We performed superselective angiography in 28 hips in 25 patients with Perthes’ disease in order to study the blood supply of the lateral epiphyseal arteries (LEAs). Interruption of the LEAs at their origin was observed in 19 hips (68%). Revascularisation in the form of numerous small arteries was seen in ten out of 11 hips in the initial stage of Perthes’ disease, in seven of eight in the fragmentation stage and in five of nine in the healing stage. Penetration of mature arteries into the depths of the epiphysis was seen in four of nine hips in the healing stage. Vascular penetration was absent in the weight-bearing portion of the femoral head below the acetabular roof. Interruption of the posterior column artery was seen where it passed through the capsule in seven hips when they lay either in internal rotation or in abduction with internal rotation.

We suggest that in Perthes’ disease the blood supply of the LEAs is impaired at their origin and that revascularisation occurs from this site by ingrowth of small vessels into the femoral epiphysis. This process may be the result of recurrent ischaemic episodes. The normal vascular anatomy of the femoral head in children has been studied using microangiography. The LEAs supply an extensive area of the capital epiphysis and are the most important arteries. We have performed superselective angiography of the medial femoral circumflex artery in order to demonstrate the blood supply in Perthes’ disease. Our aim was to examine the vascular changes in the LEAs and the possible pathogenesis of the disease as a result of circulatory disturbance at this site.

Patients and Methods

We performed superselective angiography of the medial femoral circumflex artery in 25 patients (28 hips) with Perthes’ disease. There were 22 boys and three girls with a mean age at the time of angiography of 9.2 years (4 to 15). Four patients had bilateral disease. Conventional antero-posterior and lateral radiographs were taken to determine the stage of the disease and the area of involvement of the hip. The stage at the time of angiography was classified according to Waldenstrom; 11,16 11 were in the initial stage, 8 in the fragmentation stage and nine in the healing stage. Of the 11 in the initial stage, eight presented with flattening of the femoral epiphysis. The remaining three were almost spherical. The exact age of onset of the disease was difficult to determine since it had already progressed to some extent when the patients first presented.

We used the Catterall classification to assess radiologically the extent of the involved area of the capital femoral epiphysis. The 28 hips were divided into two groups: group 1, those with a small lesion (Catterall group 2, 3 hips) and group 2, those with an extensive lesion (Catterall group 3 or 4, 25 hips) There were no Catterall group-1 hips in this study. If the lesion was not seen clearly at the time of angiography, the classification was made on follow-up radiographs. Hips were classified in the healing stage by the observation of viable bone.
The normal angiography of the LEAs was obtained from four normal hips in four boys who had tumours of the knee or leg. These children did not have any symptoms or radiological findings suggestive of Perthes’ disease. Three were aged 10 years and one 12 years. Superselective angiography of these hips showed that the posterior column artery arises as a branch of the medial femoral circumflex artery. The main trunk of the LEA was shown to arise from the posterior column artery in its distal portion and extend into the centre of the epiphysis. Small arteries branching from this artery were not shown clearly (Fig. 1).

**Method of superselective angiography.** In all 28 hips angiography was performed on the anteroposterior view in neutral rotation. In addition, it was carried out on six hips in the lateral view, seven in either internal rotation or abduction with internal rotation and on five in the abducted position. The subtraction method was used to give good visualisation.

Under general or local anaesthesia, the contralateral femoral artery was cannulated in the older children and the ipsilateral femoral artery in the younger because the bifurcation of the common iliac artery was too acute to allow the cannula to negotiate the angulation. The tip of the cannula was inserted into the origin of the medial femoral circumflex artery and 6ml of contrast medium injected manually every four seconds to fill the posterior column artery and the LEA. If this was not achieved, the catheter was inserted into the internal iliac artery and further contrast injected to see whether the posterior column artery could be filled from this site.

A cylindrical biopsy was also taken from 11 affected epiphyses immediately after angiography or at the time of operation two weeks later. Biopsy specimens were obtained manually using a 4.3 mm needle from the lateral or medial portion of the capital epiphysis including articular cartilage in an attempt to accelerate revascularisation. The specimens were fixed in formal saline. After decalcification they were sectioned and stained with haematoxylin and eosin. The histological findings were compared with the angiographic results. All radiographs, angiographs and biopsy specimens were obtained with informed consent of the patients and parents.

**Results**

**Pattern of origin of the posterior column artery and the LEAs.** In 22 of the 28 hips, the posterior column artery arose from the medial femoral circumflex artery and in the remaining six from a branch of the internal iliac artery. The
LEAs arose from the distal portion of the posterior column artery. There was no evidence of vascular impairment in the medial femoral circumflex artery, the branch of the internal iliac artery or the posterior column artery.

**Angiographic classification.** The patterns of blood supply of the LEAs revealed by angiography of both normal and Perthes’ hips were classified as follows (Fig. 2).

*Class I.* A normal pattern.
*Class II.* No filling distal to the origin of the LEA.
*Class III.* No filling of the true LEA from its origin. There was, however, vascularisation consisting of numerous newly-formed small arteries on the surface of the articular cartilage and passing through the cartilage. These were distributed to a small lateral portion of the bony epiphysis.
*Class IV.* Several arteries were seen associated with an increase in the size of the extraosseous area. Small arteries were present in the lateral bony epiphysis.
*Class V.* There was penetration of mature arteries into the depth of the epiphysis in an irregular manner.

A normal appearance of the LEA was seen only in class I. This classification probably reflects the process of revascularisation.

**Vascular changes in Perthes’ disease.** Four normal control hips had class-1 angiographic findings. None of the 28 hips with Perthes’ disease showed a class-1 pattern (Figs 3 to 6). Interruption of the true LEA at its origin was observed in 19 hips (68%) (Fig. 3b). This was noted on eight of the 11 hips in the initial stage, in six of the eight hips in the fragmentation stage and in five of the nine in the healing stage.

One of the 11 hips in the initial stage showed interruption of the LEA at its origin (class II). The remaining ten had numerous small arterial penetrations with no true LEA; four were in class III (Fig. 3b) and six were in class IV (Fig. 4b). None was in class I or class V.

Of the eight hips in the fragmentation stage one was classified as class II. The remaining seven showed numerous small abnormal arteries; two were in class III, and five in class IV (Fig. 6b). None was in class I or class V.
Figure 4a – Anteroposterior radiograph of the left hip of a 12-year-old boy showing increased density over an extensive area of the epiphysis. Figure 4b – Angiography shows a hypervascularised area in the lateral epiphysis (arrow) and an abnormal LEA which is visible only in the extraosseous portion. The angiographic classification is class IV.

Figure 5a – Anteroposterior radiograph of the right hip of a four-year-old boy. The central epiphysis has reossified with slight loss of height. Figure 5b – The lateral view shows that the anterior half of the head is involved. Figure 5c – Numerous small arteries are distributed over the entire epiphysis indicating a class-IV appearance. The normal appearance of the LEA is absent. The dotted line shows the contour of the femoral head.
Of the nine hips in the healing stage numerous abnormal arteries penetrating the viable bone were seen in five hips; one was classified as class III and four as class IV (Fig. 5c). Four hips were class V with no small abnormal arteries. Class-I and class-II changes were not seen at this stage. Increased vascularisation arising from the LEA was still present in six hips. A summary of the angiographic changes seen in the normal hips and in those with Perthes’ disease is given in Table I.

The histological appearance of a biopsy specimen is shown in Figure 3c. There was penetration of immature vascular fibrous tissue into woven bone in the vascularised area related to the small arterial penetration on angiography.

**Correlation between the vascular changes and the extent of involvement by Perthes’ disease.** There were only three hips in group I and all were in the healing stage. In two the angiographic classification was class V and in one class IV (Fig. 5c). In group 2 there were 25 hips with extensive involvement; two were in class II, seven in class III, 14 in class IV and two class V. Eleven of the group-2 hips were in the initial stage; of these one hip was classified as class II, four as class III (Fig. 3b) and six as class IV (Fig. 4b). Of the eight group-2 hips in the fragmentation stage, one was class II, two were class III and the remaining five were class IV (Fig. 6b). Of the 12 group-2 hips in the healing stage, one was class III, three were class IV and two were class V.

**Blood supply to the weight-bearing area below the acetabular roof.** Vascularisation in the lateral non-weight-bearing area of the affected femoral epiphysis was seen in 26 hips. In 18 the weight-bearing portion below the acetabular roof was avascular (Figs 3b and 4b); all of these were group 2. In the remaining eight, vascularisation was seen in the weight-bearing area below the acetabular roof (Fig. 5c); three of these hips were in the fragmentation stage, and five in the healing stage. The angiographic class of these eight hips was III in one, IV in five and V in two. The extent of involvement was group 1 in two and group 2 in six.

**Vascular changes in different positions of the hip.** Vascular changes were observed in either internal rotation or internal rotation with abduction in seven hips in either class III or class IV and compared with the findings in the neutral position. In internal rotation and internal rotation with abduction, the posterior column artery was markedly stretched and interrupted in its distal portion where it passed through the capsule. The LEAs were not filled in either position although they filled well when the hip lay in neutral position with no evidence of impairment of the blood supply. In abduction the vascularity was decreased, but there was no interruption of the blood supply (Fig. 6).

**Discussion**

The aetiology of Perthes’ disease is probably associated with impairment of the blood supply to the femoral capital epiphysis resulting in ischaemia. Although there has been much debate the exact cause of the interruption of the blood supply is still controversial. There are a number of unsolved questions such as whether this interruption occurs inside or outside the femoral head, whether impairment occurs in the arterial or venous system, and whether more than one episode gives rise to the ischaemia.

Angiographic studies of Perthes’ disease have shown interruption of the blood supply of the LEAs at the origin of their extraosseous portion. We have observed vascular impairment at the same site. This probably leads to an initial episode causing ischaemia in the femoral epiphysis, although the cause of this episode is unknown. Our study has also shown that the posterior column artery originated from a branch of the internal iliac artery in six of the 28 hips. This finding appears to be an abnormal congenital vascular pattern and not related to the pathogenesis of Perthes’ disease. There was no suggestion of vascular impairment at this site. Similar findings have been observed in normal hips of adults.

In experimental animals a Perthes'-like change in the femoral epiphysis has been produced by transitory interruption of the venous flow after intracapsular tamponade, but this study did not show fragmentation of the femoral epiphysis which is a characteristic finding of the disease. We have shown absence of true LEAs at various stages of the disease. This suggests that the interruption of the blood supply of the LEAs occurred early. This ischaemic episode may involve most or all of the epiphysis.

In immature dogs Henard and Calandruccio observed that certain positions of the hip can produce obstruction to the arterial supply to the head of the femur. In our study interruption of the blood supply to the LEAs was demonstrated where the arteries passed through the capsule when the hip was either internally rotated or abducted. This suggests that there may be a mechanical element contributing to the vascular impairment in this area.

**Table I.** Angiographic classification of the LEAs in the four normal hips and in the 28 with Perthes' disease

<table>
<thead>
<tr>
<th>Class</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
<th>Class V</th>
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<td>0</td>
<td>0</td>
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</tr>
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<tr>
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<td>2</td>
<td>5</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Healing stage</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>4</td>
<td>9</td>
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Our study also suggests that where there is penetration of small vessels indicative of revascularisation, the normal vascular pattern can be restored in the progressive stage of the disease. If there is a single ischaemic episode, revascularisation may be able to penetrate the entire epiphysis and new bone be formed throughout. Revascularisation appears to develop from newly-formed small vessels which probably originate from or around the affected LEAs not only in the hips with small involvement but also in those with an extensive lesion. No nutrient arteries with a normal appearance were detected in the femoral epiphysis. Perthes’ disease appears to develop despite spontaneous revascularisation penetrating the avascular bony epiphysis. Recurrent obstruction to revascularisation probably occurs during the repair process. Necrosis of woven repair bone and reparatory fibrous tissue, suggestive of recurrent necrosis, are common histological findings in Perthes’ disease in man and are also found in experimental studies. Incomplete infarction of the epiphysis has also been reported in histological studies in man.

Salter and Thompson speculated that the radiological phenomenon of the subchondral crescentic radiolucent line was a pathological fracture. They observed that the extent of this ‘subchondral fracture’ correlated precisely with the subsequent degree of maximum resorption. Microangiography of femoral heads with osteonecrosis in adults which...
had been resected during total hip replacement clearly showed blockage of revascularisation as a result of subchondral fracture and collapse. In our study, hips with an extensive lesion showed revascularisation of the lateral portion in the non-weight-bearing area. The weight-bearing area remained avascular implying impairment of revascularisation. We believe that in Perthes’ disease impairment of the blood supply via the LEAs takes place at the origin of the artery where it passes through the joint capsule, causing ischaemia of the femoral epiphysis. Revascularisation occurs through newly-formed small arteries arising in the area surrounding the origin of the LEAs. These vessels penetrate the involved bony epiphysis. Revascularisation is blocked by the subchondral fracture and collapse in the weight-bearing portion of the femoral epiphysis leading to the characteristic deformation seen in severe forms of Perthes’ disease.

The authors choose not to respond to the request for a conflict of interest statement.

References