COXA PLANA

A CLINICAL AND RADIOLOGICAL INVESTIGATION WITH PARTICULAR REFERENCE TO THE IMPORTANCE OF THE METAPHYSEAL CHANGES FOR THE FINAL SHAPE OF THE PROXIMAL PART OF THE FEMUR

Translated by Eva Palmgren

To my Family

CONTENTS

ACKNOWLEDGEMENTS

I should like to express my gratitude to my friend and former teacher, Professor C. GÖSTA JANSSON, M.D., who directed my interest to the radiology of skeletal diseases.

The present study was started in 1955. At this time a relatively large number of patients with coxa plana had been registered at the Radiological Department of the Orthopaedic Hospital of the Invalid Foundation, and some primary results of healing could be evaluated. I am deeply grateful to the late Professor FABIAN LANGENSKIÖLD, M.D., the former Head of the Hospital, for his initiative and valuable guidance in the early stage of the work.

Since the autumn of 1956 my work has been followed with interest and encouragement by my present chief, Professor ANDERS LANGENSKIÖLD, M.D. In the evaluation of the various aspects of the radiological course in coxa plana I have thus had the advantage of profiting by Professor Langenskiöld's extensive knowledge on the normal and pathological mechanisms of bone growth. He also revised my manuscript with constructive criticism. For all this generous aid I wish to extend my warm thanks.

My thanks are due to my colleagues at the Orthopaedic Hospital of the Invalid Foundation, who helped me with the radiological examinations and examined the patients clinically.

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Helsinki, March 1965. WALTER EDGREN

I. INTRODUCTION

Among the so-called aseptic necroses, coxa plana occupies a central position owing to the relatively frequent occurrence of the disease and owing to the fact that its location to the hip joint, which is of such great significance to the mobility of human beings, makes it a serious clinical and social problem.

The denomination coxa plana, introduced by H. WALDENSTRÖM in 1920, describes the deformity — a flattening of the femoral head — caused by the disease.

In the extensive literature concerning the radiological course in coxa plana, interest has mainly been focussed on the changes of the femoral epiphysis and the ultimate shape of the latter after the completion of healing. In spite of the great importance of the metaphysis and the greater trochanter for the modelling of the proximal end of the femur and although the trochanter, in addition, influences the statics of the hip joint, less attention has been directed to the changes in these structures.

On inspection of serial radiographs of patients with coxa plana I observed that end-results which were satisfactory from the standpoint of the shape of the femoral head were sometimes markedly impaired because the greater trochanter was abnormally large in relation to the head and neck of the femur. In certain cases the tip of the trochanter extended by several millimetres over the proximal surface of the femoral head.

It seemed a reasonable assumption that this secondary deformity was due to premature closure of the subcapital growth line of the affected hip as compared with the subtrochanteric growth line, in which growth sometimes continued for a long time after the vertical growth of the metaphysis had been arrested.

Furthermore, I observed that certain changes of the metaphysis almost constantly occurred on the radiographs of coxa plana patients. These changes, *i.e.* irregularities of the proximal surface of the metaphysis, were all of the same type, but varied in degree from case to case. They were often marked in

those cases where the growth line closed earlier on the affected side than on the unaffected side.

These observations were made at the same time as my colleague at the Invalid Foundation P. PYLKKÄNEN worked on his monograph on coxa vara infantum. At the suggestion of our chief A. LANGENSKIÖLD, epiphysiodesis of the greater trochanter was performed in some cases of coxa vara in order to arrest the growth of the trochanter and thus produce better anatomical conditions in the proximal end of the femur in cases where the subcapital growth line was considered functional. These attempts were successful in some cases.

Encouraged by the above-mentioned results, I suggested that epiphysiodesis of the greater trochanter should be performed in such cases of coxa plana where the radiographs were indicative of impending premature closure of the subcapital epiphyseal line, with resulting abnormal growth of the trochanter as compared with the shortening of the femoral neck.

II. PROBLEMS

The aim of the present investigation was to find an answer to the following questions:

1. What are the radiological end-results in coxa plana treated mainly on the principle of prevention of weight-bearing on the affected hip, using Thomas' splint as an aid?

2. What is the cause of the radiological changes observable in the metaphysis, epiphyseal plate and adjacent parts of the epiphysis?

3. What is the role of growth arrest of the femoral neck and premature closure of the subcapital epiphyseal line in the development of a disturbing hypertrophy of the greater trochanter?

4. Does coxa plana cause a valgus or a varus deformity?

5. Is it possible, by epiphysiodesis of the greater trochanter, to prevent hypertrophy of the trochanter?

III. REVIEW OF THE LITERATURE

CLINICAL AND RADIOLOGICAL INVESTIGATIONS

Till late in the nineteenth century, the understanding of juvenile diseases of the hip was limited to congenital luxation and coxitis. From the latter, heterogeneous group, disorders of a non-inflammatory nature were, however, distinguished even before the introduction of radiological methods of investigation. In 1881, FIORANI described a typical deformity of the proximal end of the femur, which HOFMEISTER, in 1894, gave the name of coxa vara. In 1897, MAYDL described two cases of arthritis deformans in adolescents. During the next few years, a large number of reports on arthritis, or osteoarthritis deformans coxae juvenalis, were published. Those of HOFFA (1901), v. BRUNN (1903), NEGRONI (1905), PREISER (1907), FRANGENHEIM (1909) and BIBERGEIL (1910) may be mentioned in this connexion because the descriptions and the illustrations suggest that some of these cases probably were coxa plana.

In 1909, SOURDAT published a study on diseases of the hip joint. From a large material he was able to distinguish 8 cases, showing typical clinical symptoms and radiological signs. These cases, by SOURDAT listed as coxa vara, seem to have been coxa plana, as judged on the basis of the description and the drawings.

In the same year (1909), H. WALDENSTRÖM described a disease which he called »der obere tuberculöse Collumherd». On the basis of 10 cases he summarized certain clinical and radiological features which still apply in the main to coxa plana. Owing to the fact that all these patients responded positively to tuberculin, WALDENSTRÖM regarded the disease as tuberculous in nature. The next year (1910), WALDENSTRÖM described the disorder in greater detail. He still regarded it as tuberculous, but he concluded: »Der obere Collumherd ist also eine so charakteristische Erkrankung, dass sie eine selbständige Gruppe der tuberculösen Coxitiden bilden kann».

In 1910, the American LEGG, the Frenchman CALVÉ and the German PERTHES independently of each other described a non-tuberculous disease

of the hip in children, which they had detected and studied during the preceding years. Both the clinical picture and the radiological course of the disease were similar in the three reports and typical of coxa plana. Thus, the disorder may be said to have been definitely recognized as a separate clinical entity.

LEGG's first paper, called »An obscure affection of the hip joint», was based on 5 cases with signs of a disease which he found was not typical of any condition previously described. He gave the following list of characteristic symptoms: age 5 to 8 years, a history of injury, limping, absence of pain, absence of constitutional symptoms, little or no spasm, absence of shortening of the limb. The radiological changes consisted of flattening of the femoral head, an irregular translucent area near the epiphyseal line, shortening and thickening of the femoral neck.

Under the name of »Pseudocoxalgie», CALVÉ described 10 cases of a disorder of the hip which he could not refer to tuberculous coxitis or any other disease of the hip previously known. The most typical clinical and radiological symptoms recorded were: age 3½ to 10 years, chronic or subacute joint symptoms with pain, a limp and reduced mobility of short duration, and signs of rickets. The radiographs exhibited flattening of the epiphysis with two or more ossific centres, hypertrophy of the neck of the femur, coxa vara in some cases, intact joint cartilage. The cases healed with preserved mobility.

PERTHES' first report included 6 cases of the disease, which he called »arthritis deformans juvenilis». He, too, emphasized the occurrence of a limp with or without joint pain and limitation of mobility as the main clinical symptoms. As the first radiological sign discernible he mentioned flattening of the femoral epiphysis and subsequent deformation, frequently cone-shaped.

In 1913, PERTHES published a second investigation: »Ueber Osteochondritis deformans juvenilis», in which 15 new cases were described in addition to the 6 earlier ones. He characterized the disease as »ein durch subchondrale Destruktionsherde bedingter, im Laufe von Jahren sich vollziehender eigenartiger Schwund der oberen Femurepiphyse». The clinical and radiological pictures tallied with his previous observations. PERTHES discussed WALDENSTRÖM's (1909, 1910) reports and stated that, according to his opinion, the »upper tuberculous focus in the collum» described by the latter was osteochondritis deformans juvenilis.

The above-mentioned, first reports were followed by a copious number of papers in which various aspects of coxa plana were discussed. A large

proportion of these papers were short case reports. Among the more important articles, those of LEVY (1911), SCHWARZ (1914), DREHMANN (1914), BRANDES (1914), LEGG (1916), TAYLOR & FRIEDER (1916) and PHEMISTER (1921) may be mentioned. On the basis of a case in which the focus in the femoral head was curetted, the last-mentioned author described the disease histologically as bone necrosis.

In 1920, SUNDT published a monograph under the name of »Malum coxae Calvé-Legg-Perthes», in which various aspects of the disorder were discussed on the basis of 66 cases.

WALDENSTRÖM, in 1922, reported a series of 22 cases which he had followed from the onset till the completion of healing. On the basis of his observations he presented his well-known classification of the different stages of the disease.

STRÅHLE, likewise in 1922, published a survey of the disease and reported his own observations on 5 cases.

RIEDEL (1922, 1923) and AXHAUSEN (1923) described the results of histological investigations.

In a study on non-tuberculous diseases of the hip, FROELICH (1923) reported 7 cases of coxa plana.

In 1924, a monograph by MØLLER, based on clinical and radiological investigation of 74 cases, was published.

CAAN, in 1924, published an extensive survey, mainly based on previous reports by other investigators. KIDNER (1926) and ROCKEMER (1927) described the results of histological studies, which revealed necrosis without any signs of inflammation.

In 1934, FERGUSON & HOWORTH reported observations made in connexion with arthrotomy and drilling of the femoral epiphysis in 21 patients. In the early stage of the disease the synovial membrane was thickened, soft and vascularized. In addition, villus formations, oedamatous and thickened periosteum and normal joint cartilage were found. The authors considered the changes to be suggestive of inflammation. HOWORTH (1948) was able to confirm his previous observations in connexion with 50 later operations. HAYTHORN (1949), who examined the tissues removed from the epiphysis of patients operated upon for coxa plana, observed aseptic necrosis of the bone and bone marrow.

SUNDT (1949) reported the results of a follow-up investigation on 153 cases of coxa plana.

BERNBECK discussed the pathogenesis of coxa plana in papers based on more than 300 cases (1948, 1950 and 1951 a and b).

In 1953, JONSÄTER published a study based on 34 cases representing different stages of the disease, investigated by needle biopsy, and 40 cases examined by arthrography of the hip joint. JONSÄTER summarized his results as follows:

The disease reaches its climax during the initial stage which, roughly speaking, implies total necrosis of the epiphysis. Since haemorrhages do not occur, the necrosis is ischaemic by nature. In this stage the epiphysis is soft. The radiological fragmentation stage is histologically a reparative stage, during which the dense areas on the radiographs correspond to necrotic bone, and the translucent areas to ingrowing reparative tissue and fresh, not yet fully mineralized bone. The epiphysis is harder than in the initial stage. The radiological reparative stage is also histologically a regenerative phase, which differs only in degree from the fragmentation stage. The epiphysis is harder than in the fragmentation stage.

HELBO (1953), too, published a monograph on coxa plana, which was based on clinical and radiological investigation of 204 cases. Of these, 66 were treated by protracted bed rest. They were compared with symptomatically treated or untreated cases. The end-results in the first-mentioned group were very good.

In 1954, GOFF's large monograph on Legg-Calvé-Perthes' syndrome was published, in which heredity, constitutional aspects, growth phenomena and methods of treatment are discussed at length. The end-results in 65 cases are reported.

In the same year, PERTTILÄ reported the results of a follow-up investigation of 33 cases, seen from 10 to 20 years after the active phase of the disease.

In 1957, RYDER *et. al.* reported a series of 104 cases, and KATZ described the end-results in 100 cases.

EVANS, in 1958, published the results of a follow-up study of 48 cases of coxa plana.

WANSBROUGH *et al.* (1959) reported their observations on a series of 129 cases, and O'GARRA (1959) the results of 25 cases.

The most recent contributions to the clarification of the clinical and radiological aspects of the disease have been published by PONSETI & COTTON (1961), RALSTON (1961), BERGSTRAND & NORMAN (1961), GOFF (1962) and MOSE (1964).

Radiological evaluation of end-results. EYRE-BROOK (1936), SJÖVALL (1943) and HEYMAN & HERNDON (1950) used various methods for the measurement of height, flattening and increase in breadth of the femoral head as compared

with the unaffected side. The results were expressed as epiphyseal index, epiphyseal quotient or comprehensive quotient.

Regarding all these methods of measurement GOFF (1954) emphasized the difficulty in defining the points of determination. In addition, he pointed out that they are of no use in bilateral cases, as was also emphasized by HELBO (1953).

GOFF evaluated the shape of the femoral head on direct inspection of the radiograms. He classified the results as *spheroidal type, mushroom type* and *irregular type* (*malum coxae juvenilis*). As an aid in determining the spherical outline of the head, he used a protractor by scratching circles at 2 mm intervals on a piece of transparent acetate.

MOSE (1964) estimated the shape of the outline of the head in the same way as GOFF, and completed this estimate with calculation of the epiphyseal quotient as suggested by SJÖVALL. *Good results* meant that the head was spherical, the outlines in the frontal and lateral views constituting circles with identical radii, and that the epiphyseal quotient was above 60 per cent. *Fair results* implied that the head was spherical, but crescent-shaped and flattened, with an epiphyseal quotient below 60 per cent. *Poor results* comprised all those heads in which the radii of the outline circles differed in the lateral and frontal views, and all heads which were not spherical, irrespective of the quotient.

AETIOLOGY AND PATHOGENESIS

The authors of the first reports on coxa plana differed in their opinions regarding the aetiology of the disease. WALDENSTRÖM considered it to be a mild form of tuberculosis. LEGG believed that it was due to trauma. CALVÉ, who detected signs of rickets in some of his patients, regarded the latter disorder as a possible cause of coxa plana. PERTHES suggested the possibility of an infectious aetiology.

Trauma

Necrosis of the femoral head, resembling coxa plana, is relatively frequent after closed reduction of a congenitally luxated hip. This has been emphasized by many authors as evidence of a traumatic aetiology of coxa plana (EDEN 1912, BIBERGEIL 1912, BADE 1913, BRANDES 1916, LEGG 1916, REHBEIN 1922, HOWORTH & SMITH 1932, LIMA, ESTEVE & TRUETA 1960, and others).

After traumatic luxation, necrosis of the femoral head has been described by ELMSLIE (1919), REHBEIN (1923), NICOLAYSEN (1931), DYES (1935), BLUMENSAAT (1936), GOLDENBERG (1938), LINDEMANN (1957), ULLOA (1963), and others.

In adolescents, necrosis of the femoral head following fracture of the neck has been reported by a number of investigators, *e.g.* AXHAUSEN (1922), REHBEIN (1923), JOHANSSON (1927), KRAFT (1931), LANGE (1932), BORNEBUSCH (1940), BRAILSFORD (1943), BERNBECK (1951 b) and RATLIFF (1962). These authors regarded their observations as evidence in favour of a traumatic aetiology.

HELBO surveyed the cases previously reported in which trauma had been indicated as the cause. Trauma was mentioned in a third of 258 case reports. In the histories of 22 per cent of his own patients HELBO detected trauma of a kind that might have had a causal relationship with the onset of the disease. He regarded trauma as a possible cause of coxa plana.

Inflammatory causes

After WALDENSTRÖM and PERTHES, many authors have suggested the possibility that coxa plana is due to inflammation. DREHMANN (1914), PHEMISTER (1921), BERNBECK (1951 b), HOLLÄNDER (1952) and others have described conditions resembling coxa plana in connexion with inflammatory processes in the hip region, in particular. CALVÉ (1910) and SUNDT (1949) observed coxa plana in association with rheumatoid arthritis. SINDING-LARSEN (1915), AXHAUSEN (1922), RIEDEL (1923) and HOWORTH (1948) described coxa plana in connexion with infections such as tonsillitis, scarlatina and influenza. JACOBS (1960) reported a study of 25 patients with synovitis in the hip joint. In 3 of these the disease later developed into coxa plana. He suggested a causal relationship between synovitis and osteochondrosis as a reasonable hypothesis.

Hereditary and constitutional factors.

A large number of authors have reported a familial occurrence of coxa plana, *e. g.* CALVÉ, SCHWARZ, BRANDES (1920), SUNDT (1949) and MONTY (1962). STEPHENS & KERBY (1946) were able to trace coxa plana through five generations in the same family. They found the disease in 28 out of 63 members investigated and regarded their results as indicative of inheritance according to Mendel's law.

JEQUIER & FREDENHAGEN (1948) described a family in which coxa plana could be traced through seven generations. In 14 cases the diagnosis was verified. These authors regarded their observations as evidence of a dominant hereditary aetiology.

Among 200 patients with coxa plana, HELBO (1953) detected 3.5 per cent who had close relatives suffering from this disease. On comparison of the frequency of coxa plana with the frequency of its familial occurrence, he concluded that the difference was significant and that the large number of familial cases could not be due to chance.

GOFF (1954) found a family history of coxa plana in 20 per cent of his 103 cases. From his own investigation and the data available in the literature he drew the conclusion that coxa plana is a mostly recessive, seldom dominant, condition with varying penetrance.

In a series comprising 129 cases, WANSBROUGH *et al.* (1959) noted a family history of coxa plana in 9.3 per cent. These authors considered the disease to be of constitutional origin and believed that minor traumata and systemic infection may, perhaps, precipitate the onset.

Coxa plana was observed in monozygotic twins by GOFF (1954), GIANNESTRAS (1954), SÖDERBERG (1957), WANSBROUGH (1959), DUNN (1960) and INGLIS (1960).

GOFF was able to demonstrate growth inhibition in his patients, inhibition of the longitudinal growth of the lower extremities in particular. Furthermore, he observed delayed skeletal age in connexion with coxa plana.

RALSTON (1961) reported that the bone age in his series was delayed by an average of 1 year and 9 months.

MAU & SCHMITT (1960) suggested that constitutional dysostotic factors may be responsible for the development of coxa plana.

Endocrine factors

Radiological changes of the hip joint resembling those seen in coxa plana were described in cretins by BIRCHER (1909) and LÄWEN (1909). LOOSER (1929) regarded these changes as due to a retarded transformation of cartilage to bone. ALBRIGHT (1938) reported favourable results with thyroid medication in similar cases.

SUNDT (1920) suggested that the cause of coxa plana is osteodystrophy due to hereditary endocrine factors, and that the onset of the disease is precipitated by trauma or infection.

Thyroid function was studied by GILL (1943) in 20 children with coxa plana, by KATZ (1955) in 32 children, by CHAPMAN (1956) in 10 children and by BEILER & LOVE (1956) in 22. In none of these investigations was hypothyroidism observed.

HOWORTH (1948) found that the basal metabolism was normal in patients with coxa plana.

Nutritional factors

CALVÉ (1910) regarded rickets as a possible cause of coxa plana. SUNDT (1920) reported that a third of his patients had a history of rickets. GOFF (1954) tested patients with osteochondrosis for alkaline phosphatase and serum cholesterol, without being able to detect any significant changes.

SCHNEIDER (1937) demonstrated a decrease in vitamin A in children with coxa plana.

BRAILSFORD (1948) stated that Legg-Perthes' disease is much more common among the poor than among the well-to-do classes. PEIĆ (1962) observed a relatively higher incidence of coxa plana among the children of manual workers. By contrast, GOFF (1954) detected no relation to economic levels or to housing or home environment.

PONSETI (1956) suggested that the disease is due to changes in the chemical composition of the ground substance of the epiphyseal plate. In biopsy specimens of the epiphyseal plate of patients with coxa plana and epiphysiolysis he observed changes resembling those seen in rats fed with a diet rich in aminonitrile. WOROBEC & NORWOOD (1956) arrived at similar conclusions regarding the cause of coxa plana.

Dysplasia of the acetabulum

BRANDES (1920), CALOT (1921), JANSEN (1923) and HILGENREINER (1933) attributed coxa plana to congenital changes of the hip joint. In support of this view, BRANDES described cases in which coxa plana developed in the unaffected hip in children with congenital luxation. Later, many authors (*e. g.* GUILDAL 1930, MORVILLE 1930, 1935, WALDENSTRÖM 1934, SEVERIN 1942, HELBO 1953) have shown that coxa plana occurs in previously entirely normal hips.

Racial factors

GOFF (1954) reported that coxa plana is rare in Negroes, Indians and Polynesians.

Circulatory factors

Many authors have suspected that disturbed circulation in the proximal end of the femur is the cause of coxa plana. Mention may here be made of AXHAUSEN's (1922) theory concerning »bland mycotic embolism».

BERNBECK (1954) regarded coxa plana as osteonecrosis due to total infarction, resulting from blockage of the epiphyseal vessels in the cartilage canals in the chondroepiphysis. He presumed that trauma or infection may lead to degenerative cartilaginous oedema, resulting in obstruction of the canals and arrest of the circulation, and that the borderline lamella between the cartilage and the bone is then penetrated so that the acid cartilaginous fluid makes its way into the bone tissue and causes decalcification, a chemical osteonecrosis.

The investigations by TRUETA (1956, 1957) regarding the supply of the proximal end of the femur are interesting in this connexion. After injection of contrast medium into autopsy material, and on the basis of sections and microradiographs, he studied the circulation in 46 cases from the eighth foetal month to 17 years. It emerged that the vessels in the ligamentum teres do not participate in the supply of the femoral head from birth until the age of 4 years, and that the blood flow through the ligamentum teres is of no importance for the supply of the femoral head until the age of about 8 years. After birth the metaphyseal vessels gradually decrease in size, and at the age of 4, they are of practically no importance for the supply. From the age of 4 to 8 years, with individual variations, the femoral head is exclusively supplied via the lateral epiphyseal vessels. These are exposed to compression by the strong lateral rotator muscles, and in extreme positions the limit of elasticity of the vessels may be exceeded. TRUETA accepts trauma as a precipitating cause, but emphasizes that the nutritional conditions in the femoral head at the coxa plana age are the primary cause of the disease. As further evidence for this theory he points out that in a study on the vascular conditions in the proximal end of the femur in Negro children of the same age, he found that the supply of the femoral head in this race occurs via the ligamentum teres earlier in life than in the white race. This would explain the rare occurrence of the disease in Negro children.

The investigations by ULLOA (1962) regarding the blood supply in the proximal end of the foetal femur confirms TRUETA's results.

The most recent contribution to research on the circulation in the proximal end of the femur is a paper by HIPP (1962), who studied this problem on the basis of angiographies in a normal series and in different pathological condi-

tions. In coxa plana he observed partial obstruction of the proximal nutrient vessels of the femoral head and relatively frequent obliteration of the ramus profundus of the medial circumflex femoral artery. During the progressive stage of the disease, in particular, the blood flow in these vessels was retarded.

EXPERIMENTAL INVESTIGATIONS

Experiments on animals have been performed in order to produce necrosis by blocking or dividing the vessels supplying the femoral epiphysis.

ISELIN (1918) divided the ligamentum teres in young dogs without any necrosis resulting. NUSSBAUM (1923) divided the ligamentum teres and the periosteum around the femoral neck in young dogs. The experiments resulted in necrosis of the head of the femur. BENTZON (1926) injected alcohol into the area surrounding the metaphysis in young rabbits and goats. A condition was caused which histologically resembled coxa plana. BERGMANN (1927) and MILTNER & HU (1933) brought about necrosis of the femoral head in rabbits, the former by removing and the latter by dividing the periosteum of the femoral neck and by dividing the ligamentum teres.

LEMOINE (1957) induced necrosis of the head of the femur in rabbits by division of the ligamentum teres, division of the nutrient vessels to the head and division of the anterior circumflex artery.

ROKKANEN (1962) was able to produce necrosis of the femoral head in rabbits, *e. g.* by tightly ligating the neck with steel wire and by dividing the ligamentum teres.

A. LANGENSKIÖLD *et al.* (1962) studied the changes following experimental dislocation of the hip in young rabbits. After closed reduction of such hips necrosis of the head and the neck of the femur was observed.

NAGURA (1937) and NAGURA & KOSUGE (1938) induced changes resembling coxa plana in rabbits by local, blunt trauma of the femoral head. From his experimental results NAGURA drew the conclusion that an aseptic necrosis is always primarily due to a subchondral fracture.

NORMAL GROWTH OF THE PROXIMAL END OF THE FEMUR

Ossification of the femur begins in the seventh foetal week with ossification of the diaphysis. At birth, or immediately after, an ossific centre develops in the distal end of the bone. In the proximal end of the femur the epiphyseal plate in the newborn child forms a coherent, crescentic line. The medial portion of the epiphyseal plate is transformed into the subcapital

epiphyseal cartilage, which forms the growth zone of the femoral neck. Initially, the neck grows almost straight in the cranial direction, but soon the lateral portion of the pre-plate also develops into the epiphyseal cartilage of the greater trochanter. Growth and modelling of the proximal end of the femur from now on occurs by reciprocal action of these two separate growth zones (MORGAN & SOMERVILLE 1960). The first effect of growth in the area of the trochanter is a decrease of the valgus position and increased growth of the proximolateral part of the bone. In the subcapital epiphysis an ossific centre develops in girls at the age of three to four months and

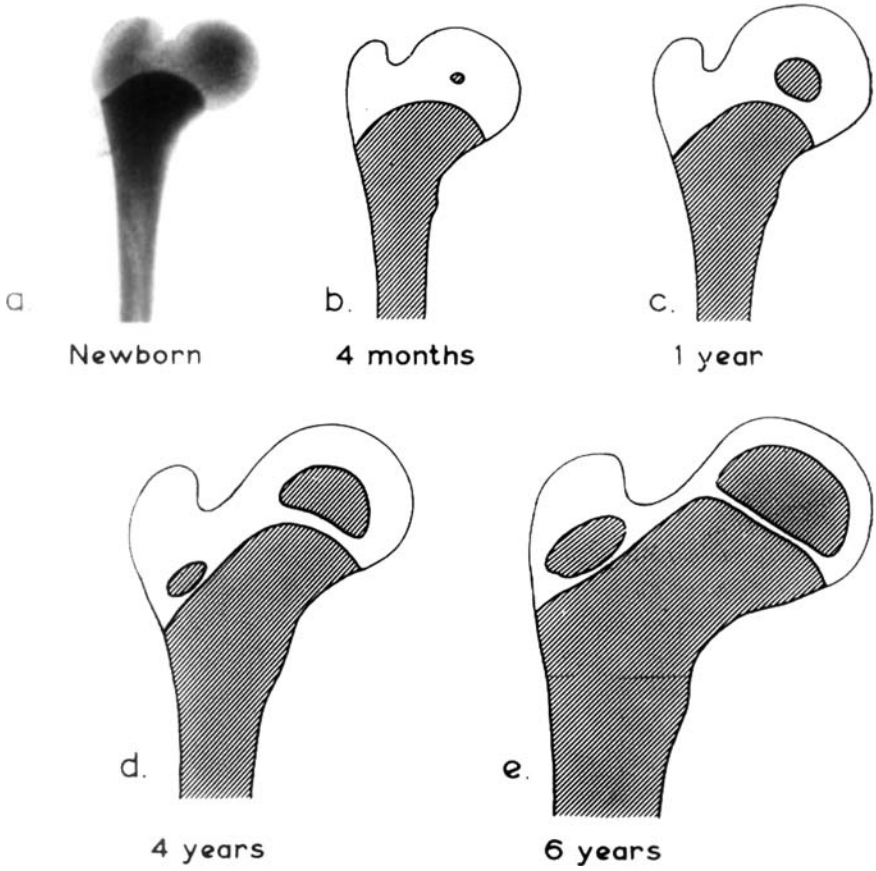

Fig. 1. The transformation of the preplate to separate growth zones for the femoral head and the greater trochanter. The development of the epiphyseal nuclei in the proximal end of the femur. *a:* radiogram of the proximal end of the femur of a still-born foetus (female) weighing 3250 g. *b—e*: drawings made on the basis of radiograms.

in boys at the age of five to six months. In the greater trochanter an ossific centre develops at the age of about four years (SCHMID & HALDEN 1949).

Fig. 1 demonstrates the transformation of the pre-plate, its division into two separate epiphyseal plates and the development of the ossific centres in the femoral head and the greater trochanter.

At the Orthopaedic Hospital of the Invalid Foundation investigations by the tetracycline method are in process regarding the growth of the proximal end of the femur in pigs. These studies have shown that the growth of the greater trochanter occurs in equal parts in the metaphysis and by apposi-

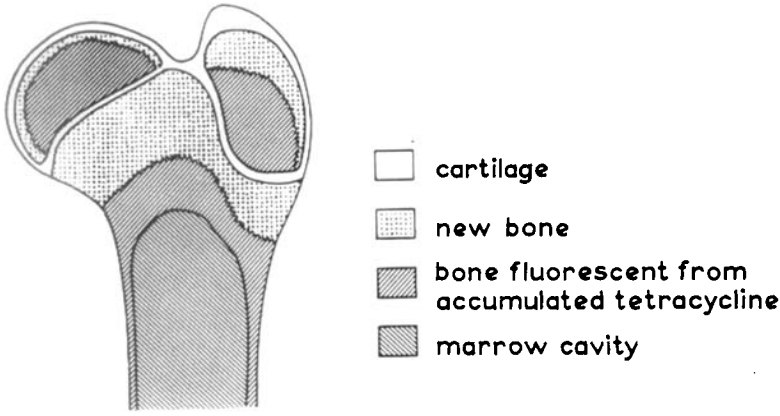

Fig. 2. Drawing of a frontal section through the upper end of the femur of a 14-week-old pig, injected eight weeks previously with tetracycline (50 mg/kg).

tion around the tip of the greater trochanter. The growth of the femoral neck occurs in the metaphysis and is markedly stronger than the growth of the subtrochanteric region. By contrast, apposition is markedly weaker in the capital epiphysis than in the greater trochanter (A. LANGENSKIÖLD & SALENIUS, unpublished observations). (See Fig. 2).

TREATMENT

Previously, the treatment of active coxa plana consisted in purely symptomatic measures, immobilization and/or weight relief by bed rest, by traction or by the use of plaster casts for some period of time (SCHWARZ 1914, BRANDES 1920, and others). Some authors, *e. g.* PERTHES (1913), CALVÉ

(1910) and NIEBER (1916), preferred early mobilization. CAAN (1924) and SUNDT (1949) maintained that the course of the disease and the end-results could not be influenced to any appreciable degree by any kind of treatment.

Later, the importance of protracted non-weight-bearing has been generally accepted, but owing to the long duration of the disease, adequate accomplishment of such treatment meets with considerable difficulties. Practical and social viewpoints have therefore determined the principles on which it is carried out.

WALDENSTRÖM (1923) introduced a compromise therapy: bed rest and traction only in cases where pain and contracture were present, otherwise weight relief by crutches or splints. Different modifications of this treatment have been used by SEVERIN (1942), LEVY & GIRARD (1942), MINDELL & SHERMAN (1951), EVANS *et al.* (1958), WANSBROUGH *et al.* (1959), and others.

BRAILSFORD (1932, cited by BRAILSFORD 1948) and DANFORTH (1934) reported good results after prolonged bed rest. This method in combination with different kinds of traction and continued by weight relief accomplished with crutches or splints has later been employed by a large number of authors, *e. g.* EYRE-BROOK (1937), GILL (1940), SJÖWALL (1943), HERNDON & HEYMAN (1952), HELBO (1953), GOFF (1954), RATLIFF (1956), RYDER *et al.* (1957), O'GARRA (1959), HOWORTH (1959), JACOBS (1960), RALSTON (1961) and MOSE (1964).

Duration of treatment. Since DANFORTH, in 1934, recommended non-weight-bearing for three to four years, views have changed somewhat on this point. Many modern authors regard one and a half to two years' weight relief as adequate. HAUGE (1956) treated his patients by non-weight-bearing for one year, but after comparison with the end-results in series treated in the same way for longer periods, he recommended weight relief for one and a half years. In his first study (1954) GOFF reported that the mean non-weight-bearing period in his series had been 27 months, but later (1959) he reduced this time to nine months combined with crutch-walking for further six months. EVANS (1958) used an average of 25 months' non-weight-bearing.

Regarding the treatment of bilateral cases, the majority of authors — even those who prefer ambulatory treatment in unilateral cases — consider hospitalization to be the only possible alternative. Bed rest is continued until either hip is trusted to tolerate weight-bearing, and then the patient is allowed to walk with the worse side in a caliper (MINDELL & SHERMAN 1951, WANSBROUGH *et al.* 1959, and others). MCKENDRY *et al.* (1960) described a brace for the treatment of bilateral cases of coxa plana, which they had used

successfully. GRIPENBERG & WALLGREN (1963) reported good results with Thomas' splints in bilateral coxa plana.

Previously, operative treatment was only resorted to in poorly healed cases, in part as a remodelling measure, in part in the form of resection of the femoral head or osteotomy for the correction of malposition (CAAN 1924).

FRÜND (1922) described 3 cases of severe deformation after coxa plana, with positive Trendelenburg signs, in which he successfully excised the joint capsule ventrolaterally, removed osteophytes by chiselling, and shifted the greater trochanter a couple of centimetres in the distal direction.

In more recent years, various procedures have been employed in order to damage the necrotic epiphysis and thus bring about its revascularization and more rapid healing. BOZSAN (1932), FERGUSON & HOWORTH (1934), v. ABERLE-HORSTENEGG (1941), HOWORTH (1948), DUBOIS (1951) and KIENZLE (1953) used drilling of the epiphysis through the greater trochanter or the femoral neck. Drilling and grafting were used by HACKENBROCH (1941), STUPNICKI (1952), BERTRAND (1954), CAMARGO (1957) and YAMAGUCHI (1959). BERNBECK (1948) and GARDEMIN (1951) evacuated the necrotic portion of the epiphysis. CATHRO & KIRKALDY-WILLIS (1963) plugged the evacuated epiphysis with spongy bone from the femoral neck or the crista iliaca, and PITZEN (1951), PETER (1955) and SALVO LECARRE (1957) drove a metal nail into the epiphysis through the greater trochanter and the femoral neck.

Subtrochanteric osteotomy was employed by SOEUR & DE RACKER (1952) and SLAVIK (1956) in order to alter the weight-bearing surface of the epiphysis.

With regard to medication, accelerated healing with thyroid preparations has been reported by EDBERG (1918), MØLLER (1924), CAVANAUGH *et al.* (1936), EMERICK *et al.* (1954), FIELDS (1959), and others, while PONSETI & COTTON (1961) observed no effect of thyroid medication.

GOFF (1954, 1959) used aureomycin and achromycin in coxa plana in order to promote growth.

Androgenous and oestrogenous hormones in combination with thyroid preparations were administered by GUÉRITÉE *et al.* (1959). KOSKINEN (1959), who noted a significant effect of human growth hormone and thyrotropin on the healing of fractures in laboratory animals, suggested the use of this treatment also in aseptic necroses. KRISTENSEN (1963) recommended anabolic steroids in the treatment of coxa plana.

IV. MATERIAL AND METHODS

MATERIAL

The material consists of all cases of coxa plana registered at the Radiological Department of the Orthopaedic Hospital of the Invalid Foundation during the years 1946—1958. The total number of cases is 276. Fifty of these were bilateral, and the total number of affected hips is thus 326. The series includes 22 patients who were adult at the time of their first visit. In these cases the process had already reached its final stage. The annual number of registrations is shown in Table 1.

TABLE 1. — *Annual number of patients with coxa plana registered at the Orthopaedic Hospital of the Invalid Foundation during the years 1946—1958 (total 276).*

Year	No. of cases	Adult at the first examination
1946	6	2
1947	3	—
1948	7	—
1949	23	5
1950	23	4
1951	32	2
1952	28	—
1953	21	2
1954	35	1
1955	28	3
1956	18	—
1957	23	2
1958	29	1
Total	276	22

Of the present cases, 246 had been remitted to the Radiological Department from the Outpatient Department of the Orthopaedic Hospital and 30 from the private practices of the doctors on the hospital staff.

The distribution of the cases geographically and according to residence and family environment is seen in Tables 2, 3 and 4.

The small number of cases registered during the first three years (Table 1) is accounted for by the exceptional conditions prevailing after the war, when the work in this hospital mainly consisted of the care of the war-wounded, and the means of communication were deficient.

Table 2 shows that the geographical distribution of the coxa plana pa-

TABLE 2. — *Geographical distribution of 274 cases of coxa plana compared with the distribution of the whole population in Finland in 1952.*

Province	Coxa plana		Whole population
	No. of cases	%	%
Nyland (Uusimaa)	63	23.0	17.0
Åbo & Björneborg (Turku & Pori)	37	13.3	15.5
Åland (Ahvenanmaa)	1	0.4	0.5
Tavastehus (Häme)	41	15.0	13.5
Kymmene (Kymi)	24	8.8	8.0
St. Michel (Mikkeli)	20	7.3	6.0
Kuopio	29	10.6	11.5
Vasa (Vaasa)	26	9.5	15.0
Uleåborg (Oulu)	21	7.7	9.0
Lappland (Lappi)	12	4.4	4.0
Total	274	100.0	100.0

tients corresponded fairly well with the distribution of the whole population over the different provinces of Finland. A slight preponderance of patients from the provinces of Nyland and Tavastehus is accounted for by proximity to the capital. The relatively smallest number of patients had been remitted

TABLE 3. — *Distribution of 274 cases of coxa plana according to residence, compared with the corresponding distribution of the whole population in 1952.*

Residence	Coxa plana		Whole population %
	No. of cases	%	
Towns and market-towns ..	98	35.8	34.0
Rural districts	176	64.2	66.0
Total	274	100.0	100.0

TABLE 4. — *Distribution of 264 cases of coxa plana according to social background, compared with the corresponding distribution of the whole population in 1950.*

Father's occupation	No. of cases	% of cases	Distribution of children in the whole population according to father's occupation, %
Workers	127	48	43
Farmers	71	27	36
Clerical or non-manual, managerial/professional	66	25	21
Total	264	100	100

from the province of Vasa (9.5 per cent against 14.6 per cent). This is due to the fact that two specialists on orthopaedics were practicing in this province during a great part of the time the present series was collected.

As is seen in Table 3, the present study did not reveal any difference in the frequency of coxa plana between the rural and urban populations.

In this series no particular social group seemed to be especially affected with coxa plana (Table 4).

METHODS

Clinical examination

The patients remitted from the Outpatient Department were clinically examined there. As a rule, gait and Trendelenburg's sign, mobility of the hip joints and the length of the lower extremities were recorded. In addition, the erythrocyte sedimentation rate was determined in many cases, and in some the protein-bound iodine in serum. No other laboratory tests were regularly performed. The data regarding these examinations were obtained from the records of the Outpatient Department, which unfortunately in many cases were incomplete.

The clinical data regarding the private patients were obtained from the respective doctors. In some cases I examined the patients myself.

When the material was collected in 1956, I found that no less than 148 patients had to be summoned to follow-up examinations. The majority of these had visited the Outpatient Department but failed to attend at the follow-up examinations. In a minor proportion of cases the treatment had been regarded as finished. Of the 148 patients 100 attended at follow-up investigations during the years 1958—1961. In about 10 cases no contact could be established, and the remainder failed to attend.

Radiological examination

All patients were radiologically examined. Among the earliest cases there are some in which radiographs were taken in only one projection, and a few where only the affected hip was examined.

On radiological examination of a chronic disease of the hip such as coxa plana, which necessitates repeated check-ups, it is of paramount importance that a standardized method is employed, so that the results of one examination are comparable with those of the next. Since 1946, a modification of the method suggested by KNUTSSON (1938) has been used in the radiological examination of the present patients as well as in other examinations of the hip performed in this Department. As a rule, both hips are simultaneously radiographed on the same film. Frontal views are taken with the patient supine, with the legs extended in 20 degrees inward rotation. In this position the neck of the femur is parallel with the film, broadly speaking, since according to v. LANZ (1950) the antetorsion angle of the femoral neck is about 20 degrees in the coxa plana age.

Lateral views are also taken with the patient supine, with about 25 degrees flexion and 50—60 degrees abduction of the femora, which are supported with wedges of sponge plastic. The knees are bent and the feet placed against each other in the median line. A maximum of symmetry is aimed at. If the pelvis is slanting owing to atrophy of the gluteal muscles, this is corrected with cushions. The central ray is focussed immediately above the symphysis. The target — film distance is 110 cm.

In cases with contracture of the hip joint, in which limited abduction makes a simultaneous examination of the two hip joints impossible in the lateral projection, these are separately studied. By keeping the pelvis aslant the desired position is achieved.

The radiographs obtained by this technique have proved satisfactory.

During the period of non-weight-bearing treatment, check-up examinations were made at intervals of three to four months, in some cases at shorter intervals. In a large number of cases the intervals between the check-ups were much longer, owing to failure of the patient to attend at the time arranged. During the last few years this drawback has been eliminated to a great extent thanks to the work done in our hospital by nurses specially trained in social care.

The first follow-up examination after the completion of non-weight-bearing treatment was made after four to six weeks. Subsequently the intervals between the examinations were gradually prolonged to one and two years at the time of completed growth.

In 50 unilateral cases orthoradiographic measurement of the length of the lower extremities was performed in connexion with the last follow-up examination.

In 12 cases the proximal end of the femur was tomographically examined in order to enable better assessment of the condition of the epiphyseal plate.

Protection against radiation

Since 1947, the patients' gonads have been protected with a lead rubber plate or a lead capsule. At the first examination the frontal view has been taken without any protecting device in order to obtain a general view of the whole pelvic skeleton.

Radiological evaluation of the end-results.

The end-results were assessed by a modification of GOFF's (1954) method.

The evaluation of the end-results was based on direct inspection of radiographs. The shape of the femoral head, the outline of the latter, the length and breadth of the femoral neck, perfection of fit with the acetabulum, the possible presence of persisting bodies and the degree of joint reactions were taken into account.

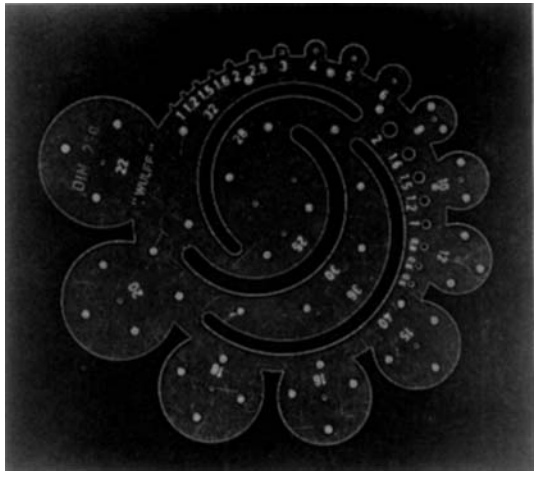

Fig. 3. Transparent plastic protractor, used in the evaluation of the shape of the femoral head and in measuring the radius of the outline in spherically healed cases.

In order to facilitate assessment of the shape of the femoral head the plastic protractor seen in Fig. 3 was used.

With the protractor placed on the radiograph the fit of the femoral head in one of the circles was estimated. If the head was spherical the length of the radius was measured. In unilateral cases the same procedure was carried out on the unaffected side.

Furthermore, the length of the femoral neck and the breadth of the metaphysis were measured in both hips in unilateral cases.

V. CLINICAL AND RADIOLOGICAL OBSERVATIONS

GENERAL CONSIDERATIONS

Sex distribution. Table 5 shows the sex distribution in the present series. The ratio males to females is 4:1.

TABLE 5. — *Sex distribution in 276 cases of coxa plana.*

	No. of cases	%
Males	223	80.8
Females	53	19.2
Total	276	100.0

Affected side. The distribution according to affected hip in this series is shown in Table 6.

TABLE 6. — *Distribution according to affected side in 276 cases of coxa plana.*

Sides	Males		Females		Total	
	No. of cases	%	No. of cases	%	No. of cases	%
Right.....	84	37	21	40	105	38
Left	97	44	24	45	121	44
Both	42	19	8	15	50	18
Total	223	100.0	53	100.0	276	100.0

Body build. The body build was subjectively assessed by inspection in 215 cases. The results are seen in Table 7.

In the cases exhibiting obesity the sella turcica was radiologically examined. No pathological changes were observed.

TABLE 7. — *Body build in 215 cases of coxa plana.*

	No. of cases	%
Normal	183	85.1
Slender	24	11.2
Stout	8	3.7

TABLE 8. — *Age at onset in 265 cases of coxa plana.*

Age in years	Males	Females	Total	%
2— 3	6	6	12	4.5
4— 5	61	10	71	26.8
6— 7	82	22	104	39.3
8— 9	41	8	49	18.5
10—11	21	5	26	9.8
12—14	3	—	3	1.1
	214	51	265	100.0

Age at onset. The patient's age at the time when the first symptoms — a limp and/or pain — were observed was regarded as the onset age. This could be established in 265 cases, as shown in Table 8.

The youngest patient was 2 years and 8 months and the oldest was 14 years old at the time of onset of the disease.

It is noteworthy that the ratio males to females is 1:1 in the youngest age group. The small number of cases does not allow of any conclusions, however.

In 11 cases the age at onset could not be established.

Duration of symptoms. The duration of symptoms at the time of diagnosis was calculated from the onset. If the diagnosis had been made at some other hospital and adequate treatment had been instituted, this point of time was regarded as the limit of the duration of symptoms. If adequate treatment had not been instituted elsewhere, this period was reckoned up to the time when the disease was diagnosed in our Hospital. Table 9 shows the duration of symptoms in the present series.

Twenty-two ultimately healed cases are omitted from Table 9. The longest duration groups consist of neglected cases, the majority of which were in a very advanced stage of reconstruction when first examined. It is seen in the table that treatment had been instituted within half a year after the onset of symptoms in less than half the cases.

TABLE 9. — *Time from onset of symptoms to diagnosis in 254 cases of active coxa plana.*

Duration of symptoms in months	No. of cases	%
0— 1	39	15.4
2— 3	43	16.9
4— 5	28	11.0
6— 7	39	15.4
8—12	32	12.6
13—24	49	19.3
25—36	24	9.4
Total	254	100.0

Mean duration 9.6 months.

Stage of the process at the time of diagnosis. On the basis of the radiographic findings at the time of diagnosis the present series was divided into groups according to JONSÄTER's (1953) modification of WALDENSTRÖM's (1923) classification. The distribution of the cases is shown in Table 10.

TABLE 10. — *Distribution according to stage of the process at the time of diagnosis in the whole series.*

Stage	No. of hips in unilateral cases	No. of hips in bilateral cases	Total no. of hips	%
Initial	101	46	147	45.1
Fragmentation	75	35	110	33.7
Reparative	33	8	41	12.6
Definitive	17	11	28	8.6
Total	226	100	326	100.0

The group »definitive stage» includes 6 bilateral cases. In one of these, the outline of one femoral head showed defects which will probably disappear later. This hip was included in the group »reparative stage». If the cases in the group »definitive stage» are omitted, the cases diagnosed in the initial stage constitute only 48.3 per cent of the series.

TREATMENT

At the Orthopaedic Hospital of the Invalid Foundation coxa plana has been conservatively treated. The main principle of treatment has been the prevention of weight-bearing on the affected hip. In unilateral cases weight

relief has been accomplished with Thomas' splint and elevation of the opposite shoe. In bilateral cases where non-weight-bearing on both hips has been conditional, this has been secured by bed rest. One of the present patients was treated by bilateral Thomas' splints. Until 1957, patients were hospitalized only if the diagnosis was uncertain or if the joint exhibited marked contracture. Since 1957, the pattern of treatment has been as follows: Hospitalization for three to eight weeks, during which the patient was kept in bed. Traction was used if contracture of the hip was present. A Thomas' splint was manufactured, fitted and adjusted. Towards the end of the hospitalization period the patient practiced walking with the splint, the fit of which was further adjusted. When the patient had learned to walk with a Thomas' splint, he was discharged and his parents were informed as to the importance of strictly following the prescriptions regarding the use of the splint. Subsequent follow-up examinations were done at the Outpatient Department. Exceptions from this routine were made in 1958, in particular, when unfortunately for certain administrative reasons admission to the hospital was difficult in many cases.

Table 11 shows the kind of treatment prescribed in the present cases. The first group comprises the patients registered during the first few years covered by this study and some bilateral cases from more recent years. At a later follow-up examination of these cases it was found that the treatment prescribed had often been so inadequately carried out that no major influence on the course of the disease can be accorded to it. These cases were classified as untreated (cf. p. 41).

TABLE 11. — *Treatment prescribed at the Orthopaedic Hospital of the Invalid Foundation.*

	No. of cases
Bed rest and/or crutch-walking at home	29
Bed rest with traction at home	4
Thomas' splint	190
Hospitalization + Thomas' splint	27
Total	250

In a number of cases diagnosed elsewhere, the patients had received some kind of treatment, as shown in Table 12, before being seen at the Orthopaedic Hospital of the Invalid Foundation.

It is seen in Table 12 that Thomas' splint had been prescribed elsewhere in very few cases.

TABLE 12. — *Treatment before the first examination at the Invalid Foundation.*

	No. of cases
Sparing use of limb, crutches	22
Bed rest	44
Traction	17
Plaster cast	21
Thomas' splint	8
Total	112

If the non-weight-bearing treatment indicated in Table 12 was regarded as adequately carried out, it was included in the data regarding the duration of such treatment.

Non-weight-bearing treatment was continued until reconstruction had advanced so far that the radiological appearance of the epiphysis was almost uniform and a large proportion of the epiphysis showed a normalized structure on the radiographs.

The duration of non-weight-bearing treatment is seen in Table 13.

TABLE 13. — *Duration of non-weight-bearing in 194 cases.*

Years	No. of cases
1	32
1—2	85
2—3	62
3—4	14
over 4	1

Mean duration of non-weight-bearing 2.3 years.

After the completion of non-weight-bearing treatment the patients were told not to strain their hips for a further two or three years, or until primary healing had been achieved. During this time, school gymnastics and athletics were forbidden.

As a rule, Thomas' splint was abandoned gradually, being used during half the day, for instance at school, while the child was allowed to walk without it during the rest of the time.

Not in a single case have we had to revoke the decision to let a patient abandon Thomas' splint. The radiological check-ups invariably showed that the epiphysis had tolerated the strain. Usually the very first follow-up examination showed increased reconstruction.

END-RESULTS

In the present study the time from the onset of the first symptoms until the last (or only) radiological examination is indicated in Table 14.

TABLE 14. — *Time from onset to last or only examination in 276 cases of coxa plana.*

Years	No. of cases	Years	No. of cases
1	18	9	20
2	12	10	23
3	15	11	16
4	20	12	10
5	26	13	9
6	35	14	1
7	27	15	4
8	24	16 or more	16

The longest period between the onset of symptoms and the last radiological examination was 36 years.

Primary healing is used in the present series to denominate the condition prevailing when the reparative process in the femoral epiphysis had advanced so far that the latter had attained its final shape, and the bony structure of the epiphysis had been normalized. In this stage the epiphysis was uniform, provided that no loose bodies remained, as in osteochondrosis dissecans.

Ultimate healing is used to denominate the stage when growth was completed.

Since the *shape of the epiphysis* does not change to any appreciable degree after primary healing has taken place, that is during the remainder of the growing period (WALDENSTRÖM 1923), the cases showing primary and ultimate healing were united when the end-results in the femoral head were assessed.

Definition of the degree of healing. The femoral head was regarded as spherical (*good result*) if its outline could be precisely fitted into one of the circles of the plastic protractor, and if the radii of the circles formed by the outline on the frontal and lateral views did not differ from each other by more than 1 mm. A difference of 1 mm between the radii on the frontal and lateral views was noted on the unaffected side in many cases.

The femoral head was evaluated as elliptical (*fair result*) if its outline was regularly convex without fitting into any of the circles of the protractor, and if the difference between the radii of the outlines on the frontal and lateral views exceeded 1 mm.

The femoral head was evaluated as irregular (*poor result*) in those cases where it could not be regarded as spherical or elliptical.

In the present series a total of 165 treated hips in 119 unilateral and in 23 bilateral cases showed either primary or ultimate healing.

TABLE 15. — *Relationship between radiological end-results and age at onset in 165 treated hips.*

End-results	Onset age in years										Total no. of hips	%
	2—3		4—5		6—7		8—9		10—14			
	No	%	No	%	No	%	No	%	No	%		
Good	10	90.9	31	64.6	30	49.2	10	31.2	—		81	49.1
Fair	1	9.1	13	27.1	14	22.9	6	18.8	1	7.7	35	21.2
Poor	—		4	8.3	17	27.9	16	50.0	12	92.3	49	29.7
	11	100	48	100	61	100	32	100	13	100	165	100

TABLE 16. — *Relationship between radiological end-results and duration of symptoms before treatment in 165 treated hips.*

End-results	Time in months												Total no. of hips	%
	0—1		2—3		4—5		6—7		8—12		13—24			
	No	%	No	%	No	%	No	%	No	%	No	%		
Good	23	74	21	65.6	9	45	16	51.6	4	18.2	8	27.6	81	49.1
Fair	6	19.5	3	9.4	8	40	5	16.2	3	13.6	10	34.4	35	21.2
Poor	2	6.5	8	25	3	15	10	32.2	15	68.2	11	38	49	29.7
	31	100	32	100	20	100	31	100	22	100	29	100	165	100

TABLE 17. — *Relationship between radiological end-results and stage of the process at the institution of treatment.*

End-results	Stage						No. of hips
	Initial		Fragmentation		Reparative		
	No. of hips	%	No. of hips	%	No. of hips	%	
Good	55	56.7	25	39.0	1	25.0	81
Fair	18	18.6	16	25.0	1	25.0	35
Poor	24	24.7	23	36.0	2	50.0	49
Total	97	100.0	64	100.0	4	100.0	165

In Tables 15, 16 and 17 the radiological end-results are correlated with onset age, duration of symptoms and stage of the process at the institution of treatment.

Enlargement of the femoral head. In order to obtain an idea of the enlargement of the femoral head in those cases where it healed as spherical, the radii of the outline circles of both heads were compared in unilateral cases. To this end the cases were divided into primarily healed (42) and ultimately healed (21). In no case was the radius of the affected head smaller than that of the unaffected head. In the group of primarily healed cases there were 7 with the same radius on both sides. The difference between the two radii varied between 1 and 5 mm, the mean being 2.6 mm. In the group of ultimately healed cases, one showed the same radius on both sides. The difference varied between 1 and 10 mm, the mean being 3.7 mm.

In the primarily healed group the radii of the affected heads exhibited a mean enlargement of 11.9 per cent, as compared with the unaffected heads. The corresponding figure for the ultimately healed group was 13.6 per cent. The corresponding mean increases in the volume of the head were 38.5 and 46.4 per cent, respectively.

Discussion. This analysis demonstrates the well known fact that the head of the femur in a coxa plana hip as a rule is enlarged, as compared with the unaffected side.

In the present analysis the flattening of the epiphysis was disregarded, while in the above-mentioned calculations of index it has been taken into account. It is very difficult, however, to estimate the degree of flattening of the epiphysis, since the height of the latter shows a wide variation even in spherically healed cases owing to a cranially directed convexity in the metaphyseal plane. In these cases the height of the epiphysis is hardly of any practical significance, however. It is the shape of the head that is decisive, irrespective of whether it is a hemisphere consisting of the epiphysis alone, or consists of a crescent-shaped epiphysis, the basal interior portion of which is filled out by the metaphysis.

Untreated cases. The present series includes both entirely untreated coxa plana hips and cases in which the treatment had been so recently instituted or so inadequately carried out that no therapeutic significance can be accorded to it. The primary or ultimate healing could be evaluated in 78 such hips (67 cases). The end-results are shown in Table 18.

The surprisingly large number of good results in this group is accounted for by the fact that it includes 4 very slight, abortive unilateral cases and 12 bilateral cases in which the better, untreated hip exhibited slight, abortive

TABLE 18. — *Relationship between radiological end-results and age at onset in 78 untreated hips.*

Onset age in years	Good	Fair	Poor	No. of hips
2— 3	1	—	1	2
4— 5	7	1	4	12
6— 7	5	5	15	25
8— 9	1	2	12	15
10—11	3	—	6	9
12—14	1	1	2	4
unknown			11	11
Total	18	9	51	78
Per cent	23	11	66	100

changes. Only two of the untreated cases which healed with spherical femoral heads showed fragmentation of the epiphysis.

If these abortive cases are omitted, the end-results in the untreated group are as follows:

Good 3.2 per cent Fair 14.5 per cent Poor 82.3 per cent

When these results are compared with the end-results in the treated group (Table 15), the difference is striking.

Cases under treatment. The series includes 61 patients (83 hips) who are still under treatment or have failed to attend at follow-up examinations. In these cases it is not possible to say anything regarding the end-result.

Special factors affecting the end-results

In the present series the following factors were found to affect the end-results:

Osteochondrosis dissecans	in	16	cases	(3	untreated)	
Subluxation	»	45	»	(30	»	)
Secondary arthrosis	»	20	»	(17	»	)

Comparison of the present observations with previous reports

Sex incidence. The data of different authors agree in that coxa plana is commoner in boys than in girls. In Table 19 the relevant figures from some of the more extensive series are indicated.

TABLE 19. — *Sex incidence of coxa plana in other series.*

Investigator	No. of cases	Males %	Females %
Levy et al. (1942)	102	91.0	9.0
Bernbeck (1951 a)	369	77.0	23.0
Sundt (1949)	153	78.0	22.0
Kite et al. (1952)	165	86.0	14.0
Helbo (1953)	204	77.4	22.6
Goff (1954)	103	83.0	17.0
Ryder et al.	104	82.2	17.8
Evans (1958)	52	75.0	25.0
Wansbrough et al. (1959)	129	81.4	18.6
Carpenter (1960)	90	84.5	15.5
Peić (1962)	189	79.4	20.6
Mose (1964)	257	80.6	19.4

The sex incidence in the present series — 80.8 per cent males and 19.2 per cent females — is in good agreement with the figures in Table 19.

TABLE 20. — *Distribution of coxa plana according to affected side in other series.*

Investigator	No. of cases	Right side %	Left side %	Both sides %
Sundt	153	40.5	47.0	12.5
Helbo, survey of the literature	250	57.0	38.0	5.0
Helbo, own series	204	48.0	44.6	7.4
Goff	103	42.5	40.0	17.5
Ryder et al.	104	42.3	42.3	15.4
Evans	58	48.2	41.5	10.3
Wansbrough et al.	129	?	?	17.8
Peić, male patients	150	56.0	38.7	15.3
Peić, female »	39	33.4	56.4	10.2
Mose	257	?	?	10.9

Affected side. The observations regarding the affected side in certain earlier series are compiled in Table 20. Most reports show a slight preponderance for the right side. SUNDT had slightly more left-sided than right-sided cases, while the two sides were equally represented in RYDER's series. PEIĆ observed a preponderance of right-sided lesions in the male patients, while the female group showed a left-sided preponderance.

In the present series a slight preponderance was noted for the left side, *i.e.* 44 per cent against 38 per cent right-sided, while the proportion of bilateral cases was 18 per cent (Table 6). Contrary to PEIĆ's finding, there was no difference between males and females in regard to the frequency of right-sided and left-sided lesions.

The frequency figures for bilateral cases indicated in the literature show a wide variation (Table 20). This may, perhaps, be accounted for by the fact that in bilateral cases the lesion in one hip is often very slight and may escape recognition, for instance if satisfactory lateral views are not available.

Age at onset. In the present series (Table 8) there was an obvious accumulation of cases in the age group 6—7 years, which was represented by 39.3 per cent.

STÅHL (1948) calculated the onset age by reducing the age at the first radiological examination by an average of three, nine or eighteen months, depending on whether the process was in the initial, fragmentation or early reparative stage at the time of the first examination. According to these calculations, the mean age at onset was 6.6 years in STÅHL's series comprising 103 hips. In other series published, the maximum frequency varies to some extent. The following authors, for instance, have observed a peak for the age groups as listed here: BERNBECK (1951 a) 8 years, WANSBROUGH *et al.* (1959) 7 years, GOFF (1954) 6 years, HELBO (1953) and PONSETI & COTTON (1961) 5—6 years, O'GARRA (1959), HERZOG (1961) and MOSE (1964) 5 years, PEIĆ (1962) 4—6 years.

Duration of symptoms from onset to diagnosis. It is seen in Table 9 that the duration of symptoms from onset to diagnosis was an average of 9.6 months in the present series.

HELBO (1953) described a group of 66 patients with coxa plana in which the duration of symptoms was an average of 3.9 months in 39 cases, an average of 6 months in 6 and 10.1 months in 21. In EVANS' (1958) series the duration was one to 36 months, the mean being 6 months. CARPENTER & POWELL (1960) indicated a mean duration of symptoms of 7 months and 24 days. In a series of 107 patients with coxa plana described by RALSTON (1961), treatment could be instituted 3 months after onset in 49 cases (46 per cent). In the series described by WANSBROUGH *et al.* (1959), comprising 106 cases, fifty per cent were diagnosed within 3 months, and in two-thirds the diagnosis was made within 6 months after onset.

Stage of the process at diagnosis. As is seen in Table 10, slightly less than half the present cases of active coxa plana were in the initial stage at the time when the diagnosis was made, 33.7 per cent were in the fragmenta-

tion stage, and 15.8 per cent exhibited more or less advanced reconstruction.

In HELBO's (1953) series of 66 patients treated by protracted bed rest, 68 per cent were initial cases and 32 per cent showed fragmentation on admission to hospital. WANSBROUGH *et al.* (1959) described 55.5 per cent of their cases as very early or early, and 44.5 per cent as advanced or very advanced. Among 90 patients examined, CARPENTER & POWELL (1960) found that 73.3 per cent had minimal or moderate, and 26.7 per cent advanced destructive changes.

End-results. In Table 21 the end-results in a number of previous studies are compiled. There are wide variations in the selection of cases, the treatment employed and the methods of evaluation of the end-results in both the series treated by bed-rest and in those treated by various non-weight-

TABLE 21. — *Relationship between end-results and treatment in different series reported in the literature.*

Investigator	No. of cases	Treatment	Results in %		
			Exellent and good	Fair	Poor
Pike 1950	11	Bed rest without splints	36	28	36
Pike »	29	Bed rest with splints	83	10	7
Mindell et al. 1951	28	Bed rest	53.5	17.7	28.8
Herndon et al. 1952	33	»	61.0	39.0	0
Helbo 1953	61	»	82.0	16.4	1.6
Goff 1954	65	»	57.0	29.2	13.8
Hauge 1956	132	»	32.6	40.0	27.4
Ratliff 1956	41	»	43.7	36.8	19.5
Evans 1958	52	»	29.0	40.0	31.0
Evans et al. 1958	24	»	62.5	20.8	16.7
Wansbrough et al. 1959	14	»	50.0	36.0	14.0
Herzog 1961	73	»	77.5	14.0	8.5
Mose 1964	78	Strict bed rest	58.0	17.0	25.0
Mose »	70	Mobile bed rest	61.0	20.0	19.0
Mindell et al. 1951	32	Crutches or walking caliper	72.0	15.5	12.5
Evans et al. 1958	24	Crutches and Snyder's sling	58.3	16.7	25.0
Herzog 1961	34	Walking caliper	44.0	24.0	32.0
Wansbrough et al. 1959	76	Taylor caliper	75.0	11.8	13.2
Wansbrough et al. „	16	Thomas' splint	25.0	75.0	25.0
Mose 1964	71	Walking caliper	45.0	17.0	38.0

bearing devices. Therefore, the table is useful only as a basis for very approximate comparisons.

MINDELL & SHERMAN (1951) found no significant difference between the end-results in their patients treated non-ambulatorily and ambulatorily, respectively, and recommended ambulatory treatment in all unilateral cases. WANSBROUGH *et al.* (1959), who were able to report very good results with Taylor's walking caliper, arrived at the same conclusion. EVANS (1958) and EVANS & LLOYD-ROBERTS (1958), who compared the late results in hospital patients and out-patients, respectively, detected no significant difference in the final radiographic appearance of the femoral head. In view of this, and considering both social and economic standpoints, they regarded ambulatory treatment as the method of choice. By contrast, HELBO (1953), who compared his excellent results with prolonged bed rest with the results of symptomatic treatment, recommended whole-heartedly the first-mentioned method. HERNDON & HEYMAN (1952) and GOFF (1954) were advocates of hospital treatment, too. HOWORTH (1959) emphasized the importance of strict weight relief for the affected hip, which does not, however, imply absolute immobilization. »It appears that early recognition of the synovitis, which precedes the development of coxa plana, with prompt institution of bed rest and non-weight-bearing, may result in abortion of at least some of the cases.»

MOSE (1964) observed an insignificant difference between the results in patients treated by strict bed rest and mobilizing bed rest, while the difference between these two groups and a group treated as out-patients was significant. He recommended treatment by bed rest.

When the present results — 49.1 per cent good, 21.2 per cent fair and 29.7 per cent poor — are compared with the figures in Table 21 showing the results in previous studies, it is found that the former are somewhat poorer than the mean level of the latter. However, even among the series treated by bed rest there are poorer results than the present ones.

Age at onset and end-results. Numerous authors have found that the end-results are better the younger the child at the time of onset of the disease (MINDELL & SHERMAN 1951, GOFF 1954, EVANS 1958, WANSBROUGH *et al.* 1959, CARPENTER & POWELL 1960, MOSE 1964, and others). Table 15 shows the same for the present series. In the group with an onset age of under 4 the result was good in over 90 per cent of cases and poor in none. In the age groups over 9 no hip healed with a spherical femoral head.

Duration of symptoms before institution of treatment and end-results. Regarding the correlation between the duration of symptoms and the *end*-results,

a large number of authors emphasize that the earlier treatment is instituted, the better are the results (BRANDES 1920, SEVERIN 1942, SJÖVALL 1943, MINDELL & SHERMAN, HELBO, GOFF, WANSBROUGH *et al.*, CARPENTER & POWELL and others).

The present observations confirm the above-mentioned observation. It is seen in Table 16 that in over half of the cases which healed with a spherical femoral head, treatment had been instituted within three months after the onset of symptoms, while only about 15 per cent of the patients with good results had been ill for over seven months when the diagnosis was made.

In the present series the mean duration of symptoms before the institution of treatment was 9.6 months (Table 9). This seems to some extent, at least, to account for the poorer end-results in this series as compared with some of those presented in Table 21. In the majority of the earlier series a shorter mean duration of symptoms has been indicated.

RALSTON (1961) arrived at deviating results in regard to the relationship between onset age and end-results. His series consisted of 43 cases of unilateral coxa plana. Treatment (recumbency with Buck's extension and daily progressive resistance exercise) was in all cases instituted within three months after the onset of symptoms. Statistical analysis of the comprehensive quotient after healing had begun showed no correlation between onset age and degree of healing. The age at onset as compared with the duration of the period of reco verygave a poor correlation, and the correlation between onset age and the maximal percentage of epiphyseal involvement was also poor.

Stage of the process at institution of treatment and end-results. This point was discussed by HELBO, HAUGE, WANSBROUGH *et al.* and CARPENTER & POWELL, among others, who found that the end-results were better if the treatment had been instituted before the process had advanced to the stage of fragmentation.

The present series (Table 17) shows a clear tendency towards better radiological end-results when treatment was instituted at an early stage of the disease.

Factors affecting the end-results. HAAS (1937), FREUND (1939), BRAILSFORD (1943), HERMODSSON (1944), SUNDT (1949), GOFF (1954), PERTTILÄ (1954), RATLIFF (1956), EVANS (1958), JENKINS (1958), FREEHAFER (1960) and MORRIS & MCGIBBON (1962) have described occasional cases in which a condition resembling osteochondrosis dissecans was associated with coxa plana. MOSE (1964) detected 8 cases of osteochondrosis dissecans in his

series. All these patients belonged to the higher age groups, and in none of these cases were there any radiological changes discernible in the epiphyseal line or the metaphysis.

In the present series osteochondrosis dissecans was detected in 17 hips, *i.e.* in 5.2 per cent of the 326 hips with coxa plana examined. In one bilateral case, osteochondrosis dissecans was also bilateral. Four of these patients belonged to the age group 5—7 years at the onset of the disease, and 12 patients were 8—11 years at onset. In 13 cases growth was completed. In 8 there were no or minimal metaphyseal changes. In 9 hips osteochondrosis dissecans developed in connexion with severe metaphyseal disturbances. In this respect the observations in the present series differ from those of MOSE.

In 7 of the cases under discussion there was a history of trauma. The experimental investigations of Å. LANGENSKIÖLD (1955) and TALLQVIST (1962) regarding the development of osteochondrosis dissecans lend strong support to the view that the cause of this lesion is a *cartilage fracture* during the growing period. In the present series of 276 cases there were 48 (17.3 per cent) in which trauma was mentioned in the history. Among those showing osteochondrosis dissecans 43.7 per cent had a history of trauma. This is strongly suggestive of a causal relationship between trauma and osteochondrosis dissecans in coxa plana.

To a certain extent this observation also lends support to NAGURA's (1937) theory that the cause of coxa plana is a compression fracture in the chondroepiphysis.

In this connexion it may also be mentioned that KIRSCH (1961) described a case of coxa plana which initially exhibited the kind of picture typical of osteochondrosis dissecans.

Regarding the frequency of subluxation in association with coxa plana there are few data in the literature. SUNDT (1949) observed subluxation in 104 out of 153 and PERTTILÄ (1954) in 17 out of 33 finally healed cases. EVANS (1958) detected subluxation in 42 out of 58 coxa plana hips. He pointed out that the subluxation was very slight in the patients with good and fair end-results.

In the present series subluxation was noted in 45 hips out of 326 (13.8 per cent). Thirty of these were untreated and poorly healed, while in 15 hips the result of treatment was poor.

That coxa plana predisposes to secondary arthrosis has been emphasized by several authors, *e.g.* MØLLER (1926), EYRE-BROOK (1936) and SUNDT (1949).

In the present series secondary arthrosis was observed in 20 cases, 17 of which were neglected. These patients sought medical aid on account of symptoms in the hip due to the complicating arthrosis. In 3 instances arthrosis developed in poorly healed cases during the time of observation.

OBSERVATIONS WITH A BEARING ON AETIOLOGICAL FACTORS

Trauma

In 10 cases (3.6 per cent) of the present series the disease was preceded by a single trauma which may be considered directly related to the development of coxa plana.

Case 22. A girl aged 3 had fallen from the second floor and injured the hip region.

Case 24. A girl aged 6 had had a traumatic luxation of the hip.

Case 43. A boy aged 11 had been run over by a sledge, resulting in contusion and immediate pain in his hip.

Case 74. A boy aged 7 had fallen from a tree and bruised the hip region.

Case 141. A boy aged 6 had fallen from a pile of wood, resulting in contusion and immediate pain in his hip.

Case 148. A boy aged 6 had bruised the hip. Ecchymoses of the hip region resulted.

Case 176. A boy aged 8 had fallen from a bar during gymnastics, resulting in immediate, severe pain in his hip.

Case 191. A girl aged 9 had bruised her hip and been hospitalized on this account.

Case 196. A boy aged 8 had fallen from a rafter in a barn and experienced immediate pain in his hip.

Case 216. A boy aged 8 had fallen from a moving merry-go-round and bruised his hip. There was immediate pain.

Furthermore, in 31 cases (11.2 per cent) a single trauma was mentioned, *e.g.* a fall during play, or when riding a bicycle or skiing, jumping from a rail or a stair, a kick by a playmate, but these everyday incidents had often occurred long before any hip symptoms were noted and must be regarded with great reserve as possible aetiological factors.

In addition, the series contains 7 cases (2.5 per cent) in which repeated trauma or excessive straining of the hip at the time of onset of symptoms can be taken into consideration. In 3 cases the child had jumped innumerable

times from a roof or a tree. In 1 case the symptoms had been preceded by intensive training in ski-jumping, in 3 cases by energetic training in bicycle-riding.

Infection

An infectious aetiology seems tenable in the following of the present cases:

Case 102. A boy aged 5 started limping and complained of pain in the left knee in association with acute tonsillitis. On radiological examination nothing noteworthy was observed in the knee. About four months later, coxa plana in an early stage of fragmentation was observed in the left hip.

Case 54. A boy aged 11 fell ill with fever and hip pain. Three months later, contracture and limitation of movement in the right hip was observed at our Hospital. A radiograph revealed marked condensation and moderate flattening of the right femoral epiphysis and a large step-shaped defect in the ventral margin of the metaphysis. Coxa plana in a late initial stage was diagnosed. (Fig. 4). The head-socket distance was increased medially and slightly narrower than normal cranially. The outline of the acetabulum was blurred. The patient was fitted with Thomas' splint. At a follow-up examination one year later the femoral epiphysis was in a stage of reconstruction. The metaphyseal defect had been filled up to some extent, but the head-socket distance was still larger than normal medially. The mobility of the hip

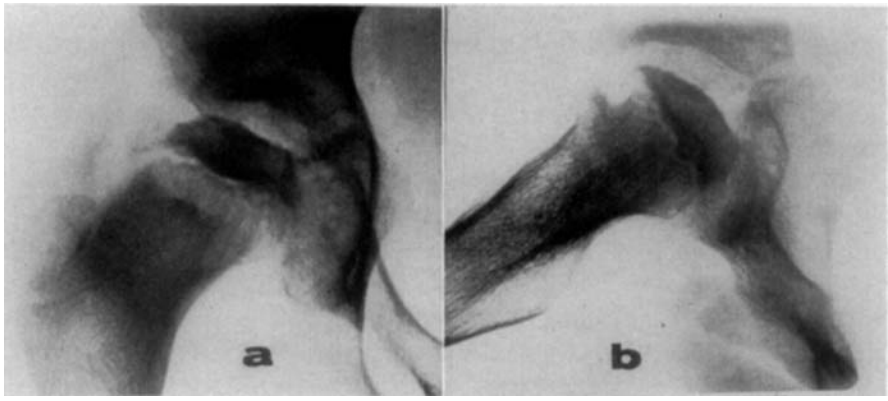

Fig. 4. Case 54, boy aged 11 fell ill with fever and hip pain. Three months later, contracture and limitation of movement in the right hip. Radiographs at this time, *a*: frontal — and *b*: lateral view. Marked condensation and moderate flattening of the femoral epiphysis and a large step-shaped defect in the ventral margin of the metaphysis.

showed some improvement. The patient has failed to attend at suggested, later follow-up examinations.

Case 149. A boy aged 3 years and 2 months developed symptoms in the left hip in connexion with acute appendicitis. Appendicectomy was performed. Three months later, radiological examination revealed signs of epiphysiolysis of the proximal femoral epiphysis. Typical coxa plana developed (Fig. 5).

Case 161. A girl aged 7 was hospitalized for one month on account of burns. Subsequently, she showed a limp. Three to four months later, coxa plana on the left side was observed.

Case 78. A boy aged 6 fell against a stump and sustained a wound in the right inguinal fold. Wound infection developed and he was treated in bed for a month. Subsequently, he showed a persistent limp. A radiological examination performed eight years later in our Hospital showed coxa plana on the right side in a late reparative stage.

Hereditary and constitutional factors

Data regarding the possible occurrence of coxa plana in the patient's family was elicited in 172 of the present cases. In 11 cases (6.4 per cent) radiologically verified coxa plana was recorded in members of the family.

TABLE 22. — *Birth order in 153 cases of coxa plana, and of all 585 children in the coxa plana families concerned.*

Birth order	No. of cases of coxa plana	No. of children	Ratio
I	51	153	1 : 3.0
II	50	141	1 : 2.8
III	24	105	1 : 4.3
IV	5	62	1 : 12.4
V	8	42	1 : 5.3
VI	1	29	1 : 29.0
VII	9	23	1 : 2.6
VIII	3	15	1 : 5.0
IX	1	9	1 : 9.0
X	1	4	1 : 4.0
XI	—	1	—
XII	—	1	—
Total	153	585	

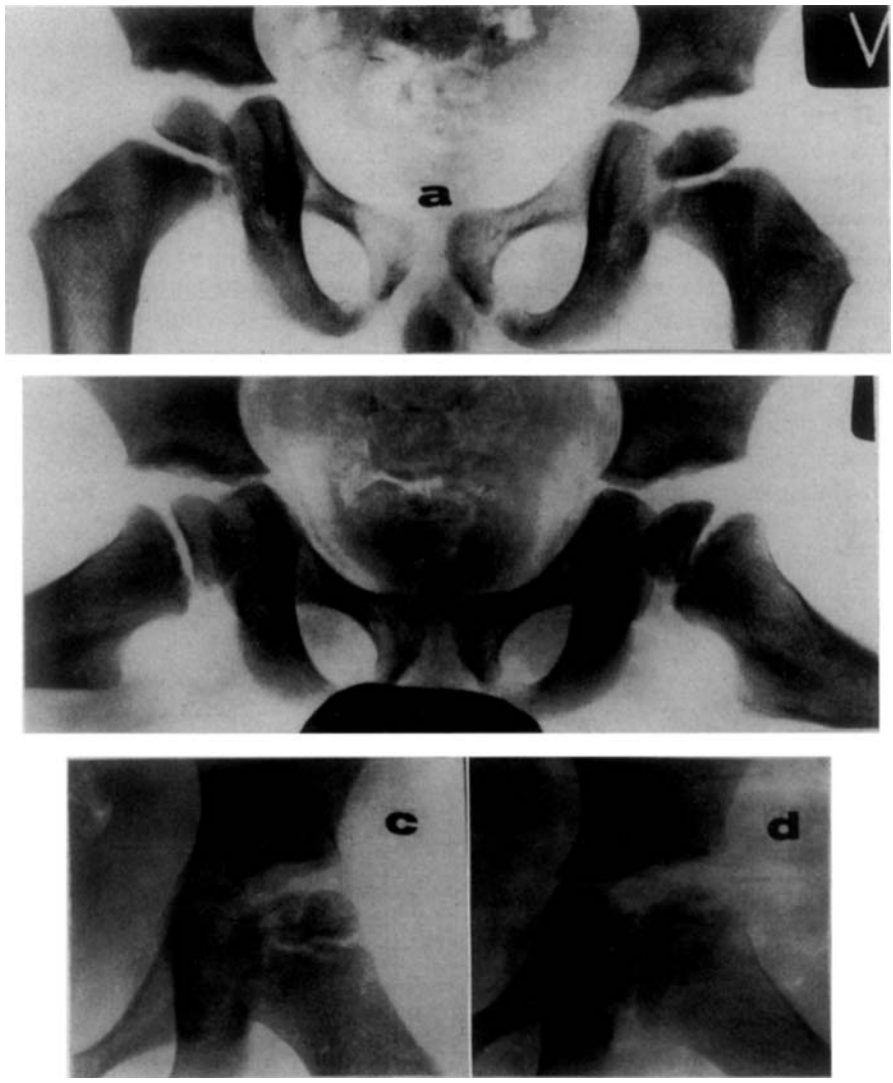

Fig. 5 a—d.

In 4 cases the disease had been diagnosed in siblings. In 1 case a cousin had coxa plana, in 3 cases a cousin of one of the parents' and in 3 cases a child of one of the patient's cousins.

Furthermore, in 9 cases some member of the patient's family had a chronic lesion of the hip of unknown aetiology.

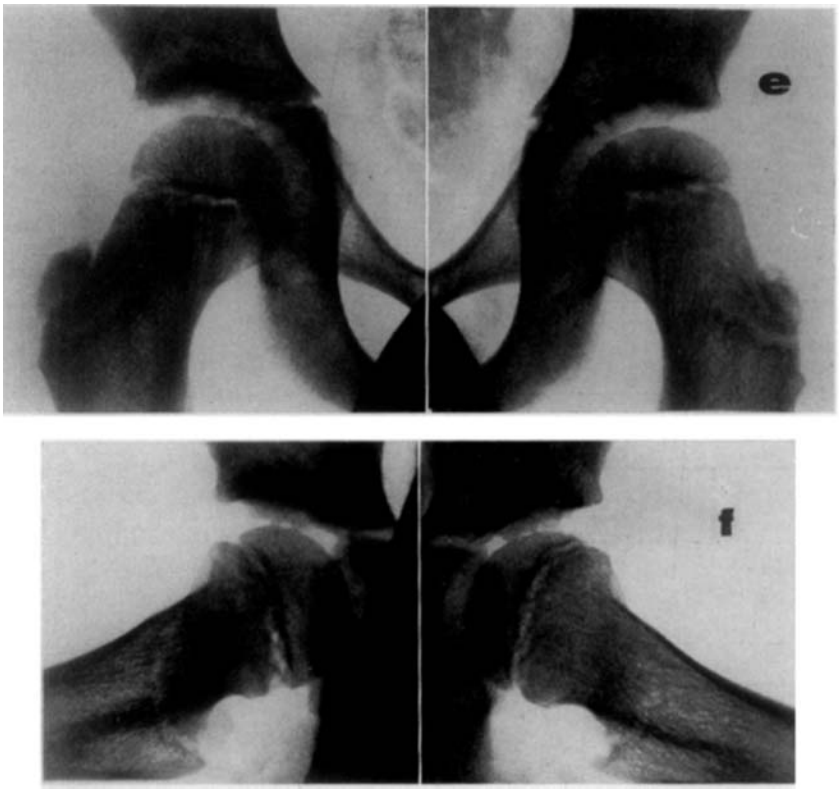

Fig. 5. Case 149, boy aged 3 years and 2 months developed symptoms in the left hip in connexion with acute appendicitis. Appendicectomy was performed. Three months later radiological examination revealed signs of epiphysiolysis of the proximal femoral epiphysis, *a:* frontal, and *b:* lateral view. Later, typical coxa plana with slight fragmentation of the epiphysis, but without metaphyseal changes developed, *c—d.* Treated with Thomas' splint. End-result good, *e—f.*

Birth order. In 153 cases the birth order of the patient was recorded. Table 22 shows the distribution of the cases by birth order groups and the relationship between these and the total number of children in the respective groups in the families concerned.

Bone age. Bone age was determined by GREULICH & PYLE's (1959) method on radiographs of the bones of the hand in 64 cases of coxa plana. Some of these patients did not belong to the present series. The cases were taken at

random and represented different stages of the disease. Table 23 shows the differences between bone age and chronological age in the cases concerned.

TABLE 23. — *Skeletal age in 64 cases of coxa plana in different stages of the disease.*

Stage	Delayed		Normal	Accelerated		Total no. of cases
	No. of cases	Months (range)	No. of cases	No. of cases	Months (range)	
Initial	6	19 (9—31)	—	—		6
Fragmentation.....	11	24 (12—48)	4	1	7	16
Reparative	11	19 (10—32)	12	1	13	24
Definitive (Growing period)	9	18 (6—30)	6	3	12 (9—13)	18
Total	37	19	22	5	11	64

Discussion

Trauma. From the survey of the literature it appeared that many authors regard trauma as a possible cause of coxa plana.

In the present series trauma was mentioned in 48 cases (17.3 per cent), but in the majority of these the statement has to be viewed with great reserve. Parents usually look for a cause when their child starts limping or complains of pain in the hip. Then the child itself, or somebody in the family or its environment, recalls a previous accident, which is considered responsible for the disease, although it may be entirely insignificant.

In regard to repeated minor traumata, it is difficult to decide whether they are of aetiological significance in coxa plana. It seems possible that they play a part as a contributory or precipitating cause.

In 3.6 per cent of the present cases, however, trauma appears to be a factor that significantly influenced the development of the disease.

Infection. As was already mentioned in the survey of the literature, a number of authors have suggested that inflammatory processes may lead to coxa plana.

The present series contains only a few cases in which an inflammatory aetiology can be suspected. In case 54 it seems probable that coxa plana developed in connexion with acute synovitis of the hip joint.

Case 149 (Fig. 5) exhibited an obvious wedge-shaped gap in the epiphyseal line ventrally. This observation corroborates the view, chiefly advanced by PONSETI & MCCLINTOCK (1956), that loss of cohesion of the cartilage matrix is the cause of the circulatory disturbance leading to necrosis of the epiphysis. This loss of cohesion may be due to inflammation.

Hereditary and constitutional factors. Of the present patients 6.4 per cent had close relatives with radiologically verified coxa plana.

HELBO calculated the incidence of coxa plana among the school children in Copenhagen as 0.44 pro mille. MOSE (1964) reported frequencies of 0.08 pro mille among the population on Zealand and 0.09 pro mille in South-Jutland. If these figures are compared with the frequency of familial coxa plana observed in the present study, *i.e.* 6.4 per cent, it seems obvious that hereditary factors play a part in the development of the disease.

One of the present patients, who came from a family with 7 children, had 3 siblings with coxa plana. The lesion was unilateral in all four cases, with typical radiological changes of the same type. One of the four was a heterozygous twin, whose twin sister was unaffected. All the children were otherwise in good health and well developed. The parents were healthy, but 2 cousins (not brothers) of the father's have a limp due to shortening of one extremity.

Birth order. GOFF (1954) found that the first child most frequently develops coxa plana. This observation was corroborated by PEIĆ (1962). Table 22 shows that in the present series, too, first and second children were most numerous among the patients with coxa plana. But this is due to the fact that the absolute number of first and second children was largest in the families concerned. These constituted about fifty per cent. The ratios between the number of coxa plana children in the different birth order groups and the total number of children in these groups show that the morbidity was not higher among first and second children than among those born later. The table shows the highest incidence in the group of seventh children, *i.e.* 1:2.6, and the lowest — 1:29 — in the group of sixth children. The wide variation is obviously due to the fact that the series analysed is too small. The study seems, however, to indicate that the birth order is insignificant.

Bone age. In Table 23 it is seen that in the cases examined bone age and chronological age coincided in 22. In 37 cases the bone age was delayed by

an average of 19 months, while in 5 cases it was in advance of the chronological age by an average of 11 months. As compared with the stage of the disease process, the bone age was most delayed in the fragmentation stage. The study confirms, however, the results previously published which indicate a delayed bone age in coxa plana. This seems to corroborate the view that constitutional factors play a part in the development of the disease.

VI. THE COURSE OF THE RADIOLOGICAL CHANGES IN COXA PLANA

JONSÄTER modified WALDENSTRÖM's (1923) classification of stages by uniting his last two groups (the growing period and the definitive stage) for the reason that the femoral head attains its final shape during the growing period. In this paper JONSÄTER's classification into initial stage, fragmentation stage, reparative stage and definitive stage is used.

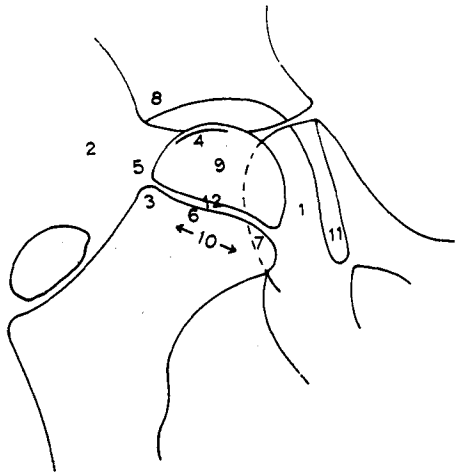

Fig. 6. Early radiological signs in coxa plana. 1: Increased head-socket distance, WALDENSTRÖM's sign (1934). 2: Bulging of the joint capsule, FERGUSON & HOWORTH (1934). 3: Rarefaction, DREHMANN (1914) or rounding, GAGE (1933) of the lateral margin of the metaphysis. 4: A strip-shaped subcortical translucent area ventro-laterally in the epiphysis, FREUND (1930). 5: Rarefactions in the lateral outline of the epiphysis close to the epiphyseal plate, FREUND (1930). 6: Band-shaped osteoporosis in the metaphysis close to the epiphyseal plate, WALDENSTRÖM (1923). 7: Translucent area in the medial metaphyseal zone, GILL (1940). 8: Changes in the roof of the acetabulum, FROMME (1921). 9: The bony epiphysis smaller than in the unaffected hip, shape and structure of the epiphysis normal, BERGMANN (1927). 10: Enlargement of the neck of the femur close to the epiphyseal plate, FÈVRE & LAGRANGE (1956). 11: The »tear-shaped phenomenon», widening of Köhler's tear-shaped figure, HALKIER (1956). 12: Thickening of the epiphyseal plate, HOWORTH (1959).

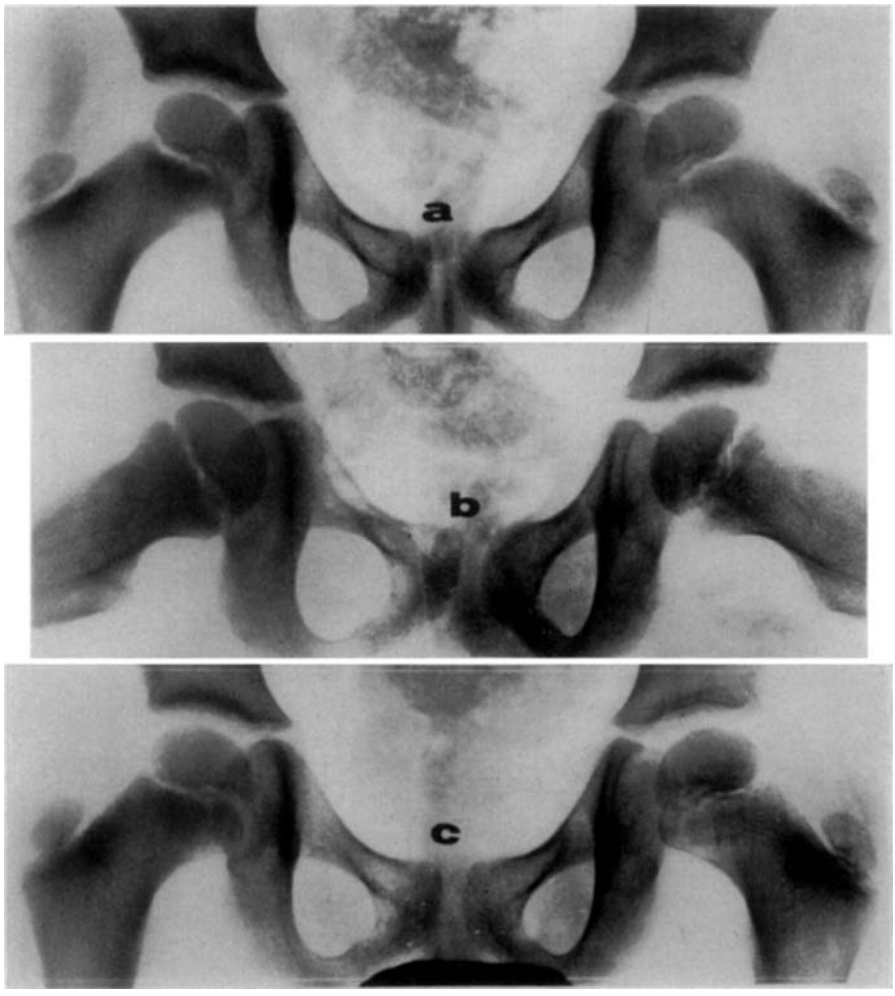

Fig. 7 a—c.

EARLY RADIOLOGICAL SIGNS

The importance of early diagnosis and early institution of treatment in coxa plana has been emphasized by the majority of authors. The recognition of early radiological signs is therefore a point of major interest.

Earlier observations

WALDENSTRÖM (1923) began his definition of the initial stage as follows: »To begin with only limping without any change in the X-ray picture».

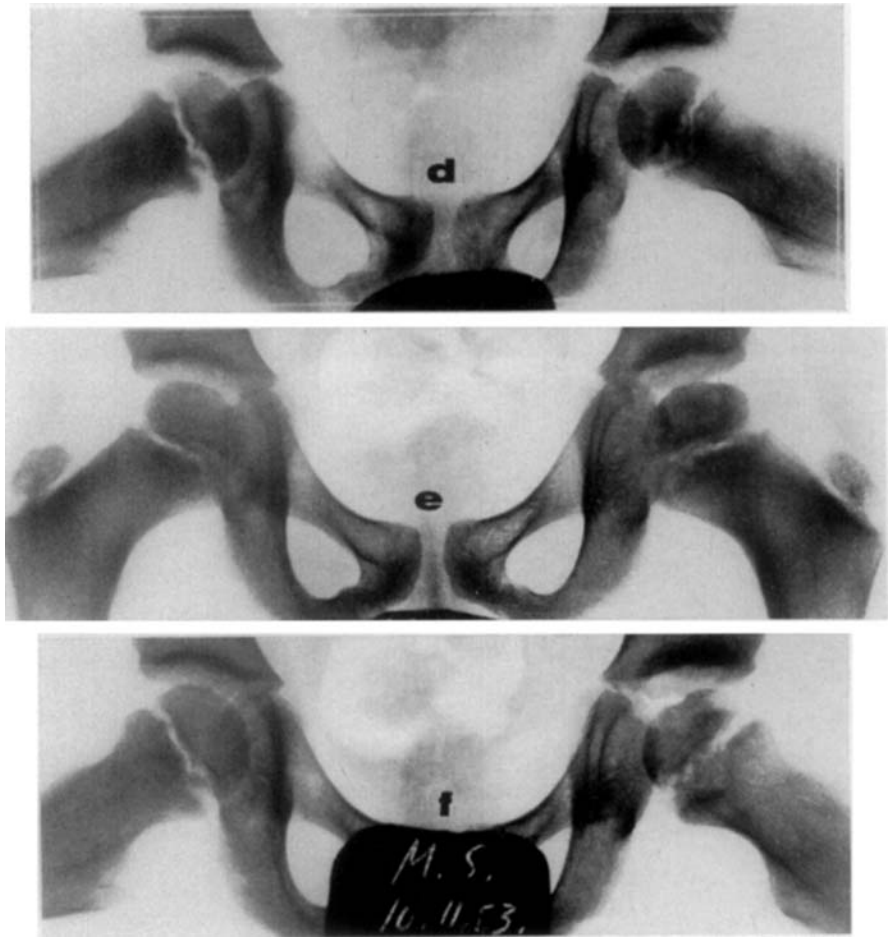

Fig. 7. Case 141, boy aged 5 years and 10 months at onset. Limping after fall from a pile of wood. *a* and *b:* 3 weeks after onset, increased head-socket distance, slight swelling of the soft parts lateral to the left hip joint, slight general osteoporosis of the hip region and thickening of the epiphyseal plate. No structural changes of the epiphysis or changes of its outline. Bed rest with extension for six weeks. *c* and *d:* 9 weeks after onset, the radiological state was the same as before. No clinical symptoms. Weight-bearing was allowed. *e* and *f:* 22 weeks after onset; a large translucent area in the ventral part of the epiphysis, which was slightly flattened. Treated with Thomas' splint for 3 years. After slight fragmentation and slight metaphyseal changes healing with spherical head.

Many other authors have come to the conclusion that coxa plana begins with a prodromal stage, during which no radiological changes, or only changes of the soft parts, are observable (FREUND 1930, FERGUSON &

Fig. 8. Case 65, boy aged 7 at onset. *a* and *b:* 3 months after onset; reduced height and breadth of the right epiphysis. The calcium concentration, outline and structure of the latter are completely normal. The head-socket distance is the same as on the unaffected left side. Later, this case showed moderate fragmentation and a good end-result.

Howorth 1934, Helbo 1953, Nova Monteiro 1954, Howorth 1959, Jacobs 1960, Ralston 1961).

Fig. 6 demonstrates early radiological signs in coxa plana of the kind previously described in the literature.

Observations on the present material

In the present series the radiological findings at the first examination were mostly typical and indisputable owing to the fact that the disease process was already advanced. But in certain cases the first diagnosis was tentative. Some of these may be described as examples of early signs.

Case 141 (Fig. 7) exhibited Waldenström's sign, *i.e.* increased head-socket distance, slight swelling of the soft parts lateral to the left hip joint, slight general osteoporosis of the hip region and thickening of the epiphyseal plate two months after onset. No structural changes of the epiphysis or changes of its outline were discernible. Later, typical changes developed.

Case 65 (Fig. 8). Three months after onset the radiographs showed reduced height and breadth of the right bony epiphysis. The calcium concentration, outline and structure of the latter were completely normal. The head-socket distance was the same as on the unaffected side. Later, this case showed moderate fragmentation and a good end-result.

Case 123 (Fig. 9). A bilateral case. At the first examination the left hip was evaluated as radiologically normal. At a check-up three months later the outline of the lateral pole of the epiphysis was irregular and exhibited a small defect. This finding corresponds to the early sign described by FREUND (1930). Later, typical changes developed.

Case 124 (Fig. 10). The only radiological sign was a small subcortical strip-shaped translucent area proximally in the epiphysis. Complete healing occurred without any other treatment than refraining from athletics and gymnastics for some weeks. This case must be regarded as uncertain.

The tear-shaped phenomenon was observed in 25 cases of incipient coxa plana. In 20 of these the phenomenon was obviously due to oblique projection caused by atrophy of the gluteal muscles on the affected side. In 5 cases it could not be established whether the projection was oblique. In a further 3 cases the tear-shaped figure was narrower on the affected side than on the unaffected side.

Discussion

Both previous reports and the present observations indicate that the early signs in coxa plana vary from case to case. The radiological picture shown by case 141 (Fig. 7 a), for instance, is not pathognomonic of coxa plana, it could just as well support a diagnosis of any kind of synovitis. In uncertain cases it is of paramount importance to institute weight relief and keep the patient under observation for a sufficiently long time. This has previously been emphasized by HOWORTH (1959), in particular.

Most often the tear-shaped phenomenon is probably due to oblique projection of the pelvis, and this sign cannot be regarded as pathognomonic of coxa plana.

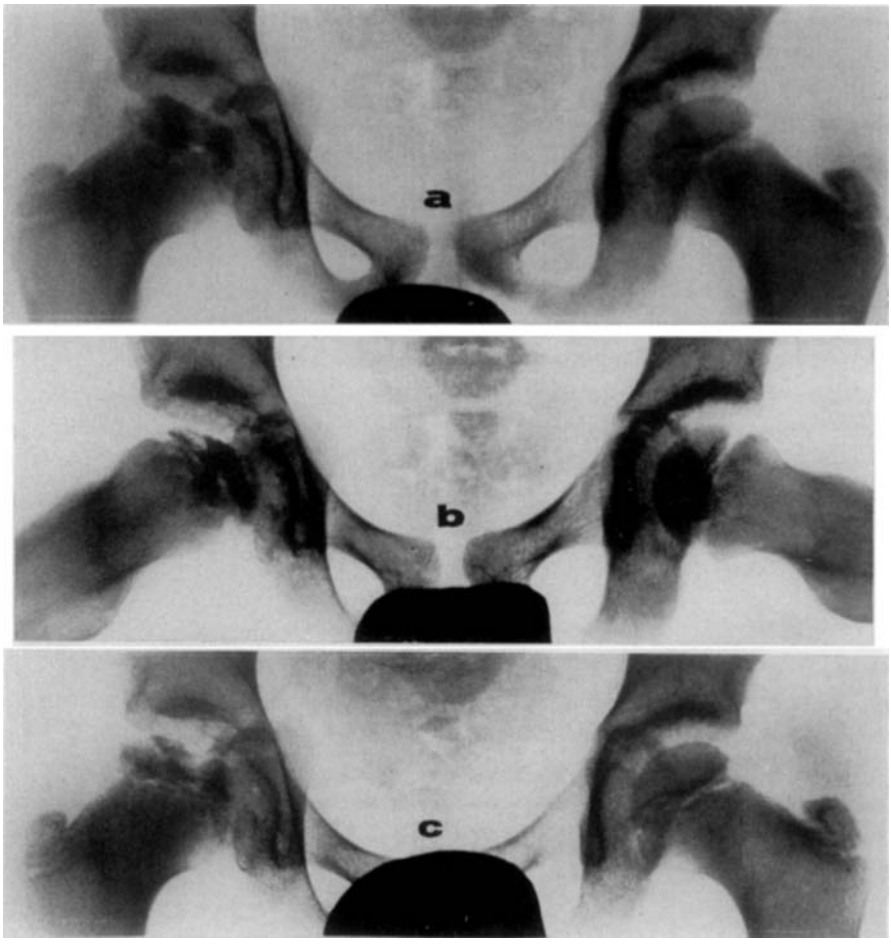

Fig. 9 a—c.

CHANGES IN THE CAPITAL EPIPHYSIS

Initial stage

Observations on the present material. This stage was counted from the onset of symptoms to the time when the bony epiphysis exhibited the first signs of fragmentation.

Duration of the initial stage. In the present series 147 hips were studied in the initial stage, *i.e.* 101 in unilateral and 46 in bilateral cases. The dura-

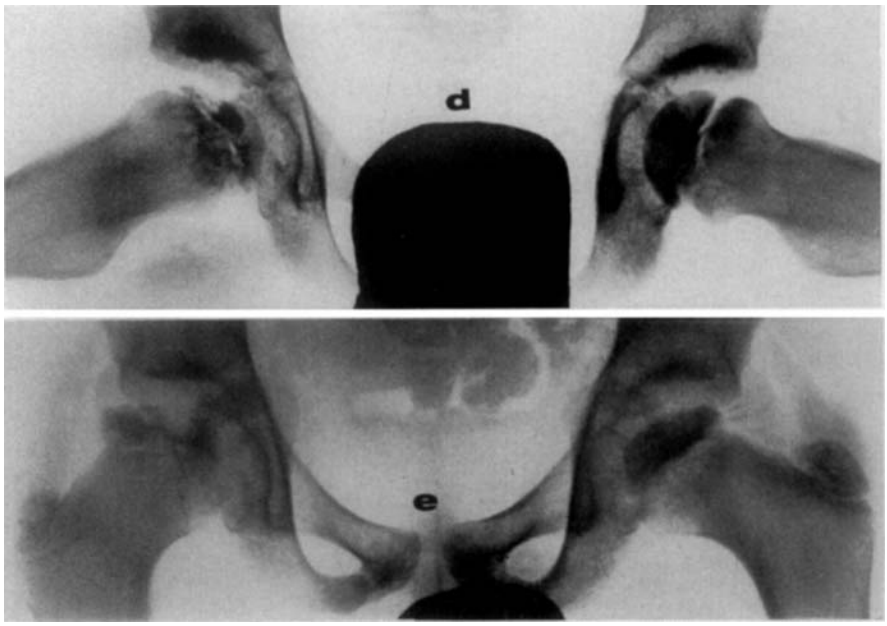

Fig. 9. Case 123, boy aged 7 years and 5 months at onset (right hip). Treated with crutches for five months before the first visit. At the first examination, *a* and *b*: the right epiphysis shows fragmentation. In the lateral outline of the left epiphysis there is a slight rarefaction which was overlooked. Thomas' splint on the right side. At a check-up three months later, *c* and *d*: the rarefaction in the lateral pole of the left epiphysis is larger. Marked metaphyseal changes on the right side. After a further three months, typical initial stage of coxa plana on the left side with flattening and condensation of the epiphysis. *e:* After 6 months in bed Thomas' splint on the left side for one year. End-result poor on both sides. Osteochondrosis dissecans in the right hip.

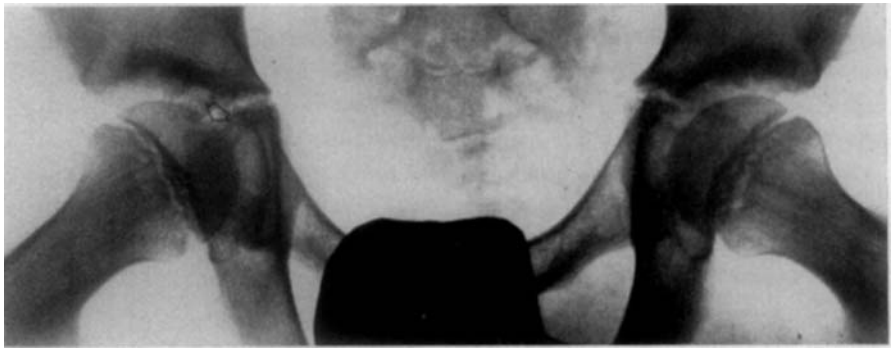

Fig. 10. Case 124, boy aged 10. The radiograph was taken four months after intermittent limping and pain in the right hip. The only radiological sign is a small subcortical strip-shaped translucent area proximally in the epiphysis on the lateral view. Complete healing occurred without any other treatment than refraining from athletics and gymnastics for some weeks.

tion of the initial stage could be evaluated in 116 cases as shown in Table 24.

Table 25 shows the relationship between the duration of the initial stage and the age at onset of the disease. It is seen that *the duration of the initial stage was independent of the onset age.*

TABLE 24. — *Duration of the initial stage in 116 cases.*

Time in months	No. of cases
under 6	57
6—12	59

Mean duration 5.6 months.

TABLE 25. — *Relationship between duration of the initial stage and onset age in 116 cases.*

Duration of initial stage in months	Onset age in years						
	2—3	4—5	6—7	8—9	10—11	12—14	No. of cases
Under 6	4	17	26	7	2	1	57
6—12	5	19	22	7	6	—	59

The radiological changes occurring during this stage in the epiphysis consisted of depressions and breaks in the outline, as a rule in its proximo-ventral portion, translucent areas, often subcortical, and flattening and condensation. These changes occurred almost invariably, except in mild, abortive cases, in which condensation was mostly absent.

The changes varied in degree from case to case, and it goes without saying that variations were seen in one and the same case, depending on how far the process had advanced.

The strip-shaped subcortical translucent area mentioned in connexion with early signs (p. 57) was a relatively frequent sign in the initial stage. It was transitory, owing to the fact that the cortical lamella was resorbed in more advanced cases, or pressed against the spongy bone. A subcortical strip of this kind shown in Fig. 11 (case 32) was observed in 43 cases. This radiograph was taken five weeks after the onset of symptoms.

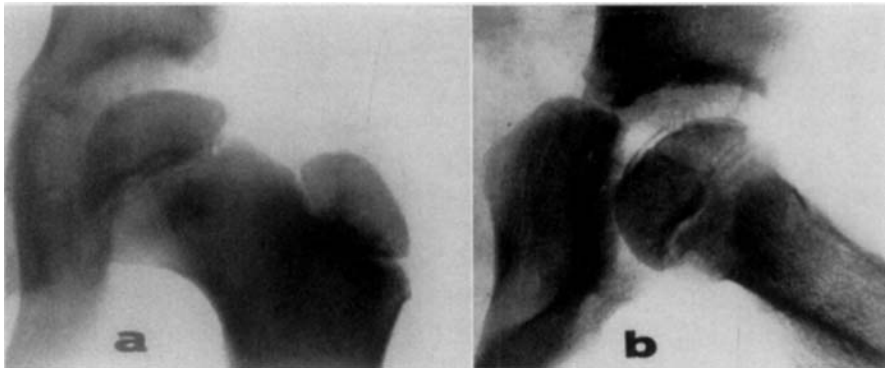

Fig. 11. Case 32, boy aged 9. Five weeks after onset. Marked subcortical strip-shaped translucent area in the lateral view, *b*.

Condensation of the epiphysis often occurred early in the initial stage. In exceptional cases it was observable immediately before, or in connexion with, fragmentation. Case 141, Fig. 7, is an example of absent condensation.

The height of the chondroepiphysis remained unaltered during the initial stage. This was shown by the fact that the distance from the epiphyseal cartilage to the socket of the acetabulum on the lateral view was the same as on the unaffected side. In some cases with a »vacuum phenomenon» on the radiographs, the height of the chondroepiphysis could be measured directly on the pictures.

Discussion of the initial stage. The duration of the initial stage was indicated by WALDENSTRÖM as a half to one year. HELBO reported a mean duration of 12 months in his series, the variation being 5 to 18 months.

BERGSTRAND (1961) observed subcortical fissures or changes of the outline in the proximal portion of the femoral epiphysis in all cases of his series. These changes were demonstrable until eight months after the onset of symptoms.

The condensation observable in the epiphysis has been discussed by many authors. AMSTAD (1916), CAAN (1924) and others ascribed it to sclerosis associated with increased deposition of calcium. WIDERÖE (1921) and LEHMANN (1940) regarded the condensation as a direct necrotic manifestation, analogous to the sequestrum in osteomyelitis.

AXHAUSEN & BERGMANN (1937) concluded that the condensation was due to compression of necrotic spongy bone.

JONSÄTER (1953), who investigated this point histologically, stated that a compression of the trabeculae obviously takes place, but he did not think

that this phenomenon accounts for the marked condensation seen in early cases, in which the epiphysis has still maintained its original shape.

BERNBECK (1954) attributed the early condensation to an increase in inorganic substance due to penetration of chondroitinsulphuric acid from the surrounding cartilaginous tissue (»Kalk-Phanerose»). The condensation seen in later stages he ascribed to compression of necrotic spongy bone.

Case 43, Fig. 12, in the present series is an example of intensive, homogeneous condensation involving the whole of the epiphysis, which showed very slight flattening. In this case it is difficult to explain the condensation as due entirely to compression. It seems more probable that it was a result of total necrosis with markedly increased calcium concentration (ivory epiphysis). A. LANGENSKIÖLD (1952) described a case of tuberculous spondylitis in which the first radiological examination showed an ivory vertebra without any loss of substance or signs of compression.

BERGSTRAND (1961) found that the breadth of the bony epiphysis in the initial stage was smaller on the affected side, and that the epiphyseal breadth

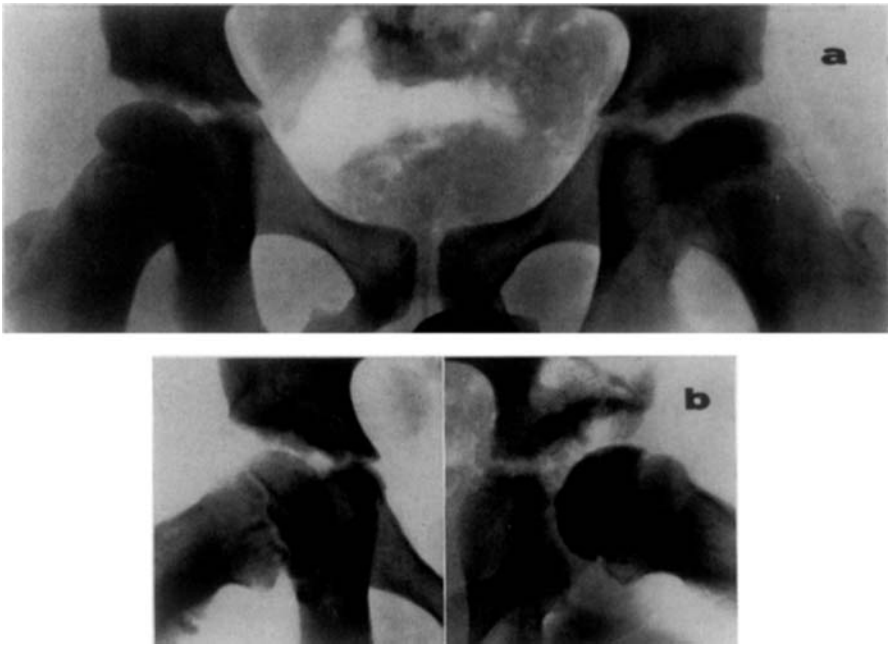

Fig. 12. Case 43, **boy aged 11 years and 3 months at onset.** *a* **and** *b:* 5 months after an obvious single **trauma. Pronounced condensation without any appreciable** flattening of **the epiphysis. Widening of the head-socket distance.**

of the unaffected hip was fairly constantly attained and exceeded six to nine months after onset. In the present study this point was not checked by systematic measurements, but in many cases BERGSTRAND's finding could be confirmed, as is shown in Fig. 8. The phenomenon is attributable to inhibited growth of the necrotic ossific centre (PONSETI & COTTON 1961).

The shape and size of the chondroepiphysis was studied arthrographically by JONSÄTER (1953) and GOFF (1954), by measurement of the epiphyseal plate-socket distance on radiographs by BERGSTRAND (1961), and in connexion with arthrotomy by HERZOG (1961). These investigations showed that the chondroepiphysis maintained its normal height throughout the initial stage, and for part of the fragmentation stage.

Fragmentation stage.

In this study the fragmentation stage was counted from the time when the first radiological signs of fragmentation of the epiphysis were discernible until the time when the first signs of reconstruction could be seen on the radiographs.

Observations on the present material. On the basis of serial radiographs the *duration of the fragmentation stage* could be evaluated in 113 of the present cases, as is seen in Table 26. The mean duration was 10.8 months.

TABLE 26. — *Duration of the fragmentation stage in 113 cases.*

Time in months	No. of cases
Under 6	27
6—12	52
12—24	34

Mean duration 10.8 months.

TABLE 27. — *Relationship between duration of the fragmentation stage and onset age in 113 cases.*

Duration of fragmentation stage in months	Onset age in years												Total no. of cases
	2—3		4—5		6—7		8—9		10—11		12—14		
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	
Under 6	2	20	7	19	8	19	7	50	3	37.5	—		27
6—12	6	60	16	43	23	53	3	21	3	37.5	1	100	52
12—24	2	20	14	38	12	28	4	29	2	25	—		34

The relationship between the duration of the fragmentation stage and the age at onset of the disease appears in Table 27. A lower onset age does not seem to shorten the duration of the fragmentation stage, which was 6 to 12 months in the majority of the cases with an onset age of 2 to 7 years. Among those who were 8 to 11 years old at the time of onset of the disease, the majority had a fragmentation stage of under 6 months, but the number of cases is too small to allow of any definite conclusions. Fragmentation was found to begin in the ventrolateral portion of the epiphysis, and is best seen on the lateral view. The structure of the epiphysis, which usually showed condensation, became irregular, less dense areas occurred and the epiphysis became fragmented. Fragmentation was often preceded by flattening of the bony nucleus, and as fragmentation progressed, the flattening was further increased.

The degrees of fragmentation showed wide variations. In some cases the epiphysis was divided into only two or three pieces, while in others the osseous centre seemed to be completely fragmented. In extreme cases only a few dense fragments were seen, or a thin condensed lamella proximal to the metaphyseal surface.

Discussion of the fragmentation stage. WALDENSTRÖM indicated the duration of the fragmentation stage as two to three years. In STÅHL's series the duration from onset of the disease to the beginning of the reparative stage was an average of 16 months, with a variation of 7 to 34 months. HELBO noted a mean duration of 26 months from the onset to the beginning of reconstruction in untreated and »symptomatically» treated patients, and 16 months in those treated by protracted bed rest. BERGSTRAND observed maximal fragmentation from 9 to 23 months after onset.

Even in the first reports on coxa plana, fragmentation of the femoral epiphysis was described. WALDENSTRÖM (1909), PERTHES (1910), SCHWARZ (1914) and others regarded the fragmentation as a sign of a destructive process. By contrast, CALVÉ (1910) believed that the epiphysis had not been uniform, but primarily separated into two or more small bony nuclei, scattered over the chondroepiphysis. He stated that a total absence of any osseous or cartilaginous destructive process was a feature typical of the disease.

In contrast to current views, FREUND (1930) maintained that the fragmentation stage, instead of being a destructive phase, is a stage of intensive reorganization, originating from the surrounding normal tissues. This was confirmed by JONSÄTER's (1953) histological investigations.

As mentioned above, the degree of fragmentation shows wide variations. For this reason O'GARRA (1959) divided his series into two groups, »anterior

Perthes' disease» and »involvement of the whole epiphysis». In a series of coxa plana patients, GOFF (1959) distinguished between a type with total epiphyseal involvement (37 per cent) and another type with partial epiphyseal involvement (50 per cent). The remaining patients (13 per cent) had bilateral lesions.

RALSTON (1959) calculated the maximal percentage of epiphyseal involvement and studied the relationship between this parameter and various others. The best correlation was noted between the extent of necrosis and the duration of the disease: the larger the proportion of the epiphysis showing fragmentation, the longer the duration. Furthermore, the investigation revealed that the smaller the portion of the epiphysis involved, the more anatomical was the end-result.

Owing to the wide variation of the radiological changes, it is very difficult to make any accurate classification of a large series on the basis of the extent of the necrosis. There are, of course, a number of typical and obvious cases in which evaluation is easy, but very often no indisputable information regarding the extent of the necrosis is obtainable from the radiographs alone. JONSÄTER's (1953) histological investigations suggest that the necrosis is total, broadly speaking.

Reparative stage

In the present study, radiological reparative stage is used to denominate the period from the appearance of the first signs of reossification of the fragmented epiphyseal bony nucleus until the time when the latter is uniform, its structure is normalized and the epiphysis has attained its final shape.

Observations on the present material. In the present series the duration of the reparative stage could be evaluated in 81 cases, as is shown in Table 28.

TABLE 28. — *Duration of the reparative stage in 81 cases.*

Duration in months	No. of cases
12—24	20
24—36	27
36—48	34

Mean duration 32 months.

TABLE 29. — *Relationship between duration of the reparative stage and onset age in 81 cases.*

Duration of reparative stage in months	Onset age in years												Total no. of cases
	2—3		4—5		6—7		8—9		10—11		12—14		
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%	
12—24..	1	14.3	7	25.0	8	28.5	4	28.5	—		—		20
24—36..	4	57.0	9	32.0	8	28.5	4	28.5	2	75.0	—		27
36—48..	2	28.7	12	43.0	12	43.0	6	43.0	1	25.0	1	100.0	34

The mean duration was 32 months. Table 29 demonstrates the relationship between the duration of this stage and the age at onset of the disease. It is seen that almost two-thirds of the cases in the lowest age group had a duration of under 36 months, while almost one-half in the older groups had a duration of over 36 months. None of the patients who were 10 to 14 years old at the time of onset had a duration of under 24 months. The different groups are too small, however, to allow of any definite conclusions.

As a rule, the first signs of reossification were seen in those areas where fragmentation began, *i.e.* in the ventrolateral portion of the epiphyseal bony nucleus. At this time resorption of necrotic bone tissue still occurred in other areas of the epiphysis. The radiographs showed both point-shaped new ossific centres in entirely demineralized areas and reconstruction of bone in the marginal areas of remaining bone fragments, in which resorption had come to a standstill and regeneration had begun. Occasionally, diffuse, homogeneous condensation was seen in entirely demineralized areas, probably representing osteoid tissue which gradually undergoes mineralization. Later, the newformed bone substance in previously entirely decalcified areas fused with the remaining reossified fragments, with the result that the ossific centre became uniform and the defects in its outline were made good. As a rule, the proximoventral portion of the epiphysis was ossified last. An increase in breadth of the epiphysis ran parallel with its reossification.

Signs of early reconstruction. In 22 of the present cases, radiographs taken late in the initial stage showed an osseous shadow in the chondroepiphysis lateral to the bony nucleus. The primary ossific centre was still uniform, although at its lateral margin the structure exhibited some rarefaction as a sign of incipient demineralization. As judged from the radiographs, the above-mentioned bone formation could not consist of a fragment detached from the uniform ossific centre. The further radiological develop-

ment clearly showed that the reformation of bone described here consisted of a fresh ossification area in the lateral portion of the chondroepiphysis, the first visible sign of the formation of new bone preceding fragmentation in the bony epiphysis proper. Fig. 13 (case 128) illustrates this phenomenon. As far as I know, this early reossification phenomenon has not previously been described in the literature.

Discussion of the reparative stage. WALDENSTRÖM (1923) indicated the duration of the reparative stage as one or two years. According to BRAILSFORD (1948), healing is completed during or after the fourth year, counted from the onset. In HELBO's (1953) series the mean duration from onset to primary healing was 52 months in untreated patients and 42 months in cases treated by protracted bed rest.

In mild abortive cases reparation of the femoral epiphysis is complete. Occasionally an epiphysis which has undergone slight fragmentation is rebuilt so as to become entirely symmetrical with the unaffected side. The present series contained only a few such cases. BRAILSFORD (1948), HELBO

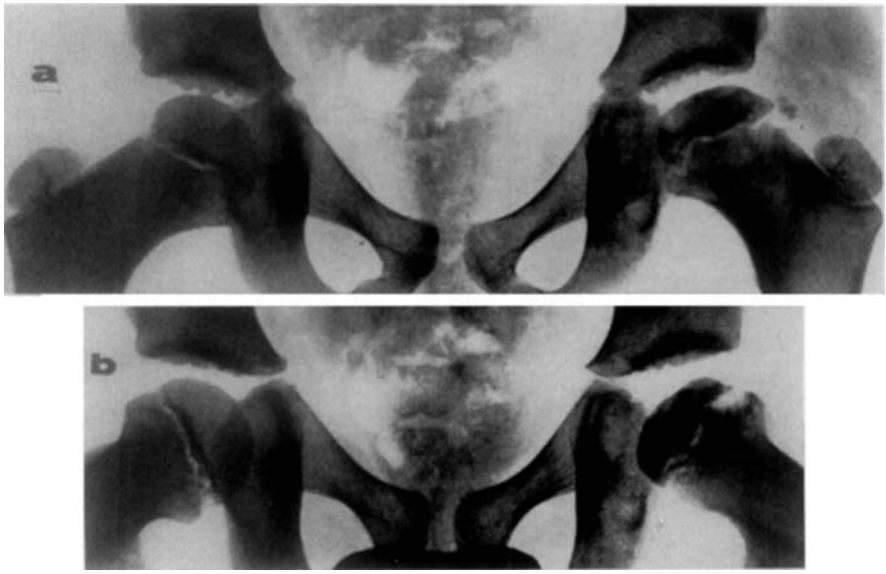

Fig. 13. **Case 128, boy aged 6 at onset. Limping for three months before the first examination.** *a:* **frontal and** *b:* **lateral view. Coxa plana on the left side, initial stage with moderate flattening and condensation of the epiphysis. A broad band-shaped translucent area across the metaphysis in the frontal view. A large furrow-shaped defect in the proximal surface of the metaphysis close to the ventral margin in the lateral view. Note the large ossification area laterally in the chondroepiphysis before the bony epiphysis shows fragmentation.**

(1953), GOFF (1954) and others have described complete radiological healing.

In the majority of cases with good end-results a slight reduction in height and increase in breadth of the epiphysis occur. In most cases of coxa plana, broadening of the epiphysis is one of the most typical features. For this reason BERGSTRAND & NORMAN (1961) suggested that the denomination coxa plana should be replaced by coxa lata.

Definitive stage

When the bony epiphysis has become uniform, the defects in its outline have been filled out and its structure has been normalized, the process is primarily healed and has reached its definitive stage, but the femoral head still increases in size throughout the normal growing period. How much the

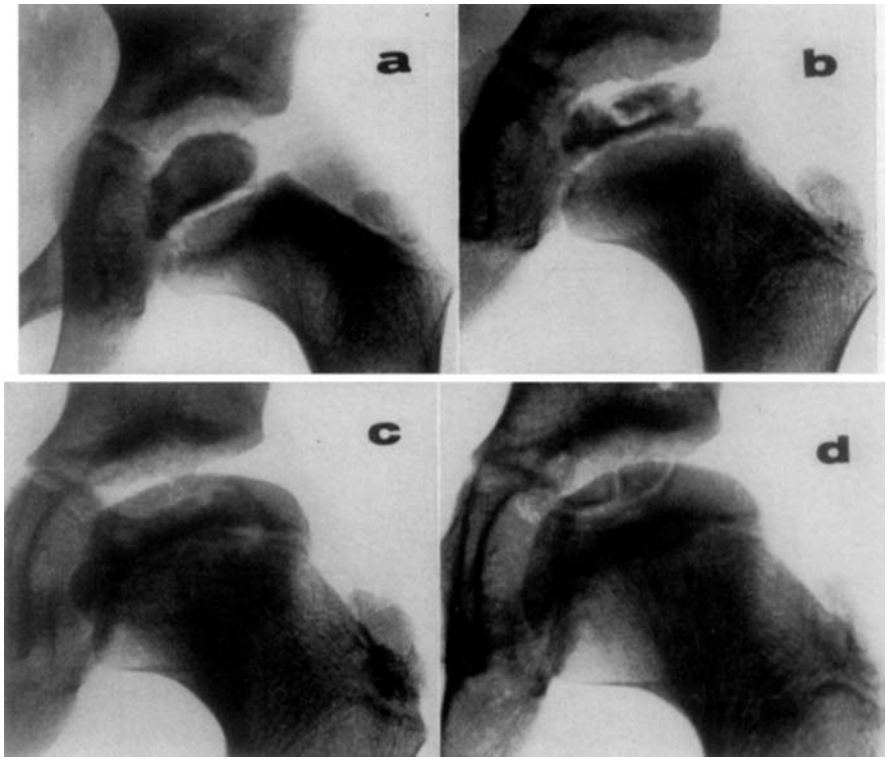

Fig. 14. Case 229, boy aged 6 years and 6 months at onset. The radiographs *a*—*d* show the development of osteochondrosis dissecans.

head will grow during the definitive stage depends, of course, on the age at which this stage is attained.

The final shape of the head shows wide variations depending on the course of the disease process. The range of this variation is best illustrated by the large number of different denominations by which different authors have described the ultimate shape: spherical, ball-formed, ovoid or oval, elliptical, mushroom-shaped, cylindrical, quadrangular or pyramid-shaped.

In the present series *the mean duration of the process of the disease was four years and four months as counted from the onset of the illness until primary healing had taken place.*

In some cases the result of the reparative process was not a uniform head, but one where one or more loose fragments remained in the proximal portion of the epiphysis (Fig. 14).

CHANGES IN THE METAPHYSIS AND THE EPIPHYSEAL PLATE

Earlier investigations

Radiological changes. In his first paper on coxa plana LEGG described radiological changes in the metaphysis which he interpreted as necrotic.

Osteoporosis revealed by focal or band-shaped translucent areas in an early stage of the disease have been described by WALDENSTRÖM (1910, 1923), PERTHES (1913), SCHWARZ (1914), DREHMANN (1914), GAGE (1933), GILL (1940), WIRZ (1953), GOFF (1954), CAFFEY (1961) and others.

GILL maintained that the metaphysis is the primary site of the process, the necrosis of the epiphysis being secondary.

BERGSTRAND (1961) described a local marginal decalcifying process (»Usurierung») in the proximal surface of the metaphysis preceding fragmentation of the epiphysis. This change he interpreted as the first visible sign of granulation tissue invading the epiphyseal bony nucleus.

MINDELL & SHERMAN (1951) were able to follow the metaphyseal changes in 53 cases. The usual change consisted of a diffuse, irregular zone of decreased density, which sometimes appeared early and sometimes late. This defect was filled in as the head of the femur healed. From their observations the authors concluded that extensive changes in the femoral neck generally indicate a poor prognosis. They found that the metaphyseal changes led to asymmetrical and retarded growth or epiphyseal arrest.

Changes resembling those described by MINDELL & SHERMAN were also reported by GOFF (1954).

MOSE (1964) observed a band-shaped area of rarefaction across the metaphysis in the frontal view. In the lateral view this area appeared like a wide gap in the anterior half of the epiphyseal plate, comprising defects both on the epiphyseal and metaphyseal side of the gap. MOSE regarded the epiphyseal defect as an early sign of fragmentation of the epiphysis, while the metaphyseal defect seemed to be attributable to delayed growth in the anterior part of the metaphysis. In the healing phase MOSE observed that the gap was filled out by bone tissue from the epiphyseal side, projecting down into the area of depression in the metaphysis. In some instances the epiphyseal line reassumed its linear appearance, in other instances the depression in the anterior area of the metaphysis was still visible as a bayonet-shaped angle in the epiphyseal line at the time of primary healing.

In an advanced stage of the disease WALDENSTRÖM (1922) observed remodelling of the metaphysis, which exhibited a cranially directed convexity, »Kuppelform». The occurrence of this deformation was emphasized by HOWORTH (1959) and by BERGSTRAND (1961).

Premature closure of the epiphyseal plate. In coxa plana, premature closure of the epiphyseal line has previously been described by MINDELL & SHERMAN (1951), HELBO (1953), GOFF (1954), EVANS (1958), BETTE (1960) and others. MINDELL & SHERMAN observed a direct relationship between the metaphyseal changes and premature fusion of the epiphyseal line and emphasized that the latter phenomenon was a cause of the ultimate deformation of the proximal end of the femur. GOFF reported that premature closure was frequent in the group which healed with a spherical femoral head, but the phenomenon was still more frequent, and closure occurred earlier, in those who healed with a mushroom-shaped head. In the group with irregular end-results fusion occurred earlier still than in the foregoing group.

EVANS (1958) reported increased antetorsion of the femoral neck and head as a common phenomenon in coxa plana owing to asymmetrical growth caused by premature closure of the epiphyseal line.

Histological changes in the epiphyseal plate and the metaphysis. LEVY (1911) assumed that coxa plana is due to destructive processes in the epiphyseal plate.

The data regarding the histological picture of the epiphyseal plate in coxa plana are scanty. Together with normal hyaline cartilage, RIEDEL (1923) observed islands of cartilage showing cell proliferation, oedema, fibrillar transformation and here and there marked atrophy of the nuclei of the cells. PONSETI (1956), who studied 2 cases of coxa plana histologically, found that the cartilage of the epiphyseal plate was fibrillated and cracked,

the cells were clustered and the line of enchondral ossification was irregular. The lesion of the epiphyseal plate appeared to be due to a loss of cohesion in the cartilage matrix. In connexion with therapeutical drilling, YAMAGUCHI (1959) studied the histology of coxa plana. With regard to the epiphyseal plate he found that the cartilage was undergoing degeneration first in parts and later as a whole. The zone of maturing cartilage was affected in different degrees in its different parts. The bone trabeculae in the metaphysis were compressed, broken or necrosed.

In fractures of the femoral neck which led to necrosis of the proximal fragment, AXHAUSEN (1922) observed necrosis of the epiphyseal cartilage.

Observations on the present material

These observations are based on inspection of the series of radiographs taken in all the present cases of active coxa plana. About 3000 radiographs were examined.

In one case increased thickness of the epiphyseal plate was seen early in the initial stage (Fig. 7, case 141). Another patient exhibited epiphysiolysis with a gap ventrally in the epiphyseal line (Fig. 5, case 149).

In mild abortive cases, in which only the proximal portion of the bony epiphysis was involved, no changes were detectable in the metaphysis. The series includes 15 such hips.

In 20 cases slight, reversible metaphyseal changes were observed. These consisted of irregularities in the proximal outline of the metaphysis and band-shaped or cyst-like translucent areas in the subcapital part of the metaphysis, which subsided in a few months without leaving any persistent metaphyseal changes. These changes were concurrent with slight or moderate signs of necrosis in the epiphysis.

The most frequent metaphyseal change observed during the initial stage was a band-shaped zone of decalcification across the proximal portion, seen in the frontal view. This phenomenon was more or less conspicuous in all initial cases, the above-mentioned abortive cases excepted.

In the lateral view about half the cases exhibited a marginal depression ventrally in the proximal surface of the metaphysis, corresponding to a furrow-shaped defect in the periphery (Fig. 13). The other half showed a wide gap in the ventral part of the epiphyseal plate, caused by a defect of the ventral metaphyseal margin.

In some cases the metaphyseal defect extended as far as to the dorsal portion of the metaphyseal surface or margin, but as a rule it was less con-

spicuous there. Occasionally this defect was circular and involved the whole periphery of the metaphyseal surface. In such cases the central portion of the latter formed a plateau-shaped elevation, surrounded by depressions. An extreme example of this is shown in Fig. 15 (case 44).

During the further course, the cases exhibiting a furrow-shaped depression in the metaphyseal surface showed a defect of the metaphyseal margin,

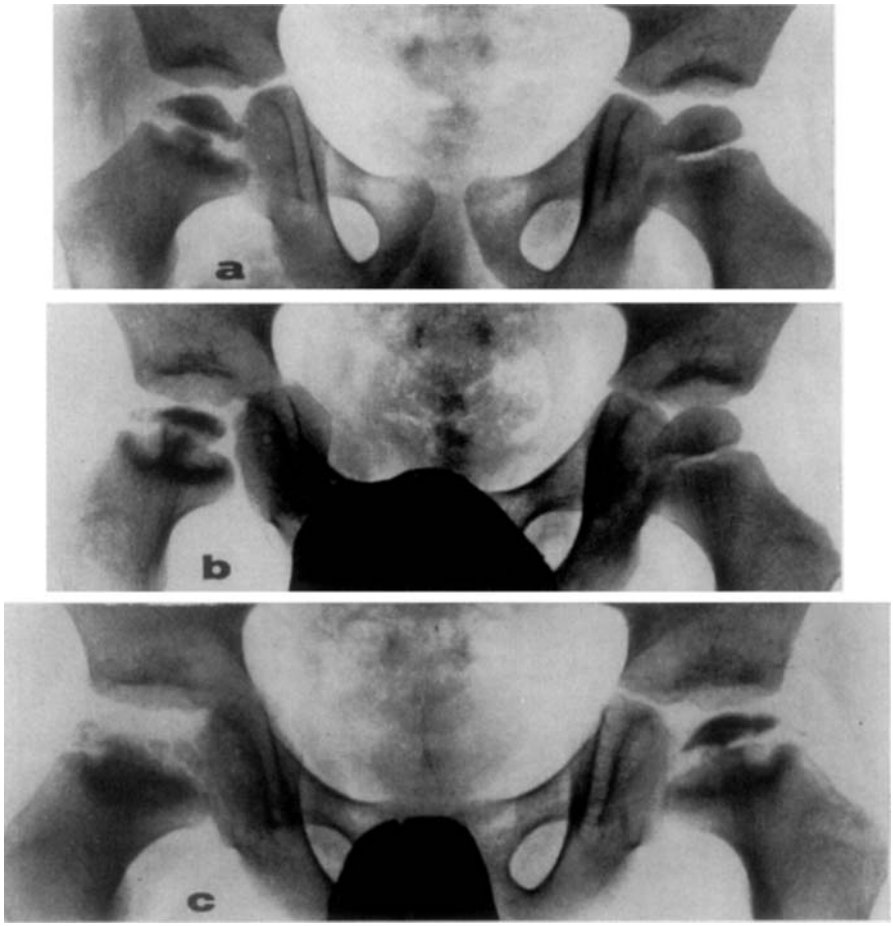

Fig. 15. Case 44, boy aged 4 at onset. The radiographs demonstrate severe, symmetrical metaphyseal changes with circular defects in the periphery of the proximal metaphyseal surfaces and plateau-shaped elevations in the central portions of the latter. Total necrosis of the epiphysis. *a:* 6 months after onset. *b:* 10 months after onset. *c:* 23 months after onset. Treated at first with Thomas' splint on the right side, then by bed rest for one and a half year and finally with Thomas' splint on the left side. End-result elliptical head on both sides.

leading to the development of a gap in the ventral portion of the epiphyseal plate. The picture resembled that seen in the group of cases primarily exhibiting a gap. (It is possible that the development of a gap in this group had been preceded by a stage in which a furrow-shaped depression in the proximal metaphyseal plane occurred.) During the fragmentation stage the defect in the metaphyseal margin was enlarged and gradually developed into an oblique, or most often a step-shaped, defect (Fig. 4). The cause of this seemed to be that ossification in the central portion of the metaphysis continued, while the growth in the area of the defect was arrested. The margins of the step-shaped defect were gradually rounded off, and at the same time the whole outline of the metaphyseal surface became irregular. During the later course the metaphyseal surface invariably developed a proximally directed convexity.

During the reparative stage, the metaphyseal defect was made good in the majority of cases in such a way that the bony epiphysis grew in the distal direction towards the defect, which was thus filled out by the epiphysis. Frequently, isolated ossific centres were seen within the metaphyseal defect. Gradually these fused with the ingrowing epiphysis. As a natural result of this series of events, the epiphyseal line assumed a more or less marked step-shaped irregularity in its ventro-lateral part (Fig. 21).

In 3 of the cases with severe metaphyseal changes, the metaphyseal defect disappeared owing to reformation of bone at its margins, so that the metaphyseal surface was levelled and the epiphyseal line became regular, although with a proximally directed convexity (Fig. 16, case 46).

Even in areas which on the radiograph did not show any metaphyseal defect proper, a tendency of the epiphysis to grow out over the metaphyseal margin was discernible, with broadening of the epiphysis resulting. This increase in breadth was often most conspicuous in the lateral portion, in which an outgrowth of the epiphysis extending far over the femoral neck, even to the greater trochanter, could be seen. This observation seems to indicate that there were metaphyseal changes also in other parts of the metaphyseal margin, although these were not visible on the radiographs.

In parallel with the ossification of the metaphyseal defect, *an increase in breadth of the metaphysis occurred.* Periosteal apposition of bone was a relatively frequent finding at the borderline between the metaphysis and the femoral neck, usually cranially to the latter.

Towards the end of the reparative stage, typical deformations in the metaepiphyseal borderline area occurred.

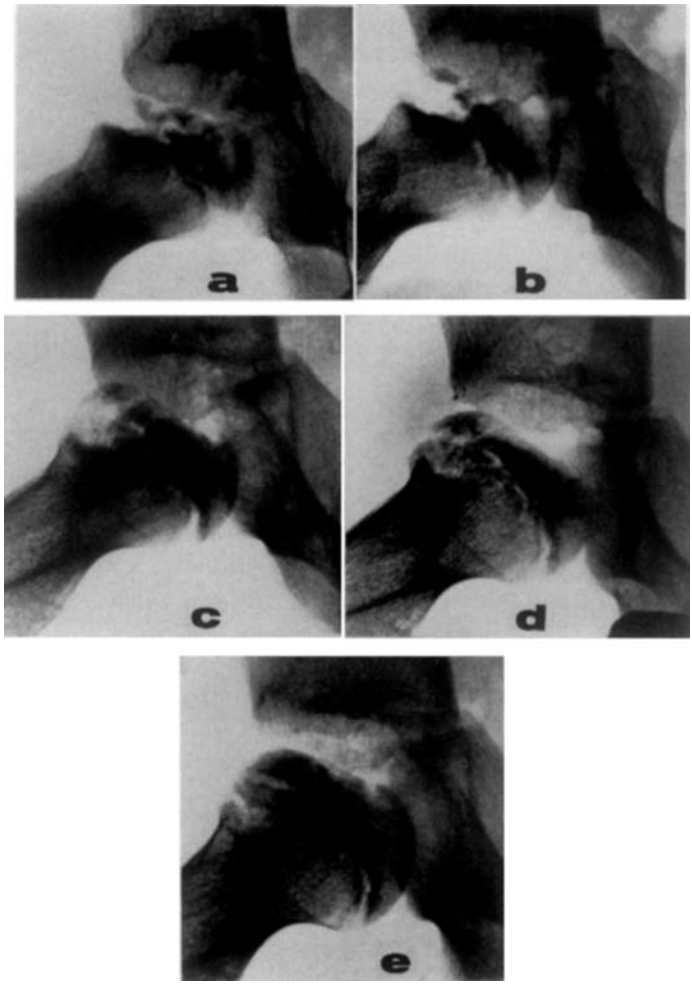

Fig. 16 a—e.

Normally, the distal surface of the femoral epiphysis and the proximal surface of the metaphysis are almost straight. Between these the epiphyseal plate is situated, appearing on the radiographs like a line or a band-shaped space between the epiphysis and the metaphysis.

In the great majority of cases in the present series various degrees of irregularity of the proximal surface of the metaphysis were observable.

As has already been mentioned, the metaphyseal surface was convex in the proximal direction. The convexity sometimes had a smooth outline and

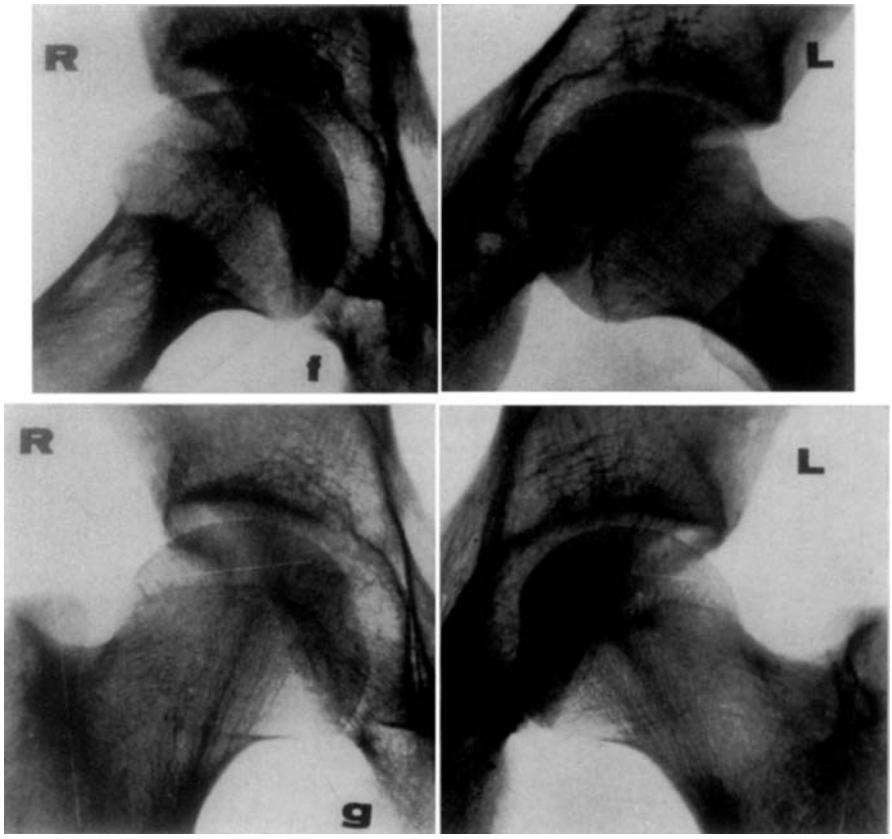

Fig. 16. Case 46, boy aged 6 years and 10 months at onset. A large, oblique metaphyseal defect seen in *a* and *b* disappeared owing to reformation of bone at its margins, as seen in *c*—*e*. The epiphyseal line became regular. The radiographs *a*—*e* were taken from 10 to 34 months after onset, at intervals of 4 to 12 months, *f* and *g* about 10 years after onset. Treatment: Thomas' splint for 2 years and 9 months. End-result good.

sometimes showed a more or less regular undulation, or the contour was entirely irregular with deep, distally directed depressions and proximally directed plateau-shaped or step-shaped elevations. The depressions were situated peripherally and corresponded to the primary marginal defects, while the elevations were more centrally located.

Often the routine radiographs did not provide an accurate concept of the changes described here. Tomograms were very informative, and on these the grave deformation of the metaphyseal surface was often striking (Fig. 17, case 262).

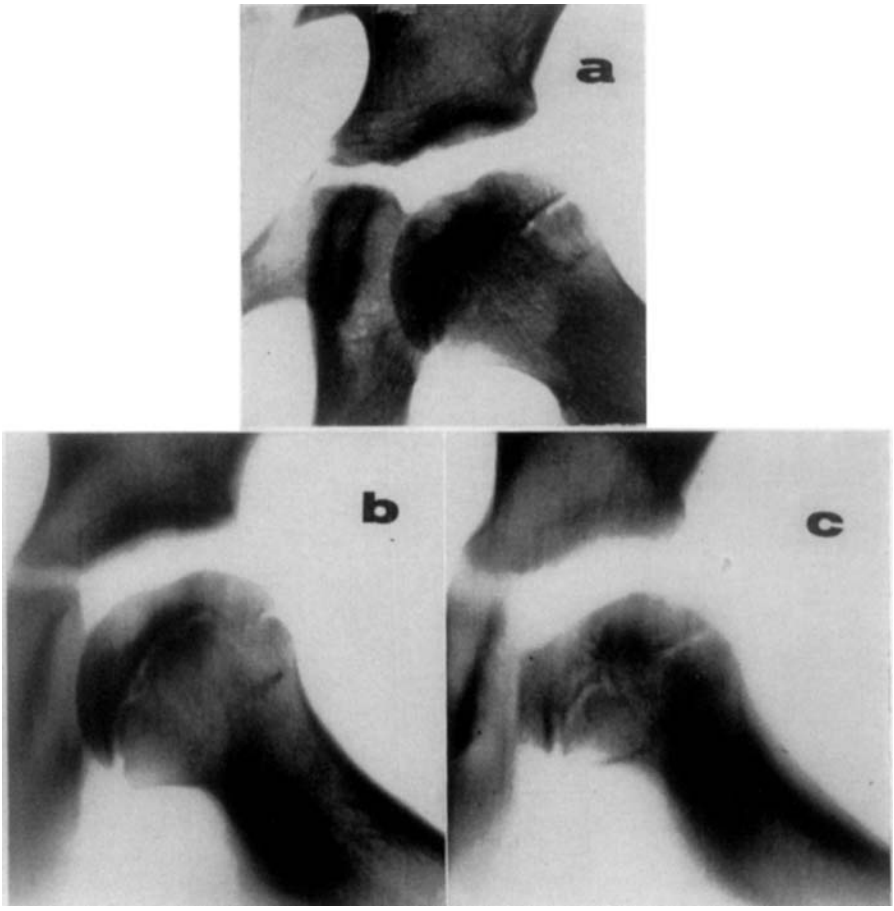

Fig. 17 a—c.

The remodelling process in the metaphysis took place in parallel with the reparative process in the epiphysis. On inspection of the large number of radiographs on which this study is based, it was clearly seen that the two processes were dependent on each other, but it was impossible to distinguish between cause and effect. In cases where fragmentation had been slight, or a coherent layer of spongy bone developed relatively soon upon the metaphyseal surface, the deformation of the metaphysis was slighter. Relatively marked irregularity of the metaphyseal surface was usually associated with a high degree of fragmentation and slow consolidation of the epiphysis. In many cases the radiographs conveyed the impression that

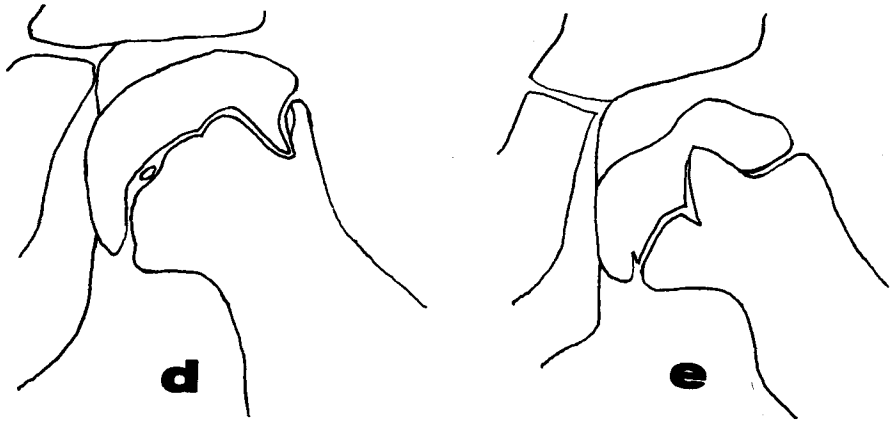

Fig. 17. Case 262, boy aged 6 years at onset. *a:* routine radiograph, *b* and *c:* tomograms with a 1 cm level distance, 5 years after onset. Late reparative stage. *d* and *e:* drawings to *b* and *c*. Routine radiographs do not provide an accurate idea of the changes in the meta-epiphyseal borderline. Tomograms give very good information regarding irregularities in the metaphyseal surface.

the elevated central portion of the metaphysis intervened with the fusion of the ossifying epiphyseal fragments, which was slow in this site. In some cases months and years elapsed before fusion of the epiphysis occurred in this area, and as a rule the layer of bone remained very thin and it seldom exhibited a normal structure (Fig. 18).

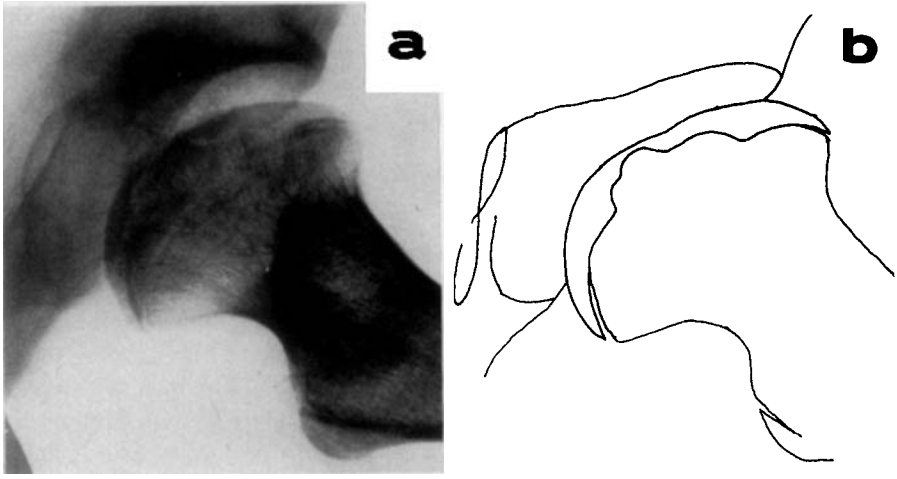

Fig. 18. Case 212, boy aged 8 at onset. *a:* 11 years after onset. *b:* drawing to *a*. The subcapital epiphyseal line is partially closed. The metaphyseal surface is very irregular with a central elevation. In the proximal part of the epiphysis the layer of bone tissue is thin and the bone structure is still irregular. Untreated case.

The distal surface of the epiphysis was congruous with the proximal surface of the metaphysis. In the cases under discussion it was thus more or less concave. The epiphysis looked like a cap on the metaphysis. In 20 cases the cap-shape was very striking (Fig. 19, case 140).

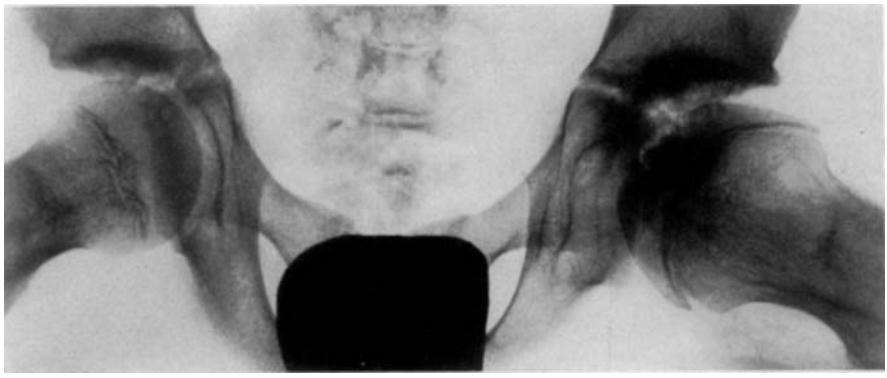

Fig. 19. Case 140, boy aged 12 years at onset. Untreated. The radiograph shows a cap-shaped epiphysis.

In parallel with the broadening of the metaphysis, inhibition of the longitudinal growth of the femoral neck was observed.

Towards the end of the reparative stage the epiphyseal plate became thinner, as was clearly seen in unilateral cases on comparison with the unaffected side.

Partial, and gradually total, *fusion of the epiphyseal line often occurred much earlier on the affected side than on the unaffected side.* As a rule, fusion occurred first in the central area, *i.e.* at the vertex of the convexity. In this case the situation of the epiphysis in relation to the femoral neck was not affected.

In many cases it was found that the epiphyseal line closed laterally, while the metaphysis continued to grow on the medial side. In such cases the epiphysis assumed a valgus position in relation to the femoral neck. Often this valgus position was only faintly discernible, but in some cases it was conspicuous (Fig. 20 a). In one case the epiphyseal line first closed medially, resulting in a varus position of the head (Fig. 20 b). In 14 unilateral cases an increased antetorsion of the neck and head was observed as compared with the unaffected side. In one of these cases the increased antetorsion involved particularly the head, and it is obvious that the epiphyseal line in

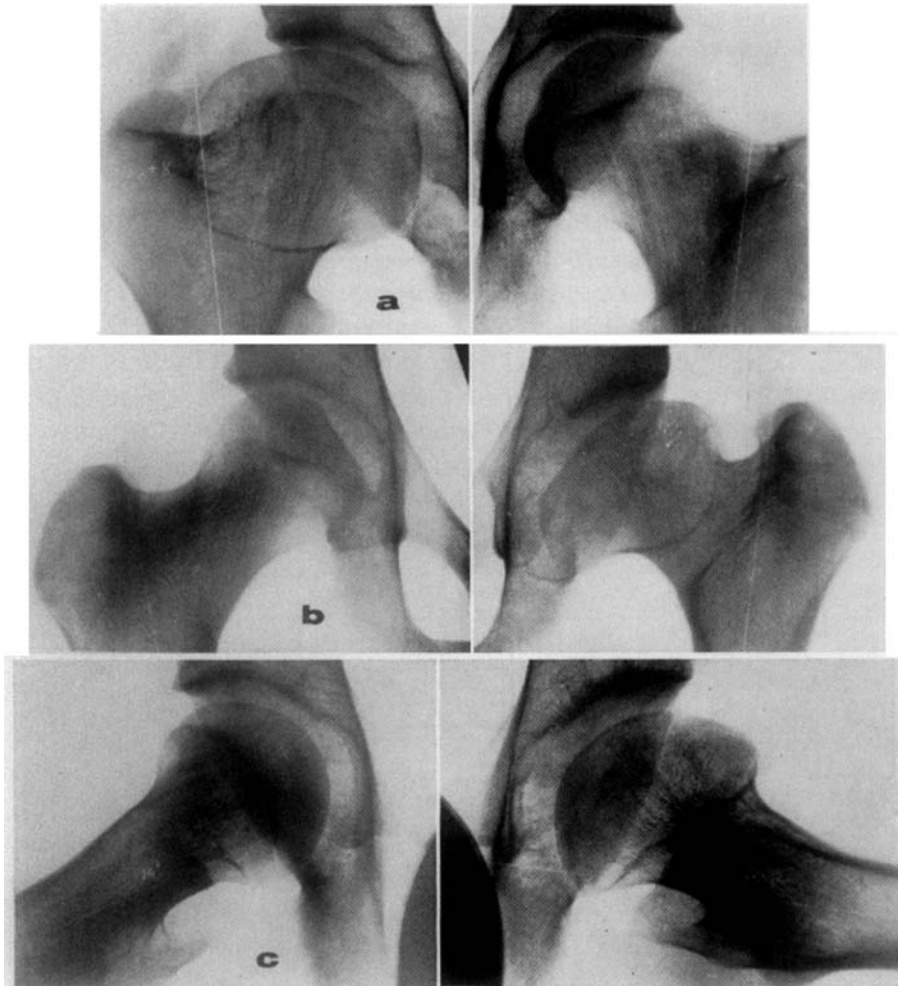

Fig. 20. Examples of asymmetrical growth owing to premature fusion of the epiphyseal line. *a:* valgus position of the femoral head after fusion of the lateral part of the epiphyseal line. *b:* varus position of the head as a result of premature fusion in the medial part of the epiphyseal line. *c:* increased antetorsion of the head owing to fusion of the anterior part of the epiphyseal line.

this case closed earlier on the ventral side (Fig. 20 c). Retrotorsion was not seen in any cases.

In order to enable evaluation of the relationship between premature closure of the subcapital epiphyseal line and the final shape of the femoral head, the present ultimately healed cases of unilateral coxa plana were

divided into two groups, depending on whether the serial radiographs revealed premature or normal closure of the epiphyseal line.

In this paper premature closure stands for fusion that had certainly taken place at least one year earlier on the affected side than on the unaffected side. Normal closure means that fusion occurred less than a year earlier on the affected side.

The group with premature closure comprises 42 cases, the group with normal closure 20 cases. (Both groups were certainly larger, but owing to long intervals between the examinations radiological evidence is lacking).

Table 30 shows the relationship between fusion of the epiphyseal line and the ultimate shape of the femoral head. It appears in the table that in more than half the cases premature closure of the subcapital epiphyseal line was associated with poor healing of the femoral head. In the group with normal closure, the 2 cases which did not heal with a spherical femoral head developed a condition resembling osteochondrosis dissecans.

TABLE 30. — *Relationship between closure of the subcapital epiphyseal line and ultimate shape of the femoral head.*

Closure of the epiphyseal line	Shape of the femoral head			Total
	Spherical No. of cases	Elliptical No. of cases	Irregular No. of cases	
Normal	18	—	2	20
Premature	5	13	24	42

From the standpoint of the end-result in the femoral head, premature closure of the epiphyseal line thus seems to be an unfavourable sign.

For the sake of evaluating the relationship between the metaphyseal changes and the closure of the subcapital epiphyseal line, the ultimately healed cases were classified according to the degree of metaphyseal changes. The classification was made at a late reparative stage, as close as possible to the time of epiphyseal fusion. The following principles of classification were used:

Small or no changes: the proximal surface of the metaphysis plane, or regularly convex.

Moderate changes: step-shaped marginal metaphyseal defect, filled up by the epiphysis, otherwise a regularly convex metaphyseal surface.

Marked changes: metaphyseal surface very irregular, the central portion plateau-shaped and elevated.

The increase in breadth of the metaphysis as compared with the unaffected side was measured in the lateral view, while the shortening of the femoral neck was measured in the frontal view.

The relevant observations are listed in Table 31, which shows that premature closure of the epiphyseal line in most cases was associated with marked metaphyseal changes. In cases with normal closure the metaphyseal changes as a rule were slight. The increase in breadth of the metaphysis and the decrease in length of the femoral neck were clearly larger in the cases showing premature closure than in those showing normal closure.

TABLE 31. — *Degree of metaphyseal changes, broadening of the metaphysis and shortening of the femoral neck in 42 unilateral, ultimately healed cases with premature closure, and 20 cases with normal closure of the subcapital epiphyseal line.*

Closure of the epiphyseal line	Degree of metaphyseal changes			Broadening of the metaphysis in mm, mean (range)	Shortening of the femoral neck in mm, mean (range)
	Slight No. of cases	Moderate No. of cases	Severe No. of cases		
Premature	3	5	34	12 (3—23)	17 (0—30)
Normal	16	4	—	4.3 (0—9)	4.5 (0—9)

It may be concluded that marked metaphyseal changes and marked broadening of the metaphysis and retardation of the longitudinal growth of the femoral neck are unfavourable prognostic signs, indicating that the epiphysis will close prematurely.

Fig. 21 (case 248) shows the radiological course in a girl who was 7 years old at the onset of coxa plana. This case represents moderate metaphyseal changes.

Discussion

The present observations concerning the *metaphyseal changes and premature closure of the subcapital epiphyseal line* agree well with the above-mentioned reports of MINDELL & SHERMAN, GOFF and EVANS. With regard to the ossification of the metaphyseal defect, there is a high degree of correspondence between the observations made in the present series and those of MOSE.

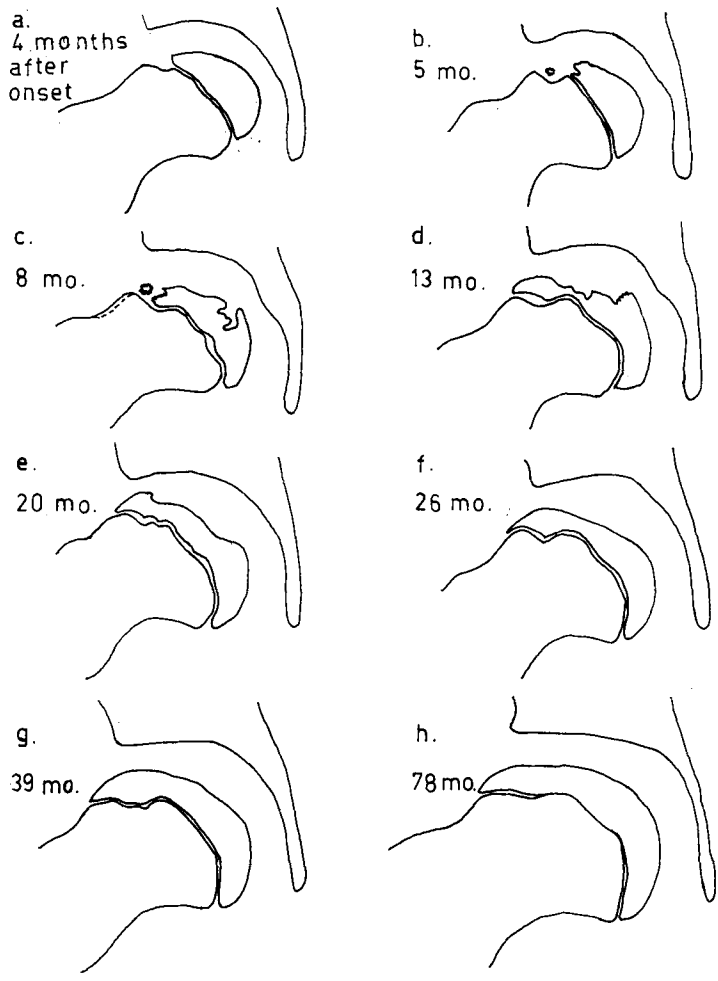

Fig. 21. Drawing based on lateral radiographs of the right hip showing the radiological course of coxa plana in a girl aged 7 years at onset of the disease (case 248). *a:* 4 months after onset; the epiphysis is slightly flattened; in the ventral margin of the metaphysis there is an oblique defect. *b:* 5 months after onset, fragmentation in the ventral part of the epiphysis; a step-shaped defect in the ventral margin of the metaphysis; there is a small ossification centre in the metaphyseal defect. *c:* 8 months after onset, fragmentation proximally in the epiphysis; the ventral part of the epiphysis is growing into the metaphyseal defect; the margins of the defect are rounded; the ossification centre in the defect is enlarged; thickening of the periosteum ventrally on the neck. *d:* increased demineralization proximally in the epiphysis; the ossification centre in the metaphyseal defect is fused with the ventral part of the epiphysis, which fills up the defect; the metaphysis is broadened. *e—g:* mineralization of the epiphysis proceeding; the borderline of the metaphyseal defect is still visible; the epiphyseal line is slightly wave-like; *h:* 78 months after onset, the central part of the epiphyseal line is closed.

The premature fusion of the subcapital epiphyseal line may begin in different areas of the growth zone. This accounts for the deviations from the normal relationship between the positions of the femoral head and neck seen in coxa plana, and it also explains the conflicting conclusions drawn by certain previous authors regarding the final deformation in this disease. Thus, LEVY (1911), for instance, described cases in which the femoral head occupied a varus position in relation to the neck and suggested that the name of the condition, osteoarthritis deformans coxae juvenilis, should be substituted by »coxa vara capitalis». DREHMANN (1914), on the other hand, reported cases with a valgus position of the femoral head in relation to the neck and suggested the denomination »coxa valga epiphysaria».

Analogous deformities following partial epiphyseal closure are known from other skeletal diseases. In tibia vara, for instance, premature closure of the medial portion of the proximal epiphyseal line of the tibia is a common phenomenon and leads to a marked increase of the varus deformity (A. LANGENSKIÖLD 1952).

The degree of the deformity under discussion is, of course, entirely dependent on how long the growth continues and how rapid it is in still functional parts of the growth zone after partial fusion of the epiphyseal line has taken place.

Thickening of the metaphysis. The cause of the increase in breadth of the metaphysis has been discussed by many authors. BERNBECK (1954) regarded this broadening in part as initial apposition of osseous lamellae caused by the irritation resulting from overgrowth of the epiphysis over the metaphyseal border, in part as appositional increase in thickness implying strengthening of weak osseous parts.

HELBO (1954) who — like many other authors — observed periosteal apposition in the lateral surface of the femoral neck, accorded no major significance to this phenomenon from the standpoint of the thickening of the femoral neck. He observed the same periosteal reaction in an early stage in patients who later exhibited good end-results, without any thickening of the neck of the femur. HELBO regarded the metaphyseal changes and weight-bearing as responsible for the increase in breadth.

In the present series there are cases in which periosteal apposition undoubtedly seemed to contribute to the increase in breadth of the femoral neck, but in the great majority of cases no such reaction of the periosteum was observable. As evaluated on the basis of the radiographs, the thickening of the femoral neck was obviously due to growth phenomena in the metaphysis, which is the region where the increase in breadth appears first and is most conspicuous.

An increase in volume of the proximal end of the femur is relatively frequent after reduction of congenital luxation of the hip, and also occurs in cases where no radiological signs of epiphyseal necrosis are observable. Such a coxa magna is attributed to local growth-stimulating phenomena, possibly elicited by the reposition trauma.

It seems probable that similar stimulating factors also play a part in coxa plana.

Formation of a step in the metaphysis. A. LANGENSKIÖLD (1952) described the radiological changes in the infantile type of tibia vara (osteochondrosis deformans tibiae). Some cases exhibited a defect in the proximal metaphysis of the tibia causing a definite step. The defect was attributed to growth arrest in one portion of the metaphysis and continued ossification in the remainder. The step-shaped defect was filled out by a broadened part of the epiphyseal cartilage, and in a later stage it was found to be occupied by the bony epiphysis (Fig. 22).

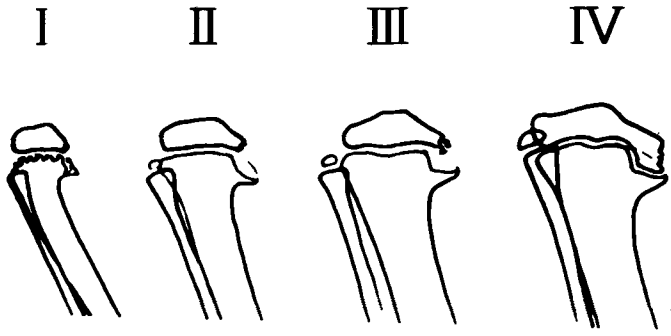

Fig. 22. Formation of a step-shaped metaphyseal defect in tibia vara. Compensatory epiphyseal growth, the defect is occupied by the bony epiphysis. (According to A. LANGENSKIÖLD, 1952).

By mechanical injury to the proximal tibial growth cartilage in rabbits, A. LANGENSKIÖLD (1955) provoked changes resembling those seen in tibia vara, *i.e.* a metaphyseal depression, compensatory growth of the epiphysis and a varus deformity (Fig. 23).

Metaphyseal defects associated with osteomyelitis and tuberculosis in children are relatively frequent. A. LANGENSKIÖLD (1955) has described compensatory epiphyseal filling of these defects and emphasized the resemblance between this regenerative process and that observed by him in tibia vara (Fig. 24).

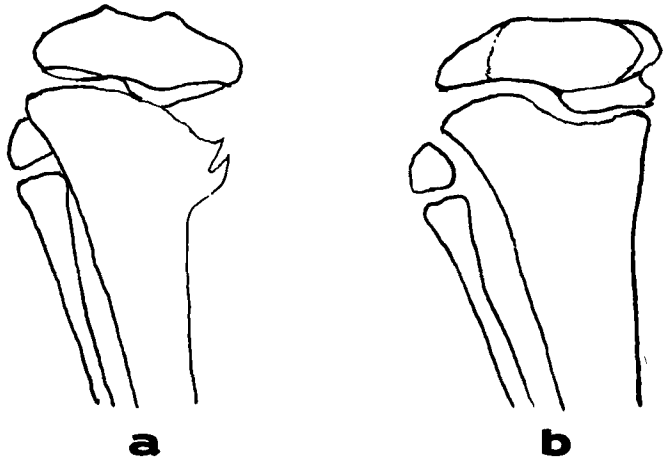

Fig. 23. a: drawing of a radiograph of the tibia of a young rabbit taken 10 days after removal of the medial part of the zone of cartilage cell columns from the proximal epiphyseal plate. There is an oblique defect in the metaphysis. *b:* drawing of a radiograph of the upper end of the tibia of a rabbit taken 16 days after removal of the medial part of the zone of cartilage cell columns from the epiphyseal plate. The metaphyseal defect is occupied by an extension of the bony epiphysis. (According to A. LANGENSKIÖLD, 1954).

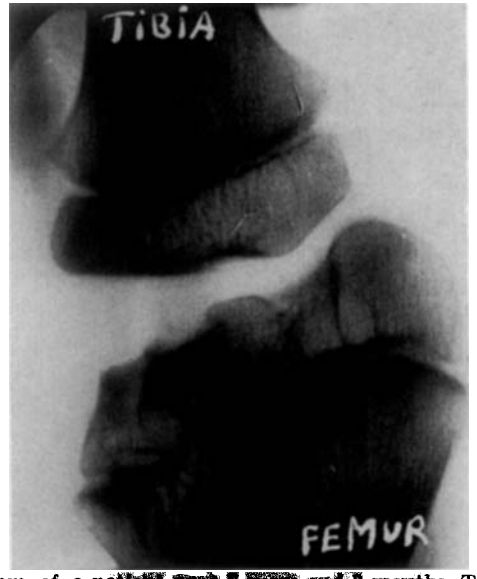

Fig. 24. A tomogram of a patient aged 7 years and 3 months. The steplike deformity had developed in the distal growth zone of the femur after osteomyelitis in infancy. Compare with *Fig. 17 b.* The resemblance of the changes is striking. (Reproduced by courtesy of LANGENSKIÖLD & RISKA, and the *J. Bone & Joint Surg.*, Am., 1964.)

A similar mechanism appears to be responsible for the development of the metaphyseal defect observed in the present study: A lesion of a limited area of the meta-epiphyseal zone leads to inhibited ossification of this area, while the ossification of the surrounding tissues continues.

Ossification defects of the vertebrae occur in juvenile osteochondrosis. Such defects of the lumbar vertebrae have been described by KNUTSSON (1948), BROCHER (1953), EDGREN & VAINIO (1957) and others. Serial radiographs have shown that local inhibition of the ossification in the cranial or caudal surfaces of the vertebral bodies initially causes a step-shaped defect, as a rule in the ventral edge of the vertebral body. As ossification progresses, the edge of the vertebral body may become completely ossified, while the original defect remains as a paradiscal depression in the site where it developed. In cases with severely disturbed ossification, the defect remains

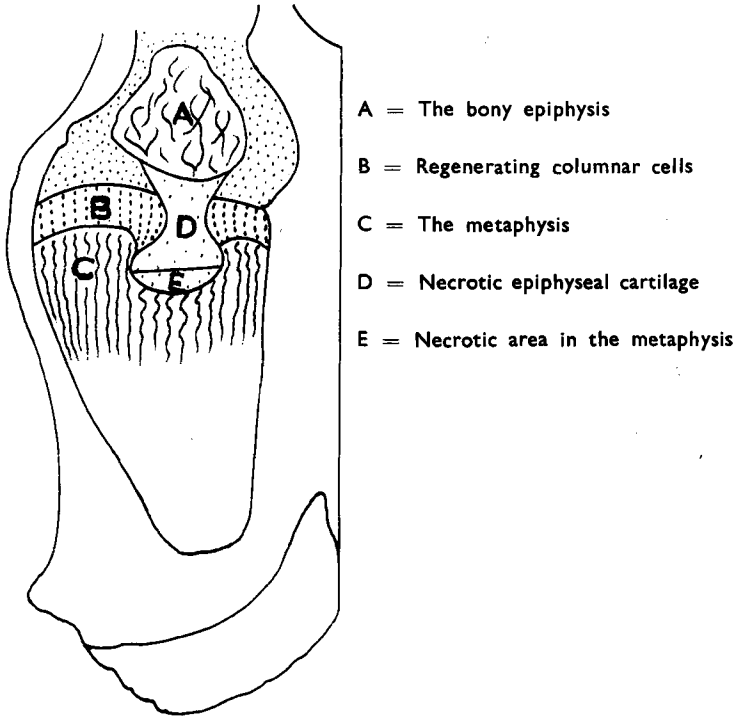

Fig. 25. Proximal end of the fibula of a rabbit transplanted to the thigh at the age of 21 days, period of observation 7 days, schematic drawing. The necrosed epiphyseal cartilage has been replaced from the sides, while it has been left behind in the metaphysis. On the metaphyseal side of the necrotic cartilage the bony trabeculae lack an osteoblast lining and in the interspaces there is detritus. (According to HEIKEL).

marginal and step-shaped and increases as the vertebra grows. These changes seem to be comparable with the metaphyseal changes in coxa plana.

In connexion with experimental epiphyseal transplantation, HEIKEL (1960) made histological studies a few days after this procedure. The central portion of the *epiphyseal cartilage exhibited extensive necrosis, caused by interruption of the vascular supply.* Subsequently, the necrotic cartilage tissue remained in the same site, while ossification continued in the surrounding non-necrotic areas. In this way a central defect in the metaphysis developed, the marginal area of which was initially more or less step-shaped (Fig. 25).

On comparison of the radiological sequence of events in coxa plana, tibia vara, experimentally induced tibia vara, inflammatory metaphyseal defects and cartilage necrosis after experimental epiphyseal transplantation, a striking resemblance emerges.

On the basis of previous reports concerning the histological changes of the epiphyseal plate in coxa plana (RIEDEL, PONSETI, YAMAGUCHI), in cases showing necrosis of the proximal fragment after fracture of the femoral neck (AXHAUSEN, 1922) and in tibia vara (A. LANGENSKIÖLD 1952), and considering the findings of HEIKEL cited above regarding the necrosis of growth cartilage, it seems obvious that *necrosis of the cartilage tissue in the epiphyseal plate is a pathogenetic factor which these disease conditions have in common.*

VII. THE PATHOGENESIS OF COXA PLANA

It is a generally accepted view, based on HAYTHORN's, JONSÄTER's and TRUETA & PINTO DE LIMA's histological investigations, among other things, that the coxa plana process is histopathologically an avascular necrosis. The primary cause of the vascular disturbance is still obscure, however.

The circulatory conditions in the proximal part of the femur have been studied by WALDENSTRÖM (1910), NUSSBAUM (1924), WOLCOTT (1943), TUCKER (1949), TRUETA & HARRISON (1953), TRUETA (1957), TRUETA & PINTO DE LIMA (1959), HIPP (1962), and others. According to TRUETA, in the coxa plana age the bony epiphysis is mainly supplied by the lateral epiphyseal arteries only, which depart from the medial circumflex artery. The central portion of the metaphysis is supplied by the nutrient artery, while the peripheral parts are supplied by the superior and inferior metaphyseal arteries, which are both branches of the medial circumflex artery. At this age there are no anastomoses between the vessels through the epiphyseal plate (TRUETA & MORGAN 1960).

On the assumption that coxa plana is an avascular necrosis of the femoral epiphysis and surrounding parts of the metaphysis and the epiphyseal cartilage, and considering the nutritional conditions in the proximal part of the femur, it may be suggested that the radiological course in this disease has the following pathogenetic background:

Owing to a cause not yet established (mycotic embolism as suggested by AXHAUSEN 1922, cartilage oedema resulting from trauma or infection as suggested by BERNBECK 1954, compression via the outer rotator muscles as suggested by TRUETA 1956), the lateral epiphyseal vessels are obstructed. This leads to more or less extensive necrosis, usually in the ventrolateral portion of the epiphysis and corresponding areas of the epiphyseal cartilage and the metaphysis. Radiologically this is evidenced by the changes described in the foregoing, *i.e.* condensation, flattening and fragmentation of the epiphyseal osseous centre and a defect in the periphery of the metaphyseal surface. In the central portion of the metaphyseal surface, which is supplied by the nutrient artery, ossification continues, which results in growth of this

part in the proximal direction. Resorption and reossification of the epiphysis start in the peripheral portions, as a rule ventrolaterally (as is demonstrated by numerous radiographs in this study), which is probably due to revascularization via the periosteum (the synovial membrane) occurring first in these areas. The fate of the metaphyseal defect is dependent on the severity of the cartilage lesion. To begin with this defect is filled out by the chondroepiphysis. If the lesion in the zone of ossification is slight, ossification commences at the margins of the metaphyseal defect which is made good, as it were, from the metaphysis. In this case the epiphyseal plate reassumes a relatively regular outline. If the lesion of the cartilage is severe, the metaphyseal defect remains and is gradually filled out by the bony epiphysis. Probably, the lesion in the epiphyseal plate is often of such a kind that contact is established between the vascular system in the epiphysis and the metaphysis, with local fusion of the epiphyseal line resulting (NOVÉ-JOSSERAND 1894, A. LANGENSKIÖLD & EDGREN 1949, TRUETA & AMATO 1960). It seems possible that the compression due to weight-bearing is a contributory cause of premature closure of the epiphyseal line (TRUETA & TRIAS 1961), particularly in cases in which the central portion of the metaphysis has grown markedly and the zone of ossification is protected only by a very thin layer of cartilage.

VIII. THE GREATER TROCHANTER

Earlier observations

In his paper of 1913, PERTHES pointed out that the greater trochanter was strikingly large in some of his cases. Similar observations were reported by AXHAUSEN (1923). These authors attributed the hypertrophy to proliferative phenomena around the greater trochanter. BIBERGEIL (1912), who also noted that the trochanter was large, correlated this finding with atrophy of the femoral head and neck.

SUNDT (1920) described in his monograph some cases of coxa plana with hypertrophy of the greater trochanter. Since in some of these the trochanter was also hypertrophic on the unaffected side, he drew the conclusion that the hypertrophy had no connexion with the disease process in coxa plana.

In his paper called »The definite form of the Coxa plana» WALDENSTRÖM (1922) described the radiological picture in his end-result group 3 as follows: »The upper pole of the caput is edgeformed and usually lower than the summit of the greater trochanter», but he offered no explanation of this observation.

CAAN (1924), also, discussed this phenomenon without being able to trace its cause.

PERTTILÄ (1954) reported that in 10 out of 33 patients with coxa plana who were followed up, the tip of the greater trochanter was situated more cranially than the proximal pole of the femoral head. The elevation of the tip of the greater trochanter was not accounted for.

GOFF (1954) and HORWITZ (1960) published radiographs of elevated greater trochanters, but did not discuss the cause of the elevation.

Observations on the present material

In the present series no radiological changes indicative of necrosis were observable in the greater trochanter.

As appeared in the foregoing, retardation of the longitudinal growth of the femoral neck and premature closure of the subcapital epiphyseal line

are of frequent occurrence in coxa plana. As a rule, the growth of the greater trochanter was not affected by the process. Since the longitudinal growth of the femoral neck may cease completely at the age of 12 to 14 years, while the growth of the greater trochanter continues until the age of 17 to 18 years and sometimes longer, it is obvious that a discrepancy between the femoral head and neck on the one hand and the greater trochanter on the other must result. On the radiographs this was discernible as a reduction of the distance between the tip of the greater trochanter and the proximal pole of the femoral head. In the present paper this distance is called the *articulo-trochanteric distance.*

For the sake of assessing the influence of the process of the disease in coxa plana on the articulo-trochanteric distance, measurements were made on the radiographs. For this purpose perpendiculars were drawn against the longitudinal axis of the femoral diaphysis so as to touch the proximal pole of the femoral head and the tip of the greater trochanter. The distance between these perpendiculars was measured in millimetres. In what follows, it is denominated ATD (Fig. 26). If the tip of the greater trochanter was situated distally of the proximal aspect of the femoral head, the ATD was regarded as positive. It was regarded as negative when the tip of the greater trochanter was situated proximally of the proximal aspect of the head of the femur.

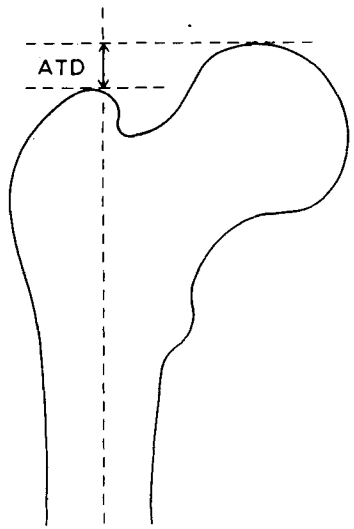

Fig. 26. Determination of the articulo-trochanteric distance, ATD.

In order to find out to what extent the ATD is influenced by different rotational positions of the femur, frontal radiographs were taken of 12 hip joints in the neutral position and in 20 degrees outward and inward rotation. The radiographs were taken by the same technique as was used in examining the coxa plana patients. In addition, a specimen was radiographed in the same way. As compared with the neutral position, the ATD was found to increase on outward rotation and decrease on inward rotation. The results of these tests are shown in Table 32. Although the differences were small, they may have influenced the measurements in the present series to some degree. The errors were reduced, however, by the radiographic technique, which was elaborated so as to attain the greatest possible degree of standardization of the projections. The severest deformities, involving the largest reductions of the ATD, were often associated with a slight outward rotation of the extremity and a limitation of the 20 degrees' inward rotation which is a condition of our standard technique. The readings for the ATD therefore represent maximum values.

TABLE 32. — *ATD in 12 normal hip joints and in a specimen in the neutral position, outward rotation and inward rotation.*

	Articulo-trochanteric distance	
	Mean in 12 normal hips, mm.	Specimen, mm.
Neutral position	21	18
Outward rotation	24	20
Inward rotation	19	14

The results obtained after the completion of growth in 81 unilateral cases of coxa plana are shown in Table 33.

TABLE 33. — *ATD in unilateral coxa plana after completion of growth in relation to shape of the femoral head.*

Shape of the femoral head	Articulo-trochanteric distance				
	Affected hips		Unaffected hips		No. of cases
	Mean	(Range)	Mean	(Range)	
Spherical	11.7	(+21— — 9)	16.0	(+24— + 6)	23
Elliptical	1.7	(+13— — 4)	15.9	(+28— +11)	17
Irregular	2.2	(+17— —19)	15.1	(+28— + 8)	41

The results obtained in the same group at an earlier stage of the disease, before closure of the subcapital epiphyseal line, are shown in Table 34.

Table 35 shows the results of measurement after the completion of growth in 20 bilateral cases of coxa plana.

From Tables 33 and 34 it appears that no reduction of the ATD was primarily present (it occurred at a late reparative stage, in part only when the femoral head had attained its final shape). The reduction of the ATD increased with poorer end-results in the head, although it was sometimes marked also in cases healing with a spherical head.

TABLE 34.

Shape of the femoral head	Articulo-trochanteric distance			
	Affected hips		Unaffected hips	
	Mean	(Range)	Mean	(Range)
Spherical	17.2	(+24— + 5)	16.8	(+26— + 8)
Elliptical	14.8	(+18— +12)	16.9	(+22— +12)
Irregular	14.1	(+26— + 5)	16.4	(+30— + 8)

TABLE 35. — *ATD in relation to ultimate shape of the femoral head after completion of growth in 20 cases of bilateral coxa plana.*

Shape of the femoral head	Articulo-trochanteric distance		No. of hips
	Mean	(Range)	
Spherical	10	(+18— — 3)	13
Elliptical	8.8	(+15— + 1)	4
Irregular	0.9	(+26— —19)	23

In Table 36 the ATD in the group with unilateral coxa plana showing premature closure of the epiphyseal line is compared with the ATD in the group showing normal closure. It is seen in the table that the ATD was markedly reduced in the group showing premature closure. In the group showing normal closure the reduction of the ATD was slight, as compared with the unaffected side. Fig. 27 shows the results of measurement of the ATD in these groups.

TABLE 36. — *ATD in unilateral, ultimately healed coxa plana with premature closure (42 cases) and normal closure (20 cases) of the subcapital epiphyseal line.*

Closure of the epiphyseal line	Articulo-trochanteric distance				No. of hips
	Affected hips		Unaffected hips		
	Mean	Range	Mean	Range	
Premature	1.3	+13— —11	16.2	+28— +5	42
Normal	13.7	+23— + 5	15.4	+24— +6	20

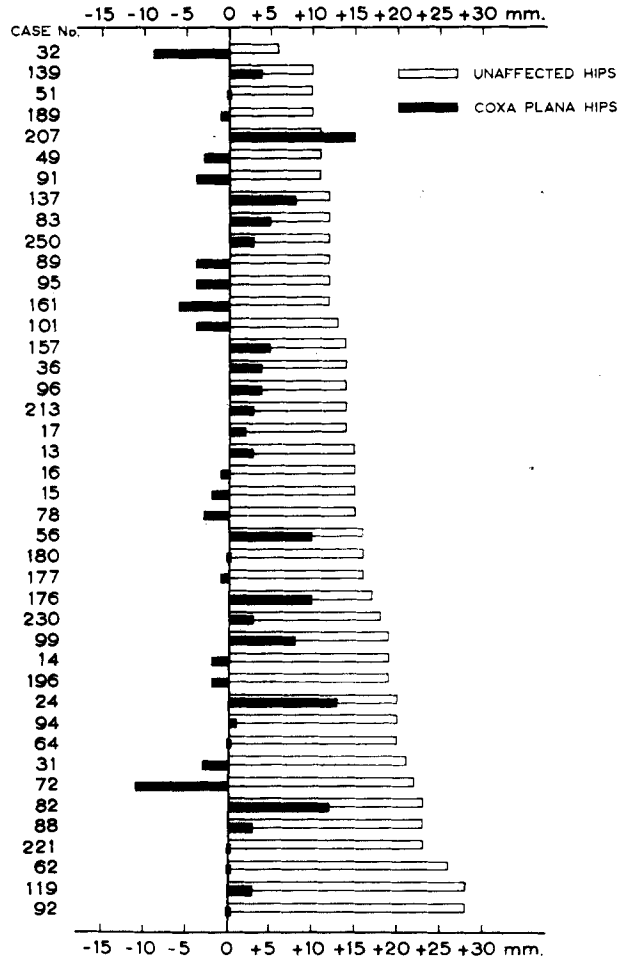

Fig. 27 a.

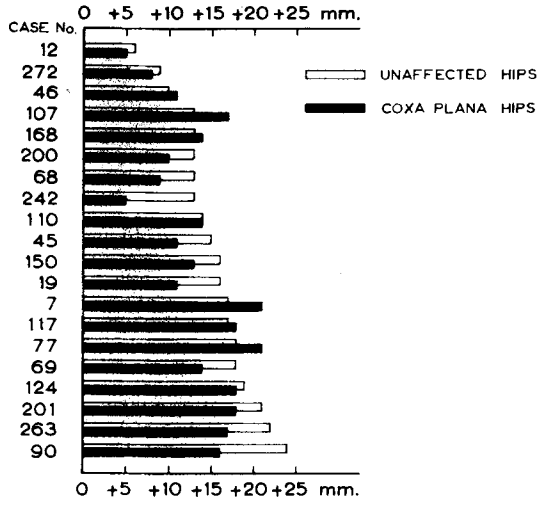

Fig. 27 b.

Fig. 27. a: the articulo-trochanteric distance in 42 cases showing premature closure of the epiphyseal line, and *b:* in 20 cases showing normal closure of the epiphyseal line.

Case 230 in the present series is a typical example of reduction of the ATD in connexion with premature closure of the subcapital epiphyseal line. The essential data are listed in Table 37.

The series includes 7 cases in which the radiographs were indicative of premature and simultaneous closure of the subcapital growth line and the growth line of the greater trochanter. In these cases, 5 of which healed

TABLE 37. — *Reduction of ATD in a typical case of coxa plana. Case 230. Boy aged 8 at onset in Sept. 1957. Right side affected.*

Date	Right hip Condition of the epiph. lines		ATD		Left hip. Condition of the epiph. lines	
	Subtro-chanteric	Subcapital	Right	Left	Subcapital	Subtro-chanteric
April 1959 ..	Open	Open (Reparat. st.)	+ 15	+ 16	Open	Open
April 1960 ..	»	Very irregular	+ 14	+ 15	»	»
Oct. 1960 ..	»	Partly closed	+ 12	+ 16	»	»
April 1961 ..	»	More closed	+ 10	+ 15	»	»
Oct. 1961 ..	»	Almost closed	+ 8	+ 17	»	»
Oct. 1962 ..	»	Closed	+ 3	+ 18	Partly closed	»
Jan. 1964 ..	Closed	»	+ 3	+ 18	Closed	Closed

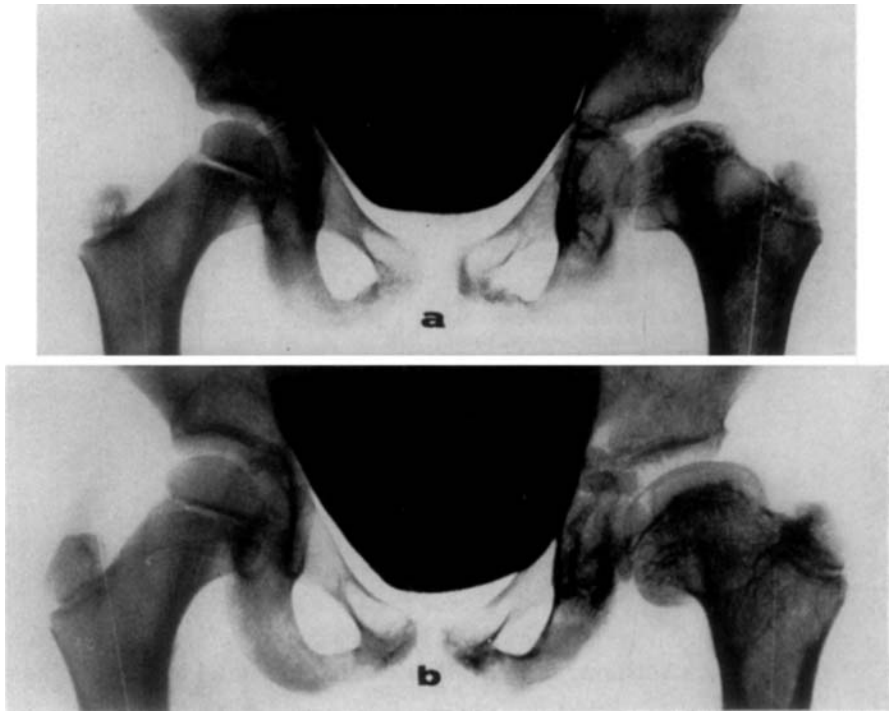

Fig. 28 a—b.

with irregular femoral heads, 1 with an elliptical and 1 with a spherical femoral head, no major reduction of the ATD occurred. On the affected side this distance was a mean of 14.6 mm (the range being from +9 to +26 mm) and on the unaffected side a mean of 15.9 mm (the range being from +7 to + 25 mm). In some of these cases the premature closure of the epiphyseal line of the greater trochanter was apparently due to the epiphysis having grown out over the cranial aspect of the femoral neck, so as to come into contact with the trochanter at its base close to the epiphyseal line. It may be assumed that vascular anastomoses between the epiphysis of the trochanter and the metaphysis had been established, with fusion of the growth line resulting in the same way as in the subcapital region. In some cases this mechanism was obviously not responsible, however, and the radiographs afford no explanation of the premature closure of the growth line of the greater trochanter.

From the observations made in the present series concerning the growth of the proximal part of the femur the conclusion may be drawn that *the*

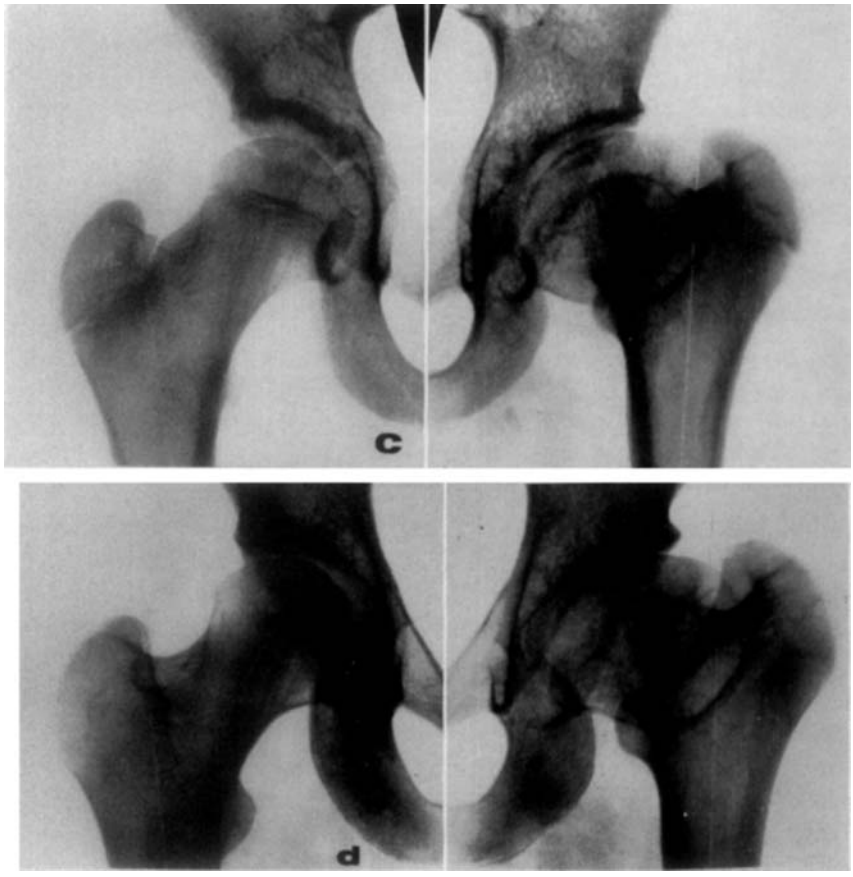

Fig. 28. Case 161, girl aged 7 years at onset. Treated for 9 months with Thomas' splint in an advanced stage of the disease. *a:* September 1954, late reparative stage, the epiphyseal line very irregular. *b:* August 1956, primary healing, the subcapital epiphyseal line partially closed, ATD + 11 mm on the left side, + 16 mm on the right side, Trendelenburg's sign negative. *c:* October 1959, the subcapital epiphyseal line closed, the subtrochanteric epiphyseal line still open. ATD left — 3 mm, right + 12 mm. Trendelenburg's sign positive to the left. *d:* February 1963, all growth lines closed, ATD left — 6 mm, right + 12 mm. The left femoral head slightly irregular, enlarged. Normal mobility in the left hip. Trendelenburg's sign positive.

elevation of the greater trochanter in coxa plana is due to retardation of the longitudinal growth of the femoral neck and premature closure of the subcapital epiphyseal line, while the growth of the greater trochanter proceeds as normal throughout the remainder of the growing period.

Fig. 28 (case 161) illustrates the elevation of the greater trochanter associated with premature closure of the subcapital epiphyseal line.

IX. THE DEFORMITY IN COXA PLANA

VARUS OR VALGUS

Earlier observations. According to HOFMEISTER's (1894) definition, coxa vara is present if the neck-shaft angle is smaller than normal (120 degrees in adults, v. LANZ-WACHSMUTH 1938). This definition does not take the greater trochanter into account, which is an integrating part of the proximal end of the femur and derives its epiphyseal plate from the same preplate as the femoral head (see p. 23). SOURDAT (1909) and CALVÉ (1910) stated that coxa plana leads to a varus deformity. WALDENSTRÖM (1910), who observed no appreciable alteration of the neck-shaft angle in coxa plana, did not regard the deformity caused by the disease as a varus deformity, but he pointed out that an apparent varus deformity may result if the medial portion of the femoral head grows in the medio-caudal direction. Many other authors have adopted the same view, *e.g.* HORWITZ (1960) and LEGER (1961). The latter wrote: »Die Perthesche Erkrankung ist zu der durch Wachstumsstörungen bedingten Coxa vara im engeren Sinne nicht zu rechnen». GOFF (1954) reported that he had not observed any varus deformity in his series except in the group »irregular type», in which no coxa vara was present at first but sometimes developed soon enough as an osteomalacia of a local character.

SUNDT (1949) measured the neck-shaft angle on the radiographs taken at follow-up investigation of his series. He found that the angle was almost invariably enlarged and concluded: »The disease has thus an unmistakeable tendency to produce a valgus position of the neck.»

PERTTILÄ (1954), too, measured the neck-shaft angle and found that the coxa valga type was clearly prevalent when the determinations were made on neutral position radiographs. He emphasized, however, that the values obtained by this method were too high. It seems probable that the large neck-shaft angles noted by SUNDT were also due to the fact that the measurements were made on radiographs taken in the neutral position, or possibly in outward rotation.

PERTTILÄ regarded functional coxa vara as being present in those cases where the greater trochanter is elevated.

EVANS (1958) determined the neck-shaft angle in a series of 52 patients. A valgus deformity was present in 6 cases, in which the angle was increased by over 10 degrees as compared with the unaffected side. In no case was any marked varus deformity noted. The author pointed out that marked shortening of the head-neck segment is shown clinically by elevation of the greater trochanter.

In the present series no major changes of the neck-shaft angle were observed. The measurements were made on radiographs taken in 20 degrees inward rotation.

LANGENSKIÖLD & SARPIO (unpublished) and LAURENT (1959) showed in animal experiments, LAURENT (1959) and PYLKKÄNEN (1960) in clinical series, that a coxa vara deformity developed when the activity of the subcapital epiphyseal plate was disturbed or prematurely came to a standstill, while a coxa valga deformity resulted when the activity of the epiphyseal plate of the greater trochanter was disturbed or prematurely ceased.

Discussion. In the *distal end of the femur* the epiphysis consists of two condyles originating from a common growth plate. If the growth process is inhibited in the growth zone of the medial condyle, while the lateral condyle continues to grow, a varus deformity develops. *Vice versa*, if the growth of the lateral condyle is inhibited, a valgus deformity results.

In certain mammals, *e.g.* the elephant, the preplate is not differentiated into a separate epiphyseal plate for the femoral head and another for the greater trochanter. The head and the trochanter grow from the same growth plate (LÜTKEN 1961). In rare cases a bony bridge is formed between the capital epiphysis and the bony nucleus of the trochanter in man, so that a common, uniform epiphyseal plate for the femoral head and the greater trochanter develops. LÜTKEN described 3 such cases and MAU (1962) also described 3. At the Orthopaedic Hospital of the Invalid Foundation I have observed 2 such cases in which the formation here described developed after closed reduction of a congenital hip luxation (Fig. 29). In such cases, similar conditions prevail in the proximal and distal ends of the femur, which justifies a comparison of these structures. The head and the greater trochanter may be compared with the two condyles. If the growth of the medial, subcapital region is arrested, while the lateral, trochanteric area continues to grow, a varus deformity develops, and *vice versa*, if the growth of the trochanteric region is arrested while the subcapital region continues to grow, a valgus deformity results.

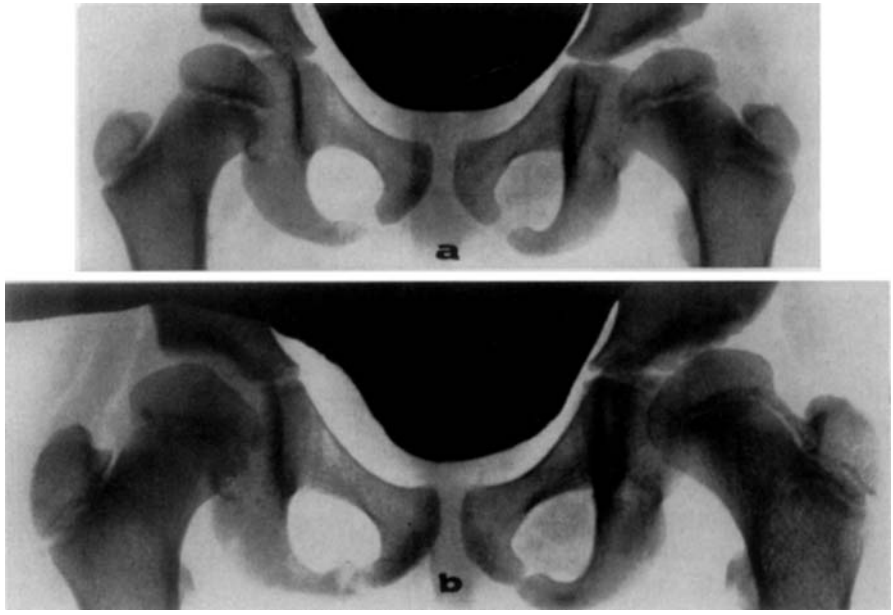

Fig. 29. Formation of a bony bridge between the capital epiphysis and the bony nucleus of the greater trochanter in a girl treated by closed reduction of a congenital hip luxation. In *b* the bridge is complete and there is a common, uniform epiphyseal plate for both the growing centres in the upper part of the femur.

Against this background, the greater trochanter cannot be ignored when assessing a possible varus or valgus deformity in the proximal end of the femur.

As appears from the foregoing, retardation of the longitudinal growth of the femoral neck and premature closure of the subcapital epiphyseal line are very common phenomena in coxa plana. By contrast, the growth cartilage of the greater trochanter remains in most cases intact and functional. The result of this is a shortening of the medial structures, head — neck, and a relative lengthening of the lateral structure, *i.e.* the greater trochanter, or in other words, *a real varus deformity.*

Fig. 30 is a diagrammatic representation of different types of varus deformity due to growth arrest in different portions of the subcapital epiphyseal plate.

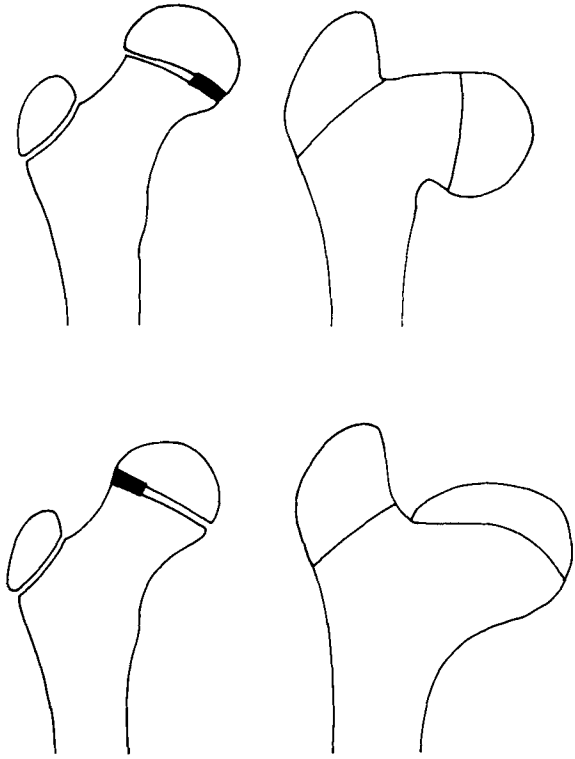

Fig. 30. Diagrammatic representation of different types of varus deformity. Above: growth arrest in the medial part of the epiphyseal plate leads to a varus deformity with diminished neck-shaft angle. Below: growth arrest in the lateral part of the epiphyseal plate; the epiphysis assumes a valgus position in relation to the shortened femoral neck; the neck-shaft angle is not appreciably influenced; this type is common in coxa plana.

X. TRENDELENBURG's SIGN

Earlier investigations

The greater trochanter is the site of insertion of the abductors of the femur, in the first place the gluteus medius and gluteus minimus. From the standpoint of the function of the pelvitrochanteric abductor muscles and the statics in the pelvic hip region, the position of the greater trochanter in relation to the pelvis is of fundamental importance. In the presence of a coxa vara deformity, *e.g.* in coxa vara infantum, the function of the pelvitrochanteric abductors is impaired owing to elevation of the greater trochanter. The abductor insufficiency is manifested by a positive Trendelenburg sign (v. LANZ-WACHSMUTH 1938, PYLKKÄNEN 1960).

Limping is a constant early symptom in coxa plana. This form of limp was described by CALVÉ *et al.* (1939) as »antalgic gait», and it differs from Trendelenburg's type. At the time of primary healing, the limp, if there is any, is of a neutral type, due to shortening of the limb (CALVÉ *et al.* 1939, GOFF 1954). If the disease leads to a varus deformity, *i.e.* if the greater trochanter is elevated in relation to the hip joint and if abductor insufficiency develops, the limp is of Trendelenburg's type.

Observations on the present material

In the present series a positive Trendelenburg sign was noted in 26 cases of ultimately healed unilateral coxa plana. In 15 of these the ATD was 0 or negative *ad* — 9 mm. In 9 cases the ATD varied between + 1 and + 5 mm. In 1 case the ATD was + 8 mm in the coxa plana hip against + 23 mm in the unaffected hip, which implies a marked reduction. In 1 case no appreciable reduction of the ATD was demonstrable (+ 12 mm against + 13 mm in the unaffected hip). In this case the positive Trendelenburg sign must be attributed to some other cause than reduction of the ATD, probably to muscular insufficiency.

Among the 26 cases with positive Trendelenburg signs there were 9 in which a retrospective examination of the clinical data and the radiographs

revealed that Trendelenburg's sign had been negative at a late reparative stage of the disease, and that no or only a slight reduction of the ATD had then been present (Fig. 28).

As far as the shape of the femoral head is concerned, the radiological end-results were mostly poor in this group. Two cases healed with spherical heads, 7 with elliptical and 17 with irregular heads.

The present observations in the cases with positive Trendelenburg signs show that *abductor insufficiency in coxa plana may develop at a very advanced stage of the disease, as a result of elevation of the greater trochanter.*

XI. PREVENTION OF DEFORMITY BY EPIPHYSIODESIS OF THE GREATER TROCHANTER

In 1961, epiphysiodesis of the greater trochanter was performed at the Orthopaedic Hospital of the Invalid Foundation in 3 cases of coxa plana. In 2 of these, the ATD was markedly reduced at the time of the operation, *i.e.* + 2 and — 4 mm in the affected hips against + 12 and + 4 mm on the unaffected side. In the latter case moderate bilateral coxa vara was already present at the time of the diagnosis. The deformity was probably due to rickets. In the third case epiphysiodesis was performed at a late reparative stage. On the affected side the subcapital epiphyseal line was partially closed, but the ATD was not reduced; it was + 12 mm on both sides. The operation was performed by PHEMISTER's (1933) method, first applied to the greater trochanter by A. LANGENSKIÖLD in 1957 (personal communication).

On follow-up examination of these patients 17, 22 and 14 months, respectively, after operation the growth line of the greater trochanter was found to be closed in all cases. In the first two cases the reduction of the ATD had increased by 2 mm on the side operated upon and was thus 0 and — 6 mm, respectively. Trendelenburg's sign was in these cases weakly positive. In the third case the ATD had been enlarged by 1 mm and was + 13 mm on both sides. Trendelenburg's sign was negative.

During the years 1963 and 1964, epiphysiodesis of the greater trochanter was performed in 20 unilateral and 2 bilateral cases of coxa plana, in which radiological studies revealed progressive reduction of the ATD or premature closure of the subcapital epiphyseal line. Thirteen of these cases do not belong to the present series.

In Table 38 the principal data regarding the patients operated upon are compiled.

Discussion

It appears from Table 36 that the reduction of the ATD was arrested by epiphysiodesis of the greater trochanter. In the cases which were followed up, the postoperative reduction of the ATD was a maximum of 2 mm except in 2 cases (J. K. and J. Y.) in which the ATD had been reduced by 4 and 3 mm, respectively. In the bilateral case L. J. the ATD on the operated, right side decreased by 1 mm during the time of observation, while the reduction of the ATD on the non-operated, left side was 13 mm during the same period. In 8 cases the ATD had been enlarged by 1 mm.

In all cases which have been followed up, the epiphysiodesis led to partial or complete fusion of the growth line of the trochanter (Fig. 31).

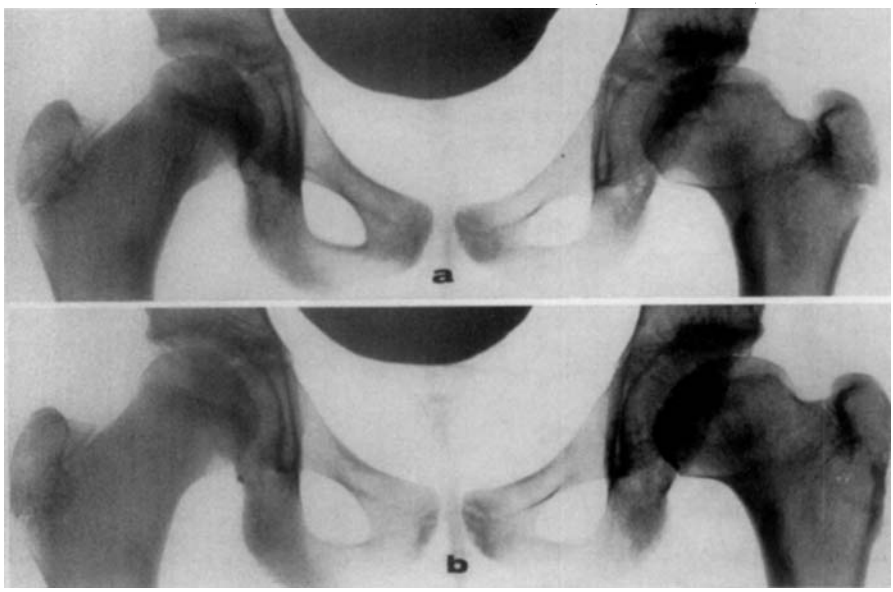

Fig. 31. Case M-L. R. (not in the present series), girl aged 6 years at onset. *a:* in June 1964, five and a half years after onset; the subcapital epiphyseal line is partially closed on the affected left side; ATD is + 3 mm and had decreased from + 7 mm in Dec. 1962. Epiphysiodesis of the left greater trochanter was performed in June 1964. *b:* Oct. 1964. The subtrochanteric epiphyseal line is closed on the left side. ATD is + 4 mm on the left side and + 8 mm on the right side, as was the case at two previous examinations. Trendelenburg's sign was negative.

In many cases the operation was performed late, when marked elevation of the greater trochanter was already present.

If epiphysiodesis of the greater trochanter is performed at a favourable point of time, it appears to be possible to prevent elevation of the trochanter and consequent abductor insufficiency.

TABLE 38. — *Coxa plana cases in which epiphysiodesis*

Case	Year of birth	Stage of process at time of oper.	Condition of epiphyseal line at time of oper.		Articulo-tro-		
					At time of operat.		Time of oper.
			Subcapital	Sub-trochant.	Unaf-fected hip	Affected hip	
L.M-L.	1947	Late repar.	Part. closed	Open	+12	+ 2	Oct. 1961
M.U.	1947	Repar.	»	»	+ 4	— 4	»
V.N.	1947	Late repar.	»	»	+12	+12	Nov. 1961
I.K.	1952	Repar.	Open, irreg.	»	+18	+12	Feb. 1963
L.J.	1949	R. Prim. heal.	Closed	»	—	+ 7	Mar. 1963
		L. »	»	»	—	+16	Un-oper.
V.T.	1948	Late repar.	»	»	+19	— 4	June 1963
E.V.	1950	Prim. heal.	»	»	+11	— 3	Oct. 1963
R.L.	1952	Repar.	Open, irreg.	»	+10	+ 1	Jan. 1964
J.Y.	1950	Late repar.	»	»	+15	+11	»
P.V.	1952	Prim. heal.	Part. closed	»	+22	+ 8	Feb. 1964
P.S.	1950	Late repar.	Open, irreg.	»	+14	+11	Mar. 1964
A.N.	1949	Prim. heal.	Part. closed	»	+14	+ 3	»
P.M.	1951	R. Repar.	Closed	»	—	0	»
		L. Prim. heal.	»	»	—	0	»
P.K.	1953	Repar.	Open, irreg.	»	+16	+ 2	»
L.M.	1953	Prim. heal.	Part. closed	»	+15	+ 9	May 1964
M-L.R.	1953	Late repar.	»	»	+ 8	+ 3	June 1964
E.K.	1950	Prim. heal.	Closed	»	+25	+ 6	July 1964
O.V.	1950	Repar.	»	»	+11	+ 8	»
A.V.	1951	Late repar.	Part. closed	»	+17	+ 1	»
A-M.L.	1951	Prim. heal.	»	»	+13	+ 4	»
R.Y.	1955	Late repar.	»	»	+17	+12	Aug. 1964
A.R.	1954	»	»	»	+14	+ 5	Sept. 1964
R.V.	1954	Prim. heal.	Open, irreg.	»	+11	— 1	»
P.M.	1955	Repar.	»	»	+16	+15	Oct. 1964
H.S.	1953	»	»	»	+12	+10	»

of the greater trochanter was performed.

chanteric distance		Condition of epiphyseal line after operation		Observation time after operation in months
After operation				
Affected hip	Unaffected hip	Subcapital	Subtrochanteric	
0	+13	Closed	Closed	17
— 6	+ 2	»	»	34
+13	+13	»	»	14
+ 8	+15	»	Part. closed.	19
+ 6	—	»	Closed	15
+ 3	—	»	Open	15
				—
— 4	+14	»	Closed	11
+ 2	+ 9	Part. closed	Part. closed	10
+ 8	+15	»	»	6
+ 6	+19	»	»	10
+11	+14	Open, irreg.	»	12
+ 4	+15	Closed	Closed	9
+ 1	—	»	»	6
— 2	—	»	»	6
+ 3	+16	Open, irreg.	»	6
+ 8	+16	Part. closed	»	4
+ 4	+ 8	Closed	»	4
+ 7	+26	»	»	3
+ 8	+11	Part. closed	»	3
+ 1	+17	Closed	»	7
+ 3	+13	»	»	4
+11	+16	»	»	6
+ 4	+15	Part. closed	»	8
				—
+16	+16	Open, irreg.	»	5
+10	+12	»	»	5

XII. SHORTENING OF THE EXTREMITY IN COXA PLANA

Observations on the present material.

On clinical examination of the present patients at the end of the growing period, a shortening of the affected extremity varying between 0.5 and 3.5 cm was observed in the majority of cases.

Orthoradiographic measurement of the lower extremities was performed in 50 non-selected, ultimately healed unilateral cases.

The following measurements were taken:

The proximal articular aspect of the femur — the distal articular aspect of the tibia = total length

The proximal articular aspect of the femur — the distal articular aspect of the femur = length of the femur

The distal articular aspect of the femur — the distal articular aspect of the tibia = length of the tibia

It appeared that a reduction in total length as compared with the unaffected side was present in all cases. The maximum reduction was 35 mm, and the mean reduction was 15 mm.

The length of the femur was reduced in all cases. The reduction varied from 2—35 mm, the mean being 16 mm.

In 16 cases the shortening of the extremity was increased by a simultaneous length reduction of the tibia, varying from 2 to 12 mm, the mean being 4 mm.

In 19 cases the shortening of the extremity was reduced by a compensatory length increase of the tibia, which varied between 2 and 7 mm, the mean being 3 mm.

In 5 cases the tibia was equally long on both sides.

Discussion

With regard to the shortening of the extremity in coxa plana, CAAN (1924) reported a maximal reduction of 4 cm. CARPENTER & POWELL (1960) observed

shortening of the limb in all cases showing extensive changes of the femoral head. The maximum reduction was 1.9 cm.

One third of the longitudinal growth of the femur occurs in the subcapital growth zone (BLOUNT 1954). It is obvious that the meta-epiphyseal disturbance of ossification in coxa plana is the main cause of the shortening of the extremity. In some cases, general atrophy, due to the non-weight-bearing treatment, may be a contributory cause, as is suggested by the fact that the tibia, too, may exhibit shortening.

MORGAN & SOMERVILLE (1960) alleged that the relatively slight shortening of the limb in coxa plana is due to the fact that the growth in the epiphyseal plate of the greater trochanter is undisturbed. This view is not corroborated by LAURENT's (1959) observations on some cases of congenital luxation of the hip joint in children, in which the growth plate of the greater trochanter was obliterated in connexion with open reduction of the femur. In these cases, an extreme valgus deformity with a long neck developed, but no appreciable reduction in length of the extremity resulted. These observations suggest that the longitudinal growth of the femur is entirely regulated by the distal and subcapital growth zones, and that the growth plate of the greater trochanter is of no significance for the length of the limb measured from the surface of the hip joint.

XIII. SUMMARY

The study was carried out on a series of patients registered at the Radiological Department of the Orthopaedic Hospital of the Invalid Foundation during the years 1946—1958. The series comprised 276 cases from the whole of Finland. Fifty cases being bilateral, the total number of coxa plana hips was 326. The ratio of males to females was 4:1. A slight preponderance for left-sided lesions was noted. The youngest patient was 2 years and 8 months old, the oldest 14 years, at onset of the disease. The highest frequency was observed in the age group 6—7 years. The mean duration of symptoms at the time of diagnosis was 9.6 months. Of the cases of active coxa plana, 48.3 per cent were diagnosed in the initial stage.

Treatment. The treatment was conservative and mostly consisted of weight relief on the affected side, accomplished with Thomas' splint, and elevation of the opposite shoe. In 27 cases the treatment by Thomas' splint was preceded by some weeks' hospitalization, during which the walking caliper was manufactured and fitted and the patient was instructed in its use. A small number of patients were treated by bed rest or by non-weight-bearing with crutches at home. The mean duration of non-weight-bearing was 2.3 years.

End-results. The end-results were classified into spherical head = good, elliptical head = fair and irregular head = poor result. In a total of 165 treated hips, the result was good in 49.1 per cent, fair in 21.2 per cent and poor in 29.7 per cent. When end-results and onset age were correlated, the results were found to be clearly better in the younger age groups. Furthermore, it was found that the shorter the duration before the institution of treatment and the earlier the stage of the disease when treatment was instituted, the better were the results.

Conditions resembling *osteochondrosis dissecans* were observed in 16 hips, *i.e.* in 5.2 per cent of the hips with coxa plana examined. In 7 of these cases there was a history of trauma, suggestive of a causal relationship.

Aetiological factors. In 3.6 per cent of the present cases the disease was preceded by a single trauma, which may be considered directly related to the

development of coxa plana. In 5 cases the disease was preceded by general or localized infection which may be regarded as a factor of aetiological significance. Radiologically verified coxa plana was encountered in 11 close relatives of those patients, numbering 172, who were particularly questioned on this point. This high frequency (6.4 per cent) seems to indicate that hereditary factors play a part in the development of the disease.

The course of the radiological changes. In the present series the mean duration of the initial stage was 5.6 months. The duration of the initial stage was independent of the onset age.

The mean duration of the fragmentation stage was 10.8 months. A lower onset age did not seem to shorten the duration of the fragmentation stage.

The mean duration of the reparative stage was 32 months. The duration of reconstruction was somewhat shorter in the younger than in the older age groups.

The mean duration of the disease was 4 years and 4 months, counted from the onset of illness until primary healing had taken place.

In mild abortive cases no changes were detectable in the metaphysis. In some cases with slight or moderate signs of fragmentation in the epiphysis, slight, reversible metaphyseal changes were observed. During the initial stage the most frequent metaphyseal change was a band-shaped zone of demineralization across the proximal portion, seen on the frontal view. This zone represents a defect in the ventral margin of the metaphysis, seen on the lateral view (Fig. 13. p. 71). During the fragmentation stage the defect was enlarged and gradually developed into an oblique, or most often a step-shaped defect, owing to the fact that the ossification of the central portion of the metaphysis continued, while growth was arrested in the area of the defect (Fig. 21. p. 86). The metaphyseal surface developed a proximally directed convexity. During the reparative stage the metaphyseal defect was filled out by the epiphysis (Fig. 21). In occasional cases with severe metaphyseal changes, the metaphyseal defect disappeared owing to reformation of bone at its margins. In such cases the metaphyseal surface was levelled and the epiphyseal line became regular (Fig. 16. p. 78—79). The epiphysis showed a tendency to grow out over the metaphysis, with broadening of the epiphysis resulting. Parallel with the ossification of the metaphyseal defect and the epiphysis, an increase in breadth of the metaphysis occurred. Inhibition of the longitudinal growth of the femoral neck was observed.

Various degrees of irregularity of the proximal surface of the metaphysis were observable. Tomograms were very informative in this respect (Fig. 17.

p. 80—81). Towards the end of the reparative stage, the epiphyseal line became thinner. Partial, and gradually total, fusion of the epiphyseal line often occurred earlier on the affected side than on the unaffected side. As a consequence of this, asymmetrical growth with malposition of the head in relation to the neck resulted in some cases (Fig. 20. p. 83).

Severe metaphyseal changes are often associated with poor healing of the femoral head, premature closure of the subcapital epiphyseal line, broadening of the metaphysis and shortening of the femoral neck.

Formation of a step in the metaphysis. Step-shaped metaphyseal defects have previously been described in tibia vara (Fig. 22. p. 88), and after osteomyelitis or tuberculosis (Fig. 24. p. 89) of the long bones. Similar metaphyseal defects have been produced in animal experiments (Fig. 23. p. 89). In juvenile osteochondrosis of the lumbar vertebrae, ossification defects have been observed which resemble the above-mentioned. A feature in common to all these defects appears to be that growth is arrested locally, while it continues in the surroundings. Initially, the metaphyseal defects are filled out by cartilage. As ossification continues, the metaphyseal defects are often filled out by the bony epiphysis. The development of the metaphyseal defects observed in the present series of coxa plana, as studied on the basis of radiographs, showed a striking resemblance to the course described above.

On the basis of the scanty observations previously reported concerning the histological changes of the epiphyseal plate in coxa plana and in necrosis following a fracture of the femoral neck in adolescence, and on the basis of reports on extensive necrosis of the epiphyseal cartilage in connexion with experimental epiphyseal transplantation, it seems obvious that necrosis of the cartilage tissue in the epiphyseal plate is a pathogenetic factor in common to the development of metaphyseal defects of the kind described here.

The pathogenesis of coxa plana. It is a generally accepted view that the coxa plana process is histopathologically an avascular necrosis. The primary cause of the vascular disturbance is still obscure.

On the basis of earlier investigations concerning the circulatory conditions in the proximal part of the femur in the coxa plana age, the bony epiphysis is supplied only by the lateral epiphyseal arteries departing from the medial circumflex artery. The central portion of the metaphysis is supplied by the nutrient artery, the peripheral parts by the superior and inferior metaphyseal arteries, which are branches of the medial circumflex artery. In coxa plana the lateral epiphyseal vessels and, probably, the metaphyseal vessels are obstructed owing to a cause not yet established, the result being

necrosis of the epiphysis and corresponding areas of the epiphyseal cartilage and the metaphysis. Radiologically, this is evidenced by condensation,flattening and fragmentation of the epiphysis and a defect in the periphery of the metaphyseal surface. The central part of the metaphysis, supplied by the nutrient artery, continues to grow. The reossification of the epiphysis starts in the periphery after revascularization via the synovial membrane. The fate of the metaphyseal defect is dependent on the severity of the cartilage lesion in the epiphyseal plate. If the lesion is slight, ossification commences at the margins of the metaphyseal defect. If the lesion is severe, the metaphyseal defect remains and is gradually filled out by the bony epiphysis. The lesion in the epiphyseal plate is often of such a kind that contact is established between the vascular systems in the epiphysis and the metaphysis, with local fusion of the epiphyseal line resulting.

The greater trochanter. Retardation of the longitudinal growth of the femoral neck and premature closure of the subcapital epiphyseal line are of frequent occurrence in coxa plana. As a rule, the growth of the greater trochanter is not affected. It is, thus, obvious that a discrepancy between the femoral head and neck on the one hand and the greater trochanter on the other must result. On the radiograms this is discernible as a reduction of the distance between the tip of the greater trochanter and the proximal pole of the femoral head, in the present paper called the articulo-trochanteric distance, ATD (Fig. 26. p. 95).

In the present study systematic measurements of the ATD were made. It emerged that the reduction of the ATD, which implies elevation of the greater trochanter, occurred late in the reparative stage or after primary healing had taken place in those cases where the subcapital epiphyseal line fused prematurely. In many cases the elevation of the greater trochanter was so marked that the tip of the trochanter reached several millimetres higher cranially than the upper pole of the head of the femur.

The deformity in coxa plana. The deformity caused by coxa plana has been discussed in the previous literature. Some authors believe that the disease leads to a varus deformity, while according to others it tends to cause a valgus deformity. In these estimates the part of the greater trochanter in the modelling of the proximal end of the femur has been disregarded. The greater trochanter is, however, an integrating part of the proximal end of the femur and cannot be ignored on assessing a possible varus or valgus deformity of the latter.

An account is given of previous experimental and clinical observations on growth phenomena in the proximal end of the femur associated with distur-

bances in the subcapital or subtrochanteric growth zones, and a comparison is made with the conditions prevailing in the distal end of the femur. The conclusion is drawn that in those relatively frequent cases of coxa plana in which the subcapital growth line is prematurely closed, and the growth of the greater trochanter continues undisturbed throughout the normal growing period, the ultimate deformity of the proximal end of the femur is a true varus deformity.

Trendelenburg's sign. In the present series a positive Trendelenburg sign was noted in 26 cases of ultimately healed unilateral coxa plana. The ATD was reduced in all but one of these cases. Among the 26 cases there were 9 in which a retrospective examination of the clinical data and the radiographs revealed that Trendelenburg's sign had been negative at a late reparative stage, and that no, or only a slight, reduction of the ATD had been present. The observations in the cases with positive Trendelenburg signs show that abductor insufficiency in coxa plana may develop at a very advanced stage of the disease, as a result of elevation of the greater trochanter.

Prevention of deformity by epiphysiodesis of the greater trochanter. Since 1961, epiphysiodesis of the greater trochanter has been performed at the Orthopaedic Hospital of the Invalid Foundation in 25 cases of coxa plana, in which radiological examinations revealed progressive reduction of the ATD, or premature closure of the subcapital epiphyseal line. In 23 cases which have been followed up, the epiphysiodesis led to partial or complete fusion of the growth line of the trochanter, and the reduction of the ATD was arrested by the operation. These results seem to justify the conclusion that if epiphysiodesis of the greater trochanter is performed at a favourable point of time, it appears to be possible to prevent elevation of the greater trochanter and consequent abductor insufficiency.

XIV. REFERENCES

v. Aberle-Horstenegg, W.: Zur Behandlung der Perthesschen Erkrankung. — Verhandl. d. deutsch. Orthop. Gesellsch. *34:* 228, 1941.

Albright, F.: Case simulating Legg-Perthes disease due to juvenile Myxoedema. — J. Bone & Joint Surg. *20:* 764, 1938.

Amstad, E.: Beitrag zum Schwund des jugentlichen Schenkelkopfes (Osteoarthritis deformans juvenilis). — Beitr. z. klin. Cir. *102:* 652, 1916.

Axhausen, G.: Die Nekrose des proximalen Bruchstücks beim Schenkelhalsbruch und ihre Bedeutung für das Hüftgelenk. — Arch.f.klin.Chir. *120:* 325, 1922.

Axhausen, G.: Ueber Vorkommen und Bedeutung epiphysärer Ernährungsunterbrechungen beim Menschen. — Münch. med. Wchnschr. *69:* 881, 1922.

Axhausen, G.: Der anatomische Krankheitsablauf bei der Koelerschen Krankheit der Metatarsalköpfchen und der Perthesschen Krankheit des Hüftkopfes. — Arch. f. klin. Chir. *124:* 511, 1923.

Axhausen, G. and Bergmann, E.: Die Ernährungsunterbrechungen am Knochen. — In Henke-Lubarsch: Handbuch d. spez. pathol. Anat. u. Histol. IX: 3: 118, Julius Springer, Berlin, 1937.

Bade, P.: Über die Beziehungen der Arthritis deformans juvenilis zum eingerenkten kongenital luxierten Hüftgelenk. — Ztschr. f. orthop. Chir. *33:* 206, 1913.

Beiler, D. D. and Love, W. H.: Thyreoid function in Legg-Perthes disease. — J. Bone & Joint Surg. *38-A:* 1320, 1956.

Bentzon, P. G. K.: Experimental studies on the pathogenesis of coxa plana. — Acta Radiol. *6:* 155: 1926.

Bergmann, E.: Theoretisches, klinisches und experimentelles zur Frage der aseptischen Knochennekrosen. — Deutsche Ztschr. f. Chir. *206:* 12, 1927.

Bergstrand, I. and Norman, O.: Die Krankheiten des Hüftgelenks im Kindesalter. — Der Radiologe *1:* 76, 1961.

Bernbeck, R.: Untersuchungen zur Pathologie und Ätiologie der Perthes'schen Krankheit. — Verhandl. d. deutsch. orthop. Gesellsch. *36:* 241, 1948.

Bernbeck, R.: Zur Pathogenese der jugentlichen Hüftkopfnekrose (Perthes-Legg-Calvé). — Arch. f. Orthop. u. Unfallchir. *44:* 164, 1950.

Bernbeck, R.: Kritisches zum Perthes-Problem der Hüfte. — Arch. f. Orthop. u. Unfallchir. *44:* 445, 1951 a.

Bernbeck, R.: Zur Pathogenese der aseptischen Hüftkopfnekrose nach Frakturen des proximalen Femurendes. — Verhandl. d. deutsch. orthop. Gesellsch. *38:* 49, 1951 b.

Bernbeck, R.: Kinderorthopädie. Georg Thieme, Stuttgart 1954.

Bertrand, P.: Technique de greffe intraépiphysaire dans le traitement de la coxa plana. — Rev. Chir. Orthop. *40:* 116, 1954.

Bette, H.: Beobachtungen und Ergebnisse bei der konservativen und operativen Behandlung des Morbus Perthes. — Ztschr. Orthop. *92:* 74, 1960.

Bibergeil, E.: Gibt es eine Osteoarthritis deformans coxae juvenilis idiopathica? — Ztschr. f. orthop. Chir. *25:* 184, 1910.

Bibergeil, E.: Weitere Mitteilungen über Osteoarthritis deformans coxae juvenilis. — Ztschr. f. orthop. Chir. *30:* 163, 1912.

Bircher, E.: Die Entwicklung und der Bau des Kretinenskelettes im Röntgenogramme. — Lucas Gräfe & Sillem, Hamburg 1909. Ref. Fortschr. Röntgenstr. *14:* 137, 1909—1910.

Blount, W. P.: Fractures in children. — Williams et Wilkins Company, Baltimore 1954.

Blumensaat, C.: Über sekundäre Schenkelkopfnekrosen nach traumatische Hüftgelenksverrenkungen. — Arch. f. klin. Chir. *185:* 720, 1936.

Bornebusch, K.: Die aseptische Caputnekrose nach Schenkelhalsfrakturen bei Jugentlichen und ihre Beziehung zum Perthesschen Krankheitsbild. — Deutsche Ztschr. f. Chir. *253:* 458, 1940.

Bozan, E. J.: A new treatment of intracapsular fractures of the neck of the femur and Calvé-Legg-Perthes disease. — J. Bone & Joint Surg. *14:* 884, 1932.

Brailsford, J. F.: Avascular necrosis of bone. — J. Bone & Joint Surg. *25:* 249, 1943.

Brailsford, J. F.: The Radiology of Bones and Joints, Ed. IV. Churchill, London 1948.

Brandes, M.: Beobachtungen zur Osteochondritis deformans juvenilis. —Deutsche Ztschr. f. Chir. *131:* 232, 1914.

Brandes, M.: Ueber Spätdeformationen bei reponierter kongenitaler Hüftgelenksluxationen und ihr Verhältnis zum Krankheitsbild der Osteochondritis deformans juvenilis. — Ztschr. f. orthop. Chir. *35:* 274, 1916.

Brandes, M.: Über Fälle von einseitiger Luxatio coxae congenita mit Osteochondritis deformans juvenilis des nicht luxierten Hüftgelenks; Zugleich ein Beitrag zur Ätiologie der Osteochondritis deformans juvenilis (Calvé-Perthes). — Arch. f. Orthop. u. Unfallchir. *17:* 527, 1920.

Brandes, M.: Nachuntersuchungen und weitere Beobachtungen zum Krankheitsbild der Osteochondritis deformans juvenilis coxae. — Deutsche Ztschr. f. Chir. *155:* 216, 1920.

Brocher, J. E. W.: Die Wirbelsäulentuberkulose und ihre Differentialdiagnose. — Georg Thieme, Stuttgart 1953.

v. Brunn, M.: Ueber die juvenile Osteoarthritis des Hüftgelenks. — Beitr. z. klin. Chir. *40:* 650, 1903.

Caan, P.: Osteochondritis deformans juvenilis coxae, Coxa plana, Calvé-Legg-Perthes Krankheit. — Ergebn. d. Chir. u. Orthop. *17:* 64, 1924.

Caffey J.: Pediatric X-ray Diagnosis, 4th Ed. — Year Book Medical Publ. Chicago 1961

Calot, F.: La maladie de Perthes (ou de Legg) n'éxiste pas. — Les centaines de cas cités sont autant de subluxations congénitales larvées et méconnues (d'après 150

observations personelles). — J. des practiciens *35:* 51, 1921. Ref. Zentralorg. f. d. ges. Chir. *14:* 327, 1921.

CALVÉ, J.: Sur une form particulière de pseudo-coxalgie greffée sur des deformations caractéristiques de l'extrémité supérieure du fémur. — Rev. de chir., Paris, *42:* 54, 1910.

CALVÉ, J., GALLAND, M. and DE CAGNY, R.: Pathogenesis of the limp due to coxalgia. — J. Bone & Joint Surg. *21:* 12, 1939.

CAMARGO, F. P.: Revascularization of the neck of the femur in Legg-Calvé-Perthes syndrome. — Clin. Orthop. *10:* 79, 1957.

CARPENTER, E. B. and POWELL, D. O.: Osteochondrosis of capital epiphysis of femur (Legg-Calvé-Perthes disease). — J.A.M.A. *172:* 525, 1960.

CATHRO, A. J. M. and KIRKALDY-WILLIS, W. H.: Treatment of Perthes' disease of the hip by cancellous bone grafting. — J. Bone & Joint Surg. *45-B:* 284, 1963.

CAVANAUGH, L. A., SHELTON, E. K. and SUTHERLAND, R.: Metabolic studies in osteochondritis of the capital femoral epiphysis. — J. Bone & Joint Surg. *18:* 959, 1936.

CHAPMAN, E. M.: Thyreoid function in Legg-Perthes disease. — New England J. Med. *255:* 289, 1956.

DANFORTH, M. S.: The treatment of Legg-Calvé-Perthes disease without weight bearing. — J. Bone & Joint Surg. *16:* 516, 1934.

DREHMANN, G.: Osteoarthritis deformans juvenilis (Perthes). — Beitr. z. klin. Chir. *91:* 642, 1914.

DUBOIS, M.: Pathogenetische und therapeutische Probleme der juvenilen epiphysären Nekrose des Hüftgelenkes (Legg-Calvé-Perthes). — Schweiz. med. Wchnschr. *80:* 1251, 1950.

DUNN, A. W.: Coxa plana in monozygotic male twins. — J. Bone & Joint Surg. *42-A:* 178, 1960.

DYES, O.: Hüftkopfnekrosen nach traumatischen Hüftgelenksluxationen. — Arch. f. klin. Chir. *172:* 339, 1933.

EDBERG, E.: Studien über die sogenannten osteochondritis coxae juvenilis. — Nord. med. Ark. *51:* 63, 1918.

EDEN, R.: Über Osteoarthritis deformans coxae juvenilis. — Deutsche Ztschr. f. Chir. *117:* 148, 1912.

EDGREN, W. and VAINIO, S.: Osteochondrosis juvenilis lumbalis. — Acta chir. Scandinav. Suppl. *227:* 1—47, 1957.

ELMSLIE, R. C.: Pseudocoxalgie following traumatic dislocation of the hip. — J. orthop. Surg. *1:* 109, 1919.

EMERICK, R. W., CORRIGAN, K. E., JOISTAD, A. H., JR. and HOLLY, L. E.: Thyroid function in Legg-Calvé-Perthes disease — a new approach to an old problem. — Clin. Orthop. *4:* 160, 1954.

EVANS, D. L.: Legg-Calvé-Perthes disease. A study of late results. — J. Bone & Joint Surg. *40-B:* 168, 1958.

EVANS, D. L. and LLOYD-ROBERTS, G. C.: Treatment in Legg-Calvé-Perthes disease. A comparison of in-patient and out-patient methods. — J. Bone & Joint Surg. *40-B:* 182, 1958.

EYRE-BROOK, A. L.: Osteochondritis deformans coxae juvenilis or Perthes' disease. The results of treatment by traction in recumbency. — Brit. J. Surg. *24:* 166, 1936.

Ferguson, A. B. and Howorth, M. B.: Coxa plana and related conditions at the hip. — J. Bone & Joint Surg. *16:* 781, 1934.

Fèvre, M. and Lagrange, J.: Réflexions sur 40 cas de coxa plana. — Rev. de chir. orthop. *42:* 44, 1956.

Fiorani, G.: Sopra una forma speciale di zoppicamento. — Gazz. d. osp. *2:* 717, 1881. Ref. Zentralbl. f. Chir. *9:* 265, 1882.

Frangenheim, P.: Zur Pathologie der Osteochondritis deformans juvenilis Coxae. — Beitr. z. klin. Chir. *65:* 19, 1909.

Freehafer, A. A.: Osteochondrosis dissecans following Legg-Calvé-Perthes disease. — J. Bone & Joint Surg. *42-A:* 777, 1960.

Freund, E.: Zur Deutung des Röntgenbildes der Perthesschen Krankheit. — Fortschr. Röntgenstr. *42:* 435, 1930.

Freund, E.: Osteochondritis dissecans of the head of the femur. — Arch. Surg. *39:* 323, 1939.

Froelich, P.: Des arthrites chroniques non tuberculeuses de la hanche du jeune âge ou coxites de croissance. — Rev. de chir., Paris, *42:* 473, 1923.

Fromme, A.: Die Ursache der Wachstumsdeformitäten. — Deutsche med. Wchnschr. — *46:* 169, 1920.

Fründ, H.: Die operative Behandlung der Osteochondritis juvenilis. — Zentralbl. f. Chir. — *49:* 896, 1922.

Gage, H. C.: A possible early sign of Perthes' disease. — Brit. J. Radiol. *6:* 295, 1933.

Gardemin, H.: Chronische Osteomyelitis der Hüfte und Perthessche Krankheit. — Münch. med. Wchnschr. *93:* 853, 1951.

Giannestras, N.: Legg-Perthes disease in twins. — J. Bone & Joint Surg. *36-A:* 149, 1954.

Gill, A. B.: Legg-Perthes' disease of the hip: Its early roentgenographic manifestations and its cyclical course. — J. Bone & Joint Surg. *22:* 1013, 1940.

Gill, A. B.: The relationship of Legg-Perthes disease to the function of the thyroid gland. — J. Bone & Joint Surg. *25:* 892, 1943.

Goff, C. W.: Legg-Calvé-Perthes Syndrome and related Osteochondroses of Youth. — Charles C. Thomas Publisher, Springfield, Ill. 1954.

Goff, C. W.: Recumbency versus nonrecumbency treatment of Legg-Perthes disease. — Clin. Orthop. *14:* 50, 1959.

Goff, C. W.: Legg-Calvé-Perthes Syndrome. An up-to-date critical review. — Clin. Orthop. *22:* 93, 1962.

Goldenberg, R. R.: Traumatic dislocation of the hip followed by Perthes' disease. — J. Bone & Joint Surg. *20:* 770, 1938.

Greulich, W. and Pyle, I.: Radiographic Atlas of Skeletal Development of the Hand and Wrist. — Stanford Univ. Press, Stanford, Calif. 1959.

Gripenberg, L. and Wallgren, G. R.: Personal communication, 1963.

Guéritée, N., Ducroquet, J. and Ducroquet, P.: Proposition d'un traitement médical original de l'osteochondrite juvénile de la hanche (ou maladie de Legg-Perthes-Calvé). — Semaine d. hôp. Paris, *35:* 3042, 1959.

Guildal, P.: Fall zur Erläuterung der Frage von Calvé-Perthes — congenitale Subluxation. — Acta orthop. Scandinav. *1:* 50, 1930.

Haas, A.: Umbau von Perthes'scher Krankheit in Osteochondritis dissecans. — Zentralbl. f. Chir. *64:* 2873, 1937.

HACKENBROCH, M.: Zur Behandlung der Wachstumsstörungen des oberen Femurendes. — Verhandl. d. deutsch. Orthop. Gesellsch. *34:* 222, 1941.

HALKIER, E.: The »Tear-shaped Phenomenon» in Calvé-Perthes disease. — Acta orthop. Scandinav. *25:* 287, 1956.

HAUGE, M. F.: The treatment of coxa plana. A follow-up examination. — Acta orthop. Scandinav. *26:* 53, 1957.

HAYTHORN, S. R.: Pathological changes found in material removed at operation in Legg-Calvé-Perthes disease. — J. Bone & Joint Surg. *31-A:* 599, 1949.

HEIKEL, H. V. A.: Experimental epiphyseal transplantation. Part II. Histological observations. — Acta orthop. Scandinav. *30:* 1, 1960.

HELBO, S.: Morbus Calvé-Perthes. — Thesis, Copenhagen, Fyns Tidendes Bogtrykkeri, Odense 1953.

HERMODSSON, I.: Über die Osteochondritis dissecans des Femurkopfes. — Acta Radiol. *25:* 269, 1944.

HERNDON, C. H. and HEYMAN, C. H.: Legg-Perthes disease, an evaluation of treatment by traction and ischial weight-bearing brace. — J. Bone & Joint Surg. *34-A:* 25, 1952.

HERZOG, F. G.: A study of Perthes' disease. — Proc. Roy. Soc. Med. *54:* 1102, 1961.

HEYMAN, C. H. and HERNDON, C. H.: Legg-Perthes disease. A method for the measurement of the roentgenographic result. — J. Bone & Joint Surg. *32-A:* 767, 1950.

HILGENREINER, H.: Beitrag zur Ätiologie der Osteochondritis coxae juvenilis. — Med. Klin. *29:* 494, 1933.

HIPP, E.: Die Gefässe des Hüftkopfes. — Ztschr. f. Orthop. *96:* Suppl. 1962.

HOFFA, A.: Verletzungen und Erkrankungen der Hüfte und des Oberschenkels. In Bergmann, v. Bruns & Mikulicz: Handbuch der praktischen Chirurgie, IV, Ferdinand Enke, Stuttgart 1901.

HOFMEISTER, F.: Coxa vara. Eine typische Form der Schenkelhalsverbiegung. — Beitr. z. klin. Chir. *12:* 245, 1894.

HOLLÄNDER, L.: Beitrag zur ätiologischen Erfassung der Perthesschen Krankheit. — Helvet. paediat. acta, *7:* 573, 1952.

HORWITZ, T.: The deformity in Legg-Calvé-Perthes disease. — Bull. Hosp. Joint Dis. *21:* 181, 1960.

HOWORTH, M. B.: Coxa plana. — J. Bone & Joint Surg. *30-A:* 601, 1948.

HOWORTH, M. B.: Coxa plana. — Arch. Pediat. *76:* 1, 1959.

HOWORTH, M. B. and SMITH, H. W.: Congenital dislocation of the hip treated by open operation. — J. Bone & Joint Surg. *14:* 299, 1932.

INGLIS, A.: Genetic implications in coxa plana. — J. Bone & Joint Surg. *42-A:* 711, 1960.

ISELIN, H.: Ueber den Zusammenhang von jugentlichem Schenkelkopfschwund und ähnlichen Deformationen mit dem Malum senile coxae und Arthritis deformans. — Korresp. — Bl. schweiz. Ärz. *48:* 1016, 1918.

JACOBS, B. W.: Early recognition of osteochondrosis of capital epiphysis of femur. — J.A.M.A. *172:* 527, 1960.

JANSEN, M.: On coxa plana and its causation. — J. Bone & Joint Surg. *5:* 265, 1923.

JENKINS, S. A.: Osteochondritis dissecans of the hip. — J. Bone & Joint Surg. *40-B:* 827, 1958.

JÉQUIER, M. and FREDENHAGEN, H.: L'hérédité de la dystrophie épiphysaire des hanches; ostéochondrite déformante juvénile; maladie de Legg-Calvé-Perthes. — Radiol. clin. (Basel) *17:* 92, 1948.

JOHANSSON, S.: Über Epiphysennekrose bei geheilten Collumfrakturen. — Zentralbl. f. Chir. *35:* 2214, 1927.

JONSÄTER, S.: Coxa plana. — Acta orthop. Scandinav. Suppl. *12:* 1—98, 1953.

KATZ, J. F.: Protein-bound iodine in Legg-Calvé-Perthes disease. — J. Bone & Joint Surg. *37-A:* 842, 1955.

KATZ, J. F.: Legg-Calvé-Perthes disease. Result of treatment. — Clin. Orthop. *10:* 61, 1957.

KIDNER, F. C.: An unusual case of Legg-Perthes disease. J. Bone & Joint Surg. *8:* 565, 1926.

KIENZLE, L.: Die Behandlung der Coxa vara epiphysaria und der Perthesschen Erkrankung durch die Becksche Bohrung. — Ztschr. f. Orthop. *83:* 270, 1953.

KIRSCH, K.: Die juvenile Osteochondrose des Hüftgelenkes. — In Hohmann, Hackenbroch & Lindemann: Handbuch der Orthopädie, IV: I: 365, Georg Thieme, Stuttgart 1961.

KITE, J. H. and FRENCH, G. O.: The early diagnosis of Flat-headed femur. — South. M. J. *45:* 581, 1952.

KNUTSSON, F.: Några smärre tekniska demonstrationer och ord om inställningstekniken för höftleden. — Nord. med. tidskr. *15:* 274, 1938.

KNUTSSON, F.: Observations on the growth of the vertebral body in Scheuermann's disease. — Acta Radiol. *30:* 97, 1948.

KOSKINEN, E. V. S.: The repair of experimental fractures under the action of growth hormone, thyrotropin and cortisone. — Ann. chir. et gynaec. Fenniae, *48:* Suppl. 90, 1959.

KRAFT, R.: Zur traumatischen Grundlage der Osteochondritis coxae juvenilis deformans. — Deutsche Ztschr. f. Chir. *233:* 345, 1931.

KRISTENSEN, H.: Et tilfaelde af Morbus Calvé-Perthes behandlet med anabolisk steroid og aflastning. — Ugesk. f. laeger, *125:* 255, 1963.

LANGE, M.: Die Gefahr der Pseudoarthrosenbildung und Femurkopfnekrose. — Ztschr. f. orthop. Chir. *57:* 531, 1932.

LANGENSKIÖLD, A.: Tibia vara (Osteochondrosis deformans tibiae). — Acta chir. Scandinav. *103:* 1, 1952.

LANGENSKIÖLD, A.: Ivory vertebra in tuberculosis. — Acta Chir. Scandinav. *104:* 373, 1952.

LANGENSKIÖLD, A.: Aspects of the pathology of Tibia vara (Osteochondrosis deformans tibiae). — Ann. chir. et gynaec. Fenniae, *44:* 58, 1955.

LANGENSKIÖLD, A.: The shape of the epiphysis compensating for metaphyseal defects. Change caused by osteomyelitis or tuberculosis. — Ann. chir. et gynaec. Fenniae, *44:* 87, 1955.

LANGENSKIÖLD, A.: Can osteochondritis dissecans arise as a sequel of cartilage fracture in early childhood? — Acta chir. Scandinav. *109:* 206, 1955.

LANGENSKIÖLD, A. and EDGREN, W.: Imitation of chondrodysplasia by localized roentgen-ray injury. — An experimental study of bone growth. — Acta chir. Scandinav. *99:* 353, 1949.

LANGENSKIÖLD, A. and RISKA, E. B.: Tibia vara (Osteochondrosis deformans tibiae). — J. Bone & Joint Surg. *46-A:* 1405, 1964.

LANGENSKIÖLD, A. and SARPIO, O.: Unpublished observation.

LANGENSKIÖLD, A., SARPIO, O. and MICHELSSON, J-E.: Experimental dislocation of the hip in the rabbit. — J. Bone & Joint Surg. *44-B:* 209, 1962.

v. LANZ, T.: Anatomische und Entwicklungsgeschichtliche Problem am Hüftgelenk. — Verhandl. d. deutsch. Orthop. Gesellsch. *37:* 7, 1950.

v. LANZ, T. and WACHSMUTH, W.: Praktische Anatomie. — J. Springer, Berlin 1938.

LAURENT, L-E.: Growth disturbances of the proximal end of the femur in the light of animal experiments. — Acta orthop. Scandinav. *28:* 255, 1959.

LEGER, W.: Die Valgus- und Varusdeformitäten der Hüfte. — In Hohmann, Hackenbroch & Lindemann: Handbuch der Orthopädie IV/1: 403, Georg Thieme, Stuttgart 1961.

LEGG, A.: An obscure affection of the hip joint. — Boston Med. & Surg. J. *162:* 202, 1910.

LEGG, A.: Osteochondral trophopathy of the hip joint. — Surg., Gynec. & Obst. *22:* 307, 1916.

LEHMANN, J. C.: Der Ablauf der Epiphysennekrosen im Röntgenbild. — Deutsche Ztschr. f. Chir. *253:* 132, 1940.

LEMOINE, A.: Vascular changes after interference with the blood flow of the femoral head of the rabbit. — J. Bone & Joint Surg. *39-B:* 763, 1957.

LEVY, R.: Beiträge zur Frage der coxitis, coxa vara und sogenannte osteoarthritis deformans juvenilis (richtiger coxa vara capitalis). — Deutsche Ztschr. f. Chir. *109:* 205, 1911.

LEVY, L. J. and GIRARD, P. M.: Legg-Perthes' disease. A comparative study of various methods of treatment. — J. Bone & Joint Surg. *24:* 663, 1942.

LIMA, C., ESTEVE, R. and TRUETA, J.: Osteochondritis in congenital dislocation of the hip. — Acta orthop. Scandinav. *29:* 218, 1960.

LINDEMANN, K.: Die juvenilen Osteochondrosen. — In Hohmann, Hackenbroch & Lindemann: Handbuch der Orthopädie, I: 169, Georg Thieme, Stuttgart 1957.

LOOSER, E.: Ueber die Ossificationsstörungen bei Kretinismus. — Verhandl. d. deutsch. Pathol. Gesellsch. *24:* 352, 1929.

LÜTKEN, P.: Bone-Bridge formation between the greater trochanter and the femoral head — a normal variation of the pattern of the ossification in the upper end of the femur in adolescence. — Acta orthop. Scandinav. *31:* 209, 1961.

LÄVEN, A.: Zur Kenntnis der Wachstumsstörungen am Kretinenskelett. — Deutsche Ztschr. f. Chir. *101:* 454, 1909.

MAU, H.: Zur Ätiologie und Pathogenese von Verknöcherungsstörungen des Schenkelhalses und-kopfes. — Ztschr. f. Orthop. *96:* 156, 1962.

MAU, H. and SCHMITT, H. W.: Der konstitutionell-dysostotische Perthes und die Skelettreifungshemmung beim eigentlichen Perthes. — Ztschr. f. Orthop. *93:* 515, 1960.

MAYDL, K.: Coxa vara und Arthritis deformans coxae. — Wien. klin. Rundschau, *11:* 153, 171 and 187, 1897.

MCKENDRY, J. B. J., CARRIE, A. V. and FOSTER, A. E.: Bilateral Legg-Calvé-Perthes disease: A new treatment. — Canad. M.A.J. *83:* 635, 1960.

Miltner, L. J. and Hu, C. H.: Osteochondritis of the head of the femur; an experimental study. — Arch. Surg. *27:* 645, 1933.

Mindell, E. R. and Sherman, M. S.: Late results in Legg-Perthes disease. — J. Bone & Joint Surg. *33-A:* 1, 1951.

Monty, C. P.: Familial Perthes' disease resembling multiple epiphysial dysplasia. — J. Bone & Joint Surg. *44-B:* 565, 1962.

Morgan, J. D. and Somerville, E. W.: Normal and abnormal growth of the upper end of the femur. — J. Bone & Joint Surg. *42-B:* 264, 1960.

Morris, M. L. and McGibbon, K. C.: Osteochondritis dissecans following Legg-Calvé-Perthes disease. — J. Bone & Joint Surg. *44-B:* 562, 1962.

Morville, P.: Comments on the X-ray findings in a patient with Calvé-Perthes disease. — Acta orthop. Scandinav. *1:* 325, 1930.

Morville, P.: Om hofteleddets anatomi og pathologi. — Nord. med. tidskr. *10:* 1331 and 1370, 1935.

Mose, K.: Legg-Calvé-Perthes' Disease. Thesis, Copenhagen, Universitetsforlaget, Aarhus 1964.

Møller, P. F.: Malum deformans coxae infantile. — Thesis, København 1924.

Møller, P. F.: The clinical observations after healing of Calvé-Perthes disease. — Acta Radiol. *5:* 1, 1926.

Nagura, S.: Das Wesen und die Entstehung der Osteochondritis dissecans Königs (bzw. der Perthes-, Köhler II — und ähnlichen Krankheiten und Veränderungen an wachsenden Knochenenden). — Zentralbl. f. Chir. *64:* 2049, 1937.

Nagura, S. and Kosuge, S.: Die Pathogenese und das Wesen der Perthesschen Krankheit. — Arch. f. klin. Chir. *191:* 347, 1938.

Negroni, G.: Dell' osteoartrite deformante giovanile dell' anca. — Arch. di orthop. Milano, *22:* 257, 1905. Cited by Bibergeil 1910.

Nicolaysen, K.: Malum coxae Calvé-Perthes-Leggs pathogenese. — Norsk mag. f. laegevidensk. *92:* 985, 1931.

Nieber, O.: Ueber Osteochondritis deformans coxae juvenilis (Perthes) Ztschr. f. orthop. Chir. *35:* 301, 1916.

Nova Monteiro, J. A.: Coxa plana. Diagnóstico e tratamento. — Rev. méd. munic. (Rio de J.). — *20:* 36, 1954.

Nové-Josserand, G.: Thèse de Médecine, Lyon 1894. Cited by Langenskiöld & Edgren.

Nussbaum, A.: Über Osteochondritis coxae juvenilis Calvé-Legg-Perthes. — Deutsche med. Wchnschr. *49:* 849, 1923.

Nussbaum, A.: Die arteriellen Gefässe der Epiphysen des Oberschenkels und ihre Beziehungen zu normalen und pathologischen Vorgängen. — Beitr. z. klin. Chir. *130:* 495, 1924.

Official Statistics of Finland VII. 1950 population census. Helsinki 1957.

O'Garra, J. A.: The radiographic changes in Perthes' disease. — J. Bone & Joint Surg. *41-B:* 465, 1959.

Peić, S.: Beitrag zur Perthesschen Erkrankung. — Ztschr. f. Orthop. *96:* 276, 1962.

Perthes, G.: Über Arthritis deformans juvenilis. — Deutsche Ztschr. f. Chir. *107:* 111, 1910.

Perthes, G.: Über Osteochondritis deformans juvenilis. — Arch. f. klin. Chir. *101,* 779, 1913.

PERTTILÄ, M.: End-results in Legg-Calvé-Perthes disease. — Ann. chir. et gynaec. Fenniae, *43:* Suppl. 5: 290, 1954.

PETER, E.: Erfahrungen mit der operativen Behandlung der Pertheschen Hüfterkrankung nach Pitzen. — Arch. f. orthop. u. Unfall-Chir. *47:* 417, 1955.

PHEMISTER, D. B.: Operation for epiphysitis of the head of the femur (Perthes' disease). Findings and result. — Arch. Surg. *2:* 221, 1921. Ref. Zentralorg. f.d.ges. Chir. *13:* 205, 1921.

PHEMISTER, D. B.: Operative arrestment of longitudinal growth of bones in the treatment of deformities. — J. Bone & Joint Surg. *15:* 1, 1933.

PIKE, M.: Legg-Perthes disease. — J. Bone & Joint Surg. *32-A:* 663, 1950.

PITZEN, P.: Beschleunigung der Heilung von aseptischen Knochennekrosen im koxalen Femurende (also von der sogenannten Coxa vara adolescentium und dem Perthes) durch Nagelung. — Ztschr. f. Ortop. *81:* 7, 1952.

PONSETI, I. V.: Legg-Perthes disease. (Observations on pathological changes in two cases). — J. Bone & Joint Surg. *38-A:* 739, 1956.

PONSETI, I. V. and MCCLINTOCK, R.: The pathology of slipping of the upper femoral epiphysis. — J. Bone & Joint Surg. *38-A:* 71, 1956.

PONSETI, I. V. and COTTON, R. L.: Legg-Calvé-Perthes disease — pathogenesis and evolution. — J. Bone & Joint Surg. *43-A:* 261, 1961.

PREISER, G.: Ein Fall von sogenannter »idiopatischer« juveniler Osteoarthritis deformans coxae (eine kongenitale Dysarthrie?). — Deutsche Ztschr. f. Chir. *98:* 613, 1908.

PYLKKÄNEN, P.: Coxa vara infantum. — Acta orthop. Scandinav. Suppl. *48:* 1—120, 1960.

RALSTON, E. L.: Legg-Calvé-Perthes disease — Factors in healing. — J. Bone & Joint Surg. *43-A:* 249, 1961.

RATLIFF, A. H. C.: Pseudocoxalgia: A study of late results in the adult. — J. Bone & Joint Surg. *38-B:* 492, 1956.

RATLIFF, A. H. C.: Fractures of the neck of the femur in children. — J. Bone & Joint Surg. *44-B:* 528, 1962.

REHBEIN, M.: Zur Ätiologie der Pertheschen Krankheit, zugleich ein Beitrag zur traumatischen Hüftgelenksluxation im Kindesalter. — Deutsche Ztschr. f. Chir. *174:* 416, 1922.

REHBEIN, M.: Beiträge zur Pertheschen Krankheit. — Fortschr. Röntgenstr. *31:* 251, 1923.

RIEDEL, G.: Beitrag zur pathologischen Anatomie der Osteochondritis deformans coxae juvenilis. — Zentralbl. f. Chir. *49:* 1447, 1922.

RIEDEL, G.: Zur pathologischen Anatomie und Ätiologie der Osteochondritis deformans coxae juvenilis. — Virchows Arch.f. path. Anat. *244:* 335, 1923.

ROCKEMER, K.: Zur Histopathogenese der Pertheschen Krankheit. — Frankfurt. Ztschr. f. Pathol. *35:* 1, 1927.

ROKKANEN, P.: Rôle of surgical interventions of the hip joint in the aetiology of aseptic necrosis of the femoral head. — Acta orthop. Scandinav. Suppl. *58:* 1—107, 1962.

RYDER, C. T., LEBOUVIER, J. D. and KANE, R.: Coxa plana. — Pediatrics, *19:* 972, 1957.

SALVO LECARRE, J. C.: Confrontación de los resultados operatorios y conservadores en la enfermedad de Legg-Calvé-Perthes. — Med. clín., Barcelona, *28:* 380, 1957.

Schmid, F. and Halden, L.: Die postfetale Differentierung und Grössenentwicklung der Extremitätenknochenkerne. — Fortschr. Röntgenstr. *71:* 975, 1949.

Schneider, E.: Zur Pathogenese der regulatorischen Wachstumsmalacien. — Arch. f. klin. Chir. *188:* 91, 1937.

Schwarz, E.: Eine typische Erkrankung der oberen Femurepiphyse. — Beitr. z. klin. Chir. *93:* 1, 1914.

Severin, E.: Über die Entwicklung von Coxa plana. — Acta chir. Scandinav. *87:* 317, 1942.

Sinding-Larsen, M.: Malum deformans coxae infantile (Calvé-Perthes sygdom). — Norsk mag. f. laegevidensk. *13:* 475, 1915.

Sjövall, H.: Zur Frage der Behandlung der Coxa plana. — Acta orthop. Scandinav. *13:* 324, 1942.

Sjövall, H.: Om Perthes sjukdom, dess diagnos och behandling. — Svenska läkartidn. *40:* 214, 1943.

Slavík, J.: Coxa plana — Morbus Maydl-Calvé-Legg-Perthes. — Albertova Sbirka, Praha 1956.

Soeur, R. and de Racker, Ch.: L'aspect anatomopathologique de l'ostéochondrite et les théories pathogéniques qui s'y rapportent. — Acta orthop. Belg. *18:* 57, 1952.

Sourdat, P.: Étude radiographique de la hanche coxalgique. — Thesis, Paris 1909.

Statistical yearbook of Finland, New series — 49th — year 1953. Helsinki 1954.

Stephens, F. E. and Kerby, J. P.: Hereditary Legg-Calvé-Perthes' disease. — J. Hered. *37:* 153, 1946.

Ståhle, L.: Malum coxae Calvé-Legg-Perthes. — Finska läk.-sällsk. handl. *64:* 244, 1922.

Stupnicki, A.: Zur operativen Behandlung der Perthesschen Krankheit mit Bohrung und Bolzung. — Ztschr. f. Orthop. *81:* 272, 1952.

Ståhl, F.: Early Coxa plana. Age studies. — Acta orthop. Scandinav. *17:* 180, 1948.

Sundt, H.: Undersøkelser over Malum coxae Calvé-Legg-Perthes. — Thesis, Kristiania 1920.

Sundt, H.: Further investigations respecting Malum coxae Calvé-Legg-Perthes with special regard to the prognosis and treatment. — Acta chir. Scandinav. Suppl. *148:* 1—101, 1949.

Söderberg, L.: Simultaneous occurrence of Coxa plana in enzygotic twins. — Acta orthop. Scandinav. *27:* 135, 1957.

Tallqvist, G.: The reaction to mechanical trauma in growing articular cartilage. — Acta orthop. Scandinav. Suppl. *53:* 1—112, 1962.

Taylor, H. L. and Frieder, W.: Quiet hip disease. — Surg., Gynec. & Obst. *22:* 158, 1916.

Trueta, J.: La etiologia de la enfermedad de Perthes. — Rev. Fac. de med., Bogotá, *24:* 743, 1956.

Trueta, J.: The normal vascular anatomy of the human femoral head during growth. — J. Bone & Joint Surg. *39-B:* 358, 1957.

Trueta, J. and Harrison, M. H. M.: The normal vascular anatomy of the femoral head in adult man. — J. Bone & Joint Surg. *35-B:* 442, 1953.

Trueta, J. and Pinto de Lima, C. S.: Estudios sobre la osteochondritis de la cabeza femoral o enfermedad de Legg-Calvé-Perthes. — Rev. Ortop. y Traum. Lat. Amer. *4:* 115, 1959.

TRUETA, J. and MORGAN, J. D.: The vascular contribution to osteogenesis. I Studies by the injection method. — J. Bone & Joint Surg. *42-B:* 97, 1960.

TRUETA, J. and AMATO, V. P.: The vascular contribution to osteogenesis. III Changes in the growth cartilage caused by experimentally induced ischaemia. — J. Bone & Joint Surg. *42-B:* 571, 1960.

TRUETA, J. and TRIAS, A.: The vascular contribution to osteogenesis. IV The effect of pressure upon the epiphyseal cartilage of the rabbit. — J. Bone & Joint Surg. *43-B:* 800, 1961.

TUCKER, F. R.: Arterial supply to the femoral head and its clinical importance. — J. Bone & Joint Surg. *31-B:* 82, 1949.

ULLOA, I.: Über die embryonale und fötale Entwicklung des Gefässystems des proximalen Femurendes und Acetabulum des Menschen. — Ztschr. f. Orthop. *96:* 306, 1962.

ULLOA, I.: Traumatische Hüftgelenkverrenkung bei Kinder. — Verhandl. d. deutsch. orthop. Gesellsch. *50:* 294, 1963.

WALDENSTRÖM, H.: Der obere tuberculöse Collumherd. — Ztschr. f. orthg. Chir. *24:* 487, 1909.

WALDENSTRÖM, H.: Die Tuberculose des Collum Femoris im Kindesalter und ihre Beziehungen zur Hüftgelenkenzündung. — Stockholm 1910.

WALDENSTRÖM, H.: Coxa plana, Osteochondritis deformans coxae, Calvé-Perthessche Krankheit, Legg's disease. — Zentralbl. f. Chir. *22:* 539, 1920.

WALDENSTRÖM, H.: The definite form of the Coxa plana. — Acta Radiol. *1:* 384, 1922.

WALDENSTRÖM, H.: On Coxa plana. — Acta chir. Scandinav. *55:* 577, 1923.

WALDENSTRÖM, H.: The first stages of Coxa plana. — Acta orthop. Scandinav. *5:* 1, 1934.

WALDENSTRÖM, H.: The first stages of Coxa plana. — J. Bone & Joint Surg. *20:* 559, 1938.

WANSBROUGH, R. M., CARRIE, A. W., WALKER, N. F. and RUCKERBAUER, G.: Coxa plana. Its genetic aspects and results of treatment with the long Taylor walking caliper. — J. Bone & Joint Surg. *41-A:* 135, 1959.

WIDERÖE, S.: Zur Ätiologie und Pathogenese des Malum coxae Calvé-Perthes. — Zentralbl. f. Chir. *48:* 158, 1921.

WIRZ, F.: Die Osteoporose als Frühsymptom der Osteochondrosis deformans coxae juvenilis Perthes. — Schweiz. med. Wchnschr. *83:* 384, 1953.

WOLCOTT, W. E.: The evolution of the circulation in the developing femoral head and neck. — Surg., Gynec. & Obst. *77:* 61, 1943.

WOROBEC, R. and NORWOOD, G.: Legg-Perthes disease. — Guthrie Clin. Bull. *26:* 40, 1956.

YAMAGUCHI, M.: A histological study of the neck of the femur in Perthes' disease (Jap.text). — Igaku Kenkyu, *29:* 317, 1959. Ref.: Exc. Med. IX, Surg. *14:II:* p. 1524, 1960.