Thickness and distribution of human femoral head articular cartilage

Changes with age

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SUMMARY The distribution of hyaline cartilage in the sagittal plane of the normal human femoral head was examined. 28 specimens from persons aged 10 to 68 years subject to post-mortem examination in a teaching hospital were anatomically orientated and x-rayed in a lateromedial projection. An optical technique was used to measure the cartilage thickness from the x-ray films thus produced. Measurements were made with a sensitivity of ± 0.01 mm using a metrological projector. 11 defined sites on each femoral head were surveyed to give the distribution of cartilage thickness. The greatest thickness was identified anterior to the zenith. Between the ages of 20 and 45 years the thickness of this zone increased with age. The thickness of femoral head cartilage was not evidently related to femoral head diameter, femur length, or body weight.

The purpose of the investigations reported in this paper was to establish the thickness and distribution of human femoral head cartilage and to examine the relationship between thickness and age. A preliminary report has been published (Armstrong, 1976).

The thickness of the articular cartilage of mammalian limbs has been found to be allometrically related to body weight, to hip-to-shoulder length, and to the area of the tibial plateau (Simon, 1970). It is also evident that cartilage thickness is inversely proportional to the relative congruence of the joint, at least in the dog (Simon *et al.*, 1973). By contrast, very few measurements have been recorded of the thickness, area, distribution, and mass of the hyaline articular cartilage that forms the bearing surfaces of human hip and knee joints. Moreover, the relationship between these variables and age has not been determined. Yet the physical properties of this cartilage are crucial to an understanding of the natural history of the most important forms of human osteoarthritis.

A view has been expressed that cartilage thinning advances from maturity to senescence (Barnett *et al.*,

1961). Measurements of the mean thickness of human humeral cartilage do not support this opinion (Meachim, 1971) and no significant alteration of patellar cartilage thickness accompanies aging (Simon, 1971). The systematic measurement of human femoral head cartilage at different ages has not been undertaken.

Materials and methods

Twenty-eight whole right femurs were obtained at post-mortem examination of 13 males and 15 females. The ages and causes of death of the subjects are given in Table 1. They were selected to exclude gross obesity, chronic renal, metabolic, and endocrine disease, articular and systemic connective tissue disease, trauma to the hip joint and adjacent structures, and a history of prolonged immobilization. Femoral heads that displayed overt fibrillation on the superior surface (Meachim and Stockwell, 1973) were also excluded.

To ensure comparability each femur was orientated with respect to the main body planes so that the centre of the hip joint contact area, approximated by the zenith of the femoral head, was in the same vertical plane as the tibiofemoral contact areas. For convenience this orientation was achieved with the specimen horizontal (Fig. 1). The femur rested on the

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Table 1 Age, sex, and cause of death of patients from whom specimens were taken for this study

Age (yrs)	Sex	Cause of death
10	F	Fallot's tetralogy
12	F	Renal failure, pneumonia
12	F	Pneumonia
13	M	Suprasellar cyst
18	F	Gunshot wounds
18	M	Gunshot wounds
20	F	Cerebral tumour
20	F	Amniotic fluid embolism
22*	M	Aortic aneurysm
26	F	Cerebral anoxia
29	M	Cerebral tumour
32	M	Cerebral haemorrhage
36	F	Subarachnoid haemorrhage
37	F	Subarachnoid haemorrhage
39	F	Cerebral haemorrhage
41	M	Cerebral infarct
43	F	Pneumonia, atheroma
43	M	Myocardial infarction
46	M	Myocardial infarction
47	F	Lung tumour
49	M	Coronary thrombosis
51	F	Intestinal obstruction
51	M	Subarachnoid haemorrhage
51	F	Bronchopneumonia
53	M	Lung tumour
55	M	Intestinal tumour
56	M	Pneumonia, fractured skull
58	M	Blastic oedema
68	F	Colon carcinoma

*Specimen deleted.

posterior condyles and a vertically adjustable support. A perspex jig representing the three perpendicular body planes was positioned so that one vertical face simultaneously touched the inferior, tibial surface of both condyles. The points of contact could be seen through the perspex as bright spots. These gave a good approximation of the centres of the tibiofemoral contact areas. An ink line, the condylar line, was drawn on the perspex to join the spots. To identify the zenith of the femoral head the jig was then transferred to the upper end of the femur. There, the height of the support was adjusted until the contact spot on the femoral head lay on the condylar line, defining the zenith. Ink marks were then taken from the perspex to the femoral head, marking the zenith and the sagittal plane.

Each femoral head was then removed and placed for 30 minutes in normal saline before reorientation,

Fig. 1 Diagram of the method of femur orientation showing ink line and points of contact of cartilage with perspex jig.

by means of the reference ink marks, on a small rotary table (Unimatic Engineers model TR46). With the zenith and circumferential limits of the cartilage marked with needles, the specimen was x-rayed in the lateromedial projection in a Faxitron 8050 cabinet. The x-rays were superimposed on a transparent polar grid and measurement sites marked at 20° angular intervals from the zenith (Fig. 2). Cartilage thickness at each site was measured with a Heuer optical projector designed for engineering metrology. Readings were taken to the nearest 0.01 mm. The maximum range of variation recorded at any site when one specimen was reorientated, x-rayed, and measured 3 times was ± 0.03 mm. Appropriate statistical analyses were made. The regression equations, correlation coefficients, and significance levels are given in Table 2.

Fig. 2 X-ray of femoral head showing measurement sites. Topmost point of femur and cartilage margins marked by needles.

Table 2 Results of multiple regression analyses of cartilage thickness variation at each site. 13 specimens from patients ranging in age from 18–45 years

Cartilage thickness at	Age	Sex (M=1) (F=2)	Height	Multiple R	P
-100°					NS
-80°					NS
-60°					NS
-40°	+	-	-	0.8260	0.025
-20°	+	-	-	0.8673	0.01
0°	+			0.7114	0.01
20°	+			0.7630	0.01
40°	+			0.6959	0.01
60°	+			0.6014	0.05
80°	+			0.6306	0.025
100°	+			0.7096	0.01

+ = an increase in the variable gives an increase in cartilage thickness; - = the opposite effect.

Note: There was no significant correlation of cartilage thickness with body weight, femoral head diameter, or femoral length.

Fig. 3 Cartilage thickness is measured against the angle from the zenith or topmost point of the femoral head; mean of all specimens over 18 years old.

Results

CARTILAGE THICKNESS IN RELATION TO ANGLE FROM THE ZENITH

A characteristic distribution of cartilage was recognized in the lateromedial radiograph of the femoral head (Fig. 2). From a thickness of approximately 1 mm on the posterior face, the cartilage increased in depth to 2 mm in the -20° to $+40^\circ$ zone (Fig. 3), i.e. immediately anterior to the zenith. Thickness then decreased again. Asymmetry was characteristic but was least pronounced in immature and young adult femoral heads. To determine whether asymmetry was unique to the plane chosen for measurement, 2 specimens were x-rayed in a range of positions representing a 3-dimensional survey of the thickness of the entire cartilage. These x-rays showed a similar pattern to those detected in the lateromedial view. There was more cartilage at any point on the anterior hemisphere compared to an equivalent point on the posterior. There was also more cartilage on the medial than on the lateral surface.

CARTILAGE THICKNESS IN RELATION TO AGE (EACH SITE) (FIGS. 4-7)

The variation of cartilage thickness at each site with age showed significant differences. In childhood and adolescence a distinction between growth and articular cartilage was impracticable. Near the articular margins (Figs. 4 and 7), there was little alteration after this age. However, towards the anterosuperior surface (Fig. 6), the cartilage thickness increased again up to age 45 years. Beyond this age the scatter of thickness measurements was large. The pattern of variation was similar at each site over the entire surface but the magnitude of variation increased gradually with distance from the articular margins to a maximum in the $+20^\circ$ to $+40^\circ$ regions. To determine whether this was peculiar to the chosen plane of measurement,

three additional sites were selected in the perpendicular anteroposterior projection. The resulting patterns of thickness variation with age were similar to those in the lateromedial projection.

The relationships of the cartilage thickness at each site to age, sex, body weight, height, femoral length, and femoral head diameter were estimated by multiple regression analyses. If the entire sample age range 10-68 years was analysed, the regression

Fig. 4

Figs. 4-7 Cartilage thickness measured against age at each measurement site.

Fig. 5

equation was heavily weighted by the four specimens from patients of less than 15 years, due to the thick layer of growth cartilage shown. Measurements made from specimens of over 45 years showed a very large scatter that could mask trends outside this age group. Thus, analyses performed on all specimens over 25 years showed no highly significant correlations between the variables. When calculations were limited to the 13 specimens in the range 18–45 years, a significant increase in cartilage thickness with age

alone at all sites on the anterior half of the femoral head was recognized, i.e. a simple linear regression (Fig. 8). If corrections for specimen sex, and height differences were performed by multiple regression analysis, a significant thickness increase was also found at -20° and -40° .

Discussion

These investigations show that the distribution of articular cartilage over the head of the adult human

Fig. 6

Fig. 7

femur is asymmetrical. The thickest cartilage is identified on the anterosuperior surface of the femoral head. The present evidence also confirms that the thickness of the adult anterosuperior zone increases with age, by contrast with the margin of the articular cartilage where there is little increase in thickness. If the measured increases are attributable to abnormal selection, i.e. bias in the choice of material, it is anticipated that the percentage increase in cartilage thickness would be the same at all sites. This is not the case. It is interesting to note that there appears to be little relationship between measures of skeletal geometry such as height, femoral length, and femoral head diameter, and the thickness of the femoral head cartilage.

There are few published records of measurements of the thickness of human articular cartilage and none that show its distribution quantitatively. Determining creep modulus for a series of femoral head cartilages, Kempson (1970) recorded measurements of cartilage thickness. Six records were from patients aged less than 50 years: the measurements conformed with the patterns reported here. In studies of the cartilage covering the head of the humerus, Meachim (1971) determined mean thicknesses over substantial areas of fixed histological preparations. Dividing his adult

sample into age groups, he identified no significant change in humeral head cartilage thickness with time. If the present results are grouped in this manner they also fail to show an increase in femoral head cartilage thickness with age. In the same way Simon (1971), who measured the ratio between patellar cartilage area in section and total cross-sectional patellar area, identified no variation with age after maturity.

A suggestion has been made that zones of cartilage that have undergone the mild, superficial fibrillation of established osteoarthritis are thicker than normal (Meachim, 1971). This view conforms with the microscopical evidence that chondrocytes near the fissures and clefts are grouped in clusters in which the increased numbers of cells indicate an increased frequency of cell division. The suggestion also conforms with biochemical evidence (Mankin, 1974) that shows an increased rate of DNA and proteoglycan synthesis in such zones by contrast with the decreasing rates in parts where cartilage loss has begun.

If zones of increasing cartilage thickness are regarded as sites of predisposition to osteoarthritis, then surveys of human femoral head cartilage such as those reported by Byers *et al.* (1970) would be expected to show fibrillation most commonly in the anterosuperior region. This is not the case. Byers and his colleagues detected fibrillation of the superior surface in only 16 of 400 femoral heads although in 15 of these cases the damage was recognized in the

Fig. 8 Diagram of femoral head showing mean cartilage thickness ± 3 SD and results of multiple regression analyses of 18- to 45-year age range.

** Significant regression of cartilage thickness against age alone, i.e. a simple linear regression. * Significant regression of cartilage thickness against age, sex, and body height with age accounting for the majority of the variation explained. See Table 2 for details of correlation coefficients and significance levels.

anterosuperior region. It is of course possible that fibrillation, whether identified by simple inspection or by the India ink painting technique, is not as sensitive or as precise a guide to established early osteoarthritis as has been suggested. There is strong evidence to support this criticism. But it is equally possible that increased cartilage thickness, an age-related change, is not a factor leading to osteoarthritis, as might be surmised from Meachim's investigations of the head of the humerus.

It is tempting to draw the results of McDevitt and Muir (1976) and Muir (1977) into the debate. Division of the anterior cruciate ligament in dogs invariably causes the rapid onset of cartilage injury in a defined area of the tibial condyle. Analysis shows an early increase in the water content of the injured thickened cartilage, and a subsequent increase in proteoglycan monomers.

It can be argued that the canine cartilage-injury model should not be accepted as an analogue of human osteoarthritis except of those instances of the human disease, such as the knee osteoarthritis of footballers, where local trauma has initiated the disorder. In the great majority of examples of human femoral head osteoarthritis the course of the disease is prolonged, the progress insidious, the initial provocative factors unknown: the contrast with the dog model is very great.

On this basis it is logical to relate the observed increase in cartilage thickness with age not to injury but to physiological mechanical factors. This view takes account of the evidence that, whereas load peaks during walking occur symmetrically on the femoral head most other loads, including those of lifting and standing-up, occur with the joint flexed and are upon the anterosuperior zone. In immature and young adult femoral heads, as we record, the asymmetry of femoral head cartilage distribution is small. But as middle age is approached asymmetry becomes pronounced with an increase in cartilage in the $+20^\circ$ to $+40^\circ$ region. No *inherent* requirement for an asymmetrical layer of cartilage is apparent but evidently, with time, a change in the quality and/or quantity of load-bearing leads to those

alterations in cartilage cell synthetic mechanisms that culminate in the thickened zones detected on systematic measurement.

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References

- Armstrong, C. G. (1976). The distribution of human femoral head cartilage. *Clinical Science and Molecular Medicine*, **50**, 1P.
- Barnett, C. H., Davies, D. V., and MacConaill, M. A. (1959). *Synovial Joints. Their Structure and Mechanics*, p. 88. Longmans, London.
- Byers, P. D., Contepomi, C. A., and Farkas, T. A. (1970). A post-mortem study of the hip joint. *Annals of the Rheumatic Diseases*, **29**, 15-31.
- Kempson, G. E. (1970). Mechanical properties of human articular cartilage. Ph.D. thesis, University of London.
- McDevitt, C. A., and Muir, H. (1976). Biochemical changes in the cartilage of the knee in experimental and natural osteoarthritis in the dog. *Journal of Bone and Joint Surgery*, **58B**, 94-101.
- Mankin, H. J. (1974). The reaction of articular cartilage to injury and osteoarthritis. *New England Journal of Medicine*, **291**, 1335-1340.
- Meachim, G. (1971). Effect of age on the thickness of adult articular cartilage at the shoulder joint. *Annals of the Rheumatic Diseases*, **30**, 43-46.
- Meachim, G., and Stockwell, R. A. (1973). The matrix. *Adult Articular Cartilage*, p. 42. Ed. by M. A. R. Freeman. Pitman Medical, London.
- Muir, H. (1977). Molecular approach to the understanding of osteoarthritis. (Heberden Oration). *Annals of the Rheumatic Diseases*, **36**, 199-208.
- Simon, W. H. (1970). Scale effects in animal joints. I. Articular cartilage thickness and compressive stress. *Arthritis and Rheumatism*, **13**, 244-255.
- Simon, W. H. (1971). Scale effects in animal joints. II. Thickness and elasticity in the deformability of articular cartilage. *Arthritis and Rheumatism*, **14**, 493-502.
- Simon, W. H., Friedenber, S., and Richardson, S. (1973). Joint congruence. A correlation of joint congruence and thickness of articular cartilage in dogs. *Journal of Bone and Joint Surgery*, **55A**, 1614-1620.