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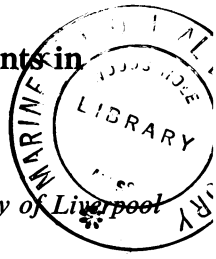

Cartilage fibrillation in shoulder and hip joints in Liverpool necropsies

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INTRODUCTION

The incidence and severity of articular cartilage fibrillation in necropsy subjects is known to increase with advancing age (Heine, 1926). This 'age-related fibrillation' often appears to have been asymptomatic during life, and requires separate consideration from the cartilage changes seen in patients with clinical evidence of osteoarthrosis. Both types of lesion show the splitting and disintegration characteristic of cartilage fibrillation, and can in their later stages be accompanied by a smooth-surfaced destructive thinning of cartilage, with abrasive wear of adjacent exposed bone (Meachim, 1972*a*). In spite of this histological similarity, they often differ in cause. Thus it would seem that osteoarthrosis of the hip is usually due to a separate causative factor, acting independently of ageing (Byers, Contepomi & Farkas, 1970). Similarly, patello-femoral osteoarthrosis is often due to a separate factor superimposed on the effects of ageing, although in some elderly subjects it may result solely from ageing (Meachim & Emery, 1974).

Quantitative and qualitative studies have recently been made of age-related cartilage fibrillation at the patello-femoral joint in a random series of necropsies in the city of Liverpool (Meachim, 1972*b*; Meachim & Emery, 1974). In the present study these observations have been extended by examining at necropsy a random series of shoulder and hip joints from the same population. The observations were again carried out by macroscopic inspection and light microscopy of cartilage surfaces which had first been painted with India ink (Bullough & Goodfellow, 1968; Meachim, 1972*b*), since these techniques make minor alterations of the surface more readily apparent, allow recognition of a number of different morphological types of cartilage change, and readily demonstrate the topographical distribution of these changes.

Quantitative study of age-related cartilage fibrillation (Meachin & Emery, 1974) has suggested that its extent and severity may differ between men and women, and perhaps also between left and right joints. The present observations were therefore confined to left shoulders and hips; representative material was examined from both sexes.

MATERIALS AND METHODS

The articular surfaces were studied in 37 left shoulder joints and 32 left hip joints from 62 white European subjects (34 male; 28 female) whose ages ranged from 12 to 94 years. The specimens were obtained from a random series of necropsies

in the city of Liverpool. Persons dying from accidents and persons dying outside hospital suddenly or unexpectedly from natural causes were included as far as practicable. Joints with evidence of rheumatoid or other inflammatory disease, previous surgery, old injury, or injury from a recent accident were excluded. Representative joints were studied from each age-decade for both sexes; 9 hips and 11 shoulders were deliberately included from persons, mainly women, in the ninth or tenth age-decade.

India ink preparations of the articular surfaces were made, and examined macroscopically and by light microscopy of the cartilage surface *en face* using the methods described previously (Meachim, 1972*b*). For some specimens vertically cut histological sections were also examined. The distribution of the various types of cartilage change thus demonstrated was then mapped on full-scale drawings separately prepared for each specimen. Measurements were transferred from the specimens to the drawings by using a graduated ureteric catheter as a flexible ruler. To allow for their curvatures the articular surfaces of the humeral and femoral heads and acetabular sockets were depicted as four or three segments separated by gaps meeting at the centre since this method gave equal representation both of radial and of circumferential lengths over the surfaces. The relatively slight curvature of the glenoid surface allowed it to be represented without separation into segments.

RESULTS

Surface morphology

The appearance of the articular surface varied from specimen to specimen, even amongst subjects of similar age. There was also variation from site to site on the same specimen. The following types of surface appearance were encountered in the India ink preparations (Figs. 1–6).

(1) Sites at which the cartilage surface was intact (Figs. 1, 4).

(2) 'Irregular' and 'parallel linear' patterns of 'minimal fibrillation', attributable to a very superficial splitting of the cartilage which did not extend deeply into the superficial layer (zone 1) of the tissue (Meachim, 1972*b*). In the shoulder and hip joints this minimal change was usually of the 'irregular' pattern (Fig. 6); a macroscopic 'parallel linear' pattern was less common and, when present, occupied shorter lengths of the surface than at the patello-femoral articulation.

(3) 'Ravines' (Figs. 3, 5) formed by vertical splitting. These usually extended more deeply into the tissue than the very superficial markings of minimal fibrillation. At a site of ravine formation the individual ravines were seen as branched or non-branched curvilinear markings or were joined together to give an incomplete or complete mosaic. Sites of ravine formation were usually contiguous with or close to a site of minimal or overt fibrillation (Fig. 3); sometimes they occurred independently. Within the ravined area itself, the intervening plaques of cartilage between the ravines were either intact or showed minimal fibrillation.

(4) Overt fibrillation (Figs. 1–3) due to frank splitting of the cartilage extending vertically down into its superficial (zone 1), intermediate (zone 2), or deep (zone 3) layer. Sometimes cartilage showed a smooth-surfaced destructive thinning without the frank splitting usually seen in overt fibrillation; such sites were uncommon in the

present necropsy series of shoulders and hip joints, and were not recorded separately from overt fibrillation of the usual type.

(5) Occasionally the old cartilage surface showed a localized rounded defect of varying depth without bone exposure in its base.

(6) A few of the specimens included sites where the full thickness of the original cartilage had been lost. Such sites usually presented a surface of exposed bone, often interspersed with plugs and plaques of new non-osseous tissue. Very occasionally a non-peripheral gap in the old cartilage was completely occupied by new fibrocartilaginous or fibrous tissue, and no bone exposure was then apparent.

(7) Peripheral sites showing fibrous covering over old cartilage, or over old bone on which there had been full-thickness cartilage loss, were sometimes seen without any accompanying osteophytosis or ossification of the old cartilage.

(8) Some of the specimens showed new bone formation at the periphery of the cartilage sheet. The detailed pattern of this osteophytosis varied, as previously observed in a study of surgical excision specimens of osteoarthritic femoral heads (Meachim, 1972*a*). Thus the osteogenesis occurred either inside or outside the perimeter of the old cartilage surface or at both positions. When inside the perimeter it was seen as ossification within surviving old cartilage, or as ossification within new non-osseous tissue which had completely replaced the old cartilage, or as both phenomena. In the case of the hip joints minor amounts of peripheral ossification were usually not recorded on the drawings.

Topographic relations of minimal and overt fibrillation

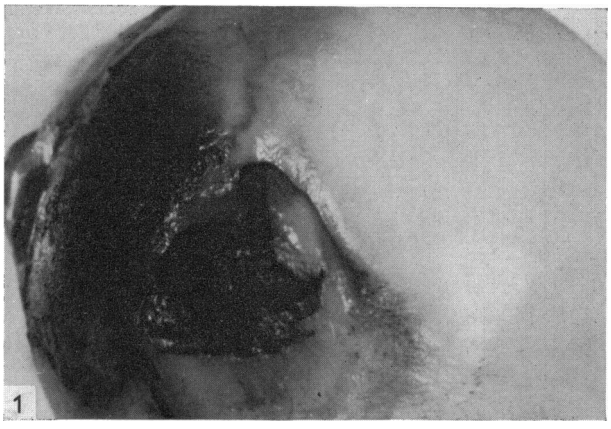
Minimal and overt fibrillation were usually, but not invariably, both present on the same specimen. Topographic distribution was first analysed separately for each. Such an analysis showed that they both had a generally similar topographical pattern when the series of humeral heads was considered as a whole. This also held true for the glenoid specimens, the femoral heads, and, to a lesser degree, the acetabular surfaces. In the account which follows they will therefore both be described together as 'fibrillation', and this term will imply either minimal or overt change unless specifically qualified. On many specimens more than one area of fibrillation was apparent. Each individual area showed minimal fibrillation only or overt fibrillation only, or a combination of minimal and overt change.

Boundaries between overt fibrillation and intact cartilage usually showed either an abrupt transition or a variable width of intervening minimal change. Occasionally there was instead an intervening area of ravine formation.

No qualitative difference in the topographical distribution of the lesions was apparent between men and women.

Humeral heads

The position of cartilage lesions on the humeral heads was mapped on drawings constructed as shown in Fig. 8. The point of intersection of the long and short axes of the articular surface was marked on the central region of the specimen. The surface was then divided into four segments by lines drawn through this central point at approximately 45° to the long axis. The junction between the peripheries of the superior and anterior segments lay close to the track of the long head of biceps at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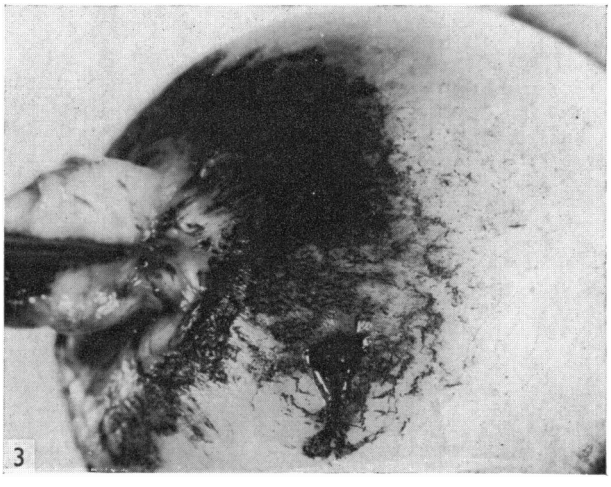
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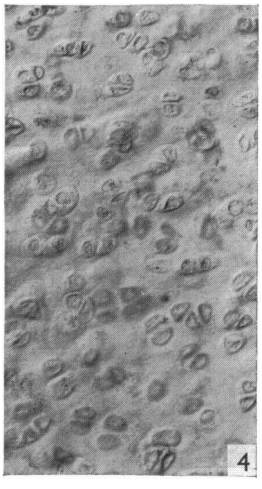
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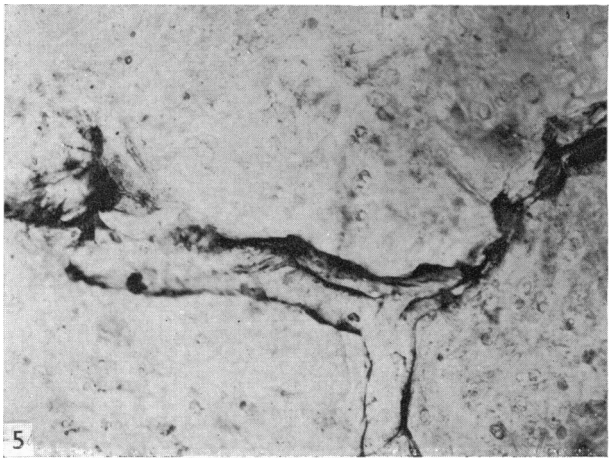
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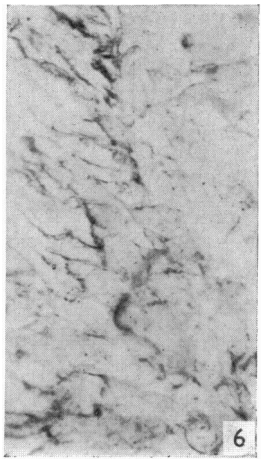
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its exit between the greater and lesser tuberosities; some of the specimens showed a notch at this junction. The distribution of cartilage lesions (Fig. 7) was studied on the central region of the articular surface, its periphery, and the mid-zone between the periphery and the centre.

Fibrillation initially involved the periphery of the humeral articular surface, on which it could occur as early as the second decade of life (Figs. 9, 11). In contrast, involvement of the central region of the humeral head was exceptional in subjects aged under 50 years, although a few of the younger subjects showed isolated foci of fibrillation on sites in the mid-zone (Fig. 14) between the centre and periphery. Peripheral foci (Figs. 9–13) occurred at the edge of the cartilage surface or a short distance inside the edge. Usually two, three, four or more separate peripheral foci were present. Peripheral fibrillation often spared a small part of the cartilage edge on the posterior side of the superior pole; otherwise any part of the periphery was susceptible, although peripheral involvement anteriorly and posteriorly tended to be more extensive than at the inferior pole. One site commonly involved was at or near the junction between the superior and anterior segments (Figs. 11, 12, 14, 19), close to the track of the long head of biceps. Peripheral fibrillation increased in its extent by centripetal and by circumferential spread. This spread appeared to be mainly by enlargement of existing areas, but sometimes seemed to have been also by the development of new foci.

Fibrillation of the central region of the humeral surface (Figs. 17, 18) was exceptional before the age of 50. In some specimens a central area of minimal fibrillation

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Fig. 1. Part of an India ink preparation of the left femoral head from a woman aged 28 years. The infero-medial segment is at the left and upper left of the picture; nearly all of this segment shows confluent blackening indicative of overt fibrillation. The fovea is just to the left of the centre and lower part of the picture; the blackening in the fovea is due simply to its content of fibrous tissue. The posterior and part of the superolateral segments are at the right and upper right of the picture; their cartilage surface is mostly intact, and shows no ink markings except on an area of minimal fibrillation at the parafoveal periphery of the superolateral segment.

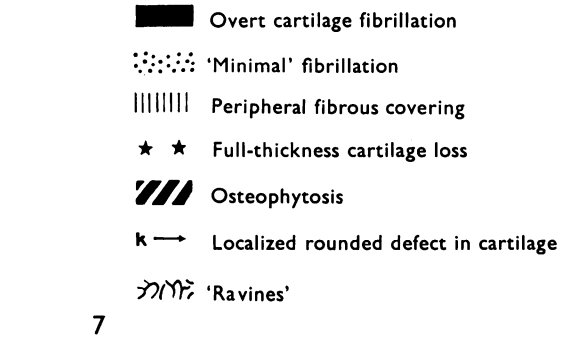
Fig. 2. Part of an India ink preparation of the left glenoid articular surface from a man aged 67 years. At the periphery there are segments of confluent blackening from overt fibrillation. The rest of the surface is partly intact and partly shows dark ink markings from minimal fibrillation and from ravine formation. This shoulder joint is also illustrated as Fig. 16.

Fig. 3. Part of an India ink preparation of the left femoral head from a man aged 60 years. The fovea is at the left of the picture. There is parafoveal blackening from overt fibrillation of the articular cartilage; at the edge of the overt fibrillation there are dark ink markings from minimal fibrillation (upper right) and from ravine formation (below centre). In many femoral head specimens no ravine formation was seen.

Fig. 4. Tangential slice of an intact cartilage surface viewed at a magnification of × 150 in transmitted light. There are no ink markings on the cartilage surface. The background texture represents the underlying cells. India ink preparation. Mid-zone of superolateral segment of the left femoral head from a woman aged 84 years.

Fig. 5. A ravine due to vertical splitting which extends more deeply into the tissue than the very superficial splitting of minimal fibrillation. Humeral head; woman aged 75 years. Ravine formation was observed in only a minority of the humeral head specimens. Tangential surface slice. India ink preparation. Transmitted light. × 150.

Fig. 6. Tangential surface slice from an India ink preparation, viewed in transmitted light, showing one example of irregular pattern of minimal fibrillation. Acetabular surface; man aged 84 years. × 150.



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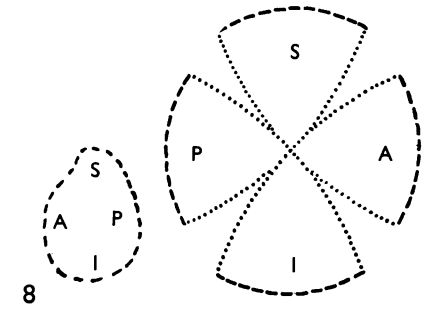

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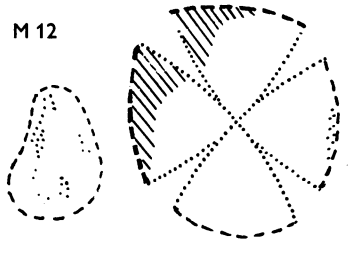

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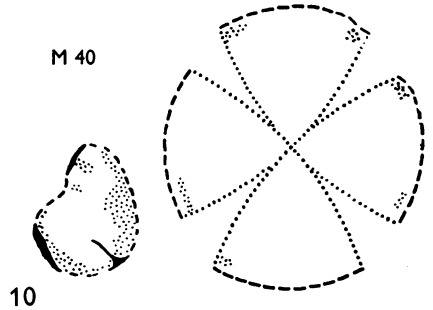

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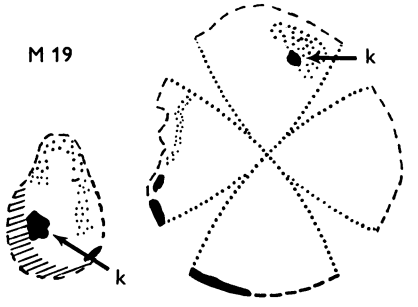

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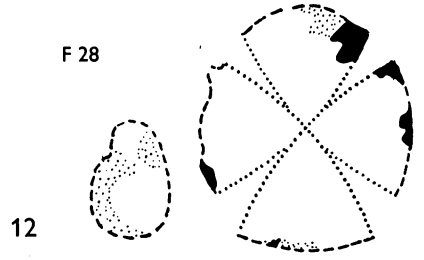

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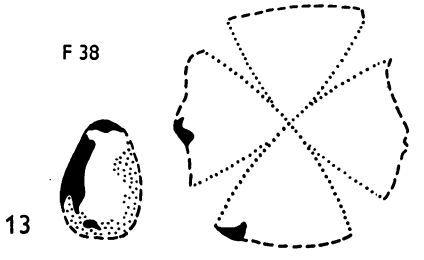

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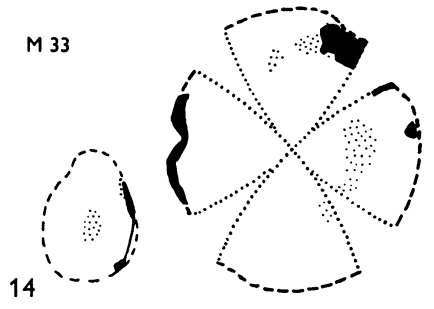
Fig. 7. Key to the symbols used to depict the cartilage lesions in the drawings of the shoulder and hip joints.

Fig. 8. Orientation of the drawings of the left shoulder joints, showing the position of the anterior (A), posterior (P), superior (S) and inferior (I) aspects.

Figs. 9–29. Representative selection from the drawings of the left shoulder joints. Symbols as in Fig. 7 and orientation as in Fig. 8. The glenoid is shown to the left of the humeral head. The letter and numbers above each glenoid give the sex (M, male; F, female) and age in years of the subject. Shown at one-half actual size.



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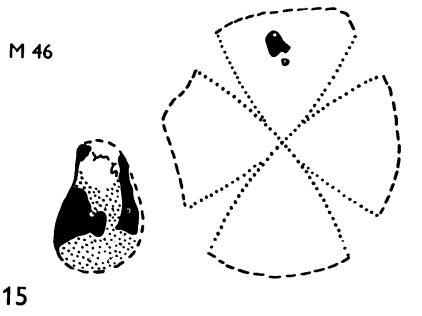

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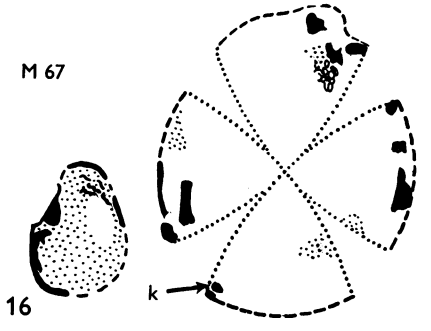

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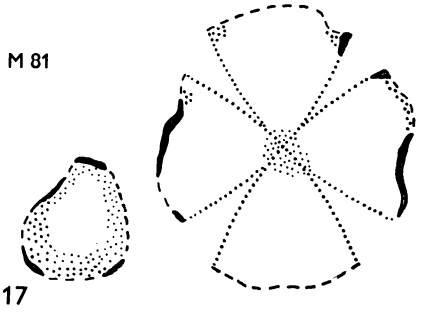

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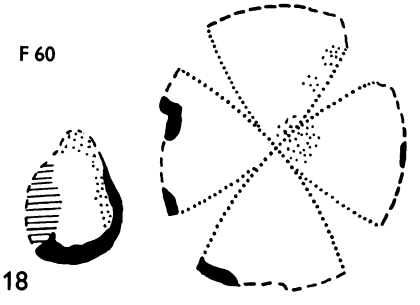

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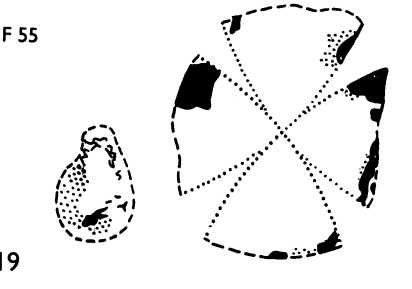

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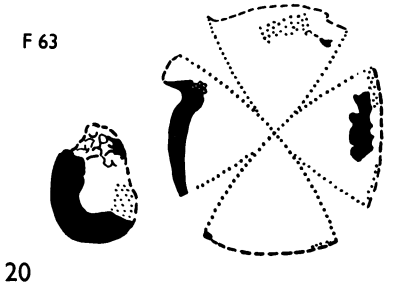

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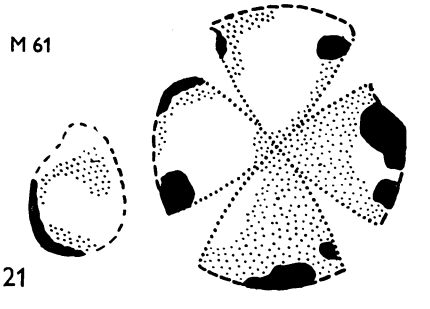

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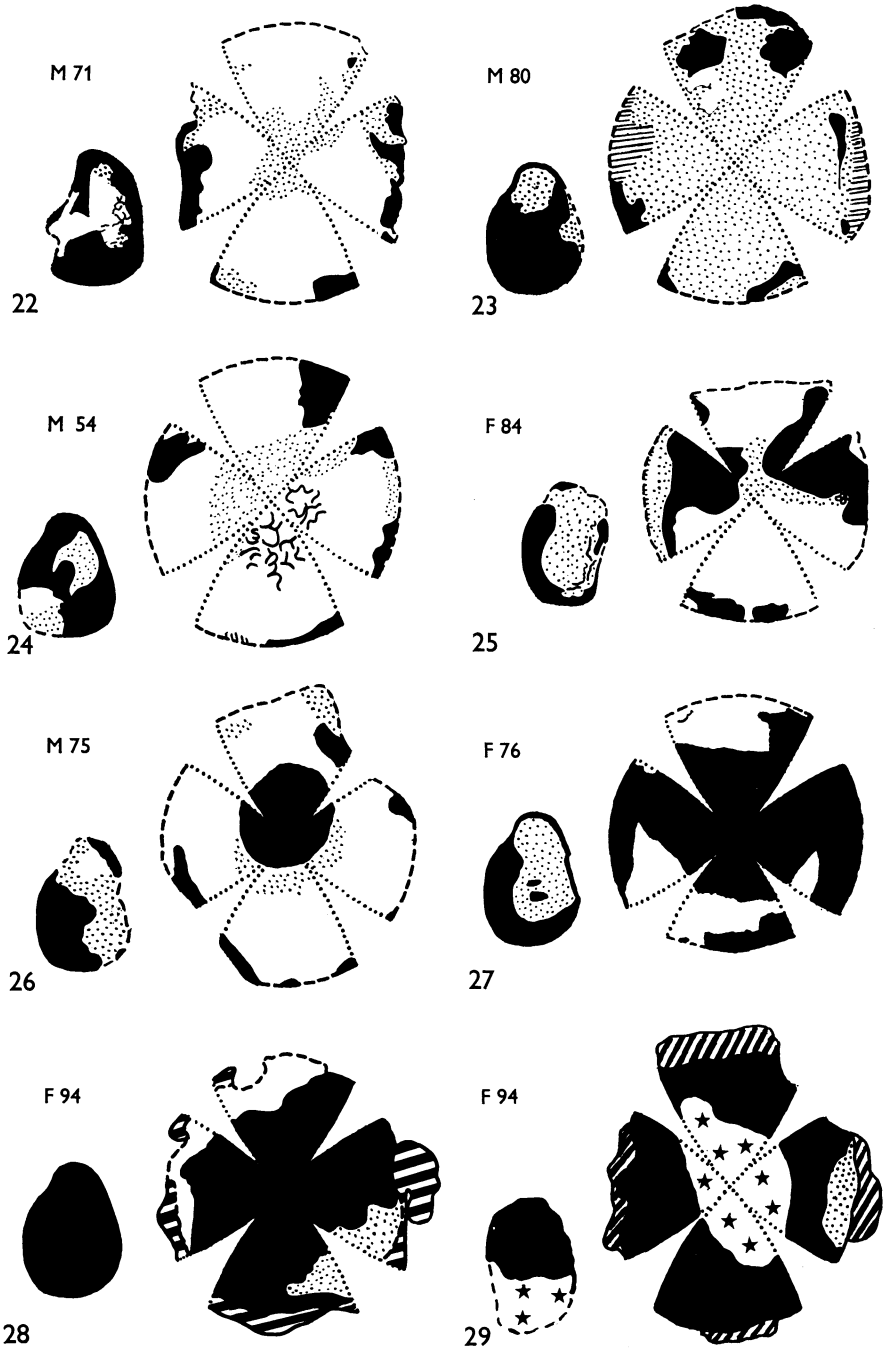
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(Fig. 17) or of overt fibrillation rimmed by minimal change (Fig. 26) was separated from the peripheral fibrillation by an intervening band of intact cartilage. This observation indicated that in at least some instances the central involvement had developed independently and not by centripetal spread from the periphery. In other specimens the central fibrillation was isolated except for a single bridge of confluence at its anterosuperior perimeter (Figs. 22, 24). In others (Fig. 25) an anterosuperior and a posterior confluence were both apparent.

In elderly subjects there was considerable variation from individual to individual, but in some of them further involvement of central and mid-zonal regions (Fig. 21) had led to a state in which most or all of the articular surface was covered by confluent fibrillation (Fig. 23).

An area of ravine formation (Fig. 24) was noted on some, but by no means all, of the humeral heads from subjects aged over 50. Such areas occurred on the central region, on the mid-zone, or at the inner aspect of the peripheral region. A few of the adult specimens had one or more peripheral sites covered by a layer of fibrous tissue (Fig. 23). Fibrous covering of part of the periphery was also seen on a specimen from a boy aged 12 years (Fig. 9). Very occasionally a localized rounded defect (Figs. 11, 16) was noted in the humeral cartilage.

Glenoid cavities

On the glenoid articular surfaces a central region of thinner cartilage and a broad peripheral region of thicker cartilage were recognized. At its outer edge, the peripheral region of articular cartilage merged with the fibrocartilage of the glenoid labrum. At its inner edge, macroscopic inspection *en face* often showed a well-defined boundary line at the junction of the peripheral region with the antero-inferior aspect of the central region, and histological examination showed a minor change in the texture of the superficial layer of the cartilage at this junction. These features often gave the superficial tissue of the antero-inferior part (Fig. 8) of the peripheral region a meniscoid appearance, although its base was firmly adherent to the underlying tissue and its inner edge was not free except where there was splitting attributable to fibrillation.

Study of the distribution of the cartilage lesions (Fig. 7) showed that fibrillation of the glenoid surface first became apparent on the peripheral region (Figs. 9–11) and along the line (Fig. 9) marking the boundary between the antero-inferior part of the peripheral region and the central cartilage; it could occur at these sites as early as the second decade of age (Figs. 9, 11). Any part of the peripheral region could be affected. There were usually two or three independent areas of peripheral fibrillation, one of which often occupied a considerable length of the glenoid circumference (Fig. 12). Peripheral involvement increased in extent by centripetal and circumferential spread.

Fibrillation of the central region of the glenoid surface usually first became apparent between the fourth and the seventh decades of age, though there was considerable variation (Figs. 14, 17). In most instances it was confluent with peripheral fibrillation, a finding consistent with an origin by centripetal spread. Occasionally it was seen as an isolated area completely surrounded by intact cartilage (Fig. 14), indicating origin as an independent central focus in such specimens.

In the majority of subjects from the age of 70 or over, most or all of the glenoid surface had become fibrillated (Figs. 23–28).

An area of ravine formation was present on a minority of the glenoid surfaces from the fourth decade onwards (Figs. 14–16, 19, 20, 22, 25). Such areas occurred on the superior or posterior and inferior parts of the surface, but were not seen anteriorly. A few of the specimens showed an area of peripheral fibrous covering on their anterior or antero-inferior aspect (Figs. 11, 18). Very occasionally a localized rounded defect (Fig. 11) was noted in the glenoid cartilage.

Bone exposure and osteophytosis in the shoulder joints

None of the shoulder joints from subjects younger than 80 years of age showed vertical progression of fibrillation to give an area of full-thickness cartilage loss with exposed bone, nor did any of them show osteophytosis at the humeral or glenoid articular margins.

In 11 left shoulders (from 2 men and 9 women) in the age-range 80–94 years the findings were as follows:

(1) One joint (Fig. 29) showed an area of bone exposure on the humeral head and opposing glenoid socket accompanied by osteophytosis at the humeral, but not at the glenoidal, margin.

(2) Two joints showed osteophytosis at both the humeral and glenoidal margins.

In two, osteophytosis was seen on the humeral head only (Fig. 28). On the humeral heads osteophytosis was often due mainly to ossification within the perimeter of the old cartilage, without any major element of bony outgrowth beyond the original articular margins.

Femoral heads

The distribution of cartilage lesions (Fig. 7) on the femoral heads was mapped on drawings constructed as shown in Fig. 30. The position of the mid-coronal plane of the specimen was first determined. Lines intersecting this plane at an angle of approximately 45° were then drawn through the fovea so as to divide the articular surface into four segments, each of which had an outer circumference of approximately equal length (Fig. 30). For each segment a parafoveal inner peripheral region, a peripheral region at the outer perimeter, and a mid-zone between the two peripheries were arbitarily recognized. Since the fovea is eccentric, the mid-coronal width of the inferomedial mid-zone was less than on the other segments (Fig. 30). The mid-coronal part of the mid-zone of the superolateral segment represents the zenith of the femoral head as seen in an anteroposterior radiograph.

On the femoral heads fibrillation first affected the following sites:

(1) The parafoveal inner peripheral region of the cartilage sheet, occurring on any segment.

(2) The peripheral region at the outer perimeter, again occurring on any segment.

(3) A localized region on the mid-zone, almost always inferomedially.

Taking the series as a whole, fibrillation developed in parallel at these three sites, although their extent of involvement relative one to another showed some variation according to the individual subject. The total amount of femoral head fibrillation also varied from individual to individual amongst persons of similar age.

Parafoveal fibrillation could occur as early as the second decade (Fig. 32). It was seen as one, or occasionally more than one, area extending for a variable distance around the parafoveal edge of the cartilage (Figs. 31–36). It became more extensive by circumferential spread and by radial enlargement toward the mid-zone. In the majority of subjects aged over 50 years a rim of fibrillation completely encircled the fovea (Figs. 37–39).

Peripheral fibrillation at the outer perimeter of the cartilage could also occur as early as the second decade (Fig. 32). It was seen as from one to four separate areas, each of which involved a variable distance around the perimeter (Figs. 31–35). It became more extensive by circumferential spread and by radial enlargement toward the mid-zone. In some of the subjects aged over 50 years a rim of fibrillation completely encircled the outer perimeter (Figs. 37, 38), but in others from one to four separate areas were still apparent (Figs. 39, 41).

Fibrillation on a region of the mid-zone was usually confluent with peripheral involvement, but was occasionally seen as an independent focus. It could occur as early as the second or third age-decade (Figs. 32, 33). The region first affected was almost always on the mid-zone of the inferomedial segment (Figs. 32–35). Half or more of the total area of this segment showed minimal or overt fibrillation in some of the subjects aged less than 50 years, and in nearly all the subjects older than this.

In the majority of subjects aged over 50 years, mid-zonal minimal or overt fibrillation was seen on part of the anterior or posterior or both segments (Figs. 38, 40, 42, 43). Mid-zonal fibrillation on the superolateral segment also occurred in a few of the older subjects, but was exceptional unless one or both of the adjacent segments was involved. Usually mid-zonal fibrillation on other segments was contiguous with the inferomedial mid-zonal involvement, and thus it often seemed to have spread by circumferential extension from the inferomedial segment on to adjacent areas of one or both of the neighbouring segments (Figs. 38, 40, 42, 43), but in some instances this explanation was not tenable. The superolateral mid-zone was usually the last region to be affected to a major extent, and thus cartilage on that part of this region which formed the zenith of the femoral head often remained intact until old age (Figs. 4, 42–44).

One or more areas of ravine formation (Fig. 38) were seen on a few of the femoral heads from subjects aged over 50 years; they occurred on the mid-zone of the anterior, posterior or superior segments or at the inner aspect of the parafoveal region. Peripheral fibrous covering (Fig. 31) was noted only occasionally. No localized rounded defects were observed in the femoral head cartilage.

Acetabular surfaces

The position of cartilage lesions (Fig. 7) on the acetabular sockets was mapped on drawings constructed as shown in Fig. 30. The articular surface was divided into a broad superoposterior segment, forming the curve of the acetabular horseshoe, and an anterosuperior and inferoposterior segment separated by the non-articular territory in the floor of the acetabular fossa. For each segment an inner periphery, an outer periphery and a mid-zone between them were arbitarily recognized. Near the outer periphery the hyaline cartilage fused with a band, usually 1–6 mm in width, of bone-based articular fibrocartilage, which was in turn contiguous with

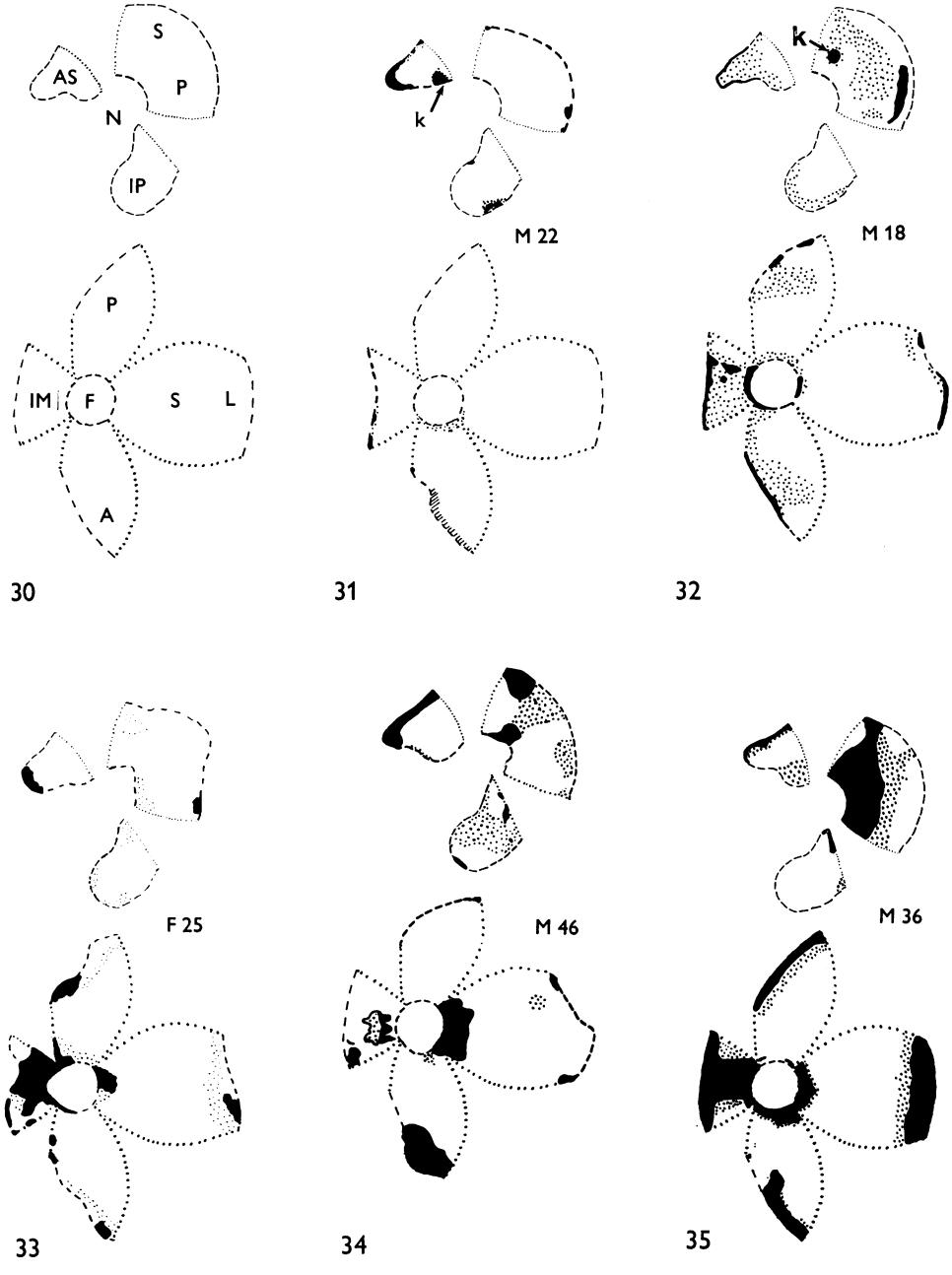
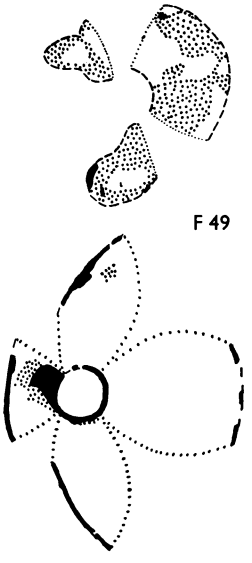


Fig. 30. Orientation of the drawings of the left hip joints, showing the position of the fovea (F) and the inferomedial (IM), anterior (A), posterior (P) and superolateral segments (S-L) of the femoral head and the position of the anterosuperior (AS), superoposterior (S-P) and inferoposterior (IP) segments of the acetabular articular surface in relation to the acetabular notch (N).

Figs. 31–47. Representative selection of the drawings of the left hip joints. Symbols as in Fig. 7 and orientation as in Fig. 30. For each joint the letter and number give the sex (M, male; F, female) and age in years of the subject. Shown at one-third actual size.



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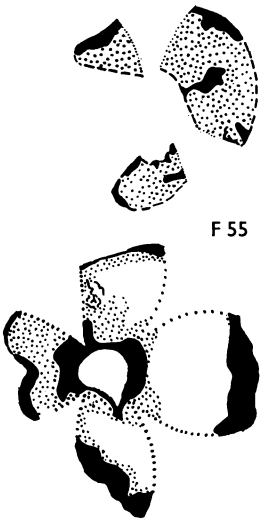

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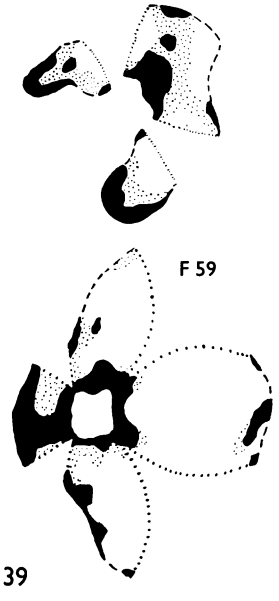

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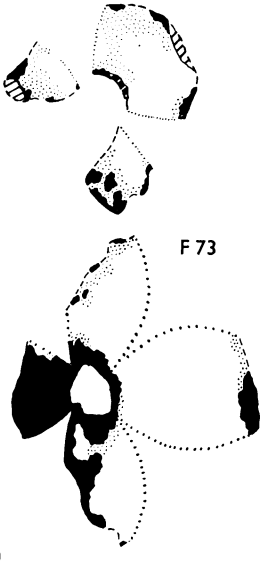

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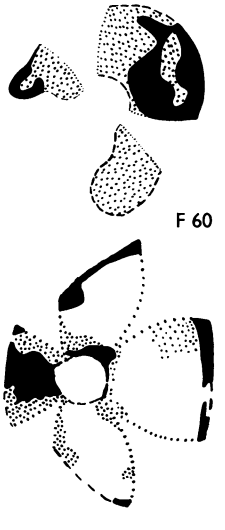

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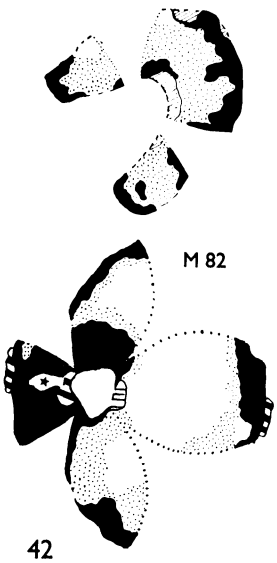

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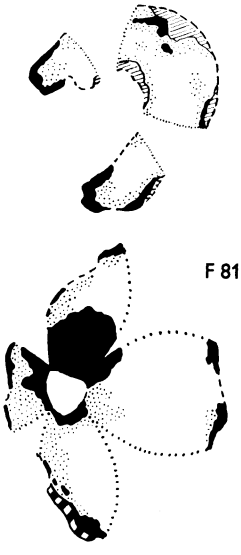
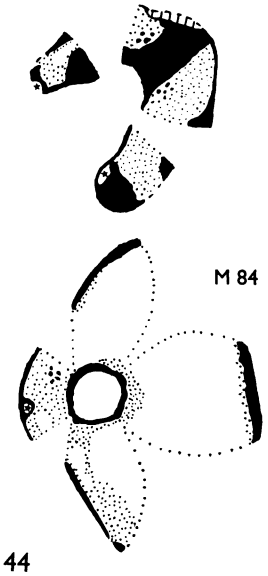


43



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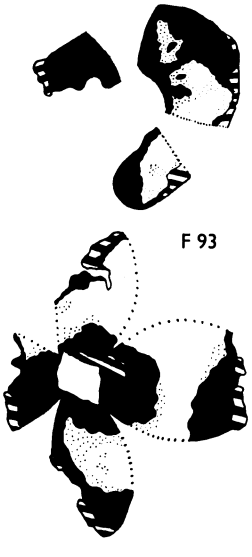

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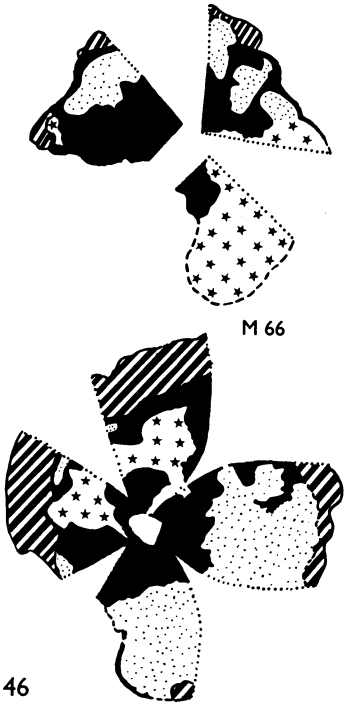

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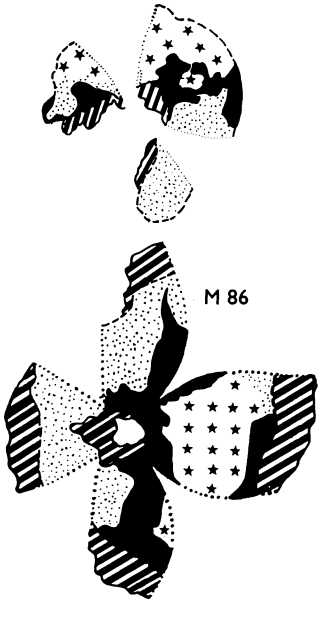

47

the fibrous labrum. A similar narrow band of articular fibrocartilage was sometimes present at the inner periphery. These narrow bands of bone-based fibrocartilage were considered to be part of the acetabular articular surface, and were therefore included as such in the drawings.

On the acetabular surface mid-zonal and peripheral fibrillation both occurred at an early age (Fig. 32), although in some of the younger subjects peripheral involvement only was seen (Fig. 31). The total extent of fibrillation showed variation from individual to individual amongst persons of the same age.

Peripheral involvement by minimal or overt fibrillation was observed at any site around the inner and outer edges, fibrocartilaginous or hyaline, of the acetabular articular surface (Figs 31–34). From two to six independent areas were seen (Figs. 31, 33), or a semi-complete or complete rim of confluent fibrillation was apparent (Fig. 37). Involvement became more extensive by circumferential and by radial enlargement.

Mid-zonal fibrillation was usually confluent with peripheral areas (Figs. 34–37), but an independent mid-zonal focus was also observed in some instances. Minimal fibrillation occurred on any part of the acetabular mid-zone. In most, but not all, subjects it tended to affect particularly the superoposterior segment and sites adjacent to this on the other two segments. In the case of mid-zonal overt fibrillation, the tendency to initial localization on this region of the acetabulum was more definite than for minimal change (Fig. 35).

An area of ravine formation was seen very occasionally in the older subjects. Some of the younger subjects showed a localized rounded defect in the acetabular cartilage (Figs. 31, 32). Peripheral fibrous covering was sometimes seen, and tended to occur especially at the outer and inner aspects of the superoposterior segment (Figs. 42–44); the histological distinction between this phenomenon and minor degrees of osteophytosis was sometimes debatable.

Bone exposure and osteophytosis in the hip joints

In the 23 subjects younger than 80 years, 1 left hip joint, from a man aged 66 years (Fig. 46), showed a large area of bone exposure on the posterior and inferomedial segments of the femoral head and on the corresponding part of the acetabulum, with femoral and acetabular osteophytosis. A further 2 of these 23 joints showed acetabular osteophytosis, without bone exposure on either surface.

In 9 left hip joints from subjects (4 men and 5 women) in the age-range 80–93 years, the findings were as follows:

(1) One joint (Fig. 47) showed a large area of bone exposure on the superolateral segment of the femoral head and on the corresponding part of the acetabulum, with femoral and acetabular osteophytosis.

(2) One joint (Fig. 44) showed very small areas of bone exposure on the inferomedial segment of the femoral head and at the tips of the anterosuperior and inferoposterior segments of the acetabular surface, without either femoral or acetabular osteophytosis.

(3) One joint showed both femoral and acetabular osteophytosis, without bone exposure (Fig. 45).

(4) Four joints showed either femoral or acetabular osteophytosis only (Fig. 40);

in 1 of these 3 subjects a small area of full-thickness old cartilage loss on the infero-medial segment was completely filled with non-osseous repair tissue.

DISCUSSION

The present observations on shoulder and hip joints, when taken in conjunction with the results of other studies of knee joints (Bennett, Waine & Bauer, 1942; Meachim & Emery, 1974), elbow joints (Goodfellow & Bullough, 1967) and lumbar apophyseal joints (Meachim, unpublished), strongly suggest that, at least in white Europeans, regions of fibrillation develop in all synovial joints and that their presence within an adult joint can be considered normal. Such a joint, will, of course, usually also have regions in which the cartilage surface is still intact. The present and previous studies also show that fibrillation is often apparent as early as the second decade of life. The phenomenon occurs both in weight-bearing and in non-weight-bearing joints. With advancing age, but subject to variation from individual to individual in its rate of development, overt fibrillation gradually involves an increasing proportion of the total surface area of an articulation (Meachim & Emery, 1974).

The above conclusions indicate that concepts of the 'wear-resistance' and 'longevity' of articular cartilage (Radin & Paul, 1972) are largely a matter of semantics. The material has in fact a poor resistance to the onset of wear at certain characteristic sites within a joint, but normally has a fairly high resistance to the effects of this wear when the whole joint is considered in terms of the tangential spread of the lesions across an articular surface and of the rate of vertical 'progression' (Byers *et al.* 1970) of destructive cartilage changes to give bone exposure similar to that of osteoarthrosis. Thus the rate of tangential spread of overt fibrillation is slow in relation to the expected total life-span of an individual (Meachim & Emery, 1974); moreover, certain regions, notably the zenith of the femoral head, often remain intact until a late stage. Similarly, the rate of vertical progression of ageing changes to give bone exposure is relatively slow, but less so in certain joints than in others. Bone exposure from ageing at the hip (Byers *et al.* 1970) and the shoulder is uncommon even in the elderly. It can, in contrast, occur in the radio-humeral component of the elbow joint (Goodfellow & Bullough, 1967) and in the patello-femoral articulation (Meachim & Emery, 1974), although it is exceptional at these sites before the sixth decade of life. The question of the part, if any, played by 'ageing' in the causation of osteoarthrosis may therefore require consideration separately for each different anatomical site, and has been discussed elsewhere (Meachim & Freeman, 1973).

The destructive cartilage lesions of ageing and of osteoarthrosis can be accompanied by attempts at remodelling and repair. In the case of the margin of adult articular surfaces there is histological evidence (Meachim, 1972*a*) that peripheral fibrous covering and osteophytosis are closely related phenomena which may both be features of fundamentally the same remodelling process. In the case of more central sites on the surface it is relevant that a segment of full-thickness loss of old cartilage does not invariably show bone exposure, but can instead occasionally acquire a covering of new fibrous or fibrocartilaginous tissue.

In the present series of hip joints minor degrees of osteophytosis were usually not recorded. For this reason the incidence of hip osteophytes in the present series is less than the total incidence given by Byers *et al.* (1970), but may be comparable to the incidence of their 'Stage 2' osteophytes.

The topography of age-related cartilage fibrillation has previously been studied in the elbow joint by Goodfellow & Bullough (1967), in the hip by Byers *et al.* (1970), in the knee by Øwre (1936) and Bennett *et al.* (1942), and in various joints by Collins (1949). The present and other recent studies (Meachim, 1972*b*; Emery & Meachim, 1973) show that cartilage fibrillation typically first develops on the periphery of an articular surface and on certain other characteristic sites such as the mid-zone of the inferomedial segment of the femoral head, the mid-zone of the acetabular surface, the medial strip of the medial patellar facet, and near the presumptive boundary between patellar and tibial articulation territories on the femoral condyles. The reason for this susceptibility of cartilage to peripheral fibrillation is not known. The phenomenon occurs regardless of the shape of the cartilage edge in vertical section (Emery & Meachim, 1973), regardless of whether the edge is hyaline or shows a narrow bone-based fibrocartilaginous band, as at the outer periphery of the acetabulum, and regardless of whether or not the edge is fused with a fibrous labrum. The effect may perhaps be accentuated where a cartilage sheet moves to and fro under the interrupted edge of the opposing sheet (Freeman, 1972), but the susceptibility of the glenoid periphery, where such movement under the humeral edge seems unlikely, suggests that a more comprehensive explanation is required. The present study also shows that susceptibility to peripheral fibrillation is found not only at the outer perimeter of cartilage sheets, but also at inner peripheries such as the parafoveal region of the femoral head and the edge of the acetabular surface adjacent to the floor of the fossa. The parafoveal and outer edges of femoral head cartilage are susceptible on any segment, and not just inferomedially where the head opposes the gap in the acetabular surface. In apparent contrast to these conclusions, the outer part of the upper tibial articular cartilage is relatively resistant to wear, but this atypical finding is attributable to a protective effect by the overlying menisci (Bullough *et al.* 1970).

A previous study of the patello-femoral joint using India ink preparations (Emery & Meachim, 1973) has shown that age-related cartilage wear is not a uniform process at this joint in terms of the morphology of the lesions, their topographic distribution, and the time-sequence of their vertical progression more deeply into the tissue. A morphological variety of lesions was again noted in the present study of hip and shoulder joints, and the main types seen were basically similar to those noted at the knee.

'Irregular minimal' and overt fibrillation show a generally similar pattern of topographical distribution at the hip and the shoulder. It is therefore suggested that one way in which overt fibrillation can develop is by the continued operation at the tissue level of fundamentally the same process as that responsible for 'irregular minimal' change. The time-sequence of this hypothetical relationship can be considered further in terms of a possible vertical progression of irregular minimal change by deeper splitting to give new foci of overt fibrillation, and in terms of a possible part played by adjacent irregular minimal change during the tangential spread of existing

foci of overt fibrillation on to adjacent areas of the surface. In the context of possible vertical progression, it does not necessarily follow that minimal fibrillation, having developed at a particular site, would then inevitably progress further to give overt change. Conversely, progression to overt change might in some other instances be so rapid that a preceding stage of minimal change would not be detectable. Concerning the tangential enlargement of existing foci of fibrillation, the present study shows that the edge of such an area is sometimes separated from nearby intact cartilage by a variable width of minimal change, suggesting that the phenomenon may have been expanding tangentially by gradual conversion of minimal into overt territory. However, it must be admitted that an area of overt fibrillation can instead abut directly on to intact cartilage, which would on this hypothesis imply either that its tangential spread had been temporarily halted, or that the hypothetical conversion process was so rapid that an interface of minimal change was not detectable.

At the hip and the shoulder, the 'parallel linear' macroscopic pattern of minimal fibrillation is less common, and tends to occupy shorter lengths of the articular surface, than at the patellofemoral joint. Ravine formation was seen in only a few of the hips and shoulders, and was by no means a constant feature of the ageing process. From the present study no conclusion was drawn concerning the relationship between the local pathogenesis of macroscopically parallel linear and ravined areas and that of overt fibrillation.

SUMMARY

A study has been made of the articular surfaces in 37 left shoulder and 32 left hip joints from a random series of necropsies on 62 white European subjects whose ages ranged from 12 to 94 years. By use of India ink preparations, the following cartilage lesions were recognized: minimal fibrillation; ravine formation; overt fibrillation; localized rounded defects in the cartilage; full-thickness loss of old cartilage, with bone exposure; peripheral fibrous covering of the articular surface; and osteophytosis. Observations were made on the topographical distribution of the cartilage changes and their patterns of development during normal ageing.

The findings, when taken in conjunction with previous studies on other joints, strongly suggest that regions of fibrillation normally develop in all synovial joints and that such regions are often apparent as early as the second decade of life Fibrillation first typically develops on the periphery of an articular surface and on certain other characteristic sites such as the mid-zone of the inferomedial segment of the femoral head and the mid-zone of the acetabular surface. With increasing age, but subject to variation from individual to individual in its rate of development, it spreads tangentially to involve an increasing proportion of the total area of an articular surface. This spread would seem to be due mainly to circumferential and radial enlargement of existing foci; new, topographically independent, foci are some times also initiated, as, for example, on the centre of some humeral heads. Tangential spread of overt fibrillation during ageing is slow in terms of expected total life-span; moreover, certain sites, notably the zenith of the femoral head, often remain intact until a late stage. Vertical progression of ageing changes to give bone exposure from full-thickness cartilage loss is uncommon at the left shoulder and hip, even in the

elderly; the findings contrast in this respect with those at the patellofemoral and radiohumeral articulations.

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REFERENCES

BENNETT, G. A., WAINE, H. & BAUER, W. (1942). *Changes in the Knee Joint at Various Ages with Particular Reference to the Nature and Development of Degenerative Joint Disease.* New York: Commonwealth Fund. London: Oxford University Press.

BULLOUGH, P. & GOODFELLOW, J. (1968). The significance of the fine structure of articular cartilage. *Journal of Bone and Joint Surgery* **50**B, 852–857.

BULLOUGH, P. G., MUNUERA, L., MURPHY, J. & WEINSTEIN, A. M. (1970). The strength of the menisci of the knee as it relates to their fine structure. *Journal of Bone and Joint Surgery* **52**B, 564–570.

BYERS, P. D., CONTEPOMI, C. A. & FARKAS, T. A. (1970). A post mortem study of the hip joint. *Annals of the Rheumatic Diseases* **29**, 15–31.

COLLINS, D. H. (1949). *The Pathology of Articular and Spinal Diseases*, pp. 74–115. London: Arnold.

GOODFELLOW, J. W. & BULLOUGH, P. G. (1967). The pattern of ageing of the articular cartilage of the elbow joint. *Journal of Bone and Joint Surgery* **49**B, 175–181.

EMERY, I. H. & MEACHIM, G. (1973). Surface morphology and topography of patello-femoral cartilage fibrillation in Liverpool necropsies. *Journal of Anatomy* **116**, 103–120.

FREEMAN, M. A. R. (1972). The pathogenesis of osteoarthrosis: an hypothesis. In *Modern Trends in Orthopaedics*, vol. 6 (ed. A. G. Apley), pp. 40–94. London: Butterworth.

HEINE, J. (1926). Über die Arthritis deformans. *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* **260**, 521–663.

MEACHIM, G. (1972*a*). Articular cartilage lesions in osteo-arthritis of the femoral head. *Journal of Pathology* **107**, 199–210.

MEACHIM, G. (1972*b*). Light microscopy of Indian ink preparations of fibrillated cartilage. *Annals of the Rheumatic Diseases* **31**, 457–464.

MEACHIM, G. & EMERY, I. H. (1974). Quantitative aspects of patello-femoral cartilage fibrillation in Liverpool necropsies. *Annals of the Rheumatic Diseases* (to be published).

MEACHIM, G. & FREEMAN, M. A. R. (1973). Ageing, degeneration and remodelling of articular cartilage. In *Adult Articular Cartilage* (Ed. M. A. R. Freeman), pp. 287–329. London: Pitman.

ØWRE, A. (1936). Chondromalacia patellae. *Acta chirugica scandinavica* **77**, supplementum 41.

RADIN, E. L. & PAUL, I. L. (1972). A consolidated concept of joint lubrication. *Journal of Bone and Joint Surgery* **54**A, 607–616.