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ON OSTEOCHONDROSIS DEFORMANS JUVENILIS CAPITULI HUMERI INCLUDING INVESTIGATION OF INTRA-OSSEOUS VASCULATURE IN DISTAL HUMERUS

BY

STEFÁN HARALDSSON

EJNAR MUNKSGAARD
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PREFACE

Osteochondrosis deformans juvenilis (O.d.j.) located in the ossifying capitulum humeri is a well defined clinical and roentgenologic entity.

Nevertheless, the disease is not widely known, and but few cases have been published.

Perusal of the literature of the last 54 years revealed only 30 verified cases. These had been described by a total of 19 authors.

No single author has reported more than 4 cases, most authors having contributed with only 1 case.

Literature studies revealed that further investigation of various problems related to this disease was desirable.

The present investigation is based on a series of 27 cases, which, though not many, is by far the largest material hitherto published.

The purpose of this investigation was to elucidate the clinical and roentgenologic features of Osteochondrosis deformans juvenilis of the capitulum humeri against the background of literature studies.

The investigation also includes an experimental study of the intra- and extra-osseous vasculature of the distal end of the humerus with special reference to the capitulum, and is based on injection experiments on cadavers of children and adults.

The aim of the experimental investigation was to shed light on some problems bearing on the nutrition of the growing chondroepiphysis and the vasculature in growing and full-grown bones, as well as on some details of etiologic interest in connection with O.d.j. capituli humeri.

All authors discussing this disease agree that it belongs to a group of affections of the growing skeleton, which in this publication will be referred to as Osteochondrosis deformans juvenilis (O.d.j.). Any conclusions, which the findings made in this investigation might justify, may therefore also hold for O.d.j. in other locations.

The literature contains descriptions of changes in 42 different endo-

chondral ossification centres in the growing skeleton which the authors have regarded as belonging to the above mentioned group of affections. Many of these publications have been criticised and will be criticised further in this paper.

In view of the observations set forth above, it was considered of interest first to give some general considerations on this group of diseases, in order *inter alia* to underline the present predominant opinion that the lesions, at least in some of these locations, are simply different local manifestations of one and the same pathologic process and not different diseases.

PART I

Chapter I

OSTEOCHONDROSIS DEFORMANS JUVENILIS

General considerations

Until the turn of the century it was widely believed that most bone lesions causing local rarefaction were of tuberculous origin. The advent of diagnostic radiology implied the availability of a method for following the course of these processes.

Roentgenologic, clinical and histologic studies gradually revealed that a number of non-tuberculous pathologic processes with many features in common were capable of occurring in ossification centres.

These lesions proved to be manifestations of a disturbance of the normal ossification process and have since been described in different endochondral ossification centres in the growing human skeleton.

Despite difference in location of these processes, many cases resemble one another in respect to their history, clinical course and roentgenographic appearance. Many authors therefore agree that at least some of these lesions independently of their location in the skeleton are local manifestations of one and the same fundamental pathologic process (ALLISON & MOODY 1915, ERLACHER 1922, LIEK 1922, FRIEDRICH 1924, CAAN 1924, NUSSBAUM 1924, WOLLENBERG 1928, PANNER 1929, BUCHMAN 1929, SMITH 1929, BELMONTE 1929, HARBIN & ZOLLINGER 1930, SAÇERDOTE 1931, WINTERSTEIN 1933, HAGEN & BISSCHOP 1933, UHRMACHER 1933, BRAILSFORD 1935, OVERTON 1935, HAAS 1937, NAGURA 1937, 1938, 1939, 1940, 1941, SCHAEFER, STRICKROOT & PURCELL 1939, ELWARD 1939, BOZSAN 1941, LANG 1941, MÜLLER 1945, DOUB 1945, COLE 1945, COLLINS 1949, PEASE 1950, SCHINZ, BAENSCH, FRIEDL & UEHLINGER 1950, CAMERA 1951, HEGEMANN 1951, HOLLÄNDER 1952, HOWORTH 1952, BENNET 1952, MASON 1953, JUD 1953, HELBO 1953, GOFF 1954, ADAMS 1956, MAU 1958 and others).

After the pioneer work of WALDENSTRÖM (1909), LEGG (1909), CALVÉ (1910) and PERTHES (1910) on the lesions of the ossification centre of the femoral head, which resulted in classification of the changes as a separate clinical entity, various names for the entire group of the affec-

tions have been discussed. As a rule, the disease in a given location has been called after the author who first described the lesion there. This terminology has contributed to the tendency to conceive each location of the disease as an independent clinical entity. As mentioned, however, it was gradually realised that these pathologic processes, at least those in some of the locations, were local manifestations of one and the same fundamental pathologic process with the result that it was considered unsuitable to use such eponyms.

Various common names have been suggested. PERTHES (1913) suggested "Osteochondritis deformans juvenilis" for the lesions of the ossification centre of the femoral head. He defended the suffix *itis* not on the grounds that he considered the lesion to be of inflammatory nature but to underline its similarity with the term osteochondritis dissecans, suggested by KÖNIG in 1888.

Since then a variety of terms have been suggested as a common name for these lesions: Osseous aseptic necrosis (AXHAUSEN 1911), osteochondropathia juvenilis parosteogenetica (ZAAIJER 1921), local malacias (MÜLLER 1925), local dyschondroplasia (BENTZON 1926), osteopathia juvenilis necroticans (HOLST & CHANDRIKOFF 1927), malakopathie (WAGNER 1928), osteochondrolysis (CORDES 1930), epiphysitis, apophysitis (ELWARD 1939), juvenile chondroepiphysitis (SCHAEFER et al. 1939), osteosis (WEINMANN & SICHER 1947), epiphysos, apophysos (FANCONI 1952), osteochondroses of youth (GOFF 1954).

The term osteochondrosis is used in *Manual of the International Statistical Classification of Diseases* etc. (1948, 1949) as a name for most of the pathologic conditions of unknown etiology in the growth centres of the skeleton.

Judging from the literature of recent years, osteochondrosis is being more and more widely accepted as a common name for the group of diseases under discussion. Thus, HOWORTH (1952) uses the term in his *Textbook of Orthopedics* and GOFF (1954) speaks of "Osteochondroses of Youth" in his comprehensive work on this subject. LINDEMANN (1957) prefers the term "juvenilen Osteochondrosen" in *Handbuch der Orthopädie*. In a large publication in 1958 MAU stated that "gegenüber den früher gebräuchlichen Ausdrücken ——— beginnt sich der Begriff der juvenilen Osteochondrosen immer mehr durchzusetzen".

The lesions appear in endochondral ossific nuclei and, according to most authors, are of non-inflammatory nature. These features are covered by the term osteochondrosis. In addition, the disease occurs only

in the growing skeleton and sometimes results in malformation. In view of the observations set forth above, in the present investigation it was decided to give preference to the term osteochondrosis deformans juvenilis (O.d.j.) as a common name for these diseases. This term distinguishes the group from other aseptic circumscript osseous lesions, *e.g.* from aseptic bone necrosis in the adult skeleton, which differ in some respects from osteochondrosis deformans juvenilis.

The essential features of the disease are, as mentioned, essentially the same independently of the location of the changes, and according to many authors the disease appears during the period of the most rapid growth of the ossification centre affected (LIEK 1922, HARTLEY 1923, BUCHMAN 1929, HIRSCH 1933, UHRMACHER 1933, OVERTON 1935, ELWARD 1939, SCHINZ et al. 1952, HOLLÄNDER 1952, BENNET 1952, LANGE 1954, ADAMS 1956, LAURENT & LINDSTRÖM 1956 and others).

According to some authors, this pronounced tendency of the disease to appear at the time of most rapid growth of the ossification centre may be ascribed to the vulnerability and decreased resistance of rapidly growing cells. This theory has been defended in particular by MURK-JANSEN (1929) who says that "der Grad der Wachstumsschwäche ist proportional der Schnelligkeit des Wachstums". Other authors have also expressed the view that the power of resistance of rapidly growing ossification centres to direct or indirect exogenic insults is decreased and that this favours the development of O.d.j. (ZAAIJER 1921, LIEK 1922, HASS 1931, UHRMACHER 1933, MÜLLER 1945, LUCK 1950, JUD 1953, LANGE 1954).

This view is assailed by GOFF (1954), who believes that those types of O.d.j. he calls "true osteochondroses" and to which he assigns *inter alia* O.d.j. capituli humeri, arise in an ossification centre that has slowed down in growth.

As mentioned, changes ascribed to this group of diseases have been described in different endochondral ossification centres, and new locations of the lesions are still being described.

It has therefore been suggested that, theoretically, the disease might appear in any endochondral ossification centre (CAAN, 1924, CHRISTIE 1926, SMITH 1929, HARBIN & ZOLLINGER 1930, HIRSCH 1933, KING 1935, BENNET 1952, MASON 1953, GOFF 1954, EFSKIND 1955, ADAMS 1956 and others). Other authors, however, have stressed that the disease seldom occurs in parts of the skeleton not subject to direct or indirect trauma, muscular strain or static stress (SMITH 1929, HARBIN & ZOLLINGER

1930, SCHINZ et al. 1950, BENNET 1952, JUD 1953, GOFF 1954 and others).

As to the fundamental pathologic changes in O.d.j., AXHAUSEN (1923) is regarded as the first to have elucidated the pathology of the disease by his histologic examination of lesions in 2 different locations. In his cases the disease was in such an early stage that he was able to study the primary pathologic changes before the appearance of secondary reactive alterations. He arrived at the conclusion that the primary pathologic process in O.d.j. consists of aseptic necrosis of the young growing, spongy tissue in the ossification centre, and that all other changes are secondary to this bone necrosis.

Many other histologic investigations have contributed to our knowledge of the pathology of the lesions in the femoral head (PERTHES 1913, PHEMISTER 1921, HEITZMANN & ENGEL 1923, RIEDEL 1923, ROCKEMER 1927, ZEMANSKY 1928, LIPPMANN 1929, KONJETZNY 1934, HAYTHORN 1948, 1949, JONSÄTER 1953) and in other ossification centres (KIDNER & MURO 1924, HOLST & CHANDRIKOFF 1927, DESSECKER 1930, HASS 1931, KONJETZNY 1934, NAGURA 1940, LUTTEROTTI 1948, PEREGALLI 1954).

It would appear, however, that no histologic investigations have been made of O.d.j. lesions located in the ossification centre of the capitulum humeri.

Perusal of the literature revealed descriptions of osseous changes ascribed on more or less well-founded grounds to this group of bone diseases. In other words, the range of bones in which these lesions have been described in the literature is wider than that generally supposed. It was therefore considered of general interest to give a survey of the locations in which changes believed by the authors to belong to the above mentioned group have been described in the literature (Table 1 and Fig. 1).

In the meantime it has, however, been discovered that in many centres endochondral ossification is normally uneven (FORSELL 1912, HELLMER 1935, KING 1935, FRANCIS & WERLE 1939, SONTAG & PYLE 1941, SCHINZ et al. 1952, GOFF 1954, CAFFEY 1956, SWOBODA 1956 and others). Some ossification centres such as in the capitulum humeri and femoral capital epiphysis are normally ossified from a single site in the chondroepiphysis (unicentric ossification). This is seen in the roentgenogram as a growing single ossific nucleus of homogeneous spongy structure and even outline. In other ossification centres, on the other

hand, uneven mineralisation is the rule during the period of growth. Here mineralisation sometimes occurs in different sites simultaneously and is then referred to as multicentric ossification. The roentgenographic appearance of such an ossification centre may resemble that of O.d.j., with which it has also often been confused. In the investigation of an ossification centre in the humeral trochlea, for example, or in the olecranon, patella, apophysis of the calcaneus and others, where normal mineralisation is often uneven, it is therefore sometimes difficult to decide on the basis of a single roentgen examination, whether the irregular appearance of the ossification centre should be regarded as normal or not. Roentgenographic follow-up will, however, often decide whether such unevenness is due to a destructive or constructive process in the ossification centre. But even then the diagnosis may offer difficulties in cases of O.d.j. in which the disease was in the healing stage already at the time of the first roentgen examination.

Broadly speaking, caution must be exercised in the establishment of the diagnosis of O.d.j. on the basis of a single roentgen examination of children with no other symptoms or signs of active disease. This holds in particular for ossification centres in which mineralisation is known to be normally uneven. Ossification centres, in which mineralisation is apt to be uneven (according to CAFFEY 1956) are given in Table 1. The ossification centre of the capitulum humeri does not belong to this category.

In the review of the locations in which changes diagnosed as O.d.j. have been described (Table 1, Fig. 1), the changes in the os lunatum, "malacia ossis lunati" described by KIENBÖCK (1910) were not included. The reason for this exclusion was that they do not fall under the heading of O.d.j., since they do not occur in a growing ossification centre but in a fully developed bone. This opinion is shared by SONNTAG (1923), KING (1935), STÅHL (1947), MERCER (1950), GOFF (1954) and ADAMS (1956). This also holds for the changes in the carpal scaphoid bone described by PREISER (1911).

It is clear from Table 1 and Fig. 1 that the disease has been claimed in various parts of the growing skeleton.

Most authors agree that in some of the locations included in Table 1 particularly the femoral head, the os naviculare pedis, the head of metatarsale II and the capitulum humeri have really been the sites of O.d.j. on the basis of necrosis of the ossification centre in question.

As to many of the other locations given in Table 1, several authors

TABLE 1

Osteochondrosis def. juvenilis and similar changes described in literature

No. in Fig. 1	Centre of ossification affected	First described by	Year of first publicat.	Normal mineralisation sometimes irregular
1	Tuberositas tibiae	OSGOOD, R. B., SCHLATTER, C.	1903	+
2	Apophysis calcanei	HAGLUND, P.	1907	+
3	Patella (main centre)	KÖHLER, A.	1908	+
4	Os naviculare tarsi	KÖHLER, A.	1908	+
5	Os tibiale externum	HAGLUND, P.	1908	
6	Epiphyses of phalanges	THIEMANN, H.	1909	+
7	Caput femoris	WALDENSTRÖM, H., LEGG, A.T.	1909	
		CALVÉ, J., PERTHES, G.	1910	
8	Tuberositas os. metatars 5.	ISELIN, H.	1912	
9	Epiphysis os. metatars. 2	FREIBERG, A. H.	1914	+
10	Epiphysis prox. humeri	ALLISON, N. and MOODY, E. F.	1915	
11	Epiphysis dist. radii	ALLISON, N. and MOODY, E. F.	1915	
12	Epiphysis dist. tibiae	ALLISON, N. and MOODY, E. F.	1915	
13	Epiphysis corp. vertebral.	SCHEUERMANN, H.	1920	+
14	Patella (accessory centre)	JOHANSSON, S., SINDING-LARSEN, M.	1921	+
15	Olecranon	ZAAIJER, J. H.	1921	+
16	Epiphysis os. metatars. 3	PANNER, H. J.	1921	+
17	Proximal tibia	ERLACHER, P.	1922	+
18	Epiphysis os. metacarp. 4—5	ERLACHER, P.	1922	+
19	Synchondrosis ischio-pub.	VAN NECK, M.	1924	+
20	Epiphysis claviculae	FRIEDRICH, H.	1924	
21	Os sesamoid. metatars. I	RENANDER, A.	1925	
22	Corpus vertebralis	CALVÉ, J.	1925	
23	Crista iliaca	BUCHMAN, J.	1925	+
24	Os cuboidale	SILFVERSKIÖLD, N.	1926	+
25	Capitulum humeri	PANNER, H. J.	1927	
26	Astragalus	DIAZ, F. G.	1928	
27	Symphysis pubis	PEIRSON, E. L.	1929	+
28	Acetabulum	FRÖLICH	1929	+
29	Epiphysis os. metatars. I	WAGNER, A.	1930	+
30	Epicondyl. med. humeri	LEGG, A. T.	1930	+
31	Epiphysis dist. ulnae	BURNS, B. H.	1931	+
32	Epiphysis os. metacarp. 3	DIETRICH, H.	1932	+
33	Metatarsal pseudo-epiphys.	BURMAN, M. S.	1933	
34	Trochlea humeri	UHRMACHER, F.	1933	+
35	Os cuneiforme mediale	BUCHMAN, J.	1933	+
36	Os pisiforme	SCHMIER, A. A., MEYERS, M. P.	1939	+
37	Acromion	CLEAVES, E. N.	1940	+
38	Tuber ischiadicum	MCMASTER, P.	1945	+
39	Capitulum radii	HERMODSSON, I.	1947	+
40	Spina iliaca ant. inf.	DE CUVELAND, E., HEUCK, F.	1951	+
41	Os cuneiforme secundum	GLANVILLE HICKS, B. T.	1953	+
42	Eminentia intercond. tibiae	CAFFEY, J.	1956	

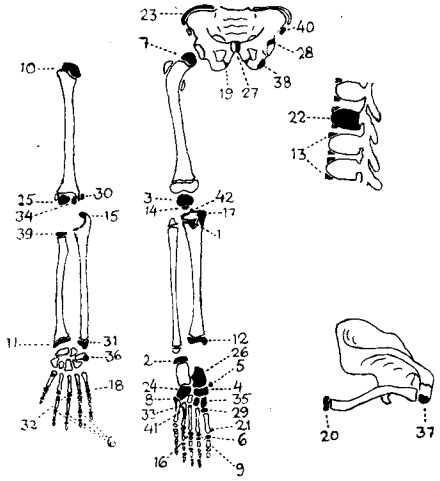


Fig. 1. Anatomical distribution of changes described by authors in Table 1.

are inclined to doubt whether the changes observed really were due to a pathologic process in the ossific nucleus.

As early as 1912, FORSELL described the normal, uneven ossification in the growing os naviculare pedis.

BERGMANN (1926) made a histologic study of the changes described by HAGLUND (1907) in the apophysis of the calcaneus. BERGMANN concluded that these changes should not be regarded as O.d.j., but as a physiologic ossification variant. This view was afterwards shared by MERLINI (1928), LANG (1941), BRAILSFORD (1953) and FERGUSON & GINGRICH (1957).

According to MÜLLER (1927), LANGE (1928), and KIMMENSTIEL, KREMSER & RICHTER (1933), the "osteochondropathy" of the medial sesamoid bone of the first metatarsal described by RENANDER (1925) should not be regarded as O.d.j. They based their view *inter alia* on histologic examinations.

SCHMORL (1930) and COLLINS (1949) do not believe that the changes in the marginal ring of the vertebral bodies described by SCHEUERMANN (1920) should be classified under the heading of O.d.j., but that the changes were secondary to intravertebral disc prolapses.

HELLMER (1935) showed that the roentgenogram described by many authors as O.d.j. in the main centre of ossification of the patella (KÖHLER 1908) was in reality a picture of a normal stage in the ossification of the patella. He expressed a similar view of the roentgenologic appear-

ance of an accessory centre of ossification of the patella described by JOHANSSON (1921) and SINDING-LARSEN (1921).

LANG (1941), CAFFEY & ROSS (1956) and others have stressed that swelling and uneven mineralisation of the ischiopubic synchondrosis (VAN NECK 1924) normally precedes the fusion in many children. They claim that supposed cases of O.d.j. in this location are often only a normal ossification variant.

RAVELLI (1955) wrote that O.d.j. of the ossification centre in the epiphysis of the clavicle, first described by FRIEDRICH (1924) and later by others, may very well occur. He stressed, however, that in the cases published the evidence available was not sufficient to establish such a diagnosis.

Changes in the ossifying tuberosity of the tibia, first described by OSGOOD (1903) and SCHLATTER (1903), is regarded by many, including LANG (1941), UHRY (1944) and WATSON-JONES (1955) as a sequela after mechanical trauma, traumatic separation of the ossification centre. Other authors such as LUTTEROTTI (1948), and SCHINZ et al. (1950) distinguish between O.d.j. in this ossification centre with fragmentation in the ossific nucleus and osteoporosis in the adjacent metaphysis on one hand and avulsion fracture on the other, in which no changes occur in the bone structure.

MÜLLER (1939), LANG (1941) and SCHINZ et al. (1950) claim that the multiple hereditary epiphyseal disorders described by RIBBING (1937) are neither identical nor related to O.d.j.

SCHINZ et al. (1950) make a clear distinction between the roentgenologic changes in the epiphyses of the toes and finger phalanges described by THIEMANN (1909) and O.d.j.

GOFF (1954) differentiates between "true osteochondroses" which occur in joint forming "pressure epiphyses" and "false osteochondroses", seen in "traction epiphyses and atavistic epiphyses".

Finally, it might be mentioned that osteochondritis dissecans (KÖNIG 1888) is regarded as belonging to the O.d.j.-group of diseases by some authors such as HAAS (1937), LANG (1941), BENNET (1952), HOWORTH (1952), GOFF (1954), HÄUPTLI (1954), TOBIN (1957) and others. Other authors, however, claim that a clear distinction should be made between this disease and O.d.j. (BERGMANN 1929, LANGE 1929, FRANCILLON 1932, KING 1935 and ADAMS 1956).

Perusal of the literature showed that these two diseases differ from one another in several respects. First there is a difference in age incid-

ence. Secondly, in course and end result they differ from one another. Thus, while O.d.j. ends in organisation of the total necrotic ossification centre with normalisation of osseous structure, in osteochondritis dissecans there is usually separation from the bone, of the circumscript, subchondral necrotic focus together with overlying joint cartilage (NIELSEN 1933).

Though the two diseases thus seem to differ from one another, KING (1935) claims that occasionally cases are seen in which the two conditions appear to merge or at least occur coincidentally.

Similarity between O.d.j. of the femoral head and O.d.j. of the capitulum humeri

It is clear from the preceding sections that opinions differ on the true nature of many of the osseous changes described.

General agreement has, however, been achieved that the lesions in the epiphysis of the femoral head represent the classical form of osteochondrosis deformans juvenilis.

Of the processes described in other ossification centres those located in the capitulum humeri are beyond doubt those which most closely resemble the classical O.d.j. of the femoral head (see Chapters III and V). The similarity between these 2 locations of the process is thus striking both with regard to the roentgenologic appearance, the clinical picture, age at onset, sex incidence, course and the obscure etiology. This similarity has been pointed out by several authors (AMSTAD 1916, PANNER 1927, KREBS 1927, HAGEN & BISSCHOP 1933, UHRMACHER 1933, WINTERSTEIN 1933, BURCKHARDT 1936, NIJST 1937, RIVAROLA 1939, MOL 1939, KOHLBACH 1940, BURROWS 1941, BOZSAN 1941, LANGE 1954 and LAURENT & LINDSTRÖM 1956).

The roentgenologic similarity between these two diseases is obvious on comparison of the two series of roentgenograms given in Fig. 2. One of the series shows various stages of the well-known roentgenographic appearance of O.d.j. of the femoral head, the other similar stages in the capitulum humeri. These two series represent some of the stages given by WALDENSTRÖM (1922) in his classification of the development of coxa plana, namely:

I. Evolutionary period
   1. Initial stage
   2. Fragmentation stage

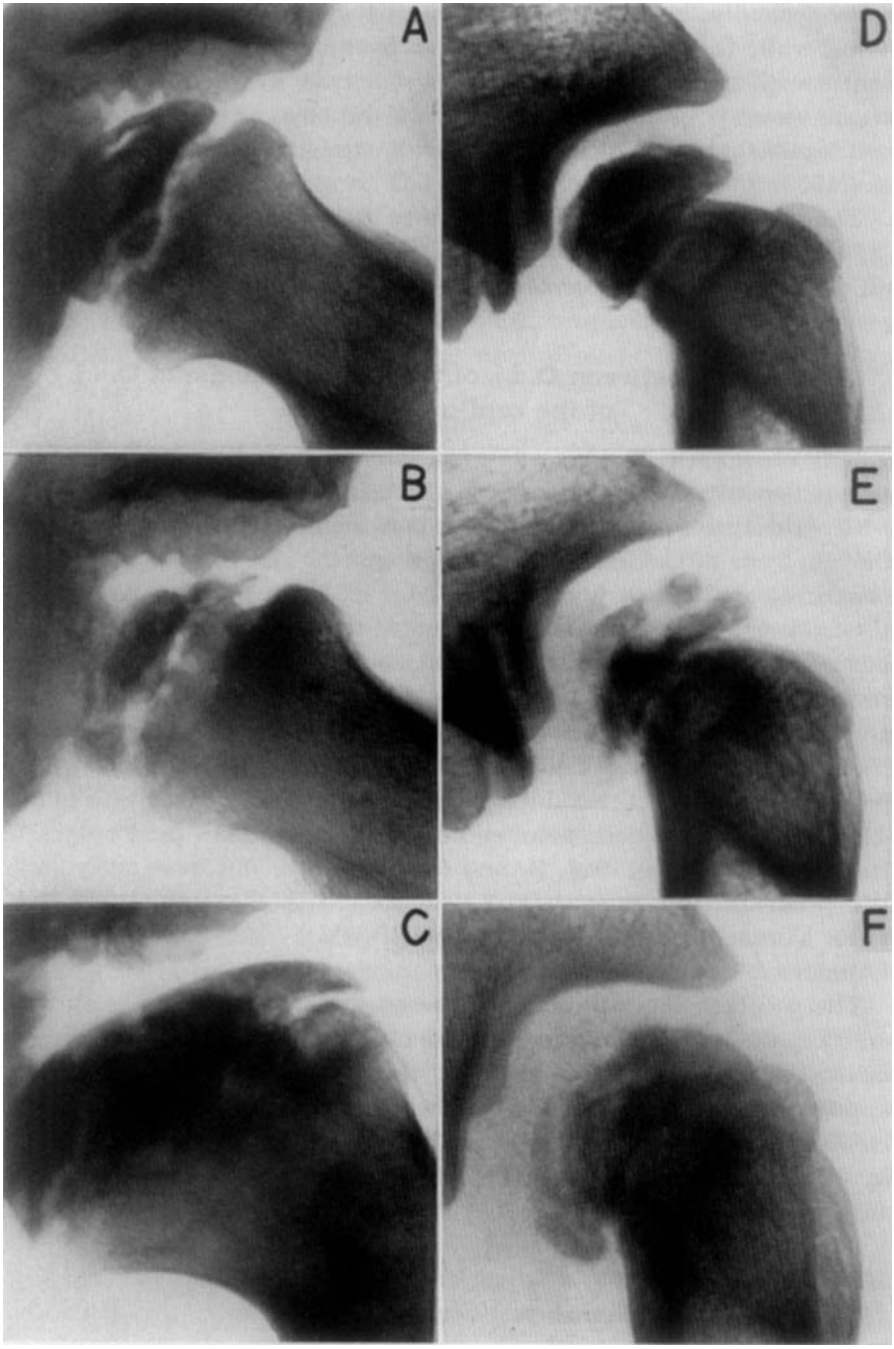


Fig. 2. The roentgenologic similarity between O.d.j. capitis femoris (Legg-Calvé-Perthes disease), Figs. A, B, C and the process in capitulum humeri, Figs. D, E and F.

II. Healing period
III. Growing period
IV. Definite stage

Hereinafter this classification will be adopted to facilitate the survey of the roentgen findings in different stages of O.d.j. of the capitulum humeri.

It is clear from Fig. 2 that the process in the capitulum humeri shows the pathognomonic roentgenographic appearance of different stages of O.d.j. Fig. 2 D shows the initial stage; Fig. 2 E, the fragmentation stage; and Fig. 2 F, the healing period (Case 22 of author's material).

As mentioned, histologic investigations by various authors have shown that O.d.j. of the femoral head is primarily an aseptic necrosis of the ossification centre. On the other hand, no histologic investigations are available of such lesions in the capitulum humeri. Nevertheless, like other investigators, the present writer is of the opinion that the above mentioned similarities between O.d.j. of the femoral head and such lesions in the capitulum humeri justify the conclusion that both are different local manifestations of one and the same pathologic process.

On the basis of our present knowledge of O.d.j., and as will be discussed in subsequent sections, there seems to be reasonable roentgenologic and clinical evidence to assume that the pathologic process in the capitulum humeri is a typical lesion of osteochondrosis deformans juvenilis, *i.e.* aseptic necrosis of a growing ossification centre.

Recapitulation

1. The literature contains descriptions of changes in 42 different endochondral, growing ossification centres, claimed by the respective authors to belong to the group of diseases, referred to in the present publication as osteochondrosis deformans juvenilis (O.d.j.).

2. The uneven normal mineralisation in many endochondral ossification centres may make it difficult to establish a diagnosis of O.d.j. on the basis of a single roentgen examination.

3. There is general agreement that O.d.j. of the femoral head is the classical form of this group of diseases.

4. Of the similar changes seen in other ossification centres, the lesions in the capitulum humeri are those that most closely resemble O.d.j. of the femoral capital epiphysis. This similarity is striking with

respect to the roentgenologic appearance, the clinical picture, sex incidence, patients' ages, course and the obscure etiology.

5. Despite the unavailability of histological evidence there appears to be reasonable roentgenologic and clinical evidence to assume that the processes in the capitulum humeri and the femoral head are simply two different local manifestations of one and the same pathologic process.

The process in the capitulum humeri is a typical manifestation of osteochondrosis deformans juvenilis.

Chapter II

THE NORMAL ANATOMY OF THE DEVELOPING DISTAL END OF HUMERUS

Interpretation of the roentgenogram of O.d.j. in the ossification centre of the capitulum humeri and of the angiograms in the experimental investigation described in Part II requires knowledge of the normal anatomy of the distal end of the growing humerus.

It is above all necessary to know the time of appearance of the ossific nucleus in the chondroepiphysis of the capitulum as well as its roentgen appearance in different ages.

Like O.d.j. in other epiphyses, O.d.j. of the capitulum occurs during the period of ossification of the epiphysis. It has never been described to occur outside of a period, which begins with the appearance of the ossific nucleus in the cartilage and ends with its fusion with the surrounding osseous tissue, which in this case consists of neighbouring ossification centres in the epiphysis and the metaphyseal bone. It should be observed that the distal end of the humerus has 3 other ossification centres, namely in the medial epidondyle, in the trochlea and in the lateral epicondyle. The two last mentioned locations are of particular interest, since they are situated close to the ossific nucleus of the capitulum.

Finally, it is necessary to know when these ossific nuclei fuse with one another, and when fusion occurs between the epiphysis and the metaphysis, because this fusion marks the end of the period in which O.d.j. is known to appear.

The humerus is ossified mainly from the ossification centre in the diaphysis, which appears already in the 6th to 8th week of intrauterine life (SCHINZ et al. 1952, BRAILSFORD 1953, KÖHLER & ZIMMER 1956, CAFFEY 1956 and others). Such ossification centres appearing early in the bones, are often called primary ossification centres, while those appearing later in epiphyses are often referred to as secondary ossification centres.

At birth the distal end of the humerus is entirely cartilaginous, but it is gradually ossified from the four secondary ossification centres, of

which that in the capitulum appears first and is afterwards followed by the appearance of ossification centres in the medial epicondyle, trochlea and lateral epicondyle in the order given.

It is known that ossification begins at different ages in different epiphyses and that the time of ossification in any one centre of ossification varies from subject to subject (Table 2).

Some authors claim that the order of appearance of the ossific nuclei of the epiphyses is not necessarily the same on both sides in one and the same individual (ELGENMARK 1946, TORGERSEN 1951, SCHINZ et al. 1952, KÖHLER & ZIMMER 1956, CAFFEY 1956 and others), while others doubt, whether any appreciable difference exists (FLECKER 1942, SCHMID & HALDEN 1949).

On roentgen examination of 554 children PRYOR (1923) was able to confirm the observation of BADE (1899) that the skeleton ossifies earlier in the female than in the male. This advance in ossification of the female skeleton applies to the appearance of the ossific nuclei and the osseous fusion of the epiphyseal lines. These observations have been verified by many authors (HASSELWANDER 1931, FRANCIS & WERLE 1939, FLECKER 1942 and ELGENMARK 1946).

As mentioned, the distal end of the humerus is ossified from 4 ossification centres. Only 2 of them partake in the formation of the actual elbow joint, namely the ossification centre appearing first in the capitulum and the ossification centre in the trochlea. The other two ossification centres, in the medial epicondyle and the lateral epicondyle do not take part in the formation of articular surfaces.

Opinions differ fairly widely on the age at which these ossification centres appear in the chondroepiphysis as well as regarding the time of fusion between the various epiphyseal ossific nuclei and between these and the metaphysis (Table 2, Table 3). This is probably due to differences in the method of determining the presence of an ossific nucleus or to the conclusions being based on too small a number of observations.

A reliable opinion of these phenomena can be obtained only from those investigations carried out on large materials and in which sex-linked differences have been taken into account.

Of the investigations of the growing distal end of the humerus, those of HASSELWANDER (1931) are based on a fairly large material. His data are founded on a roentgenologic investigation by KÖHNLE, of more than 2000 healthy children.

TABLE 2

Time of appearance of the centres of ossification in distal end of humerus

Author	Year of publicat.	Capitulum humeri	Medial epicondyle	Trochlea	Lateral epicondyle
COHN, I.	1921	Earliest at 17 months	7—11 1/2 years	8—10 years	Presence is exeption
PATERSON, R. S.	1929	1—1 1/2 years	♀ = 5—6 years ♂ = 8—9 „	10 years	14 years. Presence in 70 %
HASSELWANDER, A. (KÖHNLE, H.)	1931	In first year	♀ = 5—8 years ♂ = 7—9 „	♀ = 7—11 years ♂ = 8—13 „	♀ = 8—11 years ♂ = 9—13 „
KING, E. S. J.	1935	♀ = 9—18 months ♂ = 11—18 „	♀ = 3 1/2—6 y. ♂ = 5 1/2—7 y.	8 1/2 years	9 years
FRANCIS, C. and WERLE, P. P.	1939	♀ = From 3 months ♂ = From 3 months			
FLECKER, H.	1942	♀ = From 9 m. ♂ = „ 11 „	♀ = From 5 y. ♂ = „ 7 y.	♀ = From 7 y. ♂ = From 8 y. 9 m.	♀ = From 8 1/2 y. ♂ = „ 10 y. 7 m.
ELGENMARK, O.	1946	♀ = 1—11 months ♂ = 1—26 „			
SCHMID, F. and HALDEN, L.	1949	First year	4—9 years	8—11 years	8—12 years
SCHINZ, H. R., BAENSCH, W. E. et alia.	1952	6 w.—8 m.	4—7 years	8—9 years	12—14 years
BRAILSFORD, J. F.	1953	Early in second year	In 4th.—5th. year	In tenth year	In ninth year
CAFFEY, J.	1956	♀ = 1 m.—6 m. ♂ = 6 w.—8 m.	♀ = 27 m.—61 m. ♂ = 57 m.—84 m.	♀ = 7 y.—9 y. ♂ = 8 y.—9 y.	♀ = 12 y.—14 y. ♂ = 12 y.—14 y.

TABLE 3

Time of fusion between the various osseous structures in the distal end of the growing humerus

Author	Year of publication	Fusion between:			
		Capitulum and Trochlea	Capitulum and ext. epicond	Conjoint epiphysis and metaphysis	Int. epicondyle and metaphysis
COHN, I.	1921	11—13 years		13—14 years	
HASSELWANDER, A. (KÖHNLE, H.)	1931		♀ = From 11 y.	♀ = 12—14 years ♂ = 13—16 „	♀ = From 14 y. ♂ = „ 17 y.
KING, E. S. J.	1935	♀ = 10—16 years ♂ = 12—16 „	♀ = 10—16 y. ♂ = 12—16 y.	♀ = At 13 years ♂ = At 15 „	♀ = 10—16 y. ♂ = 12—21 y.
LANZ, T. v., WACHSMUTH, W.	1935	13—16 years	13—16 years	14—16 years	14—18 years
FLECKER, H.	1942	♀ = From 10 y. ♂ = „ 12 y.	♀ = From 10 y. ♂ = „ 12 y,	♀ = From 13 y. 4 m. ♂ = „ 14 y. 7 m.	♀ = From 10 y. ♂ = „ 12 y.
SCHINZ, H. R., BAENSCH, W. E. et alia.	1952	During 14th y.	During 14th y.	15—17 years	
BRAILSFORD, J.	1953	After 14th y.	After 14th y.	After 16th y.	After 18th y.

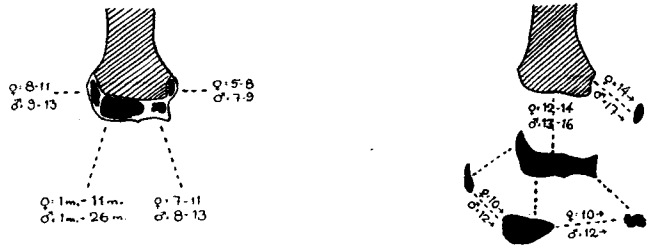


Fig. 3. Ossification of distal end of humerus.

Time of appearance of centres of ossification. Time of fusion between osseous structures.

(Figures followed by "m"=months; otherwise=years.)

The most detailed description of the roentgenographic appearance of the ossification centre in the capitulum humeri is probably that published by ELGENMARK (1946) on the basis of a material consisting of 852 healthy children, aged 0—5 years.

The following description of the ossification of the distal end of the humerus is founded to a large extent on the publications of HASSELWANDER and of ELGENMARK.

I. Capitulum humeri

ELGENMARK (1946) found that the ossific nucleus began to appear in the capitulum as early as in the first month of life in both sexes. He demonstrated the nucleus in 50 per cent of the females at 5.7 months of age and in 50 per cent of the males at 6 months of age. Ossification was demonstrable in the capitulum at 11 months in all of the girls and at 26 months in the boys (Table 2, Fig. 3). Thus, as pointed out earlier by PRYOR (1923), the advance in the ossification in females increases with the age of the children.

According to HASSELWANDER (1931), the major part of the chondroepiphysis of the capitulum is ossified by the age of 5—6 years. In addition to joint cartilage, however, part of the chondroepiphysis remains unossified for some time, namely the cartilaginous epiphyseal plate, and a relatively broad band of cartilage, extending outward as the lateral epicondyle.

In girls the epiphyseal plate between the ossific nucleus in the capitulum and the metaphyseal bone becomes fairly narrow by the age of 10 years, though bony fusion does not occur until 12 years at the ear-

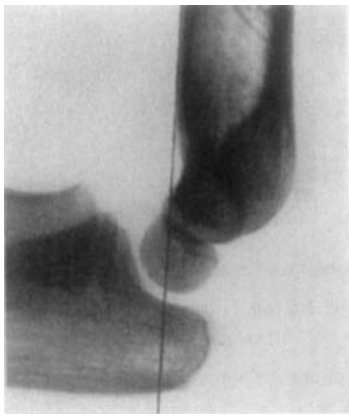


Fig. 4. Normal position of ossification centre of capitulum humeri. (See text.)

liest. At 13 years the cartilaginous line has usually disappeared and at 14 years regularly so (Table 3, Fig. 3).

In boys this fusion occurs later and proceeds over a longer period, namely between 13 and 16 years of age (Table 3, Fig. 3).

Union with the metaphysis does not occur until the ossific nucleus of the capitulum has fused with those in the trochlea and the lateral epicondyle. According to FLECKER (1942) this fusion occurs almost simultaneously on both sides, though fusion with the nucleus in the epicondyle is sometimes slightly later than with that in the trochlea. This fusion, which, according to FLECKER, occurs at 10 years or later in girls and at 12 years or later in boys thus results in the formation of a common ossification centre for the entire articular process of the distal end of the humerus and lateral epicondyle. This ossific nucleus has been called the conjoint epiphysis (Table 3, Fig. 3).

FLECKER (1942) stresses the smooth outline of the ossification centre in the capitulum, which contrasts with the physiological uneven ossification centre in the ulnar part of the trochlear cartilage.

CAFFEY (1956) does not include the ossification centre of the capitulum in the group of centres in which irregular mineralisation may normally occur.

FRANCIS & WERLE (1939) found irregular texture in some recently formed ossification nuclei of the capitulum. Similar observations were made by SONTAG & PYLE (1941), who called this phenomenon "disseminated provisional calcification".

Physiologically irregular ossification has, however, not been described

in this ossification centre during that period of life, in which O.d.j. of the capitulum is known to appear. By then the ossification is fairly well advanced, because ossification of that centre starts at a fairly early age. All authors describe the structure and outline of ossification in that centre as fairly regular during this period.

According to COHN (1921) the developing ossific nucleus of the capitulum occupies a relatively constant position. The normal position of the ossification centre is determined by WATSON-JONES (1955) by dropping a line down the front of the humeral shaft as seen in a lateral roentgenogram. Normally, the major part of the ossification centre of the capitulum will lie dorsal to this line (Fig. 4).

II. Trochlea

According to HASSELWANDER (1931) the ossific nucleus appears here at the age of 7 to 11 years in girls and between 8 and 13 years in boys (Table 2, Fig. 3).

This ossification centre frequently shows irregular mineralisation. The ossification process often appears at the same time in several spots which afterwards fuse to a single nucleus of irregular outline. This irregular mineralisation of the normal ossification centre has been described by various authors (FLECKER 1942, SCHINZ et al. 1952, TALBOT, SOBEL & MCARTHUR 1952, CAFFEY 1956).

This irregularity in the ossification of the trochlea has sometimes caused confusion in the interpretation of roentgenograms, as has been pointed out by RUCKENSTEINER (1931) and WUSTMANN (1954), for example, and will be discussed further in chapter III.

As mentioned, the ossification centre of the trochlea fuses with that of the capitulum at about or after 10 years of age in girls and at or after 12 years in boys and then, as part of the conjoint epiphysis, fuses with the metaphysis at 12 to 14 years in girls and 13 to 16 years in boys (Table 3, Fig. 3).

HASSELWANDER (1931) stresses that the ossification in the epiphysis of the trochlea occurs unusually late for a "reguläre Hauptepiphysenossifikation". He claims that ossific nuclei in "Hauptepiphysen" usually appear during the first 5 years of life, an opinion also shared by ELGENMARK (1946), who stated that only few ossific nuclei in the limbs make their initial appearance after the age of 5 years.

III. Lateral epicondyle

The ossific nucleus appears in the lateral epicondyle at the age of 8 to 11 years in girls and between 9 and 13 years in boys (Table 2, Fig. 3) (HASSELWANDER 1931).

This ossific nucleus fuses with that of the capitulum after 10 years of age in girls and after 12 years of age in boys (FLECKER 1942) (Table 3, Fig. 3). HASSELWANDER points out that once the centre has appeared this fusion occurs relatively rapidly. This rapid fusion with the ossific nucleus of the capitulum probably explains why some investigators such as COHN (1921), PATERSON (1929) (Table 2) and others assumed that the lateral epicondyle seldom, if ever, has any ossification centre of its own but is ossified by extension from the ossific nucleus of the growing capitulum. This mode of ossification is denied by HASSELWANDER (1931), FLECKER (1942) and others.

After the ossification centre of the lateral epicondyle has fused with that of the capitulum, it has became part of the conjoint epiphysis and fuses together with the latter to the metaphysis, which, as already mentioned, occurs at the age of 12—14 in girls and between 13—16 years in boys (Table 3, Fig. 3).

IV. Medial epicondyle

According to HASSELWANDER (1931) the ossific nucleus of the medial epicondyle appears at the age of 5 to 8 years in girls and between 7 to 9 years in boys (Table 2, Fig. 3).

This ossific nucleus, like that of the trochlea, shows sometimes irregular granules as the initial sign of ossification.

This ossification centre fuses directly with the humeral metaphysis without any relation to the other ossification centres in the distal end of the humerus. This fusion (HASSELWANDER 1931) occurs after 14 years of age in girls and after 17 years of age in boys (Table 3, Fig. 3).

Relation between attachment of capsule and epiphyseal plate in distal end of humerus

It might not be out of place to give a few brief remarks on the attachment of the joint capsule on the distal end of the humerus and its relation to the epiphyseal plate, *i.e.* the cartilage zone separating the ossifying epiphysis from the metaphysis.

The major part of the epiphyseal plate in the distal end of the humerus is intracapsular, but so that both epicondyles which provide important attachments for muscles, are situated extracapsulary. Therefore, part of the laterodorsal portion of the epiphysis will lie extracapsulary and the epiphyseal plate, the lateral part of which is situated proximally to the lateral epicondyle, will be situated in its laterodorsal portion proximal to the capsule, which there is attached at the posterior and lateral edge of the joint cartilage on the capitulum and lateral edge of the joint cartilage of the trochlea (Fig. 5).

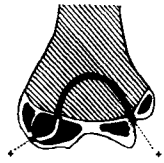


Fig. 5. × Dorsal attachment of joint capsule on distal end of growing humerus.

The only part of the capitular chondroepiphysis where the ossific nucleus of the capitulum lies against a surface that is not covered by joint cartilage is a region located laterodorsally on the chondroepiphysis. This region is limited proximally by the epiphyseal plate and medially and distally by the capsular attachment at the edge of joint cartilage (Fig. 5).

The significance of these anatomical relationships for the vascular topography of the ossification centre of the capitulum humeri will be discussed further in Part II.

Recapitulation

1. Opinions differ on the age at which the ossific nucleus of the capitulum and the other three nuclei in the distal end of the humerus first appear in the roentgenogram. According to investigations based on large materials the ossific nucleus in the capitulum appears between the ages of 1 and 11 months in girls and 1 and 26 months in boys. Corresponding data for the other 3 ossification centres in the distal end of the humerus are: medial epicondyle: girls 5—8 years, boys 7—9 years, trochlea: girls 7—11 years, boys 8—13 years and lateral epicondyle: girls 8—11 years, boys 9—13 years.

2. Opinions also differ on the time of fusion between the various parts of the distal end of the humerus. According to investigations on large series, the ossific nucleus of the capitulum fuses with neighbour-

ing nuclei in the trochlea and lateral epicondyle after 10 years of age in girls and 12 years in boys. All 3 centres fuse to form the conjoint epiphysis, which fuses with the metaphysis between the ages of 12 and 14 years in girls and 13 and 16 years in boys. The medial epicondyle fuses directly with the metaphysis from 14 years of age in girls and 17 years in boys.

3. A search of the literature failed to reveal any descriptions of irregular normal mineralisation of the ossific nucleus of the capitulum during the period, in which O.d.j. of the capitulum is known to appear. All authors described structure and outline of this nucleus as fairly regular during the period under discussion. The regular ossification of the ossific nucleus in the capitulum contrasts sharply with the irregular normal ossification of the nucleus of the trochlea.

4. The anatomic relationship between the cartilaginous epiphyseal plate and the dorsal attachment of the joint capsule on the distal end of the humerus may be of significance for the vascular topography of the ossification centre of the capitulum humeri (see also Part II).

Chapter III

SURVEY OF LITERATURE

I. Earlier investigations

As mentioned in chapter I, during the last 50 years numerous reports have been published on O.d.j. in various endochondral ossification centres.

Despite this voluminous body of literature only few cases of O.d.j. of the ossification centre of the capitulum humeri have been published. A fairly detailed description of the cases on record was therefore considered desirable, even though the clinical picture and roentgenologic appearance do not differ widely from case to case.

AMSTAD (1916) reported that ISELIN had placed at his disposal a case in which the roentgenographic appearance of the growing eminentia capitata humeri was the same as that seen in "Osteochondritis deformans juvenilis" in the femoral head and concluded that it was thus a case of "Atrophie mit vermehrter Kalkaufnahme". But AMSTAD did not give any details about the case, nor did he give any roentgenographic reproductions in support of his conclusion.

WILD (1921) described roentgenographic changes in the capitulum humeri in a 14-year old child and interpreted the changes as "Osteochondritis deformans juvenilis". The patient had 3 weeks' history of pain in the elbow, and the region over the radial part of the joint was tender to palpation. WILD, however, gave no roentgenographic reproductions or further details of the case, so that it is not possible to conclude with certainty whether the diagnosis of O.d.j. of the capitulum was really justified.

CAAN (1924) reported that he had observed "wolkige Trübungen und Aufhellungen" in the distal end of the humerus in two patients, and that the only interpretation available was "Osteochondritis deformans juvenilis". But neither did this author give any closer description of these cases, neither did he say which part of the distal end of humerus it was that was involved nor give any accompanying roentgenographic reproductions. It is therefore not possible to decide with certainty, whether these 2 cases were examples of O.d.j. of the capitulum or not.

PANNER (1927, 1929, 1930) was the first to present a clinically and roentgenologically acceptable description of O.d.j. of the capitulum humeri. His material consisted of 3 boys, who had always felt well, and who at the time of the first examination were 10, 10 and 7 years old, respectively. All 3 patients had sustained definite trauma against the elbow, the first patient 2 weeks before the first examination, the second 3 months before and the third 6 weeks before. All 3 had clear clinical symptoms in the region of the elbow. The affection was on the right side in 2 and on the left in 1.

The symptoms immediately following the trauma in all 3 cases were pain, tenderness and limitation of movement of the elbow joint.

On first examination all 3 patients showed the same type of signs. All of them were normally developed and in a good general condition. The elbow was swollen and tender to palpation and extension was impaired. In one of them flexion was also limited. Pronation and supination were normal in all. Two of the patients showed slight muscle atrophy of the arm involved. No symptoms from other joints. The patients showed no signs of infection. The tuberculin test was performed on 2 and it was negative in both.

Roentgen examination showed similar changes in all three. On first examination a change was noted in the structure of the ossification centre of the capitulum, which showed fissure-like rarefactions. A flossy less well defined outline of the ossification centre was also observed.

Roentgenologic follow-up showed that the ossification centre had become smaller than that on the contralateral side, besides its outline became deeply indented and irregular spots of rarefaction and increased density appeared.

On culmination of the roentgenologic changes the ossification centre appeared fragmented. In one of the cases PANNER also pointed out that the distance between the ossification centres in the capitulum humeri and in the capitulum of the radius on the affected side, was greater than on the other side, and he cited CALVÉ's words on vertebra plana: "too little bone, too much cartilage".

On further roentgenologic control the size, outline and structure of the ossification centre of the capitulum regained almost normal appearance. According to PANNER, healing took at most 3 years. However, on analysis of the final roentgenographic reproductions accompanying PANNER's article, it will be seen that the roentgenogram of the capitulum was not quite normal in any of his cases. In all of them the osseous

structure was somewhat uneven, and the fine, rounded outline was missing.

With reference to WALDENSTRÖM's (1922) classification of coxa plana (page 21), it will be seen that the roentgenographic reproductions given by PANNER represent all of the roentgenologic stages described by WALDENSTRÖM, except the definite stage, which is not reached until growth is complete. This similarity with O.d.j. of the femoral head (coxa plana) is also underlined by PANNER, who believes both diseases to be fundamentally identical.

In all of PANNER's cases the diseases was unilateral.

Although healing was so slow — up to 3 years — most of the symptoms disappeared much earlier without any special treatment. There was thus a certain discrepancy between the clinical symptoms and the roentgenographic changes. PANNER did not say exactly when the patients were completely free from subjective and objective clinical symptoms. In the first of his patients extension of the elbow was still slightly limited 3 years after onset of the symptoms. The second patient had slight limitation of extension and mild pain on weight-bearing one year and five months after the onset of the symptoms. As to the third patient, PANNER simply stated that he soon became symptom-free.

PANNER followed up his patients for at most 3 years and thus not until the definite stage had been reached.

At the meeting of the *Northern Association of Medical Radiology* in Copenhagen 1927, at which PANNER presented his first report of the disease, KREBS described a case of the same disease, which he published later that year. The patient was an 8-year old boy, who 2 years before the onset of symptoms had had a traffic accident. KREBS pointed out that it was not possible to say whether he had sustained any severe trauma at that time, and that at any rate no external signs of such a trauma were observed immediately after the accident. Neither could the boy remember having experienced any other significant trauma.

Two years after the accident, the boy began to complain of pain in the right elbow and trembling of the right hand, especially when writing.

When first seen by KREBS 6 months after the onset of symptoms the boy was normally developed, and he was in a good general condition. The elbow in question was swollen but not tender and both extension and flexion of the elbow were limited. Pronation and supination were normal. The tuberculin test was positive. Wasserman's test was negative. All other joints appeared normal.

The reproductions of the roentgenograms show that on that occasion the

pathologic process was in the fragmentation stage. KREBS underlined the roentgenographic similarity with O.d.j. of the femoral head.

The patient received only expectant treatment.

When last examined 18 months after the onset of the symptoms, flexion was still somewhat defective and the circumference of the joint was somewhat larger than on the other side. The roentgenographic reproductions from the final examination, 18 months after the onset of symptoms, showed that the process was in the healing stage, but the structure of the ossification centre was still irregular, its outline was deformed and it was somewhat flattened.

KREBS suggested "Maladie de Panner", a name which was afterwards generally accepted.

SMITH (1929) reported on a 4-year old boy with O.d.j. of the left femoral head. Roentgen examination of other joints was said to show a similar lesion in the ossification centre of the left capitulum humeri. SMITH stated that the elbow showed no clinical evidence of a pathologic condition but he gave no further details. He based his diagnosis on a single roentgen examination.

On closer examination of the roentgenographic reproduction given by SMITH, it is not possible to decide with certainty whether the lesions should be classified as O.d.j. of the capitulum or not.

BUSCH (1930) published a case of O.d.j. of the capitulum humeri in a 9-year old boy, who had until then felt well, and who without any previous known trauma suddenly developed a non-tender swelling of the right elbow with pain on movement of the joint.

On first examination extension and flexion of the joint were considerably limited, while pronation and supination were normal.

Tuberculosis of the joint was suspected, but roentgen examination 3 weeks after the onset of symptoms showed changes in the ossification centre of the capitulum, which were interpreted as Morbus Panner. The roentgenographic appearance of the elbow on the other side was normal.

Judging by the accompanying roentgenographic reproductions the pathologic process was in the initial stage of the evolutionary period.

The symptoms were described as having soon disappeared, but no details are given of the course of the disease or the treatment given. BUSCH stressed, however, that the disease should perhaps not be regarded only as a roentgenologic rarity but also as a disease that must be expected in practical life.

WINTERSTEIN (1933) described a 13-year old boy, who was working as an acrobat and thus strained his arms more than what can be regarded as normal for his age. He sought medical advice because of painless impairment of mobility of the right elbow.

According to this author, roentgen examination of the elbow showed

changes in the ossification centre of the capitulum humeri of the type described by PANNER (1927, 1929, 1930). WINTERSTEIN underlined that he did not believe that his patient had osteochondritis dissecans.

The accompanying roentgenographic reproductions, however, do not show total necrosis of the ossific nucleus of the capitulum, *i.e.* changes generally recognised as being pathognominic of O.d.j. of the capitulum humeri.

WINTERSTEIN's roentgenograms showed circumscript rarefactions in the radial part of an otherwise normal ossification centre in the capitulum. The roentgen appearance was thus not that of O.d.j. of the capitulum humeri. Neither did the age of the patient at onset fit in with such a diagnosis.

HAGEN & BISSCHOP (1933) contributed with a case of O.d.j. of the capitulum humeri in a hitherto healthy 8-year old boy, who had for 4 weeks complained of pain and impaired extension-flexion of the right elbow. He could not remember having sustained any trauma with certainty.

On examination the boy was found to be normally developed and in a good general condition. Pronation-supination of the elbow was normal. No local or generalised signs of inflammation were observed. Tuberculin and Wassermann tests were negative.

Roentgen examination showed changes in the ossification centre of the capitulum, which the authors described as resembling those seen in "Mb Perthes". They therefore belived that they were dealing with the disease described by PANNER.

Judging by the roentgenographic reproductions, the patient had O.d.j. of the capitulum in the initial stage of the evolutionary period.

These authors took arthrograms of the elbow joint. Judging by the accompanying reproductions, the articular surface of the capitulum was smooth, but otherwise it is difficult to form any definite opinion of the outline of the cartilaginous epiphysis from these reproductions.

They gave no further details about the case.

UHRMACHER (1933) published 2 cases of what he called "Osteochondritis deformans juvenilis" of the elbow bilaterally in 2 young girls. He believed these cases to be of the same type as that described by WILD (1921) and PANNER (1927, 1929, 1930).

But in both of the cases described by UHRMACHER it was in the ossification centres of the humeral trochlea that he observed the changes, which *inter alia* included fragmentation of the "normale glatte Kontur der Trochlea".

As mentioned (page 15), it is believed by some authors that O.d.j.

may, theoretically, occur in any endochondral ossification centre in the body. O.d.j. might thus occur in the ossification centre of the humeral trochlea just as well as in the capitulum.

But, on the other hand, the diagnosis of O.d.j. is based mainly on the roentgenographic appearance. In the ossification centre of the trochlea, unlike that in the capitulum, mineralisation is often uneven during the course of ossification, which, as mentioned, has been observed by several authors. The normal roentgenographic appearance of this ossification centre thus often resembles that of O.d.j.

It must therefore surely be difficult on the basis of a single roentgenographic examination to decide whether the process reflected in the roentgenogram should be regarded as a destructive lesion in the ossification centre of the trochlea or as a normal variant of ossification. Any such decision may require roentgenologic follow-up during a long time, and even then it may be difficult in cases of O.d.j. not examined until the process has reached the healing period. These requirements were not satisfied by UHRMACHER, so that it is not certain whether it really was a question of O.d.j. in his cases.

SCHAEFER, STRICKROOT & PURCELL (1939) and HEGEMANN (1951) published cases analogous with UHRMACHER's, as did PALTRINIERI (1953) and KHADZHIDEKOV (1954).

What was said of UHRMACHER's cases hold essentially also for those of all these authors.

KING (1935) reported that he had observed O.d.j. of the capitulum humeri in 3 children, 2 boys and a 5-year old girl. He published typical roentgenograms of a single examination of the girl. He described local symptoms in the form of pain, swelling, tenderness and impaired range of movement of the elbow and recommended that such elbows should be spared, but he gave no detailed description of these cases.

He expressed the view that the clinical and roentgenologic pictures are characteristic and that the roentgen findings strongly suggested that the disease is allied to O.d.j. of the femoral head.

In 1937 NIJST published a case of O.d.j. of the capitulum humeri in an otherwise healthy 8-year old boy, who had a 6 months' history of pain on movement of the right elbow. The boy could not remember having sustained any blow against the elbow.

On examination the patient's general condition was good, the elbow was of normal appearance and felt normal on palpation. Extension-flexion and pronation-supination of the joint were markedly limited.

The reproductions of the roentgenograms accompanying his report show O.d.j. of the capitulum humeri in the fragmentation stage. The author observed the similarity between the roentgenogram in that case and that of O.d.j. of the femoral head and diagnosed the case as Mb Panner.

On review 9 1/2 months after the onset of symptoms roentgen examination showed that the disease was in the healing period, and the patient reported that the symptoms were subsiding.

No further details were given about the case or the therapy given.

MOL (1939) reported a case of left-sided O.d.j. of the capitulum humeri in a 9-year old boy. Six months before the first roentgen examination the boy had fallen and injured his elbow, after which local clinical symptoms had appeared.

After the blow the elbow was painful and extension-flexion was diminished.

On initial examination, 6 months after the accident, the elbow was found to be slightly swollen and warm and tender to palpation over the radial part of the joint, where slight crepitations were also noted. Cubitus valgus was found to be increased compared with the other side. Extension-flexion was limited and further passive movement was painful. The E.S.R. was normal, and the tuberculin test was negative.

Accompanying roentgenograms show intermingled densities and rarefied spots in the ossification centre.

The patient was instructed to rest the joint.

The clinical symptoms gradually abated, and when last seen one year and three months after the onset of symptoms the patient felt well. The range of extension-flexion was, however, still slightly impaired and cubitus valgus was still increased. Slight muscular atrophy of that arm was noted.

The author stressed the similarity with "Legg-Calvé-Perthes" disease.

No further details are given.

SCHAEFER, STRICKROOT & PURCELL (1939) published the results of a roentgenologic examination of a series consisting of 258 patients, aged 8—15 years, with endocrine disease and of a control material consisting of 99 children of corresponding ages.

The authors found signs of "juvenile chondro-epiphysitis" in 91 (35.2 %) of the patients. Of the 91 patients, 85 showed clinical and roentgenologic evidence of primary or secondary hypothyroidism and 38 of multiple epiphyseal disorders. Only 3 of these 91 patients had clinical symptoms referable to the osseous involvement. They did not say anything about the clinical symptoms or the localisation of the osseous involvement in these 3 patients.

They stated that 3 of the patients showed changes in the ossification centre of the capitulum humeri and that the alterations were of the

type described by PANNER (1927, 1929, 1930). They gave no further details or roentgenographic reproductions of these patients.

On the basis of histologic examinations, LOOSER (1929) showed that, pathologically, O.d.j. differed widely from the irregular, retarded, endochondral ossification in hypothyroidism. O.d.j. is a degenerative process in a previously normal ossification centre. The centre is broken down and then assumes a fragmented appearance in the roentgenogram, after which it regenerates. This process usually involves only one ossification centre and is often painful. In hypothyroidism on the other hand, it is a question of multicentric endochondral ossification, which roentgenologically resembles O.d.j. These multicentric ossification centres progress slowly and gradually fuse to a single ossific nucleus of irregular outline. These ossific nuclei may be deformed secondarily on exposure to trauma or strain because they do not possess the same stability as ossification centres with unicentric homogenous ossification.

Epiphyseal disorders are often multiple in hypothyroidism. TALBOT et al. (1952) and CAFFEY (1956) called them epiphyseal dysgenesis and distinguished them sharply from O.d.j. Such distinction is also made by ALBRIGHT (1938), HOWORTH (1952) and BRAILSFORD (1953).

This does not exclude the possibility of true O.d.j. occurring in children with hypothyroidism.

With reference to the changes in the ossification centre of the capitulum humeri described by SCHAEFER et al. (1939), it must be concluded that since these authors do not give any reproductions of the roentgenograms of these cases or any further details, it cannot be concluded with certainty whether O.d.j. of the capitulum humeri was present or not. JUD (1953) is of the same opinion.

In 1939 RIVAROLA described a 6-year old boy with O.d.j. of the left capitulum humeri. Preceding trauma was suspected but not described in detail.

The patient was first examined the day after the onset of the symptoms. The region of the elbow was swollen but not tender. Extension, flexion and supination were impaired and forced passive movement of the elbow was painful.

Roentgen examination showed the disease to be in the initial stage of the evolutionary period.

Five months after the onset of the symptoms roentgen examination showed that the process was in the healing stage. The patient still had local, clinical symptoms.

When last seen one year and three months after the onset of the symptoms

the patient was symptom-free. Roentgen examination, however, still showed that the process had not completely healed.

RIVAROLA considered this to be a case of the same disease as O.d.j. of the femoral head.

In 1939 ELWARD reported on two 8-year old boys with O.d.j. of the capitulum humeri.

In one of these boys, who had hitherto been healthy, initial examination revealed impaired range of movement and pain of the right elbow. The patient's general condition was good, and he could not remember having sustained any trauma. ELWARD did not say how long the patient had had symptoms before first examination.

Reproductions of the roentgenograms taken on first examination show that the process was in the initial stage of the evolutionary period and films taken 3 months later show the picture of the fragmentation stage.

The patient was instructed to rest the joint and the symptoms gradually subsided. No further details of the course of the disease were given. On review $6\,1/2$ years after the first examination the elbow was found to be normal.

The second patient had sustained a blow against the left elbow a few days before the first examination. He complained of pain and stiffness of the left elbow. The roentgenograms taken in association with the first examination show O.d.j. in the initial stage of the evolutionary period. Check-radiography one year later show a picture of the fragmentation stage.

The patient only received expectant treatment. No further details were described.

KOHLBACH (1940) published 2 cases of O.d.j. of the capitulum humeri in a girl and a boy, both aged 5 years. He expressed the view that both had the same pathologic process as that seen in "Perthes' disease" of the hip.

The former of these 2 patients had no history of trauma. She had been referred for roentgen examination because of suspected cubital tuberculosis. For a few weeks before the first examination she had complained of pain in the right arm.

On examination extension-flexion was found to be impaired, but otherwise the joint was of normal clinical appearance. Roentgenograms taken on that occasion show O.d.j. of the capitulum in the initial stage of the evolutionary period. The roentgenographic appearance of the contralateral elbow was normal.

On review 3 years later the joint had recovered normal range of mobility. Roentgenograms taken on that occasion show that the pathologic process was healed but that it had not reached the definite stage because the child was still growing.

The second patient had fallen on the right elbow 3 days before the first

examination. Pain developed and he was referred for roentgen examination because of suspected fracture.

On examination the elbow was found to be painful on movement and its range of extension-flexion was reduced. The accompanying roentgenographic reproductions of films taken on that occasion show O.d.j. of the capitulum humeri in the initial stage of the evolutionary period. KOHLBACH stressed that the roentgenologic changes in the ossific nucleus of the capitulum in that case did not resemble those of a fresh injury and were therefore not due entirely to the trauma 3 days earlier but must have existed there before. The roentgenographic appearance of the other elbow was normal.

He gave no further details about this case.

In 1944 MAROTTOLI described a case with what he called "osteochondrosis" of the external condyle of the humerus. He described the changes as being the same as those seen in the disease described by PANNER (1927, 1929, 1930) and others.

Since MAROTTOLI's patient was a 20-year old man, in whom ossification of the distal end of the humerus had thus been concluded, it appears warranted to exclude this case as being an example of O.d.j. of the capitulum humeri.

MARCH (1944) published a case of O.d.j. of the right capitulum humeri in an 8-year old boy.

Three weeks before the first examination the boy had thrown a dart with right hand and afterwards observed diminished range of extension-flexion in the elbow on that side and slight pain on extreme movement of the joint.

The lateral part of the elbow was slightly swollen. The range of pronation and supination was normal. The accompanying reproductions of the roentgenograms taken on that occasion show that the process was in the initial stage of the evolutionary period. No further details were given of the case or of the further course.

HERMODSSON (1947) described a 13-year old boy, who 8 1/2 months after a severe blow against the right elbow, was found to have roentgenologic changes in the ossific nucleus of the capitulum humeri, which, according to the author, must "be paralleled with" those described by PANNER (1927, 1929, 1930).

Roentgen examination immediately after the blow showed no signs of primary injury to the ossificaiton centre of the capitulum humeri.

However, analysis of the films taken on the review 8 1/2 months after trauma show a picture resembling that described by WINTERSTEIN (1933) (vide supra). The lesion was thus not O.d.j. of the capitulum

humeri with the pathognomonic total necrosis of the ossific nucleus but a circumscript necrotic process radially-distal in an otherwise roentgenologically normal ossific nucleus. The age at onset also lay outside that of the 30 well-documented cases of O.d.j. of the capitulum humeri in the literature.

This case is nevertheless of considerable interest in the discussion of the etiology of aseptic necrosis in growing ossific nuclei in children, and will therefore be discussed further in association with the etiology and pathogenesis.

In a publication in 1951 HEGEMANN described "spontanen aseptischen Totalnekrosen" of the ossification centre of the capitulum humeri in 4 children, aged 5, 6, 7 and 8 years. This finding was made in the course of an examination of some 1200 "Röntgenaufnahmen" of patients who had sought medical advice because of elbow trouble.

The author, however, gave a detailed report of only one of these 4 cases, an 8-year old boy. Roentgen films from that case show O.d.j. of the capitulum humeri.

The boy, who could not remember having sustained any trauma, complained of pain in the left elbow. Examination revealed decreased range of extension of the elbow without pain on movement. The joint did not appear to be inflamed. The E.S.R. was normal. According to the author, the first roentgenogram, of which no reproduction is given, showed "unklarer Prozess am capitulum humeri".

The patient was instructed to rest the joint and, later, physical therapy was started. Three months after the first examination the patient again complained of pain in the joint, which was therefore immobilised in plaster. Those roentgenograms accompanying the article were taken 5 months after the first examination and show O.d.j. of the capitulum humeri in the beginning of the healing period.

The author mentioned that in none of his 4 cases of "aseptic total necrosis" of the ossific nucleus of capitulum humeri was any preceding trauma or mechanical strain known. All had clinical symptoms, consisting of diffuse local pain with slight swelling of the soft tissues and slight decrease in range of movement of the joint. He also mentioned that one of the patients was a girl, but he did not say how old she was.

CAMERA (1951) reported a case of O.d.j. of the right capitulum humeri in an otherwise healthy 6-year old boy without any known history of trauma.

Medical advice was sought because of one month's pain and limited mobility of the elbow joint.

Examination revealed swelling, tenderness to palpation and painful extension-flexion of the elbow.

Reproductions of roentgenograms taken 9 1/2 months after the onset of symptoms show that the process was in the fragmentation stage.

When last seen one year and two and a half month after the first examination the patient was symptom-free.

Accompanying reproductions of roentgenograms taken on that occasion show that the process was in the healing period.

Treatment had consisted of immobilisation of the joint in plaster.

Nothing is said about the further course of the disease.

SEMMELROCH (1952) contributed with a case of O.d.j. of the ossification centre of the right capitulum humeri in a 9-year old boy.

The patient, who was otherwise healthy, had strained himself while doing gymnastics some time before the onset of symptoms (nothing is said of the interval between the trauma and onset of symptoms).

By the time he was first examined, extension of the right elbow had been impaired for 2 months. The clinical examination showed the boy to be in a good general condition. The only local finding was decreased extension of elbow joint, thus no swelling or tenderness of the elbow. The E.S.R. was normal, and blood studies and chest x-ray showed nothing remarkable. The tuberculin test was positive.

Roentgen examination on that occasion (no reproductions given) was said to show a destructive process in the capitulum humeri. The reproductions given of roentgenograms taken 3 months later show the fragmentation stage of the evolutionary period. Roentgenogram of the other elbow showed nothing remarkable, but SEMMELROCH discovered an unilateral disturbance of ossification in the proximal part of the left patella, where the patient had had clinical symptoms in the form of swelling of the soft tissue and pain on movement.

The joint was immobilised.

The patient was last examined one year and eight months after onset of the symptoms. He was then symptom-free. Reproductions of the roentgenograms taken on that occasion showed that the pathologic process in the capitulum was in the healing period, but the structure, contour and size of the ossification centre had not yet recovered normal roentgenologic appearance.

BUETTI (1953) described O.d.j. of the capitulum humeri in 2 boys, aged 10 and 8 1/2 years.

The former had had some weeks' pain and swelling in the region of the right elbow before the first examination. He could not remember any blow. The reproductions of the roentgenograms taken on that occasion show that the process was in the initial stage of the evolutionary period. Roentgenograms of the other elbow revealed nothing remarkable.

Roentgen examination 3 months later showed the fragmentation stage.

The case is not described in further detail.

The other patient had, generally speaking, always been healthy. The evening

before the first examination he had fallen on his right elbow, which had swelled and become stiff and was painful, especially on movement. These symptoms were so pronounced that fracture was suspected.

Reproductions of the roentgenograms taken on that occasion, however, show O.d.j. of the capitulum humeri in the initial stage of the evolutionary period.

Roentgenograms of the other elbow, which was clinically symptom-free, showed the roentgenologic picture of O.d.j. of the capitulum humeri on the fragmentation stage. Roentgenograms of other joints showed nothing remarkable.

No further details were given.

This latter case of BUETTI is of fundamental interest, because it is the first case described in which O.d.j. of capitulum humeri was bilateral, and the process in the left elbow of that patient is the first described instance of O.d.j. of the capitulum humeri that produced no symptoms.

Similar bilateral cases of O.d.j. of the femoral head have, however, often been described. Since (see Chapter I) it has been concluded that these two diseases are probably only different local manifestations of one and the same pathologic process, O.d.j. of the capitulum humeri on both sides might occasionally be expected.

In 1953 MASON contributed with a further case of O.d.j. of the right capitulum humeri in an otherwise healthy 6-year old boy.

On first examination the patient had 3 weeks' history of pain in the elbow without by any known preceding trauma.

Flexion of the elbow was impaired and painful. Extension, supination and pronation were normal. The elbow was not swollen or tender to palpation. The patient's general condition was good. The tuberculin test was negative. Control examination of other joints revealed nothing remarkable.

The reproductions of the roentgenograms show O.d.j. of the capitulum humeri in the fragmentation stage.

The joint was immobilised in plaster for 2 months.

No further details were given. It was, however, mentioned that the clinical symptoms disappeared before the roentgenologic changes. The author also stated that he thought there was no risk for permanent deformity, because the elbow is a non-weight-bearing joint.

JUD (1953) reported a case of O.d.j. of the left capitulum humeri in a 7-year old, normally developed boy.

Eight months previously the patient had had a backward dislocation of the elbow joint on that side.

The dislocation was reduced one hour later and roentgen examination then showed nothing remarkable and particularly the ossific nucleus of the capitulum humeri was of normal appearance.

The joint was immobilised for 16 days, and the patient became symptom-free except for slight flexion contracture of that joint.

Eight months later the patient returned because of slight pain and swelling of the left elbow. Examination revealed slight tenderness to deep palpation over the capitulum humeri, decreased extension-flexion and more pronounced cubitus-valgus than on the other side. Supination was also slightly reduced. Pronation was normal. Forced passive movements of the joint were painful. On pronation-supination crepitations were heard over the radial head. The accompanying reproductions of roentgenograms show O.d.j. of the capitulum humeri in the initial stage of evolutionary period.

The elbow was periodically immobilised in plaster for 14 months, and the process was followed roentgenologically. It was observed how the process developed and passed into the fragmentation stage and how the fragmentation of the ossific nucleus of the capitulum advanced more and more, 12, 18 and 28 weeks respectively, after the onset of symptoms. At 18 weeks' check radiography showed a rarefied spot in the humeral metaphysis.

Accompanying reproductions of roentgenograms taken 9 1/2 months after the onset of symptoms (1 year and 5 1/2 months after luxation) show that the process was in the beginning of the healing period.

When last seen 1 year and 9 months after the onset of the symptoms (2 years and 5 months after luxation), the patient was symptom-free apart from a slight decrease in hyperextension of the left elbow.

Roentgenograms taken on that occasion show that the process was healed. The ossific nucleus of the capitulum had, however, not quite recovered normal roentgenographic appearance, the structure was somewhat uneven, the rounded smooth, normal outline was missing.

The patient was not followed up longer, so that nothing is known about the definite stage.

LANGE (1954) contributed with a case of O.d.j. of the right capitulum humeri in an otherwise healthy 10-year old boy.

The patient had 2 months' history of pain on movement and decreased range of mobility of the right elbow. The pain had not been preceded by any known trauma.

On examination the radial part of the elbow was found to be tender to deep palpation and flexion-extension was decreased. Pronation-supination was normal. The E.S.R. was normal. The tuberculin test was negative.

Reproductions of roentgenograms taken on that occasion show that the process was in the fragmentation stage. LANGE stressed the similarity with the roentgenogram of "Legg-Calvé-Perthes' disease", and believed the pathologic process to be the same in both diseases.

Subsequent check radiography 6 months after the onset of symptoms showed that the disease had passed into the beginning of the healing period and further roentgen control showed gradual normalisation of the structure and contour of the ossific nucleus. The last roentgenograms taken 2 years, 9 months after the onset of symptoms show an almost normal structure of the ossific nucleus,

which was, however, somewhat flattened compared with that on the other side and somewhat less rounded. The disease had thus healed with slight deformity of the capitulum but the definite stage had not yet been reached, because the patient was still growing.

The subjective and objective clinical symptoms gradually subsided and in two years the patient had recovered full movement of the joint, which was no longer painful.

The patient had been treated with 6 months' immobilisation of the elbow in plaster.

Finally, LAURENT & LINDSTRÖM (1956) published two cases of O.d.j. of the capitulum humeri in 2 boys, aged 9 and 8 years, respectively. The disease had not been preceded by any known trauma.

For some months before the first examination, the 9-year old patient had had periodic pain in the right elbow after use of that joint and for 2 weeks the elbow had been swollen and tender. Nothing is said about the range of movement in the elbow on that first examination.

Reproductions of roentgenograms taken at the first examination show that the process was in the initial stage of the evolutionary period.

Check radiography 3 months after the first examination showed the process to be in the healing period. The joint was still swollen and tender. The E.S.R. was 25 mm/1 hour. The joint was immobilised in plaster for one month, after which the subjective and objective symptoms disappeared except for slight impairment of extension.

Roentgenograms of the other (left) elbow also showed multiple rarefactions and an irregular outline of the ossification centre of the capitulum humeri, which these authors interpreted as evidence of O.d.j. The left elbow was free of clinical symptoms. Roentgen examination of this joint 3 months after the first examination (no reproductions of roentgenograms are given), were described as showing that reparative changes had taken place in the capitulum.

Check radiography 7 years and 5 months after the first examination show clinically and roentgenographically normal elbows.

The second patient of these authors had had pain in the right elbow for 1 month and swelling for 1 week before examination. On examination the radial part of the elbow was tender to palpation and extension was decreased. The E.S.R. was 21 mm/1 hour. The joint was immobilised in plaster for 3 weeks during which the swelling and tenderness disappeared.

Reproductions of roentgenograms taken one month and 3 weeks after the onset of symptoms show that the process was in the initial stage of the evolutionary period. On that occasion the E.S.R., number of leucocytes and haemoglobin content were described as normal.

Check radiography 4 1/2 months after onset of symptoms showed that the process was then in the beginning of the healing stage. Extension of the joint was still defective but the patient was otherwise free from clinical symptoms.

Roentgen examination of the other elbow showed nothing remarkable.

When last seen 2 years and 9 months after the onset of symptoms the pa-

tient had made a complete recovery. The roentgenogram showed that the process had healed. The outline and structure of the ossific nucleus of the capitulum humeri was, however, still somewhat irregular and it was somewhat larger than on the other side. Nothing is known of the definite stage in this case, because the patient was still growing on that occasion.

Recapitulation

1. Perusal of the literature of the last 54 years revealed only 19 publications describing certain cases of Osteochondrosis deformans juvenilis of the centre of ossification of the epiphysis of the capitulum humeri.

2. These publications contain altogether 30 cases of the disease.

3. No single author had described more than 4 cases, and most of them only 1.

4. Most of the cases on record were followed up for only a short time, only 2 cases for more than 3 years.

2. Frequency

The number of certain cases of O.d.j. of the capitulum humeri on record is low compared with that of the disease in other locations.

In the present investigation the diagnosis of O.d.j. of the capitulum humeri was said to be certain only if the roentgenogram was pathognomonic with involvement of the entire ossific nucleus of the growing capitulum and relatively characteristic local symptoms in otherwise healthy patients. Some of the cases described under the heading of O.d.j. of the capitulum humeri do not satisfy these requirements or have not been described in sufficient detail to be included in this survey. This applies to the cases described by AMSTAD (1916), WILD (1921), CAAN (1924), SMITH (1929), WINTERSTEIN (1933), UHRMACHER (1933), SCHAEFER, STRICKROOT & PURCELL (1939), MAROTTOLI (1944) and HERMODSSON (1947).

The cases satisfying these requirements are given in Table 4.

It is clear from the table that most of these publications were based on a single case. Only 4 described 2 cases and only 2 contributed with 3 cases each.

The largest series published is that of HEGEMANN (1951), who reported 4 cases, but gave roentgenograms of only 1 of them. He stated that he found these 4 cases in a total material of "1200 Röntgenaufnahmen des Ellenbogensgelenkes", of an undefined number of patients who had sought medical advice because of elbow trouble. Since he does

TABLE 4

Frequency of O.d.j. capituli humeri according to literature

No.	Author	Year of publication	Number of cases
1	PANNER, H. J.	1927, 1929	3
2	KREBS, C.	1927	1
3	BUSCH, E.	1930	1
4	HAGEN, J. F. and BISSCHOP, P.	1933	1
5	KING, E. S. J.	1935	3
6	NIJST, E. E.	1937	1
7	MOL, W.	1939	1
8	RIVAROLA, J. E.	1939	1
9	ELWARD, J. F.	1939	2
10	KOHLBACH, W.	1940	2
11	MARCH, H. C.	1944	1
12	HEGEMANN, G.	1951	4
13	CAMERA, R.	1951	1
14	SEMMELROCH, H.	1952	1
15	BUETTI, C.	1953	2
16	MASON, M. L.	1953	1
17	JUD, H.	1953	1
18	LANGE, J.	1954	1
19	LAURENT, L. E. and LINDSTRÖM, B. L.	1956	2
Total number of cases			30

not give the number of patients in his material, no conclusions can be drawn from his publication as to the frequency of the disease.

Most authors of cases of this disease, however, agree that it is rarely seen in the capitulum humeri.

Some authors such as PANNER (1929, 1930), BUSCH (1930), MARCH (1944), JUD (1953) and LAURENT & LINDSTRÖM (1956) have, however, suggested that the disease in this location might not be so uncommon as widely believed but that it might often escape notice because of the mildness of the symptoms it produces.

PANNER (1929, 1930) pointed out that the initial stage of the disease is readily missed in the roentgenogram and BUETTI (1953) thought that the scantiness of reports of the literature might be due to the fact that its roentgenographic appearance is easily overlooked in its early stages and wrongly interpreted in its later stages.

Judging by the literature, however, one can hardly escape the impression that O.d.j. of the capitulum humeri is a rare disease.

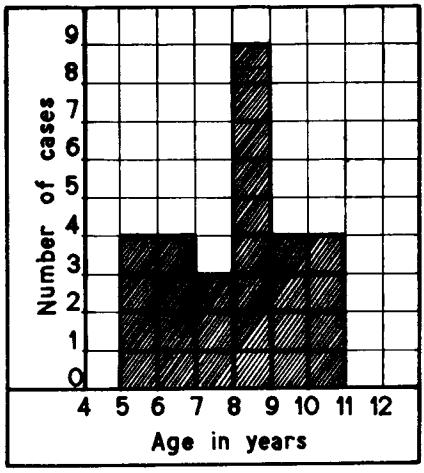


Fig. 6. Age at first examination. 28 cases from literature.

3. Age at time of first examination

Of the 30 cases on record no mention is made of the patient's ages in 2 (Fig. 6).

It is clear from Fig. 6 that the patient's ages ranged between 5 and 10 years at the time of the first examination.

The mean age was 7.6 years.

The material is too small to permit any valid conclusion on the frequency of onset in the various age-groups.

4. Sex incidence

It appears that O.d.j. of the capitulum humeri has been seen mainly in boys.

It is clear from Fig. 7 that only 3 cases have been reported in girls, which represents 10 per cent of the cases described in the literature.

As in O.d.j. in other locations such as in the femoral head, the disease was thus more common in boys.

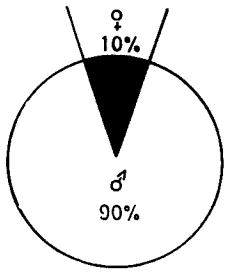


Fig. 7. Sex incidence. 30 cases from literature.

5. Side involved

Of the 30 cases on record the contralateral elbow was examined roentgenologically in 19. In the remaining 11 nothing is said as to whether the other side was examined roentgenologically or not.

In 16 of these 30 patients the disease was described as localised to the right capitulum humeri.

In 6 patients it occurred on the left side.

In 2 the disease was described as bilateral.

In 6 it is not said which side was affected.

Although the material is small, it appears that the disease is usually unilateral and more common on the right side than on the left.

6. General survey of clinical picture in cases on record

Local symptoms

All the 30 patients described in the literature had clinical symptoms of the elbow affected.

In 2 of them (BUETTI 1953, LAURENT & LINDSTRÖM 1956) check radiography showed roentgenologic changes in the contra-lateral clinically symptom-free capitulum humeri, which were interpreted as asymptomatic O.d.j. of the capitulum.

The symptoms in the cases on record are summarised in Table 5.

Table 6 also gives a detailed survey of the limitation of movements adduced from incomplete data.

It is clear from Table 5 that pain in the elbow involved was the commonest subjective symptom, and described in 29 of the 30 patients.

The pain was described as spontaneous or on movement of the elbow or both.

Tenderness to palpation was described in 12 cases.

In one case (KREBS 1927) trembling of the hand on exertion was described.

Of the objective, local symptoms, impairment of mobility was the commonest, it was reported in 29 cases.

The movement most commonly impaired was extension (Fig. 6), which was noted in 18 cases. Impaired flexion was also relatively common. Pronation-supination, when mentioned, was usually described as normal.

In 21 of the cases the elbow was described as swollen.

In one of the cases (JUD 1953) crepitations were noted over the radial part of the joint on pronation-supination.

TABLE 5

Localised clinical symptoms in cases from literature

Authors	Case No.	Subjective symptoms		Objective symptoms	
		Pain	Tenderness	Swelling	Limitation of movement
PANNER, H. J.	1	+	+	+	+
	2	+	+	+	+
	3	+	+	+	+
KREBS, C.	4	+	None	+	+
BUSCH, E.	5	+	None	+	+
HAGEN, J. F. et al.	6	+		None	+
KING, E. S. J.	7	+	+	+	+
	8	+	+	+	+
	9	+	+	+	+
NIJST, E. E.	10	+	None	None	+
MOL, W.	11	+	+	+	+
RIVAROLA, J. E.	12	+	None	+	+
ELWARD, J. F.	13	+			+
	14	+			+
KOHLBACH, W.	15	+			+
	16	+			+
MARCH, H. C.	17	+		+	+
HEGEMANN, G.	18	+		+	+
	19	+		+	+
	20	+		+	+
	21	+		+	+
CAMERA, R.	22	+	+	+	+
SEMMELROCH, H.	23	None	None	None	+
BUETTI, C.	24	+		+	
(Bilateral case)	25, a	+		+	+
	25, b	None	None	None	None
MASON, M. L.	26	+	None	None	+
JUD, H.	27	+	+	+	+
LANGE, J.	28	+	+		+
(Bilateral case)	29, a	+	+	+	+
	29, b	None	None	None	None
LAURENT, L. E. et al.	30	+	+	+	+

TABLE 6

Limitation of movement in elbow in cases from literature

Author	Case No.	Extension	Flexion	Pronation	Supination
PANNER, H. J.	1	Limitation	Normal	Normal	Normal
	2	„	„	„	„
	3	„	Limitation	„	„
KREBS, C.	4	Limitation	Limitation	Normal	Normal
BUSCH, E.	5	Limitation	Limitation	Normal	Normal
HAGEN, J. F. et al.	6	Limitation	Limitation	Normal	Normal
KING, E. S. J.	7				
	8				
	9				
NIJST, E. E.	10	Limitation	Limitation	Limitation	Limitation
MOL, W.	11	Limitation	Limitation		
RIVAROLA, J. E.	12	Limitation	Limitation		Limitation
ELWARD, J. F.	13				
	14				
KOHLBACH, W.	15	Limitation	Limitation		
	16	„	„		
MARCH, H. C.	17	Limitation	Limitation	Normal	Normal
HEGEMANN, G.	18	Limitation			
	19				
	20				
	21				
CAMERA, R.	22				
SEMMELROCH, H.	23	Limitation			
BUETTI, C.	24				
(Bilateral case)	25, a				
	25, b	Normal	Normal	Normal	Normal
MASON, M. L.	26	Normal	Limitation	Normal	Normal
JUD, H.	27	Limitation	Limitation	Normal	Limitation
LANGE, J.	28	Limitation	Limitation	Normal	Normal
(Bilateral case)	29, a	Limitation	—	—	—
	29, b	Normal	Normal	Normal	Normal
LAURENT, L. E. et al.	30	Limitation	—	—	—

In 2 cases a cubitus valgus deformity of the elbow was noted (MOL 1939, JUD 1953).

Finally, it might be mentioned that PANNER (1929, 1930) reported slight wasting of the musculature of the affected arm in 2 of his cases.

General symptoms

All cases on record occurred in otherwise healthy children. The patients were all in a good general condition without definite signs of constitutional anomalies, endocrine dysfunction or retarded development. No familial occurrence or hereditary factors have been described. Fever was not reported in any cases, but in 2 the E.S.R. was raised (LAURENT & LINDSTRÖM 1956). The blood picture and other laboratory tests, when performed, in the other cases showed nothing remarkable.

In 1 of the cases (SEMMELROCH 1952) irregular roentgenologic outline and structure of the ossification centre of one of the patellae with clear clinical local symptoms was described. SEMMELROCH is the only author who described a co-existent disorder in any ossific nucleus other than that of the capitulum humeri.

TO SUM UP, it may be said that judging by the literature, O.d.j. of the capitulum humeri produces only local symptoms.

The commonest clinical picture appears to be painful unilateral impairment of extension, sometimes also flexion, in the affected elbow, as well as local swelling and sometimes tenderness to palpation over the region of the elbow.

Pronation-supination is usually described as normal.

PANNER (1929, 1930) and MASON (1953) stressed that the clinical symptoms need not be proportional to the roentgenographic changes.

7. General survey of roentgenologic appearance in cases on record

Reproductions of roentgenograms taken in 25 of the cases in the literature closely resemble one another.

In those cases, in which the pathologic process was followed roentgenologically from what was regarded as an early stage, the first roentgenograms show only slight deviations from normal: only a more or less uneven contour of the ossific nucleus of the capitulum humeri together with some insignificant structural alterations with a juxtachondral zone of rarefaction. Other roentgenologic changes may be missing, so that PANNER (1929, 1930) and BUETTI (1953) stressed the risk of mis-interpretation of the roentgenogram at this stage, *i.e.* the risk of interpreta-

tion as a simple fissure in the ossific nucleus. PANNER (1929, 1930), KREBS (1927), NIJST (1937) and JUD (1953) also found an increased roentgenologic distance between the ossific nuclei of the capitulum humeri and the capitulum radii in relation to the other healthy side.

Roentgenographic follow-up will show that the ossific nucleus decreases in size and that its contour becomes deeply indented peripherally. At the same time the normally spongy structure of the ossification centre is replaced by diffuse dense areas alternating with irregular rarefactions. These changes increase in intensity so that the ossific nucleus appears to be fragmented. JUD (1953) also found a rarefaction in the humeral metaphysis. He is the only author who described coexistent roentgenologic changes in this part of the bone.

After a certain time, however, the pathologic process in the ossific nucleus abates and its size, contour and structure assume a normal appearance. In none of the cases in the literature, which have been followed up until healed have any severe consequent deformities of the capitulum humeri been observed.

8. Duration of clinical symptoms in cases on record

As early as 1927 PANNER stressed that the initial clinical symptoms disappear quicker than the roentgenologic abnormality of the ossific nucleus.

In most of the reports available the course of the disease has not been followed long enough or described in sufficient detail to permit any conclusions on the duration of clinical symptoms.

Some authors (PANNER 1929, 1930), BUSCH (1930), ELWARD (1939), however, stated that the most acute local symptoms soon regressed after resting of the affected elbow.

Some symptoms, such as impaired mobility, however, appear to have persisted longer.

Table 7 gives a survey of the duration of clinical symptoms reported in 10 literature cases all of which are described in sufficient detail to permit such evaluation.

It is clear from Table 7 that the duration of clinical symptoms varied between 1 year, 2 1/2 months (case No. 22) and 3 years (case No. 1 and 15).

The smallness of the material, however, reduces the value of these figures.

TABLE 7

Interval between appearence of clinical symptoms and clinical recovery
10 cases from literature

Author	Case No.	Interval	Remarks
PANNER, H. J.	1	3 years	"Barest restriction of extension"
	2	1 year, 5 months	"Trifling restriction of extension. Slight sensation of pain when unduly taxed in lifting or carrying"
KREBS, C.	4	1 year, 6 months	Slight restriction of flexion
RIVAROLA, J. E.	12	1 year, 3 months	
KOHLBACH, W.	15	3 years	
CAMERA, R.	22	1 year, 2 1/2 months	
SEMMELROCH, H.	23	1 year, 8 months	
JUD, H.	27	1 year, 9 months	Slight restriction of hyperextension
LANGE, J.	28	2 years	
LAURENT, L. E. et al.	30	2 years, 9 months	

9. Duration of roentgenologic signs of pathologic process

It is difficult to draw any conclusions on the duration of the pathologic process from the reports on record. It is true that roentgen examination will show roughly when the ossific nucleus recovers more or less normal appearance, which may be regarded as evidence that the process has healed.

A factor making it difficult to judge the duration of the process, however, is that it is not known with certainty when it begins in the ossification centre. Only in one of the literature cases were roentgenograms available of the ossific nucleus before the appearance of roentgenographic changes in the capitulum humeri. In that case the author (JUD 1953) believed that the process had started within a period of 8 months before it had produced any symptoms.

Most authors agree that the pathologic picture seen in the roentgeno-

grams on first examination does not appear to be that of a recent change. On the other hand, it is difficult to estimate with anything like certainty the age of a given aseptic bone necrosis. BRAILSFORD (1943), STÅHL (1948) and GOFF (1954) tried to determine the relationship between the roentgenographic appearance of the pathologic process and its duration in cases of O.d.j. of the femoral head, but the literature contains no data in this respect on the much smaller ossific nucleus of the capitulum humeri.

Therefore, the disease can as yet only be dated from the onset of symptoms.

In 2 of the literature cases, however, the duration of the symptoms before roentgen examination is so short that the authors (KOHLBACH

TABLE 8

Interval between onset of symptoms and initial as well as almost complete roentgenologic recovery

13 cases from literature

Author	Case No.	From onset of symptoms till roentgenologic signs of initial repair	From onset of symptoms till roentgenologic recovery
PANNER, H. J.	1	8 months	3 years
	2	5 months	1 year, 5 months
	3	1 year, 1 month	
KREBS, C.	4	1 year, 6 months	
NIJST, E. E.	10	9 1/2 months	
RIVARÒLA, J. E.	12	5 months	
KOHLBACH, W.	15		3 years
HEGEMANN, G.	18	5 months	
CAMERA, R.	22	1 year, 2 1/2 months	
SEMMELROCH, H.	23	1 year, 8 months	
JUD, H.	27	9 1/2 months	1 year, 9 months
LANGE, J.	28	6 months	10 months
LAURENT, L. E. et al.	30	4 1/2 months	2 years, 9 months

1940 and BUETTI 1953) suggested that the onset of the process must have antedated the onset of the symptoms.

In the other cases, in which it was possible to determine the interval between onset of symptoms and first roentgen examination, the interval reported varied between 2 weeks and 6 months. But even in these cases the onset of the pathologic process might have antedated the beginning of the symptoms, so that all pertinent figures must be regarded as minimum values.

On the other hand, it appears justified to assume that the pathologic process in the ossific nucleus was present at the onset of clinical symptoms. In Table 8 a survey is given of the interval between the onset of clinical symptoms and roentgenologic signs of healing, provided that sufficient data are given to permit evaluation of this interval.

It is clear from Table 8 that signs of roentgenologic healing appeared between $4^1/_2$ months and 1 year, 8 months after the onset of the clinical symptoms. This interval corresponds to WALDENSTRÖM's (1941) evolutionary period of coxa plana, which he gives as 2—4 years for the much larger ossific nucleus of the femoral head.

As is apparent from Table 8, the healing process was described as being complete 10 months, to 3 years after the onset of symptoms.

With reference to Table 8, the healing period should last between about 4 months and more than 2 years. WALDENSTRÖM (1941) estimates the healing period of coxa plana at 1—2 years.

Since only 13 literature cases (Table 8) are available for evaluation, the above mentioned data on the duration of the pathologic process in the capitulum humeri is of limited value.

10. Etiology and pathogenesis

Relation to previous trauma

Table 9 summarises data given on any previous known trauma. It also gives the interval between the trauma and the onset of local symptoms as well as the interval between trauma and the first roentgenologic examination.

It is clear from Table 9 that preceding trauma was described as known in 12 of 27 cases. In 8 of these 12 cases local symptoms appeared immediately after the trauma.

In 2 (Nos. 16 and 25, a) roentgen examination 3 and 1 day, respectively, after the trauma showed changes in the ossification centre of the

TABLE 9

Relation to previous trauma in cases from literature

Author	Case No.	Known previous trauma	Interval between trauma and appearance of symptoms	Interval between trauma and first roentgenologic examination
PANNER, H. J.	1	+	None	2 weeks
	2	+	„	3 months
	3	+	„	1 1/2 month
KREBS, C.	4	+	2 years	2 1/2 years
BUSCH, E.	5	None		
HAGEN, J. F. et al.	6	None		
KING, E. S. J.	7			
	8			
	9			
NIJST, E. E.	10	None		
MOL, W.	11	+	None	6 months
RIVAROLA, J. E.	12	+		
ELWARD, J. F.	13	None		
	14	+	None	"Several days"
KOHLBACH, W.	15	None		
	16	+	None	3 days
MARCH, H. C.	17	+	None	3 weeks
HEGEMANN, G.	18	None		
	19	„		
	20	„		
	21	„		
CAMERA, R.	22	None		
SEMMELROCH, H.	23	+		
BUETTI, C.	24	None		
(Bilateral case)	25, a	+	None	1 day
	25, b	None		
MASON, M. L.	26	None		
JUD, H.	27	+	8 months	None
LANGE, J.	28	None		
(Bilateral case)	29, a	None		
	29, b	„		
LAURENT, L. E. et al.	30	„		

capitulum humeri, that were described as being of less recent date and could thus not be ascribed only to the trauma.

In the other 10 cases with known trauma the authors considered that it was not possible to exclude the possibility of the pathologic changes in the ossification centre being due to the trauma.

As mentioned in chapter I, there seems to be reasonable roentgenologic and clinical evidence to assume that the process in the epiphysis of the capitulum humeri and the classical location of O.d.j. in the epiphysis of the femoral head are two local manifestations of one and the same pathologic process. There is wide agreement that the primary pathologic process in O.d.j. is aseptic necrosis of the spongy bone in the growing ossification centre. This osseous necrosis decreases the natural resistance of the bony tissue to strain. The result is a pathologic compression fracture within the dead bone. The cartilage, on the other hand, surrounding the necrotising ossification centre in large remains alive, as has been pointed out by HEITZMANN & ENGEL (1923), NUSSBAUM (1924), ROCKEMER (1927), LÖHR (1931), PHEMISTER (1940), HAYTHORN (1949) and others. JONSÄTER (1953), however, found cartilaginous changes of a degenerative nature localised to the basal layer of the joint cartilage in cases of O.d.j. of the femoral head.

It might be mentioned that BERNBECK (1950) suggested that secondary, chemical reactions between acid medium (chondroitinsulphuric acid) in the cartilaginous epiphysis and alcaline medium (calcium salts) in the ossific nucleus might play a rôle in the pathogenesis of O.d.j. ("heterolytische Dyskrasie").

The further course of the pathologic process is widely believed to consist of absorption of sequestrated osseous tissue by ingrowth of granulation tissue with osteoclasts. This absorption is accompanied by regeneration with new-formation of bone by osteid tissue and possibly by transformation of mesenchymal tissue to cartilage and bone. These changes, called creeping substitution by PHEMISTER (1935), result in recovery of the normal structure and strength of the spongy bone. Sometimes, the end result may be deformation of the affected part of the bone, which may be of great significance, if the ossification centre in question contributes to the ossification of a part of a joint.

The observations set forth above must be considered as the present predominant conception of the pathologic anatomy of O.d.j. In recent years, some authors have, however, expressed views differing funda-

mentally from that above. These views will be discussed later in this section.

Thus, since the pioneer work of AXHAUSEN (1923) there has been increasing agreement on the pathologic anatomy of O.d.j. On the other hand, ever since the investigations of O.d.j. of the epiphysis of the femoral head by WALDENSTRÖM (1909), LEGG (1909), CALVÉ (1910) and PERTHES (1910) no satisfactory explanation of the primary cause of the disease has become available.

In the course of years so many theories have been put forward on the etiology of the condition that there appears to be hardly any etiologic factors that have not been discussed.

In the discussion of the etiology of the condition the lesion in the epiphysis of the femoral head has received most attention, because of the often serious sequelae.

Most of the relatively few observations published on O.d.j. of the epiphysis of the capitulum humeri are based on single case reports. The largest material on record consisted of 4 cases. No histologic investigations have been published on the disease in the capitulum humeri. In many of the cases the course of the disease was not followed. It is therefore but natural that these authors could not shed new light on the obscure question of the causal factor of O.d.j. in general and of O.d.j. of the capitulum humeri in particular. In their discussion of the etiology of O.d.j. of the capitulum humeri, these authors adhere to the earlier theories of the cause of O.d.j., especially O.d.j. of the femoral head.

What has been said of the etiology of O.d.j. in general thus holds broadly for that of O.d.j. in capitulum humeri. In other words, no satisfactory explanation is available.

The theories on the etiology of O.d.j. in the capitulum humeri and in other locations, especially in the femoral head, are thus interwoven to such an extent that they are inseparable and must therefore be described under a common heading.

For perspicuity the various theories are grouped as far as possible on descriptive grounds.

The main theories of today fall into the following 3 classes:

I. Disturbed circulation of the ossification centre with necrosis of the centre.

II. Primary fracture of spongy bone tissue in ossification centre.

III. Endogenous factors (congenital, acquired, constitutional, inherited).

I. *Disturbed circulation of the ossification centre*

This theory is based mainly on the histologic and roentgenologic changes, which suggest that the primary pathologic factor consists of aseptic necrosis. All other histologic and roentgenologic changes are thus secondary to this necrosis.

Only two causes of bone necrosis are known (LANG 1941, SCHINZ et al. 1950):

I) Direct necrosis due to physical or toxic-actinic factors.

II) Indirect necrosis due to interruption of the blood supply.

Since, according to adherents of the "vascular theory", the first possibility can be excluded in this respect, there remains but one possible cause, namely disturbance of the blood supply to the ossification centre.

This theory was first put forward by LEGG (1910), PERTHES (1913) and SCHWARZ (1914), as a possible factor in the etiology of O.d.j. of the femoral head. Since then many authors discussing O.d.j. in general have accepted this theory for the etiology of O.d.j. independently of the location (ALLISON & MOODY 1915, NUSSBAUM 1924, OVERTON 1935, RYERSON 1940, LANG 1941, PEASE 1950, BERNBECK 1950, BENNET 1952, ADAMS 1956).

In further support of this theory it has been stressed that O.d.j. often occurs in those parts of growing bones, in which the blood supply is most vulnerable (CHANDLER 1940, LANG 1941, FANCONI 1952).

BENTZON (1926, 1927), MAU (1929) and BERNBECK (1950) stressed the fact that the vessels of an ossific nucleus must traverse the surrounding cartilage on their way to the endochondral ossification centre. They believe that it is this intracartilaginous part of the vessels that is so vulnerable, since compression or swelling of the cartilage in a weight-bearing area can lead to mechanical traumatisation of these intracartilaginous vessels.

Several authors have performed experiments to support the vascular theory.

NUSSBAUM's experimental investigations (1923, 1924, 1926) may be regarded as pioneer work in this field. He interrupted the blood supply to growing ossification centres with total necrosis of the ossific nucleus as a result, while the surrounding cartilage survived. He thus produced the same histologic picture as that seen in O.d.j.

Similar experiments have been carried out by other authors such as MÜLLER 1924, BENTZON 1926, MAU 1929, MILTNER & HU 1933, LERICHE

1934, KISTLER 1936, RANDLØV-MADSEN 1950 and LEMOINE 1957, all of whom claimed to have produced changes of the type seen in O.d.j. Other workers, on the other hand, such as STEWART (1933), BURROWS (1941) and LACROIX (1942) stated that although the changes observed in their experiments in the growing epiphysis resembled those in O.d.j., they were not identical.

Though adherents of the vascular theory agree that O.d.j. is due to circulatory disturbances, there is no general agreement as to the cause of these disturbances. The most important opinions of this primary cause are given in the following section.

a. *Mechanical traumatisation of the vessels with disturbance of the circulation as a consequence*

The first to assume that various types of trauma might be capable of producing circulatory disturbances with such bone necrosis as a consequence were LEGG (1910), PERTHES (1913) and SCHWARZ (1914), a view later shared by many others (AMSTAD 1916, SUNDT 1920, WIDERÖE 1921, ZAAIJER 1921, SCHOONEVELT 1921, ASCHOFF 1923, PAYR 1924, ROESNER & WEIL 1925, BENTZON 1926, ROCKEMER 1927, MURK-JANSEN 1929, MAU 1929, NAGASAKA 1930, BRAILSFORD 1935, NOVOTNY 1937, DOUB 1945, BERNBECK 1950).

In support of this theory it has been claimed that aseptic total necrosis of ossific nuclei has been described to develop after well-defined trauma during the period of growth. Changes gradually developed in the ossification centre which immediately after traumatisation show no roentgenologic signs of primary lesion. Thus ELMSLIE (1919), REHBEIN (1922), NICOLAYSEN (1931), BLUMENSAAT (1936), GOLDENBERG (1938) and QUIST-HANSEN (1945) have described necrosis of the ossific nucleus of the femoral head and JUD (1953) (vide infra) of the ossific nucleus of the capitulum humeri after traumatic dislocation of these joints in children.

Such necrosis has been reported by JOHANSSON (1927), LANGE (1932), NERAA (1938), NIELSEN (1938) and BORNEBUSCH (1940) in the ossific nucleus of the femoral head after fracture of the femoral neck in children, and HERMODSSON (1936) in the scaphoid bone nucleus of the foot after trauma to the ankle. In 1947 HERMODSSON (vide infra) also described aseptic necrosis in the ossific nuclei of the capitulum humeri and capitulum radii after ulnar fracture in a boy. In 1955 DE CUVELAND

& OTTE reported aseptic epiphyseal necrosis in the capitulum radii diagnosed 10 months after trauma.

The above mentioned cases have been regarded as arguing against the theory of primary fracture as an etiologic factor and for the theory of vascular trauma.

In this connection it might also be mentioned that roentgenograms showing O.d.j.-like changes in the femoral head after reduced congenital dislocation of the hip (LEGG 1916, MAYR 1934, FERGUSON & HOWORTH 1934, MEYER 1941, MASSIE 1951, IDELBERGER 1951 and others) have sometimes been referred to as evidence for the vascular theory. Some authors, such as HEITZMANN & ENGEL (1923), ENGELMANN (1924), BRAILSFORD (1943), STÅHL (1948) and GOFF (1954), deny that these changes are identical with O.d.j. Moreover, this will not explain similar changes on the other non-dislocated side (BRANDES 1920, SPITZY 1924, BRAILSFORD 1943 and others).

Some authors believe vascular trauma to play a rôle in the etiology of O.d.j. of the capitulum humeri.

AMSTAD (1916) referred to a case reported by ISELIN of "Mb Perthes"-like changes in the eminentia capitata of the distal end of the humerus in a child. He claimed that during the period of growth the ossification centre of the capitulum humeri in the same way as the ossification centre of the femoral head, is dependent for its blood supply on vessels from "der Epiphysengrenze", and that epiphyseal disturbances in the capitulum humeri might thus be of the same origin as O.d.j. of the femoral head, namely "dauernde Ernährungsstörung" of the epiphysis, which may in turn, be due to trauma.

In papers on epiphyseal disturbances around the elbow and osteochondritis dissecans capituli humeri LÖHR (1930, 1931) described his investigations of the circulation in the epiphysis of the capitulum humeri. On the basis of these investigations he concluded that during the period of growth mechanical traumatisation of vessels coming from behind and running to the vascularly isolated ossification centre of the capitulum humeri, can disturb the circulation in the entire centre with total necrosis as a consequence. LÖHR believes that this "Gefässdrosselung" involves mainly the venous circulation and can be caused by non-physiologic strain of the elbow, especially in individuals with poorly developed muscles. He believes that these vessels may be throttled by distension of a delicate joint capsule and ligaments. He also believes that the tendency to subluxation of capitulum radii in children may

play a contributory rôle in the development of this condition. That such epiphyseal disturbances in the capitulum humeri are of vascular origin and not due to direct traumatisation of the ossific nucleus is, according to this author, supported by his investigation of 40 young patients, who had sustained supracondylar and intraarticular fractures of the elbow joint during the period of growth. In none of them were any O.d.j.-like disturbances of the epiphyses observed, not even when the fracture line has passed through the ossification centre.

UHRMACHER (1933) more or less shared LÖHR's opinion of the primary cause of O.d.j. of the ossification centres in the distal end of the humerus. He claimed that non-physiologic strain of the elbow in children can strangle or throttle the epiphyseal vessels and disturb the circulation in the ossification centres. This would decrease the vitality in rapidly growing bone cells in the epiphyseal centre.

LANG (1941) shares LÖHR's opinion regarding the traumatic-mechanical disturbance of the circulation as a factor in the cause of disorders in the ossification centre of the capitulum humeri, but while LÖHR emphasises the disorder of the venous circulation in the epiphysis, a thought, suggested earlier by ROESNER & WEIL (1925) and ROCKEMER (1927) and recently by HULTH (1958), LANG is inclined to believe the disturbance of the arterial circulation to be of major importance. According to him, differences in the topography of the vessels explain the difference in frequency in O.d.j. in different joint epiphyses. He thus propounds that because a portion of the laterodorsal part of the distal epiphysis of the humerus is extracapsular, the ossification centre of the capitulum humeri will not be completely shut off from its blood supply, not even on wide-spread interruption of the circulation in the capsular attachment on the humerus. According to LANG, the reason why this ossific nucleus can survive such a vasculatory disorder is, that a part of the epiphyseal plate is extracapsular (Fig. 5), so that "aus dem metaphysären Periost immer noch reichlich Gefässzweige in die Epiphyse gelangen".

In view of these anatomic considerations, LANG states that "kein dem ‚Perthes' voll entsprechendes Krankheitsbild" can occur in the distal end of the humerus. He does not, however, support his assertion on investigations of the circulation in the distal end of the humerus. Moreover, the 16 cases with changes resembling those seen in Perthes' disease located in the growing capitulum humeri and described in the literature at that time had escaped the attention of LANG.

HERMODSSON (1947) described "aseptic osteonecrosis" in the ossification centre of the capitulum humeri, 8 1/2 months after fracture of the ulna of the same arm in a 13-year old boy. He underlined that this case was of great principal interest, because immediately after trauma, the roentgenographic appearance of the ossification centre in question was normal, but 8 1/2 months later it showed signs of partial, circumscript aseptic necrosis. HERMODSSON expressed the view that the primary cause of this aseptic bone necrosis was impairment of the blood supply to the ossification centre owing to trauma. As mentioned, it cannot be concluded that the patient in HERMODSSON's case really had O.d.j. of the capitulum humeri.

JUD's (1953) patient, on the other hand, definitely had O.d.j. of the capitulum humeri. His patient had a history resembling that of the patient described by HERMODSSON.

JUD's patient, a 7-year old boy, had roentgenologic and clinical, typical O.d.j. of the capitulum humeri 8 months after posterior cubital luxation. The luxation was reduced in the usual way one hour after trauma and roentgen examination showed the capitulum humeri to be of normal appearance. The trauma that had caused the dislocation had thus not produced any roentgenologically demonstrable signs of a primary lesion in the spongy structure of the ossific nucleus of the capitulum humeri.

JUD considered that there was a distinct causal relationship between the luxation and the later bone necrosis. He stressed the similarity with O.d.j. of the femoral head occurring after traumatic dislocation of the hip in children.

He suggested that the vascular system nourishing the ossification centre of the capitulum humeri during the period of rapid growth is at most sufficient to meet the requirements of the growing bone. Interruption of this blood supply during this period might therefore impair growth of the ossification centre and even cause gradual necrosis.

JUD, however, does not support his hypothesis on personal investigations of the circulation of the ossification centre of the capitulum humeri, but refers to LÖHR's (1930) publication on the vascularisation of this centre. In his article, which is based partly on injection experiments, LÖHR, however, showed, as will later be discussed, no illustrations of the intra-osseous vascular anatomy of the ossification centre of the capitulum humeri, so that his conclusions must be regarded as hypothetic.

JUD is an adherent of the theory of traumatic vascular lesion. According to him, the dislocation in the case described by him produced loss of continuity or tension in the vascularized soft tissues in the region of the elbow and the consequent disturbance of the circulation in the main vessels supplying the capitulum humeri then resulted in total ischaemic necrosis of the ossification centre.

In this connection it must, however, be mentioned that judging by a search of the literature of the last 54 years, JUD is the only author, who has described O.d.j. of the capitulum humeri after dislocation of the elbow. Dislocation of the elbow is relatively common in children. If the above method of development represented the entire explanation of epiphyseal necrosis in this case, O.d.j. of the capitulum should probably be more common than it is. In view of this argument, JUD supplements his hypothesis by the idea of pre-existing inferiority of the epiphysis suggested as early as 1921 by CALVÉ.

It is clear that these publications on single cases permit no valid conclusions on the rôle of vascular trauma in the etiology of O.d.j. of the capitulum humeri.

On the other hand, much suggests that O.d.j. of the femoral head has been caused by previous traumatic dislocation of the hip joint and femoral neck fracture in children.

In an earlier section both the clinical and roentgenologic similarity between O.d.j. of the capitulum humeri and of the femoral head was stressed and it was considered justified to assume that both diseases were different local manifestations of one and the same pathologic process.

It is therefore hardly possible to reject the assumption that vascular trauma may be able to disturb the blood supply to the growing ossification centre of the capitulum humeri with O.d.j. as a consequence.

The descriptions of the vascular anatomy of the growing capitulum humeri found in the literature are not based on satisfactory evidence. Since the theory on the rôle played by traumatisation of the circulation in the etiology of O.d.j. of the capitulum humeri (and in other locations) is founded mainly on knowledge of the vascular anatomy of the ossification centre, further knowledge of the underlying mechanism of the disease can hardly be expected until we have charted the vascular anatomy of the ossification centre of the capitulum humeri, the intraosseous and intracartilaginous as well as extraosseous. It was with this in mind

that this vascularity was studied experimentally by the author as will be described in chapter IV.

It is clear that, if the vascular theory be accepted, the disorder may also be produced by many factors other than traumatisation of the vessels discussed above. This has resulted in the elaboration of other theories on the cause of the vascular disturbances in the etiology of O.d.j.

b. *Embolism as a cause of the circulatory disorder*

The theory of an aseptic embolism of unknown origin being the cause of circulatory disorders in O.d.j. was propounded by AXHAUSEN (1923). In later publications (1926, 1954) he tried to prove that his theory explains the primary cause of O.d.j. in different locations. This theory has since been embraced to a varying degree by *inter alia* WOLLENBERG (1928) and COLLINS (1949). The latter, however, believes in the possibility of other etiologic factors, too.

REICH & ROSENBERG (1953) described changes resembling O.d.j. in epiphyses in children with chronic sickle cell anaemia. They ascribed these epiphyseal changes to embolism due to the blood disease.

But it has never been possible to demonstrate vascular occlusion due to embolism in typical O.d.j. in any location.

The experimental investigations carried out by WOLLENBERG (1928) and others to support this theory gave no convincing result. BERGMANN (1927) and KAHLSTROM, BURTON & PHEMISTER (1939) believed the results of their experiments to argue against the theory of embolism, a theory which has but few adherents.

None of the authors, who have described O.d.j. of the capitulum humeri, have accepted this theory of the etiology of the condition.

c. *Endarteritis obliterans as a cause of the circulatory disorder*

Endarteritic vascular changes have been described in O.d.j. HOLST & CHANDRIKOFF (1927), KONJETZNY (1934) and others have described such changes and believe that they are responsible for the circulatory disorder. BELMONTE (1929) speaks of physiologic endarteritis obliterans.

It should, however, be emphasised, that it has not yet been possible to prove whether such endarteritic changes were primary or secondary in the course of the disease.

Thus, no convincing evidence of the correctness of this theory is available.

None of the authors who have studied O.d.j. of the capitulum humeri believe endarteritis obliterans to be the causal agent.

d. *Neurogenic causes of the circulatory disorder*

Trophoneurotic and vasomotor disorders have been suggested as causes of O.d.j. (LANDWEHR 1923, BENTZON 1926, HARBIN & ZOLLINGER 1930, SORREL & BUFNOIR 1931, HIRSCH 1933). This opinion must be regarded as speculative, because no local nervous injury has ever been demonstrated with certainty in O.d.j.

No author has described neurogenic O.d.j. of the capitulum humeri.

e. *Local infection*

WALDENSTRÖM (1909) and PERTHES (1910) first believed infection to be the cause of O.d.j. of the femoral head but afterwards revised their opinion.

FERGUSON & HOWORTH (1934) and HOWORTH (1948, 1952) believed that subacute arthritis might cause congestion and sclerosis of the vascularised soft tissues with O.d.j. as a consequence. HULTH (1958) shared this opinion.

Some other authors have also believed infection to be of etiologic importance in O.d.j. (PLATT 1922, HEITZMANN & ENGEL 1923, KIDNER & MURO 1924, BUCHMAN 1925, PHEMISTER, BRUNSCHWIG & DAY 1930 and BORCHARD 1932).

SMITH (1929) described the roentgenographic appearance of the ossification centre of the capitulum humeri in a 4-year old boy. He expressed the view that it was a case of O.d.j., and that it was due to local infection of the growing epiphysis, whose power of resistance might have been reduced by previous trauma. However, he produced no evidence in support of this hypothesis. As mentioned, SMITH's case showed no certain clinical or roentgenologic evidence of O.d.j. capituli humeri.

ELWARD (1939), who described 2 typical cases of O.d.j. of the capitulum humeri, believed the disease to be due to infection of the epiphysis. He did not, however, discuss the question further and produced no evidence in support of his assumption.

The theory of infection being of importance as an etiologic factor of typical cases of O.d.j. has never been proved histologically or bacterio-

logically. That subacute synovitis of the hip can, according to FERGUSON & HOWORTH (1934), HOWORTH (1948, 1952) and HULTH (1958), produce circulatory disorders in the vessels running to the ossification centre of the femoral head cannot be excluded. But, as far as O.d.j. of the capitulum humeri is concerned, the present experimental investigation (see Chapter IV) showed that vessels to this ossification centre do not enter the joint cavity on their way to the ossification centre. It is therefore difficult to imagine how these vessels might be influenced by mild synovitis in the elbow joint.

None of the adherents of the infection theory have ever produced reliable cultures from cases of certain O.d.j. This is an argument against the theory, which has, practically speaking, been generally rejected.

II. *Primary fracture*

The second main theory of the etiology of O.d.j. supposes a primary discontinuity, a fracture, of the spongy bone of the ossification centre as the primary causal factor.

Such loss of continuity may presumably be caused by a single blow or strain (macrotrauma, HOLLÄNDER 1952) or repeated minor traumata with cumulative effect (microtraumata, HOLLÄNDER 1952).

This theory has been embraced by a number of workers in this field.

BOZSAN (1941) and SCHINZ et al. (1950) believe fracture to be the primary cause independently of the location of O.d.j.

WALTER (1930, 1934) speaks of "mechanisch bedingten Umbauprozessen", BRANDT (1941) and MÜLLER (1945) of "schleichende Kompressionsfrakturen" of the young spongy bone in the ossification centre.

Attempts have been made experimentally to produce O.d.j. changes by producing loss of continuity in endochondral growing ossific nuclei in young animals.

As early as 1915 ALLISON & MOODY tried to produce O.d.j. capitis femoris in young animals by direct traumatisation of the ossification centre but failed. AXHAUSEN (1923) performed similar experiments and with the same negative result.

NAGURA (1937, 1938, 1939, 1940, 1941) and NAGURA & KOSUGE (1938, 1939) have claimed experimentally to have proved the primary rôle of fracture as a cause of O.d.j. independently of the location of the lesion. They claim that the initial cause is a small loss of continuity in the growing spongy bone. Owing to the slightness of the fracture, the bone is not spared, and, according to these authors, "eigenartige Wiederher-

stellungserscheinungen", which they call "Nagurasche Erscheinung" appear. A major loss of continuity, on the other hand, produces such pronounced symptoms that the joint is spared and the fracture heals in a normal way. They do not believe that there are reliable grounds for the theory of circulatory disturbances as a cause of O.d.j.

These authors' experiments have been reproduced by BURCKHARDT & WERTHEMANN (1946) and BURCKHARDT (1948) who made similar conclusions.

In many respects NAGURA's theory resembles that propounded in 1936 by BURCKHARDT, "zweite neue traumatische Theorie".

Of those authors, who have described O.d.j. of the capitulum humeri, PANNER (1927, 1929, 1930) stressed that in all of the 3 cases described by him, the symptoms had been preceded by well defined trauma of the elbow. In all of them symptoms appeared immediately after the accident. PANNER therefore believed in a causal relationship between trauma and the roentgenologic changes. In 2 of these cases the first roentgen examinations were made 2 and 6 weeks, respectively, after the trauma, and showed fissure-like alterations in the ossification centre of the capitulum humeri. PANNER suspected, however, that the alterations could not be interpreted simply as traumatic fissures, a suspicion which proved justified on subsequent roentgen examination. He believed the rôle played by trauma in the etiology of O.d.j. of the capitulum humeri to be the incidental cause, but that an endogenous factor must be supposed to be present for changes characteristic of O.d.j. capituli humeri to be able to appear.

KREBS (1927) did not discuss the etiology of his case of O.d.j. of the capitulum humeri. The cause of that case was, however, discussed by PANNER (1929). He pointed out that the roentgenographic reproductions published by KREBS, which were taken 2 1/2 years after known trauma, showed the process in an advanced stage. PANNER therefore discussed the possibility that in that case the affection had remained latent for the 2 years between the trauma and the onset of symptoms. He therefore considered that it is not possible to exclude trauma as a possible cause in KREBS' case.

WINTERSTEIN (1933) was of the opinion that O.d.j. of the capitulum humeri is caused primarily by traumatic infraction of the bony tissue of the ossification centre, due to one or more traumata, or undue straining. According to him, once a young bone has been exposed to such trauma and the damaged tissue is strained, repair requires a long time.

MOL (1939) believed that his case of O.d.j. of the capitulum humeri which was diagnosed half a year after well defined trauma, supports the hypothesis of the significance of trauma in the etiology of the disease.

The theory of primary fracture as a cause of O.d.j. has been criticised by many. It has been claimed that anatomic and histologic studies as well as careful examination of large collections of O.d.j. of various locations have shown that fracture of the ossification centre of the spongy bone cannot be accepted as an explanation of the primary cause of the disease. It has been pointed out that the fracture theory *per se* provides no explanation for the familial, and sometimes inherited cases, and the bilateral cases of O.d.j. on record. A single trauma has often been held responsible for the development of the disease, but roentgen examination immediately or soon afterwards has often shown the picture of advanced changes undoubtedly much older than the trauma sustained. Cases of O.d.j. capituli humeri of this type have been described by KOHLBACH (1940) and BUETTI (1953) (Table 9).

In many cases of O.d.j. the patients have no history of known trauma. Thus, in 15 of 27 cases of O.d.j. of the capitulum humeri on record, the patients could not remember any definite trauma (Table 9). This, however, does not imply so very much because of the small number of cases. On the other hand, literature cases of the more common O.d.j. of the femoral head show that the findings differ as to the preceding trauma. TAYLOR & FRIEDER (1916), AMSTAD (1916), PLATT (1922), PERTHES & WELSCH (1922), SCHMIDT (1934) and HELBO (1953), for example, found a history of trauma to be missing in the majority of their cases. HELBO, for example, found preceding trauma in only one third of 204 patients. On the other hand, SCHWARZ (1914), LEGG (1916), SUNDT (1920) and GOFF (1954) reported preceding trauma in the majority of their cases, GOFF in as many as 65 % of 103 children.

It is thus clear that trauma is a relative term. Moreover, any description of trauma or denial of trauma by children must be judged with caution. It should, however, be remembered that traumata are every day occurrences in healthy children, especially boys in the ages in which O.d.j. capituli humeri (and capitis femoris) occurs.

An argument against primary fracture as a cause of O.d.j. of the capitulum humeri is offered by the work of HERMODSSON (1947) and JUD (1953). They thus showed a normal roentgenologic appearance of the growing epiphysis of the capitulum humeri immediately after severe

trauma of the bone, while 8 months later definite roentgenologic and clinical signs of aseptic bone necrosis in the ossification centre were demonstrable. It must, however, be admitted that these 2 cases are not enough to permit any valid conclusions.

LÖHR (1930) rejects the idea that simple fractures in the ossification centre of the capitulum humeri can lead to necrosis of the latter. His examinations argue against this conclusion, as already described on page 67.

LEHMANN (1940) published strong roentgenologic evidence for necrosis being primary and the fracture the secondary in O.d.j. of the femoral head. He believed the situation to be the same in O.d.j. of other locations.

v. TAPAVICZA (1940) and LANG (1941) assailed the fracture theory of NAGURA and claimed that the experiments of NAGURA and of KOSUGA did not produce O.d.j. because they did not find histologic evidence of bone necrosis.

As to strain or weight-bearing, which NAGURA and WINTERSTEIN believed to cause O.d.j. after primary fracture, it might be mentioned that in children between the ages of 4 and 10 years the capitulum humeri is generally not exposed to any major strain or weight-bearing especially as part of the physiologic strain in the joint is taken over by the trochlea.

O.d.j. of the capitulum humeri appears to be rare. Fractures in the ossification centre of the capitulum humeri, on the other hand, are relatively common. This also argues against primary fracture being the cause of O.d.j. of the capitulum humeri.

III. *Endogenous factors*

Familial and hereditary occurrence of O.d.j. is known (HELBO 1953, GOFF 1954 and others). Many bilateral cases have been described, including 2 cases of O.d.j. of the capitulum humeri (BUETTI 1953 and LAURENT & LINDSTRÖM 1956). Simultaneous occurrence of O.d.j. in different ossification centres is also known (MØLLER 1924, HELBO 1953, GOFF 1954 and others). The disease has also been found to vary with race (SUTRO & POMERANZ 1937, GOFF 1954).

This familial occurrence, heredity, multiple occurrence and difference with race cannot be explained by the etiologic theories discussed above (I and II). Therefore, many authors have discussed the possi-

bility of pre-existing endogenous factors in the ossification centre involved. Though they often admit that trauma or vascular disorders may be contributary factors in the development of O.d.j., they stress that some type of congenital malformation favouring growth disturbances of the ossification centre must be presumed. In addition, generalised disturbances and constitutional anomalies have also been considered as possible fundamental cause.

As mentioned, many of these authors also believe in the possibility of some exogenous factor (realisation factor, HOLLÄNDER 1952) capable of producing the typical picture of O.d.j. in the growing ossification centre.

CALVÉ (1921) and PERTHES (1922) believed endogenous factors to be of causal significance. Other authors sharing this view are FORSELL (1912), SUNDT (1920), BRANDES (1920), ZAAIJER (1921), ERLACHER (1922), LEHMANN (1923), SCHROEDER (1924), HOLLÄNDER (1952) and GOFF (1954).

CAAN (1924) stressed, however, that it is not easy to accept the hypothesis of congenital malformations or developmental disorders, especially in those cases in which the disease has been seen to develop in previously intact ossification centres, which has also been stressed by WALDENSTRÖM (1934), LANG (1941) and others.

SCHINZ et al. (1952) states that the hypothesis of a constitutional, genetic factor in the etiology of O.d.j. does not satisfactorily explain the special monostotic location of the affection in most cases.

The only primordial and possibly inherited factor, LANG (1941) and BREITNER & LANG (1949) are willing to recognise in the causation of O.d.j. is a possible irregularity in the "Anlage und Entwicklung" of the architecture of the epiphyseal vessels, a hypothesis already discussed in 1927 by HEINE.

CHANDLER (1940) discussed the rôle of congenital vascular anomalies in local pathologic processes in the skeleton.

Even authors who have described O.d.j. of the capitulum humeri have considered the possibility of endogenous factors.

PANNER (1929, 1930) thus expressed the view that an endogenous factor must pre-exist in the epiphysis of the capitulum humeri for the "incidental" factor, *i.e.* trauma, to produce the pathologic changes typical of O.d.j. of the capitulum. He does not, however, say anything about the possible nature of such endogenous factors.

BUSCH (1930) propounded that O.d.j. of the capitulum humeri might

be due to pre-existing structural anomalies of the distribution of the vessels in the epiphysis, and that the effect of various factors, traumatic or constitutional, might lead to the pathologic changes.

JUD (1953) took up the same line of thought. He thus presumed the possibility of a poorly developed ossification centre, the maldevelopment possibly involving the vascular system of the epiphysis as a fundamental factor in the etiology of O.d.j. of the capitulum humeri.

In HEGEMANN's (1951) 4 cases of O.d.j. of the capitulum humeri, the patients could not remember any preceding trauma or stress with certainty. He therefore believed that since the disease appears in an epiphysis, which is in a state of lively activity, there is a certain undefined "Schwäche" of the ossification centre during that period. HEGEMANN does not believe in the significance of a single trauma or mechanical stress as etiologic factors.

LANGE (1954) described a case of O.d.j. of the capitulum humeri without known preceding trauma. He does not believe trauma to play any significant rôle in the etiology, and states that if it did, the disease would be more common. He presumes that the epiphysis of the capitulum humeri is vulnerable during the period of greatest activity in the ossification centre. He does not, however, exclude the possibility of trauma as a precipitating factor.

LAURENT & LINDSTRÖM (1956) also imagined the possibility of vulnerable epiphysis of the capitulum humeri during a period of great activity. In their 2 cases of O.d.j. of the capitulum humeri, the patients could not remember any trauma. They stressed, like HEGEMANN (1951) and LANGE (1954), that the disease should be more common if trauma played a main rôle in the etiology. They also claimed that the bilateral occurrence of the disease in one of their cases argues against traumatic origin. They would not, however, exclude trauma as a precipitating factor of O.d.j. of capitulum humeri in a constitutionally weak epiphysis.

It is obvious that all of these hypotheses of endogenous factors are difficult to prove, because most of the factors under consideration cannot be checked.

Judging by the literature available, it must, however, be concluded that the factors arguing against trauma as the only cause of O.d.j. in general and O.d.j. of the capitulum humeri in particular, at the same time support the assumption of some type of pre-existing endogenous factor.

Endocrine dysfunction

Finally there is the hypothesis believing endocrine disturbances of varying types, but especially hypofunction of the thyroid gland, to be the basal cause of O.d.j. It has long been known that skeletal development is retarded in cretins and children with myxoedema juvenilis. Sometimes these patients also show changes in the endochondral ossification centres, most commonly observed in the femoral head, which roentgenologically resemble O.d.j. (BIRCHER 1909, LOOSER 1929, ALBRIGHT 1938, WILKINS 1950, TALBOT, SOBEL, MCARTHUR & CRAWFORD 1952).

As mentioned, SCHAEFER, STRICKROOT & PURCELL (1939) found "chondroepiphyseal disturbance" in 91 of 258 patients with endocrine disease during the age of growth. Of these patients, 3 had roentgenologic changes in the ossification centre of the capitulum humeri, which were classified by the authors as changes of the same type as those described by PANNER (1927). These authors presumed that O.d.j., independently of its location and including the capitulum humeri, is caused by hypothyroidism.

As pointed out on page 42, the diagnoses O.d.j. capituli humeri in their cases cannot be regarded as confirmed. No authors who have described cases of unequivocal O.d.j. of the capitulum humeri have ever found reason to suspect endocrine disturbances as a causal factor.

The suggestion of hypothyroidism playing a rôle in the etiology of O.d.j. has been criticised from many quarters. On the basis of personal investigations including histologic studies, LOOSER (1929) arrived at the conclusion that the disturbances in the ossification centre due to hypothyroidism are caused by retarded and irregular endochondral ossification and claimed that they are not identical with O.d.j. This opinion is shared by ALBRIGHT (1938), TALBOT et al. (1952) and CAFFEY (1956).

GILL (1943), HOWORTH (1948) and HELBO (1953) found no evidence of hypothyroidism in large materials of children with O.d.j. of the femoral head.

Recapitulation

1. Three main theories are available of the etiology of O.d.j. of the capitulum humeri and other endochondral ossification centres. Most authors, however, agree that none of these theories by itself can ex-

plain the entire etiology and that an interplay of various factors must be assumed.

2. The theories of the etiology fall broadly into the following groups:

I. *Circulatory disorders of the ossification centre. — Mechanical traumatisation of the vessels with circulatory disorders as a consequence.* The few reports by various authors of single cases of O.d.j. of the capitulum humeri in which the authors believed there was reason to assume a vascular lesion, are by no means sufficient to permit any valid conclusions of the rôle played by such lesions in the etiology of the disease. On the other hand, it may be regarded as established that O.d.j. of the femoral head can be caused by the effect of traumatisation on the vessels of the ossification centre without primary loss of continuity within the ossific nucleus. It is believed that the close resemblance between the roentgenologic and clinical picture of O.d.j. of the femoral head and of the process in the capitulum humeri warrant the conclusion that it is simply a question of different locations of one and the same pathologic process. It is therefore not possible to reject the theory that a mechanical traumatisation of the vessels of the ossification centre of the growing capitulum humeri may lead to circulatory disturbances in the latter with O.d.j. as a consequence. Acceptance or rejection of this theory, however, requires more detailed knowledge of the vascular anatomy of the growing capitulum humeri epihysis. A description of the topography of these vessels is given in chapter IV.

Other possible causes of these disorders of the ossific nuclei have been suggested, such as *embolism, endarteritis obliterans, neurogenic causes, infection.*

3. II. *Primary fracture* as a fundamental cause. In none of the cases of O.d.j. of the capitulum humeri on record was the disease ascribed to primary fracture. O.d.j. in this location appears to be rare. Fractures of the capitulum humeri in children, on the other hand, are relatively common and no such lesion have ever been described as resulting in roentgenologic changes of the type seen in O.d.j. In many cases of O.d.j. of the capitulum humeri the disease was not preceded by any known trauma. Two bilateral cases are on record. Judging by the histologic findings in O.d.j. of the femoral head, the aseptic necrosis is primary and the loss of continuity secondary. Many cases of O.d.j. of the femoral head have been described as familial and hereditary.

All of these observations argue against primary fracture being the cause of O.d.j. of the capitulum humeri.

4. III. *Endogenous factors.* — All of the factors arguing against trauma as the only cause of the disease, support the assumption of some type of endogenous factor.

The monostotic occurrence of O.d.j. of the capitulum humeri, however, argues against a generalised, constitutional, endogenous factor.

Endocrine dysfunction. — In none of the cases of O.d.j. of the capitulum humeri on record have the authors ever mentioned evidence of endocrine disturbances.

5. TO SUMMARISE, it may be said that O.d.j. of the capitulum humeri is probably an aseptic necrosis in the growing ossification centre. The necrosis might be ascribed to obstruction or interruption of the blood supply to the ossification centre.

The cause of these disturbances of circulation and the vascular anatomy favouring their development are, however, not properly understood.

11. Treatment and results

It is clear from Table 10 that in the cases on record the patients received either expectant treatment or immobilisation of the elbow.

PANNER (1929, 1930) believes that no actual treatment is necessary. BUETTI (1953) and JUD (1953), on the other hand, recommend immobilisation of the elbow. LANGE (1954) prefers to immobilise the elbow until signs of pronounced regeneration of the ossification centre have appeared, while LAURENT & LINDSTRÖM (1956) believe that immobilisation during the period of acute, clinical symptoms is sufficient, as long as the patient spares the elbow afterwards.

Table 10 shows that only 8 of the cases were followed until the pathologic process had healed.

Only 2 (case Nos. 13 and 29) of them were followed until the end of the period of growth. Therefore, it was only in these 2 cases that the process were followed to the definite stage in WALDENSTRÖM's sense of the term. In these 2 patients, the last roentgenograms of the capitulum humeri 6 1/2 years and 7 years, 5 months, respectively, after the onset of symptoms were described (ELWARD 1939, LAURENT & LINDSTRÖM 1956) as being of normal appearance.

In the other 6 cases (Nos. 1, 2, 15, 27, 28, 30) the last roentgenograms showed healing of the pathologic process while the ossific nucleus was not yet quite normal. All these cases were, however, still growing.

In none of the cases in literature, which have been followed up until

TABLE 10

Treatment, time of observation and results in cases from literature

Author	Case No.	Treatment	Time of observation	Results at last examination
PANNER, H. J.	1	Sparing elbow	3 years	Healed with slight deformity
	2	„	1 y., 5 m.	„
	3	„	1 y., 6 m.	Not healed
KREBS, C.	4	None	1 y., 6 m.	Not healed
BUSCH, E.	5		3 weeks	Not healed
HAGEN, J. F. et al.	6	Immobilisation	None	Not healed
KING, E. S. J.	7	Sparing elbow		
	8	„		
	9	„		
NIJST, E. E.	10		9 1/2 m.	Not healed
MOL. W.	11	Sparing elbow	1 y., 3 m.	Not healed
RIVAROLA, J. E.	12	Immobilisation	1 y., 3 m.	Not healed
ELWARD, J. F.	13	Sparing elbow	6 1/2 y.	Healed without deformity
	14		1 year	Not healed
KOHLBACH, W.	15		3 years	Healed with slight deformity
	16		None	Not healed
MARCH, H. C.	17		None	Not healed
HEGEMANN, G.	18	Immobilisation	5 months	Not healed
	19			
	20			
	21			
CAMERA, R.	22	Immobilisation	1 y., 2 1/2 m.	Not healed
SEMMELROCH, H.	23	Sparing elbow	1 y., 8 m.	Not healed
BUETTI, C.	24	Sparing elbow	3 months	Not healed
	25	„	None	„
MASON, M. L.	26	Immobilisation	3 months	Not healed
JUD, H.	27	Immobilisation	1 y., 9 m.	Healed with slight deformity
LANGE, J.	28	Immobilisation	2 y., 9 m.	Healed with slight deformity
LAURENT, L. E. et al.	29	Immobilisation	7 y., 5 m.	Healed without deformity
	30	„	2 y., 9 m.	Healed with slight deformity

healed, have any severe deformities of the capitulum humeri been observed.

Judging from the reproductions of the last roentgenograms of these cases, it appears that the deviation from normal was not less in those patients treated with prolonged immobilisation than in those who had received expectant treatment only, an observation also made by LAURENT & LINDSTRÖM (1956).

Neither does immobilisation appear to have any influence on the duration of the pathologic process.

12. Prognosis

All authors who have described this disease agree that the prognosis is good quoad sanationem.

Whether the prognosis quoad functionem in later life is equally good, is not yet known, because no patient have as yet been followed up long enough to permit any such decision.

JUD (1953) suggested that the disease might favour the later appearance of arthropathies, but no such cases have ever been published.

MASON (1953) stresses that the risk of deformity is relatively slight, because the ossification centre involved is not in a weight-bearing joint.

PART II

Chapter IV

EXPERIMENTAL INVESTIGATIONS OF THE INTRA- AND EXTRA-OSSEOUS VASCULATURE OF THE DISTAL END OF THE HUMERUS WITH SPECIAL REFERENCE TO CAPITULUM

Introduction

As mentioned in chapter III, there appears to be reasonable clinical and roentgenologic evidence that O.d.j. of the capitulum humeri is an aseptic total necrosis of the ossific nucleus in the growing capitulum.

Such necrosis is presumably due to obstruction or occlusion of the blood supply to the ossification centre.

The cause of such a circulatory disturbance and how it produces total necrosis of the largest ossification centre of the distal end of the humerus, and why such a disturbance always appears during a certain period of growth are, however, still obscure. This obscurity is due in part to lack of knowledge of the intra- and extraosseous vasculature of the distal end of the humerus in childhood and in adult age.

As to O.d.j. of the femoral head, anatomic observations and experimental investigations (vide infra) have shown that the vascular anatomy of the femoral head is such that the circulation of its ossification centre is susceptible of injury by exogenous insults.

The isolation of this part of the skeleton, as far as the circulation is concerned, during the period of growth is thus a factor that favours circulatory disturbances.

A question that unsought presents itself is whether this situation also holds for the vascular supply to the ossific nucleus of the growing capitulum humeri.

Some authors (AMSTAD 1916, BENTZON 1926, 1927, LÖHR 1930, 1931, UHRMACHER 1933, HERMODSSON 1947 and JUD 1953) believe that total necrosis of the ossific nucleus of the capitulum humeri may be caused by disturbance of its blood supply. But they do not base their assumption on any satisfactory description of the vascular topography of the capitulum, and since this vasculature has not yet been properly charted, the discussion of the etiology has been founded mainly on clinical and roentgenologic similarities between O.d.j. in the capitulum humeri and in other locations, particularly in the femoral head.

Since the theory of the disturbed circulation as a causal factor of O.d.j. in the capitulum humeri or in other locations must be founded mainly on knowledge of the vascular topography of the ossification centre, little progress can be expected in this field before the intra- and extra-osseous vasculature of the capitulum humeri in the growing and full-grown skeleton has been cleared up.

It was against the background of these considerations that this part of the present investigation was started, the purpose of which was to elucidate the vasculature of the growing and full-grown distal end of the humerus by means of injection experiments, in the hope that it would:

elucidate some problems bearing on the nutrition of the unossified distal chondroepiphysis of the humerus as well as the vasculature in osseous tissue in the growing and full-grown epiphysis and metaphysis of the distal end of the humerus. The results may also be of interest in the investigation of the vasculature of long bones in general, and

help to shed light on some details in the etiology of O.d.j. of the capitulum humeri which might also be of interest in the etiology of O.d.j. elsewhere.

Earlier investigations

Demonstration of the vascular pattern by the injection of some suitable substance was described as far back as the seventeenth century by the anatomist REGNERUS DE GRAAF (1678). This method has since been elaborated but used mainly for the investigation of the vascular system in the soft parts.

During the last 50 years or so, however, the intra-osseous vasculature of different parts of the skeleton has received increasing interest.

In 1876 LANGER described experiments in which he tried to elucidate the vasculature of the long bones by intravascular injection of dyes. That investigation together with another published in 1895 by SIRAUD and later publications by LEXER (1903, 1904), LEXER, KULIGA & TÜRK (1904) and DELKESKAMP (1906), who injected radiopaque material, form the basis of our knowledge of the pattern of the gross intra-osseous vessels.

In attempts to form a more detailed opinion of the intra-osseous vasculature in different parts of the skeleton, various investigators have since contributed with experiments using various types of injection techniques.

BELOU (1934) must be regarded as the one who has used this method most extensively. He made a systematic study of the major part of the human skeleton.

Of the various parts of the human skeleton studied in this respect, the proximal end of the femur has received most attention. Knowledge of the vasculature of this part of the skeleton has been increased by the injection experiments of LÁNG (1916), NUSSBAUM (1924, 1926), BENTZON (1926), ANSEROFF (1934), WOLCOTT (1943), TUCKER (1949), HARTY (1953), TRUETA & HARRISON (1953), JUDET, JUDET, LAGRANGE & DUNOYER (1955), HULTH (1956, 1958) and TRUETA (1957).

The vasculature of the human femoral shaft was studied by injection methods in 1921 by GRÉGOIRE & CARRIÈRE and in 1953 by LAING, while the vasculature of the distal end of the femur was studied by this method by NUSSBAUM (1923), BENTZON (1926), HARRIS (1933), ANSEROFF (1934) and ROGERS & GLADSTONE (1950).

RÖPKE (1904), who used the injection technique studied the vascular anatomy of the patella; HANSON (1926), of the vertebral body; and BENTZON (1926) and NOVOTNY (1937), of the metatarsal bones.

With this method BENTZON (1926) demonstrated vessels in the scaphoid bone of the foot and the fibula, while GRETTVE (1955) used the injection technique to elucidate the vasculature of the carpal bones.

Of other authors who have used this method to elucidate details of the intra-osseous vasculature of various parts of the skeleton mention might be made of LAURO & PURPURA (1956), who studied the vasculature of the human talus and calcaneus and DRINKER, DRINKER & LUND (1922), KALLIUS (1932), RUBASCHEWA & PRIWES (1932), OLOVSON (1941), REICHEL (1947), DE MARNEFFE (1951), BROOKES (1957), LEMOINE (1957) and DALE & HARRIS (1958) who worked with laboratory animals.

However, judging by a perusal of the literature, the vascular system in the distal end of the humerus in childhood and in adult age has received less attention. BENTZON (1926) who used contrast roentgenography, studied the intra-osseous vasculature of the distal end of the humerus in a 6-year old boy. He demonstrated a vessel in the capitulum humeri, but the position of the vessel in relation to the epiphyseal plate, which is of considerable importance, cannot be judged from his illustrations. In addition, his report was based on only a single case.

On the basis of anatomic studies and experiments with the injection technique LÖHR (1930, 1931) arrived at the conclusion that the capi-

tulum of the humerus receives its blood supply mainly from branches given off by the arteria recurrens interossea and from the art. collateralis media (Fig. 33).

However, apart from an injection experiment on a newborn, LÖHR gave illustrations of the injected vasculature of only the soft parts of the elbow.

ELETTO (1933), BELOU (1934), PELLEGRINI (1934), ANSEROFF (1934), FRACASSI (1941) and LAING (1956) investigated the intra-osseous vasculature of the human humerus by the injection technique, but they did not give any satisfactory detailed description of the vessels in the epiphysis of the distal humerus.

HARALDSSON (1957) published a preliminary communication of the intra-osseous vasculature of the distal end of the humerus with special reference to the capitulum.

Though the injection technique has been widely used since the turn of the century in investigations to elucidate the vasculature of the skeleton, many details of the circulation in the bones are still obscure.

Recapitulation

1. There are still many details in the circulation of the growing and full-grown skeleton that are not yet properly understood.

2. Knowledge of the vascular anatomy of the distal end of the humerus in childhood and adult age is incomplete.

Author's investigations

1. Methods

Demonstration of the vascular pattern in the distal end of the humerus in childhood and adult age to determine whether the vascular anatomy in these regions might be such as to favour the occurrence of circulatory disorders requires not only knowledge of the intra-osseous, intra-chondral course of the vessels, but also of the path of the vessels in the neighbourhood of the bone before they enter the latter.

In the present injection experiments performed on cadavers to elucidate this situation, the following methods were used.

A. Injection of radiopaque material into vessels supplying the distal end of the humerus, after which angiograms were taken.

This was the main method used and it was chiefly on the findings made by this technique that the results of the investigations are based.

B. Injection of acrylic mass with subsequent destruction of the soft tissues to secure acrylic casts of the extra-osseous course of the vessels of the distal end of the humerus.

This method was used in only a few cases.

A. Injection of radiopaque material and subsequent angiograms

This method was used in 29 of the 33 injection experiments. Of these 29 injection experiments the results obtained in 5 were less satisfactory and were therefore rejected. This left a total of 24 for angiographic analysis.

It was soon realised that the conventional contrast media [TEICHMANN (cit. OLOVSON 1941), SPALTEHOLZ (1911), TRUETA & HARRISON (1953) and TRUETA (1957) for example] did not demonstrate the small vessels in the ossifying epiphysis with satisfactory distinction without previous decalcification or slicing of the specimens. In the examination of ossifying epiphysis it is desirable to distinguish the vessels in the ossification nucleus from those in the unossified chondroepiphysis. However, decalcification will blur the border between the ossification nucleus and the surrounding cartilage and then, of course, it is difficult to decide which vessels belong to the nucleus of ossification and which belong to the surrounding cartilage.

In order to eliminate this disadvantage basic lead carbonate [$2PbCO_3$, $Pb(OH)_2$] was used as radiopaque material (OLOVSON 1941). The composition injected consisted of:

Plumbi subcarbonas	g 200
Sol. natrii chlor. physiolog.	g 200
Gummi arabicum	g 20

This composition is cheap, easy to prepare and easy to handle. The contrast density obtained is good and even the small vessels are distinctly visualised, even before decalcification and without slicing of the specimen.

Similar results can be achieved with mercury-emulsion as an injection mass (LEXER 1903, 1904, LEXER, KULIGA & TÜRK 1904) but the technique is more time-consuming and expensive.

In the investigation of the vasculature of adult bones it is also desirable to study the topography of the vessels before decalcification. However, in an attempt to form a clearer opinion of the blood supply, many of the specimens in the present investigation were decalcified. The pre-

parations were decalcified with a fluid described by JENKINS in 1921, because the radiopaque material used is soluble in the ordinary decalcificants, *i.e.* 5 per cent nitric acid or trichloracetic acid. In view of this instability, which is the main disadvantage of this injection mass, HARRISON (in BARCLAY 1951) claimed lead compounds to be unsuitable for injections of this type.

The contrast material was heated to +40° C, filtered through 8 layers of gauze and injected into the brachial artery. (In a 3 month old boy it was injected via the thorax into the subclavian artery.) Before injection a bandage was wound tightly round the middle section of the forearm. With an ordinary 100 ml syringe about 200 ml of the radiopaque material was injected without previous perfusion of the vessels. No attempts were made to measure the injection pressure. Contrast medium was injected until it began to flow out into the chest via the subclavian vein. In order to secure a good filling of the vessels, the injection was spread over 5 minutes. The distal end of the humerus was excised 15 minutes after the end of the injection, by when the mass had set. The preparation was fixed in 5 per cent formalin for 24 hours. The periosteum and the soft parts were removed and stereoscopic angiograms were taken, after which some of the specimens were placed in JENKIN's fluid and the decalcification process was followed roentgenologically.

In order to be sure that the vessels studied belonged only to the capitulum of the humerus most of the specimens were sawn through in the sagittal plane at the border between the capitulum and the trochlea to obtain isolated views of the capitulum and its vessels (Fig. 15). Frontal views were, however, taken of all specimens while they were still intact.

B. *Injection of acrylic mass with subsequent destruction of soft tissues*

As mentioned, the angiographic method proved suitable for investigation of the intra-osseous and intra-chondral vascular anatomy in the distal end of the humerus. However, difficulties arose as soon as the method was applied to elucidate the course of these vessels in the vascularised soft tissues adjacent the distal end of the humerus. After injection of the radiopaque material, angiograms of the elbow before dissection showed that the contrast mass filled the vascular net-work

of the soft tissues to such an extent, that it was not possible to distinguish the vessels running to the distal end of the humerus. As will later be apparent, however, in some cases it was possible to obtain contrast roentgenograms of the final segments of the extra-osseous vessels, *i.e.* just before they entered the bone. This was facilitated by less radical dissection, *i.e.* by leaving part of the soft tissue on the posterior part of the capitulum.

It was, however, considered desirable to supplement the data obtained from the contrast roentgenographic method by further information on the extra-osseous course of the vessels of the capitulum. It was also desired to check the correctness of LÖHR's (1930) hypothesis that a great part of the supply of the capitulum is taken care of by the arteria recurrens interossea and WATSON-JONES' (1952) opinion that the capitulum humeri and trochlea are supplied by vessels ascending from the lower arm. To elucidate this question acrylic mass was injected into the axillary artery of 4 adult cadavers (Specimen Nos. 15, 16, 17, 19, Table 11) and the soft parts of the elbow were afterwards macerated to obtain acrylic casts of the extra-osseous vascular anatomy of the distal end of the humerus.

The injection mass used was that described by BATSON (1955) and is essentially a methyl methacrolate monomer which is polymerized to the solid polymer at room temperature. This material has a minimum of shrinkage, it can be used at room temperature and can be readily injected. It can be coloured by various pigments. This acrylic mass makes rigid casts of the vessels and has a high resistance against the chemicals ordinarily used for maceration. The working time before the mass is set is between twenty and thirty minutes.

The method was first tried in 11 injection experiments on animal cadavers (pig's feet) before it was used on human cadavers.

A mixture of 100 c.c. of the base solution with 5 per cent Cinnebar, 20 c.c. of a catalyst and 12 drops of a promoter was prepared and injected into the axillary artery with an ordinary 100 ml syringe. Before injection a bandage was wound tightly round the forearm, a handbreadth above the wrist. To secure good filling of the vessels, the injection was spread over 5 minutes. After 45 minutes the mass had set, and the region of the elbow was excised subcutaneously. The preparation was then placed in water for 4 hours, during which the mass became hard.

On maceration of the soft tissues difficulties arose. It was soon rea-

lised that specimens of the bones of the elbow joint with acrylic casts of vessels running to the capitulum could not be obtained with conventional maceration substances such as sodium hydroxide, potassium hydroxide or hydrochloric acid. These chemicals give good maceration of the soft tissues of region of the elbow, and good reproductions of the acrylic casts of the vessels of the soft tissues, but they also destroy the bone, so that it is difficult to judge the topographic relation between the acrylic casts of the vessels of the soft tissues and the capitulum with certainty. REICHEL (1947) and DE MARNEFFE (1951) who worked with the plastic injection mass in experimental animals contented themselves with dissection of the casts of the largest soft tissue vessels running to the bones, because the maceration method they used destroyed osseous tissue.

Attempts were therefore made to devise a maceration method permitting maceration of the soft tissues of the region of the elbow without interfering with the bone or the acrylic vessel casts.

Various procedures were tried and finally a method satisfying the above requirements was elaborated. The method was essentially as follows. After injection of the acrylic mass into the elbow, the specimen was placed in broth containing gas gangrene bacteria (Cl. Welchii). In this way the soft tissues were gradually macerated without any appreciable damage to the acrylic vessel casts or to the osseous tissue, but complete maceration of the soft tissues required a fairly long time, in specimen No. 17 (Fig. 32) it took 13 months. (The method might possibly be improved by the use of anaerobic bacteria with greater proteolytic activity.)

2. Material

The material consisted of 31 human cadavers of subjects, aged 3 months to 80 years. In 2 of them (a 3-year old and 8-year old boy, Table 11) the mass was injected into both arms. The total number of preparations therefore consisted of 33. Five of these preparations proved less satisfactory and were rejected. This left 28 preparations for the present investigation.

The material is summarised in Table 11. Since the cause of death in these cases can hardly have influenced the vasculature of the distal end of the humerus, the main cause of death is not included in the table.

It is clear from the table that all 10-year age classes, from 0 to 80 years, were represented.

TABLE 11

Age- and sex distribution of injected specimens of distal humerus

Specimen No.	Age	Sex	Number of specimens at each age	Ten-year periods
13	3 months	♂	1	0—10
29, 30	3 years	♂♂	2	
21, 22	8 „	♂♂	2	
18	9 „	♂	1	
20	19 years	♂	1	11—20
7	28 years	♀	1	21—30
32	38 years	♀	1	31—40
33	44 years	♂	1	41—50
10	52 years	♂	1	51—60
9	56 „	♂	1	
28	57 „	♂	1	
31	60 „	♀	1	
14, 24	61 years	♂♂	2	61—70
12, 16	63 „	♂♀	2	
11	64 „	♂	1	
8	68 „	♂	1	
26	71 years	♂	1	71—80
15	72 „	♀	1	
17	73 „	♀	1	
19	74 „	♂	1	
6, 25	75 „	♀♀	2	
23	79 „	♂	1	
27	80 „	♂	1	
		Total	28	

The first decade, *i.e.* the period of life that O.d.j. of the capitulum humeri is known to appear, was represented by 6 specimens.

As mentioned, most authors using the injection technique in the study of the epiphyseal vasculature in growing human beings worked with epiphyses that are ossified from a single ossific nucleus: femoral head, distal femoral epiphysis. These ossific nuclei appear early in life,

the ossific nucleus of the femoral head appearing in the second to eighth month of life in boys, and the nucleus of the distal epiphysis of the femur in boys at birth (CAFFEY 1956). This implies that the period available for study of the intra-chondral vasculature of these epiphyses before the appearance of the ossification process is short. Therefore, in these epiphyses it cannot be excluded that the presence of vessels in the chondroepiphysis before ossification, may be heralds of the ossification process and that the vessels do not belong to the vasculature proper of the cartilage.

The epiphysis of the distal end of the humerus, on the other hand, is ossified from 2 main ossific nuclei, which appear at very different stages in the period of growth. Thus, while the ossific nucleus of the capitulum appears in boys at the age of 1—26 months (ELGENMARK 1946, Table 2, Fig. 3), that of the trochlea does not appear until the age of 8—13 years in boys (HASSELWANDER 1931, Table 2, Fig. 3), which is unusually late for a main ossific centre of an epiphysis.

The epiphysis of the distal end of the humerus thus provides a possibility of studying the vasculature not only in the ossifying capitulum, but also in the growing unossified chondroepiphysis of the trochlea at the same time.

The chondroepiphysis of the trochlea can be studied with respect to cartilaginous vessels for a long time before the appearance of the ossification process in the cartilage.

For these reasons the distal end of the humerus lends itself particularly well to the study of the vasculature in growing epiphysis.

3. Findings

Demonstration of injected specimens

Descriptions and illustrations of some of the injected specimens are given below.

The preparations exemplify the vasculature of the distal end of the humerus in different stages of development.

It should be mentioned that all of the photographs of the specimens injected with radiopaque material (except Figs. 8, 9 and 14) are stereoscopic.

Specimen No. 13. Boy, aged 3 months. Radiopaque medium was injected 14 hours *post mortem*. Right arm. The specimen is not decalci-

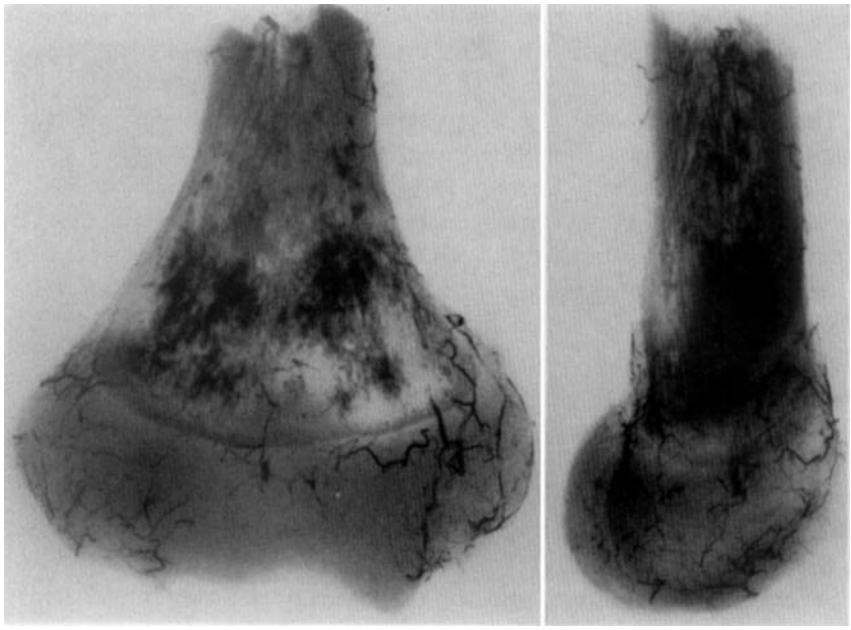


Fig. 8. *Specimen No. 13. — Age 3 months.*
Whole specimen with perichondrium in situ.

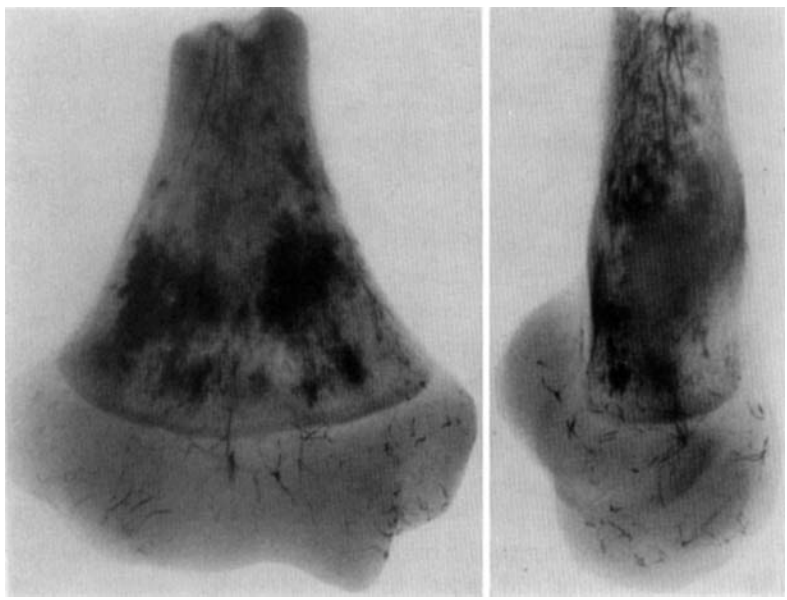


Fig. 9. *Specimen No. 13. — Age 3 months.*
Whole specimen after removal of perichondrium.

fied. Fig. 8 shows the preparation *before* removal of the perichondrium of the chondroepiphysis, and Fig. 9 *after* careful removal of the perichondrium.

The chondroepiphysis of the distal end of the humerus shows no roentgenologic signs of incipient ossification.

Although the finest details are lost in a printed reproduction, judging by Figs. 8 and 9, canals are seen here in the hyaline cartilage. They seem to enter the chondroepiphysis from that part of the surface of the latter covered by perichondrium and not taking part in the formation of the articular surface. Secondly canals, which appear to come from the margin of the metaphyseal bone dip in distal direction into the chondroepiphysis. Figs. 8 and 9 give the impression that at least most of these canals end blindly in the cartilage.

In that part of the chondroepiphysis, consisting of joint cartilage, no canals are seen.

Irregular intra-osseous patches of contrast medium are demonstrable in the ossified metaphysis.

Several vessels coming from the diaphysis pass down into the metaphysis.

Specimen No. 13 thus shows a canal system in the chondroepiphysis of the distal end of the humerus, which is demonstrable in the temporary cartilage before the appearance of any roentgenologic signs of ossification.

Specimens Nos. 29 and 30. Boy, aged 3 years. Radiopaque medium was injected into both elbows 57 hours post mortem.

Specimen No. 29, right arm, is illustrated in Figs. 10, 11 and 12, specimen No. 30, from the left arm in Fig. 13. The specimens are not decalcified. The specimens sawn in the way described (Fig. 15) are illustrated in Figs. 11, 12 and 13.

It is clear from the illustrations that the ossific nucleus of the chondroepiphysis of the capitulum was well developed in these specimens.

As to the vasculature of this ossific nucleus, Figs. 10, 11 and 13 show that 1 to 2 vessel canals enter the posterior aspect of the chondroepiphysis, traverse a great part of the cartilage and then enter the ossific nucleus from behind.

Fig. 13 (Specimen 30) shows not only the main vessel canal coming from behind and running to the ossific nucleus but also a minute vessel canal descending from the radial fossa. This canal swings round the

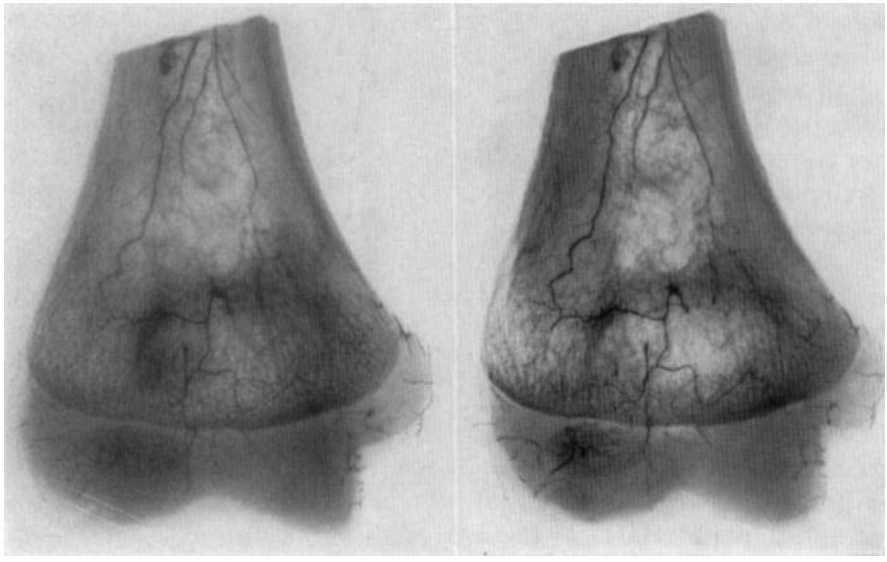


Fig. 10. *Specimen No. 29. — Age 3 years.*
Stereoscopic pictures of whole specimen.

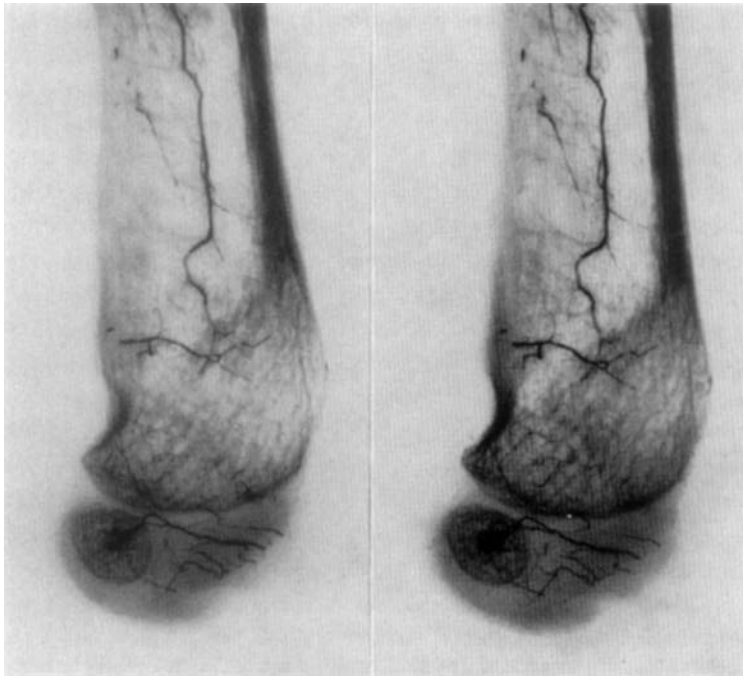


Fig. 11. *Specimen No. 29. — Age 3 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

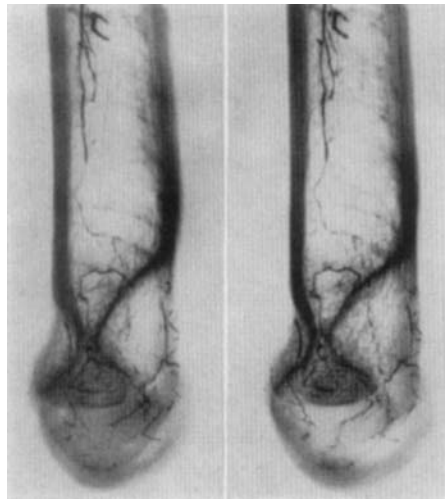


Fig. 12. *Specimen No. 29. — Age 3 years.*
Stereoscopic pictures of isolated trochlea and medial metaphysis.

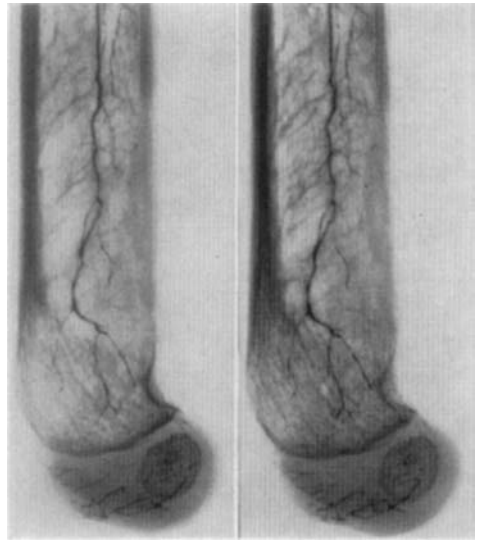


Fig. 13. *Specimen No. 30. — Age 3 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

ventral margin of the metaphyseal bone, traverses the ventral margin of the epiphyseal plate and appears to extend to the ossific nucleus.

It is clear from Figs. 10, 11 and 13 that the ossific nucleus of the capitulum in these specimens is, as far as vascularisation is concerned, isolated from the intra-osseous vascular system in the metaphysis and from the other canals in the surrounding, unossified chondroepiphysis.

The main vessels coming from behind and proceeding to the ossific nucleus of the capitulum and described above, enter the chondroepiphysis at a site, which, judging from Figs. 10, 11 and 13, is not within the elbow joint (as mentioned, the attachment of the joint capsule follows the posterior margin of the joint cartilage of the capitulum, Fig. 5).

Figs. 10, 11, 12 and 13 show contrast-filled canals inside the unossified part of the chondroepiphysis. The joint cartilage and the adjacent parts of the chondroepiphysis, however, show no canals. Judging by these illustrations, these cartilage canals do not intercommunicate but terminate blindly within the cartilage. These cartilage canals do not communicate with the ossific nucleus of the capitulum or its vessels.

The unossified chondroepiphysis of the trochlea (Figs. 10 and 12) contains a number of canals running toward the site where the trochlear ossific nucleus will appear on further development of the bone. Some of these canals enter the cartilage from the ulnar surface of the trochlea (Figs. 10 and 12). Others descend from the coronoid fossa and the olecranon fossa and pass the anterior and the posterior border respectively of the metaphyseal bone on their way down into the chondroepiphysis. These canals thus pass through the anterior and posterior outer perimeter of that region in the chondroepiphysis, which represents the epiphyseal plate, the zone of growth in length (Fig. 12).

Figs. 10, 11, 12 and 13 show intrachondreal canals in those regions of the temporary cartilage, where the ossific nuclei will later appear in the medial and the lateral epicondyles.

Figs. 10, 11, 12 and 13, like Figs. 8 and 9, thus show a number of contrast filled canals in the temporary cartilage of the distal end of the humerus before the appearance of any roentgenologic signs of incipient endochondral ossification in the trochlea or epicondyles.

But since the roentgenograms do not permit any decision as to whether the contrast-filled cartilage canals are blood-vessels or canals of some other sort, these angiographic studies were supplemented by a histologic examination of the unossified chondroepiphysis of the troch-

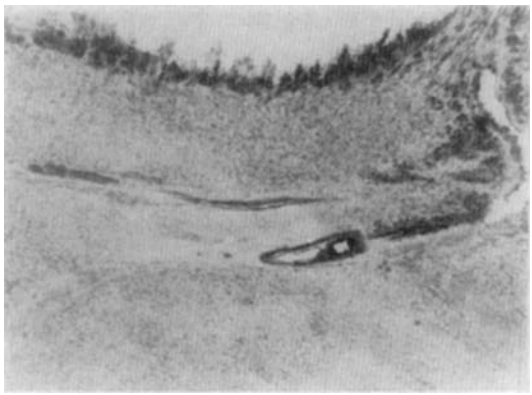


Fig. 14. *Specimen No. 29. — Age 3 years.*
Microsection from cartilaginous trochlea showing blood-vessels containing radiopaque material.

lea of Specimen No. 29. It was thus the cartilage canals shown in Fig. 12 that were examined histologically. This histologic examination (Professor Gösta Häggqvist) showed blood vessels containing injection mass alongside of vessels where the injection mass had fallen out in association with the preparation of sections (Fig. 14).

No direct communications were seen between the intra-osseous vascular net of the metaphysis on one hand and the vascular system of the epiphysis on the other, which includes vessels running to the ossific nucleus of the capitulum and the vessels in the unossified chondroepiphysis (Figs. 10, 11, 12 and 13).

A fairly large vessel runs horizontally and enters the metaphysis at the proximal edge of the coronoid fossa and afterwards branches in the metaphyseal bone (Figs. 10, 11 and 12). A vessel running into the metaphysis distally in the coronoid fossa is exemplified in Figs. 10 and 12.

Fig. 15. Method of sawing specimens. (See text.)

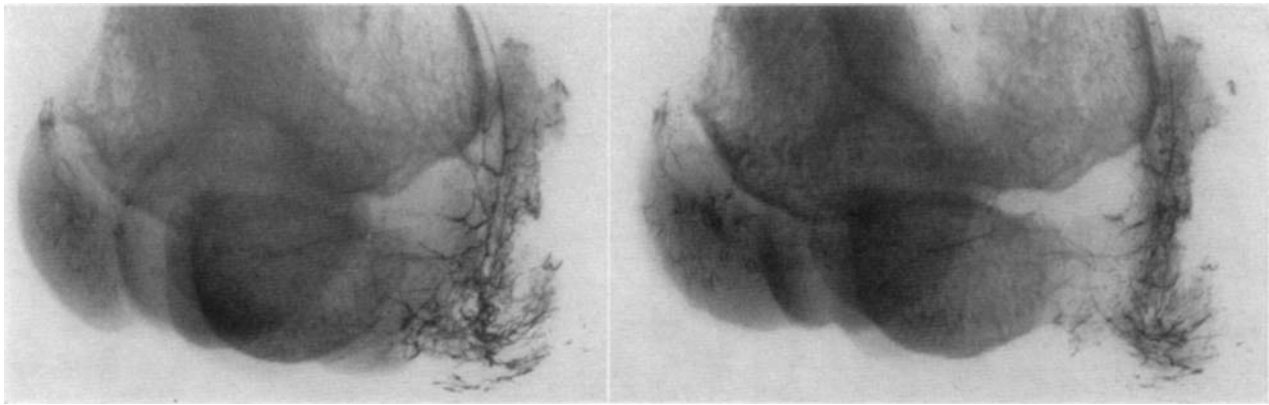


Fig. 16. *Specimen No. 21. — Age 8 years.*
Stereoscopic pictures of whole specimen with soft parts attached to back of capitulum.

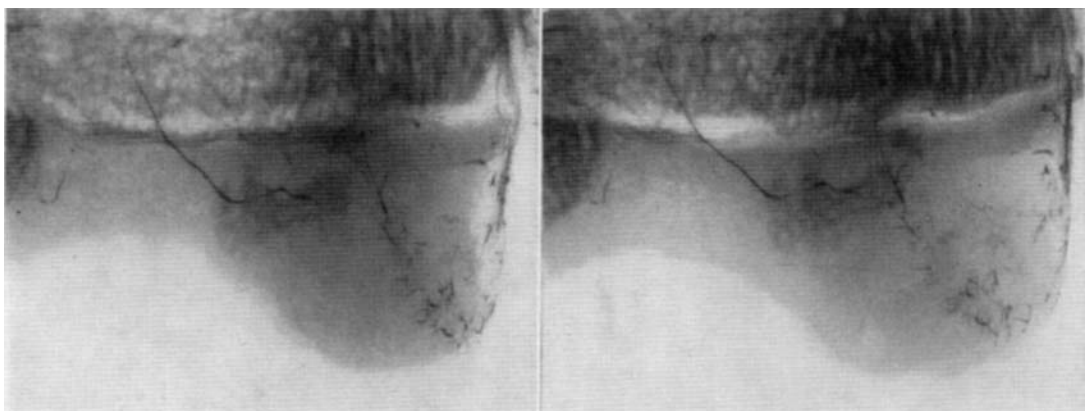


Fig. 17. *Specimen No. 21. — Age 8 years.*
Stereoscopic pictures of medial part of trochlea.

Figs. 10, 11, 12 and 13 illustrate a number of vessels extending from the diaphysis and dipping into the metaphysis where some of them communicate with metaphyseal vessels in the metaphyseal bone. The longest of these diaphyseal vessels descend almost down to the distal edge of the metaphysis (Figs. 10 and 13).

Figs. 10 and 11 show small intra-osseous metaphyseal vessels, apparently terminating within that margin of the metaphyseal bone facing the epiphyseal plate.

Specimen No. 21. Boy, aged 8 years. Radiopaque medium was injected 17 hours *post mortem* (Figs. 16 and 17). The specimen is not decalcified.

Fig. 16 was taken with soft tissue still attached to the posterior surface of the capitulum. The purpose was to show the extra-osseous course of the vessels of the ossific nucleus in the capitulum before they enter the posterior of the capitulum.

It is clear from Fig. 16 that 1 to 2 main vessels enter the chondroepiphysis from behind and afterwards enter the ossific nucleus of the capitulum. In this specimen, one of the vessels is a branch from a vessel coming from above and descending into soft tissues adjacent the posterior aspect of the humeral metaphysis and epiphysis. This vessel also gives off a branch to the distal end of the humerus proximal to the epiphyseal plate (Fig. 16). This branch thus enters the metaphyseal bone from behind. Branches from one and the same vessel thus run to the ossific centre of the capitulum and to the distal part of the humeral metaphysis.

Fig. 17 shows commencing multicentric ossification in the ulnar portion of the trochlear chondroepiphysis. This ossification centre in this specimen appears to be supplied by a vessel canal extending from the olecranon fossa and passing the posterior margin of the metaphyseal bone on its way into the chondroepiphysis. The vessel canal thus traverses the outer perimeter of the growth plate. The ossification centre is otherwise surrounded by avascular cartilage.

Specimen No. 18. Boy aged 9 years. Radiopaque medium was injected 10 hours *post mortem.* Right arm. Figs. 20 and 23 were taken before decalcification of the bone. Figs. 18, 19, 21 and 22 show the preparation after slight decalcification. The specimen sawn in the way described (Fig. 15) is illustrated in Figs. 19, 20, 21 and 22.

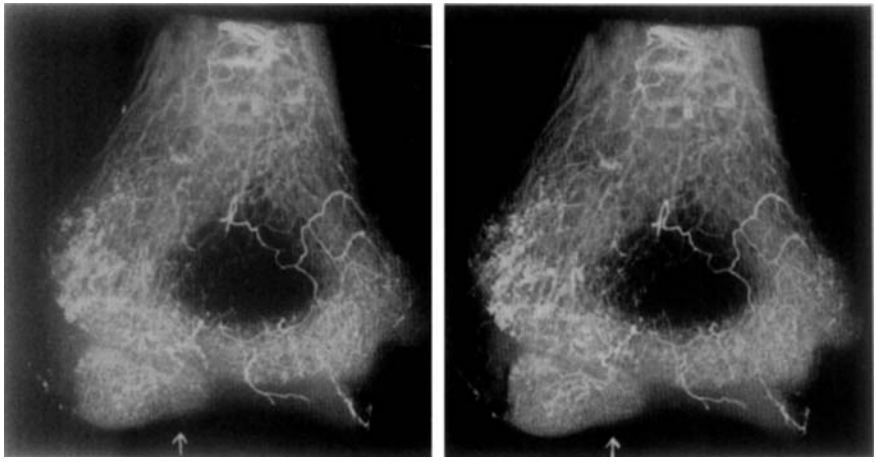


Fig. 18. *Specimen No. 18. — Age 9 years.*
Stereoscopic pictures of whole specimen.

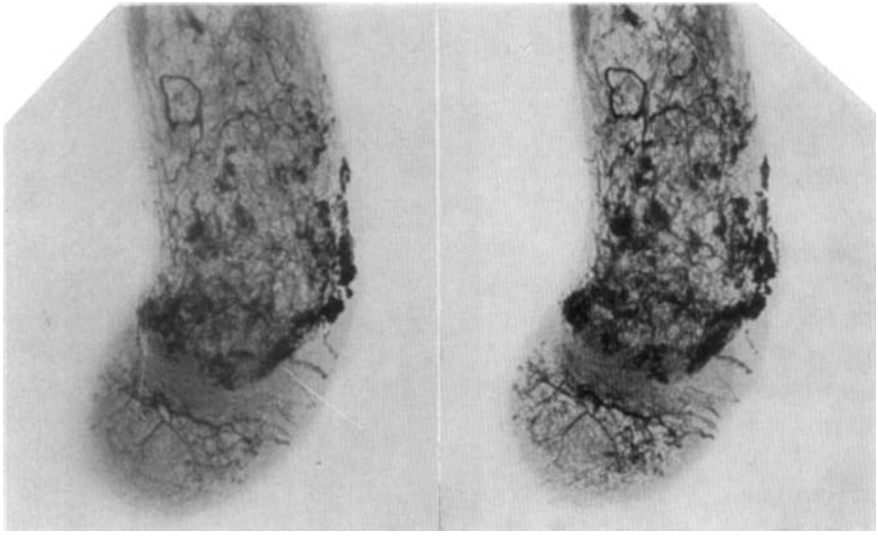


Fig. 19. *Specimen No. 18. — Age 9 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

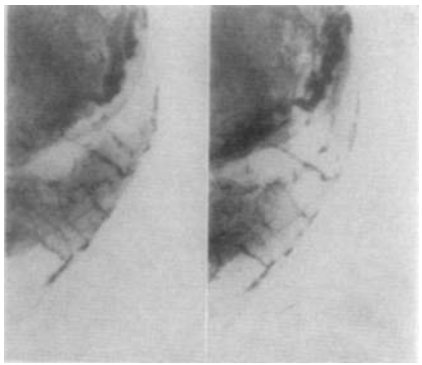


Fig. 20. *Specimen No. 18. — Age 9 years.* Stereoscopic pictures of isolated capitulum. Perichondrium *in situ* on back of epiphysis.

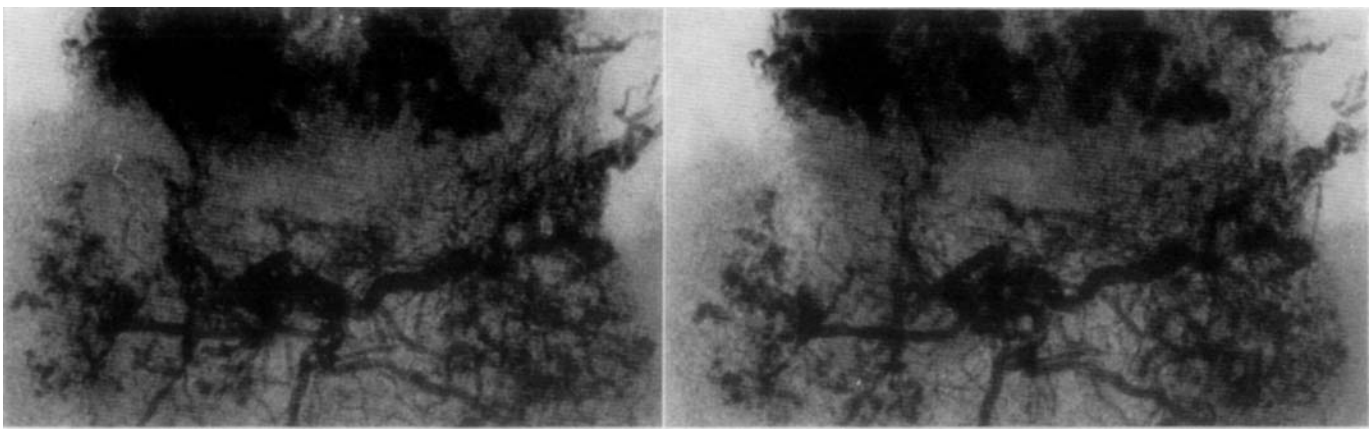


Fig. 21. *Specimen No. 18. — Age 9 years.*
Stereoscopic pictures of isolated capitulum showing vascular pattern adjoining growth plate.

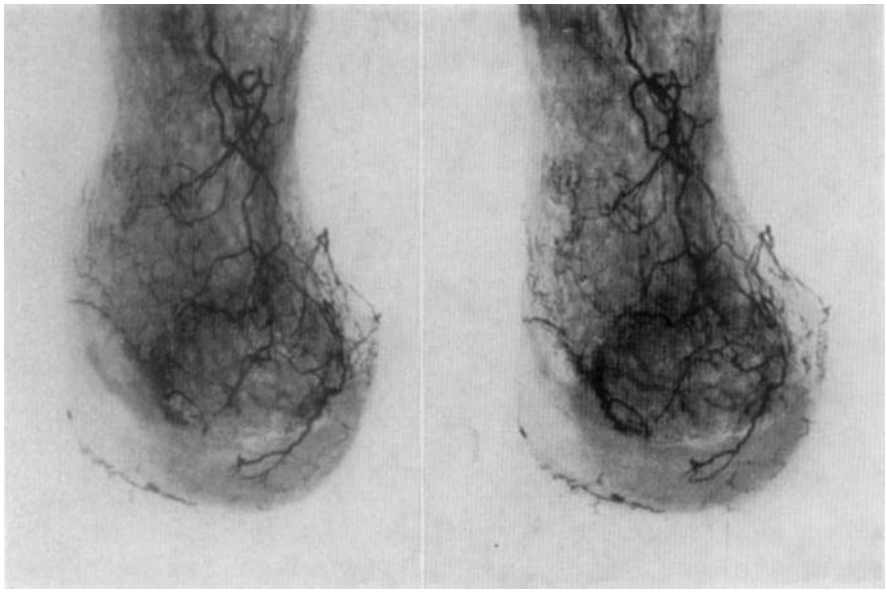


Fig. 22. *Specimen No. 18. — Age 9 years.*
Stereoscopic pictures of isolated trochlea and medial metaphysis.

Figs. 18, 19 and 20 give the impression that the ossification nucleus of the capitulum is supplied by 2 vessels coming from behind and intercommunicating within the ossific nucleus. These vessels enter the capitulum proximal to the posterior border of the joint cartilage and thus do not enter the joint cavity.

Figs. 18, 19 and 21 show not only the main vessels running to the capitulum from behind but also a vessel descending from the coronoid fossa, swinging round the ventral margin of the metaphyseal bone, traversing the anterior border of the epiphyseal plate and apparently reaching the ossific nucleus.

Judging by Figs. 18, 19, 20 and 21 the vascular system of the ossification nucleus of the capitulum, has not yet anastomosed with the vessels of the metaphysis or the rest of the epiphysis.

The ossific nucleus of the capitulum extends into the radial part of the chondroepiphysis of the trochlea (arrow in Fig. 18 indicates the crista lateralis trochleae, but this marks the border between the capitulum and the trochlea). Part of the trochlea is thus ossified from the ossification nucleus of the capitulum.

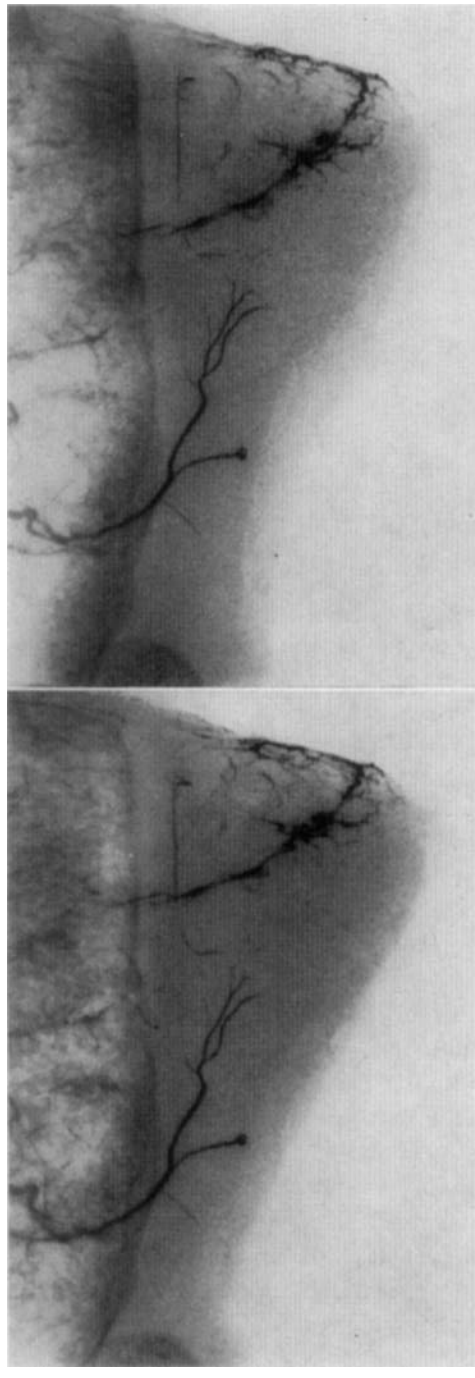


Fig. 23. *Specimen No. 18. — Age 9 years.*
Stereoscopic pictures of medial part of trochlea.

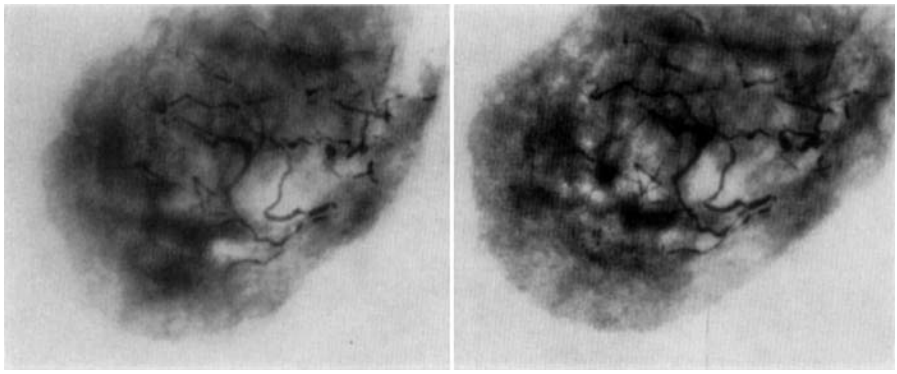


Fig. 24. *Specimen No. 20. — Age 19 years.*
Stereoscopic pictures of isolated capitulum humeri.

Figs. 18, 22 and 23 show a cartilage canal descending from the coronoid fossa and, after penetrating the ventral border of the growth cartilage, extends down into the trochlea. As is apparent from Figs. 18, 22 and 23, this vessel canal has several branches all ending blindly within the chondroepiphysis. One of these branches shows a loop-formed ending (Fig. 23). Some of the branches terminate at the future site of formation of the trochlea nucleus, as does a cartilage vessel canal, entering the chondroepiphysis from the ulnar surface of the latter (Figs. 18 and 23).

No calcified tissue is seen at the future site of the trochlear ossification nucleus in this specimen (Fig. 23).

Figs. 18 and 22 show large metaphyseal vessels which after entering the bone at the proximal and distal borders of the coronoid fossa, form a network in the metaphysis together with branches descending from the diaphysis.

Figs. 19 and 20 show 1—2 vessels, which after traversing the posterior part of the chondroepiphysis enter the osseous tissue of the metaphysis. Fig. 20, which was taken before removal of the perichondrium, gives the impression that the vessels running to the metaphysis and those extending to the ossific nucleus of the capitulum stem from one and the same main vessel.

Figs. 18, 19 and 21 show a filling of even small bone and cartilage vessels. Nevertheless, no vessel canals are seen in the central parts of the roentgenologically demonstrable epiphyseal plate.

These illustrations (Figs. 18, 19 and 21) also show the difference in the vascular pattern on the metaphyseal and epiphyseal sides of the epiphyseal plate. On the metaphyseal side the contrast appears with tuft-like endings against the cartilage. A similar vascular pattern is seen under the joint cartilage of the capitulum (Figs. 19 and 21). The intra-osseous vessels on the epiphyseal side of the epiphyseal plate, on the other hand, run parallel to the latter (Figs. 19 and 21).

Specimen No. 20. Man, aged 19 years. Radiopaque material was injected 63 hours *post mortem.* Right arm. The preparation is partly decalcified and is sawn in the way described.

As is apparent from Fig. 24 this preparation was the distal end of a full-grown humerus. The ossific centre of the capitulum has fused with the metaphyseal bone. The cartilaginous epiphyseal plate in this preparation has thus been replaced by osseous tissue.

Judging from these illustrations, the capitulum is supplied chiefly by two vessels entering the bone from behind and branching inside the bone. These vessels enter the bone posterior to the articular surface of the capitulum and thus do not appear to run intra-articularly.

In this preparation the vascular system in the capitulum is no longer isolated from the metaphysis, vessels now traversing the site of the earlier cartilaginous epiphyseal plate (Fig. 24).

Specimen No. 7. Woman, aged 28 years. Radiopaque material was injected 27 hours *post mortem.* Right arm. The preparation is partly decalcified. The specimen sawn in the way described is illustrated in Fig. 26.

It is clear from Figs. 25 and 26 that the distal end of the humerus has a rich intra-osseous vascular network in the epiphysis and in the metaphysis.

Here the vascular system of the capitulum is no longer isolated. But, as before fusion of the various ossification areas of the distal end of the humerus, the different vascular zones there can still be distinguished despite abundant intra-osseous communicating branches between these zones. One can thus distinguish vascular zones in the capitulum, trochlea and epicondyles, on one hand, and vasa nutritiae and the vascular system of the metaphysis on the other (Fig. 25).

It is clear from Fig. 25 that the vasa nutritiae together with the metaphyseal vessels form an anastomosing vascular network in the meta-

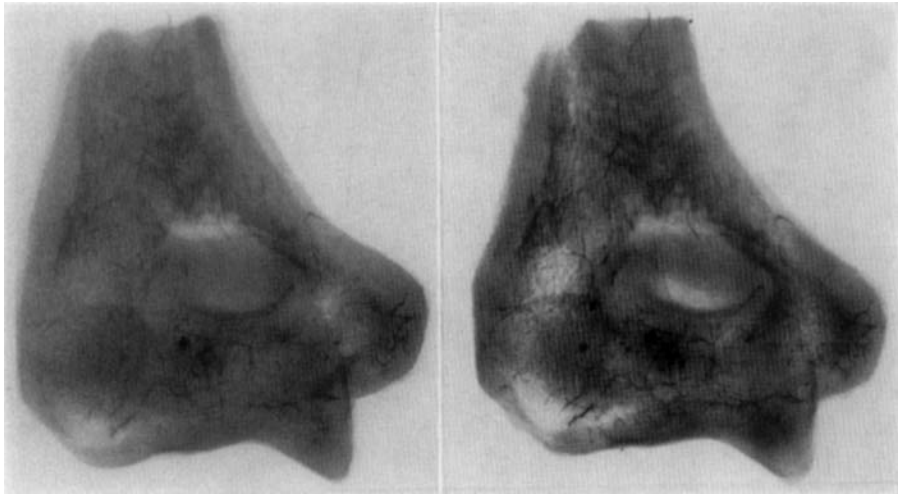


Fig. 25. *Specimen No. 7. — Age 28 years.*
Stereoscopic pictures of whole specimen.

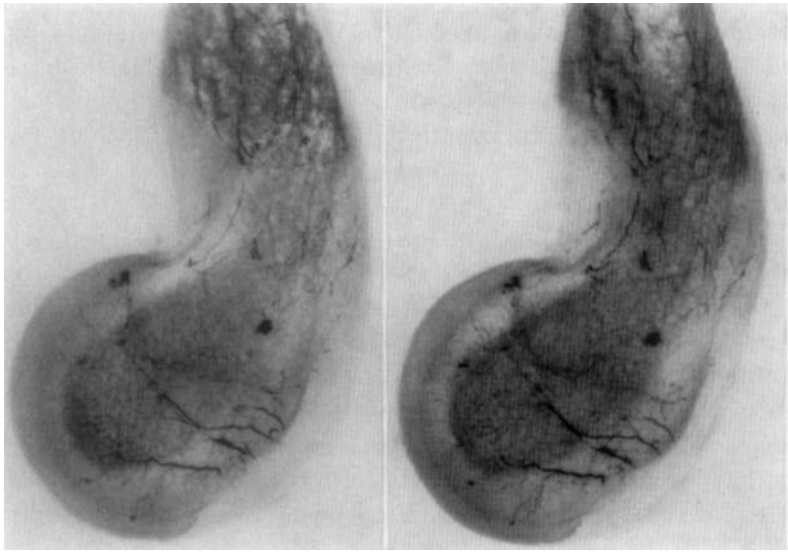


Fig. 26. *Specimen No. 7. — Age 28 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

physis. In the specimen illustrated this vascular system is in connection with the zone of epiphyseal vessels via intra-osseous communications crossing the site of the previous cartilaginous epiphyseal plate.

In Fig. 25 a relatively small vessel is seen to enter and branch within the ulnar part of the metaphysis. A few other small vessels also enter the surface of the metaphysis (Figs. 25 and 26). This specimen shows no large metaphyseal vessels of the caliber seen in the growing distal end of the humerus (Figs. 10, 11, 12, 18 and 22).

It is clear from Figs. 25 and 26 that the capitulum in this specimen is supplied mainly by 3 vessels coming from behind and entering the bone proximal to the joint surface of the capitulum to form a vascular network in the inside of the bone. As is apparent from Fig. 25, branches from the region of the vessels of the capitulum also arborise in that region of the radial part of the trochlea that has ossified from the ossific centre of the capitulum. The main vessels running to the ulnar part of the trochlea, which has been ossified from the ossification centre of the trochlea, appear to enter that part of the bone from the ulnar side and proximally (Fig. 25).

Figs. 25 and 26 show that the intra-osseous vessels underneath the joint cartilage are directed perpendicularly to this surface.

Specimen No. 10. A man, aged 52 years. Radiopaque medium was injected 40 hours *post mortem*. Right arm. Fig. 28 illustrates the specimen as sawn in the way described.

The intra-osseous vascular pattern in this specimen (Figs. 27 and 28) and in specimen No. 7 (Figs. 25 and 26) which is also from an adult, are strikingly similar.

Judging by Figs. 27 and 28 the capitulum receives its main blood supply from 2 vessels entering the bone from behind. These vessels enter the bone proximal to the joint surface of the capitulum and branch within the bone. Fig. 27, like Fig. 25, shows that the main vessel in the capitulum sends branches over into the radial part of the trochlea.

The ulnar part of the trochlea appears to be supplied mainly by vessels extending from its ulnar and proximal part (Fig. 27).

Fig. 27 shows a peculiar tortuous form of a large vessel descending from the diaphysis. Several vessels descend from the diaphysis and together with the metaphyseal vessels form a network in the metaphyseal spongiosa.

Figs. 27 and 28 show some vessels entering the surface of the meta-

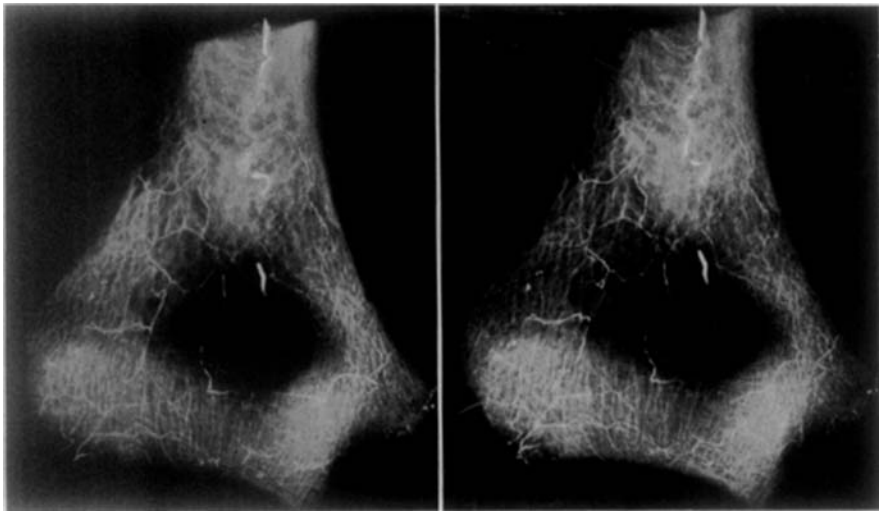


Fig. 27. *Specimen No. 10. — Age 52 years.*
Stereoscopic pictures of whole specimen.

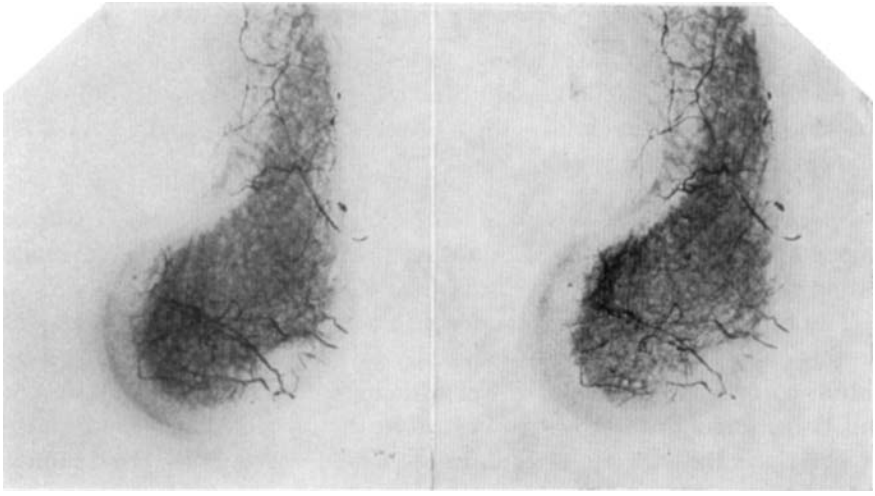


Fig. 28. *Specimen No. 10. — Age 52 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

physis. These metaphyseal vessels are relatively smaller than those seen in the distal end of growing humerus (Figs. 10, 11, 12, 18, 19 and 22).

Specimen No. 25. A woman, aged 75 years. Radiopaque medium was injected 19 hours *post mortem*. Right arm. Decalcified specimen. Sawn in way described, illustrated in Fig. 30.

This specimen shows the same topography of the intra-osseous vessels in the distal end of the humerus (Figs. 29 and 30) as the other specimens from adults (Figs. 24, 25, 26, 27, 28 and 31).

Although this preparation was from an aged woman, it contained wide vessels descending from the diaphysis down towards the metaphysis (Fig. 29). Relatively large vessels are seen entering the surface of the metaphysis (Figs. 29 and 30).

The capitulum is supplied by 2 main vessels entering the bone extra-articularly from behind and communicating with one another within the bone (Figs. 29 and 30). These vessels communicate intra-osseously with metaphyseal vessels and send off branches over to the radial part of the trochlea (Fig. 29).

Specimen No. 27. A man, aged 80 years. Radiopaque medium was injected 21 hours *post mortem*. Right arm. Not decalcified. Fig. 31 shows the preparation with soft tissues still attached to the posterior surface of the capitulum.

The capitulum appears to be supplied mainly from a descending extra-osseous vessel. The vessel branches outside of the bone and enters the capitulum from behind with 2 branches entering proximally to the margin of the joint cartilage.

Specimen No. 17. A woman, aged 73 years. Red acrylic mass was injected 36 hours *post mortem*. Right arm. Fig. 32 was taken after maceration of soft tissues.

The figure shows 2 vessels entering the capitulum from behind. The vessels enter the bone dorso-proximally to the joint surface of the capitulum corresponding to the site of attachment of the joint capsule and musculus anconaeus. The two vessels in this preparation are branches of arteria collateralis ulnaris inferior, which stems from the brachial artery proximally to the ulnar epicondyle and with its trunk forms a proximally convex arcade, crossing the dorsal surface of the distal end of the humerus dorsal to the olecranon fossa and communicating with

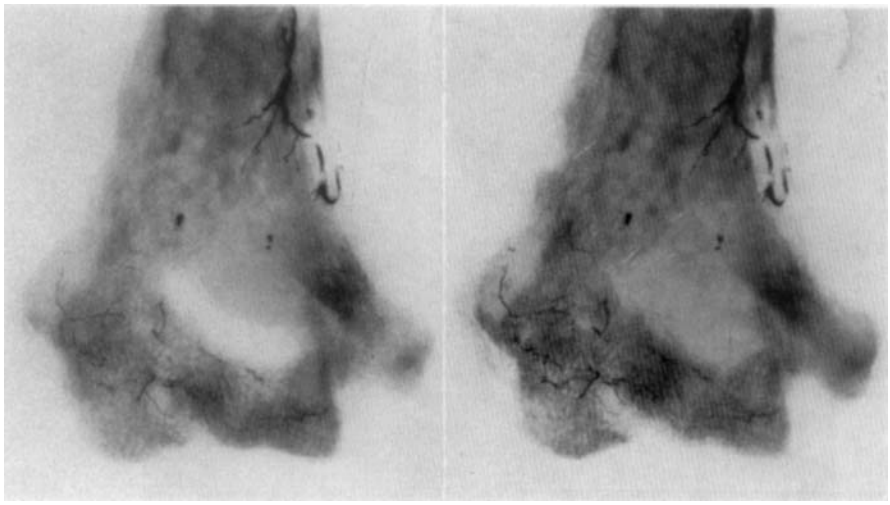


Fig. 29. *Specimen No. 25. — Age 75 years.*
Stereoscopic pictures of whole specimen.

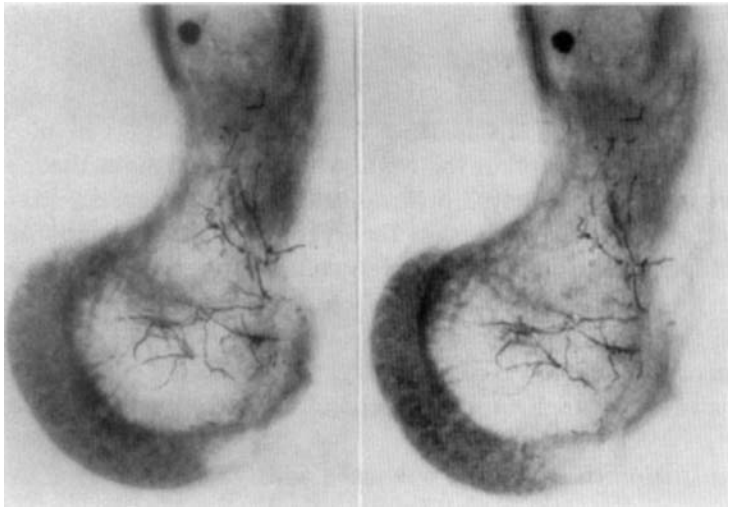


Fig. 30. *Specimen No. 25. — Age 75 years.*
Stereoscopic pictures of isolated capitulum and lateral metaphysis.

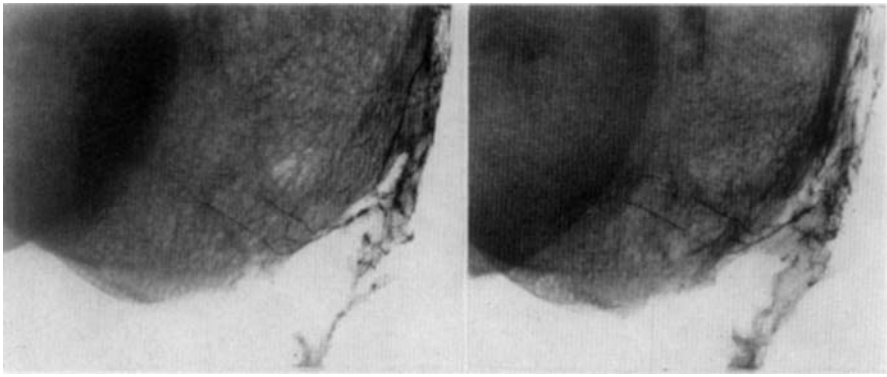


Fig. 31. *Specimen No. 27. — Age 80 years.*
Stereoscopic pictures of whole specimen with soft parts attached to back of capitulum.

the other vessels in the rete articulare cubiti. Fig. 32 thus shows communication with the arteria collateralis ulnaris superior and arteria collateralis media.

In this specimen the two branches, which enter the capitulum do not show direct communication with surrounding vessels (Fig. 32).

4. Discussion

A. *The intrachondral vasculature of the unossified distal chondroepiphysis of humerus*

The way in which the hyaline temporary cartilage in an unossified chondroepiphysis is nourished has long been the subject of discussion.

In an attempt to explain the route of nutrition, 2 main theories have been propounded, namely a diffusion theory and a vascular theory.

1. According to the diffusion theory the unossified temporary hyaline cartilage in the chondroepiphysis contains no vessels of its own and the nutrition and drainage of the chondrocytes occurs by diffusion through the intercellular substance.

2. According to the vascular theory, the unossified chondroepiphysis contains vessels, which supply the chondrocytes and remove their waste products.

The diffusion theory is the one more widely accepted. This theory is thus given preference in well-known standard textbooks on histology by various authors (SCHAFFER 1922, COWDRY 1934, PETERSEN 1935, BROMAN & HÄGGQVIST 1945, BUCHER 1948, STÖHR 1949, HAM 1953,

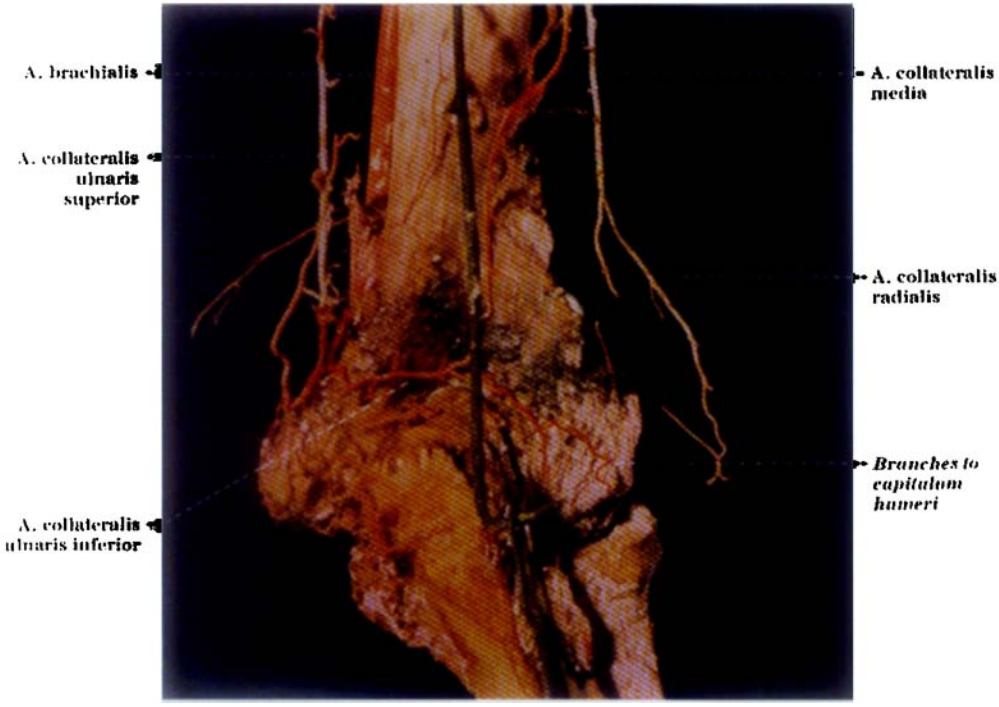


Fig. 32. *Specimen No. 17. — Age 73 years.*

Bones of right elbow-joint with acrylic casts of the extra-osseous vascular tree of the distal end of the humerus showing branches entering capitulum from behind.

BARGMANN 1956, MAXIMOW & BLOOM 1957, EINARSON, GLIMSTEDT & JANSEN 1958). This theory is also embraced by other authors (SIEGLING 1941, BACSICH & RIDDELL 1945, BERNBECK 1950 and STEIN, STEIN & BELLER 1955).

PHEMISTER (1940) and BERNBECK (1950) claim that the osmotically active hyaline cartilage tissue is nourished by diffusion of the synovial fluid, while BUCHER (1948), HAM (1953), MAXIMOW & BLOOM (1957) and EINARSON, GLIMSTEDT & JANSEN (1958) believe diffusion to occur from the vessels in the perichondrium.

Adherents of the diffusion theory are of the opinion that intrachondral vessels in the chondroepiphysis make their first appearance in connection with the development of the endochondral ossification process in the epiphysis.

The vascular theory. — Judging by the literature, this theory is less widely accepted.

The existence of vessels in the human chondroepiphysis was suggested in 1876 by LANGER.

Since then various investigators have described vessels in the unossified temporary cartilage in the epiphysis of long bones. In support of their opinions, they presented evidence produced by various methods such as serial sections and microscopy (HURREL 1935), serial sections and plastic reconstructions (HINTZSCHE & SCHMID 1933, HAINES 1934, 1937) and by injection technique (LEXER et al. 1904, v. FRIEDLÄNDER 1904, NUSSBAUM 1923, 1924, BELOU 1934, ANSEROFF 1934, WOLCOTT 1943, TRUETA 1957, HARALDSSON 1957 and TILLING 1958).

The present injection material included 6 specimens (Nos. 13, 29, 30, 21, 22 and 18, Table 11) permitting investigation of vascularisation of the unossified temporary cartilage in the chondroepiphysis of the distal end of the humerus.

Figs. 8, 9, 10, 11, 12, 13, 16, 18, 19, 20, 22 and 23 show the angiographic presence of vessels (Fig. 14) in the still unossified temporary cartilage in the epiphysis of the distal end of the humerus, but, according to BIDDER (1906), ECKERT-MÖBIUS (1924) and HURREL (1935), vascularisation of larger chondroepiphyses commences already in the 3rd to 4th month of intra-uterine life in man, such vessels arise as ingrowths from the vascular perichondrium (HINTZSCHE 1927, HURREL 1935), they present local submerges of the perichondrium (HAINES 1934, 1937).

Figs. 8, 9, 10, 12, 13, 18, 22 and 23 give the impression that these car-

tilaginous vessels stem from two origins, from the non-articular surface on the chondroepiphysis and from the surface of the already ossified metaphysis. This finding of epiphyseal vascular cartilage canals of different origin is in agreement with the opinions of BIDDER (1906) and KAJAVA (1919). TRUETA (1957) found a similar arrangement of intrachondral vessels in the unossified chondroepiphysis of the femoral head.

Figs. 8, 9, 10, 12, 18, 22 and 23 show that the cartilage vessels coming from above descend into the unossified chondroepiphysis, pass the ossifying metaphyseal border and traverse the edge of the zone of long growth, the epiphyseal plate. No direct communication is seen between the vascular system of the chondroepiphysis and the intra-osseous vessels in the metaphysis in these preparations. (See also Chapter IV, 4, F.)

Practically all of the vessel canals seen in the unossified chondroepiphysis in Figs. 8, 9, 10, 11, 12, 13, 18, 19, 22 and 23 appear to terminate blindly within the cartilage without communication with one another. This is in agreement with results obtained by BERTRAND (1923), NUSSBAUM (1923, 1924), ANSEROFF (1934) and TRUETA (1957). STUMP (1925), however, claimed to have observed inter-communicating cartilage canals in growing chondroepiphyses. LÖHR (1930) was unable to demonstrate cartilage vessels in the middle of unossified chondroepiphysis of the distal end of the humerus. Figs. 8, 9, 10, 11, 12, 13, 18, 22 and 23, however, show that the cartilage vessels extend to the middle of the massive chondroepiphysis.

In none of these preparations from growing individuals (Figs. 8, 9, 10, 11, 12, 13, 16, 18, 19, 22 and 23) were cartilage vessels seen in that part of the chondroepiphysis that persists unossified as joint cartilage in adult age. This is in agreement with the conclusions of ANSEROFF (1934) and TRUETA (1957).

It is thus clear from the injection experiments in the present investigation that vessels exist in the chondroepiphysis of the distal end of the humerus *before* epiphyseal ossification can be demonstrated roentgenologically in the corresponding part of the chondroepiphysis.

What is the nature of these intrachondral vessels? Are they the vessels of the cartilage tissue, which supply and drain the chondrocytes or are they signs ushering in an incipient ossification process?

TRUETA (1957) who studied intrachondral vessels in the unossified chondroepiphysis of the femoral head, believed these vessels to be related to the process of endochondral ossification. But TRUETA worked

with the chondroepiphysis of the femoral head, in which the ossification centre appears as a single ossific nucleus as early as 6 weeks to 6 months of age in girls and at 2—8 months in boys (CAFFEY 1956). The chondroepiphysis of the distal end of the humerus permits investigation of a more variable picture in this respect. Here the ossification of the chondroepiphysis occurs at different ages in different parts of the temporary cartilage. The ossific nucleus from which the capitulum and the radial part of the trochlea are ossified appears at the age of 1—26 months post partum in boys (ELGENMARK 1946) (Table 2, Fig. 3), while the ulnar part of the trochlea does not show signs of ossification until the age of 8—13 years in boys (HASSELWANDER 1931, Table 2, Fig. 3). In Figs. 8 and 9 intrachondral vessels are seen in both these regions of the unossified chondroepiphysis. Since the appearance of the ossific nucleus in the capitulum in Specimen 13 (Figs. 8 and 9) can be expected within the near future, it cannot be excluded that the vessels demonstrated in the chondroepiphysis of the capitulum and shown in Figs. 8 and 9 might have appeared in the cartilage for the preparation of the ossific nucleus of the capitulum and thus be due to the imminent endochondral ossification in that region. This also holds for the intrachondral vessel canals in the chondroepiphysis of the trochlea in Specimen 18 (Figs. 18, 22 and 23), an assumption which is supported on comparison between Fig. 23, where no roentgenologic signs of ossification are seen in the ulnar part of the trochlea and Fig. 17 where ossification in that location has already started.

But the situation must be different for the vessel canals in the ulnar part of the cartilaginous trochlea in the preparations from boys aged 3 months and 3 years (Figs. 8, 9, 10 and 12). What purpose do these cartilage vessels serve?

Their presence in these specimens can hardly be related to the ossification of this part of the chondroepiphysis, because they are present in the cartilage 8—13 years (Figs. 8 and 9) and 5—10 years (Figs. 10 and 12) before the appearance of the ossification nucleus in the ulnar part of the trochlea (Fig. 3). It therefore appears justified to conclude that these intrachondral vessels in the chondroepiphysis of the trochlea serve the same purpose as vessels in any tissues of the body, namely nutrition and removal of waste products from tissue cells, in this case the chondrocytes in the chondroepiphysis.

It is therefore concluded that the experiments have shown angiographically that the hyaline temporary cartilage in a growing chondro-

epiphysis has its own vessels for nutrition of this relatively large mass of cartilage.

In Specimens 29 and 30 (Figs. 10, 11 and 13) most of the numerous vascular canals in the unossified cartilage around the ossific nucleus of the capitulum appear to be independent and not to communicate with the ossific nucleus despite the advanced ossification of the latter. The ossific nucleus is supplied by only 1—2 vessel canals.

In Specimens 21 and 18 (Figs. 16, 18, 19, 20 and 23) most of the vessel canals around the ossific nucleus of the capitulum have disappeared except for the few vessel canals supplying the nucleus.

This suggests the conclusion that when the cartilaginous mass of the chondroepiphysis has been reduced by growth of the endochondral bone in the epiphyseal nucleus (nuclei), the vessels of the cartilaginous tissue disappear and only those vessels necessary for nutrition of the ossific nuclei persist in the cartilage, which they traverse on their way to the ossific nucleus.

These conclusions are in agreement with the findings of KAJAVA (1919), LASSILA (1931) and HAINES (1934, 1937) according to which part of the cartilage canals of the chondroepiphysis disappear in later stages of growth. They thus agree also with the view of ECKERT-MÖBIUS (1924), that the function of the cartilage canals is to supply large cartilages and with the opinion of HAINES (1934), HURREL (1935) and NONIDEZ & WINDLE (1949), who claimed that the primary function of cartilage canals is the nutrition of cartilages too large to be supplied by diffusion of nutriment through their substance.

It is not possible to decide on the basis of the present material whether the vessels seen in the chondroepiphysis in Figs. 8, 9, 10, 11, 12, 13, 16, 18, 19, 22 and 23 are arteries or veins. LANGER (1876), STOSS (1918) and HINTZSCHE (1927) speak of cartilage vessel canals containing an artery and vein with a capillary plexus between them that all lie in a matrix of connective tissue which serve as a base for their passage.

A loop-formed vessel-ending is seen in the end of one of the vessel canals of the trochlear chondroepiphysis in Fig. 23. TRUETA (1957) found similar terminal arrangements in cartilage vessels in the chondroepiphysis of the femoral head. He believed these to be capillaries joining the artery and the vein of the cartilage canal. He stated that this terminal arrangement was related to the preparation of the cartilage for ossification.

Recapitulation

1. The temporary hyaline cartilage in the chondroepiphysis of the distal end of the humerus contains several vessel canals before roentgenologic demonstration of ossification is possible in that part of the cartilage, where the canals may be demonstrated.

2. These cartilage vessels enter the chondroepiphysis partly from its non-articular surface and partly from the surface of the metaphysis. The last-mentioned vessels pierce the outer perimeter of the epiphyseal plate.

3. These cartilage vessel canals are not found to communicate with one another in the cartilage.

4. No vessels are found in that part of the chondroepiphysis facing the joint surface and corresponding to that part of the cartilage which in the adult bone persists unossified as joint cartilage.

5. Cartilage vessels were found in the ulnar part of the chondroepiphysis of the trochlea 8—13 years and 5—10 years before the time of appearance of the ossific nucleus in this part of the chondroepiphysis. The presence of these cartilaginous vessels therefore appears not to be due to any preparation for the ossification. It was thus roentgenologically demonstrated that the temporary hyaline cartilage in a growing chondroepiphysis has its own vessels for nutrition of the mass of cartilage

6. Some of the cartilage vessels found in the unossified chondroepiphysis of the capitulum in a 3 months old boy and in the unossified trochlear chondroepiphysis of a 9 year old boy may be regarded as playing a rôle in the imminent ossification of these regions.

7. As the mass of cartilage of the chondroepiphysis of the distal end of the humerus decreases in volume by growth of the ossification nuclei, other intrachondral vessels disappear except those supplying the ossific nuclei.

B. *Relation between appearance of bone nuclei in capitulum and trochlea and the vasculature*

No general agreement has been achieved on the relation between the appearance of bone nuclei in chondroepiphysis and the vasculature.

The most widely accepted theory of the appearance of an ossific nucleus in a chondroepiphysis is broadly as follows: At a time appropriate for each particular location the chondrocytes in the middle of the chondroepiphysis begin to hypertrophy and produce phosphatase.

After an increase of glycogen in the cytoplasm of the chondrocytes, calcification of the intercellular substance of the cartilage begins. This calcification results in nutrient materials previously able to diffuse through the matrix no longer being able to penetrate the calcified matrix, with consequent death of the chondrocytes situated in the calcified regions. During this stage capillaries grow into the calcified cartilage structure bringing osteoblasts and bone salts with them, with subsequent ossification (STEIN, STEIN & BELLER 1955).

According to this theory then, vessels do not enter an epiphyseal centre of ossification before the calcification of the cartilaginous intercellular substance (COWDRY 1934, PETERSEN 1935, BROMAN & HÄGGQVIST 1945, BUCHER 1948, STÖHR 1949, BERNBECK 1950, HAM 1953, STEIN, STEIN & BELLER 1955, BARGMANN 1956, MAXIMOW & BLOOM 1957, EINARSON, GLIMSTEDT & JANSEN 1958 and others). These authors suppose that the unossified temporary epiphyseal cartilage (chondroepiphysis) is avascular, so that ingrowth of vessels must occur at the time when the endochondral ossification process begins.

With reference to Figs. 8, 9, 10, 12, 18, 22 and 23 vessels are seen in all of these specimens in the temporary hyaline cartilage in the chondroepiphysis of the distal humerus at later sites of ossific nuclei. These vessels are present in the cartilage *before* bone salts deposited there have reached a concentration high enough to be demonstrable on the roentgenogram. Figs. 8 and 9 show vessels in those sites in the chondroepiphysis where the ossific centres of the capitulum, trochlea and epicondyles will later appear. Figs. 10, 12, 18, 22 and 23 show vessels at the sites, where the ossific nuclei of the trochlea and epicondyles will later appear. It is apparent from Figs. 10, 11, 13, 16, 18 and 19 that only a few of the many vessels in the chondroepiphysis will act as nutrient vessels to the ossific nucleus of the capitulum. Figs. 10, 11 and 13 show the ossific nucleus in a relatively richly vascularised cartilage but shows no contact with most of the cartilage vessels.

Figs. 8, 9, 10, 12, 18, 22 and 23 argue *against* the widely accepted opinion that the vessels supplying an endochondral epiphyseal ossification centre grow in in association with calcification of the latter. These illustrations show that the vessels are already present at the site in the chondroepiphysis where calcification and later ossification will occur.

It is widely believed that degenerative changes in the chondrocytes of the cartilage precede the actual ossification process in a chondroepiphysis.

Some authors (PARSONS 1905, CAREY 1922, ECKERT-MÖBIUS 1924, HARRIS 1933) believe that the degeneration of the cartilage that precedes the ossification is the result of lack of nutrition in the interior of the mass of cartilage.

Figs. 8 and 9, however, show numerous cartilage vessels located in the chondroepiphysis of the capitulum at the site where the ossific nucleus may be expected soon to appear. From Figs. 10, 11 and 13 it is clear that the ossific nucleus of the capitulum is situated in that part of the chondroepiphysis where cartilage vessels are richest.

These illustrations thus do not give the impression that the ossific nucleus of the capitulum appears in a zone of the cartilage that is less well nourished than the other parts of the chondroepiphysis of the distal end of the humerus, which will, however, become ossified later.

Figs. 18, 22 and 23 show 2 vessel canals extending to that location in the unossified chondroepiphysis of the trochlea, where ossification may soon be expected. (Preparation from a 9-year old boy.) These pictures convey the impression that the site in the chondroepiphysis of the trochlea, where the ossific centre of the trochlea will later appear is more richly supplied than the surrounding cartilage, which will later become ossified.

All these illustrations thus argue against the widely accepted opinion that lack of nutrition in the chondroepiphysis is the factor that precipitates endochondral ossification. These observations agree with HINTZSCHE's (1928) results obtained in a microscopic serial sections study of the chondroepiphysis of the distal end of the femur.

Recapitulation

1. Vessels in the distal humeral chondroepiphysis extend to the future site of the ossification nuclei of the capitulum and trochlea, before bone salts deposited there have reached a concentration high enough to be demonstrable in the roentgenogram.

2. The ossific nucleus of the capitulum appears in a region in the chondroepiphysis of the distal end of the humerus, which, judging by the present angiographic studies, is just as well vascularised as the other parts of the temporary cartilage, which will be ossified later. In a specimen from a 9-year old boy that region in the chondroepiphysis of the trochlea where the ossification centre may be expected soon to appear, was more richly vascularised than the surrounding cartilage which will not be ossified until later.

3. The observation set forth above argue against the hypothesis that the precipitating factor of endochondral epiphyseal ossification is lack of nutrition of the hyaline temporary epiphyseal cartilage (chondroepiphysis).

C. *The vasculature of the growing capitulum*

As early as 1876 LANGER described vessels supplying growing ossification centres.

Figs. 10, 11, 13, 16, 18, 19, 20 and 21 of specimens from 3, 8 and 9-year old boys show that the only vascular supply of real significance to the ossific nucleus of the capitulum are 1—2 vessel canals coming from behind and entering the capitulum dorso-proximally to the joint cartilage, at the site, where the joint capsule and the adjacent musculus anconaeus are attached on the dorsal surface of the distal end of the humerus. These vessels extend to the ossific nucleus after having traversed the unossified temporary cartilage.

The intracartilaginous course of these vessels is relatively long in specimens 29 and 30 (Figs. 10, 11 and 13) where the ossification of the chondroepiphysis is in a relatively early stage. The course of the vessels in the cartilage, on the other hand, is shorter in specimens 21 and 18 (Figs. 16, 18, 19, 20 and 21), where the ossific nucleus of the capitulum occupies almost the entire radial part of the chondroepiphysis.

Figs. 10 and 11 show aborisation of the vessels in the ossific nucleus and Figs. 18, 19 and 21 that they have formed an intercommunicating vascular network within the nucleus.

Thus, when ossification is well under way no anatomical end-vessels are seen in this ossification centre, which is in accord with the results of injection experiments in ossification centres of other chondroepiphyses performed by such investigators as NUSSBAUM (1923, 1924), HARRIS (1933), ANSEROFF (1934), DE MARNEFFE (1951) and TRUETA (1957).

In Specimens 30 and 18 (Figs. 13, 18, 19 and 21) a small vessel is seen in the anterior margin of the epiphyseal plate. This vessel extends from the ventral surface of the metaphysis and extends down into the anterior part of the ossific nucleus of the capitulum. Judging by Figs. 13 and 19, it establishes no direct communication between the intraosseous metaphyseal vessels and the capitulum, and its course is limited to a small area in the anterior margin of the ossific nucleus. Similar vessels have been described by TRUETA (1957) in association with the

ossific nucleus of the developing femoral head but they disappeared during the course of ossification and thus represented persistent cartilage vessels from the infantile period of development and not nutrient vessels proper of the ossific nucleus.

Judging by specimens Nos. 29, 30, 21 and 18 (Figs. 10, 11, 13, 16, 18, 19, 20 and 21) in these ages the vascular system of the ossific nucleus of the capitulum is not in direct communication with the intra-osseous vessels in the metaphysis or with the vessels in the rest of the distal humeral chondroepiphysis. Thus, although the vessels of the ossification centre of the capitulum are not anatomic end-vessels proper, they may be regarded as functional end-vessels during the period of growth because of the anatomic isolation of this vascular system.

Recapitulation

1. As long as the ossific nucleus of the capitulum of the humerus lies isolated in the chondroepiphysis, it appears to receive its blood supply via a few vessels entering the cartilage from behind and breaking up into a vascular network within the nucleus of ossification.

2. These vessels have a relatively long intrachondral course as long as the ossific nucleus is small, which, however, becomes shorter with increase in size of the nucleus.

3. During the period of growth the vessels of the nucleus (of ossification) are not in direct communication with the intra-osseous vessels of the metaphysis or with cartilage vessels in the surrounding non-ossified part of the distal humeral chondroepiphysis.

4. The vessels of the ossification centre of the capitulum are not anatomic end-vessels, but owing to their anatomic isolation during the period of growth, they may be regarded as functional end-vessels during that period.

D. *Vasculature of distal end of full-grown humerus, especially of capitulum*

On comparison of preparations from adults (Figs. 24, 25, 26, 27, 28, 29, 30 and 31) with preparations from growing persons (Figs. 10, 11, 13, 16, 18, 19 and 20) the vascular topography of the vessels in the full-grown capitulum appears to be essentially the same as that in growing individuals before bony union of the ossification centre of the capitulum with other nuclei in the epiphysis and to the metaphysis.

All of these illustrations as well as the other angiograms taken in the experimental study thus showed that the capitulum receives its main blood supply from 1—3 vessels entering from behind. Figs. 25, 27 and 29 show that the main vessel in the full-grown capitulum sends branches over into the radial part of the trochlea ossified from the ossific nucleus of the capitulum.

The remaining ulnar part of the full-grown trochlea receives its blood supply mainly from vessels entering the trochlea proximally and ulnarly (Figs. 25, 27 and 29). Thus, the vascular topography of this part of the bone is also essentially the same in adult age as during childhood (Figs. 10, 12, 17, 18, 22 and 23).

Figs. 24, 25, 26, 27, 28, 29 and 30 show that after bony union in the distal end of the humerus, the isolation of the capitulum vasculature is broken by intra-osseous vessels, which establish communication with the rest of the epiphysis and the metaphysis. Now the blood vessels in the epiphysis, metaphysis and diaphysis intercommunicate.

Judging from the appearance of the adult specimens (Figs. 24, 26, 28, 30 and 31) and those from 3, 8 and 9-year old children (Figs. 11, 13, 16, 19, 20 and 21) it appears that the main vessels of the ossific nucleus of the capitulum running parallel to the epiphyseal plate have not become more distant from the site of the epiphyseal plate in the bone during the period of growth. This observation supports the theory of NUSSBAUM (1924), PAYTON (1933), SIEGLING (1941), MAU (1958) and others that an epiphyseal ossification centre grows by new-formation of bone under the articular surface.

As mentioned, in the present experimental investigation the vascular topography of the capitulum was found to be essentially the same in the growing ossific nucleus as in the full-grown bone. This might be explained by the relation between the attachment of the capsule and the epiphyseal plate on the dorsal surface of the capitulum, which has been discussed in chapter II (Fig. 5). Because of this relationship the vessels coming from behind and entering the ossification centre of the capitulum have only a relatively narrow, limited region on the dorsolateral surface of the capitulum to choose for their entry into the epiphysis. This region is limited proximally by the barricade of the epiphyseal plate and distally and medially by the dorsal edge of the joint cartilage, where the joint capsule is attached (Fig. 5). The range of possibilities for the entrance from behind of the capitular vessels is therefore not so wide as for the nutrient vessels of the diaphysis of the

humerus, for example. The result is that while the site of entrance of the nutrient vessels of the humeral diaphysis varies (LÜTKEN 1950), the vasculature in the capitulum is remarkably constant.

Figs. 25, 26 and 27 show that the intra-osseous vessels situated nearest under the avascular joint cartilage are perpendicular to the joint surface. They appear to end abruptly. This is in accord with the results of histologic examinations on rabbits by HOLMDAHL & INGELMARK (1951).

As is apparent from Figs. 24, 25, 26, 27, 28, 29 and 30, this experimental investigation showed that although anastomoses have developed between the vascular system of the capitulum and the other parts of the epiphysis as well as between the vascular system of the epiphysis and metaphysis in the full-grown bone, it is still possible to distinguish between the main vascular regions in the epiphysis, metaphysis and diaphysis, seen before the bony union of the different parts of the distal end of the humerus. These findings agree with the observations of ANSEROFF (1934), who worked *inter alia* with the distal end of the humerus, and TRUETA & HARRISON (1953) and HULTH (1956) who worked with the proximal femur.

On comparison between the vasculature during the period of growth (Figs. 8, 9, 10, 18, 19, 21 and 22) and that in the full-grown bone (Figs. 24, 25, 26, 27, 28, 29 and 30), it will be seen that the vasculature of the metaphysis has decreased after conclusion of growth in length of the bone. The vasculature of the metaphysis, which is richer during the period of growth, is thus probably related to growth in length and will be discussed further under E (vide infra). The present investigation revealed no definite evidence that the remaining intra-osseous vasculature (epiphysis and vasa nutritia system) undergoes any appreciable regression after the period of growth. It was therefore not possible to support ANSEROFF's (1934) opinion of the regression of the epiphyseal vasculature after the end of growth in length.

The full-grown bone showed no definite signs of decreased calibre of the intra-osseous vessels with advancing age (Figs. 24, 25, 26, 27, 28, 29, 30 and 31). This argues against the assumption of CHEYNEL (1947), WEINMANN & SICHER (1947) and others, but agrees with the findings of TRUETA & HARRISON (1953) and JUDET, JUDET, LAGRANGE & DUNOYER (1955) in their investigation of the vasculature in the proximal end of the femur.

Recapitulation

1. The topography of the vascular system forming in the capitulum and trochlea during the period of ossification persists practically unchanged in the adult bone.

2. After the ossification of the cartilage between the various ossific nuclei of the epiphysis and of the epiphyseal plate, the intra-osseous blood vessels of the epiphysis, metaphysis and diaphysis intercommunicate.

3. The main vessels of the capitulum do not shift away from the region of the site of the epiphyseal plate during the period of growth which supports that an epiphyseal ossific nucleus grows by new-formation of bone under the articular surface and not from the epiphyseal plate.

4. The vascular topography of the capitulum is strikingly constant. This may be due to the anatomic relationship between the attachment of the joint capsule at the dorsal edge of the joint cartilage and the epiphyseal plate on the dorsal aspect of the capitulum, which leaves only a limited zone free for vessels from behind to enter the ossific nucleus of the capitulum.

5. Despite anastomoses between different vascular systems of the distal end of the full-grown humerus, it is still possible to distinguish between the main vascular regions seen before the bony union between the different parts of the end of the bone.

6. The calibre of intra-osseous vessels in the full-grown bone was not found to decrease with advancing age.

E. *Metaphyseal and diaphyseal vessels in distal end of growing and full-grown humerus*

Wide vessels (Figs. 10, 11, 12, 18, 19 and 22) and fairly wide vessels (Figs. 16, 25, 26, 27, 28 and 30) enter the metaphysis of the growing and fully grown bone.

In injection experiments on the humerus LAING (1956) was able to demonstrate only minute transcortical vessels entering the bone in this region.

During the period of growth, these intra-osseous metaphyseal vessels do not appear to be in direct communication with the intra-chondral-intra-osseous vascular system of the epiphysis, but form a metaphyseal intra-osseous vascular net together with descending vessels running down from the diaphysis as well as minute transcortical vessels from

the periosteum (Figs. 10, 11, 12, 18, 19 and 22). This is in accord with ANSEROFF's (1934) findings in injection experiments on the humerus.

The experimental study also showed that in addition to the above mentioned communication between the different vascular regions occurring in the full-grown bone, the main difference between the intra-osseous vasculature in the growing and in the fully grown distal end of the humerus, is mainly the larger absolute and relative blood supply of the metaphysis during the period of long growth of the bone (Figs. 8, 9, 10, 11, 12, 18, 21 and 22) than after conclusion of this growth in length (Figs. 24, 25, 26, 27, 28, 29 and 30). This rich vascularisation of the metaphysis, which appears to be bound only to the period of long growth of the bone, suggests the conclusion that it is related to the process of long growth, which thus apparently occurs from the metaphyseal side of the epiphyseal plate because no corresponding difference was seen between the intra-osseous vasculature of the epiphysis of the growing and of the full-grown bone (Figs. 10, 11, 13, 18, 19, 24, 25, 26, 27, 28, 29, 30 and 31).

The above conclusions on the relation between the growth in length of the epiphyseal plate and the vasculature of the metaphysis agree with those of other research workers, *e.g.* BRODIN (1955) and TRUETA (1957).

Specimens 21 and 18 (Figs. 16, 18, 19, 20 and 21) show vessels going proximal to the dorsal border of the epiphyseal plate and entering the spongiosa of the metaphysis from behind. Specimens 29 and 18 (Figs. 10, 12, 18 and 22) show vessels entering the metaphysis distally in the coronoid fossa.

In specimens 21 and 18 (Figs. 16 and 20) the vessels coming from behind to the metaphysis and vessels coming from behind to the ossification centre of the capitulum, appear to be branches of one and the same main vessel.

In these specimens then, the same main vessels supply the ossific nucleus of the capitulum and a circumscript region in the metaphysis. These findings may be of significance in the development of certain pathologic metaphyseal changes in osteochondrosis deformans juvenilis capituli humeri discussed in Chapter IV, 5 and in Part III (page 168).

A similar arrangement with epiphyseal and metaphyseal vessels stemming from one and the same extra-osseous vessel has been described in the growing distal femur by NUSSBAUM (1923) and in the growing proximal end of the femur by HARTY (1953) and HULTH (1958).

The large vessels shown in Figs. 10, 11, 12, 18 and 22 and entering the metaphysis proximally to the coronoid fossa may play a certain rôle in the nutrition of the metaphysis in the event of impairment of its blood supply from the vasa nutritia as in fractures, for example. Many trans- and supracondyle fractures of the humerus in children, however, occur distally to the entrance of these vessels into the bone, and so interrupt the blood supply via these vessels to the distal part of the metaphysis.

The juxtaepiphyseal metaphyseal vessels shown in Figs. 16, 18, 19, 20 and 21 and coming from behind and those shown in Figs. 10, 12, 18 and 22 and coming from the front will, however, owing to their distal position, probably often escape being damaged by most trans- and supracondyle fractures. These vessels must therefore be of importance for the nutrition of the distal metaphyseal fragment in trans- and supracondyle fractures of the humerus in children.

Figs. 8, 9, 18, 19, 20, 21, 25, 26, 29 and 30 show irregular opacities formed by the injection mass in the metaphysis. Similar accumulations of contrast medium in the metaphysis have been described by REICHEL (1947), who believed them to be signs of vessels, that had been ruptured by the injection pressure. TRUETA & HARRISON (1953) were of the opinion that metaphyseal irregular accumulation of contrast medium represents sinusoids of the red marrow, that had been filled with contrast mass with consequent extravasation of the latter.

Figs. 8, 9, 10, 11, 12, 13, 18, 19, 22, 25, 27 and 29 show numerous vessels coming from the diaphysis and descending into the metaphysis, where they communicate with the metaphyseal vessels. Figs. 10 and 13 show that some of these vessels, which appear to be branches of rami descendentes of the nutrient vessels of the humerus, extend almost down to the cartilaginous epiphyseal plate. This finding appears to disprove LEXER's (1904) and DRAGENDORFF's (1931) opinion that nutrient vessels do not usually extend to the epiphyseal plate, but supports the studies of LEWIS (1956), which were, however, carried out mainly on experimental animals. TRUETA & HARRISON (1953) did not find branches of the nutrient arteries to extend up into the metaphysis of the proximal end of the full-grown femur.

Specimens 33 and 10 in the present material show a peculiar coiled arrangement of the main branch of the descending part of the nutrient vessels (Fig. 27).

REICHEL (1947) found a similar arrangement of the descending branch

of the femoral nutrient vessels in rats. TRUETA & HARRISON (1953) described a similar finding in the epiphyseal and metaphyseal vessels in the proximal end of the human femur; LAING (1956), at the beginning of the ascending branch of the nutrient artery of the humerus and TILLING (1958); in the ascending and descending branches of the nutrient arteries of the growing tibia, both in man. As to this coiled arrangement of vessels, it is not possible to say with certainty whether it represents their natural shape or whether it is due to the injection pressure.

TRUETA & HARRISON (1953) suggested that there might be a correlation between this shape of vessels and haemopoiesis, but Fig. 27 shows that the coiled arrangement of the vessel is limited to the yellow marrow and thus not to any segment where haemopoiesis occurs in this age (Specimen from man, aged 52 years).

Recapitulation

1. Intercommunicating intra-osseous vessels were seen in the metaphysis of the growing and full-grown bone. This vascular net in the growing bone consisted of communications between the vasa nutritia, relatively large metaphyseal vessels, and minute transcortical vessels, while the adult metaphyseal bone showed communication also with the epiphyseal vascular system.

2. The main difference between the intra-osseous vasculature in the growing and in the adult distal end of the humerus is, that in addition to the development of epiphyseal-metaphyseal communications in adults, the calibre of the vessels in the metaphysis is larger during the period of long growth both absolutely and relatively than after conclusion of growth in length. This suggests the rôle of the metaphyseal network in the long growth and relates it to the metaphyseal side of the epiphyseal plate because no corresponding difference was found between the vasculature of the epiphysis in the developing and full-grown bone.

3. Vessels running distally of the epiphyseal plate to the ossific nucleus of the capitulum humeri and vessels running proximally of the epiphyseal plate to the metaphyseal bone may be branches of one and the same main vessel.

4. The largest metaphyseal vessels seen in the distal end of the growing humerus enter the bone in such a proximal area that they must be damaged together with the branches of the nutrient vessels in the event of many trans- or supra-condyle fractures of the humerus in children. In these fractures, the juxta-epiphyseal vessels of the metaphysis must

therefore play an important rôle in the nutrition of the distal metaphyseal fragment.

5. Intra-osseous, irregular opacities due to the injection mass were seen. Whether these masses represented ruptured metaphyseal vessels or contrast-filled sinusoids of the red marrow is not known.

6. Branches of the nutrient vessels of the humerus were seen to descend and extend almost to the cartilaginous epiphyseal plate.

7. A descending branch of the nutrient vessels in two specimens showed a peculiar coiled arrangement. This was limited to a segment in the bone, where haemopoiesis does not occur in adult age.

F. *On vessels traversing the epiphyseal plate*

In specimens from boys aged 3 months, 3, 8 and 9 years (Figs. 8, 9, 10, 12, 13, 17, 18, 19, 21, 22 and 23) vessels were seen coming from above and entering the epiphysis of the distal humerus at the ventral or dorsal margin of the ossified metaphysis. These vessels came from the surface of the bone in the coronoid fossa and radial fossa (Figs. 10, 12, 13, 18, 19, 21, 22 and 23) and olecranon fossa (Figs. 8, 9, 10, 12 and 17).

On their descent to the chondroepiphysis these vessels traverse the ventral, or dorsal, outer perimeter of the epiphyseal plate (growth plate).

In a specimen from a 3 month-old boy (Figs. 8 and 9) the vessels from the olecranon fossa appear to traverse the metaphyseal bone deep into the latter and pierce the epiphyseal plate near its centre. It should, however, be borne in mind that these vessels enter the distal end of the humerus in the mid-line, *i.e.* at the narrowest part of the bone in the olecranon fossa (Fig. 9). In lateral roentgenograms the more lateral, thicker parts of the metaphyseal bone will be superimposed on the site of entry. These vessels are thus situated at the posterior margin of the metaphyseal bone in the olecranon fossa, which agrees with the course of the vessels from the olecranon fossa, seen in a specimen from a 3-year old boy (Figs. 10 and 12) and from an 8-year old boy (Fig. 17).

This also holds for the epiphyseal vessels from the coronoid fossa in the developing bone (Figs. 12, 19 and 22), *i.e.* that in lateral views thicker metaphyseal bone will be superimposed on these vessels which will then appear to be situated deeper in the margin of the metaphyseal bone than they really are. (Compare the stereoscopic reproductions in Fig. 19.)

Figs. 8, 9, 10, 11, 12, 13, 16, 17, 18, 19, 20, 21, 22 and 23 of specimens from growing persons show no signs of a direct intercommunication between the intra-osseous vascular network in the metaphysis on one hand and the vessels of the ossific nucleus of the capitulum and the vascular system of the unossified chondroepiphysis on the other. Such intercommunications were not seen until after ossification of the epiphyseal plate (Figs. 24, 25, 26, 27, 28, 29 and 30). The findings in these specimens were thus not in accord with those of LANGER (1876), LEXER, KULIGA & TÜRK (1904), LEXER (1903, 1904), KOLODNY (1925), WOLCOTT (1943), TILLING (1958) and BROOKES (1958), who after injection experiments in man claimed to have observed direct transepiphyseal vascular communication between vascular systems in the epiphysis and metaphysis of growing long bones.

The findings in the present specimens did, however, agree with the results of injection experiments of other workers in this field such as v. FRIEDLÄNDER (1904), BERTRAND (1923), ELETTO (1933), ANSEROFF (1934), PELLEGRINI (1934) and BELOU (1934), who injected various masses into the distal end of the growing humerus, and NUSSBAUM (1923, 1924, 1926), HARRIS (1933), TUCKER (1949) and HULTH (1958), who carried out similar experiments on the growing human femur.

None of these investigators found any direct vascular communication between the growing epiphysis and the metaphysis through the cartilaginous epiphyseal plate.

TRUETA (1957), who in injection experiments studied the vasculature of the human femoral head during growth found, in children under 4 years of age, "foetal ascending metaphyseal arteries" coming from the "top of the ossifying border" and running into the chondroepiphysis and to the ossific nucleus of the femoral head, after the nucleus had appeared. Examination of the reproductions given by TRUETA (1957) will show that they are taken in one plane and that they are not stereoscopic. It is therefore not possible to draw any valid conclusion from these reproductions as to whether these "foetal ascending metaphyseal arteries" establish any direct communication between the intra-osseous vascular systems in the epiphysis and metaphysis through the epiphyseal plate.

On the basis of histologic studies various authors have tried to demonstrate the existence of communicating vessel canals between the epiphysis and metaphysis in growing long bones, but only the results of the investigators working with serial sections can be accepted as reliable. HINTZSCHE & SCHMID (1933) worked with serial sections and

plastic reconstructions of cartilage canals in chondroepiphyses in the bones of human foetuses. They found no communicating canals between the vascular system in the epiphysis and metaphysis of the distal end of the humerus. In the proximal end of the humerus they found "die Knorpel-Knochengrenze durchbohrenden Gefässkanäle" without, however, being able to show any direct transepiphyseal vascular communication between the vessels of the chondroepiphysis and the intra-osseous vascular system of the metaphysis. This also holds for the findings of HAINES (1934). He worked with serial sections and celluloid reconstructions. HAINES claimed that by the time of ossification of the chondroepiphysis the communicating canals have disappeared in man. HURREL (1935) described *inter alia* in the distal end of the humerus in human embryo, cartilage canals running from the chondroepiphysis through the zone of proliferating cartilage cell rows (epiphyseal plate). That part of these canals which ran towards the border of the metaphyseal bone was, however, nearly always entirely closed, thus without vascular communication with the intra-osseous vascular system of the metaphysis. HURREL therefore concluded that a connection between the canals in the chondroepiphysis and the metaphyseal bone are "accidental and functionless, brought about by extension of the shaft ossification into a region already invaded by canals".

Recapitulation

1. The growing epiphysis of the distal end of the humerus contains vessels, which traverse the outer perimeter of the epiphyseal growth plate both before and after ossification has begun in the chondroepiphysis.

2. In the present experiments angiography of the distal end of the humerus of growing individuals aged 3 months to 9 years, showed no definite signs of direct vascular communication through the epiphyseal plate between the vasculature in the epiphysis of the distal end of the humerus and the intra-osseous vascular network of the metaphysis.

G. *Vasculature adjoining epiphyseal growth plate*

Judging by observations made in the present investigation, the pattern of the vessels adjoining the cartilaginous epiphyseal growth plate on the metaphyseal side differs from that of the vessels adjoining the epiphyseal side of the growth plate.

It is thus clear from Figs. 18, 19 and 21 that the part of the metaphyseal vascular net bordering the cartilaginous growth plate shows a brush-like pattern towards the cartilage. Figs. 10, 11, 22 and 23 show intra-osseous metaphyseal vessels, which end abruptly short of the growth plate. Similar terminal vascular patterns are seen underneath the joint cartilage in the ossific nucleus of the capitulum (Figs. 18, 19 and 21).

On the other side of the epiphyseal plate, the epiphyseal side, the intra-osseous vessels show a different pattern. There they are more or less parallel to the epiphyseal plate (Figs. 11, 18, 19 and 21), sometimes they form small arcades.

Angiography showed no vessels in the central parts of the roentgenologically defined cartilaginous epiphyseal plate (Figs. 10, 11, 12, 13, 16, 17, 18, 19, 20, 21, 22 and 23).

In many respects the present findings regarding the relation of vasculature to the epiphyseal cartilaginous growth plate agree with those described by other workers in this field (NUSSBAUM 1923, LASSILA 1931, HARRIS 1933, ANSEROFF 1934, LEWIS 1956 and TRUETA 1957).

Recapitulation

1. The pattern of the vasculature adjoining the metaphyseal side of the cartilaginous epiphyseal growth plate in the distal humeral end is different from the intra-osseous vascular pattern adjoining the epiphyseal side of the plate.

2. The vasculature adjoining the metaphyseal border of the epiphyseal plate shows a brush-like pattern towards the cartilage.

Adjoining the epiphyseal side of the plate, were vessels, which ran parallel to the plate, sometimes in the form of arcades.

3. Angiography showed no vessels in the central parts of the roentgenologically defined cartilaginous epiphyseal plate.

H. *Extra-osseous topography of vessels of growing and full-grown capitulum*

On the basis of injection experiments and anatomic studies LÖHR (1930) believed that the capitulum was supplied mainly by branches given off by the arteria recurrens interossea and the arteria collateralis media (Fig. 33). In the reproductions accompanying his article, how-

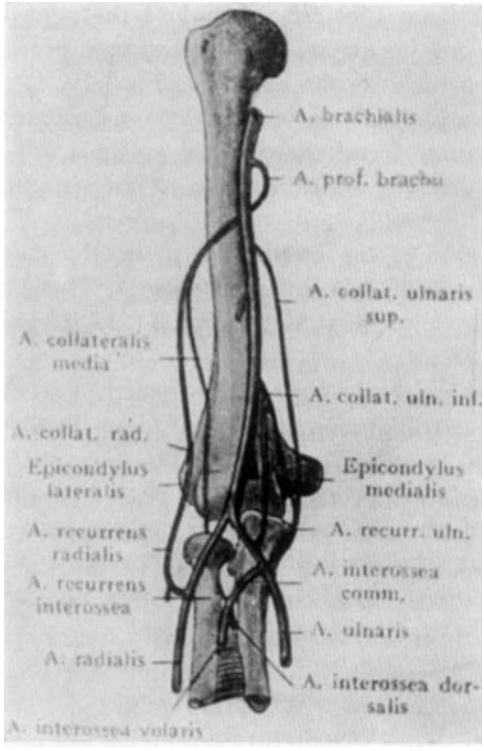


Fig. 33. After CORNING.

ever, the angiograms of the elbow do not show which vessels run to the capitulum.

In the discussion of the blood supply to the capitulum LÖHR's conclusions have been accepted by UHRMACHER (1933), LANG (1941) and JUD (1953), for example, who made no further experiments to check the validity of LÖHR's opinion.

WATSON-JONES (1952) believes the capitulum to be supplied by vessels ascending from the lower arm and crossing the elbow joint on their way to the capitulum.

Figs. 16, 20, 31 and 32 in the present publication, however, show that these specimens contain 2—3 extra-osseous vessels entering the capitulum from behind, dorso-proximally to the joint cartilage, which corresponds to the site of attachment of the joint capsule and to the site of the origin of musculus anconaeus. These vessels enter the epiphysis

at the site, which the intra-osseous angiograms (Figs. 10, 11, 13, 16, 18, 19, 20, 21, 24, 25, 26, 27, 28, 29 and 30) showed to be typical.

Judging from Figs. 16, 31 and 32, these branches to the capitulum come from a descending vessel. In Fig. 32 they appear to be branches from the arteria collateralis ulnaris inferior, which branches from the brachial artery proximal to the epicondylus ulnaris humeri and with its main branch runs in a curve proximally convex and crossing the posterior surface of the distal end of the humerus dorsally to the olecranon fossa.

In the last segment of their extra-osseous course before entering the posterior part of the capitulum, the vessels of the capitulum appeared to be without any appreciable intercommunication with the other vessels in the rete articulare (Figs. 16, 31 and 32).

It may, of course, be objected that the number of preparations with extra-osseous vessels was not enough to permit valid conclusions on the extra-osseous course of the vessels of the capitulum. But the findings in these specimens and the results of the intra-osseous angiograms which, among other things, show a striking constancy of the site of entry and of the number of the vessels of capitulum, do at least support one another.

Recapitulation

1. The intra-osseous main vessels in the capitulum humeri which enter this part of the skeleton from behind, appeared to be branches from descending extra-osseous vessels.

2. These extra-osseous vessels are, at least sometimes, branches from arteria collateralis ulnaris inferior, which runs from the brachial artery proximal to epicondylus ulnaris humeri.

3. Before entering the posterior part of the capitulum the last extra-osseous segment of these vessels appeared to be without appreciable intercommunication with the other vessels in the rete articulare.

4. Results of the study of the extra-osseous vasculature agreed with the findings in the intra-osseous angiograms. The findings in these two investigations support one another.

5. Conclusions with respect to etiology and pathogenesis of Osteochondrosis deformans juvenilis capituli humeri

It is clear from the experimental investigation (Figs. 10, 11, 13, 16, 18, 19, 20 and 21) that as long as the ossific nucleus of the capitulum of

the humerus is isolated in the chondroepiphysis, it appears to receive practically all its blood supply via a few vessels, all of which come from behind in the same direction and after an intrachondral course of varying length enter the ossific nucleus.

These vessels may be branches from one and the same extra-osseous vessel.

Judging from the present material, the vascular system of the ossific nucleus of the capitulum is not in direct communication with the intraosseous vessels in the metaphysis or with the vessels in the rest of the epiphysis. The vascular system of the ossific nucleus of the capitulum is thus isolated during the period of growth.

It appears that the vessels on which the existence of this ossific nucleus depends during this period are surely not anatomically end vessels, because they communicate with one another in the interior of the ossific nucleus. But, since they are without direct communication with the other vascular systems in the distal end of the humerus, they may be regarded as end vessels, from a point of view of function. This suggests the conclusion that although the supply to the ossific nucleus of the growing capitulum is sufficient to cope with the normal variation of physiological processes, the factors of safety are minimised owing to the anatomic isolation of the vascular system and lack of anastomoses. The ossific nucleus is thus vulnerable to any disorders in the isolated vascular system.

Reaction of the ossific nucleus of the capitulum to complete interruption of blood supply is probably the same as in any area with limited anastomotic connection, namely infarction, necrosis.

Figs. 10, 11 and 13 of a preparation from a 3-year old boy show that the ossific nucleus of the capitulum, whose volume at this age is small in relation to the surrounding chondroepiphysis, is situated in a relatively richly vascularised cartilaginous tissue. Though only 1—2 vessel canals run to the ossific nucleus, many of the vessels of the temporary cartilage run so closely to the ossific nucleus that it may be presumed that these cartilaginous vessels might play a rôle in the nutrition of the ossific nucleus in the event of circulatory disorders in the nutrient vessels proper of the ossific nucleus. Should this be so, it would help to explain why osteochondrosis deformans juvenilis in the ossific nucleus of the capitulum has never been described in such young ages as that (3 years) represented by Figs. 10, 11 and 13.

In a later stage of the period of growth than that described, the vo-

lume of the chondroepiphysis of the capitulum has decreased owing to growth of the nucleus, which then almost completely occupies the entire chondroepiphysis (Figs. 16, 18, 19, 20 and 21, from 2 boys aged 8 and 9). It is clear that here almost all of the cartilaginous vessels have disappeared from the temporary cartilage near the ossific nucleus, they are no longer necessary after decrease in volume of the cartilage. The ossific nucleus is as before supplied by only few main vessels, but is now surrounded by almost avascular cartilage only. The relatively richly vascularised temporary cartilaginous tissue seen in earlier ages (Figs. 10, 11 and 13), which might be of importance for the supply to the ossification centre in the event of disorders in the vascular system of the latter is now missing.

It is in the very stage of skeletal development shown in Figs. 16, 18, 19, 20 and 21 that Osteochondrosis deformans juvenilis capituli humeri, an aseptic total necrosis of the ossific nucleus, is known to appear. This is clear from the cases described in the literature (Fig. 6) and from the present cases (see part III, Figs. 36 and 37).

When the ossific nucleus of the capitulum is no longer isolated, *i.e.* after its bony fusion with neighbouring nuclei in the ulnar part of the trochlea and in the radial epicondylus and with the metaphysis, anastomoses develop as described, between the vascular systems of the ossific nucleus and the other epiphyseal and metaphyseal vessels (Figs. 24, 25, 26, 27, 28, 29 and 30). The above mentioned fusion with neighbouring nuclei occurs from 10 years of age in girls and from 12 in boys, while the fusion with the metaphysis occurs from 12 years in girls and from 13 years of age in boys (Fig. 3).

In none of the literature cases in O.d.j. capituli humeri has the disease started later in life than the above mentioned ages (Fig. 6), during which isolation of the vascular system of the ossific nucleus of the capitulum ceases.

This also holds for the cases of O.d.j. capituli humeri in the present investigation (Figs. 36 and 37) (see Part III).

It is clear from Figs. 10, 11, 13, 16, 19 and 20 that the vessels supplying the ossific nucleus of the capitulum run intrachondrally before they pierce the nucleus from behind. This position of the vessels in the elastic hyaline cartilage favours the development of compression injury to the vessels without loss of continuity, an observation to which much importance is attached by such authors as BENTZON (1926, 1927), MAU (1929) and BERNBECK (1950).

JUD (1953) found circumscript rarefying changes in the spongiosa of the metaphysis of the distal end of the humerus which appeared together with the pathognomonic roentgenologic epiphyseal changes in a case of O.d.j. of the capitulum humeri (similar metaphyseal roentgenologic findings are common in association with O.d.j. of the proximal end of the femur).

As to the cause of these metaphyseal changes reference is made to Figs. 16, 18, 19, 20 and 21 which show vessels coming from behind and entering proximally to the epiphyseal plate and proceeding into the metaphyseal bone. In Figs. 16 and 20 the vessels to the metaphysis and the vessels to the ossific nucleus of the capitulum branch from one and the same vessel.

The above findings suggest the conclusion that the vascular anatomy is such that a factor capable of interfering with the vascular system of the capitulum will also be able to produce circulatory disturbances in vessels supplying part of the metaphyseal bone.

The hypothesis of FERGUSON & HOWORTH (1934), HOWORTH (1948, 1952) and HULTH (1958) that synovitis is of importance in the causation of circulatory disorders in the etiology of O.d.j. of the femoral head cannot, judging by the present findings, hold good for O.d.j. of the capitulum humeri. The experimental investigation showed, as described, a marked constancy of the vascular topography in the capitulum humeri and that vessels supplying the ossific nucleus of the capitulum enter the posterior aspect of the epiphysis either at the attachment of the capsule at the posterior margin of the joint cartilage or extra-capsularly (Figs. 10, 11, 13, 16, 18, 19 and 20). The same vascular pattern of the entry of these vessels into the bone was also found in preparations from adults (Figs. 24, 26, 28, 30, 31 and 32).

Judging by these specimens then, the vessels in the capitulum never enter the joint cavity and can therefore hardly be affected by mild intraarticular disease such as subacute synovitis.

It may thus be said that the investigations suggested the following conclusions on the etiology of O.d.j. of the capitulum. During the age in which the disease is known to appear the nutrition of the ossific nucleus of the capitulum depends on a vulnerable blood supply. The vascular system of the ossific nucleus of the epiphysis during this period and until its fuses with the adjacent bone is anatomically isolated and not in communication with vessels in the other parts of the bone.

This must surely favour the development of any circulatory disorders in the ossific nucleus during the period under discussion.

These conclusions will be discussed further in association with those suggested by the clinical and roentgenologic study of a personal series of O.d.j. of the capitulum humeri in Part III.

The results of the present experimental investigation of the circulation in the endochondrally growing ossific nucleus of the capitulum humeri agree, as mentioned, in some respects with what has been found by other authors in other endochondrally growing ossific nuclei, *e.g.* the ossific nucleus of the femoral head.

It thus appears that the conclusions on the etiology and pathogenesis of O.d.j. of the capitulum may also hold for the disease in other locations, *e.g.* in the femoral head.

Recapitulation

1. During the age in which O.d.j. of the capitulum humeri is known to appear the vascular system supplying the ossific nucleus of the capitulum is vulnerable.

2. During this period the ossific nucleus of the capitulum appears to be supplied by 2—3 vessels. These vessels are not anatomic end-vessels but since they are not in communication with the other vascular systems within the bone they may be regarded as end vessels from a functional point of view.

3. Although the blood supply to the ossific nucleus in the growing capitulum is sufficient to cope with the normal variation of physiologic processes, the factors of safety are decreased during this period of life owing to the lack of intra-osseous anastomeses.

4. While the volume of the chondroepiphysis of the capitulum is large in relation to the ossification centre of the capitulum, the temporary cartilage near the ossific nucleus is relatively richly vascularised. It is believed that this might explain why O.d.j. capituli humeri has never been seen in children below 4 years.

5. However, when the volume of the chondroepiphysis of the capitulum has decreased by growth of the ossific nucleus of the capitulum, practically all the cartilage vessels have disappeared from the environments of the ossific nucleus. The nucleus is then surrounded by avascular cartilage, and supplied by only 2—3 vessels.

It is during this stage of skeletal development that O.d.j. of the capitulum is known to appear.

6. When the ossific nucleus of the capitulum is no longer isolated after bony fusion with the adjacent epiphyseal and metaphyseal bone, intra-osseous communications arise between the vascular system of the capitulum and other epiphyseal and the metaphyseal vessels. O.d.j. of the capitulum has never been observed to appear after the age, at which this fusion occurs.

7. The vessels of the ossific nucleus of the capitulum are situated in compressible temporary cartilage on their way to the nucleus.

These anatomic relationships may permit compression injury of the vessels without loss of continuity.

8. Vessels to the ossific nucleus of the capitulum and vessels coming from behind to the metaphyseal bone may be branches of one and the same soft tissue vessel. This might help to explain the metaphyseal changes observed in O.d.j. of the capitulum humeri.

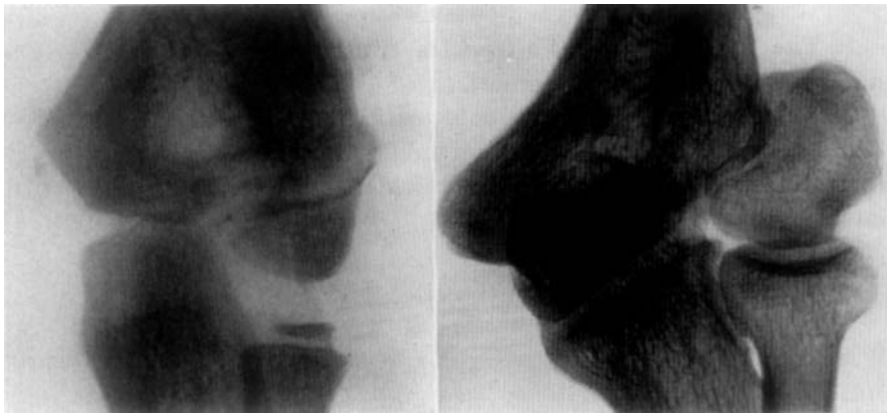
9. None of the experimental results showed any of the main vessels of the ossific nucleus of the capitulum to enter the joint cavity. This argues against mild synovitis of the elbow joint exerting a direct influence on the vessels.

10. The conclusions made regarding the etiology and pathogenesis of O.d.j. of the capitulum humeri may, to a certain extent, also hold for O.d.j. in other locations such as in the femoral head.

6. Conclusions with reference to partial ossification of the trochlea from the ossific nucleus of capitulum humeri

It is clear from Figs. 18 and 23 that the ossific nucleus of the capitulum extends into the radial part of the chondroepiphysis of the trochlea. The radial part of the trochlea, including the crista lateralis trochleae, is thus ossified from the ossification nucleus of the capitulum, an observation pointed out earlier by LANZ & WACHSMUTH (1935) and JOHNSTON & WHILLIS (1942), for example, but not mentioned in such standard textbooks as HASSELWANDER (1931), HOHMANN (1949), SCHINZ et al. (1952), BRAILSFORD (1953), BLOUNT (1954), WATSON-JONES (1952, 1955), KÖHLER & ZIMMER (1956), CAFFEY (1956) and SWOBODA (1956). Neither is it mentioned in such comprehensive works as those of PATERSON (1929), FLECKER (1942) and SCHMID & HALDEN (1949).

Since the ossification of the trochlea is thus partly dependent on the ossific nucleus of the capitulum humeri, developmental disorders, de-



G.A. Nov. 23, 1943. Age 8 years. G.A. Jan. 28, 1958. Age 22 years.

Fig. 34 A and B.

Fig. 34 A. Separation of lateral condylar epiphysis of humerus including ossific nucleus of capitulum. — Fig. 34 B. Deformity of radial part of trochlea normally ossified from ossific nucleus of capitulum.

formation or displacement of this ossification nucleus might feasibly result in malformation of the radial part of the trochlea including the crista lateralis trochleae, which might be of importance for the lateral stability of the elbow joint.

The localisation of the ossification nucleus of the capitulum would then also help to explain why the radial part of the trochlea often accompanies the fragment of the capitulum in displacement of the epiphysis of the lateral condyle in infants.

If such a displacement is not reduced or if it is redislocated after reduction (Fig. 34 A and B) the radial part of the trochlea including the crista lateralis trochleae will not be ossified because of the displacement of the ossific nucleus of the capitulum humeri. The result is a malformed trochlea with only one crista, namely the medial crista, from which the articular surface of the trochlea slopes radially-proximally (Fig. 34 B). This deformity of the trochlea together with the dislocation of the capitulum humeri results in cubitus valgus.

Fig. 34 B shows a deformity of this type in a 22 year old male after fracture of the radial condyle of the humerus at 8 years of age (Fig. 34 A).

Recapitulation

1. The radial part of the trochlea is ossified from the ossification nucleus of the capitulum humeri.

2. Developmental disorders, deformation or displacement of the ossification nucleus of the capitulum might thus feasibly result in malformation of the radial part of the trochlea humeri, including the crista lateralis.

PART III

Chapter V

CLINICAL AND ROENTGENOLOGIC REVIEW OF 27 CASES OF OSTEOCHONDROSIS DEFORMANS JUVENILIS CAPITULI HUMERI

1. Material

As mentioned (Chapter III) perusal of the literature revealed only 19 publications with a total of 30 certain cases of O.d.j. capituli humeri. Most of the authors described only a single case and none more than 4. As a rule the patients had been observed for only a short time and only 2 for more than 3 years.

It was therefore considered that a larger, and therefore more reliable, series might be of value for a closer study of the disease. The present material consists of 27 certain cases of the disease which is by far the largest series on record.

The purpose of this part of the investigation was to study the clinical and roentgenologic picture of the disease against the background of the experimental investigation (Part II) and the literature studies (Part I).

The material was collected by searching the available roentgenograms and/or records at 21 Swedish hospitals scattered throughout the country. In addition 2 cases were reported from a hospital whose roentgen material was not searched by the author for the disease (No. 22, Table 12).

These cases were thus collected from a total of 22 hospitals.

Since the disease is not so very widely known, the possibility of erroneous diagnosis could not be excluded. Therefore in 7 of the hospitals [No. 1 (Roentgendiagnostic Department 1947—1955 incl.) 2, 3, 5, 10 (from 1953), 11 and 12 in Table 12] all roentgenograms of growing elbows were checked no matter whether they had been recorded as roentgenologically normal or otherwise.

It is clear from Table 12 that the hospitals in which a search was made for the disease were of varying type and size. The periods covered by the roentgen-material examined varied from one hospital to another.

Despite a fairly thorough search O.d.j. capituli humeri was found in only 12 of the 22 hospitals (Table 12). Three new cases were reported after the end of the perusal of the roentgen-material and are included in the present series.

TABLE 12

Roentgenologic material searched for cases of O.d.j. capituli humeri

No.	Hospital	Period covered by material	Cases found
1	Lunds lasarett		
	Orthopaed. clinic	1929—1955, incl.	3
	Roentgendiagn. clinic	1934—1955, incl.	
2	Malmö allmänna sjukhus	1932—1955, incl.	1
3	Hälsingborgs lasarett	1939—March 1956, incl.	0
4	Ortopediska kliniken (Vanföreanst.), Göteborg	1930—April 1956, incl.	1
5	Göteborgs barnsjukhus	1945—April 1956, incl.	4
6	Linköpings lasarett	1937—April 1956, incl.	5 [1]
7	Ortopediska kliniken (Vanföreanst.), Stockholm	1936—April 1956, incl.	1
8	Krp. Lovisas barnsjukhus, Stockholm	1943—April 1956, incl.	4
9	Södersjukhuset, Stockholm	10-year period before May 1956	1
10	S:t Görans sjukhus, Stockholm	1938—April 1956, incl.	0
11	Barnsjukhuset Samariten, Stockholm	1946—April 1956, incl.	0
12	Sachska barnsjukhuset, Stockholm	10-year period before May 1956	0
13	Örebro lasarett	1948—April 1956, incl.	3
14	Landskrona lasarett	1934—June 1956, incl.	0
15	Halmstads lasarett	1947—1954, incl. (Search less extensive)	0
16	Borås lasarett		
	Roentgendiagn. dept.	1947—July 1956, incl.	0
	Orthopaed. dept.	1932—July 1956, incl.	1
17	Karlskrona lasarett	1938—Aug. 1956, incl. (Search less extensive)	0
18	Vanföreanstalten, Härnösand	1932—Sept. 1956, incl.	1
19	Härnösands lasarett	1949—Nov. 1956, incl.	0
20	Sundsvalls lasarett	1945—Nov. 1956, incl.	0
21	Serafimerlasarettet, Stockholm	1934—1956, incl.	0
22	Kristianstads lasarett	No search made	2
		Total	27

[1] 3 cases occurred after end of search.

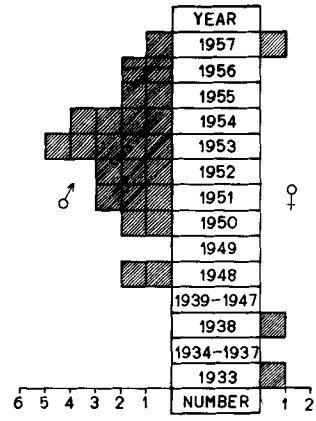


Fig. 35. Distribution among years in present series.

Fig. 35 shows the distribution of the material among years. The year given here is to be understood as the year of the first examination.

All of the patients in the present series were reviewed.

All of them had been treated at Outpatient Departments only. One patient (case 1) was admitted to hospital but not until rheumatic fever had complicated the clinical picture during the healing period of O.d.j.

Since the patients had only been treated at Outpatient Departments the notes of the symptoms and course of the disease were scanty. To compensate this lack of details careful inquiry was also made among the patients and their parents regarding such data. The review and the requestioning were carried out according to a standardised scheme. Of the review the roentgenologic examination was the most important factor. Therefore all roentgenograms taken at the review were examined by the writer. However, since the patients were scattered over a large area the clinical review and requestioning was carried out personally by the author in 14 cases; in all others except one, by orthopaedic surgeons at various hospitals and in accordance with a certain scheme.

2. Frequency

It is apparent from Table 12 that the material was collected from a wide variety of hospitals. The activity of some of these hospitals is not strictly limited to any special district and they are to a certain extent consultary institutes (e.g. University Clinics of Lund, Stockholm and Gothenburg). In addition it is clear that the periods covered by the roentgen-material varied from one hospital to another. It is therefore

not possible to say anything definite about the absolute or relative frequency of O.d.j. capituli humeri on the basis of the present material.

However, though the 21 hospitals studied included some of the largest in Sweden and since the roentgenographic material sometimes covered long periods of time (Orthopaedic Clinic, Lund 27 years, Roentgendiagnostic Clinic, Lund 22 years, Malmö General Hospital 24 years, Orthopaedic Clinic, Gothenburg 26 years, Orthopaedic Clinic, Stockholm 20 years) no more than 27 cases could be traced. As mentioned, the literature hitherto included only 30 certain cases of the disease described by 19 authors. The present cases thus bring this number up to 57.

Thus although nothing definite can be said about the frequency of the disease it can hardly be denied that it appears to be rare.

Recapitulation

Roentgen material available at 22 hospitals showed only 27 cases of O.d.j. capituli humeri. These cases together with the 30 cases reported in the literature bring the total number of cases of O.d.j. capituli humeri up to 57.

O.d.j. capituli humeri appears to be a rarely diagnosed disease.

3. Age at time of first examination

The patients' ages at the time of the first examination ranged from 4 years 11 months (case 27) to 10 years 2 months (case 15) (Fig. 36).

The mean age was 7.4 years and it was the 7 year age class that was the largest.

In Fig. 37 the ages at time of first examination in the present series are given together with those in 28 cases from the literature in which such data were available (see also Fig. 6).

It is clear from Fig. 37 that the ages ranged between 4 and 10 years. The 7- and 8-year age classes were the largest and together contained

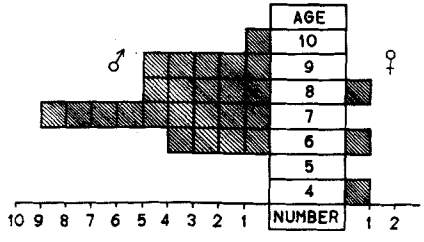


Fig. 36. Age at first examination in present series. Sex incidence.

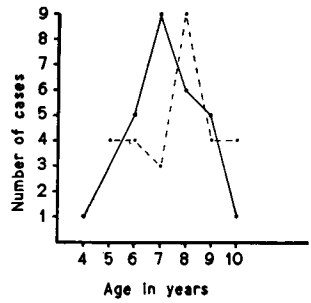


Fig. 37. Age at first examination.
——— Present series.
— — — Cases from literature.

27 (49 per cent) of a total material of 55 cases. The overall mean age at the time of first examination was thus 7.5 years.

It might be mentioned that STÅHL (1948) reported a mean age of 7.7 years at the time of the first examination in a material of O.d.j. capitis femoris.

Recapitulation

1. The age at first examination in the present material and in cases on record, varied between 4 and 10 years.

2. The mean age at time of first examination in the present material was 7.4 years. In 28 cases from the literature it was 7.6 years.

The mean age at time of first examination in a total material of 55 cases (literature cases+present cases) was 7.5 years.

4. Sex incidence

Of the present material 24 patients were boys and 3 (11.1 per cent) girls (Fig. 36).

This sex incidence is in good agreement with that found in the 30 literature cases, of which 3 (10 per cent) were seen in girls (Fig. 7).

Of the total material (literature cases+present cases) 51 of the 57 patients were boys and 6 (10.5 per cent) girls.

The disease is thus much more common in boys. This higher frequency among boys is known for O.d.j. in other locations such as caput femoris, for which HELBO (1953) gave 77.4 per cent in boys and 22.6 per cent in girls of a total material of 204 patients and GOFF (1954) described 103 patients of whom 83.5 per cent were boys and 16.5 per cent girls.

To recapitulate, O.d.j. capituli humeri is more common in boys than in girls (9:1).

5. Side affected

Of 25 of the patients of the present material the contralateral symptom-free elbow had been examined roentgenographically during the course of the disease. In 2 of the patients (cases 2 and 3) no such examination was made of the other elbow. It is true that both elbows were examined of these patients (cases 2 and 3) at the review, but by then both patients were adults and the roentgenograms were of normal appearance. Since the contralateral elbows had not been examined roentgenologically during the course of the disease in cases Nos. 2 and 3 it cannot be decided with certainty whether the disease was unilateral or bilateral in those 2 patients. Cases of latent disease in the contralateral symptom-free capitulum humeri have been described by BUETTI (1953) and LAURENT & LINDSTRÖM (1956) and corresponding findings have been reported in O.d.j. of caput femoris.

Of the 25 patients in whom both elbows were examined roentgenologically during the course of the disease the contralateral elbow was found to be of normal appearance in 24. In case 11, however, roentgenograms of the contralateral elbow showed a hazy somewhat irregular structure and irregular outline of the ossific nucleus of the contralateral left capitulum humeri. The roentgenogram resembled that seen in O.d.j. capituli humeri in the healing period. This case was therefore classified as having bilateral aseptic bone necrosis (Figs. 54, 55). But, unlike the right elbow, the contralateral elbow was symptom-free.

Of the above mentioned 25 cases the disease was right-sided in 16, left-sided in 8 and bilateral in 1. In the 2 cases in which the contralateral elbow had not been examined roentgenologically during the disease, the condition was left-sided in one and right-sided in the other.

Of the present 27 patients, 26 were right-handed and 1 left-handed. In the latter the disease was seen only in the left elbow.

First examination of case 14 also revealed roentgenologic signs of aseptic necrosis in both navicular bones of the feet. About one year before that examination the patient had had pain in the left foot. At the present review of this patient roentgenograms were made not only of both elbows but also of the feet and hips which were, however, found to be of normal appearance. Roentgen examination at the review also included examination of the feet and hips in cases 1, 3, 6, 7, 8, 10, 12, 13, 15, 16, 17, 18, 19, 20, 21, 23, 25 and 26. None showed any evidence of a pathologic condition in this locations.

In case 4 the patient had had pain in the tibial tuberosity on both sides for about 2 months before the review. Check roentgenography of the knee joints at review showed nothing abnormal.

Despite the fact that the contralateral elbow was not examined during the disease in 2 cases, it nevertheless appears justified to conclude that O.d.j. capituli humeri is as a rule unilateral and more common on the right side than on the left.

This is also in accordance with the results obtained on analysis of the literature (Chapter III, 5).

Thus in this respect, too, O.d.j. capituli humeri resembles O.d.j. capitis femoris which, as known, is usually unilateral.

Recapitulation

O.d.j. capituli humeri is usually unilateral and more common on the right side than on the left.

6. Course, clinical picture and roentgenologic findings

As will be apparent from the following, in the present material the disease ran a characteristic self-limiting course, which was practically the same in all cases. The patients had all had clinical symptoms for a varying period before the first examination. In some the symptoms had appeared after some known trauma or strain, while in others no such preceding trauma could be remembered (vide infra).

FAMILIAL OCCURRENCE of the disease was noted. Thus the boy in case 24 and the girl in case 27 were siblings.

This is the first time that O.d.j. capituli humeri has been described in more than one member of a family. Although this was only seen in 2 cases and thus not sufficient to exclude the possibility of chance, these cases might nevertheless be of interest in the discussion of the etiology of the disease (*vide infra*).

Despite careful inquiry into the family history of all cases no hereditary or familial occurrence could be revealed in any of the other cases.

None of the patients had had any similar elbow symptoms before the onset of the disease in question.

Earlier similar symptoms in other joints were denied by all except the patient in case 14 who, as mentioned, had had aseptic necrosis of the tarsal navicular bones on both sides.

One patient (case 9) had had rickets at 2 years of age and inflamma-

tion of the kidneys and pneumonia 2 years and 1 year, respectively, before the onset of symptoms in the elbow. One patient (case 11) had had pneumonia 6 years before the onset of symptoms in the elbow. One (case 23) had had poliomyelitis acuta anterior 8 years before the onset of the elbow symptoms with consequent paresis of the lower extremities. Further, apart from ordinary diseases of childhood, all patients in the present series had been in good health until the onset of the elbow symptoms.

All the patients were from apparently healthy families apart from case 7, whose father was described as having polyarthritis, case 21, whose brother had had poliomyelitis acuta anterior 7 years previously, and case 23, whose mother was in a sanatorium because of pulmonary tuberculosis at the time near the onset of the elbow symptoms in the patient.

A. Clinical picture

Local symptoms

All of the 27 patients in the present material had sought medical advice because of local symptoms in the elbow involved. In 1 patient (case 11) in whom the disease was found to be bilateral, however, the left elbow had been symptom-free.

The frequency of the most common local symptoms is given in Table 13.

The type and degree of limitation of range of movement of the elbow in cases in which such data were available are given in Table 14.

As mentioned, the material consisted of an outpatient series and therefore the notes in the records were often very brief. Data obtained from record cards were therefore supplemented by questioning the patient and his parents. As to assessment whether the disease had caused any limitation of movement or not, it may be assumed that the statement of the patient or his parents that "he could not bend or straighten the joint" must be regarded as fairly reliable in the evaluation of flexion and extension. Data obtained on requestioning were, however, not regarded as sufficient to judge any limitation of pronation-supination.

PAIN. — It is clear from Table 13 that pain in the region of the elbow was the commonest subjective symptom. All of the patients except one (case 21) had pain in the active stage of the disease. In the bilateral case (No. 11) the left elbow-joint was, as mentioned, symptom-free.

The pain was described as both spontaneous and occurring on move-

TABLE 13

Local clinical symptoms in present series

(+=present, 0=not present, —=not known)

Case No.	Subjective symptoms		Objective symptoms	
	Pain	Tenderness	Limitation of movement	Swelling
1	+	+	+	+
2	+	+	+	+
3	+	+	+	0
4	+	0	+	0
5	+	+	+	+
6	+	0	+	+
7	+	—	+	+
8	+	0	+	0
9	+	+	+	+
10	+	+	+	0
11 [1]	a. + b. 0	0 0	+ 0	+ 0
12	+	+	+	+
13	+	+	+	+
14	+	—	+	+
15	+	+	+	+
16	+	0	+	+
17	+	+	+	+
18	+	+	+	+
19	+	+	+	+
20	+	+	+	+
21	0	0	+	0
22	+	+	+	+
23	+	+	+	+
24	+	+	+	0
25	+	+	+	+
26	+	+	+	0
27	+	+	+	+
Total	26	19	27	20

[1] Bilateral case.

TABLE 14

Limitation of movement in present series

(—=not known, 0=not present)

Case No.	Extension	Flexion	Pronation	Supination	Range of movement when registered
1	Limitation	Limitation	—	—	Ext.-flex. = 155°—90°
2	Limitation	Limitation	—	—	—
3	Limitation	Limitation	—	—	—
4	Limitation	Limitation	—	—	—
5	Limitation	Limitation	—	—	Ext.-flex. = 160°—85°
6	Limitation	Limitation	—	—	Ext.-flex. = 175°—35°
7	Limitation	Limitation	Limitation	Limitation	Ext.-flex. = 145°—80° Pron.-sup. = 70°
8	Limitation	Limitation	Normal	Normal	Ext.-flex. = 170°—105°
9	Limitation	Limitation	—	—	Ext.-flex. = 180°—50°
10	Normal	Limitation	Limitation	Limitation	—
11 [1]	a. Limitation b. 0	Limitation 0	— —	— —	Ext.-flex. = 150°—90° 0
12	Limitation	Limitation	—	—	Ext.-flex. = 165°—85°, 75°
13	Normal	Limitation	—	—	Ext.-flex. = 180°—80°
14	Limitation	Limitation	Normal	Normal	40° restriction of ext.
15	Limitation	Limitation	—	—	Ext.-flex. = 175°—50°
16	Limitation	Limitation	Normal	Normal	—
17	Limitation	Limitation	Normal	Normal	Ext.-flex. = 160°—70°
18	Limitation	Limitation	—	—	10°—15° restriction of ext.
19	Limitation	Limitation	—	—	—
20	Limitation	Limitation	—	—	—
21	Limitation	Limitation	Normal	Normal	Ext.-flex. = 160°—50°
22	Limitation	Limitation	—	—	Ext.-flex. = 160°—40°
23	Limitation	Limitation	—	—	—
24	Limitation	Limitation	Normal	Normal	Ext.-flex. = 165°—65°
25	Limitation	Limitation	—	—	Ext.-flex. = 165°—45°
26	Limitation	Limitation	Normal	Normal	Ext.-flex. = 170°—60°
27	Limitation	Limitation	Normal	Normal	Ext.-flex. = 160°—75°

[1] Bilateral case.

ment in 11 cases (Nos. 1, 2, 5, 9, 12, 17, 18, 20, 22, 23, 27) as occurring only on movement in 10 cases (Nos. 3, 4, 6, 10, 11, 14, 15, 19, 25, 26) and as only spontaneous in 3 cases (Nos. 8, 13, 16). In 2 cases no data could be obtained concerning the type of the pain (cases 7 and 24).

The intensity of the pain varied from slight pain or discomfort (case 24) to such severity as to disturb the patient's sleep (cases 2, 5, 12, 17, 18, 22). One patient (case 9) reported that in the onset of the disease the pain was excruciating.

TENDERNESS localised to the region of the elbow involved was the next most common subjective symptom and was reported by 19 patients (Table 13).

In 4 patients (Nos. 1, 15, 18, 24) there was tenderness to palpation over the radial part of the elbow. In the remaining cases no such detailed data were available.

Six patients had not tenderness to palpation. In 2 cases the patients could not remember whether they had had such tenderness or not (Table 13).

LIMITATION OF MOVEMENTS. — Impairment of movement of the joint involved was the commonest objective symptom. It was the commonest of all symptoms in the present material, since it was reported by all patients. In the bilateral case (No. 11), however, the range of movement of the left, symptom-free elbow was unimpaired (Table 13).

It is clear from Table 14 that flexion was the commonest movement impaired since this was observed in all of the elbows in which the disease had produced symptoms.

Extension was reduced in 25 patients (Table 14).

In only 10 of the cases was pronation-supination of the affected joint studied during the disease before the review. Pronation-supination was normal in 8 and limited in 2 (Table 14). In 13 of the cases in which no note had been made of these movements, it was reported at the review that these movements had been normal throughout. However, as mentioned, no reliable conclusions can be drawn regarding pronation-supination from information obtained on requestioning. Therefore these data are not included in Table 14.

In cases 22 and 23 the disease was in the roentgenological healing period at the time of the review, 2 years, 1 month and 2 years, 4 months respectively after the onset of the symptoms and then pronation-supination was normal in case 22 and pronation was restricted by 10° in case 23 (Table 20).

The degree of impairment of extension and flexion in 19 cases where such reduction was recorded is given in Table 14. It is clear from the table that reduction of these movements varied from slight to pronounced restriction. All of these registrations were made at the first examination except in cases 9, 14 and 18 in which they were made 1 month and 3 weeks, 3 days, and 10 days, respectively, after the first examination. However, since the patients in cases 9 and 18 had by then only received expectant treatment (sparing of elbow) and the one in case 14 had had the joint immobilised in plaster for only a few days, the registrations of movements in these cases are also included in Table 14. In case 16 the degree of limitation of extension-flexion of the joint was registered, but since it had not been determined until after one month's immobilisation in plaster, these data are not included in Table 14.

SWELLING. — Swelling of the elbow involved was the next commonest objective symptom and was noted in 20 of the patients (Table 13).

In case 9 "fluctuant swelling over the olecranon" had been noted, in case 18 "swelling with exudate", and in case 14 "swelling of joint capsule on both sides of the olecranon". Apart from this no notes had been made as to whether the swelling was due to intraarticular or periarticular increase in circumference. In case 16 the circumference of the elbow joint involved was 1 cm longer than that of the contralateral elbow.

OTHER LOCAL SYMPTOMS. — Increased skin temperature in the region of the affected elbow compared with that on the other side was reported in 6 cases (Nos. 1, 2, 17, 19, 20, 22). Weakness of the affected arm was reported in 3 cases (Nos. 5, 22, 26), atrophy of musculature of elbow in 2 cases (Nos. 9 and 16, in the latter, however, after 1 month's immobilisation in plaster) and crepitations in the elbow joint in 2 cases (Nos. 2 and 20). Finally, increased cubitus valgus was noted on the affected side in 2 cases (Nos. 6 and 17).

Generalised Symptoms

According to available data, all of the patients were free from any generalised symptoms at the time of the first examination except the patient in case 14. The body temperature of that patient was 38.3° at the first examination, while on re-examination 3 days later after treatment with penicillin it was no longer considered necessary to check the

temperature. The E.S.R. was determined in cases 9, 11, 16, 18, 19 and 21 and in all it was found to be normal.

All of the patients were described as normally developed except the one in case 23 who, as mentioned, had paresis of the lower limbs after poliomyelitis 8 years previous to the onset of the elbow symptoms.

Judging by available data all the patients were free from signs of constitutional disorders and endocrine dysfunction.

In no ossification centres other than those in the elbows involved had any symptoms been reported on first examination.

The symptomatology in the present material was practically speaking the same as in the cases on record in the literature (Chapter III, 6).

O.d.j. capituli humeri resembles O.d.j. capitis femoris also from a symptomatologic point of view.

Duration of clinical symptoms

In an attempt to form an opinion of the duration of clinical symptoms an analysis was made of the period between the onset of the clinical symptoms and the time when the patients were free or almost free of symptoms (Table 15).

It is clear from Table 15 that the shortest duration of appreciable symptoms was 2 months (case 14) and the longest 2 years (case 24) though flexion in the latter case was still restricted 10°. But in case 23 the patient still had appreciable symptoms 2 years, 4 months after the onset of symptoms.

In the 10 literature cases in which the duration of symptoms was described (Table 7) it varied between 1 year, 2 1/2 months and 3 years.

Recapitulation

1. Familial occurrence of O.d.j. capituli humeri was described for the first time.

2. All of the patients in the present material belonged to generally healthy families and all had felt well during the period before the onset of the symptoms.

3. All had clear, local clinical symptoms. The commonest clinical picture was painful unilateral restriction of flexion-extension of the elbow joint. The elbow was usually swollen and tender to palpation. Restriction of pronation-supination was not common.

4. The clinical picture coincided with that described by other authors

TABLE 15

Duration of clinical symptoms until complete or practically complete recovery

Case No.	Duration of symptoms	Remarks
1		Permanent disability [1]
2	6 months	
3	5 months	
4	6 months	
5	1 year	
6	1 year, 2 months	
7	10 months	
8	4 1/2 months	10° restriction of extension
9	4 months	
10	1 year, 4 1/2 months	
11 [2]	a. 1 year b. No symptoms	
12	1 year	Light restriction of flexion
13	5 months	
14	2 months	
15	10 months	
16	10 months	10° restriction of flexion, slightly tender
17	1 year, 3 months	Almost symptom-free
18	1 year	
19	3 months	
20	3 months	Almost symptom-free
21	1 year	
22	1 year, 6 months	
23		Not symptom-free at review 2 years, 4 months after onset
24	2 years	10° restriction of flexion
25	1 year	
26	5 months	
27	5 months	

[1] Complicated by febris rheumatica.

[2] Bilateral case.

and the interval between onset of symptoms and disappearance of all or practically all symptoms varied between 2 months and over 2 years.

B. Roentgenologic findings

Although O.d.j. capituli humeri produces clear clinical symptoms the diagnosis cannot be established without roentgenologic examination.

The roentgenologic appearance was typical throughout and, like the clinical picture, it did not vary appreciably from case to case either.[1]

Roentgenologic analysis showed a cyclic course with destruction and regeneration, the roentgenologic appearance varying with the stage of the disease, but the appearance of each stage was one and the same in all patients.

Figs. 38, 39, 40, 41, 42, 44, 45, 46, 47, 48, 49 and 50 show a series of roentgenograms from one of the present cases (case 9). The reproductions show the typical pathognomonic roentgenographic appearance of the various stages. The appearance of the normal contralateral elbow is given in Figs. 43 and 51.

It should be mentioned that roentgenograms from case 22 in the present series were given in Fig. 2.

It is clear from Fig. 38, showing a roentgenogram taken at the first roentgen examination, that the ossific nucleus of the capitulum humeri is somewhat flattened, the contour is somewhat uneven, the normal fine rounded outline of the ossific nucleus is missing (compare with Fig. 43). On that occasion the normal spongiosa bone structure in the ossific nucleus had been partly replaced by hazy condensed parts. Distally, radially and ventrally in the ossific nucleus is a juxtachondral rarefaction, separated from the surrounding cartilage by a dense bony zone. The metaphyseal bone is of normal roentgenologic appearance.

Fig. 39 shows that the ossific nucleus had undergone further flattening, with further deformation of outline and condensation of structure.

Fig. 40 shows a more advanced stage of this process. Here the normal bony structure of the spongiosa has almost disappeared and been replaced by dense portions and rarefactions side by side, a roentgenographic appearance which is still more striking in Fig. 41 where the ossific nucleus appears to be divided into small dense fragments.

Fig. 42 shows commencing regeneration of the ossific nucleus. The

[1] Reproductions of typical roentgenograms of all cases in the present material are deposited at the University Library, Lund.

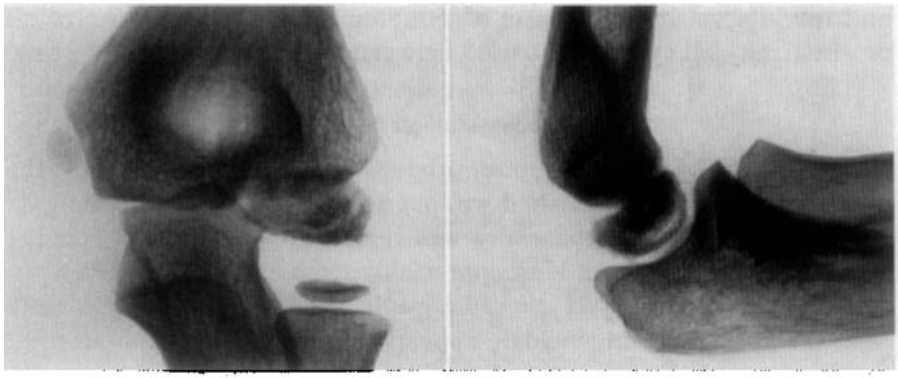


Fig. 38. *Case 9.* Oct. 22, 1951. Left. Initial stage.

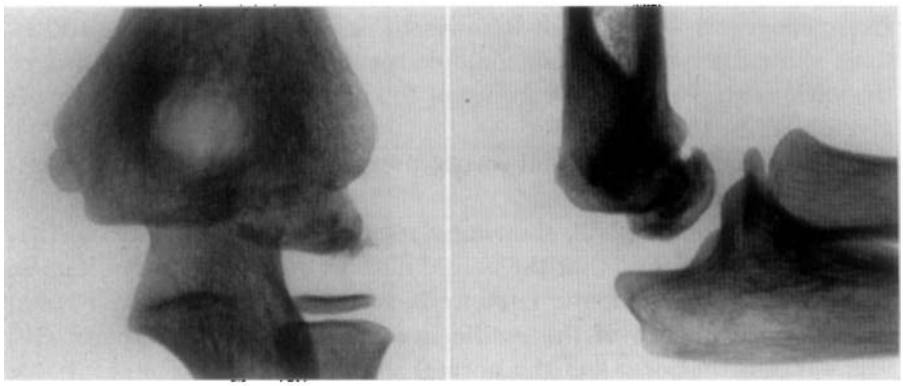


Fig. 39. *Case 9.* Nov. 15, 1951, Left. Initial stage.

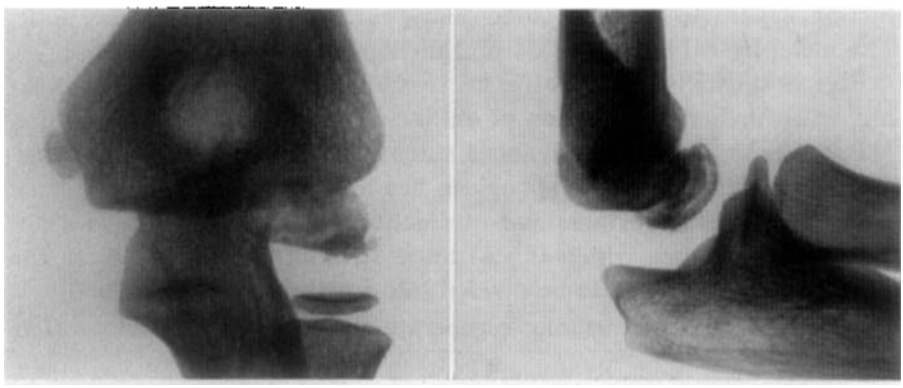


Fig. 40. *Case 9.* Dec. 28, 1951. Left. Beginning fragmentation stage.

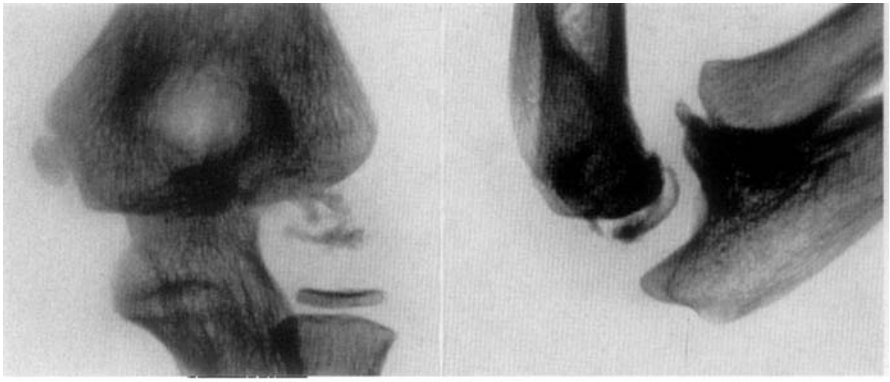


Fig. 41. *Case 9.* Febr. 13, 1952. Left. Fragmentation stage.

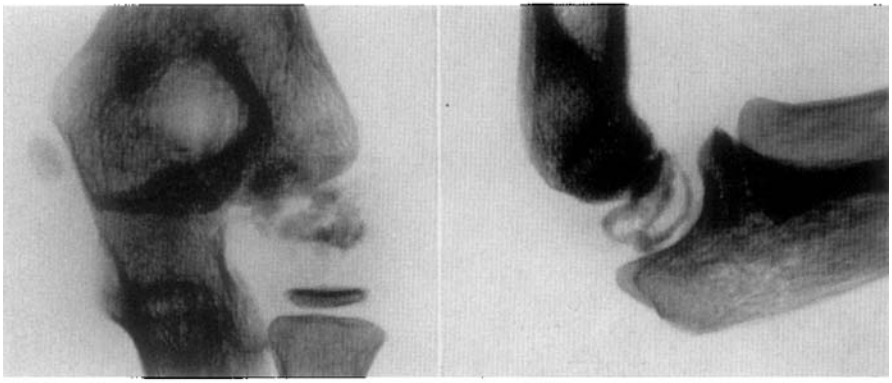


Fig. 42. *Case 9.* March 24, 1952. Left. Healing period.

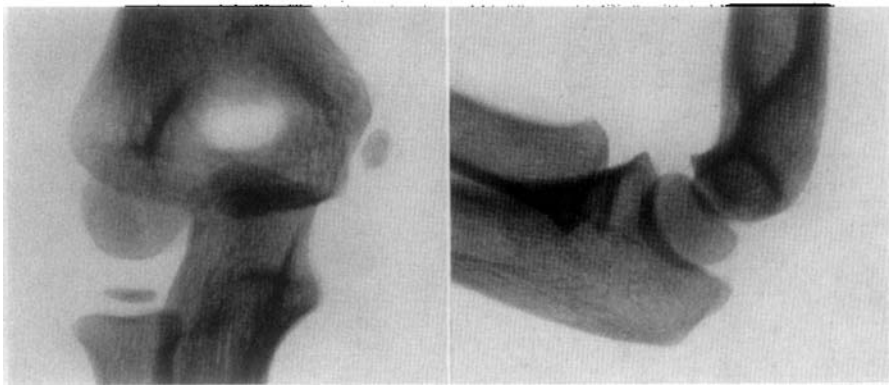


Fig. 43. *Case 9.* March 24, 1952. Right. Normal.

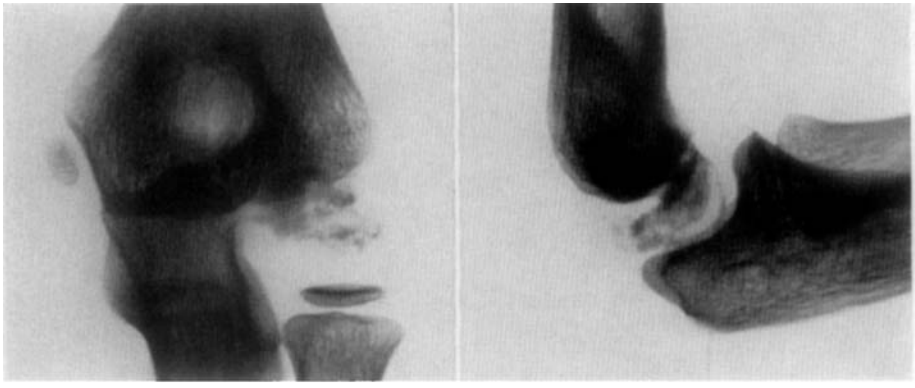


Fig. 44. *Case 9.* April 25, 1952. Left. Healing period.

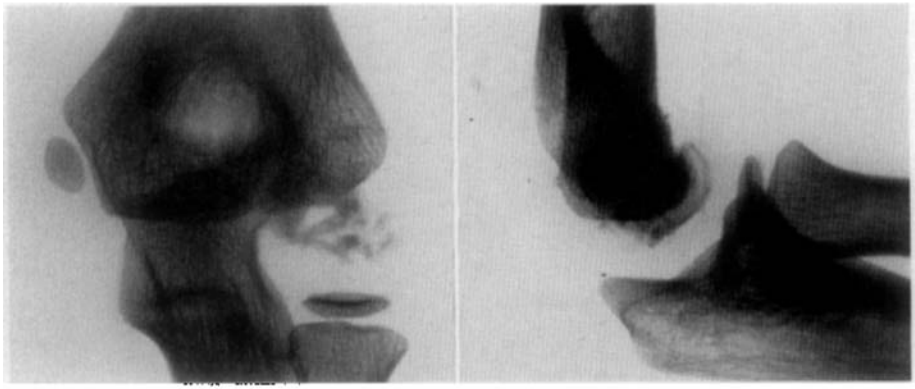


Fig. 45. *Case 9.* May 28, 1952. Left. Healing period.

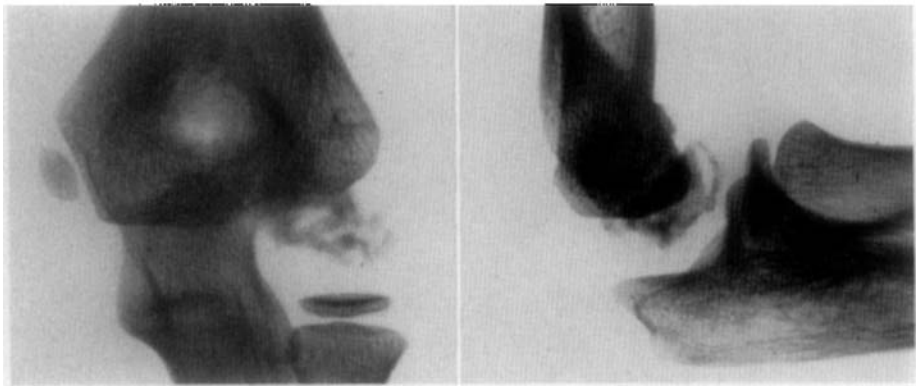


Fig. 46. *Case 9.* July 10, 1952. Left. Healing period.

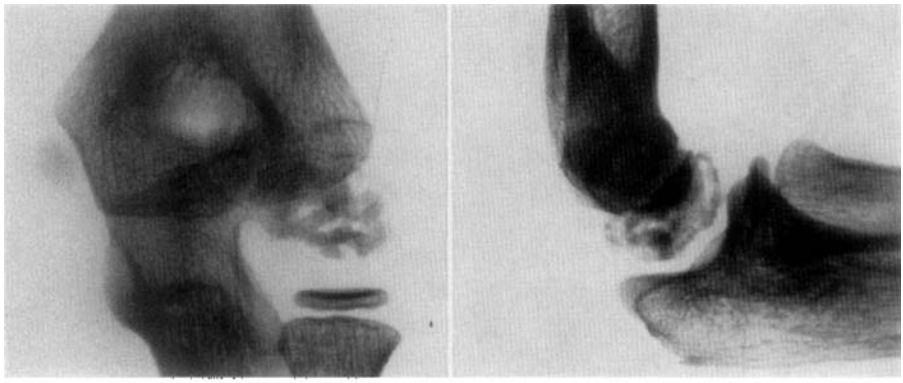


Fig. 47. *Case 9.* Sept. 10, 1952. Left. Healing period.

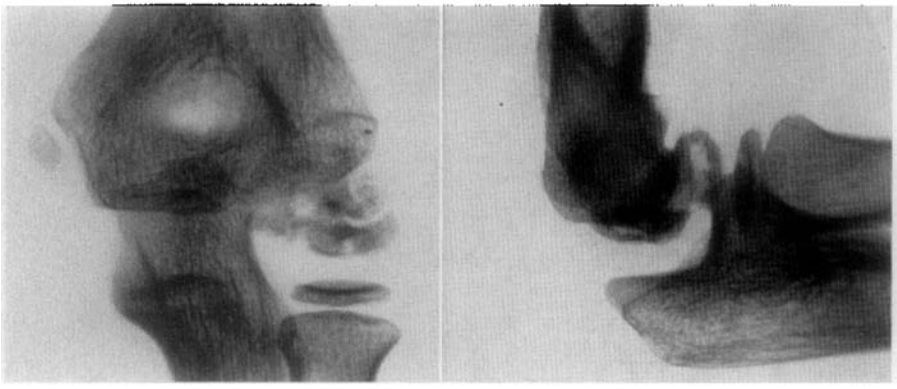


Fig. 48. *Case 9.* Jan. 27, 1953. Left. Healing period.

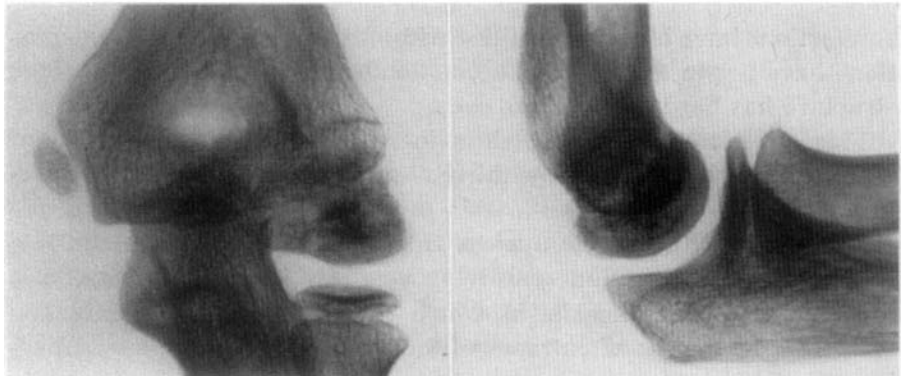


Fig. 49. *Case 9.* Aug. 31, 1953. Left. Practically healed.

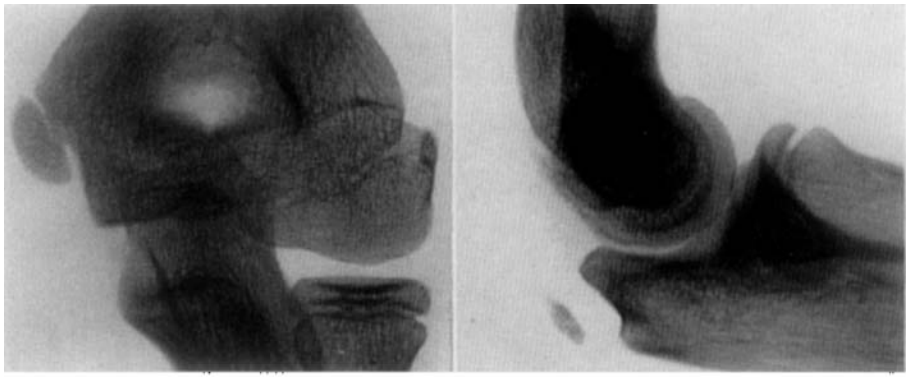


Fig. 50. *Case 9.* Jan. 2, 1957. Left. Normal. Growing period.

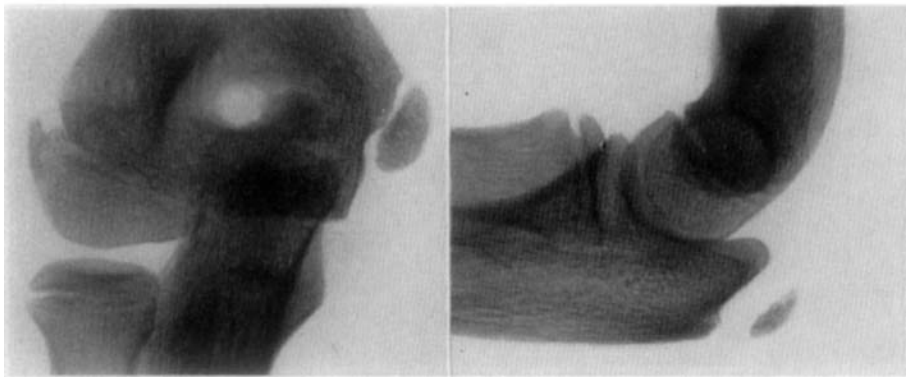


Fig. 51. *Case 9.* Jan. 2, 1957. Right. Normal elbow.

rarefactions have begun to be filled with opaque tissue, the opaque portions have begun to merge with one another to form larger units, the structure has begun to be more even.

Figs. 44, 45, 46, 47 and 48 show increasing roentgenologic signs of repair of the ossific nucleus with recovery of its size, outline and structure.

That part of the ossific nucleus filled last with opaque tissue is a juxtachondral rarefaction zone lying ventrally in the ossific nucleus, thus farthest from the site at which the experimental investigation showed to be the site of entrance of the main vessels of the ossific nucleus (Part II).

Fig. 49 shows that the process has practically healed and that the

nucleus has almost recovered normal outline and structure although it is not yet quite normal.

Fig. 50 finally shows that the ossific nucleus has recovered normal spongiosa structure and shape.

This roentgenographic series also shows a characteristic roentgenographic sign seen in most of the present cases (vide infra), i.e. that the ossification of some of the ossific nuclei on the affected side is more advanced than on the contralateral side. Thus on comparison between Figs. 42 and 43 it will be seen that the ossific nuclei in the capitulum radii and in the epicondylus medialis humeri are in a more advanced state of ossification on the affected side. Comparison between Fig. 50 and Fig. 51 will also show that, despite previous marked disintegration, the ossific nucleus of the capitulum humeri on the affected side (Fig. 50) has not only achieved the same size as the ossific nucleus on the other side, but also become still larger and is in a more advanced stage of ossification.

As mentioned, in very many respects the clinical and roentgenologic picture of O.d.j. capituli humeri closely resembles O.d.j. capitis femoris (Chapter I, Fig. 2).

Therefore, as pointed out in Chapter I, in the evaluation of the roentgenologic findings in the present material use was made of WALDENSTRÖM's (1922) roentgenologic classification of O.d.j. capitis femoris.

According to this classification Figs. 38 and 39 show the initial stage of the evolutionary period and Figs. 40 and 41 the fragmentation stage of the evolutionary period. Fig. 42 shows the beginning of roentgenologic healing period and Figs. 44, 45, 46, 47 and 48 roentgenologic signs of recovery during the healing period, the final stage of which is shown in Fig. 49. Fig. 50 shows that the ossific nucleus has become normal but since growth has not yet ended it is a question here of WALDENSTRÖM's growing period.

In this conjunction it might be mentioned that JONSÄTER (1953) found histologic signs of regeneration in O.d.j. capitis femoris already during the roentgenologic fragmentation stage, thus before WALDENSTRÖM's roentgenologic healing period. Despite these findings JONSÄTER adheres to WALDENSTRÖM's roentgenologic classification.

It is thus clear from Figs. 38, 39, 40, 41, 42, 44, 45, 46, 47, 48, 49 and 50 that the patient in case 9 showed all of the roentgenologic stages described by WALDENSTRÖM except the definite stage because the patient was not full-grown at the time of the last examination.

TABLE 16

Roentgenologic findings in present series

Case No.	Evolutionary period		Healing period	Growing period [1]	Definite stage [2]
	Initial stage	Fragmentation stage			
1	+	+	+	+	+
2	+				+
3	+				+
4	+	+	+		+
5	+				+
6	+	+	+		+
7	+	+	+		+
8	+				+
9	+	+	+	+	
10	+		+	+	
11 [3]	a. +		+	+	
	b.		+	+	
12		+		+	
13	+	+		+	
14	+			+	
15	+		+	+	
16	+	+	+	+	
17	+	+	+	+	
18	+			+	
19	+			+	
20	+			+	
21	+	+	+	+	
22	+	+	+		
23	+		+		
24	+	+	+	+	
25		+		+	
26		+		+	
27	+	+	+	+	

[1] Healed, not full-grown. [2] Healed, fusion of capitulum humeri. [3] Bilateral case.

The other cases in the present material also showed roentgenograms fitting WALDENSTRÖM's roentgenologic classification.

In cases 1, 2, 3, 4, 5, 6, 7 and 8 fusion had occurred between the ossific nucleus of capitulum humeri and the surrounding bone tissue by the time of the review, so that the roentgenograms then also showed the definite stage.[1]

The present cases showed the same roentgenogram as that described in literature cases (Chapter III, 7).

Table 16 surveys the roentgenologic findings in the present material classified according to roentgenologic stage.

It is clear from Table 16 that all of the present cases were followed until the process had healed except in cases 22 and 23. But even in those 2 cases, though the structure and outline of the ossific nucleus were not yet roentgenologically normal, the process was in the final stage of the healing period by the time of the review (Fig. 2 F from case 22).

In cases 24 and 27 the last roentgenograms were made after the present review.

KREBS (1927), PANNER (1929, 1930), NIJST (1937) and JUD (1953) described a wider roentgenologic space between the ossific nuclei of the capitulum humeri and capitulum radii on the affected side than on the contralateral side. Similar observations were made in the initial stage of cases 1, 4, 6, 8, 10, 13, 14, 15, 16, 19, 20, 23 and 24 in the present investigation. But none of the present cases were examined roentgenologically so early as to exclude the possibility of decrease in size of the ossific nucleus of capitulum humeri, but a diminution of this ossific nucleus in relation to the chondroepiphysis may explain the roentgen finding under discussion. The present material does not permit any decision as to any widening of the joint space on the affected side in an early stage, but such a finding has been described by WALDENSTRÖM (1934), JONSÄTER (1953) and others regarding O.d.j. capitis femoris. It may, however, be said that in the elbow the anatomic conditions do not favour widening of the joint space owing to swelling of soft tissues (WALDENSTRÖM 1934, HOWORTH 1948, JONSÄTER 1953), to the same extent as in the hip.

In case 27 arthrography was done of the affected elbow while the process in the capitulum humeri was in the fragmentation stage (Fig.

[1] In case 1 the patient had had rheumatic fever during the healing period of O.d.j. so that the roentgenograms made at the review were of no value for the purpose in view.

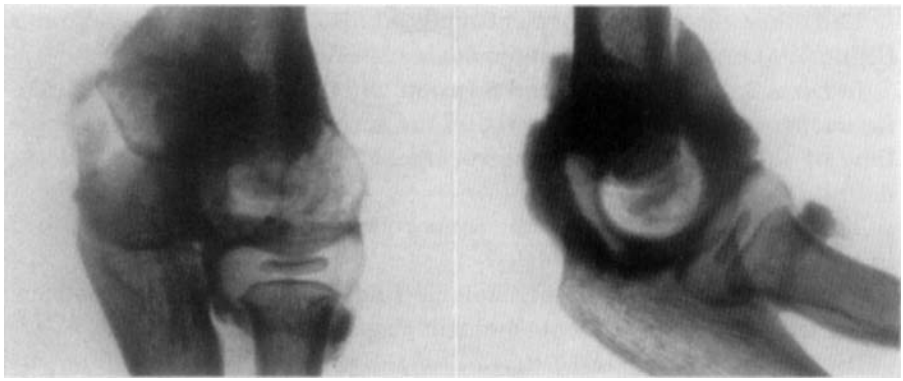


Fig. 52. *Case 27.* Febr. 6, 1958. Left. Fragmentation stage. Arthrography.

52). It is clear from the reproduction of the roentgenogram that the disintegration of the ossific nucleus had been compensated by growth of the chondroepiphysis and that the capitulum humeri had thus more or less retained its normal shape and size despite deformation of the ossific nucleus. That this was also the case in the other patients is supported by the roentgenograms taken at the review. Thus in none of the present cases that had healed by the time of the review, was any severe deformation noticed of the capitulum humeri. (As mentioned, in case 1, the patient had had rheumatic fever.)

METAPHYSEAL CHANGES. — JUD (1953) is the only author who had described pathologic roentgen findings in the structure of the metaphysis of the distal humerus in patients with O.d.j. capituli humeri. He

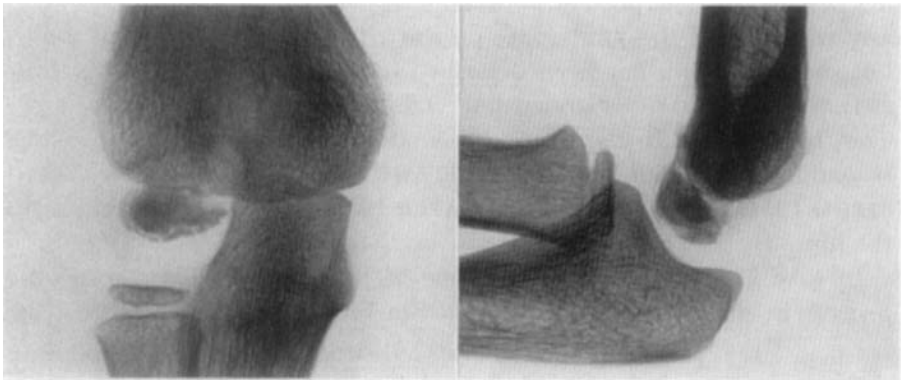


Fig. 53. *Case 20.* Aug. 7, 1954. Right. Initial stage. Changes in metaphysis.

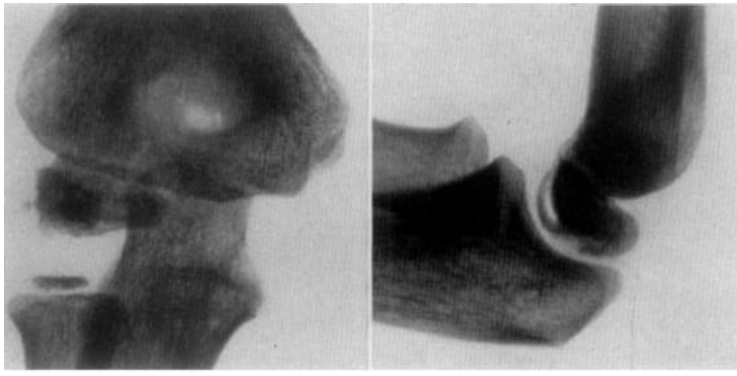


Fig. 54. *Case 11.* April 18, 1952. Right. Initial stage.

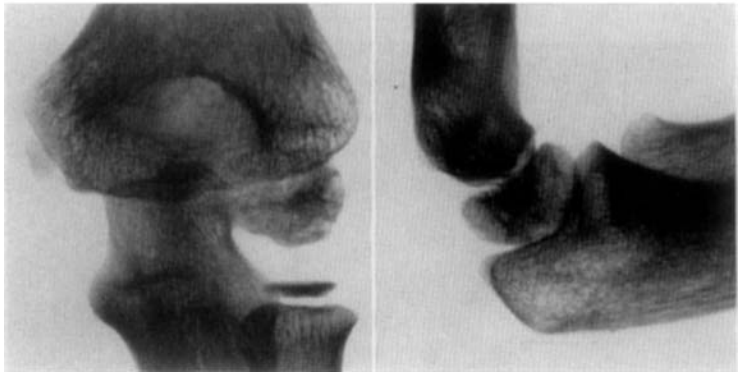


Fig. 55. *Case 11.* April 18, 1952. Left. Healing period.
Nucleus of capit. radii larger than on right side (Fig. 54).

published a roentgenogram showing a small rarefaction in the metaphyseal bone. In the present material roentgenologic signs of such metaphyseal rarefactions were seen in cases 1, 7, 11, 20 and 27 (Fig. 53). These structural changes were observed in the roentgenogram for a shorter period than the process in the capitulum and disappeared without leaving roentgenologically demonstrable signs in the metaphyseal bone.

STAGE OF OSSIFICATION. — In cases 1, 5, 6, 9, 10, 12, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26 and 27 the ossific nucleus of the capitulum radii (and sometimes also other ossific nuclei around the elbow joint) showed a more advanced stage of ossification on the affected side than on the contralateral side (Figs. 42 and 43). In cases 2

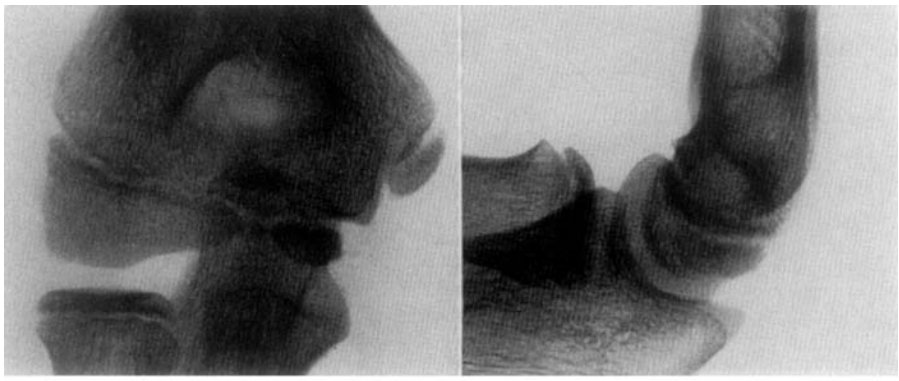


Fig. 56. *Case 11.* Jan. 4, 1957. Right. Normal. Growing period.
Nuclei of capit. humeri and radii larger than on left side (Fig. 57).

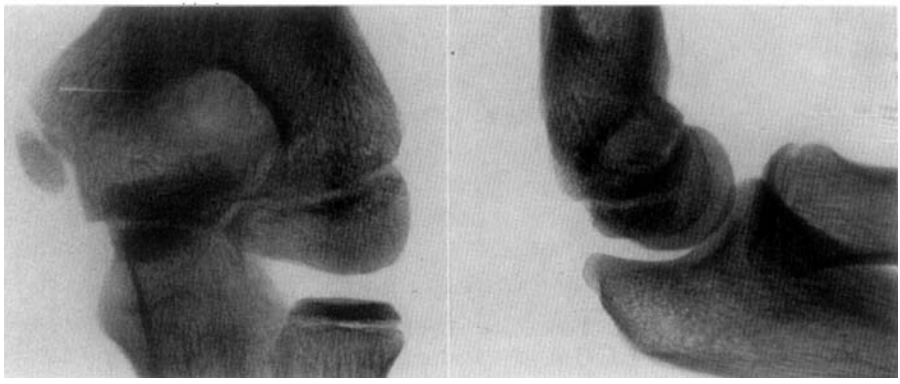


Fig. 57. *Case 11.* Jan. 4, 1957. Left. Normal. Growing period.

and 3 no roentgenograms of the contralateral elbow were available during the active period of the disease. In case 8 there was no obvious difference in size of ossific nuclei of the capitulum radii on the two sides, but the only roentgenograms available from the time of the disease dated from the initial stage of the process.

In case 11 (bilateral case) the first roentgenograms showed that the ossific nucleus of the capitulum radii on the left symptom-free side was larger than on the right side, which showed signs of the initial stage of O.d.j. (Figs. 54, 55). The last roentgenograms of this case show, however, that the ossific nucleus of the capitulum radii on the right side was now larger than that on the left (Figs. 56, 57).

Roentgenograms from the early stage in cases 10, 16 and 22 showed no definite difference in size between the ossific nuclei of the capitulum radii on either side, but roentgenograms of later stages showed that the ossification of the capitulum radii on the affected side was more advanced. These observations together with the regular appearance of this phenomenon (in 21 cases of the 25 that could be judged) suggest the conclusion that the advance in the stage of ossification of the capitulum radii (and sometimes in the other ossific nuclei of the elbow) on the affected side, occurred during and as a result of the process in the capitulum humeri. Therefore, as far as the ossific nucleus in the capitulum radii is concerned, the roentgen findings in case 11 (bilateral case) (Figs. 54, 55, 56, 57) might be explained by the assumption that the process in the asymptomatic left capitulum humeri had already passed the active stage by the time of the first examination while the process in the other elbow was at that time in the beginning of its cycle.

The above-mentioned advance in ossification on the affected side was seen also in the actual nucleus of the capitulum humeri, which despite frequently marked disintegration in the fragmentation stage, was found to increase in size and exceed that on the contralateral side during the course of healing (Figs. 50, 51, 56, 57). This was seen in cases 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 23, 25 and 26 (Table 20).

The roentgenograms of cases 2, 3, 4, 5, 6, 7 and 8 showing the elbow joints after the period of growth argue against this difference in stage of ossification between the two sides having any permanent effect on the final structure of the elbow. These roentgenograms (Fig. 58, 59) thus showed no difference between the two sides.

The above mentioned stimulation which O.d.j. capituli humeri appears to have on the other ossific nuclei around the elbow, especially the nucleus in the capitulum radii, may be of roentgen-diagnostic importance in the evaluation of the age of the disease in capitulum humeri.

The first roentgenograms of case 1 showed the initial stage of the process. Later examinations showed the typical cycle of the changes with destruction of the nucleus and regeneration with final recovery of normal roentgenologic appearance. Later check-radiograms showed deformation of the elbow joint, but since the patient had had rheumatic fever during the healing period, it might have been responsible for the final permanent deformity. This assumption was strengthened further by the absence of any such joint deformities in any of the other cases

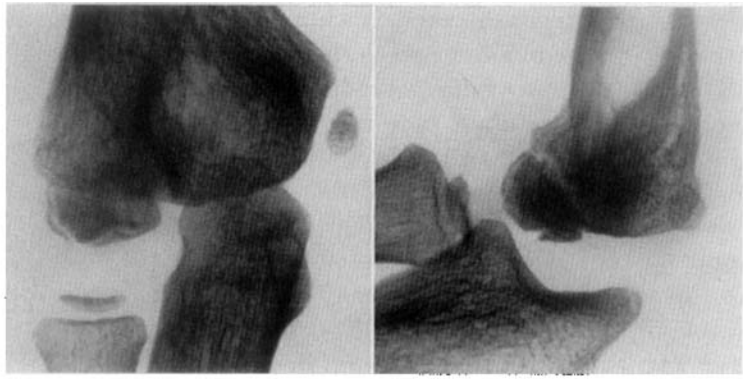


Fig. 58. *Case 5.* **March 23, 1950. Right. Initial stage.**

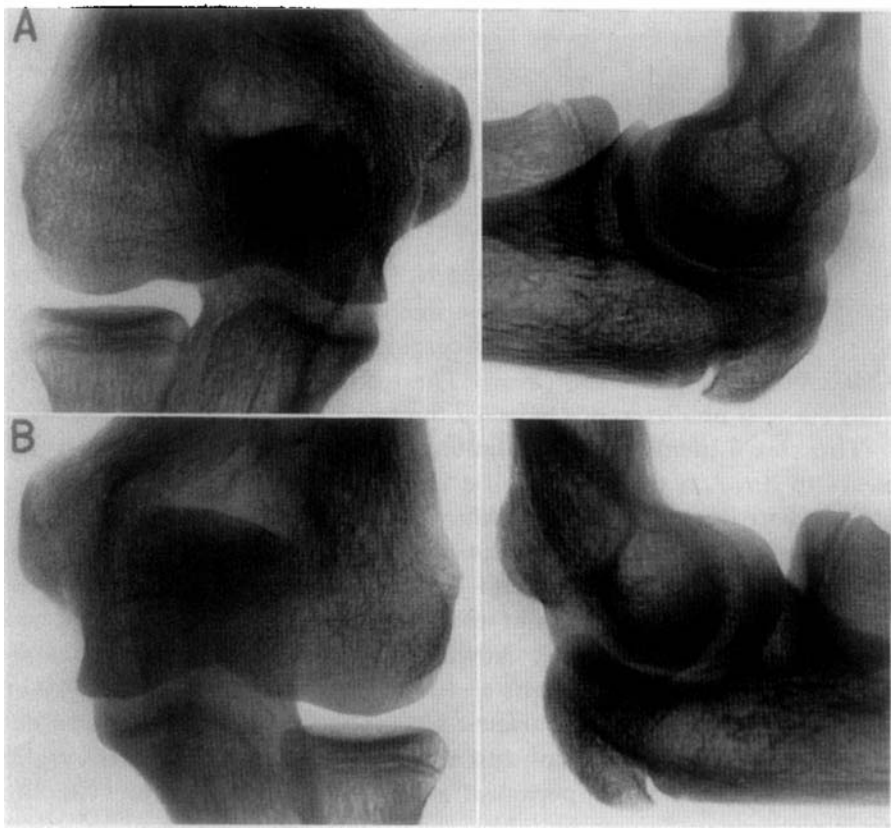


Fig. 59. *Case 5.* **Jan. 2, 1957.** Capitulum humeri full-grown. (A=right, B=left.)

in the present material or in the literature cases. In case 1, O.d.j. might have created a locus minoris resistentiae in the elbow in question and in which the rheumatic fever occurring during the healing period may have secured a footing. The other joints of the patients were normal at the time of the review.

INCREASED AMOUNT OF FLUID in the affected joint was seen in the roentgenograms with elevation of the fatty tissue dorsal to the olecranon fossa in cases 1, 4, 7, 10, 11, 13, 19 and 21, in all in the initial stage of the process.

It is obvious from what is set forth above that independently of the stage of the disease, O.d.j. capituli humeri has a typical roentgenogram. The difference between comparable stages from one case to another is so slight, that one may speak of a pathognomonic roentgenologic appearance.

Duration of roentgenologic signs of pathologic process

Below an attempt is made to judge the duration of the process from the roentgenograms available.

In the evaluation of the duration of the disease it is always difficult to say exactly when the process started, because no roentgenograms were available of the ossific nuclei before it appeared in the roentgenogram. Since it is not possible simply on the basis of roentgenograms to state with certainty how long the process has existed in a given case, the duration of the disease can only be judged from the onset of symptoms, as it must be assumed that the process existed in the ossific nucleus at the time of the onset of symptoms.

However, since the roentgenologic appearance at first examination of cases 1, 4, 5, 14, 16, 18, 19, 20 and 23 suggested that the process had been in progress for some time before it had produced any symptoms and since such earlier changes cannot be excluded in the other cases, the durations given here must be regarded as minimum values.

Table 17 includes those cases in which it was possible to evaluate the duration of the process. The table shows the interval between onset of symptoms and (a) roentgenologic signs of initial repair, and (b) until the roentgenogram of the ossific nucleus became more or less normal but nevertheless showed signs suggesting, that the healing period had recently ended, or the final stage of the healing period (Fig. 49).

In cases 11, 12, 18 and 20 roentgenograms showed that the ossific

TABLE 17

Interval between onset of symptoms and initial as well as almost complete roentgenologic recovery

Case No.	From onset of symptoms till roentgenologic signs of initial repair	From onset of symptoms till almost complete roentgenologic recovery
1	6 months	1 year, 5 months
2	—	—
3	—	—
4	4 1/2 months	—
5	—	—
6	7 1/2 months	—
7	10 months	—
8	—	—
9	8 months	2 years, 1 month
10	11 months	1 year, 4 1/2 months
11 [1]	a. 3 months b. —	— —
12	—	—
13	—	2 years, 11 1/2 months
14	—	3 years, 10 months
15	6 months	10 months
16	5 months	3 years, 9 months
17	9 1/2 months	3 years, 5 1/2 months
18	—	—
19	—	2 years, 9 months
20	—	—
21	8 months	2 years, 10 months
22	1 year, 6 months	2 years, 1 month
23	—	2 years, 4 months
24	2 years	3 years, 6 months
25	—	2 years, 5 months
26	—	1 year, 10 months
27	8 months	1 year, 4 1/2 months

[1] Bilateral case.

nucleus had recovered its normal structure and outline 4 years 10 months, 4 years 5 months, 2 years 8 months and 3 years 10 months respectively after onset of symptoms. However, since these roentgenograms no longer showed any traces of the healing process and since the interval between these roentgenograms and those taken on previous roentgen examination was long, it is not possible to say anything about the duration of the healing period in these cases.

It is clear from Table 17 that the interval between onset of symptoms and roentgenologic signs of initial repair varied between 3 months and 2 years. This period thus may correspond to the evolutionary period of the process.

Signs suggesting approximately the end of the healing period appeared at the earliest 10 months and at the latest 3 years 10 months after the onset of symptoms (Table 17).

Judging from Table 17, the healing period may be assumed to last between 4 months and more than 3 years.

Recapitulation

1. Roentgen examination of O.d.j. capituli humeri showed a typical pathognomonic roentgenogram of a process following a definite cycle with destruction and later regeneration of the ossific nucleus of the capitulum.

2. The roentgen findings in the O.d.j. capituli humeri closely resembled those in O.d.j. capitis femoris and can be classified according to WALDENSTRÖM's roentgenologic classification.

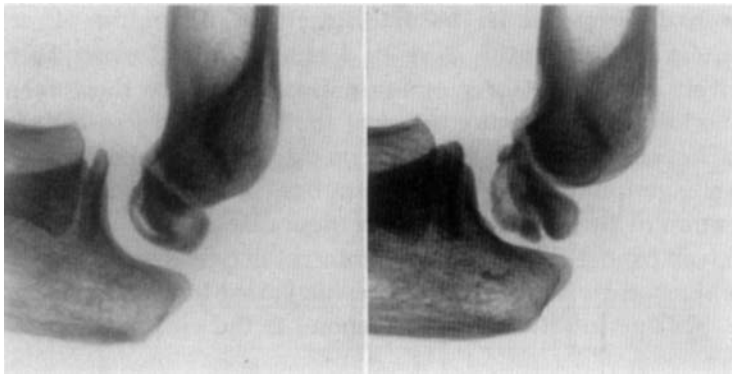
3. Arthrography performed in one case showed that despite marked disintegration of the ossific nucleus of the capitulum humeri, the capitulum chondroepiphysis retained its normal outline and size.

4. Metaphyseal rarefactions of shorter duration than the process in the capitulum were observed in 5 cases.

5. O.d.j. capituli humeri appears to stimulate growth of the ossific nuclei in the affected elbow.

6. Roentgenologic signs of increased amount of fluid in the affected joint were observed in 8 cases during the initial stage of the process.

7. In those cases in which the process had healed by the last examination the roentgenograms were of practically normal appearance in all cases, except one, in which the patient had in the meantime had rheumatic fever.



A B

Fig. 60. *Case 10.* A=May 20, 1952, initial stage. B=Dec. 30, 1952, healing period. Restitution begins dorsally.

8. The evolutionary period may be assumed to last between about 3 months and 2 years, the healing period between 4 months and more than 3 years.

7. Etiology and pathogenesis

Data available in the literature on the etiology and pathogenesis of O.d.j. in general, including O.d.j. capituli humeri, were discussed in detail in Chapter III, 10.

Relevant conclusions suggested by the present experimental investigation were discussed in Chapter IV, 5.

Below, conclusions justified by the author's material are discussed against the background of the literature and the present experimental findings.

It is clear from the present series that the similarity in the clinical and roentgenologic picture of O.d.j. capituli humeri and O.d.j. capitis femoris (in which histologic evidence of necrosis is available) is so close that it may be assumed that it is a question of two different locations of one and the same pathologic process. Therefore, despite lack of histologic evidence in cases of O.d.j. capituli humeri, the presence of necrosis of the ossific nucleus of the capitulum may be assumed. This is also supported by the present roentgenograms, which show a picture typical of epiphyseal necrosis, commencing with homogeneous condensation of the ossific nucleus, veiling of the bony structure and deformation of outline, followed by bone resorption with disintegration of the nucleus

and finally by recovery with new-formation of the spongy bone tissue. In cases 7, 9, 10 and 22 it was possible to observe that the regeneration of the ossific nucleus started from behind (Figs. 47 and 60), *i.e.*, from the region which the experimental investigation had shown to be the typical site of entry of the main vessels to the ossific nucleus.

Now, if bone necrosis be assumed, a question that unsought presents itself is whether the present material can shed any light on the cause of this necrosis.

In the present material no roentgenograms were available of the affected nucleus before the onset of symptoms except in case 11, the bilateral case, in which check radiography of the symptom-free contralateral elbow showed signs of the final stage of the healing period.

All the patients presented themselves for examination after a period of varying duration of symptoms, and the first roentgenogram always showed structural changes in the ossific nucleus such as condensation and loss of structure of the bony tissue, which suggested that the process was not recent. A further roentgen finding supporting this conclusion is that 15 of the 21 cases, which showed stimulation of ossification on the affected side (*vide supra*), showed more advanced stage of ossification already in the first roentgenograms (cases 1, 5, 6, 12, 14, 15, 17, 18, 19, 20, 21, 23, 24, 26 and 27), which shows that the process had existed some time before the first roentgen examination.

Table 18 summarises the state of affairs at the onset of the clinical symptoms, data on trauma or undue strain, the interval between the onset of symptoms and the first roentgen examination and the roentgenologic appearance of the process on that occasion.

It is clear from Table 18 that known trauma as the cause of the local clinical symptoms was reported in 14 of the cases. In the remaining 13 no such trauma could be remembered. In all the cases in which trauma was described it was said to have been directly against the affected arm. [In 27 literature cases trauma had been reported in 12 (Table 9).]

In Table 18 it is apparent that in the cases in which trauma could be remembered, local symptoms appeared immediately afterwards and therefore could be ascribed to the trauma. None of the 27 patients had had elbow trouble before.

The severity of the trauma varied from undue strain (carrying parcels, case 20) to fall from cycle onto elbow (cases 7 and 22).

The interval between the onset of symptoms and the first roentgen examination varied between 4 days and 1 year (Table 18) and the roent-

TABLE 18

Relation to previous trauma, duration of clinical symptoms before first roentgenologic examination and first roentgenologic findings

Case No.	Known previous trauma	Type of trauma	Interval between trauma and onset of symptoms	Interval between onset of symptoms and Ist. X-ray exam.	Findings at first roentgenologic examination
1	+	Twisted arm	None	10 days	Initial stage
2	+	Fall on flexed elbow	None	1 month	Initial stage
3	+	Blow against elbow	None	3 months	Initial stage
4	+	Weight lifting	None	2 weeks	Initial stage
5	0			3 weeks	Initial stage
6	0			2 months	Initial stage
7	+	Fall on elbow	None	6 months	Initial stage
8	0			2 months	Initial stage
9	0			3 months [1]	Initial stage
10	+	Fall on flexed elbow	None	2 months	Initial stage
11 [2]	a. + b. 0	Fall on flexed elbow	None	2 months —	Initial stage Healing period
12	+	Twisted arm	None	3 months, 3 weeks	Fragmentation stage
13	0			1 month	Initial stage
14	0			10 days	Initial stage
15	+	Fall on flexed elbow	None	3 months	Initial stage
16	0			4 days	Initial stage
17	+	Twisted arm	None	4 months	Initial stage
18	0			14 days	Initial stage
19	0			5 days	Initial stage
20	+	Undue strain of arm	None	2 weeks	Initial stage
21	0			1 month	Initial stage
22	+	Fall on flexed arm	None	2 months	Initial stage
23	0			3 weeks	Initial stage
24	+	Fall on arm	None	1 year	Initial stage
25	+		None	6 months	Fragmentation stage
26	0			3 months	Fragmentation stage
27	0			3 months	Initial stage

[1] Not X-rayed at first examinations. [2] Bilateral case.

gen examination showed the initial stage in 24 elbows and the fragmentation stage in 3. In case 11, the bilateral case, the contralateral symptom-free elbow showed roentgenologic signs of the healing period.

As described in Chapter III, various theories have been put forward on the still obscure etiology of O.d.j. including O.d.j. of the capitulum humeri. These theories fall into three main groups (Chapter III, 10): I. Disturbance of circulation of ossific nucleus with consequent necrosis, II. Primary fracture of ossific nucleus, III. Endogeneous factors. The conclusions drawn from the clinical and experimental studies in the present investigation will be discussed against the background of this classification.

Ad I. Circulatory disturbance. — In the experimental investigation (Chapter IV, 5) it was concluded that the ossific nucleus of the capitulum humeri has a vulnerable circulatory system during the age period, in which O.d.j. capituli humeri is known to appear. The ossific nucleus appears to receive practically all its blood supply via a few vessels coming from behind. During this period of life and until the ossific nucleus fuses with surrounding bone, this vessel system is anatomically isolated from the rest of the bone. These vessels may be regarded as functional end-vessels. Consequently although the blood supply to the growing ossific nucleus is sufficient to cope with the normal variations of physiological processes, the lack of communication with other vascular systems in the metaphysis or epiphysis during this period of life decreases the factors of safety.

This lack of communication between the vascular system of the ossific nucleus and the other parts of the distal end of the humerus enables disturbance of the circulation of the scanty vessels to the ossific nucleus to result in total interruption of nutrition to the ossific nucleus with total necrosis of the nucleus as a consequence.

The next question is whether the findings made in the present clinical series of O.d.j. capituli humeri suggest that vascular disturbances play a rôle in the pathogenesis.

Fig. 53 (case 20) shows a typical picture of O.d.j. capituli humeri in the initial stage of the evolutionary period. This figure also shows a rarefaction in the spongy bone structure of the metaphysis. Similar roentgen findings were also made in cases 1, 7, 27 and in the right elbow in case 11. These roentgen findings with metaphyseal rarefactions and necrosis of the ossific nucleus of the capitulum occurring simultaneously on either side of the epiphyseal plate may, against the background

of the experimental study, be ascribed to circulatory disturbance in a vascular area from which branches extend to these two regions. In cases 1, 7 and 27 the roentgenologic metaphyseal changes disappeared earlier than the pathologic changes in the ossific nucleus of the capitulum. (In cases 11 and 20 no roentgenograms were available to judge this point.) This may also be explained by the experimental observation, that while the ossific nucleus in this age has no intraosseous communication with other vascular systems, such communications are abundant in the metaphyseal bone (see Part II).

The clinical series thus produced findings arguing for the rôle of circulatory disorders in the pathogenesis of the disease.

If it be assumed that circulatory disorders are the cause of the pathologic changes in the disease the next question will be whether any findings suggested the possible cause of this circulatory disorder.

As mentioned, definite trauma as the cause of the symptoms was known in 14 of 27 cases (Table 18). But, if the trauma is to be held responsible, the first roentgen examination afterwards must have shown changes which correspond to the interval between the trauma and the examination. In cases 1, 4 and 20 trauma or severe strain had been reported 10, 14 and 14 days, respectively, before the first examination as the factor precipitating the symptoms. In these 3 cases, however, the first roentgenologic findings showed changes in the ossific nucleus, such as condensation and loss of structure, which must be regarded as being of longer duration than the interval between the trauma and the first examination (Fig. 53). In these cases, then, the process must have been present in the capitulum epiphysis before the trauma described.

In the remaining 11 cases in which trauma was described as the cause of the symptoms, the shortest interval between the trauma and the first roentgen examination was one month, the longest one year (Table 18). As to the rôle of trauma as a causal factor in these cases, it cannot be excluded that the trauma might have had some effect on the extraosseous or intrachondral-intraosseous course of the few isolated vessels, which the present experimental investigation showed, to be responsible for the nutrition of the ossific nucleus of the capitulum in this age.

In 13 of the present cases no trauma could be remembered. In one patient (case 11) the disease was bilateral and no trauma could be remembered against the left elbow, which had produced no symptoms. In 3 cases the process was, as mentioned, probably of longer duration than the interval between the trauma and the first examination. Two of the

patients were siblings. Only 11.1 per cent of the patients were girls. Traumatisation of the elbow is common among children, especially boys, in the age under discussion, but O.d.j. capituli humeri appears to be rare.

All this argues against the rôle of trauma as a sole primary causal factor. But it cannot be excluded that circulatory disorders due to trauma may sometimes be the cause in the same way as has been described for the process in the caput femoris (Chapter III, 10).

In all the cases with known trauma the clinical symptoms appeared immediately after traumatisation. In 3 the onset of the disease had probably antedated the trauma, but even then the trauma was the factor that precipitated the symptoms for which the patient sought medical advice. Figs. 2, 38, 53, 54, 58 and 60 show a relatively narrow juxtachondral rarefaction distally, ventrally and radially in the ossific nucleus. This finding was made in the initial stage in 23 cases and in the fragmentation stage in 2. In 2 cases, Nos. 21 and 25, this finding was missing, in the former case roentgenograms of the initial stage of the process were available, while the first roentgenograms in case 25 showed the fragmentation stage. Moreover, this finding was missing in the left asymptomatic elbow in case 11, in which the first roentgenograms showed the healing period (Fig. 55). Similar juxtachondral rarefactions have been described (WALDENSTRÖM 1934 and others) in early stages of O.d.j. in the caput femoris (Fig. 2 A). WALDENSTRÖM considered resorption of necrotic bone to be the underlying pathologic cause of this roentgen finding. In all of the present cases in which this juxtachondral rarefaction was observed, it was located disto-ventrally, sometimes radially, in the ossific nucleus (Figs. 2, 38, 53, 54, 58, 60), thus farthest from the region which the present experimental investigation showed to be the site of entry of the vessels to the ossific nucleus. In cases 1, 3, 5, 6, 8, 9, 10, 13, 14, 18, 22 and 23 the juxtachondral rarefactions were the first rarefaction seen in the spongy bone of the ossific nucleus, in cases 2, 4, 7, 11, 15, 16, 17, 19 and 20, rarefying, structural changes were seen at the same time in the posterior part of the ossific nucleus, where the vessels enter the latter, but here these rarefactions located in either end of the nucleus were without any interconnections with one another (Fig. 53).

Therefore, while the rarefactions in the posterior part of the ossific nucleus may be due to resorption of bony tissue because of ingrowth of granulation tissue, it is not probable that such ingrowth was responsible

for the rarefactions in the disto-ventral and sometimes radial part of the ossific nucleus.

This agrees with the conclusion of JONSÄTER (1953) on the basis of histologic examinations of O.d.j. in the caput femoris.

He thus found no histologic signs of such a sub-chondral thinning being due to resorption. He suggested that these rarefactions might be due to compression of the necrotic bone, after which the elastic joint cartilage with its attached ossification zone springs back into position when the pressure is removed and thereby leaves a gap.

Such a pathologic compression fracture in the necrotic spongy bone might explain why the trauma in the present material precipitated the clinical symptoms and even in the cases without known trauma it is not possible to exclude physiologic strain producing juxtachondral rarefactions in the manner described above. The only case in the present series in which roentgenograms were available of the initial stage of the disease and in which no juxtachondral rarefaction was seen was case 21. Moreover this was the only patient who had not pain in the elbow. However, since this was a single case the possibility of chance cannot be excluded.

As to other theories on the cause of the circulatory disorders in O.d.j. and presented by various authors (Chapter III, 10) it might be mentioned with reference to the hypothesis of synovitis playing a causal rôle, that some of the present cases had an increased amount of fluid in the affected joint, a sign of a pathologic condition in the synovial membrane. The present experimental investigation, however, showed (Chapter IV) that the vessels of the ossific nucleus of the capitulum do not enter the joint cavity, so that it is hardly anatomically possible for these vessels to be influenced by synovitis in the joint cavity. The above mentioned increase in the amount of fluid in the joint may be secondary to the disease in the capitulum. The disease occurred in otherwise healthy children without generalised symptoms of infection, with the exception of case 14, who had a short bout of fever at the time of the first examination. The cause of the fever was not given, it might have been due to some disease other than the process in the capitulum humeri. The higher frequency of boys in the material (and in the literature) and the limited age period during which the disease occurs argue against infection being of importance in the etiology as do the roentgenologic findings.

As to the other hypotheses on the cause of circulatory disorders, embolism, endarteritis obliterans, neurogenic factors, no observations were made in the present material in support of such theories. The practically regular monostotic occurrence of the disease, the much higher frequency among boys and the fact that the patients are otherwise healthy and normally developed argue against embolism etc. being of any causal importance.

Ad II. Fracture as a primary cause. — All the factors mentioned above as arguing against trauma as the sole primary causal factor of circulatory disorders, also argue against the hypothesis of a primary loss of continuity, a fracture, in the spongy bone of the growing ossification centre as the primary causal factor.

It may also be added that although fracture of the ossific centre of the capitulum humeri in children is not so very rare, not a single case is on record in which such a fracture was followed by O.d.j. capituli humeri. The roentgenologic appearance of the process (vide supra) also argues against primary fracture of the ossific nucleus being of etiologic importance.

Ad III. Endogeneous factors. — All the factors referred to above as arguing against trauma or fracture being a primary cause of the disease, direct one's thoughts to the possibility of some endogeneous factor. This is supported by the much higher frequency of the disease in boys, the appearance of the disease during a certain period of life, familial occurrence in cases 24 and 27, bilateral occurrence in case 11, the many cases without known trauma and the rarity of the disease in an age in which traumatisation is common.

In 24 of the 25 cases in which roentgen examination of the contralateral arm was performed, the elbow on that side was of normal appearance. Only one of the cases (case 14) showed similar simultaneous changes in other endochondral ossification centres. All the patients were in a good general condition and of normal development without signs of constitutional disorders or endocrine dysfunction. All this argues against the existence of endogeneous factors of generalised type.

As to the occurrence of the disease within a limited age period it is claimed (Chapter IV, 5) that this might be explained on the basis of the experimental investigation. That investigation showed that during the age when the disease is known to occur the ossific nucleus of the capitulum humeri is surrounded by avascular cartilage supplied only by a few isolated vessels (Figs. 16, 18, 19, 20 and 21). In younger child-

ren, on the other hand, the ossific nucleus is surrounded by relatively richly vascularised cartilage (Figs. 10, 11, 13). After the age period in which the disease is known to occur, the ossific nucleus is joined to surrounding bony tissue by bony fusion and thereby has achieved intra-osseous communications with other parts of the end of the bone (Figs. 24, 25, 27, 29 and 30).

To summarise, it may be said that the conclusions suggested by the present clinical and roentgenologic study against the background of the present experimental investigation are that O.d.j. capituli humeri is due to epiphyseal necrosis secondary to circulatory disorder. That this circulatory disorder, at least sometimes, may be of traumatic origin cannot be excluded. But much argues for the existence of some local endogeneous etiological factor.

Recapitulation

1. The roentgenologic appearance of the process as well as the close similarity between the clinical and roentgenologic picture and that of O.d.j. capitis femoris, suggests that the disease may be classified as necrosis of the ossific nucleus of the capitulum.

2. The experimental investigation showed that the vascular anatomy is such that a total necrosis of the ossific nucleus of the capitulum might be produced by circulatory disorders in a few vessels.

3. Roentgenologic rarefactions were sometimes seen in the metaphysis of the distal humerus in association with roentgenologic signs of the process in the ossific nucleus. These findings on either side of the epiphyseal plate may, on the basis of the present experimental investigation, be ascribed to circulatory disorders in one and the same vascular region, sometimes in one and the same extra-osseous vessel. The metaphyseal changes thus also suggest the rôle of a circulatory disorder in the cause of the disease.

4. Many factors in the present series argue against the rôle of trauma as the sole primary causal factor. That circulatory disorders in O.d.j. capituli humeri may sometimes be caused by trauma cannot be excluded.

5. The present experimental findings argue against the juxtachondral rarefaction pathognomonic of the initial stage of the disease being due to bone resorption. It may be due to pathologic fracture in the necrotic bone, which might explain the rôle of trauma in the causation of symptoms in the present series.

6. Increased amount of fluid in the affected joint was sometimes ob-

served. The present experimental findings, however, argue against synovitis being of etiologic importance because the vessels running to the ossific nucleus of the capitulum do not enter the joint cavity.

7. All the factors arguing against trauma or fracture as being of primary importance in the etiology, suggest some kind of endogeneous etiologic factor, which because of the frequently monostotic appearance in otherwise normal children, may be of local nature.

8. The experimental investigation helped to explain why the disease occurs only during a limited period of life.

8. Diagnosis

The diagnoses recorded on first examination are summarised in Table 19.

It is clear from Table 19 that the correct diagnosis was made on the first occasion in 17 of the cases. Of the remaining 10 cases roentgenograms showing the initial stage were available on first examination in cases 2, 3, 5, 6, 7, 8, 14, 15, 22, while in case 9 the patient was not examined roentgenologically until $1^1/_2$ month after first clinical examination and the diagnosis had thus first been made only on clinical grounds.

The risk of an erroneous diagnosis is thus greatest while the process is in the initial stage before distinct roentgenologic changes have appeared, but even then as in the other stages, the disease shows a typical pathognomonic, roentgenologic picture permitting clear-cut diagnosis. As mentioned, the diagnosis is based mainly on the roentgenogram, though the patient's age, good general condition and the local symptoms are of some help.

In cases 3, 5 and 7 the primary diagnosis of fracture was made. As mentioned, the structural changes in the ossific nucleus in the initial stage (Figs. 38, 53, 54, 58 and 60) argue against an ordinary fracture, while the juxtachondral rarefaction might be due to pathologic secondary fracture of the necrotic bone.

In cases 9 and 14 local infection was at first suspected. In the beginning the local clinical symptoms are the same as in a local infection, but the roentgenogram and absence of general symptoms of infection argue against such a diagnosis. In cases 2 and 8 first examination gave a diagnosis of anomaly or disturbance of ossification. As mentioned (Chapter II) the literature contains no description of irregular ossification of the ossific nucleus of the capitulum humeri during the years in

TABLE 19

Primary diagnoses in present series

Case No.	Primary diagnosis
1	Mb. Panner
2	Anomaly of ossification, caused by trauma?
3	Fracture of capitulum humeri
4	Aseptic necrosis, same type as in coxa plana
5	Compression-fracture of capitulum humeri
6	No diagnosis at first roentgenologic examination
7	Fracture of capitulum humeri
8	Disturbance of ossification
9	Affection of joint? Bursitis? [1]
10	Aseptic necrosis
11	Aseptic necrosis of bone
12	Necrosis of capitulum humeri. Resembles coxa plana
13	Mb. Panner
14	Arthritis
15	Osteochondritis dissecans
16	Aseptic necrosis of capitulum humeri
17	Necrosis of capitulum humeri
18	Disturbance of nutrition or aseptic necrosis. Resembling coxa plana
19	Resembles coxa plana
20	Disturbance of nutrition, resembles coxa plana
21	Osteonecrosis
22	No skeletal changes
23	Aseptic necrosis of bone
24	Mb. Panner
25	Osteochondritis
26	Mb. Panner
27	Mb. Panner

[1] Not X-rayed at first examinations.

which O.d.j. capituli humeri is known to occur. All authors describe the ossification of this ossific nucleus as regular during this period both regarding structure and outline.

As to the differential diagnosis between O.d.j. capituli humeri and Osteochondritis dissecans (primary diagnosis in case 15) of the same location, it might be mentioned that these two diseases differ from one another in various respects. In osteochondritis dissecans the pathologic changes involve only a small part of the capitulum humeri, whose structure is otherwise roentgenologically normal. NIELSEN (1933) thus found in a series of 133 patients presenting 168 instances of Osteochondritis dissecans capituli humeri, not a single case "der dafür sprach dass das Capitulum total oder subtotal necrotisch war oder gewesen war". On the other hand, both literature cases and those in the present series showed roentgenologic signs of total necrosis of the ossific nucleus of the capitulum. The two diseases also differ in age incidence. While O.d.j. capituli humeri in the literature cases and in the present cases appear between the ages of 4 and 10 years, osteochondritis dissecans of the same location appeared later, in NIELSEN's 133 cases in the great majority of cases from 13 to 17 years, rarely earlier or later.

Recapitulation

O.d.j. capituli humeri is readily diagnosed roentgenologically, since each stage of the disease has a pathognomonic roentgenographic appearance.

9. Treatment

It is clear from the survey of the literature and from the present series that O.d.j. capituli humeri runs a self-limiting course.

The treatment received by the patients in the present series is given in Table 20. The table also shows the interval between the onset of symptoms and the review, the clinical and roentgenological findings made at the review, as well as patients' ages at the time of the review.

It is clear from Table 20 that treatment varied from none (cases 23, 25 and the left elbow in case 11, bilateral case) to immobilisation of the affected elbow up to 2 months (cases 2, 7, 13). Most of the patients were treated by resting the elbow, either by immobilisation in plaster or some other kind of bandage (cases 1, 2, 7, 8, 10, 11 A, 13, 14, 15, 16) or were simply instructed to spare the elbows as much as possible (sometimes

TABLE 20

Treatment, interval from onset of symptoms to review and findings at review

(*C.h.*=ossific nucleus of capitulum humeri, *C.r.*=nucleus of capitulum radii, *Tr.*=nucleus of trochlea, *Ol.*=nucleus of olecranon)

Case No.	Treatment	Interval from onset of symptoms to review	Review		Age at time of review
			Clinical	Roentgenologic	
1	Immobilisation 1 month. Massage. Mobilisation.	22 years, 11 months	Pain in elbow on exertion. 65° restriction of extension, 60° of flexion and 30° of pronation. Forced movements painful. Muscle atrophy of arm.	*Definite stage.* Full-grown. Incongruence of ulnohumeral joint, deformity of capitulum humeri and radii. (Patient contracted febris rheumatica during healing period of O.d.j.)	28 years, 10 months
2	Immobilisation ca. 2 months	17 years, 10 months	Occasionally pain in elbow on exertion. Slight pain on forced passive ext.-flexion. Slight atrophy of forearm	*Definite stage.* Full-grown. Normal.	25 years, 10 months
3	Sparing of elbow	10 years, 8 months	Normal	*Definite stage.* Full-grown. Normal.	19 years, 8 months
4	Sparing of elbow	8 years, 4 months	Normal	*Definite stage. C.h.* fused to metaphysis. Normal.	15 years, 5 months
5	Sparing of elbow	6 years, 10 months	Normal	*Definite stage. C.h.* fused to metaphysis. Normal.	14 years, 5 months
6	Massage, sparing of elbow	8 years, 1 month	Periodic feeling of slight weakness in elbow. 5° restriction of extension	*Definite stage. C.h.* fused to metaphysis. Normal.	17 years, 6 months
7	Immobilisation 2 months	7 years, 5 months	Normal	*Definite stage. C.h.* fused to metaphysis. Normal.	14 years, 3 months
8	Immobilisation 10 days, followed by massage and exercises	6 years, 2 months	5° restriction of extension and supination	*Definite stage. C.h.* fused to metaphysis. Normal.	14 years, 11 months

9	*Sparing of elbow*	5 years, 5 months	Normal	*Growing period.* Healed. Normal. *C.h.* and *C.r.*>contralateral.	13 years, 7 months
10	Immobilisation 14 days	4 years, 7 months	Slight pain in elbow on exertion 5° restriction of flexion and 20° restriction of pronation	*Growing period.* Healed. Normal. *C.h.* and *C.r.*>contralateral.	11 years, 10 months
11 [1]	a. Immobilisation 8 days b. None	a. 4 years, 10 months b. No symptoms	a. Elbow tires soon b. Normal	*Growing period.* Healed, bilat., normal. *C.h.* and *C.r.* on right side>contralateral.	12 years, 8 months
12	Sparing of elbow	4 years, 5 months	Normal	*Growing period.* Healed. Normal. *C.h.* and *C.r.*>contralateral.	10 years, 7 months
13	Immobilisation 2 months	3 years, 9 months	Occasional slight pain in elbow	*Growing period.* Healed. Normal. *C.h.* and *Ol.*>contralateral.	11 years, 11 months
14	Immobilisation some days	3 years, 10 months	Elbow tires soon. Slight pain on forced passive ab.-adduction	*Growing period.* Uneven structure and outline of *C.h.* *C.h.*, *C.r.* and *Tr.*>contralateral.	10 years, 4 months
15	Immobilisation 1 month + 1 week	3 years, 9 months	Elbow tires soon	*Growing period.* Healed. Normal. *C.h.*, *C.r.* and *Tr.*>contralateral.	13 years, 7 months
16	Immobilisation 1 month followed by exercise	3 years, 9 months	Occasional fatigue in elbow	*Growing period.* Nearly normal. *C.h.*=Structure and outline slightly uneven. *C.h.* and *C.r.*>contralateral.	11 years, 1 month
17	Sparing of elbow	3 years, 5 months	Burning pain in elbow on exertion. Affected arm tires soon. Tender radially in elbow, slight pains on maximal supination. 5° restriction of extension.	*Growing period.* Healed. *C.h.*=Fine rounded outline missing. *C.h.*, *C.r.*, *Tr.* and *Ol.*>contralateral.	12 years, 8 months

[1] Bilateral case.

TABLE 20 (continued)

Case No.	Treatment	Interval from onset of symptoms to review	Review		Age at time of review
			Clinical	Roentgenologic	
18	Sparing of elbow	2 years, 8 months	Normal	*Growing period.* Healed. Normal. *C.h.*, *C.r.* and *Tr.*>contralateral.	12 years, 5 months
19	Sparing of elbow	2 years, 9 months	Normal	*Growing period.* Healed. *C.h.*=Fine rounded outline missing. *C.h.* and *C.r.*>contralateral.	9 years, 6 months
20	Sparing of elbow	3 years, 10 months	Elbow tires soon. 5° restriction of extension.	*Growing period.* Healed. Normal. *C.h.* and *C.r.*>contralateral.	10 years, 9 months
21	Exercises	2 years, 10 months	10° restriction of flexion	*Growing period.* Healed. Normal. *C.h.*, *C.r.* and *Ol.*>contralateral.	10 years, 7 months
22	Exercises	2 years, 1 month	Elbow tires soon. Tender over radial joint. Slight pain on forced passive extension-flexion. 5° restriction of flexion.	*Healing period.* *C.h.*=Structure and outline uneven. *C.r.* and *Ol.*>contralateral.	9 years, 5 months
23	None	2 years, 4 months	Occasionally tired, slightly painful and swollen in region of elbow. Light atrophy of arm, strength reduced. 30° restriction of extension, 10° of pronation. Slightly increased cub.valgus on affected side	*Healing period.* *C.h.*=Structure and outline uneven. *C.h.*, *Tr.* and *Ol.*>contralateral.	11 years, 1 month

24	Sparing of elbow	2 years (Last X-ray exam. 3 years, 6 months after onset)	10° restriction of flexion	(At last X-ray exam. 3 years 6 months after onset) *Growing period.* Nearly normal. *C.h.*=Structure uneven, outline slightly uneven.	8 years, 8 months
25	None	2 years, 5 months	Normal	*Growing period.* Nearly normal. *C.h.*=Structure uneven. *C.h.*, *C.r.* and *Med. epicond.*>contralateral.	10 years, 11 months
26	Sparing of elbow	1 year, 10 months	5° restriction of flexion	*Growing period.* Nearly normal. *C.h.*=Structure uneven. *C.h.* and *C.r.*>contralateral.	8 years, 10 months
27	Sparing of elbow	6 months (Last X-ray exam. 1 year, 4 months after onset)	10° restriction of flexion and 5° of extension	(At last X-ray exam. 1 year 4 months after onset) *Growing period.* Nearly normal. *C.h.*=Structure uneven, outline slightly uneven.	5 years, 2 months

with the help of a sling), (cases 3, 4, 5, 6, 9, 12, 17, 18, 19, 20, 24, 26, 27). In cases 21 and 22, however, the patients received physical exercise of the joint as the only treatment.

In cases followed until healing of the process no definite relation was found between the type of treatment received and the clinical and roentgenologic findings at the last examination. In other words, no certain difference was found between the end-results after immobilisation, sparing of the elbow, no treatment at all, and treatment with exercises only.

Neither was any definite relation found between type of treatment and the duration of appreciable clinical symptoms (Tables 20 and 15) or between type of treatment and interval between onset of symptoms and roentgenologic recovery (Tables 20 and 17). It thus appears that treatment had no demonstrable effect on the duration of appreciable symptoms, on the duration of the pathologic process or on the clinical and roentgenologic findings at the review.

Pathologic investigations on record of O.d.j. in other locations have shown, as mentioned, that the epiphysis involved is soft for a certain period and therefore sensitive to influences of form-changing nature. JONSÄTER (1953) showed that in case of O.d.j. capitis femoris the epiphysis was softest during the roentgenologic initial stage. But it is also known that the capitulum humeri forms part of a non-weightbearing joint, which in childhood is not exposed to any severe strain. Part of the physiologic pressure against the distal end of the humerus is also taken up by the ulnar part of the trochlea. All this shows that the epiphysis of the capitulum humeri is not exposed to mechanical strain of such a degree as the epiphysis of the femoral head, for example.

It is therefore believed that O.d.j. capituli humeri indicates no other treatment than sparing of the affected elbow. During the acute stage of the clinical symptoms while the patient has appreciable elbow pain, the elbow may be spared by resting it in a sling, but only as long as the subjective symptoms are acute. During that period the roentgenograms show the resistance of the epiphysis to be most decreased against influences of form changing nature, *i.e.*, while the roentgenogram shows the process in the initial and fragmentation stage, the patient should be instructed not to strain the elbow in order to decrease the risk of the process healing with deformity of the capitulum humeri. Physical treatment is not necessary, because after the pain has disappeared the child mobilises the joint spontaneously.

Recapitulation

1. 13 of the patients in the present series were treated with sparing of the elbow joint, in 10 cases the elbow was immobilised for a varying period. Two were treated with exercises from the beginning, but 3 elbows received no treatment at all.

2. No certain relationship was found between type of treatment and duration of appreciable symptoms, interval between onset of symptoms and roentgenologic recovery or findings at the review.

3. O.d.j. capituli humeri indicates no other treatment than sparing of the affected elbow. During the acute stage of the clinical symptoms the elbow should be rested in a sling, but only until the acute subjective symptoms have abated.

During the initial and fragmentation stage the patients should be instructed not to strain the elbow.

Physical treatment is hardly necessary because the child mobilises the joint spontaneously.

10. Findings at review

Prognosis

It is clear from Table 20 that the period between the onset of symptoms and the review varied between 6 months (case 27) and 22 years 11 months (case 1). As mentioned, however, in case 1 the patient had rheumatic fever during the period that the roentgenogram showed O.d.j. capituli humeri in the healing stage. In that case therefore, it cannot be excluded that the rheumatic fever might have been responsible, at least in part, for the severe joint deformity and the pronounced local clinical symptoms observed at the review. That the condition in this case was not due entirely to O.d.j. capituli humeri was confirmed further by the fact, that case 1 is the only one in the entire series in which healing of the process of the capitulum humeri was followed by severe permanent deformity. Thus, although case 1 can be used for roentgenologic evaluation of the course of O.d.j., it cannot be used for the evaluation of the late results of the disease. Case 2 is thus the oldest case that could be used in this respect (age at review 25 years 10 months) with an interval of 17 years 10 months between the onset of symptoms and review.

As to cases 24 and 27, it should be pointed out that the roentgen findings included in Table 20 were made in roentgenograms taken after the review proper.

Table 20 shows that in the first 8 cases in the table the capitulum humeri had reached the definite stage by the time of the review. Thus, cases 2 and 3 (case 1 is not used in this respect) were full-grown by the time of the review and in cases 4, 5, 6, 7 and 8 the ossific nucleus of the capitulum humeri had fused with the surrounding bone. All of these patients, who were followed until the definite stage, showed roentgenologically normal elbows at the review. Cases 3, 4, 5 and 7 also had clinically normal elbows, while in cases 2, 6 and 8 the patients had slight clinical local symptoms but of no practical importance. In these cases, in all of which ossification had been completely or almost completely concluded around the elbow joint by the time of the review, the roentgenologic appearance of both elbows was the same. As mentioned, this argues against the advance in the ossification of the affected joint during the active stage of the process and growth period (Table 20) having any influence on the final roentgenologic appearance of the elbow (Fig. 59).

In the other 19 cases in the author's series (cases 9—27 incl.) the ossification of the distal end of the humerus was not complete by the time of the review. Of these cases Nos. 22 and 23 showed the process in the roentgenologic healing period.

In cases 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 24, 25, 26 and 27 the process in the capitulum humeri had healed or almost healed by the time of the last roentgen examination. In cases 9, 10, 11, 12, 13, 15, 18, 20 and 21 the ossific nucleus of the capitulum humeri on that occasion was of normal roentgenologic appearance. In cases 14, 16, 24 and 27 the ossific nucleus of the capitulum humeri showed a somewhat uneven structure and outline at the last roentgen examination, in cases 25 and 26 the structure was uneven and in cases 17 and 19 the normal fine rounded outline of the ossific nucleus was missing.

None of these cases followed until healing of the process, and which were thus in the growing period at the last roentgen examination, showed any appreciable local clinical symptoms at the review except case 17, which on review 3 years 5 months after the onset of symptoms, showed relatively pronounced subjective symptoms, and case 10, which 4 years 7 months after the onset of symptoms showed restriction of pronation of 20°.

None of 24 [1] patients in the present material that were followed until

[1] Cases 1, 22 and 23 not included.

healing of the process or still longer showed any severe deformation of the capitulum humeri at the last roentgen examination. In those cases in which the structure and/or outline of the ossific nucleus of the capitulum humeri was uneven at the last examination, it cannot be excluded that the nuclei recovered normal appearance after the last examination, because the patients were still growing.

It thus appears that the capitulum humeri recovers its normal or practically normal roentgenologic structure and outline after the process has healed. This is in agreement with the literature cases followed until the process had healed. (See Chapter III, 7.)

It is thus clear from the literature cases and the present series, that O.d.j. capituli humeri does not result in such severe residual deformity of the epiphysis as the process sometimes does in O.d.j. of the caput femoris (coxa plana). Why the end results of O.d.j. of capitulum humeri should be better than in O.d.j. of the caput femoris might possibly be due to the difference in mechanical strain to which these two endochondral ossification centres are subjected during the period of decreased strength of the ossific nucleus.

The above assumption that the good end results of O.d.j. capituli humeri may be due to the fact that the epiphysis of the capitulum is not exposed to appreciable influences of a form-changing nature, may also suggest the value of prevention of weight-bearing in the treatment of O.d.j. of the caput femoris.

PROGNOSIS of O.d.j. capituli humeri as judged from the present series, appears to be good quo ad sanationem et restitutionem.

That an elbow that has been affected by O.d.j. capituli humeri should be more apt to develop arthrotic changes later in life was not supported by the present material. It should, however, be mentioned that the oldest patient in the present material in which this might be evaluated was only 25 years old at the time of the review.

Recapitulation

1. The interval between the onset of symptoms and the review of cases that could be used for evaluation of the end-results, varied between 6 months and 17 years 10 months.

2. At the review 7 of these patients showed the definite stage with full-grown capitulum humeri. All of them had roentgenologically normal elbows and none had any clinical symptoms of importance.

3. These patients followed until the definite stage showed the same

roentgenologic appearance of both elbows. This argues against the advance in the ossification of the affected joint having any influence on the final roentgenographic appearance of the elbow.

4. None of the cases followed until the process had healed, but in which the capitulum humeri was not full-grown by the time of the last roentgen examination, showed any severe deformation of the ossific nucleus affected. None of these patients, except 2, had any appreciable symptoms at the review.

5. Judging from the literature and the present material, O.d.j. capituli humeri does not produce any severe permanent deformity.

6. The better end-results in O.d.j. capituli humeri than of O.d.j. capitis femoris may be due to the fact, that the growing capitulum humeri epiphysis is not exposed to appreciable influences of a form-changing nature. If this assumption should be true, it may also suggest the value of prevention of weight-bearing in the treatment of O.d.j. in other locations such as in caput femoris.

7. The prognosis of O.d.j. capituli humeri is good quo ad sanationem et restitutionem.

SUMMARY

Part I

Literature studies. Since the beginning of the century, when Osteochondrosis deformans juvenilis (O.d.j.) was classified as a separate clinical entity, changes have been described in different endochondral ossification centres in the growing human skeleton and claimed by the respective authors to belong to this group of diseases.

In the meantime it has, however, been discovered that in many centres endochondral ossification is normally uneven. The roentgenographic appearance of such an ossification centre may resemble that of O.d.j., with which it has often been confused. This uneven normal mineralisation may make it difficult to establish a diagnosis of O.d.j. on the basis of a single roentgen examination.

There is general agreement that O.d.j. of the femoral head is the classical form of this group of diseases. Of the similar changes seen in other ossification centres, the lesions in the capitulum humeri are those that most closely resemble O.d.j. of the femoral capital epiphysis. Despite the unavailability of histological evidence from the lesion in capitulum humeri, there appears to be reasonable roentgenologic and clinical evidence to assume that the processes in the capitulum humeri and the femoral head are simply two different local manifestations of one and the same pathologic process. The lesion in the capitulum humeri is a typical manifestation of Osteochondrosis deformans juvenilis.

A search of the literature failed to reveal any descriptions of irregular normal mineralisation of the ossific nucleus of the capitulum during the period, in which O.d.j. capituli humeri is known to appear.

Perusal of the literature revealed only 19 publications describing certain cases of O.d.j. capituli humeri. These publications contain altogether 30 cases of the disease. No single author had described more than 4 cases and most of them only 1. Only 2 cases were followed up for more than 3 years.

An analysis was made of the material on record with reference to frequency, age at time of first examination, sex incidence, side involved,

clinical picture, roentgenologic appearance, duration of symptoms and of roentgenologic signs of pathologic process, treatment and prognosis of the disease.

The various theories on the etiology of O.d.j. of the capitulum humeri and other endochondral ossification centres are discussed under three headings: I. Circulatory disorders of the ossification centre caused by trauma, embolism, endarteritis obliterans, neurogenic causes or infection. II. Primary fracture of the ossific nucleus. III. Endogenous factors.

There appears to be reasonable evidence to assume that O.d.j. of the capitulum humeri is an aseptic necrosis in the growing ossification centre.

Part II

Experimental studies. The purpose of this part of the investigation was to elucidate some problems bearing on the nutrition of the unossified distal chondroepiphysis of the humerus, as well as, the vasculature in osseous tissue in the growing and full-grown distal end of humerus, which may also be of interest in the investigation of the vasculature of long bones in general, and to shed light on some details in the etiology of O.d.j. capituli humeri, which might also be of interest in the etiology of O.d.j. elsewhere.

Review of the earlier investigations of these problems showed that there are still many details in the circulation of the growing and full-grown skeleton, that are not yet properly understood. Knowledge of the vascular anatomy of the distal end of the humerus in childhood and adult age is incomplete.

To elucidate the above mentioned problems injection experiments were performed.

The following methods were used: A. Injection of radiopaque material into vessels supplying the distal end of the humerus, after which stereoscopic angiograms were taken. This was the main method used. B. Injection of acrylic mass with subsequent destruction of soft tissues. This method was used in few cases only.

The material used consisted of 31 human cadavers of subjects aged 3 months to 80 years. The total number of acceptable preparations was 28.

The following observations with respect to the vasculature of the distal end of the growing and full-grown humerus were made:

1. The temporary hyaline cartilage in the chondroepiphysis of the

distal end of the humerus contains several non intercommunicating vessel canals before roentgenologic demonstration of ossification is possible in that part of the cartilage where the canals may be demonstrated. It was roentgenologically demonstrated that the temporary hyaline cartilage in a growing chondroepiphysis has its own vessels for nutrition of the mass of cartilage. As the mass of cartilage of the chondroepiphysis of the distal end of the humerus decreases in volume by growth of the ossific nuclei, other intrachondral vessels disappear except those supplying the ossific nuclei.

2. Vessels in the distal humeral chondroepiphysis extend to the future site of the ossification nuclei of the capitulum and trochlea, before bone salts deposited there have reached a concentration high enough to be demonstrable in the roentgenogram. The ossific nuclei of the capitulum and trochlea appear in regions in the chondroepiphysis which, judging by the present angiographic studies, are just as well vascularised as the other parts of temporary cartilage ossified later. The present findings argue against the hypothesis that the precipitating factor of endochondral epiphyseal ossification is lack of nutrition of the hyaline temporary epiphyseal cartilage (chondroepiphysis).

3. As long as the ossific nucleus of the capitulum of the humerus lies isolated in the chondroepiphysis, it appears to receive its blood supply via a few vessels entering the cartilage from behind and breaking up into a vascular network within the nucleus of ossification. During the period of growth the vessels of the nucleus do not appear to be in direct communication with the intra-osseous vessels of the metaphysis or with cartilage vessels in the surrounding non-ossified part of the distal humeral chondroepiphysis. The vessels of the ossification centre of the capitulum are not anatomic end-vessels, but owing to their anatomic isolation during the period of growth, they may be regarded as functional end-vessels during that period.

4. The topography of the vascular system forming in the capitulum and trochlea during the period of ossification persists practically unchanged in the adult bone. After the ossification of the cartilage between the various ossific nuclei of the epiphysis and of the epiphyseal plate, the intra-osseous blood vessels of the epiphysis, metaphysis and diaphysis intercommunicate. The main vessels of the capitulum do not shift away from the region of the site of the epiphyseal plate during the period of growth, which supports the view that an epiphyseal ossific nucleus grows by new-formation of bone under the articular surface

and not from the epiphyseal plate. The vascular topography of the capitulum is strikingly constant. This may be due to the anatomic relationship between the attachment of the joint capsule at the dorsal edge of the joint cartilage and the epiphyseal plate on the dorsal aspect of the capitulum. Despite anastomoses between different vascular systems of the distal end of the full-grown humerus, it is still possible to distinguish between the main vascular regions seen before the bony union between the different parts of the end of the bone. The calibre of intra-osseous vessels in the full-grown bone was not found to decrease with advancing age.

5. Intercommunicating intra-osseous vessels were seen in the metaphysis of the growing and full-grown bone. The main difference between the intra-osseous vasculature in the distal end of the growing and full-grown humerus is that, in addition to the development of epiphyseal-metaphyseal communications in adults, the calibre of the vessels in the metaphysis is larger during the period of long growth, both absolutely and relatively, than after conclusion of growth in length. This suggests the rôle of the metaphyseal network in growth in length and relates this to the metaphyseal side of the epiphyseal plate because no corresponding difference was found between the vasculature of the epiphysis in the developing and full-grown bone. Vessels running distally of the epiphyseal plate to the ossific nucleus of the capitulum humeri and vessels running proximally of the epiphyseal plate to the metaphyseal bone may be branches of one and the same main vessel. In many trans- or supra-condyle fractures of the humerus in children the juxta-epiphyseal vessels of the metaphysis must play an important rôle in the nutrition of the distal metaphyseal fragment.

6. The growing epiphysis of the distal end of the humerus contains vessels, which traverse the outer perimeter of the epiphyseal growth plate both before and after ossification has begun in the chondroepiphysis. In the present experiments, angiography of the distal end of the humerus of growing individuals aged 3 months to 9 years showed no definite signs of direct vascular communication, through the epiphyseal plate, between the vasculature in the epiphysis and the intra-osseous vascular network of the metaphysis.

7. The pattern of the vasculature adjoining the metaphyseal side of the cartilaginous epiphyseal growth plate in the distal end of the humerus is different from the intra-osseous vascular pattern adjoining the epiphyseal side of the plate. Angiography showed no vessels in the cen-

tral parts of the roentgenologically defined cartilaginous epiphyseal plate.

8. The intra-osseous main vessels in the capitulum humeri, which enter this part of the skeleton from behind, appeared to be branches from descending extra-osseous vessels. These extra-osseous vessels are, at least sometimes, branches from arteria collateralis ulnaris inferior. Before entering the posterior part of the capitulum the last extra-osseous segment of these vessels appeared to be without appreciable intercommunication with the other vessels in the rete articulare. Results of the study of the extra-osseous vasculature agreed with the findings in the intra-osseous angiograms. The findings in these two investigations support one another.

Concerning the etiology and pathogenesis of Osteochondrosis deformans juvenilis capituli humeri the following conclusions were made on the basis of the present experimental investigation: During the age in which O.d.j. of the capitulum humeri is known to appear the vascular system supplying the ossific nucleus of the capitulum is vulnerable. During this period the ossific nucleus of the capitulum appears to be supplied by 2—3 vessels. These vessels are not anatomic end-vessels, but since they are not in communication with the other vascular systems within the bone, they may be regarded as end-vessels from a functional point of view. Although the blood supply to the ossific nucleus in the growing capitulum is sufficient to cope with the normal variation of physiologic processes, the factors of safety are decreased during this period of life owing to the lack of intra-osseous anastomoses. While the volume of the chondroepiphysis of the capitulum is large in relation to the ossification centre of the capitulum, the temporary cartilage near the ossific nucleus is relatively richly vascularised. It is believed that this might explain why O.d.j. capituli humeri has never been seen in children below 4 years. However, when the volume of the chondroepiphysis of the capitulum has decreased by growth of the ossific nucleus of the capitulum, practically all the cartilage vessels have disappeared from the environments of the ossific nucleus. The nucleus is then surrounded by avascular cartilage and is supplied by only 2—3 vessels. It is during this stage of skeletal development that O.d.j. of the capitulum is known to appear. When the ossific nucleus of the capitulum is no longer isolated after bony fusion with the adjacent epiphyseal and metaphyseal bone, intra-osseous communications have arisen between the vascular system of the capitulum and other epiphyseal and the meta-

physeal vessels. O.d.j. of the capitulum has never been observed to appear after the age at which this fusion occurs. Vessels to the ossific nucleus of the capitulum and vessels coming from behind and running to the metaphyseal bone may be branches of one and the same extraosseous vessel. This might help to explain the metaphyseal changes observed in O.d.j. of the capitulum humeri. None of the experimental results showed any of the main vessels of the ossific nucleus of the capitulum to enter the joint cavity. This argues against mild synovitis of the elbow joint exerting a direct influence on the vessels. The conclusions made regarding the etiology and pathogenesis of O.d.j. of the capitulum humeri may, to a certain extent, also hold for O.d.j. in other locations such as in the femoral head.

The radial part of the trochlea is ossified from the ossific nucleus of the capitulum humeri. Developmental disorders, deformation or displacement of the ossification nucleus of the capitulum might thus feasibly result in malformation of the radial part of the trochlea humeri, including the crista lateralis.

Part III

Clinical and roentgenologic review. This part consisted of a review of 27 cases of Osteochondrosis deformans juvenilis capituli humeri. The material was collected from a total of 22 hospitals.

The present material together with the 30 cases reported in the literature brings the total number of cases up to 57. O.d.j. capituli humeri thus appears to be a rarely diagnosed disease.

The age at first examination in the present material and in cases on record, varied between 4 and 10 years. The mean age at time of first examination in the present material was 7.4 years, in the literature material it was 7.6 years, in a total material of 55 cases (present cases + literature cases) it was 7.5 years.

O.d.j. capituli humeri is more common in boys than in girls (ratio 9:1). It is usually unilateral and more common on the right side than on the left.

Familial occurrence of the disease was described here for the first time.

All of the patients in the present material belonged to generally healthy families and all had felt well during the period before the onset of the symptoms. All had clear, local clinical symptoms. The commonest clinical picture was painful unilateral restriction of flexion-extension of

the elbow joint. The elbow was usually swollen and tender to palpation. Restriction of pronation-supination was not common. The clinical picture coincided with that described by other authors, and the interval between onset of symptoms and disappearance of all or practically all symptoms varied between 2 months and over 2 years.

Roentgen examination showed a typical pathognomonic roentgenogram of a process following a definite cycle with destruction and later regeneration of the ossific nucleus of the capitulum. The roentgen findings closely resembled those in O.d.j. capitis femoris and can be classified according to WALDENSTRÖM's roentgenologic classification. Arthrography performed in one case showed that despite marked disintegration of the ossific nucleus of the capitulum humeri, the capitulum chondroepiphysis retained its normal outline and size. Metaphyseal rarefactions of shorter duration than the process in the capitulum were observed. O.d.j. capituli humeri appeared to stimulate growth of the ossific nuclei in the affected elbow. Roentgenologic signs of increased amount of fluid in the affected joint were observed during the initial stage of the process. In those cases in which the process had healed by the last examination the roentgenograms were of practically normal appearance in all except one, in which the patient had in the meantime had rheumatic fever.

The evolutionary period may be assumed to last between about 3 months and 2 years, the healing period between 4 months and more than 3 years.

As to the etiology and pathogenesis the present clinical and roentgenologic study together with the experimental investigation suggested as follows. The roentgenologic appearance of the process as well as the close similarity between the clinical and roentgenologic picture and that of O.d.j. capitis femoris indicates that the disease may be classified as necrosis of the ossific nucleus of the capitulum. The experimental investigation showed that the vascular anatomy is such that a total necrosis of the ossific nucleus of the capitulum might be produced by circulatory disorders in a few vessels. The roentgenologic rarefactions seen in the metaphysis of the distal humerus and the process in the ossific nucleus on the other side of the epiphyseal plate may be ascribed to circulatory disorders in one and the same vascular region, sometimes in one and the same extra-osseous vessel. The metaphyseal changes thus also suggest the rôle of a circulatory disorder in the cause of the disease. Many factors in the present series argue against trauma as the sole

primary causal factor. That circulatory disorders in O.d.j. capituli humeri may sometimes be caused by trauma cannot be excluded. The present experimental findings argue against the juxta-chondral rarefaction pathognomonic of the initial stage of the disease being due to bone resorption. It may be due to a pathologic fracture in the necrotic bone, which might explain the rôle of trauma in the causation of symptoms in the present series. An increased amount of fluid in the affected joint was sometimes observed. The present experimental findings, however, argue against synovitis being of etiologic importance because the main vessels running to the ossific nucleus of the capitulum do not enter the joint cavity. All the factors arguing against trauma or fracture as being of primary importance in the etiology suggest some kind of endogenous etiologic factor, which, because of the frequently monostotic appearance in otherwise normal children, may be of local nature. The experimental investigation helped to explain why the disease occurs only during a limited period of life.

O.d.j. capituli humeri is readily diagnosed since each stage of the disease has a pathognomonic roentgenographic appearance.

The treatment (immobilisation, sparing of elbow, no treatment at all, or treatment with exercises only) had no demonstrable effect on the duration of appreciable symptoms, on the duration of the pathologic process or on the clinical and roentgenologic findings at the review. O.d.j. capituli humeri indicates no other treatment than sparing of the affected elbow. During the acute stage of the clinical symptoms the elbow should be rested in a sling, but only until the acute subjective symptoms have abated. During the initial and fragmentation stages the patient should be instructed not to strain the elbow. Physical treatment is not indicated.

The interval between the onset of symptoms and the review of cases that could be used for evaluation of the end-results, varied between 6 months and 17 years, 10 months. At the review 7 of these patients showed the definite stage with full-grown capitulum humeri. All of them showed roentgenologically normal elbows and none had any clinical symptoms of importance. These patients who were followed until the definite stage showed the same roentgenologic appearance of both elbows. This argues against the advance in the ossification of the affected joint having any influence on the final roentgenographic appearance of the elbow. None of the cases followed until the process had healed, but in which the capitulum humeri was not full-grown by the time of the

last roentgen examination, showed any severe deformation of the ossific nucleus affected. None of these patients, except 2, had any appreciable symptoms at the review. Judging from the literature and the present material, O.d.j. capituli humeri does not produce any severe permanent deformity. The better end-results in O.d.j. capituli humeri than of O.d.j. capitis femoris may be due to the fact that the growing capitulum humeri epiphysis is not exposed to appreciable influences of a form-changing nature. If this assumption should be true, it may also suggest the value of prevention of weight-bearing in the treatment of O.d.j. in other locations, such as in caput femoris. The prognosis of O.d.j. capituli humeri is good quo ad sanationem et restitutionem.

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CASE REPORTS

Reports of 27 cases in the present investigation are given in brief below.

The 30 literature cases were described in Chapter III. This publication thus gives a description of all the cases of O.d.j. capituli humeri now on record.[1]

As mentioned, the roentgenographic appearance of the disease is typical without any appreciable variation from case to case. It was therefore considered superfluous to publish all the roentgenograms of the cases described. Reproductions of typical roentgenograms of the present 27 patients are deposited at University Library, Lund, together with the records of the reviews and requestioning (in Swedish).

Abbreviations

IN. = Initial stage
FR. = Fragmentation stage
HE. = Healing period
GR. = Growing period
DE. = Definite stage
C.h. = Capitulum humeri ossific nucleus
C.r. = Capitulum radii ossific nucleus
Tr. = Trochlea ossific nucleus
Ol. = Olecranon ossific nucleus.

Case 1. (Lunds Lasarett Rtg register A.i. 21/1933). Girl, aged 6 years at first examination (17/10 1933).

Trauma. — Ten days before first examination the patient was swinging on a bar and twisted right elbow. Local joint symptoms appeared immediately.

Local symptoms. — Spontaneous pain, pain on movement, tenderness, swelling, impaired range of mobility, slight local increase in temperature. First registration of limitation of movement: Extension-flexion 155—90°. Duration of symptoms: not symptomfree at review.

Roentgen findings. — First examination showed *IN.*, later examinations: *FR.*, *HE.* and *GR.* Ossification more advanced on affected side. Metaphyseal changes. Increased amount of fluid in joint space. Signs of initial repair 6 months after onset, healed 1 year 5 months after onset of symptoms (March 1935). From 1934 increasing incongruence was noted in ulnar part of joint.

[1] After conclusion of the literature studies and while the present thesis was in press, a report of 2 further cases (from a total of 5) by KYSELKA, R. (Arch. orthop. u. Unfall-Chir. *49*: 1957—1958, pp. 379—386) was detected.

Treatment. — Cotton wool bandage, 1 month, later massage.

Complicating disease. — At end of November 1934 (during *HE.* period of O.d.j.) the patient developed, after angina, febris rheumatica with involvement of all joints. Symptoms disappeared after 14 days' bed-rest. Recurrence in October 1935. Since then no joint trouble except in elbow. Pleuritis sicca Nov. 1935 (Pediatric Clinic Lund 8/11—19/11 1935).

Local elbow symptoms did not disappear. Therefore admitted to Orthopaedic Clinic in Lund 1/7—8/7 1935 and 2/1—1/2 1936 and to Vanföreanstalten, Hälsingborg 9/6—22/10 1936. Treated with mobilisation of joint under anaesthesia and active exercise of the joint.

Review. — (22 years, 11 months after onset). Housewife, aged 28 years and 10 months. Clinical: Pain in elbow on exertion, 65° restriction of extension, 60° of flexion and 30° of pronation. Forced movements painful. Muscle atrophy of arm. Roentgenologic: *DE.*-stage Incongruence of ulno-humeral joint, deformity of capitulum humeri and radii.

Author's comment. — In this case febris rheumatica might have been at least partly responsible for the severe deformity of the joint and the pronounced local symptoms. The assumption that febris rheumatica found such a footing in the elbow joint because of O.d.j. being a locus minoris resistentiae, is strengthened by the fact that this was the only case in which the disease had produced severe permanent deformity of the joint.

Case 2. — (Malmö Allmänna Sjukhus. Rtg. reg. C.f. 73/1938). Girl, aged 8 years 1 month, at first examination (6/12 1938).

Trauma. — One month before first examination the girl had fallen on her left elbow. Elbow symptoms appeared immediately.

Local symptoms. — Spontaneous pain, which sometimes disturbed sleep, pain on movement, tenderness, swelling, impaired extension and flexion, crepitations in joint, local feeling of warmth. Duration of appreciable symptoms: 6 months.

Roentgen findings. — *IN.*-stage.

Treatment. — Cotton wool bandage and elastic bandage about 2 months.

Review. — (17 years 10 months after onset). Healthy housewife aged 25 years and 10 months. Clinical: Occassionally light pain in elbow on severe exertion. Slight pain on forced passive extension-flexion. Slight atrophy of lower arm. Roentgenologic: *DE.*-stage. Normal.

Case 3. — (Göteborgs Barnsjukhus. Rtg. registr. A.f. 257/1948). Boy, aged 9 years 3 months at first examination (7/1 1948).

Trauma. — Three months before first examination a dog had jumped against the right elbow. Elbow symptoms appeared immediately.

Local symptoms. — Pain on movement, tenderness and impaired extension-flexion. Duration of appreciable symptoms: 5 months.

Roentgen findings. — *IN.*-stage.

Treatment. — Sparing of elbow.

Review. — (10 years 8 months after onset). Healthy turner, aged 19 years, 8 months. Clinical: normal. Roentgenologic: *DE.*-stage. Normal.

Case 4. — (Ortoped. Kliniken, Vanföreanstalten, Stockholm, Rtg. reg. A.i. 5070/48). Boy, aged 7 years 1 month, at first examination (22/9 1948).

Trauma. — Weight lifting with right arm 2 weeks before first examination. Local elbow symptoms appeared immediately afterwards.

Local symptoms. — Pain on movement and limitation of extension and flexion. Duration of appreciable symptoms: 6 months.

Roentgen findings. — *IN.*, *FR.* and *HE.* Increased amount of fluid in joint. Signs of initial repair: 4 1/2 months after onset of symptoms.

Treatment. — Sparing of elbow.

Review. — (8 years 4 months after onset of symptoms). Healthy school-boy, aged 15 years 5 months. Clinical: Normal. Roentgenologic: *DE.*-stage. Normal.

Case 5. — (Krp. Lovisas Barnsjukhus, Stockholm. Rtg. reg. B.f. 7377/50). Boy, aged 7 years 8 months at first examination (23/3 1950).

No known trauma. — Onset of symptoms 3 weeks before first examination.

Local symptoms. — Right elbow: spontaneous pain, sometimes disturbing sleep, pain on movement, tenderness, swelling, limitation of movement. First registered limitation of movement: extension-flexion: 160—85°. Weakness of right arm. Duration of appreciable symptoms: 1 year.

Roentgen findings. — *IN.* Ossification more advanced on affected side.

Treatment. — Sparing of elbow.

Review. — (6 years 10 months after onset of symptoms). Healthy errand boy, aged 14 years 5 months. Clinical: Normal. Roentgenologic: *DE.*-stage. Normal.

Case 6. — (Centrallasarettet, Linköping. Rtg. reg. B.f. 12510). Boy, aged 9 years 7 months at first examination (26/4 1950).

No known trauma. — Onset of symptoms 2 months before first examination.

Local symptoms. — Right elbow: pain on movement, swelling, impaired range of movement. First registered limitation of movement: Extension-flexion: 175—35°. Slight cubitus valgus. Duration of appreciable symptoms 1 year 2 months.

Roentgen findings. — *IN.*, *FR.*, *HE.* Ossification more advanced on affected side. Signs of initial repair 7 1/2 months after onset of symptoms.

Treatment. — Massage, sparing of elbow.

Review. — (8 years 1 month after onset of symptoms). Healthy school-boy, aged 17 years 6 months. Clinical: Periodic slight weakness of elbow, 5° restriction of extention. Roentgenologic: *DE.*-stage. Normal.

Case 7. — (Lunds Lasarett, Rtg. reg. Cub.n. 34/51). Boy, aged 7 years 4 months at first examination (16/4 1951).

Trauma. — Had fallen from cycle onto right elbow 6 months before first examination. Local symptoms had appeared immediately after the fall.

Local symptoms. — Pain, swelling, limitation of movement. First registered impairment of movement: extension-flexion: 145—80°, pronation-supination 70°. Duration of appreciable symptoms: 10 months.

Roentgen findings. — *IN.*, *FR.*, *HE.* Metaphyseal change. Increased amount of fluid in joint. Signs of initial repair 10 months after onset of symptoms.

Treatment. — Cotton wool bandage 14 days, plaster splint 1 1/2 month.

Review. — (7 years, 5 months after onset of symptoms). Healthy. Son of farmer, aged 14 years 3 months. Clinical: Normal. Roentgenologic: *DE.*-stage. Normal.

Case 8. — (Centrallasarettet, Örebro. Rtg. reg. C.d. 10/51). Boy, aged 8 years 11 months, at first examination (4/6 1951).

No known trauma. — Onset of symptoms 2 months before first examination.

Local symptoms. — Right elbow: Spontaneous pain, limitation of movement. First registered limitation of movement: Extension-flexion 170—105°, pronation-supination normal. Duration of appreciable symptoms 4 1/2 months (then 10° restriction of extension).

Roentgen findings. — *IN.*

Treatment: — Cotton wool bandage 10 days. Then massage and joint exercise.

Review. — (6 years, 2 months after onset of symptoms). Healthy errand boy, aged 14 years 11 months. Clinical: 5° restriction of extension and supination of elbow. Roentgenologic: *DE.*-stage. Normal.

Case 9. — (Krp. Lovisas Barnsjukhus, Stockholm. Rtg. reg. B.i. 4555/51). Boy, aged 8 years 3 months at first examination (3/9 1951).

No known trauma. — Onset of symptoms 6 weeks before first examination.

Local symptoms. — Left elbow: Spontaneous pain on movement, "cried with pain" in the beginning. Tenderness, fluctuant swelling over olecranon, limitation of movement. First registered limitation of movement: Extension-flexion 180—50°. Slight muscle atrophy. Duration of appreciable symptoms: 4 months.

Roentgen findings. — *IN., FR., HE., GR.* Ossification more advanced on affected side. Signs of initial repair 8 months, healed 2 years 1 month after onset of symptoms.

Treatment. — Sparing of elbow.

Review. — (5 years 5 months after onset). Healthy school-boy, aged 13 years 7 months. Clinical: Normal. Roentgenologic: *GR.* Normal. *C.h.* and *C.r.*>contralateral.

Case 10. — (Centrallasarettet, Kristianstad, Rtg. reg. A.i. 10/52). Boy, aged 7 years 4 months, at first examination (8/4 1952).

Trauma. — Fell on flexed right elbow 2 months before first examination. Accident immediately followed by elbow symptoms.

Local symptoms. — Pain on movement, tenderness, limitation of flexion, pronation, supination. Duration of appreciable symptoms: 1 year 4 1/2 months.

Roentgen findings. — *IN., HE., GR.* Ossification more advanced on affected side. Increased amount of fluid in joint. Signs of initial repair 11 months after onset, almost healed 1 year 4 1/2 months after onset.

Treatment. — Immobilisation in plaster for 14 days.

Review. — (4 years 7 1/2 months after onset). Healthy school-boy, aged 11 years 10 months. Clinical: Slight pain in elbow on exertion. 5° restriction of

flexion and 20° restriction of pronation. Roentgenologic: *GR.* Normal. *C.h.* and *C.r.*>contralateral.

Case 11 (bilateral case). — (Krp. Lovisas Barnsjukhus, Stockholm, Rtg. reg. B.i. 4689/52). Boy, aged 8 years at first examination (18/4 1952).

Trauma. — Fell on flexed right elbow 2 months before first examination. Local joint symptoms appeared immediately. Left elbow: No known trauma.

Local symptoms. — Right elbow: pain on movement, swelling and limitation of movement. First registered limitation of movement: Extension-flexion: 150—90°. Duration of appreciable symptoms 1 year.

Left elbow: Never any clinical symptoms.

Roentgen findings. — First examination showed process in *IN.* in right capitulum humeri. Simultaneous control of contralateral left elbow showed changes in left capitulum humeri resembling O.d.j. in *HE.*-period. Check roentgenography one week later showed same appearance. Ossification more advanced in left elbow on first examination. Control of right elbow 3 months after onset of symptoms showed signs of initial repair. No further control of left elbow before review. Right elbow showed metaphyseal changes and increased fluid in joint.

Treatment. — Right elbow: immobilisation 8 days in plaster splint. Left elbow: not treated.

Review. — (4 years 10 months after onset of symptoms). Healthy schoolboy, aged 12 years 8 months. Clinical: Right elbow tires readily. Left elbow: normal. Roentgenologic: Right and left=*GR.* Normal. *C.h.* and *C.r.* on right side have now become larger than those on left side.

Case 12. — (Centrallasarettet, Linköping. Rtg. reg. B.i. 7809). Boy, aged 6 years 5 months at first examination (14/10 1952).

Trauma. — Twisted right arm while playing 3 months 3 weeks before first examination. Local symptoms appeared immediately after accident.

Local symptoms. — Spontaneous pain, sometimes disturbing sleep, pain on movement, tenderness, swelling, limitation of movement. Registered limitation of movement: Extension-flexion 165—85, 75°. Duration of appreciable symptoms: 1 year, then light restriction of flexion.

Roentgen findings. — *FR.* Ossification more advanced on affected side.

Treatment. — Sparing of elbow.

Review. — (4 years, 5 months after onset of symptoms). Healthy school-boy, aged 10 years, 7 months. Clinical: Normal. Roentgenologic: *GR.* Normal. *C.h.* and *C.r.*>contralateral.

Case 13. — (Centrallasarettet, Kristianstad. Rtg. reg. A.as. 3/53). Boy, aged 8 years 3 months at first examination (30/1 1953).

No known trauma. — Onset of symptoms one month before first examination.

Local symptoms. — Left elbow: spontaneous pain, tenderness, swelling, limitation of movement. First registered limitation of movement: extension-flexion 180—80°. Duration of appreciable symptoms 5 months.

Roentgen findings. — *IN., FR.* Increased amount of fluid in joint. Healed 2 years 11 1/2 months after onset of symptoms.

Treatment. — Immobilisation in plaster on 2 occasions, total 2 months.

Review. — (3 years 9 months after onset of symptoms). Healthy school-boy, aged 11 years 11 months. Clinical: Occasionally slight pain in elbow. Roentgenologic: *GR.* Normal. *C.h.* and *Ol.*>contralateral.

Case 14. — (Södersjukhuset, Stockholm, Rtg. reg. Cu.i. 132/53). Boy, aged 6 years 6 months at first examination (10/3 1953).

No known trauma. — Onset of symptoms 10 days before first examination.

Local symptoms. — Left elbow: pain on movement, swelling of joint capsule on both sides of olecranon, limitation of extension-flexion. First registered limitation of movement: 40° restriction of extension, pronation-supination normal. Duration of appreciable symptoms 2 months.

Roentgen findings. — *IN., GR.* Ossification more advanced on affected side. Roentgenologic appearance approaching normal 3 years 10 months after onset of symptoms.

Temperature 38.3° at first examination, not registered later. Aseptic necrosis in os naviculare pedis bilat. observed at second examination.

Treatment. — Immobilisation of elbow in plaster for some days.

Review. — (3 years, 10 months after onset of symptoms). Healthy school-boy, aged 10 years 4 months. Clinical: Affected elbow tired soon. Slight pain on forced passive ab-adduction. Roentgenologic: *GR.* Uneven structure and outline of *C.h.*

C.h., C.r. and *Tr.*>contralateral.

Case 15. — (Lunds Lasarett, Rtg. reg. Cub.diss. 2/53). Boy, aged 10 years 2 months at first examination (20/3 1953).

Trauma. — Fell on flexed right elbow 3 months before first examination. Local joint symptoms appeared immediately.

Local symptoms. — Pain on movement, tenderness in radial part of joint, swollen over radial epicondyle. Limitation of movement. First registered limitation of movement: Extension-flexion: 175—50°. Duration of appreciable symptoms: 10 months.

Roentgen findings. — *IN., HE.* Ossification more advanced on affected side. Signs of initial repair 6 months after onset, almost healed 10 months after onset.

Treatment. — Immobilisation in plaster 1 month 1 week.

Review. — (3 years 9 months after onset of symptoms). Healthy school-boy, aged 13 years 7 months. Clinical: Right elbow tires readily. Roentgenologic: *GR.* Normal. *C.h., C.r.* and *Tr.*>contralateral.

Case 16. — (Ortopediska Kliniken, Vanföreanstalten, Göteborg. Rtg. reg. 25627/53). Boy, aged 7 years 5 months at first examination (24/3 1953).

No known trauma. — Onset of symptoms 4 days before first roentgen examination.

Local symptoms. — Right elbow: spontaneous pain, swelling (circumference 1 cm more than on contralateral side), limited extension-flexion. Pronation and

supination: normal. Muscle atrophy of right arm (after 1 month's immobilisation in plaster). Duration of appreciable symptoms: 10 months, but still had 10° restriction of flexion.

Roentgen findings. — *IN., FR., HE.* Ossification more advanced on affected side. Signs of initial repair 5 months after onset, approaching normal appearance 3 years 9 months after onset.

Treatment. — Immobilisation in plaster 1 month. Exercises.

Review. — (3 years, 9 months after onset of symptoms). Healthy school-boy, aged 11 years 1 month. Clinical: Occasional fatigue in elbow. Roentgenologic: *GR.* Nearly normal. *C.h.*: structure and outline slightly uneven. *C.h.* and *C.r.*>contralateral.

Case 17. — (Vanföreanstalten, Härnösand. Rtg. reg. 53.560). Boy, aged 9 years 7 months, at first examination (7/10 1953).

Trauma. — Left arm twisted while playing, 4 months before first examination. Local symptoms appeared immediately after accident.

Local symptoms. — Spontaneous pain, sometimes disturbing sleep, pain on movement, tenderness, swelling, limitation of movement. First registered limitation of movement: Extension-flexion 160--70°, pronation, supination normal. Increased skin temperature, increased cubitus valgus on affected side. Duration of appreciable symptoms: 1 year 3 months.

Roentgen findings. — *IN., FR., HE.* Ossification more advanced on affected side. Signs of initial repair 9 1/2 months after onset, appearance approaching normal 3 years 5 1/2 months after onset.

Treatment. — Sparing of elbow.

Review. — (3 years 5 1/2 months after onset). Healthy school-boy, aged 12 years 8 months. Clinical: Burning pain in elbow on exertion. Affected arm tires readily. Tender radially in elbow, slight pain on maximal supination. 5° restriction of extension. Roentgenologic: *GR. C.h.*: Fine rounded outline missing. *C.h., C.r., Tr.* and *Ol.*>contralateral.

Case 18. — (Göteborgs Barnsjukhus, Rtg. reg. A.i. 33/54). Boy, aged 9 years 9 months, at first examination (27/3 1954).

No known trauma. — Onset of symtoms 14 days before first examination.

Local symptoms. — Right elbow: spontaneous pain, which sometimes disturbed sleep. Pain on movement. Tenderness over lateral epicondyle. Swelling with exsudation. Limitation of extension-flexion. First registered limitation of movement: 10—15° restriction of extension. Duration of appreciable symptoms: 1 year.

Roentgen findings. — *IN.* Ossification more advanced on affected side.

Treatment. — Sparing of elbow.

Review. — (2 years 8 1/2 months after onset of symptoms). Healthy school-boy, aged 12 years 5 months. Clinical: Normal. Roentgenologic: *GR.* Normal. *C.h., C.r.* and *Tr.*>contralateral.

Case 19. — (Göteborgs Barnsjukhus, Rtg. reg. A.i. 35a/54). Boy, aged 6 years 9 months, at first examination (20/5 1954).

No known trauma. — Onset of symptoms 5 days before first examination.

Local symptoms. — Right elbow: pain on movement, tenderness, swelling, impaired extension and flexion. Increased skin temperature locally. Duration of appreciable symptoms 3 months.

Roentgen findings. — *IN.* Ossification more advanced on affected side. Increased amount of fluid in joint. Roentgen appearance approaching normal 2 years 9 months after onset of symptoms.

Treatment. — Sparing of elbow.

Review. — (2 years 9 months after onset of symptoms). Healthy school-boy, aged 9 years 6 months. Clinical: Normal. Roentgenologic: *GR. C.h.*: fine rounded outline missing.

C.h. and *C.r.*>contralateral.

Case 20. — (Göteborgs Barnsjukhus. Rtg. reg. A.d. 42/54). Boy aged 6 years 11 months, at first examination (7/8 1954).

Strain of arm, carried a large parcel of newspapers under right arm for several consecutive days during which local symptoms developed in elbow, 2 weeks before first examination.

Local symptoms. — Spontaneous pain, pain on movement, tenderness, swelling. Impaired extension and flexion. Crepitations in joint, local increase in temperature. Duration of appreciable symptoms 3 months.

Roentgen findings. — *IN.* Ossification more advanced on affected side. Metaphyseal changes.

Treatment. — Sparing of elbow.

Review. — (3 years 10 months after onset). Healthy school-boy, aged 10 years 9 months. Clinical: Elbow tires readily. 5° restriction of extension. Roentgenologic: *GR.* Normal.

C.h. and *C.r.*>contralateral.

Case 21. — (Centrallasarettet, Örebro. Rtg. reg. C.i. 20/54). Boy, aged 7 years 10 months at first examination (24/8 1954).

No known trauma. — Limitation of movement discovered during gymnastics 1 month before first examination.

Local symptoms. — Left elbow: limited extension-flexion. Pronation and supination normal. First registered limitation of movement: extension-flexion 160—50°. Duration of appreciable symptoms 1 year.

Roentgen findings. — *IN., FR., HE.* Ossification more advanced on affected side. Increased amount of fluid in joint. Signs of initial repair 8 months after onset, healed 2 years 10 months after onset.

Treatment. — Exercises.

Review. — (2 years 10 months after onset of symptoms). Healthy school-boy, aged 10 years 7 months. Clinical: 10° restriction of flexion. Roentgenologic: *GR.* Normal.

C.h., C.r. and *Ol.*>contralateral.

Case 22. — (Krp. Lovisas Barnsjukhus, Stockholm. Rtg. reg. B.n. 20888/55). Boy, aged 7 years 6 months at first examination (3/2 1955).

Trauma. — Fell from cycle onto flexed left elbow 2 months before first examination. Local symptoms appeared immediately after accident.

Local symptoms. — Spontaneous pain, sometimes disturbing sleep, pain on movement, tenderness, swelling, limitation of movement. First registered limitation of movement: Extension-flexion: 160—40°.

Weakness of arm, increased local temperature. Duration of appreciable symptoms: 1 year 6 months.

Roentgen findings. — *IN., FR., HE.* Ossification more advanced on affected side. Signs of initial repair 1 year 6 months after onset, appearance approaching normal 2 years 1 month after onset.

Treatment. — Exercises.

Review. — (2 years, 1 month after onset of symptoms). Healthy school-boy, aged 9 years 5 months. Clinical: Elbow tires readily on exertion. Tenderness over radial joint. Slight pain on forced passive extension-flexion. 5° restriction of flexion. Roentgenologic: *HE.*

C.h.=structure and outline uneven. *C.r.* and *Ol.*>contralateral.

Case 23. — (Centrallasarettet, Örebro. Rtg. reg. C.i. 4/55). Boy, aged 8 years 10 months at first examination (22/2 1955).

No known trauma. — Onset of symptoms 3 weeks before first examination.

Local symptoms. — Left elbow: spontaneous pain, pain on movement, tenderness, swelling, limited extension and flexion. Duration of appreciable symptoms: Not symptom-free 2 years 4 months after onset.

Roentgen findings. — *IN.* Ossification more advanced on affected side. Approaching normal 2 years 4 months after onset.

Treatment: None.

Review. — (2 years, 4 months after onset of symptoms). School-boy, aged 11 years 1 month. Clinical: Occasionally tired, slightly painful and swollen in region of elbow. Light atrophy of arm, strength reduced. 30° restriction of extension, 10° of pronation. Slightly increased cub. valgus on affected side. Paresis of lower limbs after poliomyelitis. Roentgenologic: *HE.*

C.h.=structure and outline uneven. *C.h., Tr.* and *Ol.*>contralateral.

Case 24. — (Centrallasarettet, Linköping. Rtg. reg. Cu.l.m. 1). Boy, aged 7 years 8 months, at first examination (11/6 1956).

Trauma. — Fell on right elbow 1 year before first examination. Local symptoms appeared immediately.

Local symptoms. — Pain and tenderness on radial part of joint. Limitation of movement. First registered limitation of movement: Extension-flexion: 165—65°. Pronation and supination normal. Duration of appreciable symptoms: 2 years (then 10° restriction of flexion).

Roentgen findings. — *IN., FR., HE.* Ossification more advanced on affected side. Signs of initial repair 2 years after onset, approaching normal 3 years 6 months after onset. (Roentgen after review.)

Treatment. — Sparing of elbow.

Review. — (2 years after onset of symptoms). Healthy school-boy, aged 8 years 8 months. Clinical: 10° restriction of flexion. Last roentgen examination (3 years 6 months after onset of symptoms): *GR.*

C.h.=structure uneven, outline slightly uneven.

Case 25. — (Centrallasarettet, Borås, Ortoped. avd. Rtg. reg. 48.121a/56). Boy, aged 9 years at first examination (5/7 1956).

Trauma against right arm 6 months before first examination. Local symptoms appeared immediately.

Local symptoms. — Pain on movement, tenderness, swelling, limitation of movement. First registered limitation of movement: Extension-flexion 165—45°. Duration of appreciable symptoms 1 year.

Roentgen findings. — *FR.* Approaching normal appearance 2 years 5 months after onset.

Treatment: — None.

Review. — (2 years 5 months after onset of symptoms). Healthy school-boy, aged 10 years 11 months. Clinical: Normal. Roentgenologic: *GR.*

C.h.=structure uneven. *C.h.*, *C.r.* and *med. epicondyle*>contralateral.

Case 26. — (Centrallasarettet, Linköping. Rtg. reg. Cu.d.2). Boy, aged 7 years 3 months at first examination (14/6 1957).

No known trauma. — Onset of symptoms about 3 months before first examination.

Local symptoms. — Right elbow: Pain on movement, tenderness, limitation of movement. First registered limitation of movement: Extension-flexion 170—60°. Pronation and supination normal. Weakness of arm. Duration of appreciable symptoms: 5 months.

Roentgen findings. — *FR.* Ossification more advanced on affected side. Approaching normal appearance 1 year 10 months after onset of symptoms.

Treatment. — Sparing of elbow.

Review. — (1 year 10 months after onset of symptoms). Healthy school-boy, aged 8 years, 10 months. Clinical: 5° restriction of flexion. Roentgenologic: *GR.* Nearly normal.

C.h.=structure uneven. *C.h.* and *C.r.*>contralateral.

Case 27. — (Centrallasarettet, Linköping. Rtg. reg. Cu.l.m. 13). Girl, aged 4 years 11 months at first examination (16/10 1957).

No known trauma. — Onset of symptoms 3 months before first examination.

Local symptoms. — Left elbow: spontaneous pain, pain on movement, tenderness, swelling, limited extension and flexion. Pronation and supination normal. First registered limitation of movement: Extension-flexion 160—75°. Duration of appreciable symptoms: 5 months.

Roentgen findings. — *IN.*, *FR.*, *HE.* Ossification more advanced on affected side. Metaphyseal changes. Signs of initial repair 8 months after onset, appearance approaching normal 1 year 4 1/2 months after onset. (Roentgen after review.)

Treatment. — Sparing of elbow.

Review. — (6 months after onset of symptoms). Healthy girl, aged 5 years 2 months. Clinical: 10° restriction of flexion and 5° of extension. Last roentgen examination (1 year 4 1/2 months after onset of symptoms)=*GR.* Nearly normal.

C.h.=structure uneven, outline slightly uneven.