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Avascular necrosis of the proximal femur in developmental dislocation of the hip

**Incidence, risk factors, sequelae and MR imaging for
diagnosis and prognosis**

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Abstract

Avascular necrosis of the proximal femur still remains the major complication of the treatment for developmental dislocation of the hip. In a three part study I reviewed this problem.

Part I analyzed incidence, causes, and risk factors of avascular necrosis. In 105 children with 113 hips who developed avascular necrosis out of 636 consecutive patients with 823 hips treated nonoperatively for developmental dislocation of the hip in the years 1972–1976 the risk factors of avascular necrosis were determined. A method of treatment in most cases was Frejka pillow. Conventional radiographs obtained in AP views during the course of treatment and follow-up were analyzed. Avascular necrosis was found in 14 percent of the hips, classified as mild (49%), moderate (14%), and severe (37%). The differences between mild and severe cases were significant as regards age at the onset of treatment (p 0.006); with higher average age in mild forms, and degree of dislocation (p 0.01) with higher values in severe forms. The older the child was at the onset of treatment, the greater the risk of necrosis, notably if treatment was begun after 6 months of age. However, the incidence of the more severe cases was higher in the group up to 6 month of age. In general, avascular necrosis was more likely to occur in cases with high degree of initial dislocation and the differences between groups with low and high degree of dislocation were significant. In the group with highest initial dislocation the number of both mild and severe forms was high.

Part II evaluated the growth and remodeling of the hip joint with avascular necrosis after nonoperative treatment of developmental dislocation on the basis of conventional radiography. An attempt was also made to determine the correlation between the severity of necrosis as seen in conventional radiography and the clinical and radiographic appearance of the hip after completion of growth. Finally the prognostic value of conventional radiography in prediction of deformities of the proximal femur due to necrosis was estimated. 68 patients with 98 involved hips treated exclusively nonoperatively for developmental dislocation of the hip in whom avascular necrosis developed were selected for the study. The average age at the time when the final radiograph was made was 25 (18–36) years and the average follow-up period was 23 (18–35) years. 16 patients (27 hips) were examined twice after completion of growth with the time interval of 10

years, the second examination being at an average age of 30 (26–36) years. In this group also progress of signs and symptoms of degenerative changes in clinical and radiographic examination was noted. To achieve sufficient data necessary to establish indications for further operative treatment in 2 patients also CT examinations with three-dimensional surface reconstruction were performed after physseal closure. Physical examinations were performed in all patients after completion of growth. Radiographs made before the onset of treatment for developmental dislocation of the hip, during treatment, at the child's age of 4–6 years, all obtained until the cessation of growth, and at final assessment, were studied. By physical evaluation 77 hips were rated as excellent or good, being pain free or with only occasional mild pain after walking long distances, with a good range of hip motion and negative Trendelenburg sign. The reasons for 21 fair or poor clinical end-results were pain, mostly with activity, and limp due to pain and abductor weakness.

By radiographic evaluation in this group there were 50 hips rated as excellent or good, and 48 hips rated as fair or poor. In 29 hips excellent or good clinical findings at final review contrasted with fair or poor radiographic scores. In no case fair or poor clinical end-result coexisted with excellent radiographic ones. In the group examined twice after completion of growth with the time interval of 10 years no difference in clinical score was found in 14 hips, whereas in 10 hips there was deterioration of result.

CT examinations with three-dimensional surface reconstruction showed more clearly the deformity of the proximal femur when comparing to anteroposterior radiography. Also the portion of the femoral head not covered by the acetabular roof was better visible.

The course of remodeling of the hip during growth was different than expected (predicted from prognostic classification used) in 19 hips, where the classification based on radiographs appeared to be misleading.

Acetabular dysplasia was regarded as a major contributing factor in the development of early arthritis responsible for poor clinical results in this study. Deformities of the proximal femur together with acetabular dysplasia were responsible for poor radiographic results.

Changes in the lateral part of the metaphysis during

avascular necrosis appeared to be the most frequent site of metaphyseal involvement. In this type also the course of remodeling different than expected from prognostic classification was most common.

It was proven that in cases of avascular necrosis complicating the nonoperative treatment of developmental dislocation of the hip long-term follow-up is necessary since deformity of the hip may not be completely evident until the child passes through the years of rapid growth.

Part III of the study intended to evaluate the diagnostic capacity of MRI as compared to conventional radiography to identify changes in the proximal femoral epiphysis and metaphysis and subcapital growth plate disorders due to avascular necrosis in developmental dislocation of the hip. Also depiction of the growth zones of the normal proximal femur and changes in the subcapital growth plate in MRI during

development were of interest. Two groups of subjects were analyzed, first with no hip disease (36 children with 58 hips) and second with avascular necrosis complicating treatment of developmental dislocation of the hip (24 children with 33 hips). MRI appeared to be superior to radiography in assessment of subcapital growth plate and its damage in most of the hips. T1-weighted images together with FLASH 3D FAT SAT showed to be sufficient for the analysis of the growth plate and its damage.

On the basis of the analysis of MR pictures a method of mapping the subcapital growth plate allowing for location of damage was presented. It is to be hoped that with gradual improvement of surgical techniques the removal of damage from the subcapital growth plate may become feasible with careful MRI mapping out using the method presented in this study.

Terms, abbreviations, and statistics

- ATD* articulo-trochanteric distance.
- AVN* avascular necrosis of the proximal femur complicating the treatment of developmental dislocation of the hip.
- CT-3D* computed tomography examination with three-dimensional surface reconstruction from computed tomography scans.
- DDH* developmental dislocation of the hip; previous term CDH congenital dislocation of the hip; term congenital has been replaced by developmental and approved by all major orthopedic societies since many hips actually dislocate at a variable time after birth, and some do not dislocate until the child begins to walk (Coleman 1994).
- MRI* magnetic resonance imaging.
- Physis* in this study used alternatively to subcapital growth plate (longitudinal growth plate) of the proximal femur.
- SGP* subcapital growth plate, longitudinal growth plate, physis.

Statistics

Data were analysed in interval scale (e.g., angles), and ordinal scale (e.g., indexes). The observed distributions of my data differ significantly from the normal distribution. Therefore, the Spearman's rank order correlation coefficient was used to compare the ranks of individuals (or items) on selected two ordinal variables.

Comparisons between groups were made by using the Kruskal-Wallis one-way ANOVA, which is distribution-free and assume that the variable is ordinal.

Differences between frequencies were tested by using Gauss u-test.

The level of significance was 0.05.

Incidence, causes, and risk factors of avascular necrosis complicating the nonoperative treatment of developmental dislocation of the hip

Introduction

Although the incidence of avascular necrosis of the proximal end of the femur has declined it still remains the most severe complication of the treatment for developmental dislocation of the hip in children (Kalamchi and MacEwen 1980, Tönnis 1982, Lempicki et al. 1990, Kruczynski et al. 1990). Abnormal growth of the hip in this condition often leads to early arthrosis and disability (Cooperman et al. 1980, Rogala et al. 1988).

The goal of management with patients with DDH must always be to improve on the natural history of the disease process. Patients who have developed severe AVN as a result of treatment of DDH have an overall poorer prognosis than those who were not treated (Massie 1951, Cooperman et al. 1980, Kalamchi and MacEwen 1980). Early recognition of a progressive abnormality of the growing hip, prediction of resulting deformity, and choice of the correct treatment is of vital importance for these patients.

Incidence and causes of avascular necrosis (review of literature)

The rates of necrosis in the nonoperative treatment of DDH reported in the literature vary between 0 and 100 percent: Kalamchi and McFarland (1982) 0 percent, Ramsey et al. (1976) 0 percent, Grill et al. (1988) 2.38 percent, Tönnis et al. (1982) 0.4–32 percent, Salter et al. (1969) 5–30 percent, Lempicki et al. (1990) 14 percent, Palmen (1984) 19 percent, Weiner et al. (1977) 6–53 percent, Polakowski et al. (1953) 100 percent.

The incidence of AVN has been reduced recently by abandonment of forced reduction and the use of modern abduction devices, but it is still a substantial complication. It has been almost universally recognized that necrosis of the femoral head is a sequel of therapy and occurs with every form of hip splintage although Herold (1978) reported necrosis in 12 cases and Ferrer et al. (1991) in 34 cases of untreated dislocation of the hip. Necrosis may be also found in a contralateral hip of a child treated due to unilateral dislocation (Salter et al. 1969, Gage and Winter 1972, Gore 1974, Westin et al. 1976, Fisher and Cary 1978, Visser 1984, Kruczynski 1987, Thomas et al. 1989).

For some time there was divergence of opinion as

to whether the trauma of reduction or the retention in abduction was responsible for AVN. Today it is believed that multiple causes are involved (Tönnis 1987). Strangulation of the afferent blood vessels undoubtedly plays a major role. Several studies confirmed the negative influence of forced abduction and internal rotation on the blood flow in vessels supplying the proximal femur (Kolaczowski et al. 1953, Nicholson et al. 1954, Schoenecker et al. 1978, Law et al. 1982, Calvert et al. 1984). Also the statistics presented by Tönnis (1982) demonstrate that in the majority of cases the method of retention is to be blamed.

On the other hand the pressure exerted on the femoral head after reduction may exceed the physiologic limit. Animal experiments suggest, that depending on the severity of the pressure, compression of the nutrient vessels in the compressible cartilage leads to ischemic necrosis of the capital center of ossification (Crego and Schwartzmann 1948, Król 1963, Salter et al. 1969, Schoenecker et al. 1978); e.g., in the Lorenz position it was also the pressure exerted on the femoral head that led to pressure necrosis and local disorders of circulation. Gardiner and Duncan (1992) assume, that the smaller epiphysis in splinted infants may be secondary to altered blood supply due to increased pressure between the femoral head and acetabulum and increased tension of the adductor muscles in the thigh.

It has been also suggested by several authors that a circumscribed pressure on the femoral head has the same potential as a generalized pressure for precipitating ischemic necrosis (Somerville and Scott 1957, Pavlik 1957, Michel 1972, Tönnis 1987). This kind of pressure may be exerted by an inverted labrum and deformed acetabular rim (Tönnis 1987).

Yet another explanation for the cause of AVN is that the blood vessels supplying the femoral head may be susceptible to occlusion by direct pressure as shown by Bernbeck (1949). This view was also expressed by Tucker (1949), Kolaczowski et al. (1953), Nicholson et al. (1954), Mittelmeier (1961), Papadopoulos (1972), Ogden (1974), Westin et al. (1976) and confirmed experimentally in several studies. Usually the medial circumflex artery is compressed at the posterior acetabular rim or by the iliopsoas muscle.

Moreover, individual predisposing anatomic factors should be considered as was suggested by Wertheimer and Lopes (1971), Chung (1976) and Batory (1981).

Criteria for the diagnosis of avascular necrosis

Salter et al. (1969) gave 5 criteria for the diagnosis of AVN:

1. failure of appearance of the ossific nucleus of the femoral head during 1 year or longer after reduction;
2. failure of growth of an existing ossific nucleus during 1 year or longer after reduction
3. broadening of the femoral neck within 1 year after reduction;
4. increased radiographic density of the femoral head followed by the radiographic appearance of fragmentation;
5. residual deformity of the femoral head and neck when reossification is complete. These deformities include coxa magna, coxa plana, coxa vara, and a short, broad femoral neck.

Several authors indicate, that the condensation-fragmentation sequence, typical for Perthes disease is rare in AVN in DDH (Kohler and Seringe 1982, Kruczynski 1987).

Gage and Winter (1972) found, that in most cases AVN did not involve all of the epiphysis and part of it continued to grow. They established the following criteria for the diagnosis of partial necrosis of the femoral head:

1. residual deformity of the femoral head 2 years or more after closed reduction (usually a mild flattening of the medial aspect of the head);
2. abnormalities in a specific area of the epiphysis visible on roentgenograms made within 1 year of reduction. The changes occasionally progress to fragmentation of the epiphysis in the area that later shows residual deformity but more commonly are manifested as a failure of ossification of the nucleus in the corresponding area one year following reduction;
3. radiographic evidence of viability of the remainder of the femoral head.

When the criteria of Salter et al. for total necrosis are used, only 1 of the 5 points is required to make the diagnosis. When the criteria for partial necrosis are used, however, all 3 conditions have to be met (Gage and Winter 1972).

Table 1. General characteristics of 105 patients (113 hips) with avascular necrosis

	Number of hips
<i>Sex</i>	
Boys	17
Girls	96
<i>Hip</i>	
Right	60
Left	53
Unilateral	97
Bilateral	8
<i>Degree of dislocation</i>	
Dislocation	74
Subluxation	25
Dysplasia	7
Not defined*	7

* In 7 hips (out of 113 hips) treatment was started before 3rd month of age and in these children the radiograms were not performed. Thus in these hips the degree of initial dislocation was not defined.

Aim of the study

The aim of the study was to evaluate the incidence and risk factors of AVN complicating the nonoperative treatment of developmental dislocation of the hip.

Patients and methods

Risk factors for AVN were determined in 105 children who developed AVN in 113 hips out of 636 consecutive patients with 823 hips treated nonoperatively for DDH at the Orthopedic Department, Academy of Medicine (present name Karol Marcinkowski University of Medical Sciences) in Poznan in the years 1972–1976 (Table 1). All children were treated with a Frejka pillow (Lempicki et al. 1990). In a few instances requiring prolonged treatment, the Ortolani splint was used after an initial period of treatment with the Frejka pillow. In 4 children closed reduction was performed after a period of treatment with the Frejka pillow followed by immobilization in a plaster cast for a few weeks.

The only criterium for selection of patients to this study was AVN complicating the nonoperative treatment for developmental dislocation of the hip.

Conventional radiography

Conventional radiographs obtained in AP views during the course of treatment and follow-up were analyzed. The consecutive radiographs were investigated for signs of AVN which were established according to the criteria of Salter et al. (1969), Gage and Winter

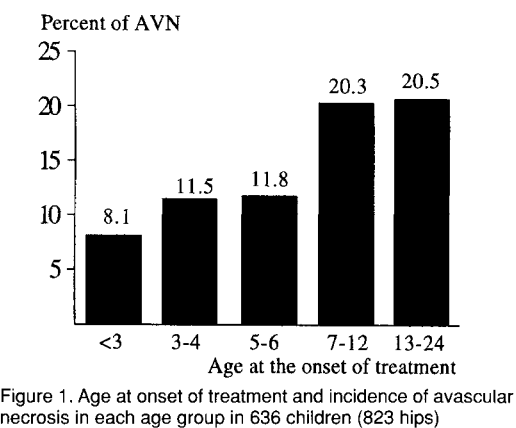


Figure 1. Age at onset of treatment and incidence of avascular necrosis in each age group in 636 children (823 hips)

Table 2. Percent of avascular necrosis in relation to type (Kruczynski) and severity of changes, and mean age at the onset of treatment in each type

Severity	Type	n	%	Mean age (months)
Mild	I	55	49	7.9
Moderate	II	16	14	7.3
Severe	III-V	42	37	
	III	21	18	5.1
	IV	3	3	6.0
	V	18	16	4.2

Table 3. Age at the onset of treatment for developmental dislocation of the hip and the percentage of avascular necrosis in each age group in relation to severity of changes in 636 children (823 hips)

Months	< 3 n=99	3-4 n=353	5-6 n=143	7-12 n=146	13-24 n=82
Mild	1.7	4	4.4	14	15
Moderate	2.7	0.5	2.9	2.5	4.8
Severe	3.7	7	4.5	4.3	1.2

Table 4. Initial dislocation and the percentage of avascular necrosis in each group in relation to severity of changes in 567^a children (724 hips)

	Degree of dislocation				
	1 n=174	2 n=224	3 n=181	4 n=94	5 n=51
Mild	2.6	5.1	5.9	18	16
Moderate	0	1.3	1.6	6.9	2.3
Severe	1.7	5	8.6	4	7

^a In 69 children with 99 hips treatment was started before 3rd month of age and in these children the radiograms were not performed. Thus in these hips the degree of initial dislocation was not defined.

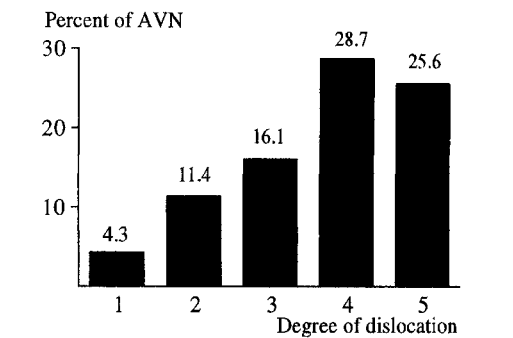


Figure 2. Initial dislocation and the incidence of avascular necrosis in each group in 567^a children (724 hips)
^a in 69 children with 99 hips treatment was started before 3rd month of age and in these children radiography was not performed. Thus in these hips the degree of initial dislocation was not defined.

(1972), and Bucholz and Ogden (1978). To describe the grade of dislocation the five-degree scale of Lempicki (1989) was used and to assess the severity of necrosis my own five-type classification was applied (Kruczynski 1987, see Appendix). No additional radiographs were made for this part of the study.

Results

Avascular necrosis was found in 113 (14 percent) of 823 hips. Mild and moderate changes were found in 63 percent of hips with AVN (Table 2), i.e., 10 percent hips treated due to DDH.

The differences between mild and severe cases were significant as regards age at the onset of treatment (p 0.006) with higher average age of children with the mild forms, and degree of dislocation (p 0.01) with higher average values in hips with severe changes.

The older the child was at the onset of treatment, the greater the risk of necrosis, notably if treatment was begun after 6 months of age and the difference was statistically significant (p 0.05) (Figure 1). However, the incidence of the more severe cases was higher in the group up to 6 month of age (Table 3).

In general AVN was more likely to occur in cases with high degree of initial dislocation (Figure 2). The differences were significant between groups 1 and 4 (p 0.01), 1 and 5 (p 0.01), 2 and 4 (p 0.02), 2 and 5 (p 0.02), and 3 and 5 (p 0.02). In the groups with highest initial dislocation the number of both mild and severe forms was high (Table 4).

Discussion

General considerations on incidence of avascular necrosis

The incidence of AVN is one of the main factors in assessment of the methods of treatment of DDH, but there is still some disagreement on which radiographic changes in the proximal femur are related to AVN. It should be noted that in the literature often only the more severe disturbances are taken into consideration. However, it remains to be seen whether the mild alterations of the ossific nucleus are not also likely to leave sequelae (Tönnis 1982). It is even more interesting when considering the suggestions that the slight irregularity of the capital center of ossification constitutes a readaptation to the changed conditions of pressure obtained in the acetabulum after reduction (Król 1963, Salter et al. 1969, Tönnis 1982), the "remodeling defect" of Schede (1940, 1950). Salter et al. (1969) described "temporary irregular ossification" as a reaction to temporary reduced blood flow, not regarding these changes as AVN. According to Tönnis (1987) the difference lies exclusively in the degree of the pressure which causes these alteration, and in the extent of the pathologic reaction. He includes even the mildest alterations in the appearance of the ossific nucleus after reduction as AVN and states that the term "remodeling defect" should not be retained. In the present study I also included changes considered by other authors as transient disorders of vascularization but the impact of this on patient outcome has yet to be determined. Most probably the reports on incidence of AVN are disturbed by its wide variety of radiographic presentations (Król 1963, Kohler and Seringe 1981, Kruczynski 1987, 1995). In numerous instances of open reduction of dislocated hips previously subjected to "atraumatic" closed management, Tönnis found deep cartilage lesions in the femoral head without signs of necrosis being detectable in the capital center of ossification. When analyzing the material I found cases with disturbances being dormant for many years giving deformity when the growth was accelerated and hence indicating previous physal damage. I agree with Kalamchi and MacEwen (1980) that these cases should be included into the study because there was no other explanation for the appearance of the deformity.

On the other hand the femoral head is sometimes composed of two or more centers of ossification and care must be taken not to misinterpret as necrosis structural disturbances or irregularities of growth also called "developmental defect" (Weickert 1970, Tönnis 1987). A "developmental defect" is said to exist when the development of the femoral head is

disturbed from the outset. These defects would account for higher percentage of total disturbances (Tönnis 1987) although I could not recognize them in my material.

In conclusion therefore I may state, that it is not possible to deduce confidently from the incidence of necrosis how atraumatic our methods of nonoperative treatment really are. Only analysis of the late results yields definitive information (Kalamchi and MacEwen 1980, Tönnis 1982, Lempicki et al. 1990).

Risk factors of avascular necrosis

Age at the onset of treatment: My study shows that the older was the child at the onset of treatment, the more frequent was the complication of AVN. I also noticed proportionally higher incidence of more severe forms of AVN in the younger children. Several authors has pointed to the vulnerability of the hip joints of young children (Becker 1969, Salter et al. 1969, Fisher 1978, Palmén 1984). However, Gage and Winter (1972) observed AVN more frequently in older children. In contrast, in the study of Gregosiewicz and Wosko (1988) the frequency of occurrence of necrosis in all age groups seemed to be similar. But in the group of youngest children, severe necrosis was almost three times more frequent than the mild type. Also in the view of the findings of Kalamchi and MacEwen (1980) the most severe forms of AVN appear in the younger age group, from birth to 6 months. This is also consistent with the findings of Salter et al. 1969 and Ogden 1974. In the group analyzed by Grill et al. (1988) with treatment started within the first 3 months of life, the rate of AVN was only 50 percent of the rate that occurred if treatment was begun between the third and sixth month of life. In their opinion which is generally accepted the earlier the treatment started, the better result achieved.

Interesting is the collective statistics of the Commission for the Study of Hip Dysplasia of the German Society for Orthopedics and Traumatology, based on 3316 cases, which indicate that with the atraumatic methods used today, age is not a significant factor in the occurrence of AVN (Tönnis 1982).

Degree of dislocation: My study proves that AVN was more likely to occur and the number of severe changes was increased in cases with high degree of initial dislocation. In the series investigated by Salter et al. (1969) the incidence of AVN of the femoral head was found to be higher in complete dislocation.

Tönnis (1982) assumes that it is the degree of the dislocation—that commonly worsens with advancing age—not the age of the child that has an essential in-

fluence on the incidence of necrosis. In contrast Gregosiewicz and Wosko (1988) and Cooperman et al. (1980) found no interesting relationship between the height of displacement of the femoral head and AVN.

Method of treatment: To draw a meaningful comparison of the rates of AVN associated with different treatment methods, it is first necessary to subdivide the population into comparable groups, regarding age, grade of dislocation and grade of AVN, thus making it possible to compare cases that had similar initial features (Tönnis 1982). Very few studies follow this steps making the comparison of results not reliable. In the present study the method of treatment was almost uniform and I subdivided the material according to the age at the start of treatment and the degree of dislocation.

Frejka pillow, used in patients being investigated in this study has been criticized by Tönnis (1984) for inducing maximum abduction. It should, however, be noted that this method of treatment starting with a "soft" Frejka pillow, did not force the hip into maximum abduction and motion of the legs in the hips, although limited were still possible. In a few children in this study closed reduction with subsequent immobilization in a plaster cast after a period of abduction treatment was applied with no bearing on the final result.

The highest incidence of AVN noted in the literature is associated with one-stage manual reduction especially vulnerable when followed by immobilization in a frog-leg position. The rate of necrosis is mostly above 30 percent reaching even 100 percent in some studies (Polakowski et al. 1953, Przychodzki 1953, Dooley 1964, Salter et al. 1969, Gonczarowa et al. 1970, Gage and Winter 1972, Cooperman et al. 1980, Buchanan et al. 1981). It seems very likely to me, that in these cases all three causes of AVN, i.e., strangulation of the afferent blood vessels, pressure exerted on the femoral head and occlusion of the blood vessels by a direct pressure play some role.

The incidence of necrosis is significantly reduced in older infants with the implementation of traction prior to reduction (Crego and Schwartzmann 1948, Gizycka 1953, Scott 1953, Salter et al. 1969, Gage and Winter 1972, Weiner et al. 1977, Fisher and Cary 1978, Kalamchi and MacEwen 1980, Buchanan et al. 1981, Visser 1984) although there is controversy about the exact place for prereduction skin traction (Kahle et al. 1990), e.g., there is no consensus regarding the amount of weight to use, the direction of pull to exert, and the duration of traction required (Coleman 1994). The fact that procedures making use of preliminary stretching, such as overhead traction,

introduced by Crego in 1954, decrease the proportion of necrosis can only be explained by the fact that the pull of the soft tissue structures, even despite the unphysiologic frog position, is then substantially diminished as it was proven by Petit (1955).

At present it is obvious that hip reduction by non-operative treatment should not be a one-step action but a continuous process stimulated by spontaneous kicking movements of the leg in hip abduction until the hip becomes stable, thus avoiding AVN (Grill et al. 1988). The Pavlik harness which in many places is the standard for nonoperative treatment and adopts all these requirements is associated with AVN in 0 to 15 percent of the cases (Kalamchi and MacFarlane 1982, Ramsey et al. 1976, Tönnis 1982, Grill et al. 1988).

In order to introduce the least traumatic procedure, Tönnis (1987) found arthrography performed at the start of treatment as being able to identify the very high-risk dislocations and thus helping to avoid measures that would be inappropriate for a given patient. I could not confirm this since arthrography had been used only occasionally in my series.

It was beyond the scope of this investigation to analyze the predisposing anatomic factors for the cause of AVN but I could not establish any correlation between the sex and increased risk of avascular necrosis. Such a correlation was suggested by Chung (1976) in children with Legg-Calve-Perthes disease. The proposed reason was deficient vascular anatomy of the proximal femur encountered in several cases in boys, and found earlier also by Wertheimer and Lopes (1971). The sex ratio in my patients was similar in the group treated due to dysplasia and in the subgroup with avascular necrosis.

In conclusion than, considering the fact that even the mild alterations of ossific nucleus were included, the rate of AVN in my study is on par with other methods of nonoperative treatment of DDH used at that time. The risk of AVN in the analyzed material increased with age and also with the higher degree of dislocation. As far as the cause of AVN in this patients is concerned it seems that the strangulation of the afferent vessels or occlusion by direct pressure from the iliopsoas muscle was most likely.

Review of the literature and own study proves, that the problem of AVN in DDH is not yet solved, and a threat to the growing hip, although much diminished, still exists. Previous research has largely focused on etiology and pathogenesis of changes in the proximal femur (Król 1963, Salter et al. 1969, Schoenecker et al. 1978, Batory 1981, Calvert et al. 1984, Tönnis 1987); radiographic presentation has also been discussed a lot (Salter et al. 1969, Gage and Winter 1972, Bucholz and Ogden 1978, Kalamchi and

McEwen 1980, Kohler and Seringe 1981, Siffert 1981, Thomas et al. 1982, Zwierzchowski and Zwierzchowska 1985, Kruczynski 1987, Tönnis 1987). Nevertheless, long term clinical and radiographic follow-up is required but have been reported

only in few studies (Massie 1951, Gage and Winter 1972, Fisher and Cary 1978, Cooperman et al. 1980, Thomas et al. 1982, Kruczynski 1987). This is the aim of the second part of this quest.

Growth and remodeling of the hip joint with avascular necrosis after nonoperative treatment of developmental dislocation of the hip

Introduction

Growth of the normal proximal femur

To understand the deformities occurring during growth of the proximal femur due to avascular necrosis, the growth in normal conditions should first be clarified. It continues as a complex process, starting from initially single cartilaginous proximal end of the femur, than progressively divides into several growth cartilages, each one playing a specific role (Figure 3):

- *the growth cartilage of the epiphysis* is oriented radially around the secondary ossification center; it is poorly organized and the rate of growth from radial apposition of cells is much slower than that of the SGP (Iannotti 1990).
- *the subcapital growth plate of the neck* which is made up of three major components:
 - a cartilaginous component with different histologic zones, contributing to longitudinal growth;
 - a bony metaphysis, active in the removal of the cartilaginous matrix, the formation of bone, and the histologic remodeling of the secondary spongiosa (Iannotti 1990);
 - a fibrous component made up of the groove of Ranvier, contributing to an increase in the diameter, or latitudinal growth, of the growth plate, and the perichondrial ring of LaCroix, a strong mechanical

support at the bone-cartilage junction of the growth plate.

- *the femoral neck isthmus*, connecting in infancy the trochanteric and femoral growth zones along the lateral border of the neck as a residual of their former common origin; it contributes to the lateral width of the neck (Siffert 1981, Grybos 1983).
- *the greater trochanter* with appositional growth in a manner identical to the femoral head
- *the trochanteric growth plate* similar in function to the SGP of the femoral neck; it contributes to bony enlargement of the neck.

The lesser trochanteric epiphysis minimally contributes to the overall shape of the proximal femur but through the attachment of the iliopsoas muscle, there is a mechanical effect on the development of the rest of the proximal femur (Ogden 1995).

Anatomy and function of the growth plate cartilage

The growth plate cartilage begins at the top of the reserve zone and ends with the last intact transverse septa at the bottom of the cell columns in the hypertrophic zone (Brighton 1978). The cartilage is divided histologically into three zones: the reserve, the proliferative, and the hypertrophic, each with characteristic histologic and biochemical features (Figure 4).

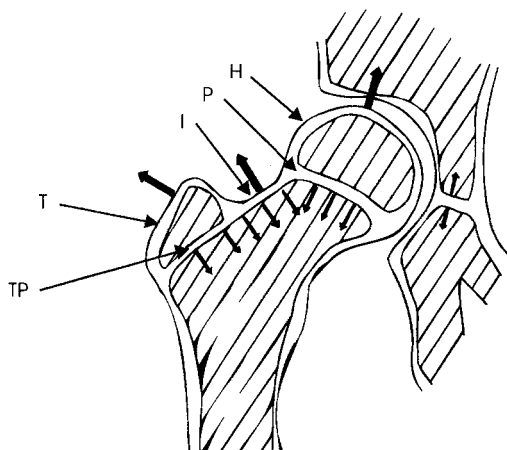


Figure 3. Growth zones of the proximal femur: H – growth cartilage of the epiphysis; P – subcapital growth plate; I – femoral neck isthmus; T – growth cartilage of the greater trochanter; TP – greater trochanteric growth plate

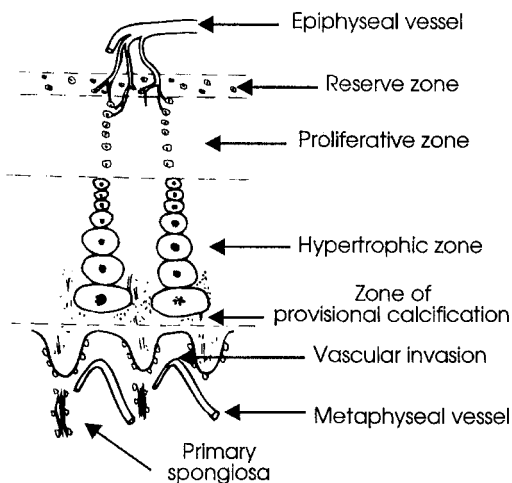


Figure 4. Diagram of the subcapital growth plate of the proximal femur.

Reserve zone. The zone is located just below the secondary center of ossification. Cellular proliferation in the reserve zone is sporadic (Kember 1960) and the O_2 tension is low [~ 21 mmHg] (Brighton and Heppenstall 1971). The reserve zone does not actively participate in longitudinal growth. The function of the reserve zone is not clear, and appears to be that of matrix production and storage of nutrients and raw materials that will be used in the lower zones of the physis (Farnum and Wilsman 1988).

Proliferative zone. The zone is characterized by longitudinal columns of flattened cells. The uppermost cell in each column is the progenitor cell. It is not derived from the reserve zone cells. The oxygen tension of the proliferative zone is higher than in any other zone [57 mmHg] (Brighton and Heppenstall 1971). The presence of rich glycogen stores and high oxygen tension support aerobic metabolism in the proliferative zone chondrocyte. The cells have a low ionized and total calcium content, and the mitochondria are primarily involved in ATP production (Iannotti 1990). The function of the proliferative zone is matrix production and cellular division, which together contribute to longitudinal growth.

Hypertrophic zone. The histologic characteristic of the hypertrophic zone is cellular enlargement to 5 times the size of the proliferative zone cell (Brighton et al. 1973, Hunziker et al. 1987). It is the more active end of the physis. Traditionally it has been divided into two parts, the upper, or zone of maturation, and the lower, or zone of degradation with the lowest also called the zone of provisional calcification. The oxygen tension in the hypertrophic zone is very low (24 mmHg) and its functions are to prepare the matrix for calcification and to calcify it. This vigorous activity is supported by anaerobic metabolism and subsequent depletion of glycogen stores. ATP is no longer formed in the mitochondria (Robertson 1990) which are initially active in calcium accumulation and matrix vesicle secretion. In the lower hypertrophic zone mitochondrial calcium is released and matrix mineralization occurs. The factors responsible for mitochondrial calcium accumulation and release are not clearly understood at present. The ultimate fate of the hypertrophic zone cell is cell death.

Vascular supply to the proximal femur

Vascular supply to the proximal femur has been thoroughly described by several authors (Tucker 1949, Howe et al. 1950, Trueta 1957, Crock 1965, Lauritzen 1974, Ogden 1974, Chung 1976).

Crock (1965) described arteries of the proximal femur on the basis of a three-plane analysis and on standard anatomic terminology, as follows:

- the arterial ring of the femoral neck (extracapsular); the femoral circumflex arteries are the principal vessels forming the ring;
- the ascending cervical branches of the arterial ring of the femoral neck; these arteries give rise to metaphyseal and epiphyseal branches;
- the arteries of the round ligament.

Chung (1976) completed the picture and described the intraarticular arterial ring, formed by the 4 ascending cervical arterial groups. The intraarticular ring in his study was more often incomplete in boys and that may explain in part the greater susceptibility of boys to Legg-Calve-Perthes disease. According to Crock (1965) and Chung (1976) the basic extracapsular and transcapsular arterial pattern to the proximal femur is established at birth and probably persists throughout life.

The age dependent differences in the significance of vessels responsible for vascular supply to the proximal femur in children were pointed out by Trueta (1957). The differences defined 5 chronological phases with lateral epiphyseal arteries gradually becoming the major blood supplier to the epiphysis, a finding not fully supported by Crock (1965), Lauritzen (1974), and Ogden (1974). According to Trueta there are no anastomoses within the epiphysis and no vessels cross the SGP when already formed. Before the formation of SGP he found vessels passing the layer of cartilage cells later responsible for the creation of the physis.

Ogden (1974) found numerous vessels, arteriosinoidal in appearance, crossing the subcapital growth plate, usually in more peripheral regions, in children up to 18 months of age. As the growth of the ossification center of the epiphysis continued, the number of vessels observed traversing the growth plate was reduced. No traversing vessels were observed once a subchondral bone plate was juxtaposed to the epiphyseal side of the growth plate. Chung (1976) suggests that careful inspection from different angles reveals no vessels passing through the central epiphyseal plate but rather through the peripheral perichondrial fibrocartilaginous complex.

The specific model of vascular supply to the proximal femur, with no anastomoses within the epiphysis, few or finally no vessels crossing the growth plate, and possible anatomic anomalies may explain the susceptibility to deficient blood supply to this area in children.

Nutrition of the growth cartilages of the proximal femur

The proliferative layer of the growth cartilage of the epiphysis receives its nutritional diffusion from the

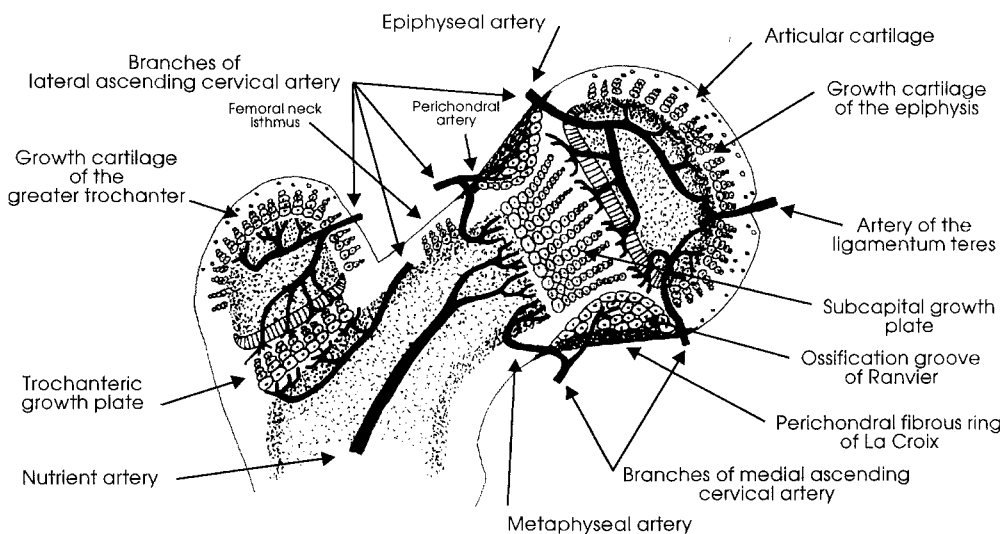


Figure 5. Structure of the proximal femur and its blood supply.

joint fluid, so it is independent of blood supply to the epiphysis. The deeper layers of the epiphyseal cartilage, most notably those concerned in the process of endochondral ossification, are supplied from vessels to the bony centrum (Siffert and Feldman 1980) (Figure 5).

Each of the 3 components of the SGP has its own distinct nutrient supply (Trueta and Morgan 1960, Chung 1976):

Cartilaginous component. Although vascular channels pass through the reserve zone, they do not appear to supply it. Epiphyseal arteries via small arterial branches arising from the main artery in the secondary center of ossification, and passing through small cartilage canals in the reserve zone, supply the top of the cell columns in the proliferative zone (Trueta and Morgan 1960). Each small arterial branch supplies the top portion of from 4 to 10 cell columns. The proliferative zone, therefore, is well supplied with blood. There are no arterial branches from the epiphyseal penetrate the cartilage portion of the growth plate beyond the uppermost part of the proliferative zone. This means no vessels pass through the proliferative zone to supply the hypertrophic zone. The nutrients to the hypertrophic cells are delivered by diffusion.

Bony metaphysis. The region of bony metaphysis is richly supplied with blood both from terminal branches of the nutrient artery as well as from metaphyseal arteries arising from the ascending cervical arteries. The capillary loops of the main nutrient vessel end at the last cartilaginous transverse septum of the bone-cartilage interface of the growth plate. The vessels

turn back on themselves to form a venous return (Morgan 1959, Brookes and Landon 1963, Anderson and Parker 1966). The vessels do not penetrate the hypertrophic zone of the growth plate.

Fibrous component. It is richly supplied with blood from several perichondral arteries.

The peripheral cartilage cells of the greater trochanteric epiphysis receive nourishment from the vessels of the attached muscles and tendons.

The proliferative cells of the longitudinal growth plate of the greater trochanter receive vascularity that penetrates its bony end plate from the trochanteric epiphysis. The metaphyseal side of the longitudinal growth plate of the greater trochanter receives vascularity from the descending metaphyseal arteries (arising from the lateral ascending cervical artery) and from nutrient artery.

The epiphyseal vessels are thus responsible for permitting longitudinal growth of the femoral neck to occur, whereas the metaphyseal vessels nourish the osteoprogenitor cells, which lay down bone on the cartilage matrix (Shapiro 1987). The SGP, deprived of its epiphyseal vascularity, may be delayed or fail to grow at all (Siffert 1981).

Radiographic presentation of avascular necrosis of proximal femur after nonoperative treatment of developmental dislocation of the hip (review of classifications)

To describe the radiographic picture of AVN several classifications in recent years have been proposed in the literature.

Tönnis and Kuhlmann (1968) described a four-grade classification with the first three grades describing the changes in the capital ossific nucleus with progressing severity and grade 4 where also metaphyseal involvement was present.

Bucholz and Ogden (1978) paid more attention to possible damage to the SGP as a result of localized vascular disturbance. In the first type only the epiphysis is affected whereas in more severe types the lateral, whole, and medial parts of the physis are involved. Robert and Seringe (1982) propose a classification similar to that of Bucholz and Ogden with an additional group V, where isolated changes in the metaphysis are present.

Kalamchi and MacEwen (1980) developed another four-group classification of vascular changes. In group I changes affect the ossific nucleus, in group II, III, and IV signs of lateral, central and whole physeal damage are present, respectively. Their evaluation of skeletal mature patients revealed, that the classification of the vascular changes used in their study is helpful in predicting the natural history of development of the hip. I introduced some modifications to the classification of Kalamchi and MacEwen (Kruczynski 1987). In my five-type prognostic classification the first two types describe epiphyseal alterations, the rest both epiphyseal and metaphyseal changes.

But as Robert and Seringe (1981) state, AVN may also take radiographic forms different from those described in the prognostic classifications.

Aims of the study

The aims of the study were:

- to evaluate the growth and remodeling of the hip joint with avascular necrosis on the basis of conventional radiography;
- to determine the correlation between the severity of avascular necrosis as seen in conventional radiography and the clinical and radiographic appearance of the hip after completion of growth;
- to estimate the prognostic value of conventional radiography in prediction of deformities of the proximal femur due to avascular necrosis.

Patients

A retrospective review was carried out of patients treated exclusively nonoperatively for DDH at the Department of Orthopedics, Academy of Medicine (present name Karol Marcinkowski University of Medical Sciences) in Poznan between 1957–1976, in

Table 5. General characteristics of 68 patients (98 hips) with a sequelae of avascular necrosis

Sex	
Male	9
Female	59
Hip	
Right	46
Left	52
Dislocation	
Unilateral	35
Bilateral	33
Avascular necrosis	
Unilateral	38
Bilateral	30
Degree of dislocation	
Dislocation	72
Subluxation	16
Dysplasia	2
Contralateral hip	8

whom AVN developed. The criteria for selection of patients for the study were complete radiographic documentation and an examination after the end of growth.

68 patients with 98 involved hips fulfilled the criteria and were available for the study (Table 5). All the patients were seen for physical and radiographic examination. The average age at the diagnosis of DDH in this series was 10 (1–27) months. All patients were treated initially with the Frejka pillow. Only in a few instances requiring prolonged treatment, the Ortolani splint was used after an initial period of treatment with the Frejka pillow. The average duration of treatment was 15 (6–35) months. In 12 hips in 11 patients closed reduction after initial abduction treatment was necessary followed by retention in a hip spica cast in 80°–100° flexion, 50°–80° abduction and about 20° internal rotation for 6–8 weeks. After the spica cast a Frejka pillow was continued until the development of the hip was considered satisfactory.

The average age at the time when the final radiograph was made was 25 (18–36) years and the average follow-up period was 23 (18–35) years. 16 patients (27 hips) were examined twice after completion of growth, the second examination being at an average age of 30 (26–36) years. In this group also progress of signs and symptoms of degenerative changes in clinical and radiographic examinations was noted.

To achieve sufficient data necessary to establish indications for further operative treatment in 2 patients also CT examination was performed after physeal closure.

Methods

Physical examination

At final assessment the patients were inquired as regards general condition, hip pain (occasional, constant, with activity), limp, restriction of activity, and the need for external support. Also occupation whether standing or sitting, deliveries, and a subjective estimation of the hip status were recorded. Examination included hip movement with attention to joint contracture, presence of Trendelenburg and Duchenne signs, leg length, and limp.

Radiographic examination

Radiographs were analyzed obtained in AP views:

- before the onset of treatment for DDH
- during treatment
- at the age of 4–6 years
- all made until the cessation of growth
- at final assessment; at that time also the radiographs with Rippstein's technique were made.

In evaluation of radiographs the principles of Müller's (1956) osteometry were adopted.

On radiographs made before the onset of treatment the degree of dislocation was described in a five degree scale according to Lempicki (1989). On radiographs made during treatment of DDH the appearance, extent, and severity of AVN was analyzed. Radiographs made at the age of 4–6 years were chosen for evaluation since at that age the process of AVN was already at the healing stage in most of the cases.

The parameters studied on radiographs made at the age of 4–6 years and at final assessment aimed to describe the development of proximal femur, acetabulum, and position of femoral head within the acetabulum (for method of measurements see Appendix, Figure 27). At the age of 4–6 years the following parameters were studied:

Proximal femur:

- projected neck-shaft angle;
- modified epiphyseal index of Eyre-Brook (1936);
- head-neck index of Heyman and Herndon (1950);
- articulo-trochanteric distance (ATD) of Edgren (1965);
- epiphysis-shaft angle of Alsberg (1899);
- femoral head sphericity of Mose (1964).

Acetabulum:

- acetabular index of Heyman and Herndon (1950);
- acetabular angle of Hilgenreiner (1925).

Head within acetabulum:

- acetabulum-head index of Heyman and Herndon (1950);
- CE angle of Wiberg (1939);

- lateral displacement angle of Labaziewicz (1979).

In addition to the parameters described at the age 4–6 years, at the final examination were also estimated: true neck-shaft angle, true anteversion angle, and head-shaft index of Kruczynski (1987), all describing the development of the proximal femur. True neck-shaft angles and true anteversion angles were calculated from the conversion table of Rippstein after measuring on radiographs projected neck-shaft and anteversion angles.

At the final examination the measurement of epiphyseal index, acetabular angle, and epiphysis-shaft angle of Alsberg was not possible due to the closure of the SGP and triradiate cartilage.

CT examinations with three-dimensional surface reconstruction from CT scans were performed with a Siemens Somatom scanner with a slice thickness of 2 mm. First scan was obtained at the level of acetabular roof and at most 20 scans were made.

Avascular necrosis of the proximal femur was diagnosed according to the criteria depicted in the first part of the study. To classify the changes within the proximal femur the classification of Kruczynski (1987) was used (see Appendix, Figures 22–26).

Radiographic signs of prearthrosis of the hip were assessed according to De Seze and Lequesne (1956). These were osteophytes around the fovea capitis, osteophyte giving the picture of double bottom of the acetabulum, and osteophyte giving picture of marginal rim. The width of the joint space was also recorded.

Angles and indexes of normal hips and those of dysplastic hips without AVN measured and calculated at 4–6 years and after completion of growth were taken from the study of Heyman and Herndon (1950), Edgren (1965), Szczekot (1974), Kruczynski (1987), and Wierusz-Kozłowska (1995).

The clinical and radiographic results were evaluated according to the modified criteria accepted at the XXXVI Congress of the Polish Orthopedic Association (Tables 6, 7).

Results

Clinical status at final examination

Out of 68 patients available for the examination after the completion of growth 9 regarded their clinical status as unsatisfactory. The reasons in all of them were pain, mostly with activity, and limp due to pain and abductor weakness. All patients were able to walk and none required a cane.

27 patients had occasional pain, especially with activity. 2 patients (type III and V avascular necrosis) had constant pain and they frequently used analgesics

Table 6. Criteria for evaluation of clinical end-results

Points	4	3	2	1
<i>Range of motion</i>				
Flexion	>110°	76°–110°	41°–75°	0°–40°
Abduction	> 30°	16°–30°	5°–15°	< 5°
Internal rot.	> 25°	16°–25°	5°–15°	< 5°
<i>Gait</i>				
Limp	none	mild	moderate	significant
T sign		—	±	+
D sign		—	±	+
<i>Pain</i>				
	none	none	mild after walking	constant during walking
<i>Physical activity</i>				
Restriction of activity	unrestr.	unrestr.	weariness after walk; slight	early weariness; significant
<i>Clinical grade:</i>				
excellent	14 points			
good	12–13 points			
fair	8–11 points			
poor	4–7 points			
Any contracture: in flexion < 20°, in abduction < 5°, ext.rot. < 20° decreases the final result by 1 grade. Greater contracture qualify the result as poor.				

Table 7. Criteria for evaluation of radiographic end-results

	Excellent	Good	Fair
Shenton line	uninterrupted	uninterrupted	slightly interrupted
Lateral displacement angle	< 20°	20°–25°	> 25°
Wiberg CE angle	> 25°	20°–25°	15°–19°
Anteversion angle	10°–40°	0°–9° 41°–50°	51°–60°
Neck-shaft angle	120°–140°	110°–119°	141°–155°
Acetabular roof	well developed head, well covered	slight uncovering, irregular ossification	uncovering irregular ossification
Femoral head shape	round	round, flattening	significant flattening, spherical
Position of the greater troch.	below center of the head	below the head surface	on the level of the head surface
Femoral head-acetabulum centering	head well centered	head well centered	decentering hip, no subluxation
Cases outside the above limits are classified as 'Poor'			

Table 8. Clinical signs and symptoms in 68 patients (98 hips) in relation to the type of avascular necrosis

Type of avascular necrosis	n	Signs and symptoms		Pain		Limp due to		T & D	Leg shortening	Negative subjective estimation
		no	>1	occasional	constant	pain	T-sign +			
I	43	29	3	8	–	1	–	3	4	1
II	10	3	–	5	–	–	–	1	1	–
III	26	9	10	9	1	2	2	9	9	3
IV	6	2	2	4	–	2	1	1	4	1
V	13	3	6	7	1	3	3	7	5	4

Table 9. Occurrence of pain in the hip associated with pregnancy in 21 women (26 hips) in relation to type of avascular necrosis (AVN)

Type of AVN	I	II	III	IV	V
Pain unchanged	9	5	3	1	3
Pain increased	–	–	1	2	2

(Table 8). In 6 patients the pain was bilateral, in 23 unilateral and always present in hips with previous AVN.

17 patients described their occupation as mostly standing and 10 of them had painful hips. 5 women correlated increased hip pain with pregnancy and delivery, whereas 16 women observed no difference (Table 9).

A good range of motion was observed in all except 2 hips of type III and V. 2 hips had contractures, one in flexion and external rotation and the other in external rotation. 16 hips had some loss of abduction.

Leg-length discrepancy was present in 23 patients and did not exceed 2 cm.

Radiographic parameters during growth and at final follow-up; CT-3D examination

The values of parameters varied according to the site of radiographic changes of avascular necrosis (Table 10). The main differences between types I and II at the age of 4–6 years were observed in the values of the epiphyseal index and head-neck index, with lower values in type II although the differences were not statistically significant. During the final examination the main difference was in the value of ATD with no sta-

Table 10. Values of parameters measured at age 4–6 years (a), and at final assessment (b) in 68 patients (98 hips). Mean values in mm or degrees, SD

Type of avascular necrosis											
I		II		III		IV		V			
a	b	a	b	a	b	a	b	a	b	a	b
A	146 6.7	137 7.8	140 10	134 5.7	144 11	138 12	131 8.1	123 9.4	128 16	126 12	
B		129 6.8		129 11		128 8.4		118 7.4		118 11	
C		38 8.1		35 7.3		41 14		33 11		39 11	
D	41 6.5		37 6.4		43 6.6		34 8.0		30 5.9		
E	159 21	164 20	147 15	164 20	158 19	155 22	155 12	158 14	134 24	126 40	
F	18 12	22 8.3	17 2.2	16.2 4.1	16 8.4	13 14	15 4.9	9.5 11	8.2 16.3	4.4 18	
G	73 6.8		73 8.0		76 8.2		51 8.9		59 23		
H	26 4.0	32 4.4	29 6.4	33 6.8	28 3.1	32 5.5	23 3.9	29 4.6	23 4.1	26 6.5	
I	21 6.3		20 6.1		19 3.7		23 3.9		24 7.6		
J	80 10	79 9.3	89 7.2	76 3.8	82 7.8	79 7.4	89 5.9	84 8.4	80 8.4	71 13	
K	20 7.2	26 9.8	25 5.7	22 4.9	21 6.0	27 8.6	23 6.0	23 10	18 5.0	19 12	
L	27 3.9	19 4.4	26 3.4	20 3.0	25 4.7	20 4.8	26 2.4	21 6.2	30 4.6	22 7.1	
M	1.7 0.9	1.8 0.8	1.8 0.6	1.4 0.7	1.4 0.8	2.2 1.4	2.8 1.3	2.5 1.7	2.3 1.2	5 2.2	
N		199 18		193 14		202 24		189 8.8		220 26	
O		4.6 0.7		4.9 0.3		4.2 1.0		3.5 1.4		3.7 1.0	
P		3.9 1.0		3.8 0.8		3.2 1.0		3.0 1.3		2.5 0.8	
Statistically significant differences P 0.05											
A	Neck-shaft angle		a) I–IV, I–V, III–V;		b) I–IV, I–V, III–IV, III–V						
B	True neck-shaft angle				b) I–IV, I–V, II–V, III–V						
C	True angle of anteversion										
D	Epiphyseal index		a) I–V, III–V								
E	Head-neck index		a) I–V		b) I–V, II–V						
F	ATD		a) I–V		b) I–III, I–V						
G	Alsberg angle		a) I–IV, II–IV, III–IV, III–V								
H	Acetabular index		a) III–V		b) I–V						
I	Acetabular angle										
J	Acetabulum-head index										
K	Wiberg CE angle										
L	Lateral displacement angle										
M	Head sphericity (Mose)				b) I–V, II–V III–V						
N	Head-shaft index										
O	Clinical end-result				b) I–V						
P	Radiographic end-result										

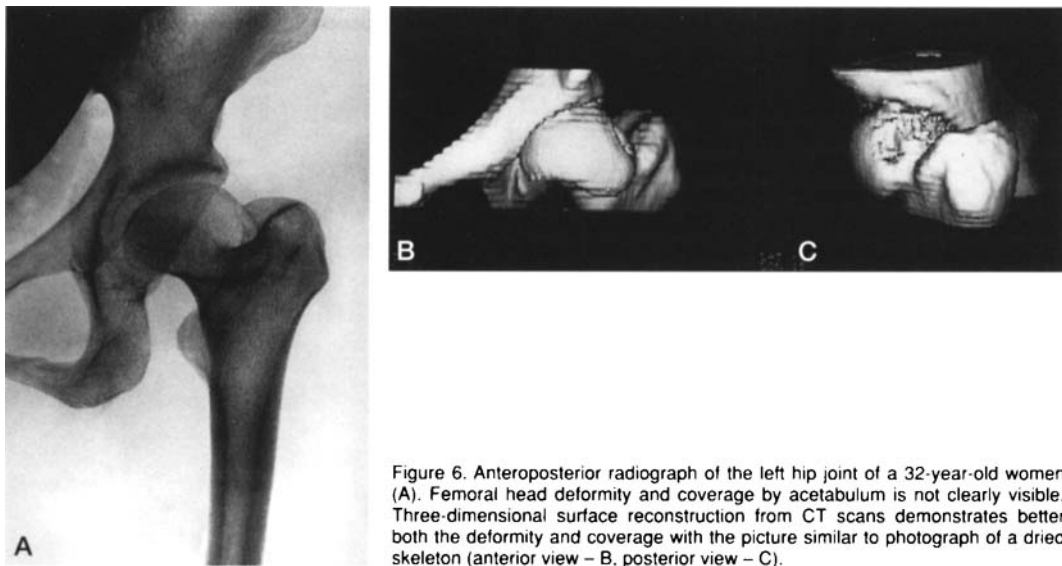
tistical significance. Epiphyseal index was below the normal values in all types and the differences were statistically significant (p 0.05). As indicated by ATD, there was an overgrowth of the greater trochanter in hips with types IV and V with true coxa vara in these types. Also in type III more than 1/3 of hips showed ATD values below the normal. Epiphysis-shaft angle of Alsberg showed medial tilt of the SGP in types IV and V and lateral tilt in type III changes.

The acetabulum was shallow in types IV and V as described by acetabular index but the values improved in all types as compared to results at 4–6 years. At this age also values of acetabular angle differed from normal in all types demonstrating poor development of the acetabulum. Head coverage described by acetabulum-head index deteriorated during growth and was below the average normal head coverage (90 according to Heyman and Herndon 1950) in all types.

The average Wiberg CE angle was within normal values during growth and at final evaluation in all types except type V. The lateral displacement angle improved during growth and was on average within the normal values in all types. Coxa magna was found in most of the hips as measured by head-shaft index with highest values in type V with significant irregularity in this type. In type V, all analyzed parameters indicated poor development of the hip.

Comparison of values of angles and indexes measured in hips with type I changes of AVN in this study with those in hips treated due to DDH without complication (from the study of Wierusz-Kozłowska 1995) showed statistically significant differences in acetabular index, acetabulum-head index, lateral displacement angle, head sphericity, and head-shaft index, with inferior values in hips with AVN (p 0.05).

CT examinations with three-dimensional surface reconstruction demonstrated more clearly the defor-



mity of the proximal femur when comparing to anteroposterior radiography. Also the portion of the femoral head not covered by the acetabular roof was better visible (Figure 6).

Clinical and radiographic end-results

On clinical evaluation 77 hips were rated as excellent or good, being pain free or with only occasional mild pain after long distance walking, with a good range of hip motion and negative Trendelenburg sign. 21 hips were rated as fair or poor with moderate or severe pain, limp, positive Trendelenburg and Duchenne sign, and leg length discrepancy.

On radiographic evaluation 50 hips were rated as excellent or good, and 48 hips as fair or poor (Table 11) (examples in the Appendix).

In 29 hips excellent or good clinical findings at final review contrasted with fair or poor radiographic

score in these patients. In no case did fair or poor clinical end-result coexist with excellent radiographic one, and only in two cases with good (Table 12).

The radiographic parameters most often responsible for poor radiographic score were ATD, acetabular index, acetabulum-head index, Wiberg CE angle, head-shaft index, and head sphericity. The low values of radiographic parameters most often found in cases with poor clinical score were acetabular index, acetabulum-head index, and Wiberg CE angle (Table 13).

There was little correlation between most of the radiographic and clinical findings. Reduction or reversal of articulo-trochanteric distance, however, showed positive Trendelenburg sign in half of the cases. Loss of sphericity (irregular heads in Mose classification) was associated with some reduction of hip motion in all cases, mostly flexion, abduction and rotation.

In the group reexamined after completion of growth with the time interval of 10 years no differ-

Table 11. Clinical and radiographic end-results in 68 patients (98 hips) with a sequelae of avascular necrosis in relation to the severity of necrosis

Type of AVN	Excellent		Good		Fair		Poor	
	C	R	C	R	C	R	C	R
I	37	13	3	17	2	8	1	5
II	9	2	1	5	0	3	0	0
III	12	3	6	6	7	10	1	7
IV	2	1	1	1	1	1	2	3
V	5	0	1	2	4	3	3	8

C clinical; R radiographic.

Table 12. Clinical versus radiographic end-results in 68 patients (98 hips) with a sequelae of avascular necrosis

Radiographic end-result	Clinical end-result				
	Excellent	Good	Fair	Poor	Total
Excellent	19	0	0	0	19
Good	26	3	1	1	31
Fair	14	5	6	0	25
Poor	6	4	7	6	23
Total	65	12	14	7	98

Table 13. Values of parameters in hips with a sequelae of avascular necrosis with poor clinical results and in hips with poor radiographic results. Mean SD

	Clinical		Radiographic	
Neck-shaft angle	135	9.7	134	15
True neck-shaft angle	128	9	124	11
True angle of anteversion	36	8.4	42	14
Head-neck index	157	21	141	43
ATD	14	12	10	20
Acetabular index	28	3.6	26	6.6
Acetabulum-head index	68	8.7	71	11
Wiberg CE angle	13	8	15	8.6
Lateral displacement angle	24	6.4	23	6.1
Head sphericity (Mose)	2.6	2.1	4	2
Head-shaft index	204	31	220	24

Table 14. Comparison of clinical and radiographic scores in 16 patients (27 hips) with a sequelae of avascular necrosis, reexamined after completion of growth with the time interval of 10 years

Type of AVN	No difference		Deterioration		Improvement	
	C	R	C	R	C	R
I	8	9	1	1	1	
II	2	2		1	1	
III	4	7	4			1
IV		1	3	2		
V			2	2	1	1

C clinical; R radiographic.

ence in clinical result was found in 14 hips, whereas in 10 hips there was a deterioration of the score, by one point in 5 hips and by two or more points in 5 hips. The reason for deterioration was pain in 8, limp in 5, and decreased motion in 3 hips (Figure 7). 3 hips improved by one point during the 10 year period (Table 14). In the same group the radiographic score showed no difference in 19 hips, deterioration by one point in 6 hips, and improvement by one point in 2 hips. The reason for deterioration was diminished value of the Wiberg CE angle, lateral displacement angle and cystic-sclerotic changes of the acetabular roof.

The ratio of mild and moderate to severe cases in hips treated by closed reduction was 1.0 comparing to ratio 1.2 in hips where no closed reduction was performed. In hips treated by closed reduction 9 were rated excellent or good and 3 fair or poor in clinical score. In radiographic score 5 were rated excellent or good and 7 fair or poor.

Radiographic signs of prearthrosis were found in

32 hips. The most common sign was double bottom in 23 hips, marginal rim in 9 hips, and osteophytes around the fovea capitis in 4 hips. In 9 hips the joint space was narrowed. More than one sign was noted in 12 hips. Clinical symptoms in these hips were pain during or after walking, limitation of movement, and limp due to pain. The radiographic signs of prearthrosis were noted in 6 hips in type I of AVN, 2 in type II, 13 in type III, 3 in type IV, and 8 in type V. Clinical symptoms were noted in 3 hips in type I, 2 in type II, 8 in type III, 3 in type IV, and 7 in type V.

The highest average scores in clinical and radiographic examinations were in types I and II and lowest in types IV and V (Table 10).

The course of remodeling of the hip during growth was different than expected (predicted from prognostic classification used) in 19 hips. Most often the difference was found in cases with metaphyseal involvement; in 10 hips in type III, 3 in type IV, and 3 in type V (Figure 8).

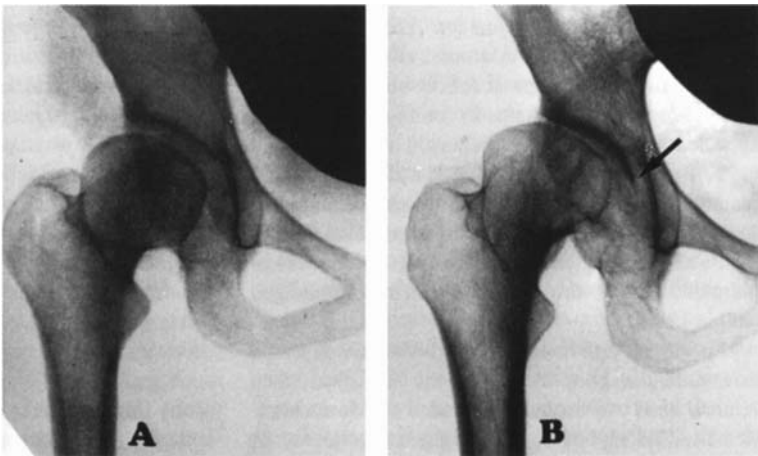


Figure 7. Example of deterioration of radiographic presentation of the hip, reexamined after completion of growth with the time interval of 10 years: at the age of 24 years (A); at the age of 34 years (B) prearthrotic "double bottom sign" (arrow) and decreased joint space.

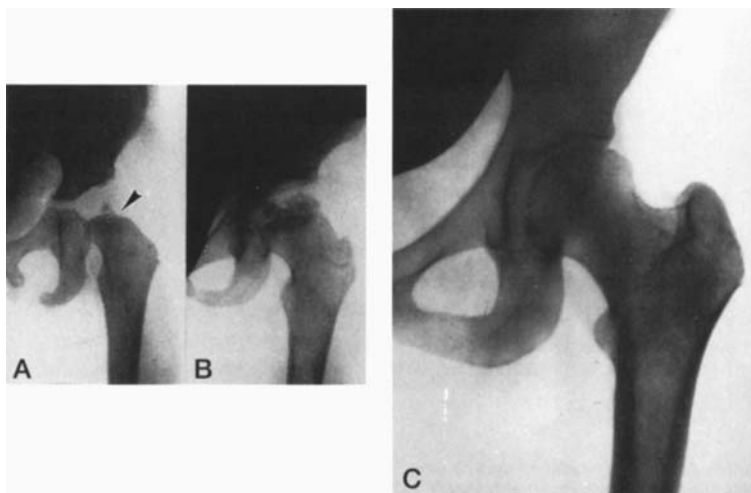


Figure 8. Anteroposterior radiograph of the left hip of a 18-month-old girl shows AVN of the epiphysis with lateral epiphyseal notching and metaphyseal involvement (arrow head) (A). At the age of 4 years 11 months epiphysis is fragmented with no clear metaphyseal involvement (B). Anteroposterior radiography of the left hip at final follow-up at the age 21 years shows development different than expected from the prognostic classification (C).

Discussion

Clinical status at final examination

The clinical disability of hips with a sequel of AVN is in most cases due to an early arthrosis (Massie 1952, Cooperman et al. 1980, Kalamchi and MacEwen 1980, Robinson and Shannon 1989). Deformed femoral head and persistent acetabular dysplasia are significant causative factors (Cooperman et al. 1980, Kalamchi and MacEwen 1980). In many hips followed in this study one sign or symptom of disability was noted but in more severe cases with metaphyseal involvement coexistence of several signs and symptoms was found.

Pain gradually becomes the major complaint in sequel of AVN and was found in more than one-third of hips evaluated, being responsible for negative estimation of health status by 9 patients. Generally pain prevailed in hips with severe changes due to AVN but was also present in half of the hips following type II changes, thus proving that the joint was more affected as compared to type I changes. Fisher and Cary (1978) reported on pain in around one-fifth of their patients but the mean age at follow-up was 15 years so deterioration of hip status in coming years may be expected. In all cases of Kalamchi and MacEwen (1980), graded as poor pain was a dominant complaint and these patients frequently used analgesics. In some patients observed by Cooperman et al. (1980) pain was disabling enough to produce functional impairment prohibiting jobs requiring prolonged weight-bearing.

Hip motion although decreased was still assessed as excellent or good in most of the hips even when femoral head was irregular as graded by Mose classification. The explanation may be that it is not the ir-

regularity itself that decreases the motion but rather the combination with articular cartilage damage due to arthrosis. With inevitable advance of arthrotic changes over time rapid decrease of range of motion in most deformed hips is likely.

In bilateral AVN cases evaluation for leg shortening caused by the disease was inadequate as both legs were affected. Probably due to this reason leg length inequality found in patients in this study was low as compared to other studies (Kalamchi and MacEwen 1980, Thomas et al. 1982) and equalization procedures were not considered. Limp when present was attributed to pain or positive Trendelenburg sign but not to leg shortening.

Radiographic parameters during growth and at final follow-up; CT-3D examination

Changes in the epiphysis alone represented by type I and II produced very little deformities after the end of growth providing there was no damage to the physis at follow-up. Although appositional growth of the femoral head was disturbed as shown by low values of epiphyseal index measured at the age of 4–6 years, there was no influence on final appearance. Similarly, the ultimate length to width ratio of the femoral head-neck complex in these two types represented by head-neck index was within the normal values. The development of acetabulum in these hips was adequate and the lateral head coverage was sufficient as demonstrated by acetabulum-head index and Wiberg CE angle. On the other hand using as a reference measurements in hips treated due to DDH without complication (Wierusz-Kozłowska 1995), significantly inferior values of parameters describing development of acetabulum, femoral head coverage, spher-

ricity, and head enlargement were observed in hips with AVN. In the series evaluated by O'Brien et al. (1986) epiphyseal changes alone were found to be of no prognostic value in the absence of physal closure. Kalamchi and MacEwen (1980) stated that these changes have little bearing on the final result, other than producing a slight loss of height and coxa magna, as seen also in my study. However, they warn that it is extremely difficult to predict with certainty the result in a patient whose hip show changes in the ossific nucleus but no visible early signs of physal involvement. I agree with their observation, that the damage to the physis may remain "dormant" for many years. As the child passes through the years of rapid growth, the physal arrest becomes obvious. Likewise, according to Fisher and Cary (1978) and Cooperman et al. (1980) fully-developed deformity seen at maturity probably cannot be predicted during the initial stages of AVN. For this reason, patients must be carefully followed until skeletal maturity.

The formation of cystic lesions in the metaphysis in the course of AVN detected on radiography, was used in this study to distinguish severe changes. There is no clear explanation in the literature for this finding. According to Katz (1973) unmineralized cartilage matrix may be responsible but the size of the cyst mostly exceeds the growth potential of the cartilage at a given time. At the same time activity in the germinal layer should be preserved, which in severe AVN is hardly to be expected due to disturbed vascularity on the epiphyseal side. Katz agrees with Brashear (1963) that metaphyseal cysts may represent resorption of the necrotic area in the metaphysis rather than unmineralized cartilage matrix.

The most active point of physal growth corresponds to the region of the junction of SGP and the femoral neck isthmus, the area called "vertex" by O'Brien et al. (1986). This area was damaged in hips of type III AVN changes, with early radiographic signs depicted by increased epiphysis-shaft angle of Alsberg. The common finding in final radiographic measurements was a short femoral neck, with normal or even increased neck shaft angle, a presentation also noticed by Kalamchi and MacEwen (1980). On the other hand low ATD values and short femoral neck in these hips caused functional coxa vara with positive Trendelenburg sign as a consequence. The combination of this signs together with leg-length discrepancy may lead to limp and fatigue after walking even short distances (Porat et al. 1994). Acetabular development on an average was slightly below normal. Interesting were observations on lateral head coverage, good as represented by acetabulum-head index and Wiberg CE angle but uncovered as seen on CT-3D reconstruc-

tion which is due to lateral tilt of the femoral head into valgus ("head in neck" position of Jansen 1925 or "subcapital coxa valga" of Jones 1977).

There is general agreement that total physal damage as the result of AVN produce the worst results (Bucholtz and Ogden 1978, Fisher and Cary 1978, Kalamchi and MacEwen 1980, Siffert 1980, Tönnis 1987, Robinson and Shannon 1989, Thomas et al. 1989, Keret and MacEwen 1991, Kruczynski 1995). Radiographic parameters after completion of growth demonstrated poor status of hips in this type in my study and signs of prearthrosis were already present in most. Femoral head deformity, short and broad femoral neck were constant findings in this group and loss of head-acetabular congruity resulted in adaptive malformation of both structures.

The term coxa magna used in the literature to describe the pathologic enlargement of the femoral head has been so far not clearly defined. The head-shaft index used in this study appears to be useful in measurement of femoral head size providing the femoral shaft is untouched by the disease itself or the way the hip was treated. Mayer (1977) used contralateral hip as reference but this is available only in unilateral cases. Normal femoral head as a reference was also used by Fisher and Cary (1978), Cooperman et al. (1980), Papavasiliou and Pigot (1983). Gamble et al. (1985) recognized coxa magna when difference was minimum 15 percent and Kallio (1988) stated that the difference should amount at least 2 millimeters. Considering the fact, that in DDH, Legg-Calve-Perthes, and some other diseases in children both hips may be affected by the disease itself or as a result of treatment, the use of the opposite hip may be misleading.

In the present study, the value of acetabulum-head index decreased with age in all groups showing inferior head coverage, contrary to observations of Szulc (1977) on DDH cases treated without complication. On the other hand the value of the lateral displacement angle improved with age in all groups, proving that head is sitting deeper in acetabulum, the phenomenon also noticed by Bernardczyk (1972) in hips without AVN. This contradiction may be explained by the presence of coxa magna in all groups, and the fact that in many hips also development of the acetabulum was inferior as represented by acetabular index.

The selection of patients for operative treatment due to late sequelae of DDH is a particularly difficult problem. Careful supervision of head-acetabular relationship is necessary so that preventive surgical measures can be taken to correct preosteoarthrotic deformity (1987). In the present study CT-3D pictures of the hips at final follow-up appeared to be helpful, demonstrating femoral head deformity better than ra-

diography. Also the coverage of the femoral head was better assessed but some error should be expected since simulation of forward tilting of the pelvis characteristic for standing upright position was not possible on my workstation. This method of assessment applying Johnson's et al. (1986) radiographic principles was introduced with CT-3D technique by Ferran et al. (1994).

I found CT-3D pictures to be indicated in strictly selected cases with extensive hip deformity as a sequel of AVN and only when surgery was discussed. The risk of increased dose of radiation should always be considered but collimation of the x-ray beam in a CT scanner allows for delivering the same amount of radiation at each level, and the doses to different sections are not additive (Peterson et al. 1981). On the other hand CT-3D allows for multiple views of the hip without increasing the radiation dose. For these reasons the amount of radiation with CT scan and 3D reconstruction seems to be reasonable if the method is rigorous and there is close surgical collaboration (Ferran et al. 1994).

Clinical and radiographic end-results

Comparison between clinical and radiographic end-results showed better clinical outcomes in most of the hips. The fact that in one-third of the hips at final examination excellent or good clinical result co-existed with fair or poor radiographic one may be explained by still too short follow-up to reveal clinical sequel of joint deformity. On the other hand in the group examined twice after completion of growth, the period of 10 years was sufficient for deterioration of clinical status in 1/3 of the hips, even by two or more points in clinical score. Considering the fact, that the patients were 36 years at the oldest the prognosis in these hips is poor what confirms remarks of Fisher and Cary (1978) that abnormalities due to severe AVN may reasonably be expected to lead to premature arthrosis and functional impairment in adult life. Interestingly enough, the radiographic picture in these hips showed in 10 years period much slower deterioration, a fact which may be interpreted by already originally lower score in these hips. On the other hand no conclusions could be drawn regarding the improvement of hip status in some hips in this group since no specific responsible factors could be found.

My analysis of poor clinical results showed clearly that parameters most often responsible were acetabular index, acetabulum-head index, and Wiberg CE angle, all describing inferior development of acetabulum and femoral head coverage, namely acetabular dysplasia. It is interesting to note that there was less correlation between the poor clinical result and pa-

rameters describing the development of proximal femur, being more clear when assessing poor radiographic results. In cases reported by Cooperman et al. (1980) the deformities produced in AVN that correlated with the development of arthrosis were loss of sphericity of the femoral head, persistent lateral and proximal subluxation, irregularity of the medial femoral head, and acetabular dysplasia. Kalamchi and MacEwen (1980) point, that partial uncovering of the femoral head in the acetabulum will leave a smaller area of contact between the two articular surfaces thus increasing the pressure per unit area which will lead to arthrosis. This concept is on par with the biomechanical principles of the stress on the hip joint presented by Kummer (1969).

The ratio of mild and moderate to severe cases was slightly lower in cases where closed reduction with anesthesia was performed, comparing to the group with abduction treatment alone. This may indicate that closed reduction was more aggressive. On the other hand all these hips had previous treatment with a Frejka pillow and reduction in most cases was easily obtained but unstable thus requiring more rigid positioning in the acetabulum. Tönnis (1982, 1987) in his retrospective analysis found cases of AVN after manual reduction being the most severe but in his group also hips reduced with no preparative measures were included. In my study the child was always adapted to the position of immobilization during initial preparative treatment and I blame this treatment together with the manipulation or subsequent immobilization for increase in severity.

Only in some hips with radiographic signs of prearthrosis a poor prognosis should be expected. Bruszewski et al. (1973) stated that radiographic prearthrotic changes progress to arthrosis in particularly unfavorable biomechanic conditions, found in 10 out of 80 hips in the material they analyzed with a long-term follow-up. In my study prearthrotic changes were found on radiograms in around one-third of hips. The most common symptom in these hips was pain during or after walking and some limitation of motion, mostly flexion and rotation. Only in few hips there were more than one radiographic sign of prearthrosis coexisting with more than one clinical symptom and in my opinion in these hips a threat of arthrosis is most likely.

The sequence of condensation-fragmentation-repair typical for Legg-Calve-Perthes disease is occasionally observed in AVN in DDH (Kohler and Seringe 1981) and I found it only in around one-tenth of the hips with type II pattern. In contrast to this findings Fisher and Cary (1978) found fragmentation to be the most frequent radiographic observation.

Changes in the metaphysis and in the SGP are the main cause of late abnormality and were responsible for deterioration of radiographic results in my study. O'Brien et al. (1986) identified metaphyseal changes only in patients who subsequently manifested abnormal patterns of growth of the proximal physis, but I could find also excellent or good radiographic results in hips with previous metaphyseal involvement.

This study based on radiographic observations of the growth of the proximal femur proves that prognosis of development based on radiography is not fully reliable. Thomas et al. (1989) even suggest, that the current classifications of AVN have no predictive value. Those presented by Bucholtz and Ogden (1978) and Kalamchi and MacEwen (1980) have increased our understanding of the patterns of vascular impairment, but approximately one-quarter of patients from the study of Thomas et al. (1989) and one-fifth in my study had a course of remodeling different than predicted. This fact may explain the scatter in the data obtained from radiographic measurements and was probably the reason for lesser value of statistic analysis of differences in mean values of parameters between groups. The course of remodeling different than expected was most frequent in type III cases with lateral involvement of the metaphysis. Also Kalamchi and MacEwen (1980) and Keret and MacEwen (1991) found this group of AVN the most unpredictable in outcome, and as in my study, this form of metaphyseal involvement was the most common one. The unusual, misleading presentation of different radiographic forms of AVN which vary from those presented in the literature should also be considered (Robert and Seringe 1981). Thus, the search should be continued for the diagnostic modality being able to specify more precisely disturbances in the hip before the deformity occurs. This is the aim of the third part of this quest.

Conclusions

Development of hips that have undergone even mild avascular necrosis following nonoperative reduction of developmental dislocation of the hip is different from that without avascular necrosis. The difference lies in femoral head enlargement and sphericity, acetabular development, and femoral head coverage with inferior values in hips with avascular necrosis.

The classification of the vascular changes used in this study permits for prognosis of the final deformity in most of the hips, although it should be interpreted with caution. In one-fifth of cases it appeared misleading.

Acetabular dysplasia was a major contributing factor to the development of early arthrosis responsible for poor clinical results. Deformities of the proximal femur together with acetabular dysplasia were responsible for poor radiographic results.

Pain in the hip appeared to be the most significant symptom in early arthrosis of the hip, whereas decrease of motion was less pronounced.

Changes in the lateral part of the metaphysis during avascular necrosis complicating nonoperative treatment of developmental hip dislocation appeared to be the most frequent. In this type also the course of remodeling different than expected from prognostic classification was most common.

Long-term follow-up is necessary in cases of avascular necrosis complicating nonoperative treatment of developmental hip dislocation since deformity of the hip may not be completely evident until the child passes through the years of rapid growth.

Growth zones of the proximal femur in MRI;

radiography versus MRI in detection of subcapital growth plate disorders due to avascular necrosis in developmental dislocation of the hip

Introduction

Injury to the subcapital growth plate of the proximal femur may result in leg-length inequality and deformity. Unfortunately, damage to the growth cartilage is not directly detectable with radiography. Physeal abnormality on plain films may be diagnosed in most cases only when growth deformity has already developed. Therefore, it is important to explore ways of detecting the early changes that precede growth arrest or to evaluate the size and location of the physeal damage once present.

Several methods for assessment of the growth plate has already been described. Carlson and Wenger (1984) presented a technique for transferring the information of a physeal bar obtained from the polytomograms onto a useful cross-sectional map. When a map is constructed, one can accurately assess the size and location of the bony bridge.

On bone scintigraphy the physis in growing children is normally seen as a linear band of increased uptake (Murray 1980). Closure of the growth plate is seen as an area of absent activity. The technique of apex view of growth centers developed by Howman-Giles et al. (1985) provides a functional map of the plate such that abnormal growth areas are displayed and their site, size, and position are obtained. Scintigraphy is positive early in the course of the disease, even when the radiographs are normal. A disadvantage of bone scintigraphy with technetium phosphates, the most commonly used radiopharmaceutical, is the lack of specificity.

In the technique developed by Porat et al. (1987) the growth plate is assessed on CT with sections through the region of the epiphyseal plate. CT depict the developing area of sclerosis that represent the metaphyseal-epiphyseal bony bridge and the percentage of closure of the epiphyseal plate is calculated on the console.

MR imaging, which can demonstrate early vascular and cartilaginous changes, is not merely an alternate method for imaging physeal growth arrest but is also the most satisfactory modality for diagnosing possible physeal abnormalities prior to osseous abnormalities (Lawson et al. 1994). MRI scans can differentiate soft tissue structures that cannot be differentiated by CT (Harcke 1990). Axial, coronal and sagittal planar sections can be obtained, depending on the informa-

tion desired. According to Coleman (1994) indications for MRI studies remain poorly defined but add greatly to our "backup" diagnostic armamentaria when properly used.

Aims of the study

The aims of the study were:

- to identify the MRI method for depiction of the growth zones of the normal proximal femur;
- to describe the appearance of the subcapital growth plate of the proximal femur in MRI during growth;
- to evaluate the diagnostic capacity of MRI as compared to conventional radiography to identify changes in the proximal femoral epiphysis and metaphysis and assess subcapital growth plate disorders due to avascular necrosis in developmental dislocation of the hip;
- to find MRI sequences adequate for imaging the subcapital growth plate disorders;
- to develop a method of mapping the damaged subcapital growth plate.

Patients and methods

The study consisted of two parts:

Part A. Designed to present the appearance of normal growth cartilages of the proximal femur in MRI. The subjects of the study were children and adolescents in different ages with no disease of the hip joint. There were 36 children with 58 hips investigated at the Center for Diagnostic Imaging, Karol Marcinowski University of Medical Sciences in Poznan for diseases around the hip, but not involving the hip joint itself. Subjects analyzed were divided into 3 groups regarding the physiologic development of the proximal femur (Batory 1982) and the typical findings in MRI (Table 15).

Part B. Designed to present the subcapital growth plate disorders due to AVN on radiography and in MRI. Changes in the epiphysis and metaphysis were also observed. There were patients treated nonoperatively (4 patients with 5 hips) or operatively (20 patients with 28 hips) due to DDH at the Department of Orthopedics and Department of Pediatric Orthope-

Table 15. Distribution of 58 normal hips in 3 age groups

Group	Age	No. of hips
I	< 5	10
II	5–12	34
III	>12	14

dics, Karol Marcinkowski University of Medical Sciences in Poznan, and complicated by AVN. All together there were 24 patients (33 hips) treated in the years 1985–1991. The average age of the patients at the time of evaluation was 10 (4–14) years (Table 16).

Conventional radiography

In children analyzed in part B conventional radiographs of the hips were obtained in AP views before the MRI examination.

Magnetic resonance imaging

The MRI examinations were performed with 1.0 T unit (Magnetom Impact, Siemens). The hip joints were examined with the use of partial-volume body Helmholtz coil as a receiver coil. The standard pulse sequence used was coronal, Spin-Echo (SE) (TR, TE/500, 15), matrix 256x256 with 4 mm section thickness.

In some patients additional sequences were used: FLASH 3D FAT SAT (TR, TE, FA/48, 7, 40); SE T1 FAT SAT (TR, TE/590, 15); FLASH 2D (TR, TE, FA/500, 18, 10); FISP 3D (TR, TE, FA/30, 10, 40); SE T2 (TR, TE1, TE2, FA/1850, 20, 90, 90); FISP 3D and FLASH 3D FAT SAT allowed for a section thickness of 1–1.5 mm.

Except for the coronal plane some of the examinations were also performed in sagittal and oblique planes. In case of physeal damage to provide more accurate data about its size and location a grid or scale were used on images on each of the planes. To outline the cross-sectional shape of the subcapital growth plate an examination in the plane of the plate was also performed. On each section of coronal and sagittal planes the extent of physeal damage was determined with the use of the grid or scale.

Because of the intrinsic motion sensitivity of MRI, immobilization was essential. This was easily obtained in the older cooperative children; in the younger children, some form of sedation usually with per rectum administered chloral hydrate was required.

The MR images in Part A were reviewed to establish the characteristics of the growth zones in the proximal femur with special interest in the presenta-

Table 16. Distribution of 33 pathologic hips in 3 age groups

Group	Age	No. of hips
I	< 5	1
II	5–12	30
III	>12	2

tion of the subcapital growth plate with respect to the visibility, the width, and continuity on the serial images. Variations in the appearance of the subcapital growth plate according to the patient's age were also noted.

The radiographs, and MR images obtained with each pulse sequence from an individual patient from group B were examined and classified blindly according to the visibility of the subcapital growth plate and the visibility of the damage to the growth plate on a 5 point scale.

Visibility of the subcapital growth plate:

- 0 – not visible;
- 1 – fair or hypointense line on T1-weighted images;
- 2 – good except localized disturbance;
- 3 – good;
- 4 – picture not clear.

Visibility of the subcapital growth plate damage:

- 0 – not visible;
- 1 – fairly visible disturbance in growth plate presentation;
- 2 – well detectable disturbance in growth plate presentation (good visibility);
- 3 – well delineated bony bridge (good visibility);
- 4 – picture not clear.

The presentation of the shape of the subcapital growth plate on coronal and sagittal planes was described as straight linear, arched or irregular.

Avascular necrosis of the epiphysis was diagnosed on radiography according to the criteria depicted in the first part of the study. Bone marrow changes indicative of avascular necrosis were that of greatly decreased signal intensity within the marrow area (Vogler and Murphy 1988).

All MR investigations were supervised by one engineer according to a strict formula adopted before the start of the research. Radiograms and MR images were assessed separately and then the findings were compared.

Results

Part A

Appearance of the normal proximal femur in MRI. The ossification center of the femoral head and that of

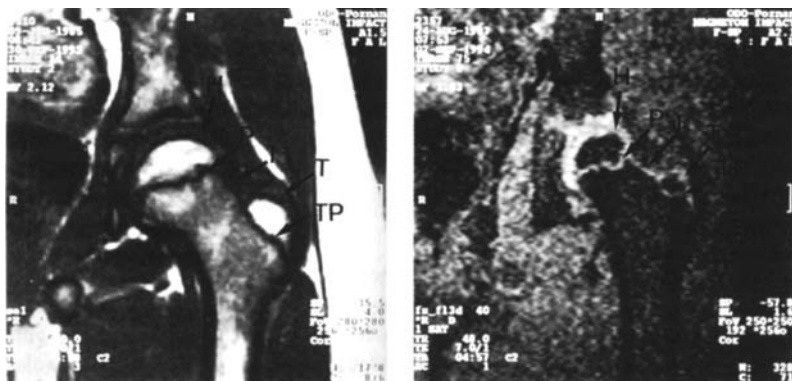


Figure 9. Normal hip joint of a 8-year-old girl is demonstrated on T1-weighted image (500, 15) (left) and of a 7-year-old girl on gradient-echo image (48, 7, 40) (right).

The following anatomical structures are identified: growth cartilage of the femoral head (H), subcapital growth plate (P), femoral neck isthmus (I), growth cartilage of the greater trochanter (T), growth plate of the greater trochanter (TP).

the greater trochanter were visible on T1 weighted images as areas of high signal intensity. On gradient echo images these structures were hypointense. The metaphysis of the growing subjects on T1 weighted images was hypointense when comparing to ossification centers and also hypointense on gradient echo images. After the completion of growth the epiphysis and metaphysis became of equal signal intensity.

All 5 growth zones of the proximal femur were visible on MR images. The appearance of the growth cartilage varied with the pulse sequence used (Figure 9). The signal intensity of the cartilage, however, was consistent in all patients, regardless of age, when imaged with a given sequence.

The growth cartilage of the femoral head and the greater trochanter. On T1-weighted images the hemispherical growth cartilage of the femoral head and greater trochanter consisted of two distinguishable layers, i.e., layer of intermediate signal intensity

placed on hypointense line regarded as the zone of provisional calcification. Differentiation of the specific histologic zones of the growth cartilage was impossible. In some hips in Group I a hypointense zone separating the articular cartilage and the growth cartilage within the hemispherical cartilage covering the bony center of the femoral head was visible (Figure 10).

On sequences using fat saturation the hemispherical cartilages were of high signal intensity as were all other cartilaginous structures.

During the process of growth the cartilage surrounding the bony centers decreased in thickness.

Subcapital growth plate. Group I. In the subgroup of younger children in this group the cartilage between the ossific nucleus and the metaphysis on T1-weighted images was visible as an hourglass configuration band of intermediate signal intensity between the ossific nucleus and the metaphysis (Figure 11). On se-

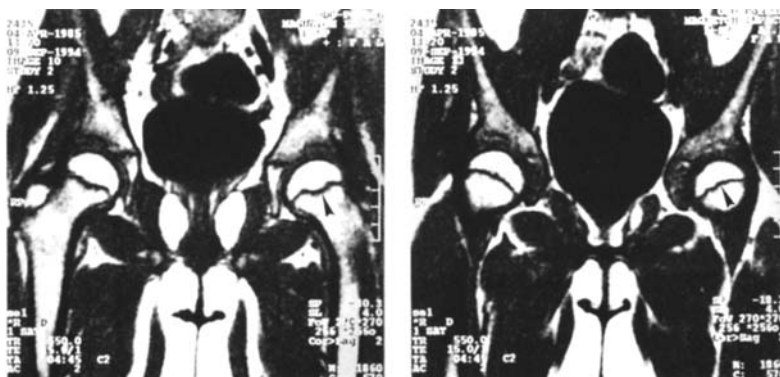


Figure 10. Normal hip joint anatomy of a 4-year-old boy (Group I). Coronal T1-weighted image (500, 15) shows hypointense line separating the articular cartilage and growth cartilage of the femoral head (arrows). Subcapital growth plate (P) is visible as a band of intermediate signal intensity (arrow).



Figure 11. Normal hip joint of a 3-year-old boy (Group I). Coronal T1-weighted image (540, 15) shows: thick cartilage covering the ossific nucleus (H), subcapital growth plate of an hourglass configuration (P), femoral neck isthmus (I).

Figure 12. Normal hip joint anatomy of a 9-year-old girl (Group II). Coronal T1-weighted image (550, 15) displays the subcapital growth plate as an irregular line of low signal intensity in the posterior part of the femoral head (arrow head) (A). In the anterior part the course of the growth plate is more regular (arrow head) (B).



quences using fat saturation this area was of high signal intensity. When assessing the older children in this group the growth plate was visible as a line with signal intensity depending on sequence used and similar to that in the younger subgroup (Figure 10).

Group II. The growth plate presented itself on MR images in several shapes. On coronal views prevalent course was irregular in the posterior and arched in the anterior part of the femoral head (13 hips) (Figure 12) but single arched was also a common finding (6 hips). Also straight line course and irregular were observed. On sagittal plane the course was usually single arched.

On T1-weighted images in this group the typical finding in the area of the growth plate was a band of low signal intensity (27 hips) (Figure 12). Only in a few instances a band of intermediate signal intensity between the hypointense epiphyseal bone plate and the hypointense zone of provisional calcification was found (7 hips). This presentation was more often in sagittal plane.

On sequences using fat saturation the growth plate was always well visible as a band of high signal intensity lying between the hypointense bone margin of the epiphysis and metaphysis (Figure 13). Gradual thinning of the growth plate with age was observed on these images.

Group III. In this group further thinning of the growth plate was observed and in the older subjects in this group the growth plate was no longer visible.

On T1-weighted images the area of the growth plate was visible as a band of low signal intensity (7 hips). In 2 hips a three-line appearance of the plate was found. In 5 hips of the oldest subjects in this group there was only a thin hypointense line better visible in the central part than on the periphery of the plate (Figure 14).

When sequences using fat saturation were done, the band was still hyperintense in 11 hips, whereas in 3 the plate was invisible.



Figure 13. Normal hip joint anatomy of a 7-year-old girl. On gradient-echo image (48, 7, 40) the subcapital growth plate is visible as a band of high signal intensity (P) lying between the hypointense bone of the epiphysis (E) and metaphysis (M).



Figure 14. Normal hip joint anatomy of a 17-year-old boy. Coronal T1-weighted image (500, 15) depicts a thin hypointense line in the area of the subcapital growth plate (P) better visible in the central part than on the periphery.

Table 17. Visibility of the subcapital growth plate of the proximal femur in 24 patients (33 hip joints)

	n	Visibility of the growth plate			
		Good	Fair	Not visible	Picture not clear
Radiography	33	6	21	6	0
MR					
T1-weighted image	33	2	28	3	0
T2-weighted image	3	2	0	0	1
T1 FAT SAT	20	9	8	2	1
FISP 3D	7	1	2	0	4
FLASH 2D	17	2	7	0	8
FLASH 3D FAT SAT	24	14	0	1	9

Trochanteric growth plate. The trochanteric growth plate behaved similar to subcapital growth plate as regards age and MRI sequences used.

Femoral neck isthmus. The growth cartilage here was visible on T1-weighted images as a zone of intermediate signal intensity in continuity with the hemispherical cartilage covering the bony center of the femoral head and greater trochanter (Figure 11). It was placed on the line of low signal intensity with the picture

similar to that of growth cartilage of the femoral head and greater trochanter. On sequences using fat saturation the cartilage was hyperintense.

Part B

Visibility of the subcapital growth plate on radiography and MRI. The appearance of the growth plate depended on the age of the subject, the pulse sequence used in MRI, and the extent of damage to the proximal femur (Table 17). The influence of the first two factors was similar to that observed in the normal subjects.

The subcapital growth plate damage on radiography and MRI. Damage to the growth plate on radiography was visible as an erased area in the line of the growth plate (14 hips) or there was no growth plate line at all (8 hips). In 4 hips there was already a caput valgum presentation of the head-neck relationship and in further 3 hips a slight valgus tilt was visible (Figure 15 A).

The extent of growth plate damage visible on MR images ranged from focal disturbance of signal intensity (23 hips) to the picture of premature complete closure of the growth plate (4 hips) (Figure 15 B, C).



Figure 15. Anteroposterior radiography of the hip joints of 8-year old girl (A). Subcapital growth plate line is invisible in the left hip. In the right hip caput valgum presentation of the head-neck relationship is shown. Coronal T1-weighted image (500/15) shows focal disturbance of signal intensity in the right subcapital growth plate (arrow head) and more advanced lesion in the left (arrow head) (B). T1-weighted FAT SAT image (590/15) confirms the findings in the right hip (arrow head). In the left hip the subcapital growth plate is invisible (arrow head) (C).

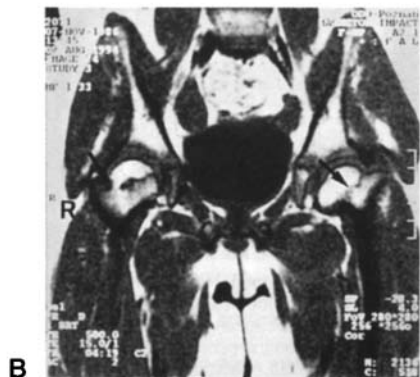


Table 18. Visibility of the damage to the subcapital growth plate of the proximal femur in 24 patients (33 hip joints)

		Visibility of the damage			
		Good	Fair	Not visible	Picture not clear
	n				
Radiography	33	22	0	11	0
MR					
T1-weighted image	33	24	0	9	0
T2-weighted image	3	2	0	0	1
T1 FAT SAT	20	10	0	3	7
FISP 3D	7	2	1	0	4
FLASH 2D	17	2	2	1	12
FLASH 3D FAT SAT	24	10	2	2	10

The appearance of the physeal damage varied with the pulse sequences used (Table 18). In 18 hips on T1-weighted images the union between the epiphysis and metaphysis was of high signal intensity suggesting yellow marrow within a bridge, whereas in 2 hips the dark band was present suggesting fibrous bridge or the one of cortical bone characteristics (Figure 16 A). On images using fat saturation it was impossible to distinguish between a fibrous and bony bridge and the damage to the physis was visible as a hypointense connection between the epiphysis and metaphysis of different size crossing the hyperintense plate (Figure 16 B). In 3 hips the damage not visible on radiography and T1-weighted images was visible on images using fat saturation (Figure 17).

Evaluation of the size and location of the growth plate damage. On the basis of 24 cases the following method of evaluation of the size and location of growth plate damage on T1-weighted MR images was developed (Figure 18, 19):

- coronal and sagittal serial cuts of the femoral head are obtained with a grid or scale;

- an outline of the cross-sectional shape of the physis is obtained from an oblique cut passing through its most distal part (with a grid or scale);
- outline of the cross-sectional shape of the physis is superimposed on a graph paper (to ease the procedure a quadrangle containing an outline of the cross-sectional shape of the physis is formed);
- the extent of growth plate damage is analyzed on each of the coronal and sagittal cuts and plotted each time on the outline of the physis (or on a quadrangle) drawn on the graph paper;
- after completion of calculations made on each cut the cross-sectional drawing of the growth plate damage is done.

The method presented above proved to be useful also on gradient echo images.

Radiography versus MRI in assessment of the growth plate and its damage. Regardless the assessment result of the visibility of the growth plate on radiography and MR images, the comparison of the data obtained showed more valuable information being provided by MRI in 24 hips, no difference in 8, and better by radiography in 1 hip.

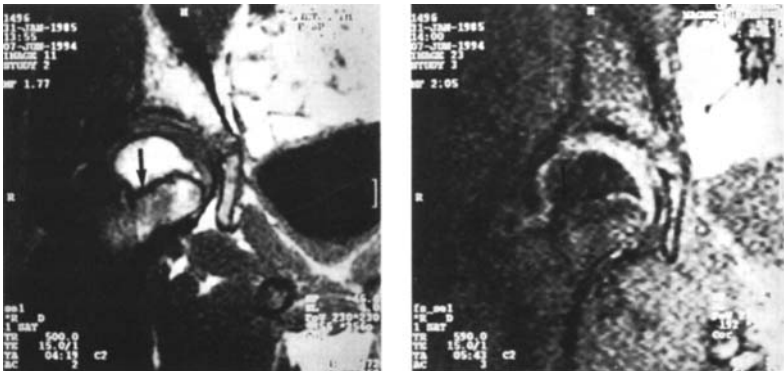
As regards the visibility of the damage to the growth plate more valuable information was provided by MR images in 20 hips, no difference in 12 hips, and better by radiography in 1 hip.

In 4 hips with complete closure on radiography MRI was able to reveal a line on certain slices, thus proving that the closure of the growth plate was not complete.

Necrosis of the epiphysis on radiography and in MRI. Necrosis of the epiphysis was visible on radiography in 5 hips.

In MRI necrosis was observed in 9 hips involving a part (8 hips) or all of the femoral head (1 hip) (Figure 20). In 4 hips the changes were not detectable on radiography. The appearance of the necrotic area varied

Figure 16. Coronal T1-weighted image (500, 15) of a 9-year-old boy shows hypointense area in the lateral part of the subcapital growth plate (arrow) (A); on T1-weighted FAT SAT image (590, 15) the subcapital growth plate in the lateral part is not visible (arrow) (B).



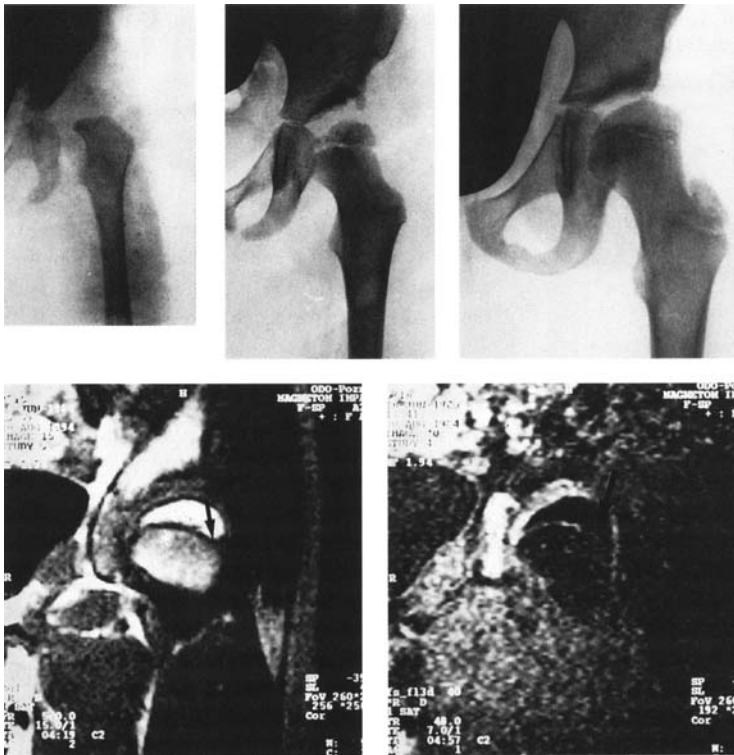


Figure 17. Anteroposterior radiography of the left hip joint of a 5-month-old girl shows developmental dislocation (A). On radiographs at the age of 3 years and 5 months (B), and 9 years (C) the epiphysis tilts laterally (shelf plasty at the age of 2 years). In MR coronal T1-weighted image (500, 15) no damage to the subcapital growth plate is visible (D). On coronal FLASH 3D FAT SAT (48, 7, 40) the picture of subcapital growth plate is interrupted, and normal hyperintensity of lateral part of the growth plate is lost (arrow) (E).

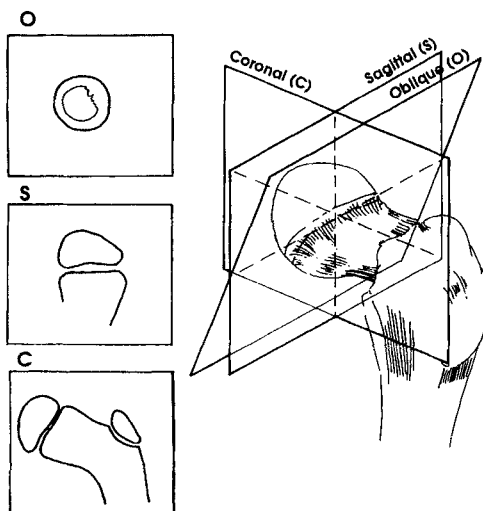


Figure 18. Three-dimensional model of assessment of subcapital growth plate shows planes of MR serial images [coronal (C), sagittal (S), and oblique in a plane of the growth plate (O)].

with the pulse sequence used. On T1-weighted images the changes were hypointense whereas on sequences using fat saturation the picture was less homogeneous, mostly mixture of high and low signal intensity.

Metaphyseal changes. Changes in the metaphysis were visible on radiography in 5 hips as a cystic lesion of different size. In MRI the metaphyseal changes were found in 7 hips. On T1-weighted images these changes were of intermediate signal intensity, whereas on sequences using fat saturation the changes were of high signal intensity similar in appearance to the cartilage. In 2 hips changes in the metaphysis were not visible on radiography while in MRI were found both on T1-weighted images and on gradient echo images (Figure 21).

Discussion

Appearance of the proximal femur in MRI

The diagnosis of avascular necrosis in developmental dislocation of the hip has so far been based mainly on conventional radiographs. The present study is the first in which findings in radiography and MRI have been compared.

The development of the normal and pathologic proximal femoral end has been intensively studied on radiography for many years (Taussig et al. 1976, Grybos 1983, Tönnis 1984). Siffert and Feldman (1980) and Siffert (1981) gave excellent description

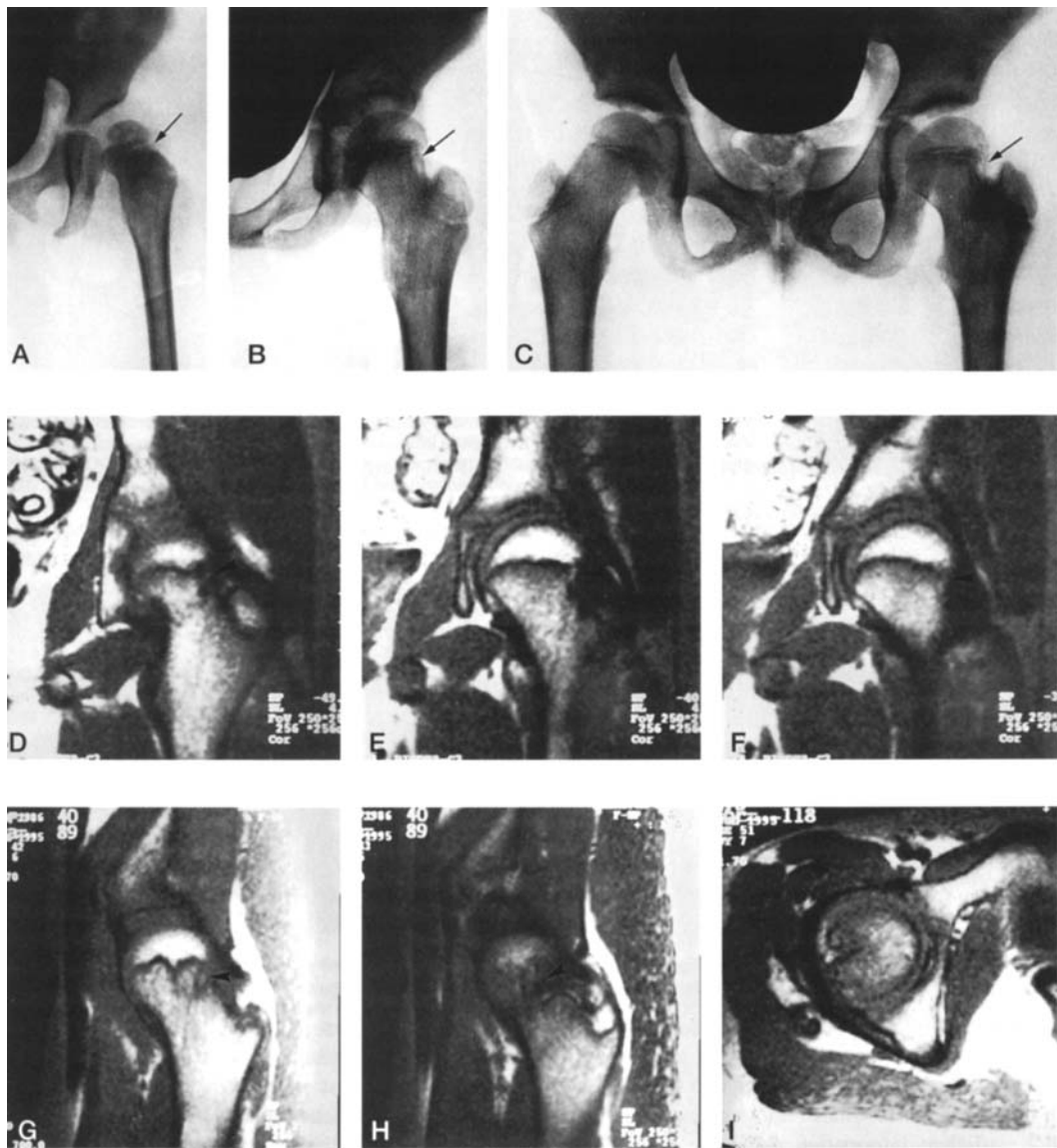
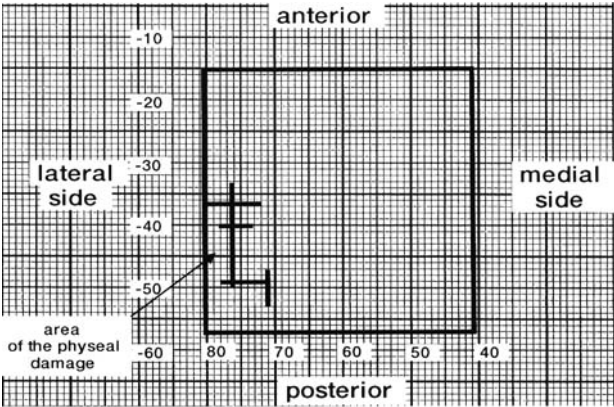


Figure 19. Anteroposterior radiography of the left hip joint of a 2-year-old girl shows lateral notching of the metaphysis (A). At 8 and 9 years of age translucent area in the lateral metaphysis is visible with lateral tilt of the epiphysis (B, C). Selected images from serial coronal (D–F) and sagittal (G–H) T1-weighted images (500, 15) of the hip joint show physeal damage (arrow heads). The extent of damage on each cut is calculated with a scale. Oblique cut in a plane of the subcapital growth plate gives an outline of the plate (I). Physeal damage from each cut is plotted as a line on the outline of the physis drawn on the graph paper. Cross sectional drawing of the physeal damage finished on a graph paper (J).



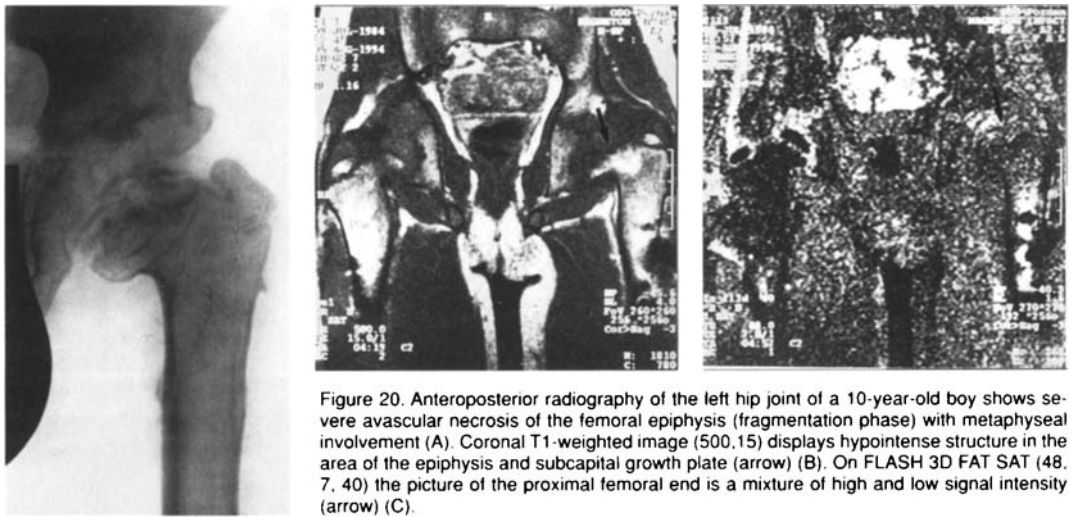


Figure 20. Anteroposterior radiography of the left hip joint of a 10-year-old boy shows severe avascular necrosis of the femoral epiphysis (fragmentation phase) with metaphyseal involvement (A). Coronal T1-weighted image (500, 15) displays hypointense structure in the area of the epiphysis and subcapital growth plate (arrow) (B). On FLASH 3D FAT SAT (48, 7, 40) the picture of the proximal femoral end is a mixture of high and low signal intensity (arrow) (C).

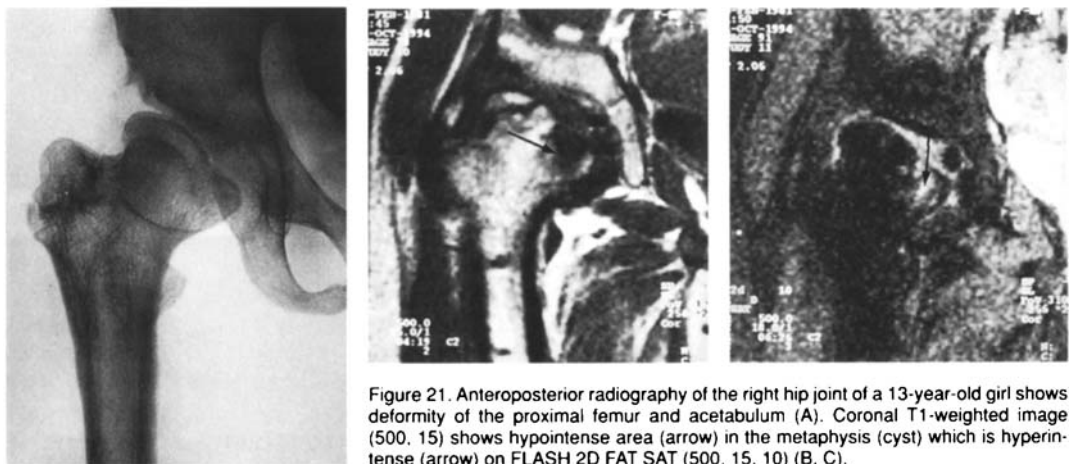


Figure 21. Anteroposterior radiography of the right hip joint of a 13-year-old girl shows deformity of the proximal femur and acetabulum (A). Coronal T1-weighted image (500, 15) shows hypointense area (arrow) in the metaphysis (cyst) which is hyperintense (arrow) on FLASH 2D FAT SAT (500, 15, 10) (B, C).

of its growth zones. Since radiography does not provide sufficient information about the cartilaginous part the search was for methods able to visualize the cartilage. The alternative diagnostic measure appeared to be MR imaging. Johnson et al. (1989) presented MR imaging anatomy of the infant hip and several authors could trace the pathology in DDH (Johnson et al. 1988, Bos et al. 1988, 1989, Guidera et al. 1990, Fisher et al. 1991 Wierusz-Kozłowska et al. 1994).

The introduction of MR imaging opened a new era also in the evaluation of growth of the femur in normal and pathologic conditions. Harcke et al. (1992) and Synder (1992) gave a detailed description of the behavior of growth plates of the distal femur and proximal tibia during growth. The knees they evaluated were divided into 4 developmental groups accord-

ing to general MRI characteristics. The attempt in this study was to show the pattern of MR imaging appearance of the proximal femur. Considering the behavior of the subcapital growth plate (Batory 1982) I distinguished 3 developmental groups and the formation of growth plate was the borderline between the first and second group at below 5 years of age at the latest. In all children at the time of investigation the ossific nucleus was already present and its signal was high on T1-weighted images proving that yellow marrow had developed. The nucleus was well delineated by bone margin of low signal intensity formed by hemispherical zone of provisional calcification and the bone end plate at the basis of the ossific nucleus.

In contrast to the ossific nucleus the signal of the metaphysis was hypointense, although its intensity was still greater than that of the muscle. In the third

group after closure of the growth plate the signal of the metaphysis equaled that of the epiphysis, the findings which were previously observed by other authors (Vogler and Murphy 1988, Harcke et al. 1992, Synder 1992). The ossific nucleus of the greater trochanter was hyperintense on T1-weighted images from its appearance with a signal intensity similar to that of the femoral head.

Growth cartilages of the proximal femur in MRI

The cartilage covering the bony center which consists of two different zones (McKibbin and Holdsworth 1967, Siffert and Feldman 1980) in most hips was homogenous on T1-weighted images. Only in a few it was possible to distinguish the hypointense zone separating the articular cartilage and the growth cartilage within the hemispherical cartilage covering the bony center of the femoral head, a finding also noticed in distal femur by Jaramillo and Hoffer (1992).

The femoral neck isthmus described as a separate growth zone by Siffert (1981) and Grybos (1983) was well delineated in all hips as a band of intermediate signal intensity on T1-weighted images and hyperintense on sequences using fat saturation. The band was in continuity with the growth cartilages of the femoral head and greater trochanter and also with the subcapital growth plate and the growth plate of the greater trochanter, supporting the theory of the uniform growth cartilage presented by Taussig et al. (1976). The continuity of these cartilages in MRI in infancy was also noted by Johnson et al. (1989).

The subcapital growth plate cartilage when already formed was well visible on T1-weighted images as a band of intermediate signal intensity only in the younger age group, lying between the hypointense lines representing epiphyseal bone plate and zone of provisional calcification. In older children this presentation was found occasionally and usually only the hypointense band was visible. I could not find explanation for this, except that the growth plate seldom presented itself as a straight line band visible mostly in the Group I. The most common finding was arched or irregular course. With the slice thickness of 4 mm it was possible that the picture of the growth cartilage was a collection of signal from the cartilage itself and two neighboring hypointense lines from the epiphysis and metaphysis. This disadvantage was also noted in CT where a structure or lesion that only partially occupies the thickness of a slice is subject to partial volume averaging which has the effect of misrepresenting the true density of the lesion or allowing it to be undetected (Harcke 1990). Also other technical rea-

sons should be considered since I used only the body Helmholtz-type coil. It is to be investigated whether new generation of CP Flex coil and CP Array coil could change the growth plate presentation on T1-weighted images, however, these disturbances had little impact on the further results of the study.

In older children, in Group II and III, as a result of progressive ossification of the cartilaginous epiphysis, the thickness of the subcapital growth plate is gradually reduced, and the distinction between cartilage and bone may be most satisfactorily obtained by utilizing fat saturation technique in which the cartilage is hyperintense in relation to the osseous structures. This finding was observed also in the knee by other authors (Synder 1992, Lawson et al. 1994).

When a physal closure begun it commenced at the periphery of the plate and the cartilage was still visible at the center. Of interest in this context is the study by Dvonsch and Bunch (1983) which indicates, that ossification of the subcapital growth plate normally begins on the lateral side and progresses medially. Harcke et al. (1992) and Synder (1992) gave the details concerning the MR picture of growth plate closure in the knee. They termed the disappearance of the growth plate signal as a "drop-out sign" which may be present due to physiologic or pathologic conditions as well as to technical reasons. With the use of fat saturation sequences I was able to differentiate between the possible causes of this finding.

The hemispherical cartilage and growth plate of the greater trochanter were well visible on T1-weighted images as a bands of intermediate signal intensity and of high signal intensity on gradient echo images. Gradual thinning was observed during growth with final closure of the growth plate in the third group. The MR picture of growth plate in this area may be useful in assessment of epiphyseodesis which was presented in the knee by Synder (1992).

Visibility of the subcapital growth plate and its damage on radiography and in MRI

In the patients analyzed in this study MRI was never used as a primary diagnostic technique. The initial attempt of diagnosis or suspicion of a growth plate injury was based on radiographs. Radiographic changes most suggestive of physal disturbance were an erased area in the line of the growth plate or already distorted head-neck relationship. The image of the growth plate on MRI varied greatly depending on the sequence used, extent of damage and age of the subject. On T1-weighted images the appearance of the growth cartilage was mostly fair as it was earlier demonstrated on healthy individuals. On sequences utiliz-

ing fat saturation, e.g., T1 FAT SAT and FLASH 3D FAT SAT the visibility of the growth plate was much improved. Since FLASH 3D gives more possibilities for further studies I find this sequence together with T1-weighted images as being sufficient for the assessment of the growth plate and its damage, a finding which was also confirmed by Harcke et al. (1992) in the distal femoral physis.

In several hips with no growth plate line on radiography MRI was able to reveal a line on certain slices, thus proving that the closure of the growth plate was not complete but these remnants are probably of no value for further growth.

The assessment of the growth plate was of lesser value when comparing to radiography in case where there was still a fragmentation phase of the femoral head. In this case the epiphysis was poorly visible on MRI and distinction of the growth plate was not clear, whereas on radiography there were already bony fragments in the epiphysis with irregular growth plate visible.

MR imaging has proved to be the modality of choice for the evaluation of the traumatized physis before the development of any osseous bridge (Lawson et al. 1994). It would be interesting to demonstrate any changes in the proliferative zone of the growth cartilage but differentiation of the specific histologic zones of the growth plate has not yet been demonstrated by MR imaging. Jaramillo et al. (1990) with gadolinium enhancement were able to indicate the development of vascularity through the plate, which preceded the formation of a bony bridge. The early phase of growth disturbance was found in some hips in this study, being recognized on fat saturation sequences but yet invisible on radiography and T1-weighted images. The bony bridges containing yellow marrow were best visible as hyperintense structures on T1-weighted images, and when hypointense, were recognized as fibrous or cortical. On gradient echo images I was able to demonstrate the lesion without possibility to differentiate between different types of bridges (bony with yellow marrow, dens bone and fibrous). Also according to Jaramillo et al. (1990) differentiation between a small bony bridge and a fibrous bridge is not possible with MR imaging, because both are hypointense on all sequences.

Deformities of the proximal femur following partial destruction of the growth plate depend on the position of a bridge that forms between the epiphysis and metaphysis. Accurate delineation of the site, number and size of bridges within the growth plate may help in prediction of the imminent growth disturbance. Each of existing methods of defining the bridges have certain disadvantages. The most com-

mon one is radiation exposure, and this alone may be a compelling reason to consider one technique over another, as it is with Carlson and Wenger (1984) polytomographic method in comparison to that developed in my study, both having similar principles. Another drawback of these two methods is that the subcapital growth plate is considered here as a one-plain structure. Since it is rather spherical, certain error is possible to occur when plotting the damage determined from each cut into the cross-sectional shape of the physis. Also some anterior or posterior tilt may influence the result. However, in my opinion, in most cases only slight distortion may be expected.

Porat (1987) CT technique is useful in flat and horizontally oriented growth plates and thus of no use in subcapital growth plate in proximal femur.

Several works attempted to assess, with positive result, whether MR imaging provides information on growth plate lesions beyond that obtained with other radiographic techniques (Havranek and Lizler 1991, Jaramillo et al. 1990, Jaramillo and Hoffer 1992, Snyder 1992). Radiographic assessment still remains imperative in the evaluation of avascular necrosis both to document the pattern of healing and monitor the development of the hip. In the present study, as well as found previously, more information concerning both the visibility of the growth plate and its lesion was provided by MRI. Of interest in this context is a case in which radiography gave more valuable information. One explanation for this finding is that in case of epiphyseal necrosis with metaphyseal involvement all these structures together with hypointense physal line were of similar signal intensity. On radiography dense necrotic area of the epiphysis was easier differentiated from the growth plate.

Necrosis of the epiphysis and metaphyseal changes on radiography and in MRI

The usefulness of MR imaging in assessment of avascular necrosis of the femoral head is already well recognized. Several studies have shown, that MR imaging can depict early AVN of the femoral head in adults by demonstrating greatly decreased signal from within the marrow cavity (Beltran et al. 1988, Grimm et al. 1989, Markisz et al. 1987, Mitchell et al. 1987, Sarrat et al. 1988, Hauzeur et al. 1989, Seiler et al. 1989, Lee et al. 1990, Tervonen et al. 1992, Lafforgue et al. 1993). MR imaging also appeared to be a very useful diagnostic tool in the Legg-Calve-Perthes disease (Scoles et al. 1984, Henderson et al. 1990, Hoffinger et al. 1991, Hoffinger et al. 1993). Johnson et al. (1988) as well as Kruczynski et al. (1995) mentioned its diagnostic value in AVN in DDH. In the

present study necrosis of the proximal femur was already at its late stages and in half of the hips the changes were not detected on radiography. The presentation of the necrotic area was typical on T1-weighted images, while on gradient echo images the necrotic area was inhomogenous mixture of high and low signal intensity. The question remains to be answered whether the high signal on gradient echo images represents healing processes.

Metaphyseal cysts on radiography are a typical finding in severe AVN although its origin and significance are still under debate (Brashear 1963, Tönnis and Kuhlmann 1969, Bucholtz and Ogden 1978, Hirohashi et al. 1980, Thomas et al. 1982, Kruczynski 1987). MR imaging appeared to be a useful tool in detection of metaphyseal involvement in cases analyzed in this study. Intermediate signal intensity on T1-weighted images and high on gradient echo images may suggest a cartilage filling up the cyst. This appearance confirms the concept of disturbed metaphyseal ossification but its influence on further growth is not fully predictable and should be carefully observed individually (Kruczynski 1995). Focal extension of the growth cartilage into the metaphysis was seen in 5 patients out of 25 investigated by Jaramillo et al. (1990) due to physeal fracture suggesting in his opinion growth plate dysfunction. Hoffinger et al. (1993) on patients with "metaphyseal" cysts in the course of Legg-Calve-Perthes disease demonstrated that the picture on plain radiographs in many cases may be misleading. In around half of the hips with "metaphyseal" changes on radiography MRI revealed no metaphyseal involvement and in half of the hips negative on radiography MRI showed positive. On the other hand with the physeal irregularities present, and because of the partial volume averaging used by MRI software, fine distinction between physis and metaphysis may prove difficult in the anterior portion of the femoral head.

Clinical significance

Angular deformities of the extremities following partial destruction of the growth plate are well known. The deformities may depend on the position of an osseous bridge that forms between the epiphysis and metaphysis. Several surgical procedures have been described that attempt to correct these deformities with the use of silicon rubber (Bright 1974), fat (Langenskiöld 1981), dead cartilage allograft plugs (Osterman 1972) or bone cement (Mallet 1975,

Mallet et Rey 1978) as a fillers. Transplantation of live chondrocytes into the gap with theoretical possibility of retaining its potential for growth is also promising (Kawabe et al. 1987). The success of these procedures depends on accurate delineation of the site, number and size of the bridges within the growth plate.

Due to the vascular anatomy of the proximal femur this locus is so far not an encouraging place for surgical excision of a physeal bar. Correction of anatomic and functional proportions between the femoral head, the neck, and greater trochanter may be achieved by trochanteric epiphyseodesis (Pylkkanen 1960, Gage and Cary 1980), distal and lateral transplantation of the greater trochanter (Jani 1969, Wagner 1978, Kelikian et al. 1983, Tauber et al. 1980, Fernbach et al. 1985, LLOYD-Roberts et al. 1985), or femoral osteotomies (Pauwels 1973, Wagner 1977, 1978). Detailed planning before the operation is necessary and confirmation of the presence and location of physeal damage may help to predict future deformity, thus optimizing the choice of corrective procedure.

It is to be hoped that with gradual improvement of surgical techniques removal of bony bridges from the subcapital growth plate may become feasible with careful MRI mapping using the method presented in this study.

Conclusions

Magnetic resonance imaging appeared to be superior to radiography in assessment of subcapital growth plate and its damage in most of the hips.

The most common presentation of the area of subcapital growth plate of the proximal femur on MR images is that of hypointense band on T1-weighted images and hyperintense band on sequences using fat saturation.

T1-weighted images are best for visualization of bony bridges containing yellow marrow crossing the growth plate.

The sequences using fat saturation are good for visualization of any damage to the growth plate without possibility do distinguish between fibrous or osseous changes.

T1-weighted images together with FLASH 3D FAT SAT seem to be sufficient for the analysis of the growth plate and its damage.

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References

- Alsberg A. Anatomie und klinische Betrachtungen über Coxa vara. *Z Orthop* 1899; 6: 106-171.
- Anderson C E, Parker J. Invasion and resorption in endochondral ossification: an electron microscopic study. *J Bone Joint Surg (Am)* 1966; 48: 899-914.
- Batory I. Ätiologie der pathologischen Veränderungen des kindlichen Hüftgelenkes. Enke, Stuttgart 1982.
- Batory I. Die Ätiologie des Luxationsperthes und seine Beziehung zu dem typischen Morbus Legg-Calve-Perthes. *Z Orthop* 1981; 119: 498-503.
- Becker F. Die konservative Behandlung der Hüftdysplasie und Hüftverrenkung. *Z Orthop* 1969; 106: 173-178.
- Beltran J, Herman L J, Burk J M, et al. Femoral head avascular necrosis: MR imaging with clinical-pathologic and radionuclide correlation. *Radiology* 1988; 166: 215-220.
- Bernardczyk K. Studies on the moulding effect of the acetabular roof on the formation of the dysplastic hip joint during growth. Thesis. Academy of Medicine, Poznan, Poland 1971.
- Bernbeck R. Untersuchung Über Pathologie und Ätiologie der Pertheschen Krankheit. *Verh Dtsch Orthop Ges*, 36 Kongr 1948. *Z Orthop* 1949; 78: 241-251.
- Bos C F A, Bloem J L, Oberman W R, Rozing P M. Magnetic resonance imaging in congenital dislocation of the hip. *J Bone Joint Surg (Br)* 1988; 70: 174-178.
- Bos C F A, Bloem J L, Verbout A J. Magnetic Resonance imaging in acetabular residual dysplasia. *Clin Orthop* 1989; 265: 207-217.
- Brashear M R. Epiphyseal avascular necrosis and its relation to longitudinal bone growth. *J Bone Joint Surg (Am)* 1963; 45 (7).
- Bright R. Operative correction of partial epiphyseal plate closure by osseous bridge resection and silicone rubber implant: experimental study in dogs. *J Bone Joint Surg* 1974 (Am); 56: 655-664.
- Brighton C T, Sugioka Y, Hunt R M. Cytoplasmic structures of the epiphyseal-plate chondrocytes. Quantitative evaluation using electron micrographs of rat costochondral junctions with special reference to the fate of hypertrophic cells. *J Bone Joint Surg* 1973 (Am); 55: 771-784.
- Brighton C T. Clinical problems in epiphyseal plate growth and development. American Academy of Orthopaedic Surgeons Instructional Course Lectures, XXIII. St Louis, CV Mosby, 1974.
- Brighton C T. Structure and function of the growth plate. *Clin Orthop* 1978; 136: 22-32.
- Brighton C T. The growth plate. *Orthop Clin* 1984; 15 (4): 571-595.
- Brighton C T, Heppenstall B R. Oxygen tension in zones of epiphyseal plate, the metaphysis and diaphysis. An in vivo and vitro study in rats and rabbits. *J Bone Joint Surg (Am)* 1971; 53: 719-728.
- Brookes M, Landon D N. The juxta-epiphyseal vessels in the long bones of fetal rats. *J Bone Joint Surg (Br)* 1963; 46: 336-345.
- Bruszewski J, Bernardczyk K, George J. Radiographic long-term follow-up of prearthrotic changes in unilaterally overloaded hip (in Polish). *Chir Narz Ruchu i Ortop Pol* 1973; 67-72.
- Buchanan J R, Greer R B, Cotler J M. Management strategy for prevention of avascular necrosis during treatment of congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1981; 63: 140-146.
- Bucholz R W, Ogden J A. Patterns of ischemic necrosis of the proximal femur in nonoperatively treated congenital hip disease. Proceedings of the 6th Scientific Meeting of the Hip Society; Mosby, St.Louis 1978: 43-63.
- Calvert P T, Kernohan J G, Sayers D C J, Catteral A. Effects of vascular occlusion of the femoral head in growing rabbits. *Acta Orthop Scand* 1984; 55: 526-530.
- Carlson W O, Wenger D R. A mapping method to prepare for surgical excision of a partial physeal arrest. *J Pediatr Orthop* 1984; 4: 232-238.
- Chung S M K. The arterial supply of the developing proximal end of the human femur. *J Bone Joint Surg (Am)* 1976; 58: 961-970.
- Coleman S S. Developmental dislocation of the hip: evolutionary changes in diagnosis and treatment. *J Pediatr Orthop* 1994; 14: 1-2.
- Cooperman D R, Wallenstein R, Stulberg D. Post-reduction avascular necrosis in congenital dislocation of the hip. Long-term follow-up study of twenty-five patients. *J Bone Joint Surg (Am)* 1980; 62: 247-259.
- Crego C H, Schwartzman J R. Follow-up study of the early treatment of congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1948; 30: 428-442.
- Crock H V. A revision of the anatomy of the arteries supplying the upper end of the human femur. *J Anat* 1965; 99:77-88.
- De Seze S, Lequesne M. *Revue du Rhumatisme* 1956; 23: 201.
- Dooley B J. Osteochondritis in congenital dislocation and subluxation of the hip. *J Bone Joint Surg* 1964; 46: 198-203.
- Dvnoch V M, Bunch W H. Pattern of closure of the proximal femoral and tibial epiphyses in man. *J Pediatr Orthop* 1983; 3: 498-501.
- Edgren W. Coxa plana. *Acta Orthop Scand (Suppl 84)* 1965.
- Eyre-Brook A L. Osteochondritis deformans coxae juvenilis or Perthes disease. *British J Surg* 1936; 24: 166-182.
- Farnum C E, Wilsman N J. Lecitin-binding histochemistry of intracellular and extracellular glycoconjugates of the reserve cell zone of growth plate cartilage. *J Orthop Res* 1988; 6:166.
- Fernbach S K, Poznanski A K, Kelikian A S, Tachjadian M O, Marzano M J. Greater trochanteric overgrowth: development and surgical correction. *Radiology* 1985; 154: 661-664.
- Ferran J L, Diméglio A, Leboucq N, Couture A, De Rosa V. Three-dimensional computed tomography of the infantile hip. *J Pediatr Orthop* 1994; 3: 131-134.

- Ferrer A, Castillo F, Edez-Paredes E. Non iatrogenic avascular necrosis in CDH. Proceedings of the 10th meeting of the European Paediatric Orthopaedic Society, Paris, France 1991; 50.
- Fisher R, O'Brien T S, Davis K M. Magnetic resonance imaging in congenital dysplasia of the hip. *J Pediatr Orthop* 1991; 11: 617-622.
- Fisher R L, Cary J M. Avascular necrosis complicating congenital dislocation of the hip. *Internat Orthop* 1978; 2: 229-240.
- Gage J R, Winter R B. Avascular necrosis of the capital femoral epiphysis as a complication of closed reduction of congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1972; 54 (3): 373-388.
- Gage J, Cary J. The effects of trochanteric epiphyseodesis on growth of the proximal end of the femur following necrosis of the capital femoral epiphysis. *J Bone Joint Surg (Am)* 1980; 62 (5): 785-794.
- Gamble J G, Mochizuki C, Bleck E E, Rinsky L A. Coxa magna following surgical treatment of congenital hip dislocation. *J Pediatr Orthop* 1985; 5: 528-533.
- Gardiner H M, Duncan A W. Radiological assessment of the effect of splinting on early hip development: results from randomised controlled trial of abduction splinting vs sonographic surveillance. *Pediatr Radiol* 1992; 22 (3): 159-162.
- Gizycka I. Continuous traction in the treatment of congenital dislocation of the hip in children (in Polish). *Chir Narz Ruchu Ortop Pol* 1953; 18: 91-95.
- Gonczarova M N, Mirzoeva I J, Jarosevskaja E N. Characteristic of morfologic changes in the femoral head post reduction of congenital hip dislocation (in Russian). *Ortop Travmat Protezir* 1970; 31 (2): 10-15.
- Gore D R. Iatrogenic avascular necrosis of the hip in young children. A review of six cases. *J Bone Joint Surg (Am)* 1974; 56 (3): 493-502.
- Gregosiewicz A, Wosko I. Risk factors of avascular necrosis in the treatment of congenital dislocation of the hip. *J Pediatr Orthop* 1988; 8: 17-19.
- Grill F, Bensahel H, Canadell J, Dungal P, Matasovic T, Vizkelety T. The Pavlik harness in the treatment of congenital dislocating hip: report on a multicenter study of the European Paediatric Orthopaedic Society. *J Pediatr Orthop* 1988; 8: 1-8.
- Grimm J, Hopf Ch, Higer H P. Die Femurkopfnekrose. Diagnostik und morphologische Analyse mittels Röntgen, Szintigraphie, Computertomographie und Magnetresonanztomographie. *Z Orthop* 1989; 127: 680-690.
- Grybos J. Developmental model of the femoral neck. Autonomic growth process in the femoral neck and risk of disturbances after operative treatment on the hip. Academy of Medicine, Krakow 1983.
- Guidera K J, Einbecker M A, Berman C G, Ogden J A, Arrington J A, Murtagh R. Magnetic resonance imaging evaluation of congenital dislocation of the hips. *Clin Orthop* 1990; 261: 96-101.
- Harcke T H. Imaging of the immature skeleton. Lowell and Winter's Pediatric Orthopaedics, J B Lippincott Co, Philadelphia 1990.
- Harcke H T, Synder M, Caro P A, Bowen J R. Growth plate of the normal knee: evaluation with MR imaging. *Radiology* 1992; 183: 119-123.
- Hauzeur J P, Pasteels J L, Schoutens A, Ninsenkamp A, Appelboom T, Chochrad I, Perlmutter N. The diagnostic value of magnetic resonance imaging in non-traumatic osteonecrosis of the femoral head. *J Bone Joint Surg (Am)* 1989; 71: 641-649.
- Havranek P, Lizler J. Magnetic resonance imaging in the evaluation of partial growth arrest after physeal injuries in children. *J Bone Joint Surg* 1991 (Am); 73: 1234-1241.
- Henderson R C, Renner J B, Sturdivant M C, Greene W B. Evaluation of magnetic resonance imaging in Legg-Perthes disease: a prospective blinded study. *J Pediatr Orthop* 1990; 10: 289-297.
- Herold H Z. Avascular necrosis of the femoral head due to malposition in untreated congenital dislocation of the hip. *Internat Orthop* 1979; 2: 293-296.
- Heyman C, Herndon C. A method for measurement of the roentgenographic results in the Legg-Perthes disease. *J Bone Joint Surg (Am)* 1950; 32: 767-778.
- Hilgenreiner H. Zur Frühdiagnose der angeborenen Hüftgelenkverrenkung. *Med Klin* 1925; 21: 1385-1388, 1425-1429.
- Hirohashi K. Perthes disease. A classification based on the extent of epiphyseal and metaphyseal involvement. *Int Orthop* 1980; 4: 47-55.
- Hoffinger S A, Rab G T, Salamon P B. "Metaphyseal" cysts in Legg-Calve-Perthes disease. *J Pediatr Orthop* 1991; 11: 301-306.
- Hoffinger S A, Henderson R C, Renner J B, Dales M C, Rab G T. Magnetic resonance evaluation of "metaphyseal" changes in Legg-Calve-Perthes disease. *J Pediatr Orthop* 1993; 13: 602-606.
- Howe W W, Lacey T II, Schwartz R P. A study of the gross anatomy of the arteries supplying the portion of the femur and acetabulum. *J Bone Joint Surg (Am)* 1950; 32: 856-858.
- Howman-Giles R, Trochei M, Yeates K. Partial growth plate closure: apex view on bone scan. *J Pediatr Orthop* 1985; 5: 109-111.
- Hunziker E B, Schenk R K, Cruz-Orive L M. Quantitation of chondrocyte performance in growth plate cartilage during longitudinal bone growth. *J Bone Joint Surg (Am)* 1987; 69: 126-173.
- Iannotti J P. Growth plate physiology and pathology. *Orthop Clin North Am* 1990; 21: 1-117.
- Jani L. Die Entwicklung des Schenkelhalses nach der Trochanter-Versetzung. *Arch Orthop Unfallchir*, 1969; 66: 127-134.
- Jansen M. Platte Hüftpfanne und ihre Folgen. *Coxa plana, valga, vara und malum coxae*. *Z Orthop* 1925; 46: 2-42.
- Jaramillo D, Hoffer F A. Cartilaginous epiphysis and growth plate: normal and abnormal MR imaging findings. *Am J Radiol* 1992; 158: 1105-1110.
- Jaramillo D, Hoffer F A., Shapiro F, Rand F. MR imaging of fracture of the growth plate. *Am J Radiol* 1990; 155: 1261-1265.
- Jaramillo D, Shapiro F, Hoffer A, Winalski C, Koskinen M, Frasso R, Johnson A. Posttraumatic growth plate abnormalities: MR imaging of bony-bridge formation in rabbits. *Radiology* 1990; 175: 767-773.
- Johnson C, Wenger D, Robert J, Burke S, Roach S. Acetabular coverage: three dimensional anatomy and radiographic evaluation. *J Pediatr Orthop* 1986; 6: 548-558.

- Johnson N D, Wood B P, Jackman K V. Complex infantile and congenital hip dislocation: assessment with MR imaging. *Radiology* 1988; 168: 151-156.
- Johnson N D, Wood B P, Noh K S, Jackman K V, Wesaesson P L, Katzberg R W. MR imaging anatomy of the infant hip. *Am J Radiol* 1989; 153: 127-133.
- Jones D. Sub-capital coxa valga after varus osteotomy for congenital dislocation of the hip. A report of six cases with minimum follow-up of nine years. *J Bone Joint Surg (Br)* 1977; 59: 152-158.
- Kahle K W, Anderson M B, Alpert J, Stevens P M, Coleman S S. The value of preliminary traction in the treatment of congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1990; 72: 1043-47.
- Kalamchi A, MacFarlane R. The Pavlik harness: results in patients over three months of age. *J Pediatr Orthop* 1982; 2: 3-8.
- Kalamchi A, MacEwen G D. Avascular necrosis following treatment of congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1980; 62: 876-888.
- Kallio P E. Coxa magna following transient synovitis of the hip. *Clin Orthop* 1988; 228: 49-56.
- Katz J F. Metaphyseal cysts in coxa plana. A hypothetical explanation for their evolution. Proceedings of the 12th Congress of the International Society of Orthopaedic Surgery and Traumatology, Tel Aviv, Israel 1972: 221-224.
- Kawabe N, Ehrlich M, Mankin H. Growth plate reconstruction using chondrocyte allograft transplants. *J Pediatr Orthop* 1987; 7: 381-388.
- Kelikian A, Tachdjian M, Askew M, Jasty M. Greater trochanteric advancement of the proximal femur: a clinical and biomechanical study. In: The hip. Proceedings of the 11th Open Scientific Meeting of the Hip Society. St. Louis, CV Mosby 1983; 77-105.
- Kember N F. Cell division in endochondral ossification: a study of cell proliferation in rat bones by the method of tritiated thymidine autoradiography. *J Bone Joint Surg (Br)* 1960; 42:824.
- Keret D, MacEwan D G. Growth disturbance of the proximal part of the femur after treatment for congenital dislocation of the hip. *J Bone Joint Surg (Am)* 1991; 73 (3): 410-423.
- Kohler R, Seringe R. Osteochondrite Primitive de la Hanche. Expansion Scientifique Française, Cahiers d'enseignement de la SOFCOT, Paris 1981.
- Kolaczkowski J, Baczyk K, Strzeczewski H. The influence of position of lower extremities on arterial circulation in the hip (in Polish). *Chir Narz Ruchu Ortop Pol* 1953; 18: 47-50.
- Krol J. Avascular necrosis of the hip after operative treatment of congenital hip dislocation with particular interest in influence of pressure and immobilisation on articular bony ends (in Polish). PZWŁ, Warszawa 1963.
- Kruczynski J. Rebuilding of the hip with avascular necrosis after nonoperative treatment of congenital hip dislocation. Thesis, Academy of Medicine, Poznan, Poland 1987.
- Kruczynski J, Lempicki A, Wierusz-Kozłowska M. Radiographic picture of avascular necrosis complicating nonoperative treatment with the Frejka pillow of congenital dislocation of the hip. *Chir Narz Ruchu Ortop Pol* 1990; 55 (4,5,6): 335-341.
- Kruczynski J. Avascular necrosis after nonoperative treatment of developmental hip dislocation. *Acta Orthop Scand* 1995; 66 (3): 239-244.
- Kruczynski J, Ziemiński A, Borkowski W. Changes within subcapital growth plate due to avascular necrosis after treatment for CDH in radiography and MRI. *Chir Narz Ruchu Ortop Pol* 1995; 60 (5): 377-383.
- Kummer B. Die Beanspruchung der Gelenke, dargestellt am Beispiel des menschlichen Hüftgelenkes. Read at the Verh DGOT, 55 Kongr Kassel 1968. Enke, Stuttgart, 301-311.
- Lafforgue P, Dahan E, Chagnaud Ch, Schiano A, Kasbarian M, Acquaviva P C. Early-stage avascular necrosis of the femoral head: MR imaging for prognosis in 31 cases with at least 2 years of follow-up. *Radiology* 1993; 187: 199-204.
- Langenskiöld A. Surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981; 1: 3-11.
- Lauritzen J. The arterial supply to the femoral head in children. *Acta Orthop Scand* 1974; 45: 724-736.
- Law E G, Heistad D D, Marcus M L, Mickeson M R. Effect of the hip position on blood flow to the femur in puppies. *J Pediatr Orthop* 1982; 2: 133-137.
- Lawson J, Keller M, Rattner Z. Recent advances in pediatric musculoskeletal imaging. Read at Sartoris D.: Advances in musculoskeletal imaging. *Radiol Clin North Am* 1994; 32 (2): 353-375.
- Lee M J, Corrigan J, Stack J P, Ennis J T. A comparison of modern imaging modalities in osteonecrosis of the femoral head. *Clin Radiol* 1990; 42: 427-432.
- Lloyd-Roberts G., Wetherill M., Fraser M.: Trochanteric advancement for premature arrest of the femoral capital growth plate. *J Bone Joint Surg (Br)*, 1985; 67 (1): 21-24.
- Labaziewicz L. Angle of the distance between the femoral head and the acetabular floor (in Polish). *Chir Narz Ruchu Ortop Pol* 1979; 44: 385-387.
- Lempicki A. Radiologischer dislokationswert des dysplastischen Hüftgelenks beim Kind. *Beitr Orthop Traumat* 1989; 33: 236-267.
- Lempicki A, Wierusz-Kozłowska M, Kruczynski J. Abduction treatment in late diagnosed congenital dislocation of the hip. *Acta Orthop Scand (Suppl 236)* 1990; 61: 1-30.
- Mallet J, Rey J C. Traitement des épiphysiodèses partielles traumatiques chez l'enfant par désépiphysiodèse. *Int Orthop* 1978; 1: 309.
- Mallet J. Les épiphysiodèses traumatiques de l'extrémité inférieure du tibia chez l'enfant: leur traitement avec désépiphysiodèses. *Rev Chir Orthop* 1975; 61: 5-9.
- Markisz J A, Knowles R, James R, Altchek D W, Schneider R, Whalen J P, Cahill P T. Segmental patterns of avascular necrosis of the femoral heads: early detection with MR imaging. *Radiology* 1987; 162: 717-720.
- Massie W K. Vascular epiphyseal changes in congenital dislocation of the hip. Results in coxa plana and congenital dislocation without vascular changes. *J Bone Joint Surg (Am)* 1951; 33: 284-304.
- McKibbin B, Holdsworth F W. The dual nature of epiphyseal castilage. *J Bone Joint Surg (Br)* 1967; 2 (49): 351-361.
- Meyer J. Legg-Calve-Perthes disease. *Acta Orthop Scand (Suppl 167)* 1977; 1-131.

- Mitchell G P. Problems in the early diagnosis and management of congenital dislocation of the hip. *J Bone Joint Surg (Br)* 1972; 54: 4-12.
- Mitchell D G, Rao V M, Dalinka M K, Spritzer E, Alavi A, Steinberg M E, Fallon M, Kressel H Y. Femoral head avascular necrosis: Correlation of MR imaging, radiographic staging, radionuclide imaging, and clinical findings. *Radiology* 1987; 162: 709-715.
- Mittelmeier H. Beitrag zur funktionellen Therapie und Spontanreposition der angeborenen Luxationhüfte mit Spreizhöschen und Pavlik-Bandage. *Arch Orthop Trauma Surg* 1961; 52: 465-522.
- Morgan J D. Blood supply of the growing rabbit's tibia. *J Bone Joint Surg (Br)* 1959; 41: 185.
- Mose K. Legg-Parthes-Calvé disease. *These Méd, Copenhagen-Aarhus* 1964.
- Murray I. Bone scanning in the child and young adult. *Skeletal Radiol* 1980; 5: 65-76.
- Müller M. Ischiometrie radiologique. *Rev Chir Orthop* 1956; 24: 124-133.
- Nicholson J T, Koppel H P, Mattei F A. Regional stress angiography of the hip. A preliminary report. *J Bone Joint Surg (Am)* 1954; 36: 503-510.
- O'Brien T, Millis M B, Griffin P P. The early identification and classification of growth disturbance of the proximal end of the femur. *J Bone Joint Surg (Am)* 1986; 68 (7): 970-980.
- Ogden J A. Editorial. Femoral shaft fractures. *J Pediatr Orthop* 1995; 15: 1-2.
- Ogden J A. Changing patterns of proximal femoral vascularity. *J Bone Joint Surg (Am)* 1974; 56: 941-950.
- Österman K. Operative elimination of partial premature epiphyseal closure: an experimental study. *Acta Orthop Scand (Suppl 147)* 1972.
- Palmen K. Prevention of congenital dislocation of the hip. *Acta Orthop Scand (Suppl 208)* 1984; 1-107.
- Papadopoulos J S. Osteolytische Druckdefekte am Schenkelhaus und Hüftköpfen bei extremer Spreizbehandlung. *Z Orthop* 1972; 110: 182-186.
- Papavasiliou V A, Piggott H. Acetabular floor thickening and femoral head enlargement in congenital dislocation of the hip: lateral displacement of femoral head. *J Pediatr Orthop* 1983; 3: 22-27.
- Pauwels F. *Atlas zur Biomechanik der gesunden und kranken Hüfte*. Springer-Verlag, Berlin Heidelberg New York 1973.
- Pavlik A. Die funktionelle Behandlungsmethode mittels Riemenbügel als Prinzip der konservativen Therapie bei angeborener Hüftgelenksverrenkung der Säuglinge. *Z Orthop* 1957; 89: 341-352.
- Peterson H, Klassen R, MacLedd R, Hofman A. The use of computerised tomography in dislocation of the hip and femoral neck anteversion in children. *J Bone Joint Surg (Br)* 1981; 63: 198-208.
- Petit P. Osteochondritis in congenital dislocation of the hip. *J Bone Joint Surg (Br)* 1955; 37: 513.
- Polakowski L, Senger A, Strzyzewski H. Avascular necrosis of the hip as a sequel of conservative treatment of congenital dislocation (in Polish). *Chir Narz Ruchu Ortop Pol* 1953; 18: 61-66.
- Porat S, Nyska M. Assessment of bony bridge by computed tomography: experimental model in the rabbit and clinical application. *J Pediatr Orthop* 1987; 7: 155-160.
- Przychodzki M, Piatkowski S, Swiderski G. Results of conservative treatment of congenital hip dislocation in children (in Polish). *Chir Narz Ruchu Ortop Pol* 1953; 18: 61-66.
- Pylkanen P. Coxa vara infantum. *Acta Orthop Scand (Suppl 48)* 1960;
- Ramsey P L, Lasser S, MacEwen G D. Congenital dislocation of the hip. Use of the Pavlik harness in the child during the first six months of life. *J Bone Joint Surg (Am)* 1976; 58: 1000-1004.
- Robert H, Seringe R. Les osteochondrites au cours du traitement de la luxation congenitale de la hanche. Read at Cahiers d'enseignement de la SOFCOT, Expansion Scientifique Francaise, Paris 1981; 103-112.
- Robert H, Seringe R. Les troubles de developpement de l'extremite supérieure du femur apres traitement de la luxation congenitale de hanche. *Rev Chir Orthop* 1982; 68: 425-439.
- Robertson W W Jr. Newest knowledge of the growth plate. *Clin Orthop* 1990; 253: 270-278.
- Robinson H J, Shannon M A. Avascular necrosis in congenital hip dysplasia: the effect of treatment. *J Pediatr Orthop* 1989; 9: 293-303.
- Rogala P, Radziejowski M, Kruczyński J. Results of the operative treatment of the dysplastic joint with osteochondrosis. *Chir Narz Ruchu Ortop Pol* 1988; 53 (3): 224-229.
- Salter R B, Kostuik J, Dallas S. Avascular necrosis of the femoral head as a complication of treatment for congenital dislocation of the hip in young children. *Canadian J Surg* 1969; 12: 44-61.
- Sarrat P, Acquaviva P C, Lafforgue P, Lopez-Vasquez M, Bernard P, Bouscarle B. Ostéonécrose aseptique de la tête fémorale: apports de l'imagerie par résonance magnétique. *Ann Radiol* 1988; 31: 133-139.
- Schede F. Die Ergebnisse unserer Behandlung der angeborenen Hüftverrenkung. *Z Orthop* 1940; 71: 3.
- Schede F. Die unblutige Behandlung der angeborenen Hüftverrenkung. *Verh Dtsch Ges, 37 Kongr* 1949. *Z Orthop* 1950; 79: 133-140.
- Schoenecker P L, Bitz M, Whiteside L A. The acute effect of position of immobilisation on capital femoral epiphyseal blood flow. A quantitative study using the hydrogen wash-out technique. *J Bone Joint Surg (Am)* 1978; 60: 899-904.
- Scoles P V, Yoon Y S, Makley J T, Kalamchi A. Nuclear magnetic resonance imaging in Legg-Calve-Perthes disease. *J Bone Joint Surg* 1984 (Am); 66: 1357-1363.
- Scott J C. Frame reduction in congenital dislocation of the hip. *J Bone Joint Surg (Br)* 1953; 35: 372-374.
- Seiler J G, Christie M J, Homra L. Correlation of the findings of magnetic resonance imaging with those of bone biopsy in patients who have stage I or II ischemic necrosis of the femoral head. *J Bone Joint Surg (Am)* 1989; 71: 28-32.
- Shapiro F. Epiphyseal disorders. *New Engl J Med* 1987; 317: 1702-1709.
- Siffert R S. Patterns of deformity of the developing hip. *Clin Orthop* 1981; 160: 14-29.

- Siffert R, Feldman J. The growing hip. The dynamic development of the normal adult hip and deformed hip of Legg-Calve-Perthes' disease. *Acta Orthop Belg* 1980; 46: 443-476.
- Sommerville E W, Scott J C. The direct approach to congenital dislocation of the hip. *J Bone Joint Surg (Br)* 1957; 39: 623-648.
- Synder M. The value of MRI in imaging of growth plates around the knee joint. Thesis, Lodz 1992.
- Szczekot J. Standards for the radiological indexes of the hip joint in children (in Polish). *Chir Narz Ruchu Ortop Pol* 1974; 29: 67-71.
- Szulc A. Rebuilding of the dysplastic hip joint after conservative treatment. Thesis. Academy of Medicine, Poznan, Poland 1977.
- Tauber C, Ganel A, Horoszowski H, Farine I. Distal transfer of the greater trochanter in coxa vara. *Acta Orthop Scand* 1980; 51: 661-666.
- Taussig G, Delor M, Masse P. Les alteration de croissance de l'extremite supérieure du femur, leur apport a la connaissance de la croissance normale. *Essei de deductions therapeutiques. Rev Chir Orthop* 1976; 62: 91-210.
- Tervonen O, Mueller D M, Matteson E L, Velosa J A, Ginsburg W W, Ehman R L. Clinically occult avascular necrosis of the hip: prevalence in an asymptomatic population at risk. *Radiology* 1992; 182: 845-847.
- Thomas C L, Gage J R, Ogden J A. Treatment concepts for proximal femoral ischemic necrosis complicating congenital hip disease. *J Bone Joint Surg (Am)* 1982; 64: 817-828.
- Thomas I H, Dunin A J, Cole W G, Menelaus M B. Avascular necrosis after open reduction for congenital dislocation of the hip: analysis of causative factors and natural history. *J Pediatr Orthop* 1989; 9: 525-531.
- Tönnis D. Congenital hip dislocation—avascular necrosis. Georg Thieme Verlag, New York 1982.
- Tönnis D. Die angeborene Hüft-dysplasie und Hüftluxation im Kindes und Erwachsenen-alter. Springer Verlag, Berlin 1984.
- Tönnis D. Congenital dysplasia and dislocation of the hip in children and adults. Springer; Berlin 1987.
- Tönnis D, Kuhlmann G P. Untersuchungen über die Häufigkeit von Hüftkopfnecrosen bei Spreitzhosenbehandlung und verschiedenen konservativen Behandlungsmethoden der angeborenen Hüftdysplasie und Hüftluxation. *Z Orthop* 1968; 106: 651-672.
- Trueta J, Morgan J D. The vascular contribution to osteogenesis. I. Studies by the injection method. A zonal analysis of inorganic and organic constituents of the epiphysis during endochondral calcification. *J Bone Joint Surg (Br)* 1960; 42: 97-109.
- Trueta J. The normal vascular anatomy of the human femoral head during growth. *J Bone Joint Surg (Br)* 1957; 39: 358-394.
- Tucker F R. Arterial supply to the femoral head and its clinical importance. *J Bone Joint Surg (Br)* 1949; 31:82-93.
- Visser J D. Functional treatment of congenital dislocation of the hip. *Acta Orthop Scand (Suppl 206)* 1984; 55: 1-109.
- Vogler J B, Murphy W A. Bone marrow imaging. *Radiology* 1988; 168: 679-693.
- Wagner H. Operative lengthening of the femur. *Clin Orthop* 1978; 136: 125-142.
- Wagner H. Prinzipien der Korrekturosteotomie am Bein. *Orthopäde* 1977; 6: 145-177.
- Weickert H. Zur Differenzierung von Femurkopfnekrose und Femurkopfaufbaustörung bei der Behandlung der Luxationshüfte. *Z Orthop* 1970; 107: 440-447.
- Weiner O S, Hoyt W A, O'Donnell H W. Congenital dislocation of the hip. The relationship of premanipulation traction and age to avascular necrosis of the femoral head. *J Bone Joint Surg (Am)* 1977; 59: 306-311.
- Wertheimer L G, Lopes S L F. Arterial supply to the femoral head. A combined angiographic and histological study. *J Bone Joint Surg (Am)* 1971; 53: 545-556.
- Westin G W, Ilfeld F W. Total avascular necrosis of the capital femoral epiphysis in congenitally dislocated hips. *Clin Orthop* 1976; 119: 93-98.
- Wiberg G. Studies of dysplastic acetabula and congenital subluxation of the hip joint with special reference to the complication of osteoarthritis. Kungl. Boktryckeriet., Stockholm 1939.
- Wierusz-Kozłowska M, Ziemiński A, Kruczyński J, Krasny I. Usefulness of MRI in the diagnosis of selected hip diseases in children and adolescents (in Polish). *Chir Narz Ruchu Orthop Pol* 1994; 59: 429-434.
- Wierusz-Kozłowska M. Development and rebuilding during growth of the dysplastic hip treated due to congenital hip dislocation and prognostication of the results. Thesis (in press), Academy of Medicine, Poznan, Poland 1995.
- Zwierzchowski H., Zwierzchowska D. Changes in the proximal femoral epiphysis and metaphysis in children treated with the Frejka pillow and Koszla abduction device (in Polish). Proceedings of the XXV Meeting of the Polish Orthopedic and Traumatologic Society, PZWL, Lodz 1985; 459-461.

Appendix

Classification of avascular necrosis of the proximal femur complicating the treatment of developmental dislocation of the hip (Kruczynski 1987)

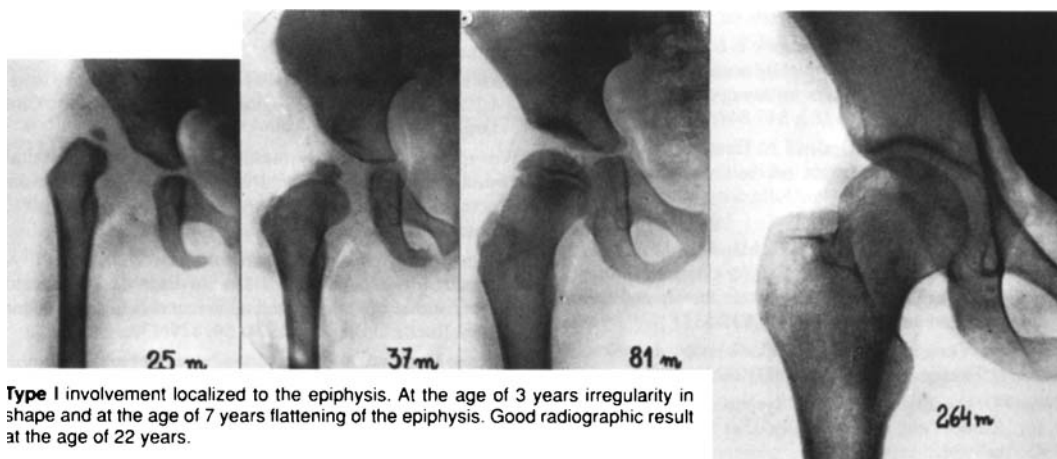
Type I: involvement of the epiphysis, no fragmentation, mild changes.

Type II: involvement of the epiphysis with fragmentation, moderate changes.

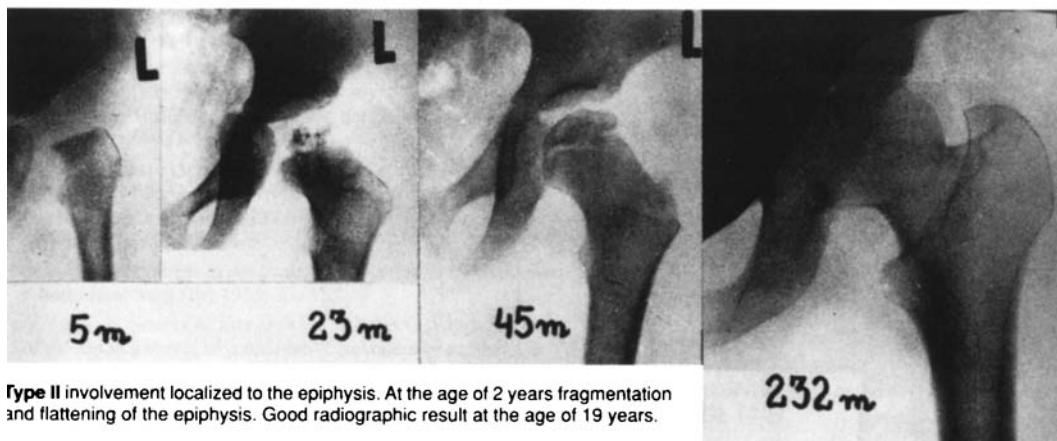
Type III: involvement of the epiphysis and the lateral part of the metaphysis under the physis, severe changes.

Type IV: involvement of the epiphysis and the medial metaphysis under the physis, severe changes.

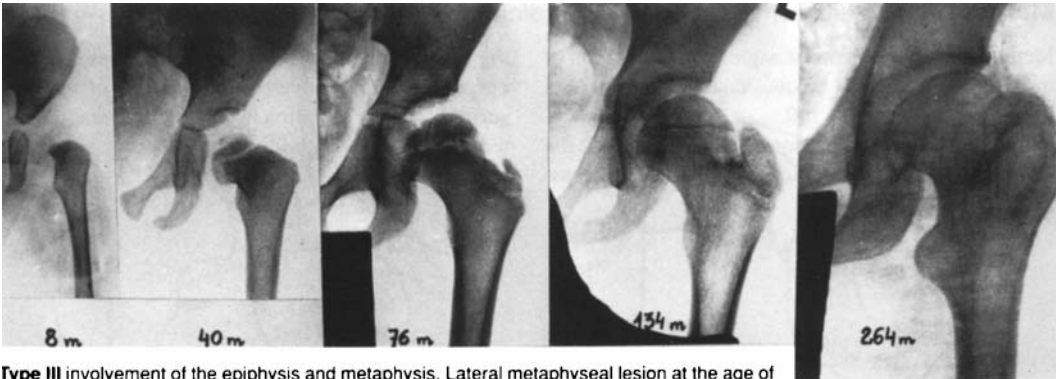
Type V: involvement of the epiphysis and the whole metaphysis under the physis, severe changes.



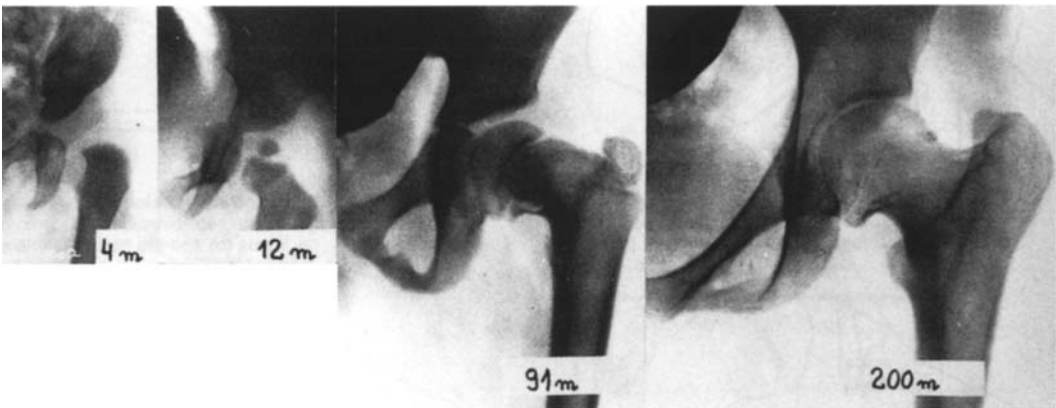
Type I involvement localized to the epiphysis. At the age of 3 years irregularity in shape and at the age of 7 years flattening of the epiphysis. Good radiographic result at the age of 22 years.



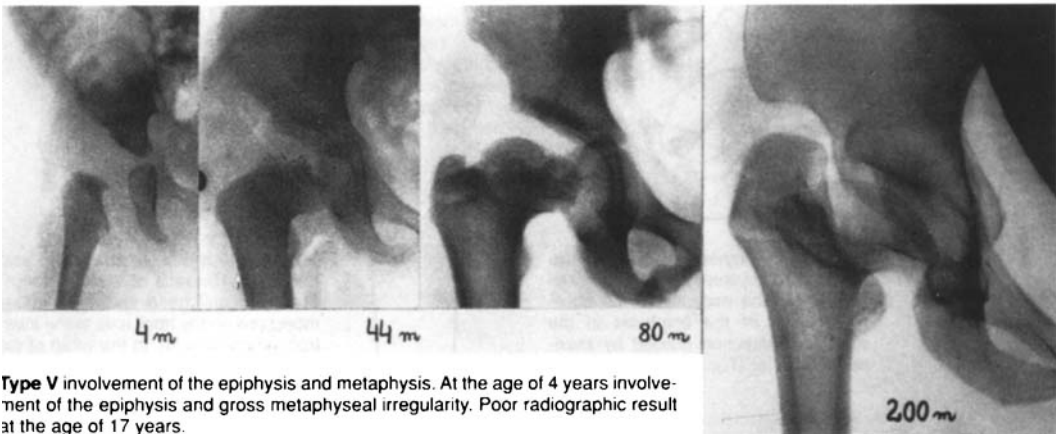
Type II involvement localized to the epiphysis. At the age of 2 years fragmentation and flattening of the epiphysis. Good radiographic result at the age of 19 years.



Type III involvement of the epiphysis and metaphysis. Lateral metaphyseal lesion at the age of 5 years. At the age of 11 years the line of the physis is horizontal and metaphyseal cystic lesion still visible. Fair radiographic result at the age of 22 years with the epiphysis tilted into valgus angulation and uncovered.



Type IV involvement of the epiphysis and metaphysis. At the age of 8 years medial metaphyseal lesion with medial physeal damage. Fair radiographic result at the age of 17 years.



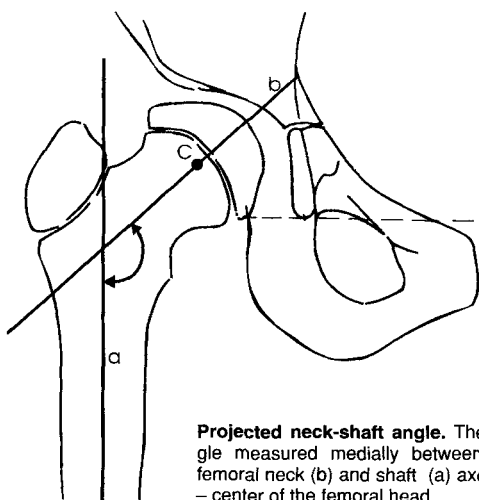
Type V involvement of the epiphysis and metaphysis. At the age of 4 years involvement of the epiphysis and gross metaphyseal irregularity. Poor radiographic result at the age of 17 years.

Method of measurements of angles and calculation of indexes

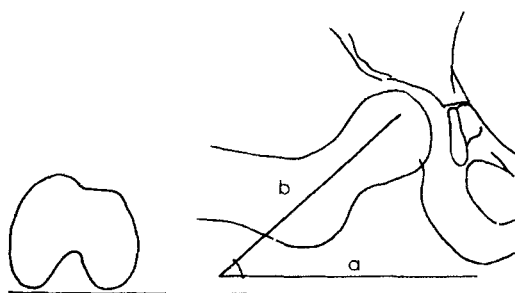
Method of measurements of angles and calculation of indexes are illustrated below. Values of all indexes should be multiplied by 100.

In assesment of femoral head sphericity the Mose's plate with concentric circles of 2 mm increas-

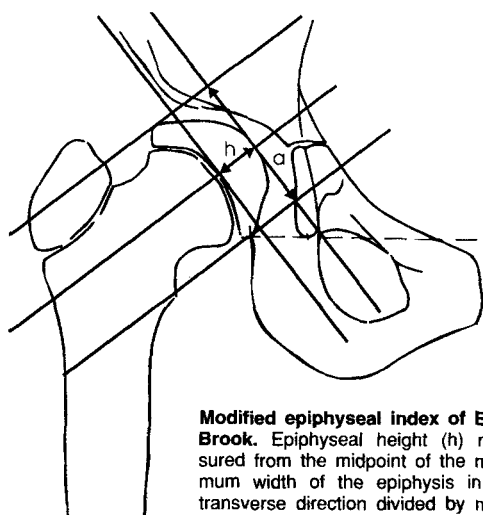
ing radius was used. The head was 'spherical' when one of the circles covered the contours of the joint surface (a 2 mm variation in radial length was permitted); with greater variation the head was classified as 'irregular'.



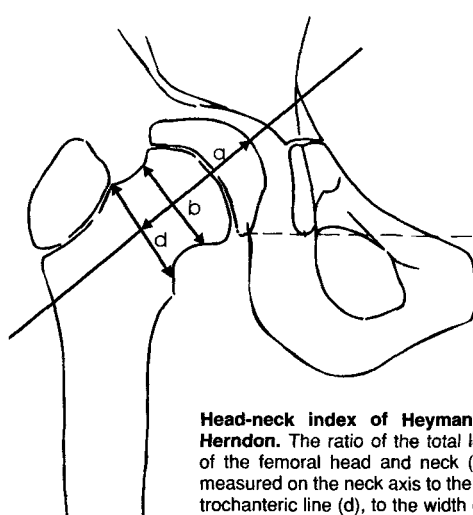
Projected neck-shaft angle. The angle measured medially between the femoral neck (b) and shaft (a) axes; C – center of the femoral head.



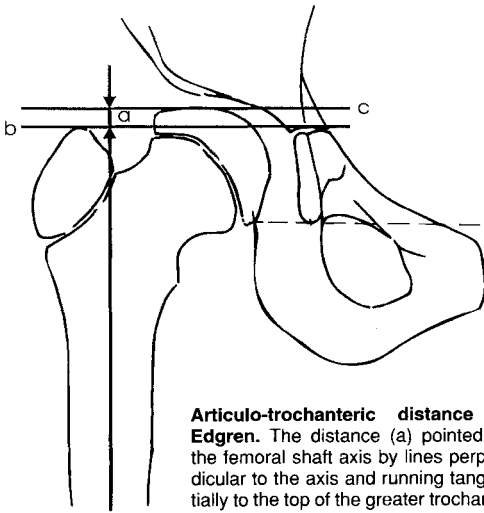
Projected anteversion angle. The angle measured between the femoral neck axis (b) and the horizontal plane defined by the reference bar (a) of the apparatus in which the patient was placed.



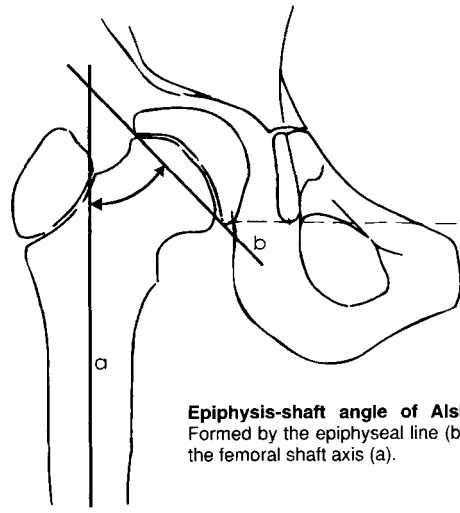
Modified epiphyseal index of Eyre-Brook. Epiphyseal height (h) measured from the midpoint of the maximum width of the epiphysis in the transverse direction divided by maximum width (a) (Tönnis 1984).



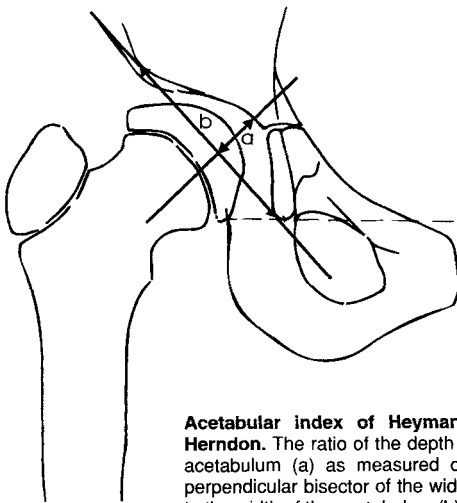
Head-neck index of Heyman and Herndon. The ratio of the total length of the femoral head and neck (a) as measured on the neck axis to the intertrochanteric line (d), to the width of the neck (b).



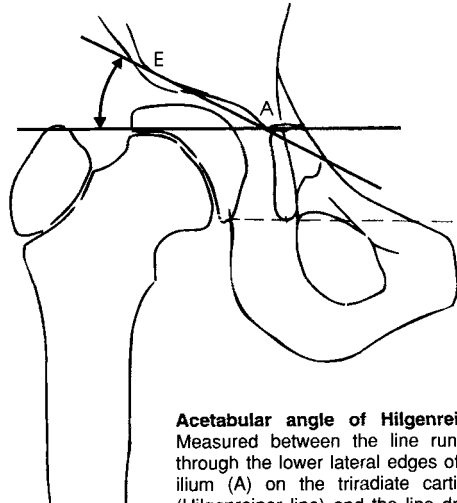
Articulo-trochanteric distance of Edgren. The distance (a) pointed on the femoral shaft axis by lines perpendicular to the axis and running tangentially to the top of the greater trochanter (b) and top of the femoral head (c).



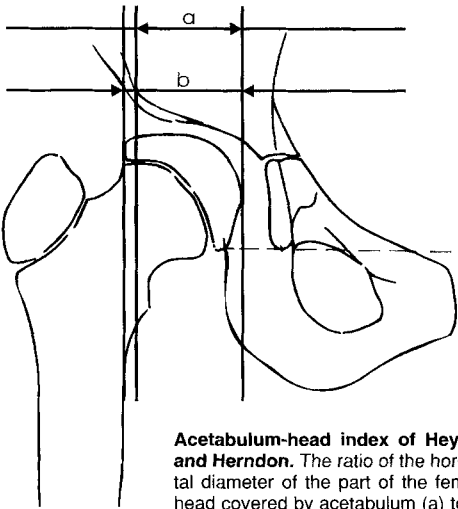
Epiphysis-shaft angle of Alsberg. Formed by the epiphyseal line (b) and the femoral shaft axis (a).



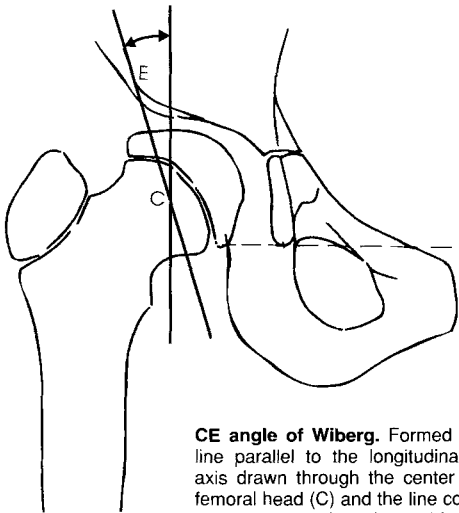
Acetabular index of Heyman and Herndon. The ratio of the depth of the acetabulum (a) as measured on the perpendicular bisector of the width line to the width of the acetabulum (b) measured from the most lateral point of the superior acetabular rim to the lateral edge of the teardrop.



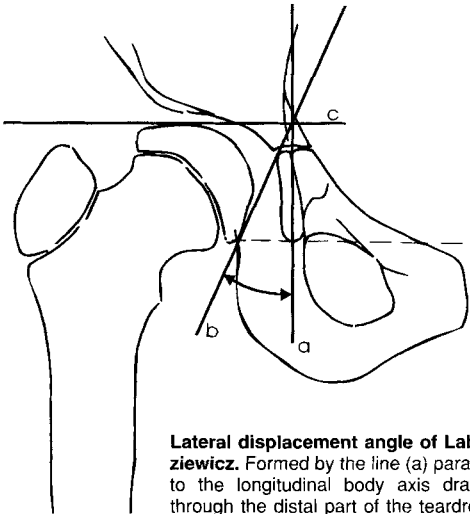
Acetabular angle of Hilgenreiner. Measured between the line running through the lower lateral edges of the ilium (A) on the triradiate cartilage (Hilgenreiner line) and the line drawn through the point on the ilium (A) and the superior acetabular rim (E).



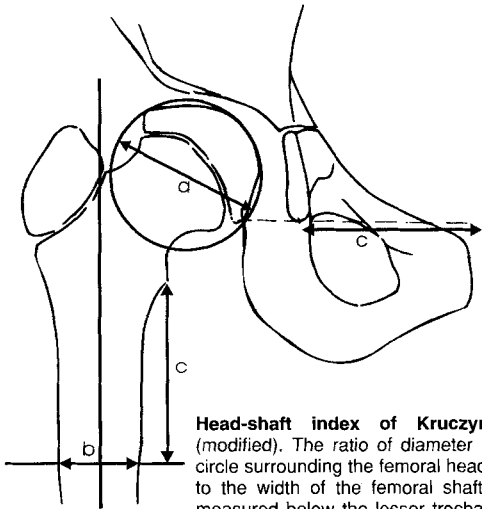
Acetabulum-head index of Heyman and Herndon. The ratio of the horizontal diameter of the part of the femoral head covered by acetabulum (a) to the horizontal diameter of the entire head (b).



CE angle of Wiberg. Formed by the line parallel to the longitudinal body axis drawn through the center of the femoral head (C) and the line connecting the center of the femoral head (C) with the edge of the acetabular roof (E).



Lateral displacement angle of Labaziewicz. Formed by the line (a) parallel to the longitudinal body axis drawn through the distal part of the teardrop, and line (b) drawn from the point of intersection with a horizontal line (c) running through the top of the femoral head, to the medial end of the spina colli.



Head-shaft index of Kruczynski (modified). The ratio of diameter of a circle surrounding the femoral head (a) to the width of the femoral shaft (b) measured below the lesser trochanter at the distance equal half the length between teardrops (c). W 185, SD 12.1