

## Cartilage fibrillation at the ankle joint in Liverpool necropsies

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(Accepted 2 December 1974)

Studies of the wear-resistance, and other mechanical properties, of articular cartilage are usually made on tissue samples removed from their natural environment and tested in the laboratory *in vitro*. An alternative approach is to observe how human articular cartilage behaves as a material when mechanically tested in the 'biological rig' *in vivo*. With this in mind studies have been made of articular cartilage lesions found in the course of a random series of necropsies in Liverpool, using synovial joints from a range of age groups. Methods have been developed for mapping the distribution of cartilage lesions and quantifying their extent by a surface point-counting technique. The study has been aided by the use of preparations painted with India ink (Bullough & Goodfellow, 1968) to demonstrate the lesions and their morphology *en face* (Meachim, 1972*a*). Results from the patello-femoral, shoulder and hip joints in Liverpool subjects have been described elsewhere (Emery & Meachim, 1973; Meachim & Emery, 1973; Meachim & Emery, 1974). The present paper describes the articular surfaces of the ankle joint at necropsy in the same population and discusses the observations with reference to data from other joints.

### MATERIAL AND METHODS

The articular surfaces were studied in 45 left ankle joints from white European subjects (24 men; 21 women) whose ages ranged from 21 to 92 years. The specimens were obtained from a random series of necropsies in the city of Liverpool. Joints with evidence of rheumatoid or other inflammatory disease, or of old or recent injury, were excluded. The cartilages were examined in the unfixed state, using preparations kept moist with physiological saline. They were painted with India ink, using a technique described in detail elsewhere (Emery & Meachim, 1973), and then examined *en face* by stereomicroscopy at  $\times 10$  while still *in situ*. The nature and distribution of the cartilage lesions was recorded. Where indicated, tangential slices were cut free-hand from the cartilage surface, mounted in saline, and further examined *en face* by transmission light microscopy at magnifications up to  $\times 150$  (Meachim, 1972*a*). Vertically sawn tissue blocks of bone and cartilage were taken for fixation and conventional histological examination. On some specimens artificial splits were made by pricking the cartilage surface with a round pin (Bullough & Goodfellow, 1968); the resulting splits generally had an elongated, straight appearance when viewed *en face*, and ran parallel to the dominant direction of the superficial cartilage collagen.

## RESULTS

The following terms will be employed when describing the topography of the cartilage lesions (Fig. 1): the 'central' articulation territory of the opposing cartilage surfaces, comprising the main articulation between the distal tibia and the superior aspect of the talus; the *medial* and *lateral* 'malleolar' territories, comprising the articulations on the medial and lateral sides of the talus; the 'boundaries' between the central and the malleolar territories; and the 'periphery' of the cartilage sheets. Macroscopic inspection and histology confirmed that the cartilage covering the central and the two malleolar surfaces of the talus formed a single continuous sheet, as did that covering the central and medial malleolar surface of the distal tibia. In contrast, the cartilage on the lateral malleolar surface of the fibula was not continuous with that of the central articulation territory of the distal tibia.

*Overt fibrillation.* Overt fibrillation was indicated by frank splitting and fraying of the cartilage, with disintegration of the articular surface, and showed as a confluent or semi-confluent blackening in India ink preparations (Meachim, 1972a). Its extent varied considerably, even amongst individuals of similar age. Its topographical distribution, characteristic for the series as a whole, is depicted in Fig. 2. At the ankle, as in other joints, fibrillation was basically a focal process (Fig. 3). Foci of overt fibrillation were extremely common in the adult ankle joints, including joints from subjects in the third and fourth age decades (Fig. 4). With increasing age overt fibrillation tended in general to become more extensive, but there was considerable variation between individuals (Figs. 5-12). Taking the series as a whole, overt fibrillation was qualitatively and quantitatively similar on the talar

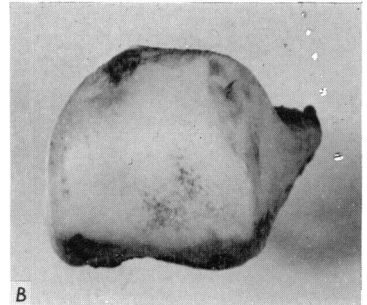
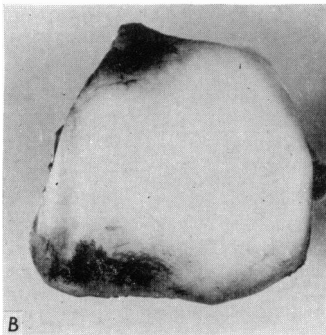
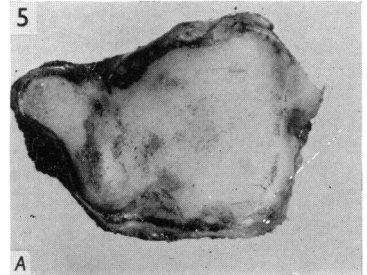
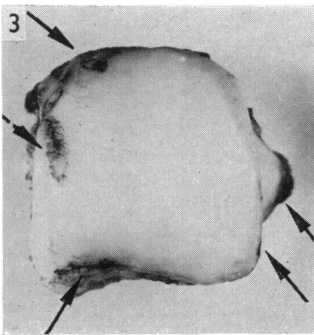
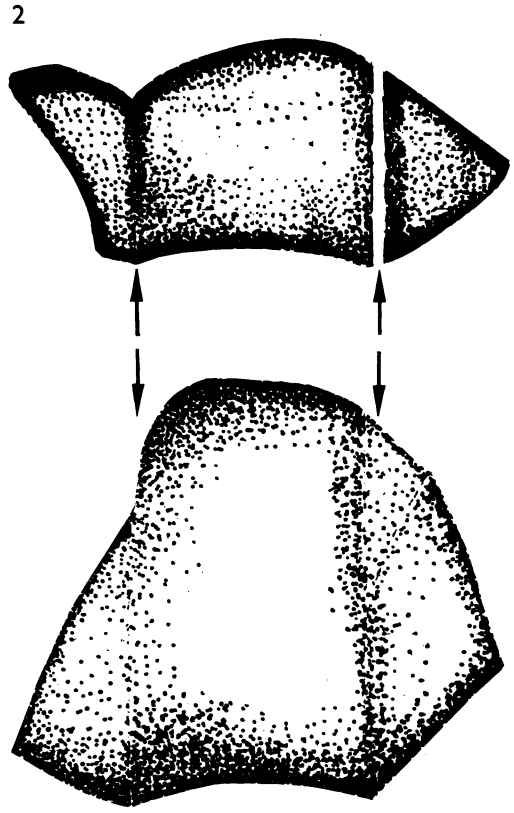
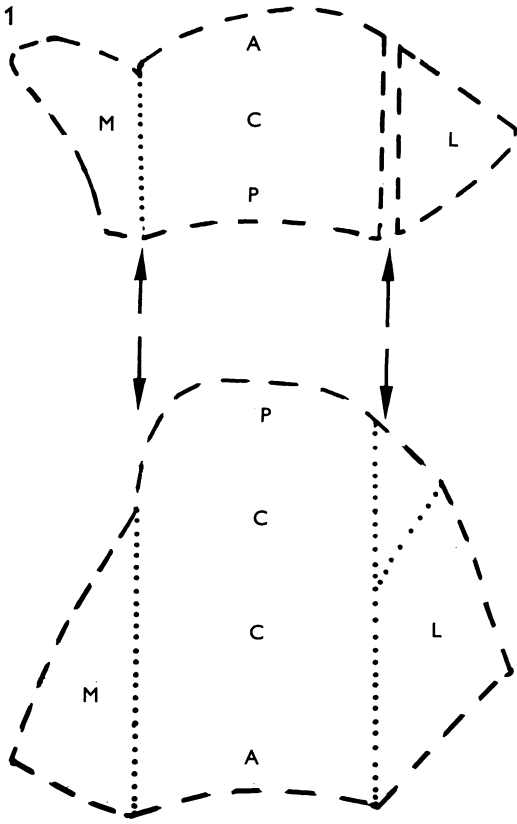
Fig. 1. Diagram depicting the terms used in describing the topography of the lesions on the distal tibia with lateral malleolus (above), and on the talus (below). Central territory on superior aspect of talus (CC) and its opposing central tibial territory (C). Anterior (A) and posterior (P) peripheries of the main bearing surface. Medial (M) and lateral (L) malleolar surfaces. Junctions between main and malleolar surfaces (arrows). In this diagram and in Fig. 2, convex and concave parts of the cartilage sheets are drawn as if laid flat, and thus the talus appears larger than in the subsequent photographs.

Fig. 2. Two-dimensional representation of the articular cartilages of the ankle joint, drawn as in Fig. 1, showing the differing susceptibility to overt fibrillation from one site to another. The arrows indicate the position of the abrupt changes in surface contour at the junctions between the main and the malleolar articulation territories (Fig. 1). High concentration of dots = high incidence of overt fibrillation: no dots = low, although not zero, incidence.

Fig. 3. India ink preparation of left talus, viewed from above. Woman aged 40 years. Small foci of overt fibrillation, stained black, are seen (arrows) at the periphery of the articular cartilage and near the junction between the superior and medial faces. In contrast, the cartilage of the central territory of the superior aspect is intact and unstained.  $\times 1$ .

Figs. 4A and 4B. India ink preparation of left distal tibial (A) and opposing talar (B) cartilage. Man aged 30 years. Areas of overt fibrillation, stained black, at the cartilage periphery. In this and some of the subsequent illustrations, pieces of para-articular fibrofatty tissue are included in the specimens, and it should be noted that this normal para-articular tissue also stains black.  $\times 1$ .

Figs. 5A and 5B. Left distal tibial (A) and opposing talar (B) cartilage. Woman aged 58 years. Areas of black staining due to overt fibrillation. Areas of less intense staining (stippled appearance) indicative of a mainly minimal fibrillation on parts of the central territory of the superior aspect of the talus and opposing tibia. India ink preparation.  $\times 1$ .



face and on the opposing tibial and fibular surfaces of the joint (Fig. 2). It especially and initially affected the periphery of the cartilage sheets (Figs. 3 and 4) and the boundaries between the central and malleolar articulation territories (Fig. 3). The non-peripheral parts of the malleolar surfaces, particularly that of the fibula, were next involved. The central territory of the articulation was the least susceptible to overt fibrillation (Fig. 11), although some of the subjects showed small patches of overt change even on this part of the joint (Fig. 10). However, even in persons over 70 years old, only a minority showed areas of overt fibrillation of any size (Figs. 8 and 12) on one or other of the opposing faces of this part of the articulation.

*Minimal fibrillation.* Minimal fibrillation represented a very superficial splitting of the cartilage surface, without the frank disintegration seen in overt fibrillation, and showed *en face* as dark markings against a pale grey background (Fig. 5) in India ink preparations (Meachim, 1972*a*). The concept that vertical extension of this minimal splitting is one of the phenomena that can lead to the development and spread of overt fibrillation has been discussed elsewhere, with particular reference to shoulders and hips (Meachim & Emery, 1973). This concept does not necessarily imply that vertical extension to overt change is inevitable: e.g. at the ankle joint minimal fibrillation (Figs. 5 and 6), unlike overt fibrillation, could occur at any site on the articular surfaces, while with increasing age, extensive involvement of the central territory with minimal fibrillation (Fig. 9) was much more common than overt change here. It would seem that minimal surface splitting on the central territory has only a low potential for vertical extension into overt fibrillation.

The surface markings of minimal fibrillation often, but not invariably, exhibited *en face* orientation when examined by light microscopy (Meachim, 1972*a*). Sometimes orientation was also apparent to the naked eye. At the ankle joint two sorts of markings in macroscopically apparent parallel alignment were encountered (Figs. 14 and 15). One sort showed an orientation macroscopically unrelated to the *en face* direction taken by splits made artificially into the nearby cartilage surface

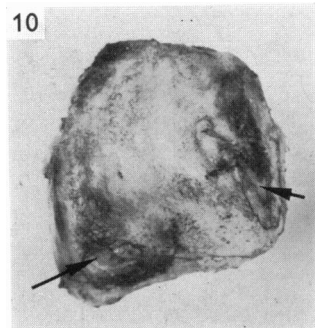
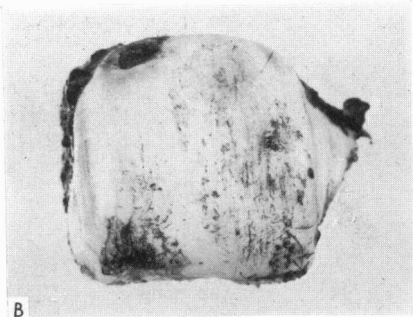
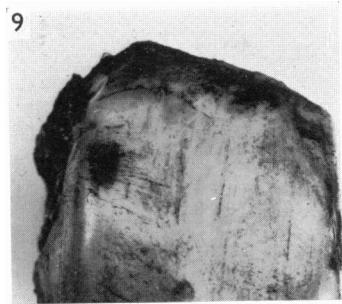
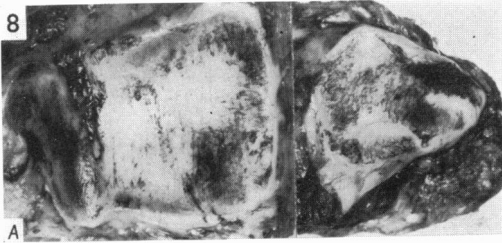
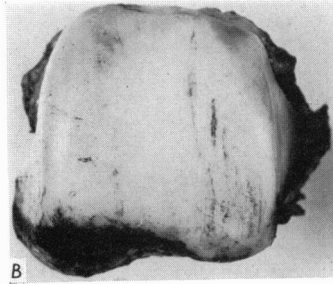
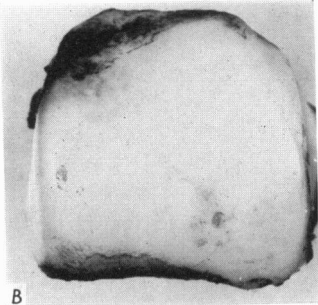
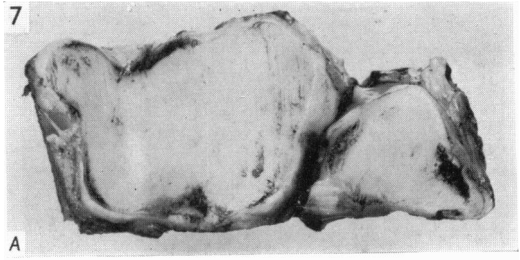
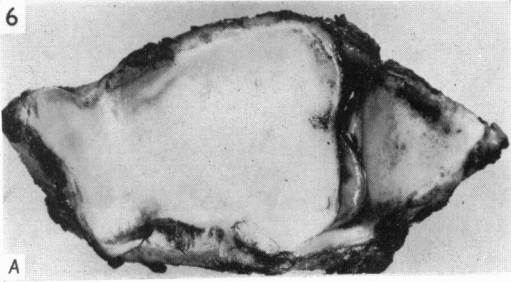
Figs. 6*A* and 6*B*. Articular cartilage of left distal tibia and lateral malleolus (*A*) and of talus (*B*) from a man aged 55 years. Peripheral overt fibrillation. Also some more central foci of mainly minimal fibrillation, seen particularly on the lateral malleolus.  $\times 1$ .

Figs. 7*A* and 7*B*. Left distal tibia with lateral malleolus (*A*) and opposing talus (*B*) from a man aged 46 years.  $\times 1$ . The lateral part of the talar surface is shown at a higher magnification in Fig. 15.

Figs. 8*A* and 8*B*. Left distal tibia with lateral malleolus (*A*), showing overt fibrillation affecting a major part of the lateral malleolar surface, nearly all the medial malleolar surface and some of the central tibial territory. Opposing talus (*B*). Man aged 56 years.  $\times 1$ .

Fig. 9. Part of the superior aspect of the left talus from a man aged 63 years. Areas of overt fibrillation at the periphery. Extensive minimal fibrillation on the central territory. Note the 'minimal' markings in two types of parallel alignments, one (right) anteroposterior and the other (left) approximately mediolateral.  $\times 1$ . See also Figs. 14 and 15.

Fig. 10. Superior aspect of left talus from a man aged 73 years. The central territory showed extensive minimal fibrillation with overt patches. There was a small area of full-thickness uncalcified cartilage loss at the anterior end of the boundary between the central and medial malleolar articulation territories (arrow), affecting both the talar and its opposing tibial surfaces, and a second area (arrow) on the lateral malleolar face of the talus. The lateral malleolus from this specimen is shown in Fig. 13.



(Fig. 15). Examples of this were encountered more commonly on central than on malleolar territories, and when present on the central territory often affected both the talar and the opposing tibial surfaces of the same specimen. On the central territory this sort of macroscopic marking showed an obvious relationship to the direction of flexion-extension movement (Fig. 15), and its orientation *en face* on this territory was consistent with track markings from abrasive-adhesive wear. The second sort of macroscopic markings were, in contrast, parallel to the artificial splits viewed *en face* (Fig. 14), and unrelated to the direction of joint movement. Such markings might be found anywhere on the ankle joint surfaces (Fig. 14). This sort of orientation was parallel to the dominant *en face* direction of the surface collagen. The majority of the specimens exhibited one or both sorts of macroscopically apparent orientation. When both were present they were usually on separate regions of the specimen, but in a few instances (Fig. 9) both sorts were macroscopically apparent at the same site. In some of the specimens, none of the minimal fibrillation markings showed alignments in a form which was macroscopically apparent.

*Full-thickness uncalcified cartilage loss.* Cartilage lesions of the type characteristic of surgical excision specimens from osteoarthritic hip and other joints (Meachim, 1972*b*) were seen in only 1 of the 20 subjects aged 71-92 years, and in none of the subjects younger than this. This single specimen, from a man aged 73 years, showed a non-peripheral area of full-thickness uncalcified cartilage loss on the opposing faces of the lateral malleolar articulation territory, with grooving of exposed calcified tissue (Fig. 13). The appearances indicated active abrasive wear of 'osteoarthritic' type (Meachim, 1972*b*). There was no obvious osteophytosis.

In contrast, in approximately one-third of 27 ankles from subjects over 60 years of age there were destructive-remodelling 'ageing' lesions of peripheral cartilage segments. These lesions showed one or more of the following features: full-thickness

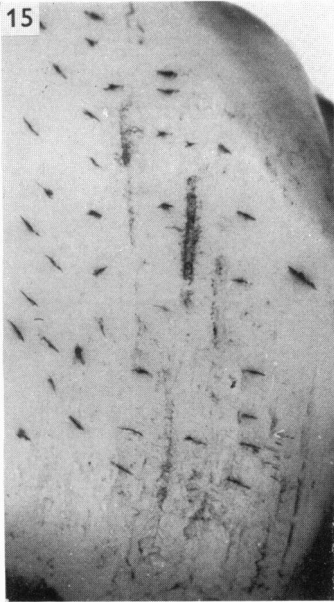
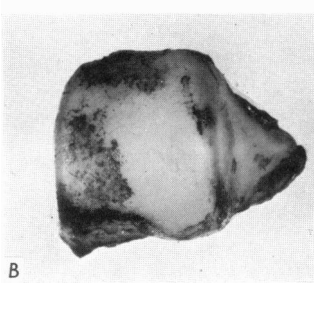
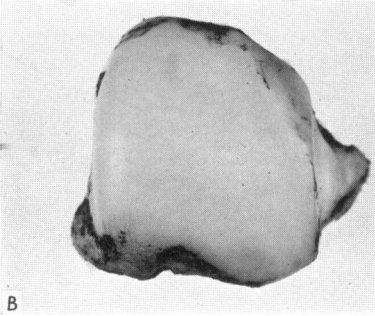
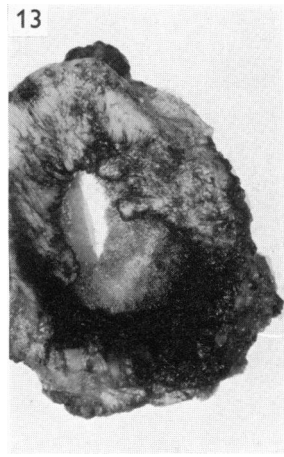
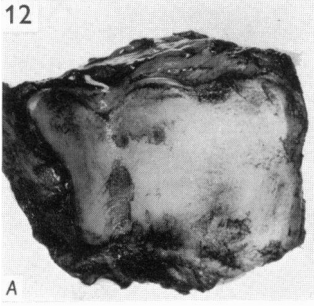
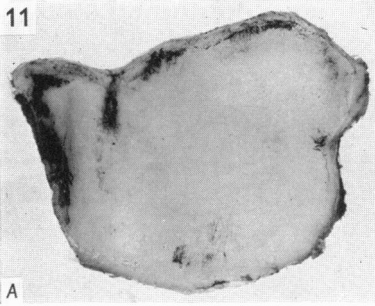
Figs. 11*A* and 11*B*. Left distal tibia (*A*) and opposing talus (*B*). The specimen is from a man aged 71 years, but most of the cartilage on the central territory is intact (unstained). Compare with Fig. 10.  $\times 1$ .

Figs. 12*A* and 12*B*. Left distal tibial (*A*) and opposing talar (*B*) cartilage from a woman aged 83 years. The joint was unusually small. Overt fibrillation involves part of the central territory of the superior aspect of the talus, although on the rest of this territory the cartilage is intact.  $\times 1$ .

Fig. 13. Lateral malleolus from the specimen also illustrated in Fig. 10. The centre of the malleolar articular surface showed full-thickness loss of the uncalcified cartilage, with grooving (linear highlight) of exposed calcified tissue indicative of abrasive wear. The rest of the cartilage on the lateral malleolus showed overt and minimal fibrillation.  $\times 2$ .

Fig. 14. Angle between medial malleolar and adjacent part of distal tibial surface. Woman aged 40 years. The short dark lines are artificially produced splits. The area of confluent blackening (centre) is an area of overt fibrillation. Below and to the right of this area there are fine parallel markings of minimal fibrillation. Such macroscopically apparent parallel markings are aligned in the same direction as the artificial splits.  $\times 3$ .

Fig. 15. Part of the superior aspect of the talus shown in Fig. 7*B*, but with artificial splits now made into the cartilage surface. There are macroscopically apparent parallel markings of minimal fibrillation, of the sort which are aligned in the same direction as that of flexion-extension movement, and unrelated to that of the artificial splits.  $\times 3$ . On microscopy this sort of marking has a different appearance from that of the markings shown in Fig. 14.



loss of a peripheral segment of uncalcified cartilage, down to the level of calcified cartilage or bone; fibrous covering over a peripheral segment of uncalcified cartilage or over the peripheral sites of full thickness uncalcified cartilage loss; peripheral intracartilaginous ossification; minor degrees of osteophytic lipping. At the histological level these features represented closely related phenomena (Meachim & Emery, 1973). They were not considered to be 'osteoarthritic' in nature, since the segments of peripheral cartilage loss were covered by fibrous tissue and did not show evidence of active abrasive wear of exposed calcified tissue.

#### DISCUSSION

The present findings indicate that foci of articular cartilage fibrillation are extremely common in the adult ankle joint. The same holds true for the patello-femoral (Emery & Meachim, 1973), radio-humeral (Goodfellow & Bullough, 1967), shoulder (Meachim & Emery, 1973) and hip articulations (Byers, Contepomi & Farkas, 1970; Meachim & Emery, 1973). Thus, fibrillated sites are to be regarded as a normal feature of an adult synovial joint; such a joint will, of course, usually also have regions on which the cartilage surface is intact. Since cartilage fibrillation is so frequent a finding in adult joints, it must often, although not invariably, be an asymptomatic process without clinical significance.

The changes in human articular cartilage with day-to-day use can be considered in terms (1) of the onset of overt fibrillation, (2) of its spread tangentially across the cartilage surfaces, and (3) of its vertical progression into the cartilage towards the bone.

*Onset of overt fibrillation.* At the ankle joint the peripheral parts of the cartilage sheets were particularly susceptible to overt fibrillation. Such peripheral susceptibility was also seen at the patello-femoral, shoulder and hip joints (Emery & Meachim, 1973; Meachim & Emery, 1973). In contrast, most, although not all, peripheral segments of the upper tibial articular cartilages are relatively immune, but this apparent exception may be due to the protective effect of the overlying menisci (Bullough, Munuera, Murphy & Weinstein, 1970). The reason for the generally high susceptibility to overt fibrillation of the peripheral parts of the cartilage sheets is debatable. One possibility is that there may be a low frequency of cartilage-to-cartilage contact at the edge of articulations, and that insufficiently frequent contact during normal day-to-day use may cause the cartilage to 'degenerate' (Bullough, Goodfellow & O'Connor, 1973), or else make it unusually susceptible to mechanically-induced wear from such contacts as do occur. An alternative possibility is that the cartilage periphery is mechanically damaged by excessive 'ploughing' under the interrupted edge of its opposing sheet (Freeman & Meachim, 1973).

The present study shows that the ankle joint cartilages were also susceptible to overt fibrillation at the boundaries between their 'central' and 'malleolar' articulation territories. A similar phenomenon has been observed on the distal femoral surface at the boundary between its patellar and tibial articulations (Emery & Meachim, 1973).

In hip and knee joints, non-peripheral sites often affected by overt fibrillation



included the inferomedial segment of the femoral head, the posterosuperior segment of the acetabulum, and the medial strip of the medial patellar facet (Emery & Meachim, 1973; Meachim & Emery, 1973). It is possible that insufficiently frequent cartilage-to-cartilage contact at these sites might have been responsible (Bullough *et al.* 1973).

*Spread of overt fibrillation across the cartilage surfaces.* The tendency for overt fibrillation to spread tangentially across a cartilage surface with increasing age varies with the individual (Meachim & Emery, 1974), and with the anatomical site. At the patello-femoral articulation, for example, overt fibrillation can involve eventually a major proportion of the opposing cartilage surfaces, but even at this susceptible site the rate of tangential spread was slow in terms of a person's total life-span (Meachim & Emery, 1974). The present findings indicated that at the ankle overt fibrillation showed a relatively low tendency to spread across the cartilage surfaces, although minimal fibrillation was often widespread in older subjects.

*Vertical progression of cartilage fibrillation.* The tendency of fibrillation to spread vertically, leading eventually to full-thickness cartilage loss with bone exposure, varied from individual to individual and from one anatomical site to another. It is, of course, this vertical progression of wear which is crucial from the functional point of view. In the present series of ankle joints, only 1 out of 20 specimens from subjects over 70 years old showed anywhere a region of full-thickness loss of uncalcified cartilage with abrasive wear of exposed calcified tissue, like that seen in patients with clinically diagnosed osteoarthritis (Meachim, 1972*b*). Some of the older ankle joints showed full-thickness loss of peripheral segments of uncalcified cartilage, but with fibrous covering of the affected segments. This was not considered an 'osteoarthritic' phenomenon because calcified tissue was not exposed and gave no evidence of abrasive damage as it might have done had the calcified cartilage been exposed at some earlier date and subsequently buried under new fibrous tissue. Thus we may conclude that in Liverpool the articular cartilage of the normal ankle joint gives good service for at least 80 years and exhibits a high resistance to vertical wear of the osteoarthritic type. This is also the case in the shoulder and hip joints (Meachim & Emery, 1973). In contrast, at the patello-femoral articulation of elderly Liverpool subjects, osteoarthritis resulting solely from an 'ageing' process would seem to be relatively common (Emery & Meachim, 1973; Meachim & Emery, 1974).

The present findings from necropsy specimens suggest that clinical osteoarthritis of the ankle is not simply a consequence of ageing, but that some additional factor is required, such as fracture, neuropathy or rheumatoid disease. There is also the hypothetical possibility that a minority of the population has inherently defective ankle joint cartilage.

#### SUMMARY

The articular surfaces have been examined in 45 left ankle joints from a random series of adult necropsies in the city of Liverpool.

Foci of *overt* fibrillation, with frank splitting of the articular cartilage surface, were extremely common in the ankle joint and were often already apparent in young adults. With increasing age overt fibrillation tended on the whole to become

more extensive, but this tendency was partly obscured by considerable variation between individuals. It initially and especially affected the periphery of the cartilage sheets and the boundaries between the central and malleolar articulation territories. Possible explanations for this peripheral susceptibility are discussed. One or more sites on the non-peripheral parts of the lateral and medial malleolar surfaces were next in order of susceptibility. The central territory of the articulation was the least susceptible to overt fibrillation.

*Minimal* fibrillation, with a very superficial splitting of the cartilage surface, could occur at any site on the ankle joint surfaces. With increasing age, extensive involvement of the central territory by this minimal change was much more common than extensive overt fibrillation here. The surface markings of minimal fibrillation sometimes showed a macroscopically apparent orientation *en face*. In one sort these were parallel to flexion-extension movement and consistent with track markings from abrasive-adhesive wear. In a second sort they were parallel to the dominant *en face* orientation of the superficial cartilage collagen.

The topography and natural history of cartilage fibrillation at the ankle joint is discussed with reference to Liverpool data from other joints.

Some of the older ankles showed peripheral segments of fibrous tissue-covered cartilage loss. This feature was attributed to 'ageing' rather than osteoarthritis. Only 1 of 20 left ankles from subjects more than 70 years old showed any region of full-thickness uncalcified cartilage loss with abrasive wear of 'osteoarthritic' type. Thus it would seem that in the majority of the Liverpool population the articular cartilage of the normal ankle joint has a high resistance to the type of vertical wear found in clinically demonstrable osteoarthritis. The practical implications of this conclusion are discussed.

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