AVASCULAR NECROSIS
AFTER SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

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Necrosis in the femoral head after slipping of the upper femoral epiphysis has always been regarded as serious. This paper is a study of hips with slipped upper femoral epiphyses that showed evidence of necrosis either when first seen or during treatment. The objects were to find out whether the necrosis affected bone or articular cartilage, or both, whether there was any common factor in the patients in whom necrosis occurred, and to what extent treatment influenced the ultimate state of the hip.

REVIEW OF LITERATURE

The reported incidence of avascular necrosis after slipping of the upper femoral epiphysis treated by different methods has varied. Details of patients showing necrosis are not often mentioned, and few radiographic findings have been shown in the reports. As Jerre (1950) observed, many authors have not distinguished between avascular necrosis of the femoral head and early necrosis of articular cartilage unaccompanied by any radiological signs of necrosis of bone; nor has a distinction always been made between early necrosis of articular cartilage and secondary degenerative arthritis.

The term "aseptic" necrosis of bone appears to have been used first by Axhausen (1922), who described it in the femoral head after fracture of the femoral neck in a child; he also thought that similar changes occurred after slipping of the upper femoral epiphysis. Waldenström (1930) recognised the existence of a type of joint change after slipping of the upper femoral epiphysis which involved necrosis of the articular cartilage without radiological evidence of avascular necrosis of the capital epiphysis. His paper described three such cases that he had seen in 1923. Jones and Roberts (1934) clarified the radiological diagnosis of avascular necrosis of bone in terms of contrasting bone densities caused by different blood supplies; the area of bone that is deprived of its blood supply retains its calcium salts and hence its density on radiography, in contrast to the surrounding bone which retains its normal blood supply and undergoes disuse demineralisation with calcium absorption and which therefore appears rarefied in radiographs.

Moore (1945a) gave the first detailed description of the pathology of avascular necrosis in the femoral epiphysis after slipping, from a careful study of two specimens obtained at operation. He also reported one patient with necrosis of the articular cartilage of both hips in a series of forty-four slipped upper femoral epiphyses. Ponseti and Barta (1948) reported three patients with necrosis of the articular cartilage after slipping of the upper femoral epiphysis, and Jerre (1950), in a series of 200 slipped femoral epiphyses—treated mainly by closed reduction and plaster immobilisation—found twenty hips with avascular necrosis and nine with necrosis of the articular cartilage. Hall (1957) reviewed 173 hips with slipping of the femoral epiphysis from various orthopaedic centres; twenty-seven of these showed avascular necrosis of the capital epiphysis, and three had necrosis of the articular cartilage.

CLINICAL MATERIAL

The material on which this paper is based is drawn from a series of 100 cases of slipped upper femoral epiphysis treated at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, and at the Prince of Wales Orthopaedic Hospital, Cardiff, in twenty-five years. Of these, twenty-one hips in twenty patients developed necrosis of the bony epiphysis (Table I)
or the articular cartilage (Table II). The diagnosis of avascular necrosis of bone was made on the radiological finding of increased density of the epiphysis in six hips. The diagnosis of articular cartilage necrosis—based on the radiological finding of diminished joint space and the clinical findings—was made in fifteen hips and confirmed at operation in three. In some patients the changes of articular cartilage necrosis were diagnosed when the patient was first examined. In others—and in all with bone necrosis—the changes developed within the first year. Figures 1 and 2 show examples of the two types of necrosis.

**FIG. 1**

The two types of necrosis after slipping of the upper femoral epiphysis. Figure 1 (Case 8) shows necrosis of the articular cartilage with loss of joint space and porosity of the head. Figure 2 shows necrosis of the upper half of the bony epiphysis, but with a good joint space (Case 3).

The small number of hips with avascular necrosis of the epiphysis is in contrast to that in other published series. Perhaps this can be explained in part by the failure of some authors to distinguish between the two types of necrosis. It is also possible that some bone necrosis may have been missed in this series by infrequent radiography, but this is unlikely because the radiographic changes of altered density usually become apparent in the early months, and most of these patients were in hospital at that time undergoing radiographic examination at monthly intervals.

The two forms of necrosis will be considered separately because, although they cannot be separated into two distinct pathological types, they appear as separate clinical entities with different radiological appearances.

**AVASCULAR NECROSIS OF THE CAPITAL EPiphYSIS**

There seems general agreement that if the blood supply to the capital epiphysis is impaired, partial or total death will occur in the tissues of the epiphysis depending on how much the blood supply is diminished. Waldenström (1934) separated the capital epiphysis from its connections with the femoral neck and the ligamentum teres, and then replaced it on the neck and held it there by means of a bone peg hoping that it would unite as a free bone graft. He did this twice but avascular necrosis of the head occurred in both. Zemansky and Lippmann (1929) cut the artery of the ligamentum teres in adolescent rabbits and found that this caused anaemic bone necrosis in the femoral head. Harris and Hobson (1956) showed, also in rabbits, that necrosis followed experimental displacement of the upper femoral epiphysis.
TABLE I
CLINICAL DETAILS IN SIX PATIENTS (SIX HIPS) WITH AVASCULAR NECROSIS OF THE FEMORAL HEAD AFTER SLIPPING OF THE EPIPHYSIS

<table>
<thead>
<tr>
<th>Case number</th>
<th>Sex</th>
<th>Age in years</th>
<th>Avascular necrosis Onset</th>
<th>Duration</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>15</td>
<td>At 3 weeks</td>
<td>3 months</td>
<td>Good 1 year and 16 years later</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>14</td>
<td>At 5 weeks</td>
<td>13 months</td>
<td>Good 1½ years and 9 years later</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>16</td>
<td>At 6 weeks</td>
<td>5 months</td>
<td>Good function 5 years later</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>9</td>
<td>Before 3 months</td>
<td>2 years</td>
<td>Good 15 years later</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>16</td>
<td>Before 5 months</td>
<td>1 year</td>
<td>Good function 2 years later</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>13</td>
<td>At 4 months</td>
<td>—</td>
<td>Density of head improving 14 months later</td>
</tr>
</tbody>
</table>

TABLE II
DETAILS OF FOURTEEN PATIENTS (FIFTEEN HIPS) WITH NECROSIS OF CARTILAGE AFTER SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

<table>
<thead>
<tr>
<th>Case number</th>
<th>Sex</th>
<th>Age in years</th>
<th>Early findings</th>
<th>Late findings</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>M</td>
<td>16</td>
<td>Reduced</td>
<td>Very limited</td>
<td>Very limited movements, Flexion contracture</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>15</td>
<td>Reduced</td>
<td>Slightly limited</td>
<td>Practically none</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>16</td>
<td>Normal</td>
<td>Restricted by pain</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>18</td>
<td>Reduced</td>
<td>Very limited</td>
<td>Diminished</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>16</td>
<td>Normal</td>
<td>Restricted by pain</td>
<td>Progressive loss</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>13</td>
<td>Normal</td>
<td>Limited</td>
<td>Very diminished</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>11</td>
<td>Right: Normal</td>
<td>—</td>
<td>Very diminished</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left: Normal</td>
<td>—</td>
<td>—</td>
<td>Very diminished</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>14</td>
<td>Normal</td>
<td>Limited by spasm</td>
<td>Diminished</td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>13</td>
<td>Good</td>
<td>Limited by pain</td>
<td>Diminished at 2 years</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>20</td>
<td>Normal</td>
<td>Very limited</td>
<td>Arthritis at 2 years</td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>15</td>
<td>Slight loss</td>
<td>Some limitation</td>
<td>Poor</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>14</td>
<td>Normal</td>
<td>—</td>
<td>Diminished at 1 year</td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>15</td>
<td>Normal</td>
<td>Restricted</td>
<td>Good at 15 months</td>
</tr>
<tr>
<td>20</td>
<td>F</td>
<td>15</td>
<td>Greatly reduced</td>
<td>None</td>
<td>—</td>
</tr>
</tbody>
</table>
The anatomical vascular pattern in man has been studied by dye injection and by naked-eye dissections of anatomical specimens, and opinion has differed as to the relative importance of the various vascular sources, particularly at different ages. Trueta (1957) supported the view that the lateral epiphysial vessels and the foveal vessels in the ligamentum teres form the main blood supply to the femoral head during adolescence.

Some authors have observed that avascular necrosis can be caused by displacement of the epiphysis alone, without treatment. Moore (1945b) stated that “epiphysial necrosis may occur naturally with minimal displacement.” In the present series no patient showed evidence of sclerosis of the head when first examined radiologically; all the patients received some form of treatment before the sclerosis appeared and, although in this paper it has been assumed that treatment was responsible for the necrosis, the possibility that it was a direct sequel of displacement cannot be disproved.

All the six patients with avascular necrosis of the femoral head had considerable displacement when first seen, with a normal joint space and bone density, and with symptoms of less than four weeks’ duration which were mostly attributed to a recent fall. In four patients movements in the affected hip were restricted at first by pain and spasm.

The site of the sclerosis in the femoral head varied. In one patient treated by manipulation and nailing the whole head became dense. The upper half of the head became avascular in three patients, two of whom were treated by manipulation and nailing, and the other by open reduction and nailing. In two patients the necrosis occurred in a central area. One of these patients was treated by medial rotation plasters after traction (Fig. 3) and the other was treated by traction, during which the head was distracted from the acetabulum, and this probably occluded the vessels in the ligamentum teres and led to the central necrosis. The three patients who showed increased density of the upper half of the femoral epiphysis had probably suffered rupture of the lateral epiphysial vessels while retaining the vessels in the ligamentum teres. The patient in whom the whole head became dense either must have lost both sources of blood supply or had an underdeveloped vessel in the ligamentum teres: this occurs in some people and makes the epiphysis particularly vulnerable.

In all the hips the position of the epiphysis was improved and in two the displacement had been slightly overcorrected into the valgus position.

The findings in this small series agree with those of Jerre (1950), who found that a successful manipulative reduction was more likely to cause avascular necrosis than an unsuccessful manipulation, and that severe epiphysial displacements after injury showed a relatively high incidence of avascular necrosis.

The necrosis in three patients showed within six weeks; this is rather earlier than is usually reported. Two of the six patients recovered normal bone density within four months; both had started non-weight-bearing exercises immediately after the insertion of a Smith-Petersen nail. This contrasts with three other patients who took over twelve months to regain normal density and who had not been treated by immediate mobilisation. This gives some support to the view expressed by Watson-Jones (1943) that “revascularisation is promoted best by active non-weight-bearing exercise.”

The fate of the articular cartilage after necrosis of the bony epiphysis probably influences the outlook for joint function as much as the amount of bony deformity resulting from collapse of necrotic bony tissue. In the experimental work on rabbits by Harris and Hobson...
(1956), separation of the capital epiphysis caused necrosis of the bone; the articular cartilage showed cellular changes and the intercellular substance became fibrillated. The number of pathological reports on human specimens in the literature is small, and the two described by Moore (1945a) are the only ones showing in microscopic detail the bone and cartilage changes in an upper femoral epiphysis that had developed avascular necrosis from slipping. In the first, a boy aged twelve, the bone of the epiphysis underwent necrosis but most of the articular cartilage appeared alive on section, although a little thinner than normal. The second specimen

![Fig. 4](image1)

![Fig. 5](image2)

Figs. 4 to 7
Case 1—A boy of fifteen was found to have a slipped upper femoral epiphysis with a history of two weeks (Fig. 4). The femoral head became dense, but this improved with mobilisation after pinning. Sixteen years later the hip was satisfactory with a good range of movement (Figs. 5 to 7).

![Fig. 6](image3)

![Fig. 7](image4)

was obtained from a man aged twenty-one; there was non-union between the necrotic epiphysis and the metaphysis; most of the articular cartilage over the dead bone had become necrotic, but one area of cartilage survived. The changes found in both specimens were late findings. There was a considerable difference in age between the two patients, and the older one had no evidence of the development of secondary sexual changes. The fact that most of the articular cartilage survived in the specimen from the younger boy is significant, as Moore suggests, and recalls the changes found in Perthes' disease in which the articular cartilage
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survives well, and is indeed sometimes hypertrophied. Patients with Perthes' disease are admittedly younger, but it is probable that in adolescents the nutrition of the articular cartilage resembles that in younger children. That the cartilage obtains its nutrition from the synovial fluid rather than directly from the underlying blood vessels in the subchondral layer is supported by physiological and pathological studies (Bauer, Ropes and Waine 1940; Landells 1957). Landells believed that direct contact of blood vessels with the articular cartilage causes death of the cartilage cells. If it is true that the nourishment of the articular cartilage is derived from the synovial fluid, then the onset of bone necrosis would not be expected to cause complete death of the articular cartilage provided the synovial membrane and vessels in the joint capsule carried on their normal physiological role. This was shown to be the late result in four of the patients in this series (Figs. 4 to 12, Cases 1 and 2), each patient having a reasonable joint space, a good range of movement and a fairly well shaped head.

In this series it is shown that avascular necrosis can follow a successful manipulation of a recent severe epiphyseal slip, but that the prognosis is not necessarily bad because the

Figs. 8 to 12
Case 2—A boy of fourteen. Figure 8 shows the appearance of the hip on admission. Nine weeks after manipulation and nailing the head is dense (Fig. 9) but nine years later the hip is satisfactory (Fig. 10) with a good range of movement (Figs. 11 and 12).
articular cartilage can survive, especially if aided by early mobilisation. Osteoarthritis may develop later, but at least there can be several years of good painless function. This prognosis is very different from that of necrosis of articular cartilage, which is extremely poor.

**Necrosis of Articular Cartilage**

Since Waldenström first described necrosis of the articular cartilage in three hips in 1930, sixteen other cases have been recorded in the literature. In this series there was necrosis of

<table>
<thead>
<tr>
<th>Initial findings</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long history of symptoms</td>
<td>8</td>
</tr>
<tr>
<td>Short history of symptoms</td>
<td>6</td>
</tr>
<tr>
<td>Severe displacement</td>
<td>10</td>
</tr>
<tr>
<td>Slight displacement</td>
<td>5</td>
</tr>
<tr>
<td>Diminished joint space</td>
<td>5</td>
</tr>
<tr>
<td>Normal joint space</td>
<td>10</td>
</tr>
</tbody>
</table>

the articular cartilage in fifteen hips. Because of the frequency of the occurrence and poor prognosis, this complication should be more widely recognised.

This complication presents in many respects like an inflammatory lesion of the hip: indeed, for a time two of the hips in this series were suspected of being tuberculous until disproved by biopsy. However, in all patients in whom the temperature, white cell count, and blood sedimentation rates were recorded they were normal. Four of the fourteen patients had arthrodesis performed within one year of first being seen; in three of these an intra-articular operation revealed necrosed articular cartilage but with healthy bleeding cancellous bone beneath it. The finding of this normal bone beneath the necrotic cartilage tends to confirm that the articular cartilage necrosis was not caused by a diminished blood supply. Waldenström expressed this view in 1930 and he pointed out that the articular cartilage of the acetabulum is also necrosed although its blood supply is not affected by the slipping of the femoral epiphysis. Also, as Moore (1945b) observed in his patient with bilateral necrosis of the articular cartilage, the epiphysis takes part in the disuse atrophy and appears rarefied, indicating that the blood supply is intact. Moore considered the cause to be severe non-specific inflammatory reaction, whereas Wilson (1949) thought it due to a partial loss of blood supply. Waldenström (1930) believed the explanation lay in the damage done to the synovial and capsular attachments superiorly by manipulation with resultant loss of synovial secretion.

The early findings in the patients in this series are shown in Table III. It will be seen that in five hips the joint space was already diminished at the first examination, and one of these

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**Fig. 13**

Case 20—A girl of fifteen who presented with a nine months' history of limping had a fibrous ankylosis, because of necrosis of articular cartilage which occurred naturally and before treatment.
was already ankylosed (Case 20, Fig. 13). Three cases in the literature also showed evidence of loss of joint space on admission, and this suggests that sometimes the prognosis is poor regardless of the type of treatment.

Figure 14 shows the combination of treatments used. When traction—which varied from skin traction to a powerful skeletal pull—was prolonged for more than seven weeks it was also considered to be immobilisation in the classification of treatment. Thus immobilisation varied from rigid fixation in plaster or on a frame to skin traction in a Thomas’s splint, which—although it allows a limited amount of movement—cannot be regarded as mobilisation.

CONSIDERATION OF ADVERSE FACTORS IN TREATMENT

**Immobilisation**—It is of special interest that necrosis of the articular cartilage developed in two so-called “healthy” hips while the opposite hips were being treated for a slip. In both cases the “healthy” hip was immobilised only during the treatment of the opposite hip. In Case 19 the right hip first developed a slip and was treated by manipulation and immobilisation on a frame for nine months. It appeared to do well and the child had walked on it for a year when the left femoral epiphysis slipped. He was placed on an abduction frame for three months and during this time developed loss of joint space and permanent loss of movement in the right hip. The second, Case 13 (Figs. 15 to 17), was a girl who sustained a severe slip of the right femoral epiphysis which was treated by manipulation and immobilisation in a double spica for six months. Both hips showed marked loss of joint space and porosity of the head, and movements remained greatly restricted despite mobilisation for twelve months.

Moore (1945h) described a similar patient who had bilateral epiphysial displacement. Both hips were treated by traction and plaster, and both developed fibrous ankylosis. Jerre (1950) also reported an analogous case in which the unaffected hip became stiff, with loss of joint space. Hall (1957) reported a patient with bilateral slipping who showed loss of joint space in both hips after being on an abduction frame for ten weeks.

Twelve of the fifteen hips in this series were treated by immobilisation for over eight weeks, and in Jerre’s (1950) series six of the nine patients showing articular cartilage necrosis had been treated by immobilisation and one by traction for eight weeks. Also the three original patients described by Waldenström (1930) were treated by immobilisation in plaster after manipulation. If the articular cartilage derives its nutrition from synovial fluid it may be assumed that rigid immobilisation, by producing atrophy of the synovial membrane and diminished synovial secretion, causes impaired nourishment of the articular cartilage. Müller (1929) suggested that nutrition of the articular cartilage was affected by the alternate physical processes of suction and pressure, which drew and expelled fluid from the adjacent network.

The recent biochemical studies on articular cartilage by Eichelberger, Roma and Moulder (1959) are important. Experimental cartilage atrophy, produced by denervation and disuse,
Case 13—A girl of eleven with bilateral necrosis of articular cartilage. The radiograph on admission (Fig. 15) shows a severe slip on the right, but only slight on the left. The right hip was manipulated, and a double plaster spica applied, and removed after nine months (Fig. 16). After a further ten months of mobilisation the joint space on both sides is very poor (Fig. 17).
was studied. Simple immobilisation of a joint produced atrophy of the articular cartilage with changes in its physical state, particularly in its extracellular component. Disuse atrophy of articular cartilage was reversible, and the authors believed that this was explained by the viability of the chondrocytes. This in turn was controlled by several factors, one of which was chondroitin-sulphate production. But this is not yet fully understood. If it is linked with synovial fluid production, then there may be a point when the synovial tissue becomes so atrophied by prolonged immobilisation that the changes in the articular cartilage are no longer reversible.

Howorth (1949) reported the frequent finding of non-specific inflammatory changes in the synovial membrane after slipping of the epiphysis. It is possible that these inflammatory changes contribute to the impairment of the nutrition of the articular cartilage.

**Traction**—Traction in some form was used in ten of the fifteen hips which developed necrosis of the articular cartilage, and the head was distracted from the acetabulum in three hips which must have damaged the vessels in the ligamentum teres and in the synovial and capsular attachments. One hip was distracted with

only fifteen pounds pull. Hall (1957) showed that traction alone produced very little correction in a slip, and the importance of its harmful influence has not been sufficiently recognised.

**Early osteotomy**—This was used in one patient (Case 9, Figs. 20 and 21) in this series. This
boy of sixteen had a severe slip partially corrected by traction and the remaining varus deformity corrected by a subtrochanteric osteotomy six weeks later. The hip was immobilised in a plaster spica for fifteen weeks. Nine months after this there was considerable loss of joint space with a fibrous ankylosis. With early osteotomy there is not only the danger that the operation may damage further an already precarious blood supply but also that it necessitates prolonged immobilisation. Osteotomy should, therefore, be deferred until a good range of movement has been obtained.

DISCUSSION

Early necrosis of the articular cartilage after slipping of the upper femoral epiphysis gives a very poor prognosis. Four of the hips in this series were arthrodesed within twelve months of first presenting. Probably the most important cause of this poor outlook is the loss of the smooth articular cartilage, but there may also be fibrosis in the capsule. The latter was demonstrated by Howorth (1949) in what he called the "residual stage" after slipping of the epiphysis. While it is accepted that good results can be obtained by a variety of methods of treatment, it must also be remembered that in any large series there will be some failures, and that there is no way in which these poor results can be predicted. It is difficult to understand why two hips with similar slips and similar treatment should give different results: this and many other aspects of the problem remain unsolved. Perhaps the explanation lies in the individual response to injury at an age when so many body changes are occurring as growing tissues alter to the mature adult state. Anatomical variations may also account for some of the differences.

SUMMARY AND CONCLUSIONS

1. Avascular necrosis of the bony epiphysis or necrosis of the articular cartilage of the hip joint—without bony necrosis—can occur after a slipped upper femoral epiphysis.
2. In avascular necrosis of the bony epiphysis the prognosis depends upon the degree of revascularisation that occurs and upon survival of the articular cartilage. The articular cartilage can survive and a good functioning hip result especially if aided by mobilisation without weight bearing.
3. The prognosis after necrosis of the articular cartilage is poor. This complication occurs more often when conservative treatment is used.
4. A certain number of hips will show poor results no matter what treatment is used.
5. Nutrition of the articular cartilage is probably by the synovial fluid.
6. Strong traction may damage the soft-tissue structure of the hip joint.
7. It is not advisable to perform an osteotomy soon after a slip of the epiphysis. It is better to wait until good function is assured in the joint.

I wish to thank the surgeons at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, and the Prince of Wales Orthopaedic Hospital, Cardiff, for permission to study their patients. In particular I am grateful to Mr Dillwyn Evans, Dr E. Mervyn Evans and Mr Robert Roaf for their helpful criticism and encouragement. I would also like to thank Mr Beverley Southern and Mr G. Haddock for the photographs.

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