A HISTOLOGICAL STUDY OF AVASCULAR NECROSIS OF THE FEMORAL HEAD AFTER TRANSCERVICAL FRACTURE

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The association of transcervical fractures of the femur with avascular necrosis of the capital fragment was recognised many years ago. Nevertheless several basic questions, such as the incidence of avascular necrosis, the extent and pattern of bone death and revascularisation and also the role played by avascular necrosis in non-union of these fractures, remain unanswered. In the hope that a histological study might throw some light on these and other problems still under discussion, all femoral heads removed at operation or necropsy from patients with displaced transcervical fractures have been examined since January 1958.

MATERIAL AND METHODS

The material falls into the following groups: 1) fifty femoral heads taken at necropsy from patients with transcervical fractures; 2) seventy-eight femoral heads removed at primary prosthetic arthroplasty; 3) sixty heads excised as a secondary procedure when the fracture became redisplaced or failed to unite. Femoral heads which were infected, had post-irradiation osteitis or inadequate histological preparation were not included in this study. Twelve heads in which the fracture united but secondary arthroplasty was done for late segmental collapse of the head are discussed separately in this issue of the Journal (page 777).

For comparison the upper end of the femur was examined in necropsies selected at random in forty-six female and four male subjects ranging from sixty-four to eighty-six years of age with an average of seventy-four.

Preparation of specimens—Each femoral head was fixed in 10 per cent formal saline for at least two weeks. Coronal slices about four millimetres thick were cut on a band saw and radiographs were taken on Kodak K.P.5 or Kodalith film using a Victor Raymax 50 machine at twenty-five kilovolts and five ampères. The bone slabs were subsequently decalcified in formic citrate buffer (Meyer 1956), washed overnight and then processed by a double embedding method (Russell 1956). After embedding in paraffin whole sections of the femoral head, or head, neck and trochanter, were cut on a Jung microtome (model K) and stained with haemalum and eosin.

In some cases the ligamentum teres was also available; it was cut into labelled serial blocks and, after processing, was stained by haemalum and eosin. Specimens of special interest were also stained to demonstrate elastica (Weigert and Orcein methods), mucopolysaccharides (PAS), connective tissue (Masson's trichrome), reticulin (Gordon and Sweet), amyloid (congo red) and by some of Lendrum, Fraser, Slidders and Henderson's stains (1962) for demonstrating fibrin (M.S.B., Masson 44/41, Yellowsolve 1).

BONE CHANGES IN "NORMAL" ELDERLY PATIENTS

It is widely recognised that from early adult life onwards some "physiological" bone necrosis occurs, especially in subchondral bone and in the interstitial lamellae of the cortex, and that this increases with advancing age and with deterioration of the vascular supply (Jaffe and Pomeranz 1934, Rutishauser and Majno 1951, Sherman and Selakovitch 1957). It was therefore essential to examine control material to find out the pattern and degree of osteocyte loss in "normal" femoral heads of patients of similar ages to those with transcervical fractures. Fifty upper femora were taken at random from necropsies in those over sixty-four years of age; the only cause for exclusion from the series was radiotherapy to the pelvic organs or infiltration by tumour. Bone death is recognised histologically by the presence of empty bone lacunae; before the osteocytes actually disappear the whole cell may lose its basophilia and be seen as a faint pink shadow in the lacuna. In these control cases the degree
Figure 1—The bone trabecula from a 74-year-old patient with a normal hip shows patchy osteocyte loss. There are nuclei in the fat cells of the marrow.

Figure 2—There is complete loss of osteocytes in this necrotic bone trabecula. The marrow is also necrotic and there is loss of nuclear staining. (Haemalum and eosin, ×150.)

Figure 3—Living bone and marrow is in contrast to Figure 4 in which there is evidence of old necrosis in the central part of the trabecula devoid of osteocytes. New living bone has been laid down on the surface. Many dilated capillaries are seen in the revascularised marrow. This femoral head was removed three years after fracture. (Haemalum and eosin, ×85.)
of osteocyte loss was always greater in the Haversian bone of the inferior cortex of the neck and in the subchondral bone plate than in spongy bone. Absence of osteocytes in trabecular bone was essentially patchy and it was unusual to find a whole trabecula or even the area bounded by a cement line to be completely devoid of cells (Figs. 1 and 2). In two femoral heads some subchondral trabeculae showed a basophilic matrix and were almost devoid of cells apart from small protrusions of live bone on the surface.

None of these control femoral heads showed any evidence of those marrow changes which were found to precede or accompany loss of osteocytes in the fracture series. In practice it was not difficult to distinguish between bone necrosis produced by sudden ischaemia after fracture and that of "physiological" necrosis.

Unexpectedly, in almost two-thirds of the cases, small excrescences of fibre bone were present at the tips or on the surfaces of trabeculae (Fig. 6). Usually few, occasionally they were abundant. More rarely, fibre bone strands were seen in the marrow spaces (Fig. 5) and were distributed unevenly in the head and sometimes also in the neck of the femur. In rather less than a third of the cases and particularly in one there was bizarre bone formation. Here, bone forming on the surface of trabeculae appeared to follow the borders of adipose cells so that these became partly—and sometimes completely—surrounded by bone, giving a curiously spiky appearance (Fig. 7). Both of these patterns in the normal femora are important because, when seen after injury, they might wrongly be regarded as a reaction to it.

Examination of articular cartilage on femoral heads with no osteoarthritis showed that, in normal elderly patients there was sometimes a slight patchy loss of chondrocytes from the cartilage lacunae, especially in the deeper layer.

STUDY OF FEMORAL HEADS REMOVED WITHIN FIFTEEN DAYS OF TRANSCERVICAL FRACTURE

Forty-nine femoral heads were removed from patients undergoing primary arthroplasty and ten were taken from patients treated by internal fixation who died within fifteen days of the fracture. Table 1 shows the details.

<table>
<thead>
<tr>
<th>Number of days after fracture</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of specimens</td>
<td>.</td>
<td>.</td>
<td>6</td>
<td>8</td>
<td>10</td>
<td>8</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td></td>
<td>59</td>
</tr>
</tbody>
</table>

HISTOLOGICAL CHANGES

Fibrin and haemorrhage were present at the fracture site in all cases and, in some femoral heads removed within twenty-four hours of injury, there was very slight proliferation of fibroblasts in the area. This reaction often increased in subsequent days and by the fifth was sometimes extensive and accompanied by an increase in capillaries. Foamy macrophages appeared in damaged marrow from about the fourth day onwards and later there was formation of oil cysts ringed by macrophages or sometimes by giant cells (Fig. 8). It was rare to see evidence of new bone formation at the fracture site or even of plumping of osteoblasts on the surface of trabeculae in less than five days, and in only one case at four days was one minute area of new bone apparent. Bone formation (Fig. 9) on the surface of trabeculae at the fracture site and in the marrow spaces was quite noticeable by the thirteenth day in some specimens.

Changes in marrow spaces—These begin to be recognisable from two days onwards. The first evidence of ischaemia was a peculiar agglomeration of the marrow, most readily recognisable in areas of haemopoiesis where large spaces appeared, surrounded by blood-forming cells.
Irregular strands of fibre bone are seen in the marrow and on the surface of trabeculae in the femoral heads of normal elderly patients. (Haemalum and eosin, ×100.)

A normal femoral head with bone forming on the surface of a trabecula and enclosing within it fat cells. (Haemalum and eosin, ×195.)
These cells, from four days onwards, died, lost their nuclei and became eosinophilic. Often, in necrotic femoral heads removed many weeks after the fracture, these changes were seen in a faint and ghostlike manner (Figs. 10 to 12). A similar but less striking alteration sometimes occurred in fatty marrow and was accompanied by loss of lipocyte nuclei (Figs. 13 and 14). From three or four days onwards, necrosis of small blood vessels in marrow (Fig. 14), capsular tags or the foveal end of the ligamentum teres (Fig. 15) could be recognised by absence of nuclei, homogeneity and increased eosinophilia of the walls.

Changes in bone—Loss of osteocyte nuclei from the fragmented bone trabeculae crushed at the fracture site was present to some extent from the fourth and fifth days, but it was notable that it was rarely complete until the fourteenth day or even later. Osteocyte death in uncrushed trabeculae surrounded by apparently necrotic marrow was slower and could not be discerned until about the thirteenth or fourteenth day. It was complete, or nearly complete, at approximately three or four weeks. There was no evidence of pre-existing trabecular death in the femoral heads removed soon after fracture.

In assessing the state of the femoral head, fibroblast proliferation, plumping of osteoblasts on bone surfaces and new bone formation were taken to indicate a blood supply in the area or immediately adjacent to it. Agglomeration with necrosis of haemopoietic marrow, total loss of lipocytes of fatty marrow, the presence of necrotic marrow blood vessels, complete absence of cellular reaction at the fracture site and osteocyte loss in the uncrushed trabeculae were thought to indicate ischaemia. In an occasional case at the fourth or fifth day in which all the above changes except osteocyte loss were present throughout the head, it was possible to deduce tentatively that necrosis had occurred. In general, however, and especially when these changes were not uniform, no conclusion could be reached until the tenth day or later.
At this time marrow necrosis in affected areas appeared to be complete, the boundary between dead and living marrow was more readily defined and osteocyte loss was becoming apparent: but the changes were even more pronounced by the sixteenth day. In practice, provided there was ample material, there was no real problem in deciding the extent of necrosis in femoral heads removed more than sixteen days after the fracture. Usually three or four blocks of the femoral head were available for examination. It seems foolhardy, however, to give a firm opinion on smaller amounts of material, such as bone cores, until osteocyte loss can be expected to be complete, often three weeks or more from the time of fracture.

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**Fig. 10**

Figure 10—Normal haemopoietic marrow. Figure 11—Necrotic haemopoietic marrow five days after fracture showing agglomeration and loss of nuclei. Figure 12—Complete loss of nuclei with the shadows of agglomerated marrow spaces in a dead head seventeen weeks after injury. (Haemalum and eosin, × 100.)
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DISCUSSION

Various interpretations have been placed on the slow disappearance of osteocytes from bone lacunae after ischaemia. Some believe that until the osteocytes disappear the bone is alive, and it has been suggested that if the blood supply can be restored within fifteen days bone death will not occur (Patrick 1960). Woodhouse (1962a and b), however, produced bone necrosis in the femoral heads of dogs after temporary occlusion of the blood supply for only twelve hours. Sevitt (1964) suggested that the late persistence of osteocytes, for example at eighteen days, might be the result of necrosis some days after injury. It is not, however,
necessary to accept this explanation, for osteocyte loss was not complete until three weeks after the interruption of the blood supply to the femoral head of dogs (Bonfiglio 1954, Brown and Catto 1964). The consensus seems to be that, in spite of the slow osteocyte loss, the bone is dead shortly after the vascular injury (Sherman 1947, Sherman and Phemister 1947, Phemister 1948, Bonfiglio 1954, Campbell 1961), whether this is at the time of fracture (Crawford 1960) or at the time of manipulation and internal fixation, and whether it is caused by actual tearing of vessels or, as has been more recently suggested (Smith 1959, Woodhouse 1962a), by their occlusion from torsion. If it is generally accepted that bone death quickly follows the vascular injury, then a more accurate application of descriptive terms would greatly simplify the interpretation of the voluminous literature. In spite of admonitions by Phemister (1948, 1949a and b), Sherman (1947), Hodges (1954) and others, there is a general tendency to write about "late" avascular necrosis (Whitman 1945; Christophe, Howard, Potter and Driscoll 1953; Cave 1960), in fact it is the clinical recognition of this complication that is late. In particular, the term "late segmental necrosis" indicates a false conception of the processes involved and "late segmental collapse" is more suitable.

Similarly there is a tendency to reserve the term avascular necrosis for its clinical manifestations and to imply that it is some way different from dead bone: "If we are correct that in the vast majority of these fractures with definite displacement of the femoral heads are dead and have to be revascularised, it is not surprising that there is a continuing high incidence of avascular necrosis" (Cleveland and Fielding 1954). The term "viable head" is sometimes used (Compere and Wallace 1942, Brindley 1963) without clearly indicating whether this is a femoral head which has never been dead or one which has been revascularised—an important distinction.

**STUDY OF FEMORAL HEADS REMOVED MORE THAN SIXTEEN DAYS AFTER FRACTURE**

**VIVABLE FEMORAL HEADS**

Some necrosis is known to occur in any bone immediately adjacent to a fracture (Phemister 1930, Hatcher 1952, Ham and Leeson 1961), and an arbitrary ruling had to be made to separate femoral heads in which the amount of bone death at the fracture site might be considered within usual limits, from those in which it was excessive. McLean and Urist (1954) said that at least half a centimetre of shaft is damaged on either side of a closed, non-comminuted fracture. Femoral heads which showed death of bone confined to half to one centimetre from the fracture line have been considered to be alive. The narrow margin of dead bone which commonly surrounded the nail track was also regarded as normal and ignored. There were in all eighteen femoral heads judged to be alive by these criteria (Table II). Eight showed a slight increase in the depth of dead bone at the upper edge of the fracture site and two a similar increase at the lower edge where the inferior cortex of the neck presented a small spur.

It is not intended to imply that all the blood vessels to the live heads were intact, but simply that sufficient viable vessels remained to prevent necrosis except at the fracture site. It was found both from studying the operation notes and from examining the femoral heads histologically that any remaining retinacular vessels were almost invariably attached to the lower part of the head and were, presumably, the inferior metaphyseal group. In only one femoral head was there histological evidence of live vessels in the still attached superior part of the retinaculum. In one specimen the vessels of the ligamentum teres alone were sufficient to nourish the whole femoral head in which the surgeon had specifically noted this to be the only remaining soft-tissue attachment. Similar observations were made by Schmorl (1924), Santos (1930), Phemister (1948) and Sevitt (1964).

In the live femoral heads removed from sixteen days to ten months after injury viable callus was invariably present at the fracture site. In the absence of good fixation the presence of a live head was not, however, a guarantee of bony union (Charnley, Blockey and Purser
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1957). In one unfixed fracture there was fibrous union eighteen weeks after the injury and in four other patients in whom immobilisation was inadequate dense collagen and fibrocartilage were conspicuous at the fracture site.

NECROSIS OF THE WHOLE FEMORAL HEAD

Thirty-six femoral heads were completely dead. In fifteen of these no revascularisation had taken place and at the time of removal, which ranged from sixteen days to forty-two weeks

**TABLE II**

<table>
<thead>
<tr>
<th>Details of all femoral heads removed</th>
<th>Remained under sixteen days</th>
<th>Remained over sixteen days</th>
<th>Total</th>
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<tbody>
<tr>
<td>Failed nails</td>
<td>0</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Primary replacement</td>
<td>49</td>
<td>29</td>
<td>78</td>
</tr>
<tr>
<td>Necrospy</td>
<td>10</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
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<td>109</td>
<td>168</td>
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<table>
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<tr>
<th>Details of femoral heads removed after sixteen days</th>
<th>Alive</th>
<th>Foveal Triangle spared</th>
<th>Complete necrosis</th>
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<tr>
<td></td>
<td>Lower head spared</td>
<td>Foveal triangle spared</td>
<td>Revascularised</td>
</tr>
<tr>
<td>Failed nails</td>
<td>2</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Primary replacement</td>
<td>10</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Necropsy</td>
<td>6</td>
<td>1</td>
<td>3</td>
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<tr>
<td>Total</td>
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<td>6</td>
<td>18</td>
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</table>

<table>
<thead>
<tr>
<th>Sex and ages of subjects with femoral heads removed after sixteen days</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
<th>Average age (years)</th>
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<tr>
<td>Live heads</td>
<td>4</td>
<td>14</td>
<td>18</td>
<td>78</td>
</tr>
<tr>
<td>Completely dead</td>
<td>6</td>
<td>30</td>
<td>36</td>
<td>72</td>
</tr>
<tr>
<td>Partly dead</td>
<td>7</td>
<td>48</td>
<td>55</td>
<td>74</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>92</td>
<td>109</td>
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</tr>
</tbody>
</table>

after fracture, with an average of eleven weeks, the head remained totally necrotic without any vestige of cellular reaction (Table II). In the remaining twenty-one heads, which were removed from three weeks to three years after fracture, with an average time of twenty-nine weeks, some revascularisation had occurred, but in thirteen this was very slight and consisted only of a tiny zone in the foveal region or at the inferior margin of the fracture line where a shred of soft tissue was attached. In only eight of the thirty-six femoral heads was there notable revascularisation, which varied from about 15 per cent to almost complete. In one case there was early fibrous union between a dead and unrevascularised head and a live neck (Case 5).
In fifty-five femoral heads necrosis was partial. The most common pattern (forty-nine cases) was of a wedge of living bone, with its base in the subchondral region of the fovea, varying in size from a few trabeculae to more than half the head. The size of this wedge was roughly classified as large (more than 33 per cent) (Fig. 16), medium (10–33 per cent) and small (less than 10 per cent) (Fig. 17); the numbers in each group are shown in Table II. It was clear that these areas were nourished from the ligamentum teres. In six cases the lower part of the head was spared, the upper part being dead. In most of these the living bone included that around the fovea but in one it barely impinged on this zone. In five, retinacular tissue containing live blood vessels was recognised histologically and it is likely that in this group some blood supply came from the inferior metaphysial arteries and probably, though not invariably, also from the ligamentum teres. In all cases in which there was partial survival of

![Figure 16](image1.png)  ![Figure 17](image2.png)

**FIG. 16**—A large vascular wedge of living bone with its base on the foveal area is shown. The remainder of the bone is necrotic. **FIG. 17**—A small vascular foveal wedge remains in the femoral head.

the femoral head some revascularisation of dead bone had occurred. It was difficult to reach any conclusion about the rate of revascularisation because there was such a wide variation in the amount of initially living bone marrow. As might be expected, in general, those femoral heads with a large, living foveal wedge became revascularised more rapidly, the process being virtually complete sometimes as early as four to eight weeks after the fracture, whereas those with a small, living foveal wedge sometimes showed only small areas of revascularisation after forty-two weeks.

There was no histological evidence in either the complete or partly necrotic femoral heads that necrosis had occurred in more than one episode apart from slight local damage at the fracture site.

**HISTOLOGICAL FEATURES OF REVASCULARISATION**

Revascularisation of the marrow was recognised by proliferation of fibroblasts and leashes of capillaries with groups of foamy macrophages (Figs. 18 and 19). This was usually followed
Figure 20—Both osteoclasts and osteoblasts are seen on the surface of dead bone. The marrow has been revascularised. (Haemalum and eosin, $\times 125$.) Figure 21—No cellular reaction is seen in relation to dead bone here. The marrow is mostly fibrous and only small capillaries are present. (Haemalum and eosin, $\times 60$.)
by osteoclasts of dead bone and laying down of new bone on the surface of dead trabeculae (Fig. 20), though sometimes all cellular reaction appeared to be inhibited by the formation of poorly vascularised fibrous tissue in the marrow spaces (Fig. 21). Often osteoclasts was slight, the striking feature being the laying down of new bone (Fig. 22).

In the early months after fracture the marrow changes were so clear cut that it was possible to distinguish at a glance living bone surrounded by normal fatty or haemopoietic marrow and dead bone with a few surface excrescences of new bone surrounded by very cellular, proliferative marrow. Later the marrow cellularity decreased but central cores of unresorbed dead bone within trabeculae persisted for several years. This made it possible, even when revascularisation was far advanced, to deduce with a fair degree of certainty the border between bone which, having never been dead, contained osteocytes throughout (Fig. 3), and that which had been dead and now, after marrow revascularisation, was covered by living bone (Fig. 4). If resorption had been unusually active the area of dead bone could appear to be smaller than it had been initially. It is emphasised that clearly defined areas of bone necrosis entombed in living bone were not seen in the femoral heads of elderly controls but they were a feature in bones of young adults with caisson disease.

The patterns of revascularisation were the same in the completely and partly dead femoral heads. In most cases revascularisation was confined to a wedge-shaped area around the fovea. This had its base on the articular surface and advanced into the marrow in a narrower spearhead (Fig. 23). When it reached the fracture line there was usually a shallow triangle of bone still remaining necrotic in the inferior part of the head and a larger area of necrosis in the superior part of the head. Sometimes a small contribution to revascularisation was made by vascular attachments at the inferior margin of the fracture site but these were not usually enough on their own to supply any sizeable area and any spurs of the inferior cortex of the neck were slow to regain a blood supply. The upper weight-bearing part of the femoral head, and especially the subchondral region, was almost invariably the last area to become revascularised (Figs. 26 and 31).

In most of these capital fragments it was clear that no contribution to the restoration of a blood supply had been made from the neck across the fracture site because there was still a mass of dead trabeculae and fibrin at the fracture site (Fig. 24). In the primary arthroplasty group and two of the necropsy cases this can be explained by the absence of fixation. In the group in which pinning failed, inadequate immobilisation or continuing impaction of the dead head may have destroyed attempts by granulation tissue to bridge the fracture line. In seven of the redisplaced fractures there were fragments of dead callus (Fig. 25) lying at the fracture site indicating attempted union and revascularisation. In the forty cases in which pieces of the neck were available for study, although the depth of bone necrosis varied from a few

**Fig. 22**

Twelve weeks after fracture much new bone containing osteocytes has formed on the surface of the dead bone which has empty lacunae. (Haemalum and eosin, × 70.)
millimetres to more than a centimetre there was always very active revascularisation and much formation of new bone on the surface of dead trabeculae. In five completely or partly necrotic femoral heads there was evidence of a vascular contribution from the neck with attempted union and these are described below.

DISCUSSION

The normal vascular pattern of the femoral head and neck in adults has been widely investigated (Kolodny 1925; Wolcott 1943; Tucker 1949; Trueta and Harrison 1953; Judet, Judet, Lagrange and Dunoyer 1955) and the nomenclature adopted by Trueta and Harrison for the blood vessels has been used in this paper. The femoral head is supplied with blood by cervical vessels which cross the marrow spaces from below, by the ligamentum teres (medial epiphyseal artery arising from the obturator artery) and chiefly by the retinacular arteries which run along the neck beneath the synovium. The superior retinacular group gives off first of all superior metaphysial branches and then a larger lateral epiphyseal artery which within the head...
forms an arcade of vessels which run parallel to but above the line of the old epiphysial cartilage plate. These vessels supply the medial and upper parts of the head while the lowest third of the head is supplied by the inferior metaphysial arteries running in the inferior retinaculum. When a fracture occurs the cervical vessels are ruptured and the femoral head then depends for its nutrition on any surviving retinacular blood vessels—though Badgley (1960) believes that all are ruptured in displaced fractures—and on those of the ligamentum teres. As described above it was concluded that there were usually, in these displaced fractures, much destruction and damage of retinacular attachments whether at the time of fracture or after manipulation but that when vessels survived they were more often in the lower than in the upper part of the head (Harty 1953, Merle d’Aubigné and Cormier 1956, Boyd 1957, Hulth 1958a and b, Mathon 1959) although Claffey (1960) believes the inferior metaphysial arteries are always torn. In general, the blood supply of the femoral head depends to a great extent on the medial epiphysial arteries in the ligamentum teres and to a lesser extent on the retinacular vessels, especially the inferior ones. It was apparent that, although in injection studies (Trueta and Harrison 1953, Cheynel 1954, Judet et al. 1955) anastomosis was demonstrated between the various groups of vessels, in practice such blood supply as remained was frequently insufficient to nourish the whole head. The amount of the head which was supplied by the medial epiphysial arteries alone varied from a few trabeculae at the fovea to the entire head. The contribution from any remaining inferior metaphysial arteries was sometimes sufficient alone, or aided by the ligamentum teres vessels, to keep alive the lower half of the femoral head. It is seen that the area of the head most vulnerable to necrosis is the upper weight-bearing area. This histological finding is supported by many other studies such as venography (Hulth 1958a and b), phosphorus32 injection and autoradiography (Boyd, Zilversmit and Calandruccio 1955; Boyd and Calandruccio 1963), tetracycline maps (Woodhouse 1962b) and most recently by Sevitt’s (1964) elegant necropsy radio-opaque injection patterns.

Some revascularisation may occur across the fracture line but when there is no significant contribution from the foveal (medial epiphysial) vessels it is extremely slow and often of small amount. This was seen in the twelve examples of late segmental collapse discussed in greater detail elsewhere in this issue. In eleven of these patients with united fractures much of the head remained dead several years after injury. It was unlikely that the delay and incompleteness of revascularisation was brought about by necrosis of the distal femoral neck fragment because, in the neck samples examined from redisplaced fractures, there was invariably, in spite of varying degrees of initial necrosis, very active marrow revascularisation and much reossification and callus formation (Judit et al. 1955). In comparison with the slow revascularisation across the fracture, the rate of revascularisation from the medial epiphysial arteries, though variable, was quicker, and often continuing, although occasionally it was not, and then the revascularisation front consisted of dense non-osteoblastic fibrous tissue surrounding the dead trabeculae. Small additional contributions to revascularisation were sometimes seen at the periphery of the head where there had been reattachment of soft tissue. It is, however, the arteries of the ligamentum teres which play an important part in the revascularisation of the necrotic or partly necrotic femoral heads and this is in agreement with Sevitt’s findings in 1964. It was very striking that the last area of the femoral head to become revascularised was almost invariably the upper subchondral region (Bonfiglio 1954, Boyd 1957). Small pockets of necrotic bone were still present (Figs. 26 and 31) in two patients with bony union of their fractures (Cases 1 and 2) and in one with fibrous union (Case 4).

The association of non-union and redisplacement of transcervical fractures with an avascular femoral head is well known. Phemister (1949a) found non-union to be four times more common in fractures with a necrotic capital fragment than in those with a live one; and in a recent follow-up study (Brown and Abrami 1964) all of the femoral heads excised and submitted to histology because of redisplacement were, initially, completely or partly
necrotic. The interpretation of this association between non-union and necrosis is difficult. It has been suggested (Compere and Wallace 1942) that inadequate reduction and poor immobilisation are factors leading to death of the head and that fibrous union may result in

Figs. 26 to 28
Case 1—The gross specimen (Fig. 26), slab radiograph (Fig. 27) and histological section (Fig. 28) all show bony union which has occurred in an initially almost completely necrotic head. Only a very small subchondral zone of bone (marked with an arrow in Figure 26) remains dead in the upper head, and it is surrounded by thickened trabeculae.

extension of necrosis in a partly necrotic head (Coleman and Compere 1961). The evidence, however, is unconvincing and there were no histological features in this material to suggest more than one episode of ischaemia. It is more likely that necrosis of the capital fragment
Case 2—Slab radiograph shows early bony union thirteen weeks after fracture in a femoral head more than half of which had initially been dead. Revascularisation was almost complete.

Figure 30—Case 3. Callus is seen bridging the fracture line. (Haemalum and eosin, 30.) Figure 31—Case 4. Fibrous union has occurred and the head which was at the beginning half necrotic has revascularised except for a few tiny upper subchondral pockets marked with arrows. This is the only fracture in the series which was probably, at least initially, undisplaced.
contributes to non-union by failure of callus formation on the head side though it is certainly not the sole cause of non-union (Barnes 1962b, 1964; Nicoll 1963). As is shown below, fractures with a completely or partly necrotic capital fragment may unite.

FRACTURE HEALING IN NECROTIC FEMORAL HEADS

Although in some live or revascularised heads there was early callus formation adjacent to the fracture site, in most of these there still remained broken trabeculae and fibrin which, not yet organised, separated the head from the neck. In five cases femoral heads which had been partly or totally necrotic showed progress towards union. This seems a very small number but in many the femoral head had been removed because of redisplacement of the fracture and most of the specimens from necropsy or primary prosthetic arthroplasty were removed in the early weeks after fracture (Table III).

**TABLE III**

**Time Between Fracture and Removal of Femoral Head**

<table>
<thead>
<tr>
<th></th>
<th>2-4 weeks</th>
<th>5-8 weeks</th>
<th>9-12 weeks</th>
<th>13-26 weeks</th>
<th>27-52 weeks</th>
<th>12-18 months</th>
<th>19-24 months</th>
<th>More than 24 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failed nails</td>
<td>10</td>
<td>7</td>
<td>12</td>
<td>9</td>
<td>14</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Primary replacement</td>
<td>12</td>
<td>12</td>
<td>—</td>
<td>4</td>
<td>—</td>
<td>1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Necropsy</td>
<td>13</td>
<td>3</td>
<td>—</td>
<td>1</td>
<td>1</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>22</td>
<td>12</td>
<td>14</td>
<td>15</td>
<td>6</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**Case 1**—A seventy-six-year-old woman suffered a transcervical fracture of the neck of her right femur which was fixed with a sliding nail plate. She died of bronchopneumonia two years and three months later following prosthetic arthroplasty for late collapse of the left femoral head after a transcervical fracture four years before. There was sound bony union of the right femur. On histological examination the head, which in the beginning had been almost completely avascular, only a small foveal area being spared, had revascularised, apart from a small superior subchondral area which was demarcated by fibrous tissue and edged by broad trabeculae (Figs. 26 to 28).

**Case 2**—An eighty-three-year-old woman with osteoporosis and senile dementia sustained a displaced subcapital fracture of her right femur, which was fixed by a sliding nail plate. She died of bronchopneumonia thirteen weeks later. On microscopy it was seen that a large foveal wedge of bone (33 to 50 per cent of the head) had remained alive. Revascularisation was almost complete but some tiny pockets of dead bone remained in the subchondral region in the upper segment of the femoral head. There was abundant vascular callus on both sides bridging the fracture and early bony union had occurred (Fig. 29).

**Case 3**—This seventy-seven-year-old woman fell and fractured the neck of her femur. At necropsy eight weeks later her death was attributed to bronchopneumonia. There had been sparing of a medium-sized foveal wedge and a spread of revascularisation to much of the head although a shallow subchondral saucer of bone in the upper segment and a spur of the inferior cortex remained necrotic. In places there was a gap between the area revascularised from the neck across the fracture line, but the fracture line itself was already bridged by vascular callus (Fig. 30) and there seemed no reason why, given time, bony union should not have occurred.

**Case 4**—A seventy-three-year-old man fell and fractured the neck of his femur twenty-two months before his death. This fracture, unlike the others, was presumably initially impacted.
but later became partially displaced. No treatment had been given and at necropsy there was fibrous union of the fracture in its inferior part, the upper part of the fracture line remaining ununited. Two small subchondral areas of bone necrosis were still present in the upper segment of the head more than half of which had initially been necrotic (Fig. 31). Had this patient’s fracture been treated by internal fixation it seems possible that more satisfactory union might have occurred.

Case 5—This seventy-six-year-old woman sustained a displaced fracture of her femoral neck which was manipulated and fixed by a Smith-Petersen nail. Eleven months later the nail was extruded and, although there was no redisplacement of the fracture, the head and a small attached trimming of the neck were removed when a prosthesis was inserted. Microscopy showed that the entire head had been necrotic and that only about one millimetre depth of dead marrow at the fovea had been replaced by dense fibrous tissue. Some revascularisation of the fracture line (Figs. 32 and 33) and lower part of the head had occurred from the neck and in places new bone had formed. Some revascularised marrow spaces contained dense collagen and here and there along the fracture line nodules of hyaline or fibrocartilage were present, probably indicating a barely sufficient blood supply. While it seemed possible that the fracture might eventually unite it appeared unlikely that any substantial part of the head would be revascularised without aid from the fovea; later the necrotic bone might have collapsed.

DISCUSSION

While it has been generally accepted that union may occur between a dead femoral head and a live neck (Axhausen 1922, Santos 1930, Palmer 1934, Phemister 1934, Sherman and
Phemister 1947, Charnley et al. 1957, Sevitt 1964) it is usually assumed (Nicoll 1963) that this is later inevitably followed by collapse of the weight-bearing segment.

This certainly seems true of a completely dead head which has revascularised solely across the fracture line with no or almost no assistance from the medial epiphyseal (ligamentum teres) arteries. It is, however, not necessarily true of dogs (Tovee and Gendron 1954, Bonfiglio 1954, Brindley 1963) nor of human femoral heads which are initially almost completely necrotic and have become revascularised both from across the fracture line and above from the foveal region. These femoral heads appear capable of uniting and of being entirely revascularised sometimes with the exception of subchondral pockets in the upper part which are too small to allow any serious collapse of the joint surface. It seems that bony union and a revascularised head may result from prolonged adequate fixation and revascularisation both from across the fracture line and from the ligamentum teres vessels, especially when the initial necrosis is only partial. When necrosis is complete and the contributions from the medial epiphyseal arteries negligible then, although union may occur, usually revascularisation is slow and incomplete and late segmental collapse follows. With inadequate fixation, union is unlikely, especially when the head is dead and bridging callus is only forming from the neck.

If these findings have been correctly interpreted then some difference in prognosis may be expected between completely and partly dead heads; but unfortunately, although many methods have been used to try and diagnose ischaemia of the femoral head at the time of operation, they are not capable of distinguishing between complete and incomplete necrosis; these include injection of dyes (Price 1962), radioactive substances (Tucker 1950, Arden and Veal 1953, Boyd et al. 1955, Arden 1958, Laing and Ferguson 1959, Boyd and Calandruccio 1963), vascular assays both venous (De Haas and McNab 1956, Hulth 1958a and b, Dahlgren 1959, Harrison 1962, Johansson 1962) and arterial (Rook 1953, McGinnis, Lottes and Reynolds 1958) and more recently oximetry (Woodhouse 1962a). In some studies the assessment of the vascular state of the femoral head depends on a bone sample sometimes taken at random and sometimes specifically from the upper segment of the head (Boyd and Calandruccio 1963) because this is the area most liable to necrosis. While this type of study is of very considerable interest the limitations of attempting to determine the ultimate fate of the head from a small sample is realised by these authors. As has been said above, although the upper part of the head may be dead, if there is a live subfoveal wedge and good fixation, bony union and a revascularised head may result. It is emphasised that initial necrosis of only part of the femoral head does not appear to be associated with late collapse of the upper segment. An avascular superior segment bone core alone is therefore probably insufficient evidence to justify immediate prosthetic replacement. Indeed a bone sample taken from the subfoveal region, though perhaps impractical because of the danger of damaging blood vessels, might give more information on the ultimate prognosis. If a bone core from this site is ischaemic then this almost certainly indicates a totally dead head which, though it may unite, is unlikely to become revascularised completely without ingrowing vessels from the ligamentum teres. Collapse of the dead bone may occur later and about half these patients develop joint symptoms severe enough to warrant further operation (Boyd 1957).

**INCIDENCE OF AVASCULAR NECROSIS AFTER TRANSCERVICAL FRACTURE**

All except two of the twenty-nine patients undergoing primary prosthetic arthroplasty more than sixteen days after fracture had this operation done because there was unavoidable delay in treatment. This group of twenty-seven patients, and also the necropsy group of twenty, are unselected in respect of avascular necrosis. Of the twenty-seven femoral heads removed at primary arthroplasty ten were thought to be alive as were six of twenty removed at necropsy, which is a total of sixteen out of forty-seven or 34 per cent (see Table IV). This figure may be inaccurate because the numbers are small and the delay in treatment may have caused further
damage to the blood vessels supplying the capital fragment. It is hoped eventually to collect enough material to obtain a true percentage of avascular necrosis after transcervical fracture.

DISCUSSION

The incidence of avascular necrosis as assessed in clinical studies is very variable. When patients were treated by lengthy immobilisation an apparent increase of radiological density of the femoral head in the ununited fracture indicated its death (Santos 1930, Phemister 1934). Phemister (1934) found an incidence of 65 per cent of necrosis in forty-nine patients, the femoral heads of seventeen being examined histologically. Early walking has made the radiological diagnosis of necrosis of the femoral head in ununited fractures almost impossible

| TABLE IV |
| FINDINGS IN THE HEADS REMOVED AFTER SIXTEEN DAYS |

<table>
<thead>
<tr>
<th></th>
<th>Live</th>
<th></th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Per cent</td>
<td>Number</td>
<td>Per cent</td>
<td>Number</td>
</tr>
<tr>
<td>Replacement*</td>
<td>10</td>
<td>37</td>
<td>14</td>
<td>52</td>
<td>3</td>
</tr>
<tr>
<td>Necropsy</td>
<td>6</td>
<td>30</td>
<td>12</td>
<td>60</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>34</td>
<td>26</td>
<td>55</td>
<td>5</td>
</tr>
<tr>
<td>Failed nails</td>
<td>2</td>
<td>3</td>
<td>28</td>
<td>47</td>
<td>30</td>
</tr>
</tbody>
</table>

* Two late primary prosthetic arthroplasty cases excluded because avascular necrosis was suspected clinically.

(Boyd 1957). The lowest incidence of avascular necrosis is assessed by many authors by the prevalence of late segmental collapse in the united fracture, Garden (1961) finding 15 per cent, Hargadon and Pearson 24 per cent, Cleveland and Fielding 24·4 per cent and Brown and Abrami 28 per cent, in patients followed for more than a year, and Green (1960) 34·5 per cent in all fractures followed for two to nine years. Linton (1944) has pointed out that the incidence of late segmental collapse increases as the length of follow-up increases; from 30 per cent of a two to three-year follow-up to 56 per cent in three to seven years after fracture in his own series, and Cauchox and Rey (1963) found that their collapse rate increased from 25·5 per cent at one year to 37·9 per cent after two years. Recently Charnley et al. (1957) in a series of thirty-three cases of displaced fracture treated by a compression screw considered, because of extrusion of the screw, that some degree of vascular damage was present in two-thirds of the cases. In addition to clinical studies, Boyd and Calandrucio (1963) examined by autoradiography femoral heads removed at primary and secondary arthroplasty after phosphorus32 administration and found that two-thirds showed loss of some vascularity; Woodhouse (1962a and b) said the same on examining femoral heads removed at primary arthroplasty from patients given tetracycline. In twenty-four specimens removed at necropsy Sevitt (1964) found arteriographic and histological evidence of some degree of femoral head necrosis in twenty-one.

In the small unselected group of femoral heads (forty-seven) in the present study it appears that about one-third remained viable and about two-thirds were partly or completely necrotic (Table IV).

CORRELATION OF HISTOLOGICAL AND RADIOGRAPHIC APPEARANCES

Ununited fractures—Although Santos (1930) and Phemister (1934, 1939, 1940, 1943, 1948) found that necrotic ununited femoral heads showed, usually within six months of injury, a relative increase in density compared with the adjacent osteoporotic pelvis and distal femur,
this was not seen in the clinical radiographs in this series since early mobilisation presumably prevented the development of local osteoporosis. Necrosis of the femoral head may not be attended by any change in radiological density (De Haas and MacNab 1956, Boyd 1957, Charnley et al. 1957, Bonfiglio and Bardenstein 1958, Bessler and Muller 1961, Hulth 1961, Woodhouse 1962a). Certainly in these patients with dead femoral heads the extrusion of the nail and disruption of the fracture could not have been forecast on the radiographic appearances. After revascularisation of dead marrow there may be osteoclastic resorption of dead bone and laying down of new bone on the surface of dead trabeculae. The density to x-rays of the reossifying area depends on the ratio of these two activities. While Santos (1930), Phemister (1939, 1940, 1943, 1948, 1949) and Sherman and Phemister (1947) described a decrease of density in the revascularised area of united femoral heads it was recognised by them that this was probably because of lack of function in the hip. In the revascularising capital fragments of this series slab radiographs showed no evidence that revascularisation was accompanied by increased radiotranslucency; indeed there was commonly a slight increase in the trabecular thickness except in the subfoveal zone which in the normal contains thin and scanty trabeculae. This lack of porosis in the reossified bone was probably also the result of relatively early walking. In some patients there was much broadening of the reossified trabeculae and this was specially so at the fracture line when it was covered by dense collagen and fibrocartilage and at the vascularisation front when it had become more fibrous and avascular histologically (Figs. 34 to 36). To summarise, there was a tendency towards an absolute increase of density to x-rays in the revascularised area (Woodhouse 1962a), slight and unremarkable in clinical radiographs in most cases but more marked where the process appeared to have stopped. This was caused by broadened trabeculae where new bone had been laid down on the surface of unresorbed dead bone as described by Hulth (1961) and in rabbits by Bobechko and Harris (1960). Slight marrow calcification was seen in only two femoral heads (Fig. 37).

United fractures—No great alteration in density of the slab radiographs of the necrotic and revascularising femoral heads was found in two of the three patients whose fractures were progressing towards bony union, as in Figure 29, for example. In Case 1 a very small subchondral area of bone which remained necrotic was edged by dense fibrous tissue and broad bone trabeculae (Figs. 26 to 28). In twelve patients with bony union and late segmental collapse reported elsewhere in this issue it was notable that alteration of the contour of the weight-bearing surface was the first radiological evidence of bone necrosis (Bessler and Muller 1961, Barnes 1962a, Woodhouse 1962a). A zone of increased radiological density caused by thick reossified trabeculae was often found later in the most proximal part of the revascularised area, especially when this had become densely fibrous and apparently without any possibility of progress. The very thick trabeculae, causing increased density to x-rays, and found when further vascularisation is frustrated may be analogous to those seen in relation to the bone ends in a pseudarthrosis (Judet, Judet and Roy-Camille 1958).

THE LIGAMENTUM TERES

Clinical appearances—In thirty-four of the eighty-nine patients who had excision of a femoral head more than sixteen days after injury a note stated whether or not the ligamentum teres bled on section. In many there was bleeding from the acetabular end when there was none from the foveal end of the ligament. Although the ligament from four of the five completely dead heads which had not become revascularised did not bleed and those from four of five live heads bled briskly, in the remaining twenty-four there was no very close correlation between the amount of bleeding found at operation and the state of vascularity or revascularisation of the femoral head.
Histological appearances—In only twenty-four of 109 femoral heads removed more than sixteen days after fracture was the whole ligamentum teres available for study but in a further forty-five a small stump was still attached at the fovea. For comparison the ligament was examined from fifty femoral heads selected at random at necropsy from patients over the age of sixty-four, fifteen from necropsies on infants and children under fourteen, and nine from the intervening ages. More than half the ligaments from the elderly controls showed hyaline
A HISTOLOGICAL STUDY OF AVASCULAR NECROSIS OF THE FEMORAL HEAD

Figure 38—Sclerotic blood vessels with concentric fibrosis and very narrow lumina are shown in the ligamentum teres. (Haemalum and eosin, ×140.)

Figure 39—A completely obliterated vessel in the ligamentum teres is seen adjacent to several vascular channels in an 18-month-old child. (Haemalum and eosin, ×225.)

Figure 40—A completely obliterated vessel in the ligamentum teres still shows elastica in the wall. (Weigert's elastica, ×225.)

Figure 41—A vein from the ligamentum teres of a 4-month-old infant shows early hyaline sclerosis. (Haemalum and eosin, ×315.)
sclerosis chiefly affecting small veins. These appearances have been described in detail by Elmore, Malmgren and Sokoloff (1963) in synovial blood vessels from many sites especially in the acetabular fat pad and prepatellar fat. Sclerosis in the blood vessels of the ligament was mentioned briefly by Chandler and Kreuscher (1932) and by Nordenson (1938). The earliest change found in the present series was an eccentric infiltration of hyaline material between the smooth muscle fibres of the media of venules. Later, the whole media became replaced by hyaline material (Fig. 38) and eventually the lumen might become obliterated. The vessel was then a solid mass of hyaline substance bounded by the external elastic lamina (Figs. 39 and 40). Perivascular fibrosis was not seen. The distribution of these lesions was patchy, a group of obliterated vessels sometimes lying adjacent to normal ones. Special stains showed that the hyaline material was not amyloid, fibrin or any of the material described by Lendrum et al. (1962) as being intermediate between fibrin and collagen, but it did, in all its stages, stain as collagen. The hyalinisation was sometimes accompanied by reduplication of elastic laminae.

Sclerosis was found also in the vessels of the ligamentum teres in two of the fifteen children aged four months and eighteen months respectively (Figs. 41 and 39), in the few ligaments at intermediate ages and in the ligaments and retinaculum after fracture. The reason for describing these patchy sclerotic vascular changes is that they might, in the absence of control material, be ascribed to damage of the blood vessels at the time of fracture, or be thought in their later obliterative phase to involve principally arteries and thereby influence the viability or revascularisation of the femoral head. While the number of ligaments available after fracture is too small to attempt any correlation with the vascular state of the head, it seems unlikely that obliteration of small groups of veins would have any notable effect.

It was striking that there was no vascular thrombosis in any of the sixty-nine ligaments, although occasionally a thrombosed vessel was seen in an attached retinacular tag at the periphery of the head. The only remarkable vascular change was that the vessels of the foveal stump of the ligamentum teres were completely necrotic in ten out of eleven necrotic femoral heads which had failed to revascularise and in three in which revascularisation was confined to tiny areas at the periphery of the head at the fracture line. In one of the cases in which the fovea was necrotic the whole ligamentum teres was also available and showed necrosis of all tissue to a depth of about half a centimetre, the more proximal parts of the vessels being alive and patent.

**Changes in the Articular Cartilage**

**Degenerative changes**—The fifty normal elderly controls were examined in relation to degenerative joint changes. The articular cartilage of none of the femoral heads was entirely normal. The least severe changes were those of superficial flaking and of fibrous replacement of the surface cartilage around the fovea and at the periphery of the head. Osteoarthritic changes of fibrillation and cartilage loss were most frequent in the lower head. Three patients showed advanced osteoarthritis with osteophyte and "cyst" formation. Of the 109 femoral heads removed after fracture none showed advanced osteoarthritis but there was no other appreciable difference in respect of degenerative changes between this and the control group.

**Loss of chondrocytes**—In normal elderly subjects some patchy loss of chondrocytes occurred especially in the deeper layers of the articular cartilage. In the femoral heads removed after fracture, changes in the cartilage covering necrotic bone were usually slow to develop and the patchy normal loss of chondrocytes was rarely exceeded until months and sometimes years had passed. The loss exceeded normal in twenty-five of the 109 femoral heads and was most marked in the deep zone of the weight-bearing area of the head. In only one head, removed more than three years after fracture, was there almost complete loss of chondrocytes. It was remarkable that in all these specimens the cartilage kept its normal depth and contour and showed no evidence of disintegration (Hatcher 1952, Hulth 1961).

Nicoll (1963) perhaps took too gloomy a view in suggesting that the articular cartilage of a dead femoral head is "doomed from the start." Cellular loss is usually slow and Phemister
A HISTOLOGICAL STUDY OF AVASCULAR NECROSIS OF THE FEMORAL HEAD

(1934) believed that if revascularisation of the underlying bone occurred reasonably quickly the cartilage would survive. Even if it eventually dies it may fail to disintegrate. The minor cartilaginous changes seen in these femoral heads with normal contours seemed unlikely to give rise to symptoms. This is in contrast to the severe changes which may occur in late segmental collapse once deformity of the joint surface is established. In these cases while the cartilage covering the still unvascularised upper segment usually retains its thickness, that covering the revascularised bone at the periphery of the head may show severe osteoarthrosis. Vascularisation of the cartilage from below, resumption of endochondral ossification, formation of osteophytes, osteoarthritic “cysts” and sometimes formation of a new joint surface may occur—changes which inevitably give rise to joint symptoms.

SUMMARY

1. Loss of osteocytes in the bone trabeculae of the femoral heads of “normal” elderly patients was patchy and distinguishable from that resulting from avascular necrosis after fracture.
2. Changes in the haemopoietic marrow were the earliest and most sensitive indicators of ischaemia, loss of osteocytes rarely being complete until three or four weeks after fracture.
3. In 109 femoral heads removed more than sixteen days after fracture the viability could be determined by histological means. All of these had suffered some damage to the vascular supply but in a number the head remained alive apart from the region of the fracture line. These heads were nourished by the blood vessels of the ligamentum teres and sometimes by retinacular arteries, usually of the inferior group.
4. Some femoral heads became completely necrotic following fracture, others were only partly affected. A variable amount of the subfoveal region commonly remained alive and it was from this site that revascularisation spread into the head. The upper segment of the femoral head least often remained alive and its subchondral region was usually the last to revascularise.
5. In a group of unselected femoral heads a third remained alive following fracture and two-thirds were partly or completely necrotic.
6. Femoral heads which were partly necrotic appeared capable of uniting and completely revascularising, there being invasion of the necrotic bone by vessels from across the fracture line and from the ligamentum teres. This contrasted with the completely necrotic femoral heads described elsewhere in this issue which united but in the absence of proliferation of ligamentum teres vessels failed to revascularise completely and developed late segmental collapse.
7. Avascular necrosis did not appear to be the sole cause of non-union.
8. Necrotic bone showed no alteration in radiological density. Reossifying bone in areas of revascularisation sometimes caused an absolute increase of radiodensity especially when associated with halted revascularisation. This increase of radiological opacity was the result of deposition of new on dead bone with broadening of the trabeculae. Marrow calcification was minimal.
9. Obliterative sclerosis of venules in the ligamentum teres was found in “normal” patients even in infancy. No thrombosis was seen in the ligaments following fracture but where the femoral heads were completely necrotic and not revascularised the ligaments were often also necrotic.
10. There appeared to be no increase in degenerative changes in the articular cartilage of the femoral heads following fracture compared with fifty elderly controls. Some loss of chondrocytes in the deep zone of the weight-bearing area was found in about a quarter of the femoral heads. In only one head was the cartilage almost completely acellular. An almost normal depth and a smooth contour of the articular cartilage were retained.

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REFERENCES


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