OSTEOARTHRITIS OF THE HIP: A STUDY OF THE NATURE
AND EVOLUTION OF THE DISEASE

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Despite the efforts of many workers to increase our understanding of osteoarthritis, effective measures have been devised only for the treatment of the advanced stage of the disease; such treatment usually involves either the total suppression of the affected joint by arthrodesis or the more or less complete removal inherent to arthroplasty. The vogue acquired by these operations proclaims our failure to arrest, and even more to reverse, the progress of the disease before the joint has been completely disorganised, a process which entails prolonged suffering and frustration.

The investigation here reported was initiated under the belief that, as has happened in other conditions, a better understanding of the problems involved might lead to new therapeutic advances. We were conscious from the beginning of the skill and authority of those who had preceded us in the study of osteoarthritis but the persistence of many conflicting views and important gaps in the knowledge on fundamental points seemed to justify further work. Many previous investigations have been directed to the tissue changes produced by the disease and, as methods of research have become increasingly refined, the pathological studies have also been made more penetrating, the study of the cell giving place to that of the molecule. Surgeons, however, have the unique opportunity of seeing the disease as it affects the joint as a functional whole and not as an ailment of a particular tissue such as cartilage, bone, capsule, synovial membrane or synovial fluid.

We have selected the hip because it is the commonest clinical site of severe osteoarthritis and the investigation has embraced not only the disorganised joint of the final phase of the disease but the various stages of its evolution from the normal. Our description is limited, largely by considerations of length, to the femoral head; we have found that the changes in the acetabulum are similar and like mirror images of those occurring in the other member of the articulation.

In the pages that follow we bring the results of our analysis mainly for the consideration of our surgical colleagues who are daily confronted with the responsibility of treating patients suffering from osteoarthritis of the hip.

MATERIAL

This investigation is based upon the following human material: 1) Ninety-one post-mortem examinations of the hip. This material, analysed in Table I, showed all variations from that of normal health to advanced osteoarthritis. 2) Forty-five femoral heads removed at operation from osteoarthritic hips. 3) Radiographs obtained from eighty selected patients suffering from osteoarthritis of the hip in whom it was possible to follow the progress of the disease over several years.
METHODS OF INVESTIGATION

The tissue studies reported below were made with particular reference to the region of the joint in which the lesions occurred: essentially it has been a correlation of pathology and joint topography, and the methods used were as follows.

Dissection and macroscopic examination—The post-mortem examination of hip joints permitted not only the collection of material but the observation of the site and the intra-articular relationship of the various pathological lesions.

Radiography—Radiographs in at least two planes at right angles to each other were made of all specimens. Many of the specimens were then sawn up into slices whose thickness and plane of section varied from specimen to specimen and these were then submitted to further radiography. The extent of cartilage lesions was frequently indicated by the insertion of radio-opaque markers into the cartilage prior to radiography.

Histology—Written and photographic record was kept of the exact area of the joint from which any material was taken for histological examination. Sections embedded either in paraffin or low viscosity nitrocellulose were stained by routine colorimetric techniques; haematoxylin and eosin, Van Gieson. Histological techniques using toluidine blue, colloidal iron (Hale) and periodic acid—Schiff (McManus) were employed for the investigation of the mucopolysaccharide content of the articular cartilage.

Investigation of the vascular pattern of the osteoarthritic femoral head—The methods of vascular injection and the radiographic, transparency and special histological techniques used in this study to demonstrate blood vessels within bone have already been described in a previous paper (Trueta and Harrison 1953). Histological examination of injected tissues has been of inestimable value in interpreting the vascular arrangements in sections cut from uninjected material.

SOME PRELIMINARY CONSIDERATIONS OF THE ANATOMY OF THE HIP JOINT

Before describing our pathological findings it is considered necessary to emphasise some anatomical features of the hip joint.

The human hip is classed as an enarthrosis or a ball-and-socket joint, but certain features of the acetabulum considerably reduce the implications suggested by this definition. With the exception of the fovea capitis the femoral head is entirely covered by hyaline cartilage; but the acetabulum is much less completely lined. There the cartilage has the shape of an inverted horseshoe between whose two limbs is the acetabular fossa (Fig. 1) which contains a pad of fat covered by synovial membrane. Such an arrangement of the acetabulum entails that in any position of the joint a considerable part of the surface of the head of the femur is opposed to the soft tissues filling the acetabular fossa and not to acetabular articular cartilage. This fact may be demonstrated in sections of fresh cadaveric hip joints. It ensures that a considerable area on the infero-medial surface of the femoral head is prevented by lack of articular contact from the transmission of joint stress. Such sections also demonstrate that anteriorly, superiorly and, to a lesser extent, posteriorly, the marginal articular cartilage of the femoral head tends to project laterally outside the acetabulum. Only in the very extremes of movement can most of this peripheral cartilage of the head be brought into articulation with the acetabular cartilage.

Throughout this article the articular cartilage on the head of the femur which in the normal ranges of movement articulates with the cartilage of the acetabulum will be

![Fig. 1](Sketch of the right hip bone showing the extent of the articular cartilage and the acetabular fossa.)
designated as occupying the pressure area; that which does not is termed the cartilage of the non-pressure area. The differentiation between the pressure and non-pressure areas on the surface of the head may be traced across into one of the main trabecular systems of the cancellous bone of the head. Before epiphysial fusion the spongiosa of the head has a close-mesh structure which permits little differentiation of individual systems. Nevertheless a band of trabeculae (a., Fig. 2), which it is desired to draw attention to in later decades, can already be defined in the metaphysis, running through the lower part of the femoral neck from the corticalis below, up towards the epiphysial plate. After fusion of the epiphysis this trabecular system can be traced upwards until it reaches the articular surface of the head in its superior part. A large range of variation exists, and it is not exceptional to find adolescent patterns of trabeculation even in later decades of life. The more typical of the trabecular patterns in adult femoral heads takes the shape of a column with its upper part spreading mushroom-like and which in frontal sections (Fig. 3) appears like a fan. We shall name this

![Fig. 2](image1.png)

**Fig. 2**
Radiograph of a coronal section from a femoral head, subject aged fourteen years. a. = bone trabeculae referred to in the text.

![Fig. 3](image2.png)

**Fig. 3**
Radiograph of a coronal section from a femoral head, subject aged eighty-six years. a. = system of bone trabeculae referred to in the text.

group of trabeculae the pressure system because they are concerned with the transmission of forces from the trunk to the lower limbs and from the lower limbs to the trunk; indeed they can be traced into continuity across the joint with a group which runs within the ilium up towards the sacro-iliae joint. As indicated in Figure 4 the distribution of these trabeculae allows articular cartilage on the surface of any head to be subdivided into: 1) that which overlies the trabeculae of the pressure system; 2) that on the infero-medial aspect of the femoral head, medial to the pressure system; and 3) that at the edge of the femoral head, lateral to the pressure system. By studying hip joint specimens and slab radiographs subsequently obtained from them it is possible to show that the areas just described have the following significance. The cartilage which overlies the pressure system is that which articulates with the acetabular cartilage and therefore occupies the pressure area. The part of the cartilage infero-medial to the pressure system is that which faces the acetabular fossa and will be designated the medial non-pressure area. The cartilage lateral to the pressure system is that which projects externally out of contact with the acetabular cartilage. It will be designated the peripheral non-pressure area. The diagram in Figure 5 depicts a distribution of the pressure and non-pressure areas which is commonly present in the normal adult femoral head.

One final definition necessary for the description which follows is that of the pressure segment. By this we mean that portion of the femoral head traversed by the trabeculae of the pressure system.

At the time when this work was started it was found that no detailed description of the
Fig. 4
Antero-posterior radiograph of a femoral head. The surface cartilage is subdivided, as described in the text, into areas (1), (2) and (3) relative to the pressure system of trabeculae.

Fig. 5
Distribution of the pressure and non-pressure areas commonly present in the normal adult femoral head. Trabeculae of the pressure system are shown in red. Non-pressure areas are cross-hatched in black.
normal vascular pattern of the femoral head, based upon radiographic or photographic evidence, existed in the literature. Such information being necessary as a control material for the present study, the normal vascular anatomy was investigated and the results have already been published (Trueta and Harrison 1953).

**Fig. 6**

The articular cartilage shows an early stage of degeneration with disruption of its superficial or tangential layer. 

*Fig. 7*

Advanced stage of cartilage degeneration; the appearance of collagen fibres and fissuration is typical of the state of "fibrillation."  

*Comment*—For the anatomical reasons presented above the hip affords excellent opportunities for a controlled investigation of the effects of stress and strain on the tissues of the joint and the distribution and thickness of the bone trajectories of the pressure system are an important guide to such a study.

In a comparative survey Walkhoff (1904) showed that this system is exclusive to *the*
adult human; none of the lower animals or apes, nor even the femoral head of the Neanderthal man, possesses it. The factor which appears to be responsible for the organisation of the pressure system is the locking of the hip joint in extension, an event inherent to the erect position peculiar to man. Without discussing the merits or otherwise of those theories which seek to explain the factors conditioning the internal architecture of bone there is considerable evidence to support those who, like Thoma (1907), Jansen (1920) and Carey (1921), attribute to pressure the role of directing the configuration of the spongiosa. Thoma (1907) showed that the determining role of pressure is bound up not only with the quantity of the load but also the period of time during which it is acting. It seems probable to us that the duration of weight bearing, habits of stance and the relatively restricted joint usage of adult life as opposed to those of youth and adolescence, may explain the tendency to increasing definition of the pressure system in the mature human. This concentration of the load explains the thinning of trabeculae in the lightly stressed segment of the head, particularly that part which is opposed to the acetabular fossa. As will become apparent in the following sections, a progressive evolution of the pressure system occurring in the hip joints of some adults appears to be incompatible with continued joint health.

RESULTS

The findings of this investigation are integrated and presented below as an account of the evolution of an osteoarthritic hip joint from a normal one. We believe that this is preferable to a detailed description of the isolated pathological data because therapeutic advances will probably be possible only when a complete understanding of pathogenesis has been achieved.

Without exception, in our material, the initiation of the osteoarthritic process took place in the articular cartilage. If the disease progresses, this initial stage is followed by osteophyte formation, flattening of the femoral head, eburnation, necrosis, sclerosis, cyst formation and extrusion of the head. These different phases of osteoarthritis will now be detailed.

The nature, frequency and distribution of the cartilage lesion—The earliest macroscopic change in the cartilage is the replacement of its normal, smooth, shiny surface by a mat irregular one which feels softer than normal when pressed upon by a probe. As the cartilage becomes further altered its surface continuity is lost and it presents a filamentous appearance; fragments of cartilage become separated and lie free in the joint cavity. Microscopically the early cartilage lesion consists of changes in the non-calcified part of the cartilage which result in the condition known as fibrillation (Figs. 6 and 7). Histochemically these changes are seen to be accompanied by a reduction in metachromasia of the cartilage.

Every femoral head examined post-mortem in subjects of fourteen years or over showed some area of cartilage which was suffering from the changes just mentioned. In the second decade the area of macroscopic cartilage degeneration was small, but beyond this age all grades of extent and severity were encountered (Figs. 8 and 9). An analysis of our post-mortem material from subjects aged fourteen to a hundred years showed that 71 per cent of femoral heads had cartilage degeneration confined to the non-pressure areas whereas in only 3 per cent was the same change restricted to the pressure areas. In the remaining 26 per cent the cartilage lesion was present in both areas. Figure 10 illustrates how the localisation of the affected cartilage was achieved.

The degree of cartilage degeneration is not uniform within the imprint of the acetabular fossa on any one femoral head. In some places circumscribed loss of cartilage produces ulcers, in others it is a diffuse velvety change which provides the contrast to normal cartilage. As shown in Figures 8 and 9 it is usual to find the rest of the cartilage—namely that of the pressure area—in an excellent state of preservation.

The term "ageing" is frequently applied to this cartilage degeneration but it must be emphasised that these changes are neither restricted to the elderly, nor could any direct
Examples of the degenerate cartilage visible on femoral heads examined post-mortem. Figure 8—Subject aged eighty-two. Figure 9—Subject aged seventy-nine. In each figure, a. = posterior surface, b. = anterior surface, c. = superior surface, d. = medial surface. In both specimens the cartilage degeneration is confined to the non-pressure areas. It is relatively mild in Figure 8 and severe in Figure 9. Note that the cartilage of the principal weight-bearing surface depicted in view c. is well preserved.
relationship be established between the age of the subject and the severity of the cartilage degeneration.* The comparison of the cartilage lesions of thirty-four femoral heads with their respective radiographs strongly suggested that the more evident the articular degeneration within the non-pressure areas the more clearly visible was the pressure system on radiography. Those femoral heads which had a diffuse infantile type of trabecular architecture showed the least cartilage degeneration and the greatest cartilage preservation.

Comment. Most investigators believe that the articular cartilage is the first tissue to show change in a joint which is becoming osteoarthritic. Nichols and Richardson (1909), Allison and Ghormley (1931), Lang (1934), Bennett et al. (1942) and Collins (1949) are among those who have expressed this opinion whose truth has been seen in this present study. We have

not, however, found any reference to the fact that the commonest sites of this change are those parts of the joint exposed but little, if at all, to the stresses and strains of joint function; indeed the opinion which is frequently reiterated in the literature is that "excessive wear and tear" is the causative factor of cartilage degeneration. Our somewhat surprising findings have forced us to consider that if excess of joint pressure is deleterious to hyaline cartilage the lack of pressure is an even more compelling cause of its degeneration.

The necessity for use and compression of cartilage in order to maintain its continued health strongly suggests that mechanical forces are concerned with cartilage nutrition. We have described elsewhere (Trueta and Harrison 1953) a system of vessels found immediately

* Detailed study of the early stages of osteoarthritis of the hip is not possible in material recovered from operation, for patients are not subjected to surgery until the disease is far advanced. For this reason we have investigated the inception of the osteoarthritic process in post-mortem examination of the hips of persons dying from a variety of causes. Such joints exhibited every degree of severity of the disease from the slightly affected to that indistinguishable from the appearance seen at arthroplasty. We believe that the lesions described in this report as constituting the early stage of the disease based upon post-mortem studies are truly the forerunners of advanced osteoarthritis; one can clearly see the evidence of their earlier existence and distribution amidst the bizarre distorted joint wreckage that is removed at operation on living patients.

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beneath the calcified zone of the articular cartilage, an arrangement that could be well suited for the supply of nutrients. Study of a series of papers from Swedish workers such as those by Ingelmark and Saaf (1948), Ingelmark (1950), Ekholm (1951) and Ekholm and Norbäck (1954) shows not only that materials can in fact pass into cartilage from its subchondral surface but that joint function materially increases this passage. This does not in any way decry the nutritive value of synovial fluid. It seems probable to us that the intermittent pumping action of alternate pressure and rest is essential for adequate nourishment of articular cartilage and we have found that Müller had suspected this in 1929. Certainly the weight-bearing areas contain cartilage of greatest thickness and mucopolysaccharide content.

Joint pressure which is excessive either in quantity or duration would prevent the adequate tissue-fluid exchange of the cartilage and thereby be likely to interfere with normal metabolism and promote its degeneration. This concept enables one to understand the

![Photomicrograph showing increase in depth of the calcified cartilage. The section depicts articular cartilage in an early stage of degeneration and as the calcified layer is traced from left (a,) to right (a,+) across the field it is seen to increase in depth and spread widely into the previously uncalcified cartilage. At b, a blood vessel has advanced deep into the calcified layer. The connection between the vessel and the marrow circulation cannot be seen in this section. (H. and E. × 38.)](image)

mechanism of degeneration of that cartilage in the human hip joint which is excluded from the intermittent compression of movement and weight bearing, and also the experimental observations of Fisher (1929), and of Bennett and Bauer (1937), that degeneration occurred in the cartilage of the trochlear surface of the rabbit’s femur after displacement of the patella. Similarly Pollicard (1936) and Retterer (quoted by Franceschini 1944) found thinning of articular cartilage after inactivity of a joint.

After these considerations of the initial cartilage degeneration we shall follow the subsequent fate of, first, the cartilage of the non-pressure areas and, later, that of the pressure area.

**The sequelae of the early cartilage lesion within the non-pressure area**—Following the degeneration and reduction in depth of the non-calcified part of the cartilage, the calcified zone increases in thickness and the darker staining bands within it increase also in depth and number (Fig. 11). Calcification thus moves surfacewards into the radial zone of the
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Fig. 12
Vascular invasion of degenerate articular cartilage. Figure 12—At a., an artery passes superficially into the thickened calcified zone (H. and E. - 39). Figure 13—Higher magnification of the artery shown in Figure 12 (× 200). (Injection mass barium).

Fig. 13

Fig. 14
Formation of bone and marrow within degenerate articular cartilage. Figure 14—Coronal section of a femoral head. Whereas the articular cartilage of the pressure area is well preserved, that of the medial non-pressure area is degenerate. The trabeculae of the pressure system are clearly visible. (H. and E.). Figure 15—A part of the enclosed area of Figure 14 shown at higher magnification. Bone and marrow have been formed within the degenerate articular cartilage. a. = subchondral bone plate. b. = surface covering of fibrocartilage. (H. and E. - 70.)

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non-calcified cartilage. As the process continues the subchondral blood vessels enter the calcified cartilage (Figs. 12 and 13) and progress towards the joint surface, their rate of advance being paralleled by that of the cartilage calcification. It thus comes about that where once there was a normal non-calcified cartilage which became degenerate, there is now a highly vascular marrow, calcified cartilage and bone (Figs. 14 and 15). The vessels spread widely into the cartilage advancing towards and parallel to the joint surface, usually but not always (Figs. 16 and 17) preceded by a vanguard of calcification. A layer of fibrocartilage in varying degrees of degeneration covers the vascular marrow and the newly formed bone; these tissues which have been laid down at the expense of the degenerate articular cartilage constitute the osteophyte. We use the term osteophyte to indicate any new bone and marrow formed within a degenerate articular cartilage and do not restrict it as do other writers to projecting masses of bone and cartilage found at the periphery of the articular surfaces. The evidence for such a definition is presented below.

**The topography and vascular anatomy of the osteophyte**—The earliest osteophytes visible are those found at the junction of articular cartilage and synovial membrane. On the femoral head such junction occurs at the margin of the fovea capitis medially and at the periphery of the articular cartilage laterally. The area of origin of these two groups of osteophyses is represented diagrammatically in Figure 18.

It has already been pointed out that the origin and growth of an osteophyte depends upon the proliferation of blood vessels into the degenerate articular cartilage and
in these peripheral osteophytes the vessels come from the adjacent subsynovial tissues. This we term extra-capital vascularisation (Figs. 19 and 20). Other osteophytes invisible to naked eye examination of the external surface of the head are forming elsewhere in the medial non-pressure area and may be seen in slab radiographs and histological preparations (Fig. 21). The vessels in such osteophytes are derived from the subchondral vessels; this we term intra-capital vascularisation (Fig. 22). Initially an osteophyte is often vascularised exclusively from either one of these two sources, depending on its position, but ultimately most receive their blood from both the intra- and extra-capital vessels.

As an osteophyte increases in size its growth is preceded by the intra-cartilaginous spread of the original vascular penetration and by the appearance of new vessels from whichever source did not contribute initially; these groups of vessels and the bone they form gradually
tend to coalesce. It is quite common to find, as depicted in Figure 23, the original articular cartilage and subchondral bone plate of the non-pressure area persisting beneath the osteophyte; extra-capital vessels are then chiefly responsible for the new growth of tissue and arteries can be seen within the osteophyte running parallel and superficial to the old articular cartilage (Fig. 24). Within the osteophyte the pattern of the smallest vessels is frequently sinusoidal; in these instances the marrow of the osteophyte is red (Figs. 25 and 26). The expansion of the osteophyte in a medial direction is, as everywhere, preceded by the spreading of the blood vessels into the adjacent articular cartilage (Fig. 24). Lateral growth is probably associated with a distinctive vascular arrangement found at the projecting margins of the peripheral osteophytes. Here, a great number of blood vessels are present in the subsynovial tissue which lies between the peripheral osteophyte and the femoral neck and these give rise to

Fig. 21—Coronal section of a femoral head. The articular cartilage has disappeared from the pressure area; that of the non-pressure areas shows osteophyte formation at a, b, and c. (H. and E.) Figure 22—Intra-capital vascularisation. Photomicrograph of the osteophyte b. in Figure 21. a—subchondral bone plate. b, b, b—vessels serving the sinusoidal bed of the osteophyte. (Section 400μ thick, unstained preparation. Injection mass barium preceded by Berlin blue. - 8.)

Fig. 23—Coronal section of osteoarthritic femoral head removed at arthroplasty. A large osteophyte, a, grows on and over the cartilage of the medial non-pressure area. Part of the original articular cartilage persists beneath the osteophyte. (H. and E.)
a series of parallel straight vessels which course along the deep surface of the osteophyte (Fig. 27). Examination of this surface shows bone lamellae being laid down by intramembranous ossification parallel to these vessels.

![Fig. 24](image)

The vascular tree within an osteophyte of the medial non-pressure area. An artery running superficial and parallel to the subchondral bone plate, a., distributes branches to the osteophyte which grows not only towards the joint surface but also at its medial extremity, b. Intra-capital vascularisation is beginning at c. (Spalteholz preparation, injection mass barium. × 8.5.)

![Fig. 25](image)

A photograph (Fig. 25) and a radiograph (Fig. 26) of a coronal slice from an osteoarthritic femoral head removed at arthroplasty. The osteophyte of the medial non-pressure area, a., is seen to contain red marrow.

We must emphasise that without exception the spread of osteophyte formation is limited to those areas within the joint which do not transmit weight: the tendency of osteophytes to grow into the free spaces of the joint outside the weight-bearing areas allows maximum
growth inferiorly (Fig. 28). In advanced osteoarthritis these large inferior osteophytes coalesce with those of the medial non-pressure area and with the ring of osteophytes situated at the margin of the joint cartilage (Fig. 29). As will be seen in this figure, the peripheral circle of osteophytes projecting from the edge of the femoral head forms one wall of a groove or gutter which runs around the neck and is occupied by synovial and subsynovial tissue (Fig. 30).

Comment—Osteophytes have frequently been considered the most characteristic features of osteoarthritis; indeed the disease has been named hypertrophic arthritis with reference to these structures. There has been considerable speculation about their pathogenesis and significance and the findings of this investigation clash with several ideas that have previously been put forward. We see no justification for referring to osteophytes as “marginal” structures; they grow in any area of low joint stress, of which the peripheral border of the articular cartilage is but one. They cannot therefore be considered the resultants of various mechanical stimuli which might operate at the junction of the articular cartilage and synovial membrane (Bennett and Bauer 1937), nor are they in any way secondary to articular cartilage degeneration occurring more centrally (Fisher 1922 and 1929, Bennett et al. 1942).

The osteophyte is the result of an attempt to revitalise degenerating cartilage, a degeneration which, as pointed out in the preceding comment, is most commonly due to malnutrition caused by the lack of an adequate and alternating pressure. This inadequacy of pressure is dictated by the joint anatomy and restricted function, and the osteophytes mark the site of a biological response to a tissue sick from under-work. There is a close similarity between the sequence of events responsible for the normal growth of bone and that
which produces the osteophyte. The inception and growth of primary and secondary ossification centres and the ossification at the epiphysial plate resembles osteophyte production in that a penetration of blood vessels into a degenerating and calcified cartilage is followed by the formation of bone. In osteoarthritis, however, the stimulus to this vascular invasion is apparently not a growth hormone but a metabolic disorder taking place in situ.

Pommer, and Lang (1934), understood the significance of the vascular invasion of cartilage in osteoarthritis, but in their theory of the disease they failed to appreciate that it is the lack of pressure on cartilage rather than an excess which most commonly promotes this process and which exclusively allows it to flourish.

**The sequela of the early lesion within the pressure area** - Under the influence of pressure and joint movement the degenerate articular cartilage of this area becomes progressively thinned until only the basal layers of the radial or even the calcified zone may remain. Such changes are responsible for the progressive decrease of joint space seen in the radiographs. This degenerate cartilage also is invaded by the subchondral vessels, but the bone which is produced thereby is sclerotic and this process of osteogenesis tends to be effaced by certain further events which will be described below.

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**Fig. 29**
The black areas indicate diagrammatically the distribution of the osteophytes in advanced osteoarthritis; at the bottom right the block of coalesced osteophytes is shown detached from the head. The point of the arrow G lies in the gutter which circles the femoral neck between it and the overhanging marginal portions of the osteophytes.

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**Fig. 30**
An osteoarthritic femoral head removed by section through the neck of the femur. The specimen lies upon the fovea capitis and presented to the camera is the cut surface of the neck, a, and the projecting tips of the marginal osteophytes, b. The synovial membrane, c, occupies the interval between these two structures.
The arterial pattern of the osteoarthritic femoral head—The response of the small vessels adjacent to the degenerate articular cartilage with which we have so far been concerned is accompanied by changes in the larger arteries within and around the osteoarthritic femoral head. In advanced osteoarthritis the outstanding feature is the state of hypervascularity which exists when compared with the normal; this occurs with the retention of the overall arterial pattern of the normal state (Figs. 31 and 32). Apart from the normal vascular pathways there is in osteoarthritis a series of vessels entering and leaving the head in the depths of the gutter between the peripheral osteophytic ring and the femoral neck. The vascular foramina through which these vessels pass can only be seen when the fibro-fatty synovial tissue which fills the gutter has been removed (Fig. 33). In contrast to this new vascularisation of the head, the medial epiphysial blood flow must often be reduced in importance as the ligamentum teres becomes compressed by the growth of the foveal osteophytes; commonly the ligament is reduced to an attenuated strand.

Comment—Quite a few investigators have considered that a vascular disorder is responsible for the etiology and pathogenesis of osteoarthritis; many have favoured a concept of ischaemia. Wollenberg (1909) put forward this view and Goldhaft et al. (1930) reaffirmed it. Bürger and Müller (1921), and Phemister (1940), suggested that primary changes of the subchondral vessels might result in nutritional interference with articular cartilage. Strangeways (1929) and Lacapère and Drieux (1952) considered that such interference might follow a decrease in the calibre of the synovial arteries with a resulting change in the synovial fluid. Pridie (1952) stressed that an original vascular catastrophe within the neck of the femur is responsible for osteoarthritic changes within the head. Therapeutic implications of the supposed ischaemic state have been made, among others, by Venable and Stuck (1946), Cheynel (1947) and Camera (1952).

We have been fortunate to have at our disposal techniques which have revealed the
vascular tree within the bone and soft tissues of the joint at the various stages of the disease. These have shown a state not of ischaemia but of vascular profusion and dilation. This is to be seen at its greatest in the advanced stages of osteoarthritis, but angiographic studies earlier in the disease have shown that this in no way represents a reaction to an earlier ischaemia. The osteoarthritic process from its inception entails the appearance and growth of new blood vessels, and as it evolves so does the vascular tree in and around the bone. We are now investigating whether the pain so characteristic of osteoarthritis is dependent upon this particular feature of the disease.

![Image](image.jpg)

**Fig. 33**
The same specimen as depicted in Figure 30 after removal of the synovial membrane and subsynovial connective tissue. A series of foramina are now visible in the depths of the gutter, a., between the femoral neck and the marginal osteophytes.

**THE DESTRUCTIVE PHASE**

The osteoarthritic process up to the stage we have just described has been in its productive phase, wherein a state of hypervascularity has been produced in response to a stimulus provided by the degenerating articular cartilage. This response has been accompanied by the formation of osteophytes which, from a modest beginning in the non-pressure areas, later reach their greatest development after certain occurrences within the pressure segment have further decreased the congruency of the joint. These take place in the second or destructive phase of the disease.

**The flattening of the femoral head**—As osteoarthritis develops in the hip there is a progressive increase in the load that a part of the joint is called upon to bear. This is because muscle spasm, capsular thickening and anatomical incongruity steadily decrease the range of movement of the joint and therefore only a restricted segment of the pressure area is used for weight bearing. The bony architecture of the head, rarefied and weakened by the hyperaemia already described, receives this increased load whose crushing effect is heightened by the progressive loss of resilient articular cartilage from the pressure area. As joint movements are lost and the pressure area becomes more circumscribed the cancellous bone of the head evolves further away from the pattern of youth and the pressure system of trabeculae becomes denser, appearing radiographically more distinct, partly because of the rarefaction of the surrounding bone. The result of this increase in the load and weakening...
of the support is that the head starts to decrease in height from successive trabecular fractures. At first only the superficial trabeculae break down but these are followed by fractures of deeper strata. In this way the top of the femoral head becomes flattened (Figs. 34 and 35); the bone of the collapsed segment increases its density in the radiographs.

**Characteristics of the pressure area in advanced osteoarthritis**—The combination of excessive pressure and friction which operates in the pressure area destroys the full thickness

of the non-calcified cartilage and denudes the upper surface of the femoral head (Fig. 36). The appearance of this area is never uniform because four different tissues may be found there; these are the calcified zone of the cartilage (Fig. 37), necrotic bone lamellae (Fig. 38), living lamellae, and aggregations of newly formed cartilage of varying degrees of cellularity.
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It is the first two tissues which endow the surface with its shiny smooth "eburnated" appearance; the newly formed cartilage is either set slightly below the surface in small "pits" or projects superficially as sproutings (Fig. 39).

The tissues of the pressure segment of the head of femur after flattening has occurred. The marrow, bone trabeculae and the vascular pattern of this area all have distinctive features and, as will be made apparent in the discussion, these may possibly have a common origin. The several appearances will now be detailed.

![Fig. 37]

Photomicrograph of part of the eburnated surface of the pressure area from a severely osteoarthritic femoral head. 

- calcified cartilage
- dilated venule within the fibrous marrow. The greater part of the thickened bone appears healthy; in the immediate subchondral area there are some acellular lamellae.

(H. and E. × 54.)

![Fig. 38]

Photomicrographs of two parts of the pressure area of a severely osteoarthritic femoral head. In both fields the articular surface is constituted by sclerotic and necrotic bone lamellae which extend deeply and irregularly into the head. (H. and E. × 37.)

The marrow is completely different from either haemopoietic or fatty marrow and its general appearance is best described as fibrous with variations depending upon degrees of cellularity and types of fibre component. A further constant feature is the presence of intercellular fluid highly suggestive of a state of oedema. The bone trabeculae are thicker than normal and more sinuous in shape and consist of healthy lamellae sometimes covering dead bone; they constitute the sclerotic bone whose density is so apparent in clinical and slab radiographs. The vascular pattern of the region, in addition to the arterial hyperaemia evidenced in Figure 31, is dominated by a state of venous engorgement which is represented...
among the small vessels by large numbers of dilated veins and sinusoids (Figs. 40 and 41). These vessels have the same general configuration as normal sinusoids (Trueta and Harrison 1953) but differ in the increased calibre of the individual vessels and the size of the meshwork which they form by their anastomoses (Figs. 42 and 43).

The three tissue appearances just described are not spread diffusely throughout the head but are found restricted to the upper part of the pressure segment. Study of a large number of preparations has impressed us with the frequency with which the sclerotic bone and fibrous marrow has the shape of an inverted cone whose apex lies inside the head and whose base is formed by a portion of the naked eburnated surface of the pressure area (Figs. 44 to 46).

Two further and most important features of this part of the osteoarthritic head must now be mentioned: the cysts of osteoarthritis and the presence of necrotic bone and marrow.

![Image](https://example.com/image.png)

**Fig. 39**

A photomicrograph of part of the pressure area illustrating how the bone marrow differentiates into fibrocartilage. This newly formed cartilage projects superficially beyond the bone margin into the joint cavity.

(H. and E. × 45.)

**The cysts of osteoarthritis**—We shall apply the term cyst to those transradiant areas which may often be seen in clinical radiographs of osteoarthritic hips. Their appraisal is more accurate if the osteoarthritic head is sectioned and the several slices radiographed; additional information is obtained if the plane of section is varied in different specimens. By this method of investigation combined with microscopic study the following conclusions have been drawn. Every osteoarthritic head in our series which had been removed at operation contained cysts. The cysts were found restricted to the upper part of the pressure segment and lay within the conic areas of dense bone deep to the eburnated articular surface. The number of cysts present in any one specimen was variable; it is usual to find several; the solitary cyst is less common. The cysts are frequently cup-shaped or pyriform and whereas the former obviously communicate with the joint cavity the "stoma" of the latter (Fig. 47) may be missed by the plane of section used in preparing the material for slab radiography or microscopy. Under such circumstances the structure might seem to be a discrete spherical lesion within the bone, separate from the joint cavity (Figs. 26, 28 and 51); but in many such cases we have been able to demonstrate the actual communication by varying the plane of section.

We desire to relate the topographic and histological features of the cysts to the tissue appearances already described as obtaining in this zone. Thus the morphology of the fibrous
OSTEOARTHRITIS OF THE HIP: A STUDY OF THE NATURE AND EVOLUTION OF THE DISEASE

Venous engorgement within the pressure segment of the osteoarthritic femoral head. Slab radiographs (upper photographs) and angiograms (lower photographs) from the right and left femoral heads of a subject aged seventy-seven. The right femoral head (Fig. 40) was well preserved. The left (Fig. 41) was the site of advanced osteoarthritis. The multiple punctate opacities within both angiograms are collections of sinusoids; the conical area, a., in the osteoarthritic specimen is an accumulation of dilated sinusoids and veins. (Both specimens injected by the same technique. Injection mass barium preceded by Berlin blue.)

Photomicrographs showing normal sinusoids (Fig. 42) from the femoral neck compared with congested sinusoids from the pressure segment of the same osteoarthritic femoral head (Fig. 43). (Both photographs made from an unstained section, 400μ thick. ×20. Injection mass barium preceded by Berlin blue.)
Sclerotic bone in advanced osteoarthritis. A slab radiograph (Fig. 44), and a photomicrograph (Fig. 45) of a coronal slice taken from a femoral head removed at arthroplasty. The outline of the sclerotic bone which occupies the upper part of the pressure segment of this femoral head is triangular. Within this zone, in Figure 44, there is a transradiant area which microscopy shows to be filled by dense fibrous and fibrocartilaginous tissue (a., Fig. 45). The fragment of bone, b., surmounting this area and projecting into the joint was found to be completely necrotic.

The upper half of a sagittal section of a severely osteoarthritic femoral head; the small diagram shows the plane of section. A triangular shaped complex of sclerotic bone and fibrous marrow is present within the head. One corner of the triangle lies deeply, a.; the other two (a', a'') lie at the articular surface. Two large areas of fibrous tissue showing varying degrees of cavitation lie within the triangle; such fibrous tissue is transradiant and appears as a cyst in radiographs.
tissue within the cysts is as varied as that elsewhere in the pressure segment and may be loose and myxoid, dense (Figs. 48 to 50) and on occasions fibrocartilaginous. The former varieties are associated with intercellular fluid, often under tension, and the latter with anatomical conditions exposing the tissue to the stimuli of joint movement (Figs. 44 and 45).

The vascular pattern of the cysts is distinctive. Dilated vessels of sinusoidal pattern occupy the conical areas which we have described within the pressure segment. As the cysts are approached the degree of dilation increases, reaching its greatest at their edge where a layer of large thin-walled anastomosing venules lines the bony wall of the cyst (Figs. 51 and 52); a number of slender arterioles can be traced into continuity with these, the transition between the two often being somewhat abrupt. The fibrous tissue of the cyst contains a few of both these types of vessels but the paucity therein is most striking when compared with the profusion that exists around its walls and in the surrounding tissue.

The bony wall of the cyst is formed by thickened trabeculae. Osteogenesis is frequently evident there and tends to be most marked around the “neck” of the cyst whereas it is usually absent in the “fundus.”

**Necrotic bone and marrow**—Reference has already been made to the necrotic bone which is to be found on the surface of the pressure area. Such tissue is often in unbroken continuity with zones of necrosis extending for a considerable depth into the upper part of the pressure segment. It is common to find necrotic tissue scattered irregularly throughout this part of the head after flattening has occurred, and, judging from histological appearances, the necrotic process is an aseptic one. The necrotic bone and marrow may remain but little changed, they may be ground into bone “sand,” or they may be the site of osteoclastic removal and osteoblastic replacement (Fig. 53). The commonest situation for necrotic bone is within and between the cysts that have just been described. In the present investigation the almost invariable finding of necrotic bone on the surface of the pressure area in advanced osteoarthritic femoral heads has led us to believe that at this advanced stage of the disease ischaemic changes are a usual occurrence.

**Comment**—The osteoarthritic head during the first or hyperaemic phase has been productive of new joint tissue but in its second or destructive phase the joint tends to disintegrate. What has been responsible for these changes is, we believe, the transmission of an excessive amount of pressure through an excessively limited area of the head for an excessively long time. This severe compression falls upon a head whose structure has been weakened by hyperaemia and from which has been removed the normal resilient and protective covering of articular cartilage. The consequence of these circumstances is the collapse of the head and in its turn this produces further important changes in internal structure.

That the zones of necrotic bone exposed on the surface of the pressure area must be attributed to infarction consequent to collapse is shown by the co-existence there of other zones of living trabeculae; if attrition were the causative factor the surface layers would be uniformly dead. Moreover, the dead lamellae often extend in unbroken continuity into the head to strata which would be unaffected by the polishing action of joint movement. The presence of calcified cartilage upon the surface shows that the flattening of the head is not due to a wearing away but is in fact an impression.

To judge by the devitalised remnants found inside and on the surface of the conical areas of the pressure segment, it is very probable that a series of infarctions has occurred there. The necrotic remnants alternate with vital tissue and with a very marked hypervascularisation which is to be found within the same area of collapse; it appears likely that the layers of new bone which lead to the sclerotic aspect of this part of the head have been laid down in response to scattered necrosis. We have seen that the cysts are aggregations of fibrous tissue, and the process of new bone formation is held at bay in their walls. A possible cause of this arrest of revitalisation is as follows. The cysts lying deep to the eburnated bone and unprotected by cartilage are within the area of transmission of greatest pressure, and,
Slab radiograph showing a large cyst beneath the pressure area of an osteoarthritic femoral head. The cyst is pyriform and communicates with the joint cavity by a small stoma. The plane of section, sagittal, is indicated in the small diagram.

The contents of the cyst. The photomicrographs show three different types of tissue from a cyst within an osteoarthritic femoral head. In Figure 48 there are abundant connective tissue fibres. In Figure 49 the framework is made looser by the presence of intercellular fluid, which in Figure 50 has accumulated and formed small cavities. (H. and E. x 100.)
because of their communication with the joint cavity, a pressure disturbance will probably enter this area of the head at every step and movement; this may be the factor defeating vascular penetration. That the cysts within the femoral head and the acetabulum can heal if the mechanical forces are redistributed is sometimes evidenced in radiographs showing the progress of osteoarthritic hips after a Girdlestone pseudarthrosis (Figs. 54 and 55), a McMurray osteotomy or an arthrodesis,
Extrusion of the femoral head—The flattened surface of the upper segment of the head of the femur as much as its smoothness favours the displacement of the head laterally, and this increases the width of the joint space infero-medially. The consequent lack of contact acts as a further stimulus to the production of new osteophytes in the non-pressure area of

![Image](image_url)

**Fig. 53**
The enclosed area of Figure 38 seen at higher magnification. *a.* = an osteoclast lies in a marrow space next to the necrotic bone, *b.*

(H. anE. d. × 300.)

![Image](image_url)

**Fig. 54**
Osteoarthritic hip (Fig. 54) treated by excision of the femoral head and neck (Fig. 55). Note that the large cyst present in the ilium before operation is subsequently replaced by bone.

Fig. 55

From the point of view of the joint stresses the outward displacement of the head further accelerates the process of disintegration. At this stage the restricted pressure zone of the head and its acetabular counterpart are situated marginally, as in congenital subluxation of the hip, but the two contact areas are flat. Under these circumstances the load is distributed.
over a further reduced area, and as joint movement becomes increasingly lost the load is carried by the same trabeculae for ever-increasing periods of time. The crushing effect of this overload is felt by even deeper bone trabeculae, with consequent extension of the events and tissue changes already referred to (Fig. 56).

There is a reconstructive or healing process present within the osteoarthritic femoral head. We have already referred to osteoclastic removal of necrotic bone and to osteogenic activities within the pressure segment, and at the many places where the fibrous marrow comes to the surface at the pressure area it differentiates into a fibrocartilage which is usually degenerate but at times is almost hyaline in nature. Most commonly these sproutings of cartilage remain discrete (Fig. 39), but, especially in patients whose hip joints have been placed at rest or when mechanical conditions have been changed, these cartilaginous outgrowths become confluent and may resurface the previously naked and eburnated bone. In such specimens the pressure segment usually contains only a few small cysts and little evidence of marrow oedema, fibrosis and bone sclerosis.

**SUMMARY**

Osteoarthritis, as seen in the hip, is a disease which eventually embraces all the tissues of the joint but begins as a reaction of the juxta-chondral blood vessels to a degeneration of the articular cartilage; this reaction results in a hyperaemia of the bone. To our surprise we found that daily use preserves rather than "wears out" articular cartilage; indeed inadequate use is the commonest cause of cartilage degeneration and ensuing vascular invasion. To this factor are added the effects of excessive pressure in the many patients who require surgical treatment for advanced osteoarthritis of a hip where some anatomical incongruity. This etiology based on cartilage suffering does not exclude, but indeed explains, the osteoarthritis implanted on joints of a normal shape which have been previously affected by acute or chronic inflammation or by hormonal dysfunction, such as acromegalic osteoarthritis. The stimulus to vessel growth and invasion is the same in all these cases—namely cartilage damage. Once the vessels have entered the cartilage the bone and marrow of the osteophyte are inevitably laid down. What is so damaging in osteoarthritis seems to be not the degeneration of the cartilage but the vigorous and persistent attempt at repair, an attempt which aggravates the already disordered function of the joint not only by osteophyte formation but by the hypervascularity which weakens the structure of the bone beyond the point where it can carry its increased load. The collapse that follows provokes further reparative efforts with the same deplorable results. The osteoarthritic process thus appears to be an attempt to transform a decaying joint into a youthful one and for this, as in the miraculous rejuvenation depicted in Goethe's *Faust*, a high price must ultimately be paid.

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