Aust N Z J Surg* 1998;68(9):647–649.
89. Kartenbender K, Cordier W, Katthagen BD. Long-term follow-up study after corrective Imhauser osteotomy for severe slipped capital femoral epiphysis. *J Pediatr Orthop* 2000;20(6):749–756.
90. Maneatis T, Baptista J, Connelly K, et al. Growth hormone safety update from the National Cooperative Growth Study. *J Pediatr Endocrinol Metab* 2000;13(Suppl 2):1035–1044.
91. Gunal I, Ates E. The HLA phenotype in slipped capital femoral epiphysis. *J Pediatr Orthop* 1997;17(5):655–656.
92. Wong-Chung J, Al-Aali Y, Farid I, et al. A common HLA phenotype in slipped capital femoral epiphysis? *Int Orthop* 2000;24(3):158–159.
93. 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(7):1000–1007.

94. Kordelle J, Millis M, Jolesz FA, et al. Three-dimensional analysis of the proximal femur in patients with slipped capital femoral epiphysis based on computed tomography. *J Pediatr Orthop* 2001;21(2):179–182.
95. Galbraith RT, Gelberman RH, Hajek PC, et al. Obesity and decreased femoral anteversion in adolescence. *J Orthop Res* 1987;5(4):523–528.
96. Mirkopulos 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(3):268–273.
97. Kitadai HK, Milani C, Nery CA, et al. Wiberg's center-edge angle in patients with slipped capital femoral epiphysis. *J Pediatr Orthop* 1999;19(1):97–105.
98. Loder RT, Mehbod AA, Meyer C, et al. Acetabular depth and race in young adults: a potential explanation of the differences in the prevalence of slipped capital femoral epiphysis between different racial groups? *J Pediatr Orthop* 2003;23(6):699–702.
99. Kordelle J, Richolt JA, Millis M, et al. Development of the acetabulum in patients with slipped capital femoral epiphysis: a three-dimensional analysis based on computed tomography. *J Pediatr Orthop* 2001;21(2):174–178.
100. Chung SM, Batterman SC, Brighton CT. Shear strength of the human femoral capital epiphyseal plate. *J Bone Joint Surg Am* 1976;58(1):94–103.
101. Fishkin Z, Armstrong DG, Shah H, et al. Proximal femoral physis shear in slipped capital femoral epiphysis–a finite element study. *J Pediatr Orthop* 2006;26(3):291–294.
102. Pritchett JW, Perdue KD. Mechanical factors in slipped capital femoral epiphysis. *J Pediatr Orthop* 1988;8(4):385–388.
103. 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.
104. Casey BH, Hamilton HW, Bobechko WP. Reduction of acutely slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1972;54(4):607–614.
105. Loder RT. Unstable slipped capital femoral epiphysis. *J Pediatr Orthop* 2001;21(5):694–699.
106. Peterson MD, Weiner DS, Green NE, et al. Acute slipped capital femoral epiphysis: the value and safety of urgent manipulative reduction. *J Pediatr Orthop* 1997;17(5):648–654.
107. Herman MJ, Dormans JP, Davidson RS, et al. Screw fixation of Grade III slipped capital femoral epiphysis. *Clin Orthop* 1996;322:77–85.
108. Cowell HR. The significance of early diagnosis and treatment of slipping of the capital femoral epiphysis. *Clin Orthop* 1966;48:89–94.
109. Matava MJ, Patton CM, Luhmann S, et al. Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of initial presentation and treatment. *J Pediatr Orthop* 1999;19(4):455–460.
110. Green WT. Slipping of the upper femoral epiphysis: diagnostic and therapeutic considerations. *Arch Surg* 1945;50:19–33.
111. Ledwith CA, Fleisher GR. Slipped capital femoral epiphysis without hip pain leads to missed diagnosis. *Pediatrics* 1992;89(4 pt 1):660–662.
112. Skaggs DL, Roy AK, Vitale MG, et al. Quality of evaluation and management of children requiring timely orthopaedic surgery before admission to a tertiary pediatric facility. *J Pediatr Orthop* 2002;22(2):265–267.
113. Rattey T, Piehl F, Wright JG. Acute slipped capital femoral epiphysis. Review of outcomes and rates of avascular necrosis. *J Bone Joint Surg Am* 1996;78(3):398–402.
114. Stanitski CL. Acute slipped capital femoral epiphysis: treatment alternatives. *J Am Acad Orthop Surg* 1994;2(2):96–106.
115. Loder RT, Greenfield ML. Clinical characteristics of children with atypical and idiopathic slipped capital femoral epiphysis: description of the age-weight test and implications for further diagnostic investigation. *J Pediatr Orthop* 2001;21(4):481–487.
116. Klein A, Joplin RJ, Reidy JA, et al. Roentgenographic features of slipped capital femoral epiphysis. *AJR Am J Roentgenol* 1951;66(3):361–374.
117. Klein A, Joplin RJ, Reidy JA, et al. Management of the contralateral hip in slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1953;35-A(1):81–87.
118. Steel HH. The metaphyseal blanch sign of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1986;68(6):920–922.
119. Billing L, Eklof O. Slip of the capital femoral epiphysis: revival of a method of assessment. *Pediatr Radiol* 1984;14(6):413–418.
120. Cohen MS, Gelberman RH, Griffin PP, et al. Slipped capital femoral epiphysis: assessment of epiphyseal displacement and angulation. *J Pediatr Orthop* 1986;6(3):259–264.
121. Loder RT. Effect of femur position on the angular measurement of slipped capital femoral epiphysis. *J Pediatr Orthop* 2001;21(4):488–494.
122. Billing L, Bogren HG, Wallin J. Reliable X-ray diagnosis of slipped capital femoral epiphysis by combining the conventional and a new simplified geometrical method. *Pediatr Radiol* 2002;32(6):423–430.
123. Morrissy RT. General considerations. In: Morrissy RT, ed. *Slipped capital femoral epiphysis*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2002:1–18.
124. Southwick WO. Osteotomy through the lesser trochanter for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1967;49(5):807–835.
125. 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(1):85–95.
126. Guzzanti V, Falciglia F. Slipped capital femoral epiphysis: comparison of a roentgenographic method and computed tomography in determining slip severity. *J Pediatr Orthop* 1991;11(1):6–12.
127. Umans H, Liebling MS, Moy L, et al. Slipped capital femoral epiphysis: a physeal lesion diagnosed by MRI, with radiographic and CT correlation. *Skeletal Radiol* 1998;27(3):139–144.
128. Castriota-Scanderbeg A, Orsi E. Slipped capital femoral epiphysis: ultrasonographic findings. *Skeletal Radiol* 1993;22(3):191–193.
129. 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.
130. Kallio PE, Mah ET, Foster BK, et al. Slipped capital femoral epiphysis. Incidence and clinical assessment of physeal instability. *J Bone Joint Surg Br* 1995;77(5):752–755.
131. Kallio PE, Paterson DC, Foster BK, et al. Classification in slipped capital femoral epiphysis. Sonographic assessment of stability and remodeling. *Clin Orthop* 1993;294:196–203.
132. Acosta K, Vade A, Lomasney LM, et al. Radiologic case study. Bilateral slipped capital femoral epiphysis, acute on the left and preslip on the right. *Orthopedics* 2001;24(8):737, 808–809, 811–812.
133. Futami T, Suzuki S, Seto Y, et al. Sequential magnetic resonance imaging in slipped capital femoral epiphysis: assessment of preslip in the contralateral hip. *J Pediatr Orthop B* 2001;10(4):298–303.
134. Lalaji A, Umans H, Schneider R, et al. MRI features of confirmed "preslip" capital femoral epiphysis: a report of two cases. *Skeletal Radiol* 2002;31(6):362–365.
135. Fragniere B, Chotel F, Vargas Barreto B, et al. The value of early postoperative bone scan in slipped capital femoral epiphysis. *J Pediatr Orthop B* 2001;10(1):51–55.
136. Rhoad RC, Davidson RS, Heyman S, et al. Pretreatment bone scan in SCFE: a predictor of ischemia and avascular necrosis. *J Pediatr Orthop* 1999;19(2):164–168.
137. Gordon JE, Abrahams MS, Dobbs MB, et al. Early reduction, arthrotomy, and cannulated screw fixation in unstable slipped capital femoral epiphysis treatment. *J Pediatr Orthop* 2002;22(3):352–358.
138. Lee KE, Pelker RR, Rudicel SA, et al. Histologic patterns of capital femoral growth plate fracture in the rabbit: the effect of shear direction. *J Pediatr Orthop* 1985;5(1):32–39.
139. Rudicel S, Pelker RR, Lee KE, et al. Shear fractures through the capital femoral physis of the skeletally immature rabbit. *J Pediatr Orthop* 1985;5(1):27–31.
140. Segal LS, Weitzel PP, Davidson RS. Valgus slipped capital femoral epiphysis. Fact or fiction? *Clin Orthop* 1996;322:91–98.
141. Shanker VS, Hashemi-Nejad A, Catterall A, et al. Slipped capital femoral epiphysis: is the displacement always posterior? *J Pediatr Orthop B* 2000;9(2):119–121.

142. Aronson J, Tursky EA. The torsional basis for slipped capital femoral epiphysis. *Clin Orthop* 1996;322:37–42.
143. Griffith MJ. Slipping of the capital femoral epiphysis. *Nurs Mirror Midwives J* 1976;143(14):47–49.
144. Nguyen D, Morrissy RT. Slipped capital femoral epiphysis: rationale for the technique of percutaneous in situ fixation. *J Pediatr Orthop* 1990;10(3):341–346.
145. Rab GT. The geometry of slipped capital femoral epiphysis: implications for movement, impingement, and corrective osteotomy. *J Pediatr Orthop* 1999;19(4):419–424.
146. DeAngelis NA, Busconi BD. Hip arthroscopy in the pediatric population. *Clin Orthop* 2003;406:60–63.
147. Futami T, Kasahara Y, Suzuki S, et al. Arthroscopy for slipped capital femoral epiphysis. *J Pediatr Orthop* 1992;12(5):592–597.
148. McCarthy J, Noble P, Aluisio FV, et al. Anatomy, pathologic features, and treatment of acetabular labral tears. *Clin Orthop* 2003;406:38–47.
149. Leunig M, Casillas MM, Hamlet M, et al. Slipped capital femoral epiphysis: early mechanical damage to the acetabular cartilage by a prominent femoral metaphysis. *Acta Orthop Scand* 2000;71(4):370–375.
150. Ganz R, Parvizi J, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Orthop* 2003;417:112–120.
151. Agamanolis DP, Weiner DS. Slipped capital femoral epiphysis: a pathologic investigation into light microscopy, histochemistry and ultrastructure. *Orthop Trans* 1985;9:496–497.
152. Agamanolis DP, Weiner DS, Lloyd JK. Slipped capital femoral epiphysis: a pathological study. II. An ultrastructural study of 23 cases. *J Pediatr Orthop* 1985;5(1):47–58.
153. 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(1):40–46.
154. Guzzanti V, Falciglia F, Stanitski CL, et al. Slipped capital femoral epiphysis: physeal histologic features before and after fixation. *J Pediatr Orthop* 2003;23(5):571–577.
155. Ippolito E, Mickelson MR, Ponseti IV. A histochemical study of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1981;63(7):1109–1113.
156. Lacroix P, Verbrugge J. Slipping of the upper femoral epiphysis. *J Bone Joint Surg Am* 1951;33(2):371–381.
157. Mickelson MR, Ponseti IV, Cooper RR, et al. The ultrastructure of the growth plate in slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1977;59(8):1076–1081.
158. Ponseti IV, McClintock R. The pathology of slipping of the upper femoral epiphysis. *J Bone Joint Surg Am* 1956;38-A(1):71–83.
159. Wattleworth AS, Heiple KG, Chase SW, et al. Pathology of slipped capital femoral epiphysis. *Instr Course Lect* 1972;21:174–181.
160. Chung SM. The arterial supply of the developing proximal end of the human femur. *J Bone Joint Surg Am* 1976;58(7):961–970.
161. Crock HV. A revision of the anatomy of the arteries supplying the upper end of the human femur. *J Anat* 1965;99:77–88.
162. Maeda S, Kita A, Funayama K, et al. Vascular supply to slipped capital femoral epiphysis. *J Pediatr Orthop* 2001;21(5):664–667.
163. Brodetti A. The blood supply of the femoral neck and head in relation to the damaging effects of nails and screws. *J Bone Joint Surg Br* 1960;42(4):794–801.
164. Blanco JS, Taylor B, Johnston CE II. Comparison of single pin versus multiple pin fixation in treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 1992;12(3):384–389.
165. Stasikelis PJ, Sullivan CM, Phillips WA, et al. Slipped capital femoral epiphysis. Prediction of contralateral involvement. *J Bone Joint Surg Am* 1996;78(8):1149–1155.
166. Boero S, Brunenghi GM, Carbone M, et al. Pinning in slipped capital femoral epiphysis: long-term follow-up study. *J Pediatr Orthop B* 2003;12(6):372–379.
167. Carney BT, Weinstein SL. Natural history of untreated chronic slipped capital femoral epiphysis. *Clin Orthop* 1996;322:43–47.
168. Hansson G, Billing L, Hogstedt 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.
169. Ordeberg G, Hansson LI, Sandstrom S. Slipped capital femoral epiphysis in southern Sweden. Long-term result with no treatment or symptomatic primary treatment. *Clin Orthop* 1984;191(191):95–104.
170. Gudmundsson G. Intertrochanteric displacement osteotomy for painful osteoarthritis of the hip. *Acta Orthop Scand* 1970;41(1):91–109.
171. Keener JD, Callaghan JJ, Goetz DD, et al. Twenty-five-year results after Charnley total hip arthroplasty in patients less than fifty years old: a concise follow-up of a previous report. *J Bone Joint Surg Am* 2003; 85-A(6):1066–1072.
172. Lloyd-Roberts GC. Osteoarthritis of the hip; a study of the clinical pathology. *J Bone Joint Surg Br* 1955;37-B(1):8–47.
173. Ranawat CS, Atkinson RE, Salvati EA, et al. Conventional total hip arthroplasty for degenerative joint disease in patients between the ages of forty and sixty years. *J Bone Joint Surg Am* 1984;66(5):745–752.
174. Solomon L. Patterns of osteoarthritis of the hip. *J Bone Joint Surg Br* 1976;58(2):176–183.
175. Goodman DA, Feighan JE, Smith AD, et al. Subclinical slipped capital femoral epiphysis. Relationship to osteoarthrosis of the hip. *J Bone Joint Surg Am* 1997;79(10):1489–1497.
176. Murray RO. The etiology of primary osteoarthritis of the hip. *Br J Radiol* 1965;38:810–824.
177. Stulberg SD, Cordell LD, Harris WH, et al. Unrecognized childhood hip disease: a major cause of idiopathic osteoarthritis of the hip. *The hip: proceedings of the third open scientific meeting of the Hip Society*. St. Louis, MO: C.V. Mosby Company, 1975:212–228.
178. Resnick D. The 'tilt deformity' of the femoral head in osteoarthritis of the hip: a poor indicator of previous epiphysiolysis. *Clin Radiol* 1976;27(3):355–363.
179. Smith JT, Price C, Stevens PM, et al. Does pediatric orthopedic subspecialization affect hospital utilization and charges? *J Pediatr Orthop* 1999;19(4):553–555.
180. Meier MC, Meyer LC, Ferguson RL. Treatment of slipped capital femoral epiphysis with a spica cast. *J Bone Joint Surg Am* 1992;74(10):1522–1529.
181. Carney BT, Weinstein SL, Noble J. Long-term follow-up of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1991;73(5):667–674.
182. Betz RR, Steel HH, Emper WD, et al. Treatment of slipped capital femoral epiphysis. Spica-cast immobilization. *J Bone Joint Surg Am* 1990;72(4):587–600.
183. Lowe HG. Avascular necrosis after slipping of the upper femoral epiphysis. *J Bone Joint Surg Br* 1961;43(4):688–699.
184. Weiner DS. Bone graft epiphysiodesis in the treatment of slipped capital femoral epiphysis. *Instr Course Lect* 1989;38:263–272.
185. Ferguson AB, Howorth MB. Slipping of the upper femoral epiphysis. *JAMA* 1931;97(25):1867–1872.
186. Schmidt TL, Cimino WG, Seidel FG. Allograft epiphysiodesis for slipped capital femoral epiphysis. *Clin Orthop* 1996;322:61–76.
187. Adamczyk MJ, Weiner DS, Hawk D. A 50-year experience with bone graft epiphysiodesis in the treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 2003;23(5):578–583.
188. Howorth B. The bone-pegging operation for slipping of the capital femoral epiphysis. *Clin Orthop* 1966;48:79–87.
189. Melby A, Hoyt WA Jr, Weiner DS. Treatment of chronic slipped capital femoral epiphysis by bone-grafted epiphyseodesis. *J Bone Joint Surg Am* 1980;62(1):119–125.
190. Weiner DS. Epiphysiodesis in slipped capital femoral epiphysis. *J Pediatr Orthop* 1986;6(6):754–755.
191. Weiner DS. Open bone graft epiphysiodesis for slipped capital femoral epiphysis. *J Pediatr Orthop* 1990;10(5):673–674.
192. Weiner DS. Use of open bone-graft epiphysiodesis in the treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 1998;18(1): 136–137.
193. Weiner DS, Weiner S, Melby A, et al. A 30-year experience with bone graft epiphysiodesis in the treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 1984;4(2):145–152.
194. Weiner DS, Weiner SD, Melby A. Anterolateral approach to the hip for bone graft epiphysiodesis in the treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 1988;8(3):349–352.

195. Irani RN, Rosenzweig AH, Cotler HB, et al. Epiphysiodesis in slipped capital femoral epiphysis: a comparison of various surgical modalities. *J Pediatr Orthop* 1985;5(6):661–664.
196. Rao SB, Crawford AH, Burger RR, et al. Open bone peg epiphysiodesis for slipped capital femoral epiphysis. *J Pediatr Orthop* 1996;16(1): 37–48.
197. Rostoucher P, Bensahel H, Pennecot GF, et al. Slipped capital femoral epiphysis: evaluation of different modes of treatment. *J Pediatr Orthop B* 1996;5(2):96–101.
198. Herndon CH, Heyman CH, Bell DM. Treatment of slipped capital femoral epiphysis by epiphyseodesis and osteoplasty of the femoral neck. A report of further experiences. *J Bone Joint Surg Am* 1963;45:999–1012.
199. Ward WT, Wood K. Open bone graft epiphyseodesis for slipped capital femoral epiphysis. *J Pediatr Orthop* 1990;10(1):14–20.
200. Morrissy RT. Slipped capital femoral epiphysis technique of percutaneous in situ fixation. *J Pediatr Orthop* 1990;10(3):347–350.
201. Lee FY, Chapman CB. In situ pinning of hip for stable slipped capital femoral epiphysis on a radiolucent operating table. *J Pediatr Orthop* 2003;23(1):27–29.
202. Aronson DD, Peterson DA, Miller DV. Slipped capital femoral epiphysis. The case for internal fixation in situ. *Clin Orthop* 1992;281:115–122.
203. Goodwin RC, Mahar AT, Oswald TS, et al. Screw head impingement after in situ fixation in moderate and severe slipped capital femoral epiphysis. *J Pediatr Orthop* 2007;27(3):319–325.
204. Gonzalez-Moran G, Carsi B, Abril JC, et al. Results after preoperative traction and pinning in slipped capital femoral epiphysis: K wires versus cannulated screws. *J Pediatr Orthop B* 1998;7(1):53–58.
205. Morrissy RT. In situ fixation of chronic slipped capital femoral epiphysis. *Instr Course Lect* 1984;33:319–327.
206. Morrissy RT. Principles of in situ fixation in chronic slipped capital femoral epiphysis. *Instr Course Lect* 1989;38:257–262.
207. Walters R, Simon SR. Joint destruction: a sequel of unrecognized pin penetration in patients with slipped capital femoral epiphysis. *The hip: proceedings of the eighth open scientific meeting of the Hip Society.* Vol. 145–164. St. Louis, MO: The C.V. Mosby Company, 1980.
208. Goodman WW, Johnson JT, Robertson WW Jr. Single screw fixation for acute and acute-on-chronic slipped capital femoral epiphysis. *Clin Orthop* 1996;322:86–90.
209. Stevens DB, Short BA, Burch JM. In situ fixation of the slipped capital femoral epiphysis with a single screw. *J Pediatr Orthop B* 1996;5(2):85–89.
210. Orr TR, Bollinger BA, Strecker WB. Blind zone determination of the femoral head. *J Pediatr Orthop* 1989;9(4):417–421.
211. Moseley C. The "approach-withdraw phenomenon" in the pinning of slipped capital femoral epiphysis. *Orthop Trans* 1985;9:497.
212. Stambough JL, Davidson RS, Ellis RD, et al. Slipped capital femoral epiphysis: an analysis of 80 patients as to pin placement and number. *J Pediatr Orthop* 1986;6(3):265–273.
213. Bassett GS. Bone endoscopy: direct visual confirmation of cannulated screw placement in slipped capital femoral epiphysis. *J Pediatr Orthop* 1993;13(2):159–163.
214. Lehman WB, Grant A, Rose D, et al. A method of evaluating possible pin penetration in slipped capital femoral epiphysis using a cannulated internal fixation device. *Clin Orthop* 1984;186:65–70.
215. Tokmakova KP, Stanton RP, Mason DE. Factors influencing the development of osteonecrosis in patients treated for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 2003;85-A(5):798–801.
216. Aronson DD, Loder RT. Slipped capital femoral epiphysis in black children. *J Pediatr Orthop* 1992;12(1):74–79.
217. Crawford AH. Slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1988;70(9):1422–1427.
218. de Sanctis N, Di Gennaro G, Pempinello C, et al. Is gentle manipulative reduction and percutaneous fixation with a single screw the best management of acute and acute-on-chronic slipped capital femoral epiphysis? A report of 70 patients. *J Pediatr Orthop B* 1996;5(2):90–95.
219. Lehman WB, Menche D, Grant A, et al. The problem of evaluating in situ pinning of slipped capital femoral epiphysis: an experimental model and a review of 63 consecutive cases. *J Pediatr Orthop* 1984;4(3):297–303.
220. Riley PM, Weiner DS, Gillespie R, et al. Hazards of internal fixation in the treatment of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1990;72(10):1500–1509.
221. Karol LA, Doane RM, Cornicelli SF, et al. Single versus double screw fixation for treatment of slipped capital femoral epiphysis: a biomechanical analysis. *J Pediatr Orthop* 1992;12(6):741–745.
222. Kruger DM, Herzenberg JE, Viviano DM, et al. Biomechanical comparison of single- and double-pin fixation for acute slipped capital femoral epiphysis. *Clin Orthop* 1990;259:277–281.
223. Kibiloski LJ, Doane RM, Karol LA, et al. Biomechanical analysis of single- versus double-screw fixation in slipped capital femoral epiphysis at physiological load levels. *J Pediatr Orthop* 1994;14(5): 627–630.
224. Snyder RR, Williams JL, Schmidt TL, et al. Torsional strength of double- versus single-screw fixation in a pig model of unstable slipped capital femoral epiphysis. *J Pediatr Orthop* 2006;26(3):295–299.
225. Early SD, Hedman TP, Reynolds RA. Biomechanical analysis of compression screw fixation versus standard in situ pinning in slipped capital femoral epiphysis. *J Pediatr Orthop* 2001;21(2):183–188.
226. Miyanji F, Mahar A, Oka R, et al. Biomechanical comparison of fully and partially threaded screws for fixation of slipped capital femoral epiphysis. *J Pediatr Orthop* 2008;28(1):49–52.
227. Stanton RP, Shelton YA. Closure of the physis after pinning of slipped capital femoral epiphysis. *Orthopedics* 1993;16(10):1099–1102; discussion 1102–1093.
228. Ward WT, Stefko J, Wood KB, et al. Fixation with a single screw for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1992;74(6):799–809.
229. Wong-Chung J, Strong ML. Physeal remodeling after internal fixation of slipped capital femoral epiphyses. *J Pediatr Orthop* 1991; 11(1):2–5.
230. Hansson LI. Osteosynthesis with the hook-pin in slipped capital femoral epiphysis. *Acta Orthop Scand* 1982;53(1):87–96.
231. Kumm DA, Lee SH, Hackenbroch MH, et al. Slipped capital femoral epiphysis: a prospective study of dynamic screw fixation. *Clin Orthop* 2001;384:198–207.
232. Macafee AL. Long handled punch for the insertion of Newman's pins in slipped upper femoral epiphysis and intracapsular fractures of the femur. *Injury* 1981;13(1):81.
233. 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 B* 1996;5(3):151–157.
234. O'Brien ET, Fahey JJ. Remodeling of the femoral neck after in situ pinning for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1977;59(1):62–68.
235. Jones JR, Paterson DC, Hillier TM, et al. Remodelling after pinning for slipped capital femoral epiphysis. *J Bone Joint Surg Br* 1990;72(4): 568–573.
236. DeLullo JA, Thomas E, Cooney TE, et al. Femoral remodeling may influence patient outcomes in slipped capital femoral epiphysis. *Clin Orthop Relat Res* 2007;457:163–170.
237. Ballard J, Cosgrove AP. Anterior physeal separation. A sign indicating a high risk for avascular necrosis after slipped capital femoral epiphysis. *J Bone Joint Surg Br* 2002;84(8):1176–1179.
238. Jerre R, Billing L, Karlsson J. Loss of hip motion in slipped capital femoral epiphysis: a calculation from the slipping angle and the slope. *J Pediatr Orthop B* 1996;5(3):144–150.
239. Kennedy JG, Hresko MT, Kasser JR, et al. Osteonecrosis of the femoral head associated with slipped capital femoral epiphysis. *J Pediatr Orthop* 2001;21(2):189–193.
240. Dietz FR. Traction reduction of acute and acute-on-chronic slipped capital femoral epiphysis. *Clin Orthop* 1994;302:101–110.
241. Aprin H, Goodman S, Kahn LB. Cartilage necrosis due to pin penetration: experimental studies in rabbits. *J Pediatr Orthop* 1991;11(5): 623–630.
242. Sternlicht AL, Ehrlich MG, Armstrong AL, et al. Role of pin protrusion in the etiology of chondrolysis: a surgical model with radiographic, histologic, and biochemical analysis. *J Pediatr Orthop* 1992;12(4):428–433.

243. Ingram AJ, Clarke MS, Clarke CS Jr, et al. Chondrolysis complicating slipped capital femoral epiphysis. *Clin Orthop* 1982;165: 99–109.
244. Koval KJ, Lehman WB, Rose D, et al. Treatment of slipped capital femoral epiphysis with a cannulated-screw technique. *J Bone Joint Surg Am* 1989;71(9):1370–1377.
245. Vrettos BC, Hoffman EB. Chondrolysis in slipped upper femoral epiphysis. Long-term study of the aetiology and natural history. *J Bone Joint Surg Br* 1993;75(6):956–961.
246. Zionts LE, Simonian PT, Harvey JP Jr. Transient penetration of the hip joint during in situ cannulated-screw fixation of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1991;73(7):1054–1060.
247. Bellemans J, Fabry G, Molenaers G, et al. Pin removal after in-situ pinning for slipped capital femoral epiphysis. *Acta Orthop Belg* 1994;60(2):170–172.
248. Denton JR. Fixation with a single screw for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1993;75(3):469.
249. Sanders JO, Smith WJ, Stanley EA, et al. Progressive slippage after pinning for slipped capital femoral epiphysis. *J Pediatr Orthop* 2002;22(2):239–243.
250. Carney BT, Birnbaum P, Minter C. Slip progression after in situ single screw fixation for stable slipped capital femoral epiphysis. *J Pediatr Orthop* 2003;23(5):584–589.
251. Laplaza FJ, Burke SW. Results after preoperative traction and pinning in slipped capital femoral epiphysis: K wires versus cannulated screws. *J Pediatr Orthop B* 1999;8(1):72–73.
252. Jerre R, Karlsson J, Romanus B, et al. Does a single device prevent further slipping of the epiphysis in children with slipped capital femoral epiphysis? *Arch Orthop Trauma Surg* 1997;116(6–7):348–351.
253. Plotz GM, Prymka M, Hassenpflug J. The role of prophylactic pinning in the treatment of slipped capital femoral epiphysis—a case report. *Acta Orthop Scand* 1999;70(6):631–634.
254. Nonweiler B, Hoffer M, Weinert C, et al. Percutaneous in situ fixation of slipped capital femoral epiphysis using two threaded Steinmann pins. *J Pediatr Orthop* 1996;16(1):56–60.
255. Gregory P, Pevny T, Teague D. Early complications with external fixation of pediatric femoral shaft fractures. *J Orthop Trauma* 1996;10(3):191–198.
256. Fish JB. Cuneiform osteotomy of the femoral neck in the treatment of slipped capital femoral epiphysis. A follow-up note. *J Bone Joint Surg Am* 1994;76(1):46–59.
257. Canale ST, Azar F, Young J, et al. Subtrochanteric fracture after fixation of slipped capital femoral epiphysis: a complication of unused drill holes. *J Pediatr Orthop* 1994;14(5):623–626.
258. Schmidt R, Gregg JR. Subtrochanteric fractures complicating pin fixation of slipped capital femoral epiphysis. *Orthop Trans* 1985;9:497.
259. Baynham GC, Lucie RS, Cummings RJ. Femoral neck fracture secondary to in situ pinning of slipped capital femoral epiphysis: a previously unreported complication. *J Pediatr Orthop* 1991;11(2):187–190.
260. Canale ST, Casillas M, Banta JV. Displaced femoral neck fractures at the bone-screw interface after in situ fixation of slipped capital femoral epiphysis. *J Pediatr Orthop* 1997;17(2):212–215.
261. Chen CE, Ko JY, Wang CJ. Premature closure of the physeal plate after treatment of a slipped capital femoral epiphysis. *Chang Gung Med J* 2002;25(12):811–818.
262. Khan FA. Treatment of slipped capital femoral epiphysis with severe displacement (report of 14 hips in 12 non Caucasian patients). *Afr J Med Sci* 1995;24(2):189–194.
263. Kendig RJ, Field L, Fisher LC III. Slipped capital femoral epiphysis, a problem of diagnosis. *J Miss State Med Assoc* 1993;34(5):147–151.
264. 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(3):294–302.
265. Greenough CG, Bromage JD, Jackson AM. Pinning of the slipped upper femoral epiphysis—a trouble-free procedure? *J Pediatr Orthop* 1985;5(6):657–660.
266. Kahle WK. The case against routine metal removal. *J Pediatr Orthop* 1994;14(2):229–237.
267. Crandall DG, Gabriel KR, Akbarnia BA. Second operation for slipped capital femoral epiphysis: pin removal. *J Pediatr Orthop* 1992;12(4):434–437.
268. Lee TK, Haynes RJ, Longo JA, et al. Pin removal in slipped capital femoral epiphysis: the unsuitability of titanium devices. *J Pediatr Orthop* 1996;16(1):49–52.
269. Hagglund G. The contralateral hip in slipped capital femoral epiphysis. *J Pediatr Orthop B* 1996;5(3):158–161.
270. Ross PM, Lyne ED, Morawa LG. Slipped capital femoral epiphysis long-term results after 10–38 years. *Clin Orthop* 1979;141(141): 176–180.
271. Aronsson DD, Karol LA. Stable slipped capital femoral epiphysis: evaluation and management. *J Am Acad Orthop Surg* 1996;4(4):173–181.
272. Givon U, Bowen JR. Chronic slipped capital femoral epiphysis: treatment by pinning in situ. *J Pediatr Orthop B* 1999;8(3):216–222.
273. Jerre R, Hansson G, Wallin J, et al. Long-term results after realignment operations for slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1996;78(5):745–750.
274. Aadalen RJ, Weiner DS, Hoyt W, et al. Acute slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1974;56(7):1473–1487.
275. Hall JE. The results of treatment of slipped femoral epiphysis. *J Bone Joint Surg Br* 1957;39-B(4):659–673.
276. Phillips SA, Griffiths WE, Clarke NM. The timing of reduction and stabilisation of the acute, unstable, slipped upper femoral epiphysis. *J Bone Joint Surg Br* 2001;83(7):1046–1049.
277. 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):1–8.
278. Aronsson DD, Loder RT. Treatment of the unstable (acute) slipped capital femoral epiphysis. *Clin Orthop* 1996;322:99–110.
279. Schmitz M. Treatment. In: Morrissy RT, ed. *Slipped capital femoral epiphysis*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2002:19–38.
280. Cheng JCY, Tang N. Decompression and stable internal fixation of femoral neck fractures in children can affect outcome. *J Pediatr Orthop* 1999;19(3):338–343.
281. Holmberg S, Dalen N. Intracapsular pressure and caput circulation in nondisplaced femoral neck fracture. *Clin Orthop* 1987;219:124–126.
282. Ng GPK, Cole WG. Effect of early hip decompression on the frequency of avascular necrosis in children with fractures of the neck of the femur. *Injury* 1996;27(6):419–421.
283. Soto-Hall R, Johnson LH, Johnson RA. Variations in the intra-articular pressure of the hip joint in injury and disease. *J Bone Joint Surg Am* 1964;46(3):509–516.
284. Stromqvist B, Nilsson LT, Egund N, et al. Intracapsular pressures in undisplaced fractures of the femoral neck. *J Bone Joint Surg Br* 1988;70:192–194.
285. Woodhouse C. Dynamic influences of vascular occlusion affecting development of avascular necrosis of the femoral head. *Clin Orthop* 1964;32:119–129.
286. Swiontkowski MF, Tepic S, Perren SM, et al. Laser Doppler flowmetry for bone blood flow measurement: correlation with microsphere estimates and evaluation of the effect of intracapsular pressure on femoral head blood flow. *J Orthop Res* 1986;4:362–371.
287. Vegter J, Klopper PJ. Effect of intracapsular hyperpressure on femoral head blood flow. *Acta Orthop Scand* 1991;62(4):337–341.
288. Herrera-Soto JA, Duffy MF, Birnbaum MA, et al. Increased intracapsular pressures after unstable slipped capital femoral epiphysis. *J Pediatr Orthop* 2008;28(7):723–728.
289. Frymoyer JW. Chondrolysis of the hip following Southwick osteotomy for severe slipped capital femoral epiphysis. *Clin Orthop* 1974;99: 120–124.
290. Zupanc O, Antolic V, Iglic A, et al. The assessment of contact stress in the hip joint after operative treatment for severe slipped capital femoral epiphysis. *Int Orthop* 2001;25(1):9–12.
291. Richolt JA, Teschner M, Everett PC, et al. Impingement simulation of the hip in SCFE using 3D models. *Comput Aided Surg* 1999;4(3): 144–151.

292. 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(3):135–143.
293. Martin PH. Slipped epiphysis in the adolescent hip: a reconsideration of open reduction. *J Bone Joint Surg Am* 1948;30(1):9–19.
294. Crawford AH. The role of osteotomy in the treatment of slipped capital femoral epiphysis. *Instr Course Lect* 1989;38:273–279.
295. Fish JB. Cuneiform osteotomy of the femoral neck in the treatment of slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1984;66(8):1153–1168.
296. Nishiyama K, Sakamaki T, Ishii Y. Follow-up study of the subcapital wedge osteotomy for severe chronic slipped capital femoral epiphysis. *J Pediatr Orthop* 1989;9(4):412–416.
297. Dunn D. Severe slipped capital femoral epiphysis and open repolacement by cervical osteotomy. *The hip: proceedings of the third open scientific meeting of the Hip Society*. St. Louis, MO: C. V. Mosby Company, 1975:115–126.
298. 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.
299. Barros JW, Tukiama G, Fontoura C, et al. Trapezoid osteotomy for slipped capital femoral epiphysis. *Int Orthop* 2000;24(2):83–87.
300. DeRosa GP, Mullins RC, Kling TF Jr. Cuneiform osteotomy of the femoral neck in severe slipped capital femoral epiphysis. *Clin Orthop* 1996;322:48–60.
301. Fron D, Forgues D, Mayrargue E, et al. Follow-up study of severe slipped capital femoral epiphysis treated with Dunn's osteotomy. *J Pediatr Orthop* 2000;20(3):320–325.
302. Velasco R, Schai PA, Exner GU. Slipped capital femoral epiphysis: a long-term follow-up study after open reduction of the femoral head combined with subcapital wedge resection. *J Pediatr Orthop B* 1998;7(1):43–52.
303. Leunig M, Slongo T, Ganz R. Subcapital realignment in slipped capital femoral epiphysis: surgical hip dislocation and trimming of the stable trochanter to protect the perfusion of the epiphysis. *Instr Course Lect* 2008;57:499–507.
304. Leunig M, Slongo T, Kleinschmidt M, et al. Subcapital correction osteotomy in slipped capital femoral epiphysis by means of surgical hip dislocation. *Oper Orthop Traumatol* 2007;19(4):389–410.
305. Ziebarth K, Zilkens C, Spencer S, et al. Capital realignment for moderate and severe SCFE using a modified Dunn procedure. *Clin Orthop Relat Res* 2009;467(3):704–716.
306. Wiberg G. Surgical treatment of slipped epiphysis with special reference to wedge osteotomy of the femoral neck. *Clin Orthop* 1966;48:139–152.
307. Gage JR, Sundberg AB, Nolan DR, et al. Complications after cuneiform osteotomy for moderately or severely slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1978;60(2):157–165.
308. Barmada R, Bruch RF, Gimbel JS, et al. Base of the neck extracapsular osteotomy for correction of deformity in slipped capital femoral epiphysis. *Clin Orthop* 1978;132:98–101.
309. Kramer WG, Craig WA, Noel S. Compensating osteotomy at the base of the femoral neck for slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1976;58(6):796–800.
310. Clark CR, Southwick WO, Ogden JA. Anatomic aspects of slipped capital femoral epiphysis and correction by biplane osteotomy. *Instr Course Lect* 1980;29:90–100.
311. Southwick WO. Compression fixation after biplane intertrochanteric osteotomy for slipped capital femoral epiphysis. A technical improvement. *J Bone Joint Surg Am* 1973;55(6):1218–1224.
312. Southwick WO. Biplane osteotomy for very severe slipped capital femoral epiphysis. *The hip: proceedings of the third open scientific meeting of the Hip Society*. St. Louis, MO: The C. V. Mosby Company, 1975:105–114.
313. Southwick WO. Slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1984;66(8):1151–1152.
314. Imhauser G. Pathogenese und therapie der jugendlichen huftenkopfosung. *Z orthop Ihre Grenzgeh* 1957;88:3–41.
315. Colyer RA. Compression external fixation after biplane femoral trochanteric osteotomy for severe slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1980;62(4):557–560.
316. Ito H, Minami A, Suzuki K, et al. Three-dimensionally corrective external fixator system for proximal femoral osteotomy. *J Pediatr Orthop* 2001;21(5):652–656.
317. Scher MA, Sweet MB, Jakim I. Acute-on-chronic bilateral reversed slipped capital femoral epiphysis managed by Imhauser-Weber osteotomy. *Arch Orthop Trauma Surg* 1989;108(5):336–338.
318. Diab M, Daluvoy S, Snyder BD, et al. Osteotomy does not improve early outcome after slipped capital femoral epiphysis. *J Pediatr Orthop B* 2006;15(2):87–92.
319. Spencer S, Millis MB, Kim YJ. Early results of treatment of hip impingement syndrome in slipped capital femoral epiphysis and pistol grip deformity of the femoral head-neck junction using the surgical dislocation technique. *J Pediatr Orthop* 2006;26(3):281–285.
320. Millis MB, Murphy SB, Poss R. Osteotomies about the hip for the prevention and treatment of osteoarthrosis. *Instr Course Lect* 1996;45:209–226.
321. Millis MB, Poss R, Murphy SB. Osteotomies of the hip in the prevention and treatment of osteoarthritis. *Instr Course Lect* 1992;41:145–154.
322. Salvati EA, Robinson JH Jr, O'Down TJ. Southwick osteotomy for severe chronic slipped capital femoral epiphysis: results and complications. *J Bone Joint Surg Am* 1980;62(4):561–570.
323. Mamisch TC, Kim YJ, Richolt JA, et al. Femoral morphology due to impingement influences the range of motion in slipped capital femoral epiphysis. *Clin Orthop Relat Res* 2009;467(3):692–698.
324. Ireland J, Newman PH. Triplane osteotomy for severely slipped upper femoral epiphysis. *J Bone Joint Surg Br* 1978;60-B(3):390–393.
325. Rao JP, Francis AM, Siwek CW. The treatment of chronic slipped capital femoral epiphysis by biplane osteotomy. *J Bone Joint Surg Am* 1984;66(8):1169–1175.
326. Merchan EC, Na CM, Munuera L. Intertrochanteric osteotomy for the treatment of chronic slipped capital femoral epiphysis. *Int Orthop* 1992;16(2):133–135.
327. Parsch K, Zehender H, Buhl T, et al. Intertrochanteric corrective osteotomy for moderate and severe chronic slipped capital femoral epiphysis. *J Pediatr Orthop B* 1999;8(3):223–230.
328. Sugioka Y. Transtrochanteric anterior rotational osteotomy of the femoral head in the treatment of osteonecrosis affecting the hip: a new osteotomy operation. *Clin Orthop* 1978;130:191–201.
329. Sugioka Y. Transtrochanteric rotational osteotomy in the treatment of idiopathic and steroid-induced femoral head necrosis, Perthes' disease, slipped capital femoral epiphysis, and osteoarthritis of the hip. Indications and results. *Clin Orthop* 1984;184:12–23.
330. Masuda T, Matsuno T, Hasegawa I, et al. Transtrochanteric anterior rotational osteotomy for slipped capital femoral epiphysis: a report of five cases. *J Pediatr Orthop* 1986;6(1):18–23.
331. Castro FP Jr, Bennett JT, Doulens K. Epidemiological perspective on prophylactic pinning in patients with unilateral slipped capital femoral epiphysis. *J Pediatr Orthop* 2000;20(6):745–748.
332. Kumm DA, Schmidt J, Eisenburger SH, et al. Prophylactic dynamic screw fixation of the asymptomatic hip in slipped capital femoral epiphysis. *J Pediatr Orthop* 1996;16(2):249–253.
333. Yildirim Y, Bautista S, Davidson RS. Chondrolysis, osteonecrosis, and slip severity in patients with subsequent contralateral slipped capital femoral epiphysis. *J Bone Joint Surg Am* 2008;90(3):485–492.
334. Schultz WR, Weinstein JN, Weinstein SL, et al. Prophylactic pinning of the contralateral hip in slipped capital femoral epiphysis: evaluation of long-term outcome for the contralateral hip with use of decision analysis. *J Bone Joint Surg Am* 2002;84-A(8):1305–1314.
335. Bertani A, Launay F, Pauly V, et al. Complications of prophylactic pinning for unilateral upper femur epiphysis slipping: retrospective analysis of 62 operated cases. *Rev Chir Orthop Reparatrice Appar Mot* 2008;94(4):392–398.
336. Emery RJ, Todd RC, Dunn DM. Prophylactic pinning in slipped upper femoral epiphysis. Prevention of complications. *J Bone Joint Surg Br* 1990;72(2):217–219.

337. Laplaza FJ, Burke SW. Epiphyseal growth after pinning of slipped capital femoral epiphysis. *J Pediatr Orthop* 1995;15(3):357–361.
338. Hurley JM, Betz RR, Loder RT, et al. Slipped capital femoral epiphysis. The prevalence of late contralateral slip. *J Bone Joint Surg Am* 1996;78(2):226–230.
339. Sorensen KH. Slipped upper femoral epiphysis, clinical examinations concerning the aetiology. *Acta Orthop Scand* 1969;40(5):686.
340. Denton JR. Progression of a slipped capital femoral epiphysis after fixation with a single cannulated screw. A case report. *J Bone Joint Surg Am* 1993;75(3):425–427.
341. Segal LS, Davidson RS, Robertson WW Jr, et al. Growth disturbances of the proximal femur after pinning of juvenile slipped capital femoral epiphysis. *J Pediatr Orthop* 1991;11(5):631–637.
342. Whiteside LA, Schoenecker PL. Combined valgus derotation osteotomy and cervical osteoplasty for severely slipped capital femoral epiphysis: mechanical analysis and report preliminary results using compression screw fixation and early weight bearing. *Clin Orthop* 1978;132:88–97.
343. Carlioz H, Vogt JC, Barba L, et al. Treatment of slipped upper femoral epiphysis: 80 cases operated on over 10 years (1968–1978). *J Pediatr Orthop* 1984;4(2):153–161.
344. Howorth B. Slipping of the capital femoral epiphysis. History. *Clin Orthop* 1966;48:11–32.
345. Wells L. Common lower extremity problems in children. *Prim Care* 1996;23(2):299–303.
346. Krahn TH, Canale ST, Beaty JH, et al. Long-term follow-up of patients with avascular necrosis after treatment of slipped capital femoral epiphysis. *J Pediatr Orthop* 1993;13(2):154–158.
347. Notzli HP, Chou LB, Ganz R. Open-reduction and intertrochanteric osteotomy for osteonecrosis and extrusion of the femoral head in adolescents. *J Pediatr Orthop* 1995;15(1):16–20.
348. Scher MA, Jakim I. Intertrochanteric osteotomy and autogenous bone-grafting for avascular necrosis of the femoral head. *J Bone Joint Surg Am* 1993;75(8):1119–1133.
349. Sugano N, Takaoka K, Ohzono K, et al. Rotational osteotomy for nontraumatic avascular necrosis of the femoral head. *J Bone Joint Surg Br* 1992;74(5):734–739.
350. Sugioka Y, Hotokebuchi T, Tsutsui H. Transtrochanteric anterior rotational osteotomy for idiopathic and steroid-induced necrosis of the femoral head. Indications and long-term results. *Clin Orthop* 1992;277:111–120.
351. Trumble SJ, Mayo KA, Mast JW. The periacetabular osteotomy. Minimum 2 year followup in more than 100 hips. *Clin Orthop* 1999;363:54–63.
352. Dean GS, Kime RC, Fitch RD, et al. Treatment of osteonecrosis in the hip of pediatric patients by free vascularized fibula graft. *Clin Orthop* 2001;386:106–113.
353. Ko JY, Meyers MH, Wenger DR. "Trapdoor" procedure for osteonecrosis with segmental collapse of the femoral head in teenagers. *J Pediatr Orthop* 1995;15(1):7–15.
354. Waldenstrom H. On necrosis of the joint cartilage by epiphyseolysis capitis femoris. *Acta Chir Scand* 1930;67:936–946.
355. Hughes LO, Aronson J, Smith HS. Normal radiographic values for cartilage thickness and physeal angle in the pediatric hip. *J Pediatr Orthop* 1999;19(4):443–448.
356. Cruess RL. The pathology of acute necrosis of cartilage in slipping of the capital femoral epiphysis. A report of two cases with pathological sections. *J Bone Joint Surg Am* 1963;45:1013–1024.
357. Maurer RC, Larsen IJ. Acute necrosis of cartilage in slipped capital femoral epiphysis. *J Bone Joint Surg Am* 1970;52(1):39–50.
358. Tudisco C, Caterini R, Farsetti P, et al. Chondrolysis of the hip complicating slipped capital femoral epiphysis: long-term follow-up of nine patients. *J Pediatr Orthop B* 1999;8(2):107–111.
359. Mankin HJ, Sledge CB, Rothschild S, et al. Chondrolysis of the hip. *The hip: proceedings of the third open scientific meeting of the Hip Society*. St. Louis, MO: The C. V. Mosby Company, 1975:127–135.
360. Kennedy JP, Weiner DS. Results of slipped capital femoral epiphysis in the black population. *J Pediatr Orthop* 1990;10(2):224–227.
361. Spero CR, Masciale JP, Tornetta P III, et al. Slipped capital femoral epiphysis in black children: incidence of chondrolysis. *J Pediatr Orthop* 1992;12(4):444–448.
362. El-Khoury GY, Mickelson MR. Chondrolysis following slipped capital femoral epiphysis. *Radiology* 1977;123(2):327–330.
363. Mandell GA, Keret D, Harcke HT, et al. Chondrolysis: detection by bone scintigraphy. *J Pediatr Orthop* 1992;12(1):80–85.
364. Lowe HG. Necrosis of articular cartilage after slipping of the capital femoral epiphysis. Report of six cases with recovery. *J Bone Joint Surg Br* 1970;52(1):108–118.
365. Kitakoji T, Hattori T, Ida K, et al. Arthrodiatasis for chondrolysis with hinge abduction: a case report. *J Pediatr Orthop B* 2000;9(3):198–200.
366. Brinker MR, Rosenberg AG, Kull L, et al. Primary total hip arthroplasty using noncemented porous-coated femoral components in patients with osteonecrosis of the femoral head. *J Arthroplasty* 1994;9(5):457–468.
367. Chandler HP, Reineck FT, Wixson RL, et al. Total hip replacement in patients younger than thirty years old. A five-year follow-up study. *J Bone Joint Surg Am* 1981;63(9):1426–1434.
368. Duffy GP, Berry DJ, Rowland C, et al. Primary uncemented total hip arthroplasty in patients <40 years old: 10- to 14-year results using first-generation proximally porous-coated implants. *J Arthroplasty* 2001;16(8 Suppl 1):140–144.
369. McAuley JP, Szuszczewicz ES, Young A, et al. Total hip arthroplasty in patients 50 years and younger. *Clin Orthop* 2004(418):119–125.
370. Crowther JD, Lachiewicz PF. Survival and polyethylene wear of porous-coated acetabular components in patients less than fifty years old: results at nine to fourteen years. *J Bone Joint Surg Am* 2002;84-A(5):729–735.
371. Perez RE, Rodriguez JA, Deshmukh RG, et al. Polyethylene wear and periprosthetic osteolysis in metal-backed acetabular components with cylindrical liners. *J Arthroplasty* 1998;13(1):1–7.
372. Benaroch TE, Richards BS, Haideri N, et al. Intermediate follow-up of a simple method of hip arthrodesis in adolescent patients. *J Pediatr Orthop* 1996;16(1):30–36.
373. Callaghan JJ, Brand RA, Pedersen DR. Hip arthrodesis. A long-term follow-up. *J Bone Joint Surg Am* 1985;67(9):1328–1335.
374. Mowery CA, Houkom JA, Roach JW, et al. A simple method of hip arthrodesis. *J Pediatr Orthop* 1986;6(1):7–10.
375. Scher DM, Jeong GK, Grant AD, et al. Hip arthrodesis in adolescents using external fixation. *J Pediatr Orthop* 2001;21(2):194–197.
376. Schoenecker PL, Johnson LO, Martin RA, et al. Intra-articular hip arthrodesis without subtrochanteric osteotomy in adolescents: technique and short-term follow-up. *Am J Orthop* 1997;26(4):257–264.
377. Sponseller PD, McBeath AA, Perpich M. Hip arthrodesis in young patients. A long-term follow-up study. *J Bone Joint Surg Am* 1984;66(6):853–859.
378. Farkas A. New operative treatment of tuberculous coxitis in children. *J Bone Joint Surg Am* 1939;21:323–333.
379. Price CT, Lovell WW. Thompson arthrodesis of the hip in children. *J Bone Joint Surg Am* 1980;62:1118–1123.
380. Thompson FR. Combined hip fusion and subtrochanteric osteotomy allowing early ambulation. *J Bone Joint Surg Am* 1956;38(1):13–21.
381. Amstutz HC, Sakai DN. Total joint replacement for ankylosed hips. Indications, technique, and preliminary results. *J Bone Joint Surg Am* 1975;57(5):619–625.
382. Kilgus DJ, Amstutz HC, Wolgin MA, et al. Joint replacement for ankylosed hips. *J Bone Joint Surg Am* 1990;72(1):45–54.
383. Lubahn JD, Evarts CM, Feltner JB. Conversion of ankylosed hips to total hip arthroplasty. *Clin Orthop* 1980;153:146–152.