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HARDNESS OF THE SUBCHONDRAL BONE OF THE TIBIAL CONDYLES IN THE NORMAL STATE AND IN OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS

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During the last decade several authors have advanced the theory that bone changes come before cartilage destruction in osteoarthritis as alterations in bone could significantly affect the pressures exerted on the cartilage.

Cartilage destruction is one of the hallmarks of rheumatoid disease. Scant information is available on the histologic features of the subchondral bone in rheumatoid arthritis. Murray (1969) has radiologically demonstrated subarticular decalcifications as early signs of rheumatoid arthritis. To the best of our knowledge no investigations on the mechanical properties have been presented.

We have embarked on an investigation the aim of which has been to analyse biomechanical, biochemical and morphologic features of subchondral bone in rheumatoid arthritis in comparison with normal bone and that in osteoarthritis.

This presentation concerns the biomechanical part which deals with the measurement of hardness of subchondral, trabecular bone.

MATERIAL

Specimens have been taken from the medial and lateral condyles of the tibia and have been obtained at autopsy, amputation and reconstructive surgery.

Normal: Twenty-three subjects with an age range from 20 to 90 years (see Table 1).

The normal structure of the tibial condyles was ascertained by naked eye observation and radiology.

Cause of death was myocardial infarction (8), cerebral haemorrhage (3), cancer not affecting the skeletal system (9), and various diseases (3).

Table 1. Distribution according to age, sex, disease and localization of bone specimens for hardness tests.

Age	Normal				Osteoarthritis				Rheumatoid arthritis			
	M		F		M		F		M		F	
	MC	LC	MC	LC	MC	LC	MC	LC	MC	LC	MC	LC
20-29	1		3	1								
30-39			1									
40-49	3	2	1	1								
50-59	2	1	1						3		2	1
60-69	2	1	1	1	3	2	2		2		2	1
70-79			4	3	3	2	2		2	1	4	
80-89	3	3			2	1	3				1	
>90	1						1					
Total	12	7	11	6	8	5	8		7	1	9	2

MC = medial condyle, LC = lateral condyle.

M = males, F = females.

All individuals had been fully active until shortly before death. In this group 23 medial and 13 lateral condyles were tested.

Osteoarthritis: Sixteen subjects with an age range from 62 to 91 years (see Table 1).

The diagnosis was verified by naked eye observation and radiology and based on cartilage destruction, osteophytes, subchondral cysts and sclerosis.

Ten cases were obtained at autopsy, the cause of death being myocardial infarction (5), pulmonary embolism (1), cerebral haemorrhage (1), pneumonia (2), and oesophageal cancer (1). Six cases were obtained at amputation for vascular disease.

All individuals had been mobile until shortly before the specimens were taken.

In this group 16 medial and 5 lateral condyles were tested.

Rheumatoid arthritis: There are three factors to be considered when investigating bone specimens in rheumatoid arthritis:

1. the stage of the disease,
2. steroid treatment, and
3. immobilization.

Due consideration has been taken of these three factors in the selection of specimens from the 16 cases which have been investigated. Of these, 15 were classified as classical rheumatoid arthritis according to the criteria of the American Rheumatism Association (1958) and one as definite.

A general feature of rheumatoid arthritis is osteoporosis (Gardner 1972). Previously this has been believed to be in part due to steroid treatment (Chandler et al. 1958, Edström 1961), but according to McConkey et al. (1962) and Saville & Kharmosh (1967) the use of corticosteroids did not significantly increase the frequency of osteoporosis.

In our investigation seven of the 16 cases had received steroid treatment for various lengths of time during the 5-year period prior to the taking of the specimen.

All individuals had been actively mobilized. In two there had been no weight-bearing.

The age range for the 16 cases was 53 to 86 years (see Table 1).

Six were obtained at autopsy the cause of death being cardiovascular in all. Two specimens were taken at amputation because of circulatory failure and the remaining eight at reconstructive surgery.

In this group 16 medial and 3 lateral condyles were tested.

METHOD

The condyles were removed in one piece with a margin of 2-3 cm subchondral bone. Immediately after removal the specimens were frozen to -20° Centigrade, a procedure which according to Sedlin (1965) and to Sedlin & Hirsch (1966) has no adverse effects on the physical properties of bone.

Before submitting the specimens to the hardness test they were thawed in Ringer's solution. After thawing the lower transected bone area was further prepared with a hand saw to a plane that was parallel to the weightbearing articular surface. The cartilage of the weightbearing area was then carefully removed with a chisel and knife to the calcified zone. (To ascertain that this zone had been reached without interfering with the subchondral bone, the zone was studied under a microscope. Histologic verification of the exactness of this procedure was later obtained). The remainder was carefully ground away in Ringer's solution and the subchondral cortical bone was removed so that trabecular bone was reached (Figure 1). In this way one area of about 1 cm diameter within each condyle could be prepared for the hardness tests. These were carried out on wet bone in room temperature with normal humidity.

Hardness of a material can be determined by applying a specified load over a certain time and calculating the ratio of load to indentation area. Practically, this can be done by pressing a steel ball (Figures 2 a, 2 b) according to Brinell or a

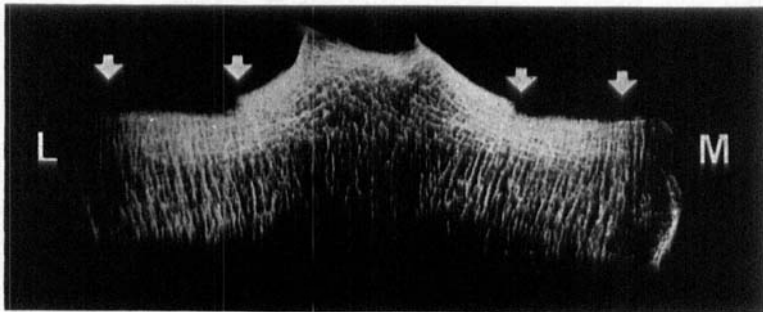


Figure 1. X-ray of 5 mm thick slice of lateral (L) and medial (M) condyles of normal tibial plateau. Cartilage and subchondral bone plate ground away between arrows.

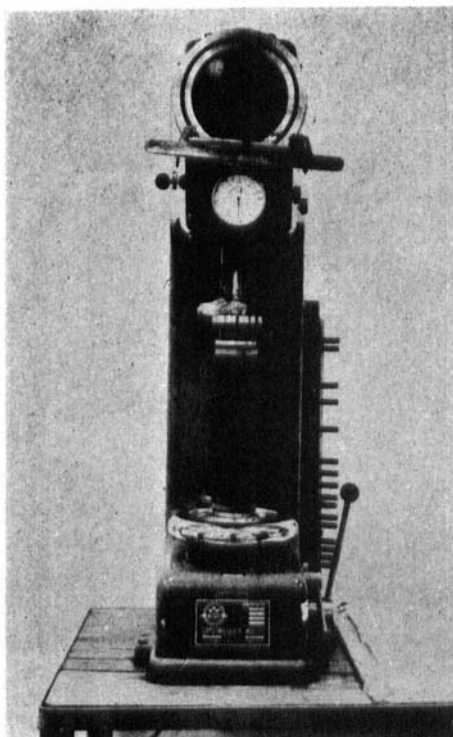


Figure 2 a. Micro-hardness tester (Otto Wolpert Werke) used for hardness measurements of subchondral bone in tibial plateaus.

pyramidal-formed diamond according to Vickers into the substance. We have used the Brinell method as described by v. Weingraber (1952), (Brinell 1900).

A steel ball with the diameter D in mm is pressed for a specific time with a load P in kp into the material (Figure 3). An indentation area A is formed and measured in mm^2 . The Brinell value (HB) as measured in kp/mm^2 is the ratio of the load P to the indentation area A , i.e.

$$A = \frac{\pi}{2} D (D - \sqrt{D^2 - d^2}) = \pi D t$$

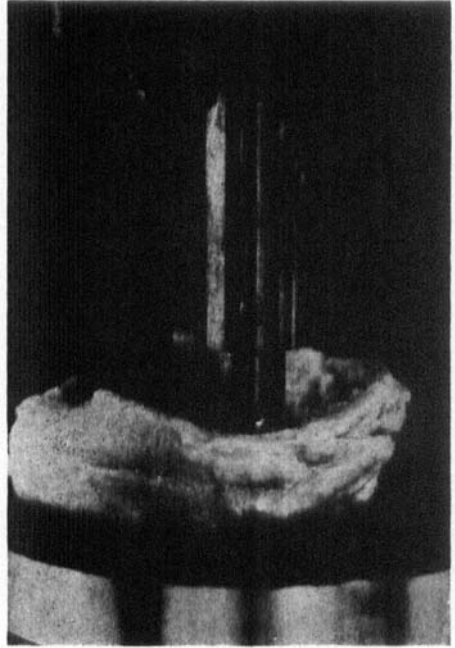
In this investigation the diameter of indentation area d has been used for the calculation.

The Brinell value can thus be written :

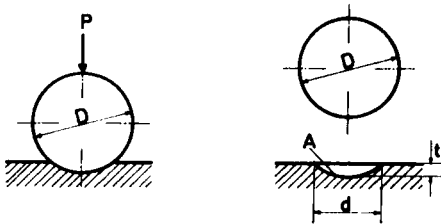
$$HB = \frac{2 P}{\pi D (D - \sqrt{D^2 - d^2})}$$

For this investigation a steel ball with the diameter 5 mm has been used, and the load was 10 kp. The time for indentation was 15 seconds. This time was found suitable as the indentation area progressed during 5 seconds after which it re-

Figure 2 b. Close-up of steel ball for indentation test according to Brinell. The ball is pressed into the subchondral bone covered with pressure sensitive tape after removal of the cartilage and subchondral bone plate within the weightbearing area of the tibial plateau.



HARDNESS TEST ACCORDING TO BRINELL



HARDNESS, $HB = \frac{P}{A} = \frac{\text{FORCE}}{\text{INDENTATION AREA}}$

$A = \frac{\pi}{2} D(D - \sqrt{D^2 - d^2}) = \pi D t$

D = DIAMETER OF BALL

d = DIAMETER OF INDENTATION

t = DEPTH OF INDENTATION

$HB = \frac{2P}{\pi D(D - \sqrt{D^2 - d^2})} \text{ [kp/mm}^2\text{]}$

Figure 3. Schematic demonstration of hardness test according to Brinell.

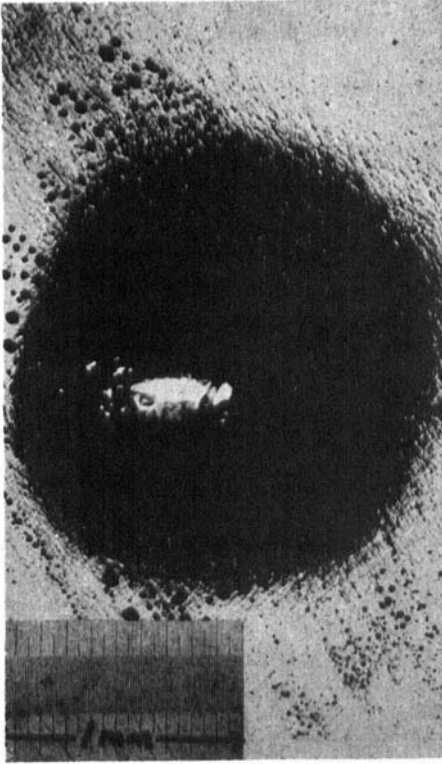


Figure 4 a. Indentation area (shadowed black zone) as maintained on the pressure sensitive tape after indentation into hard material.



Figure 4 b. Same as 4 a, but with indentation in softer material.

Table 2. Mean Brinell hardness for normal bone, and that in osteoarthritis and rheumatoid arthritis' at various ages. Increase statistically significant at 95 per cent level.

(Graphic demonstration in Figures 5-7).

Age	Normal	Osteoarthritis	Rheumatoid arthritis
20-29	5.7		
30-39	5.5		
40-49	5.6		
50-59	7.3		4.4
60-69	6.3	4.1	4.4
70-79	8.5	3.8	4.4
80-89	9.1	4.3	5.8
>90	5.7	3.1	

remained unchanged. Three indentations were carried out on different sites, and the mean Brinell value was calculated.

The elasticity of bone is such that after impact a restitution to normal is so fast that the estimation of the indentation diameter can become jeopardized. For this reason the area to be tested was covered with a polyester pressure sensitive tape which together with its adhesive layer measured 0.06 mm (Minnesota Mining and Manufacturing Comp.). Thus the indentation in the bone could be maintained on the tape (Figures 4 a, 4 b) and measurements were made in a measuring microscope in two directions. Mean calculations were made.

In order to exclude the possibility of the tape deranging the true values from bone, tests were carried out on more homogenous organic materials such as oak, birch and fir with and without the tape. The values obtained showed no significant differences.

Statistical method: Student's T-test. One-way analysis of variance. (Statistical analysis = Ulf Runze, B.A.).

RESULTS

There was no significant difference in the Brinell values for the three indentations in each condylar area tested. In the normal group the hardness increased significantly (95 per cent level) with increasing age, see Table 2 and Figure 5. No difference in sexes was observed and this applied as well to the osteoarthritis and rheumatoid arthritis groups.

For the osteoarthritis and rheumatoid arthritis groups the hardness was significantly lower (99.9 per cent level) than in the normal state in corresponding age groups (see Table 2 and compare Figures 5 to 7). The mean difference in hardness values is seen in Table 3.

The tests on the lateral condyle in the normal group showed

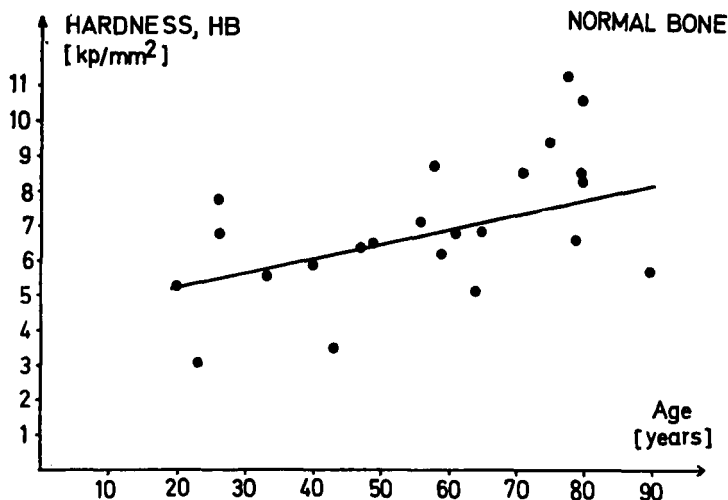


Figure 5. Hardness of normal subchondral bone of medial condyle of tibia in relation to increasing age. no. = 23.

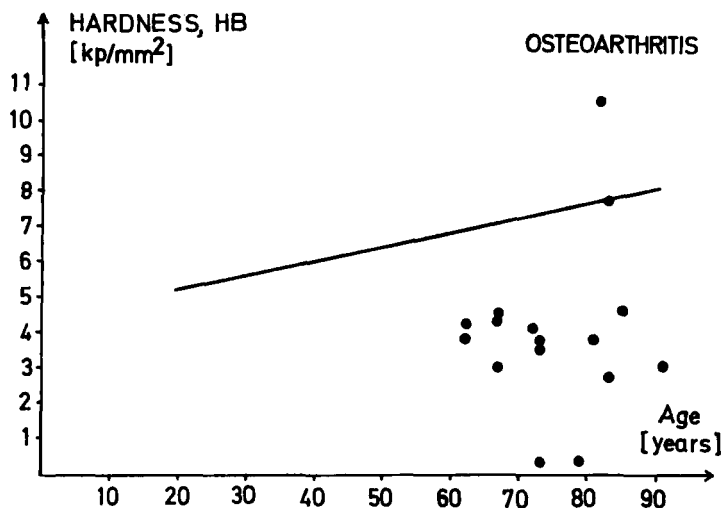


Figure 6. Hardness of subchondral bone of medial condyle of tibia in osteoarthritis. no. = 16. Regression line represents normal bone (cf. Figure 5).

significantly (99.9 per cent level) lower hardness values when compared to the medial condyles (Figure 8).

No conclusions can be made as regards the difference in hardness of the lateral condyle between each group because of the small number tested.

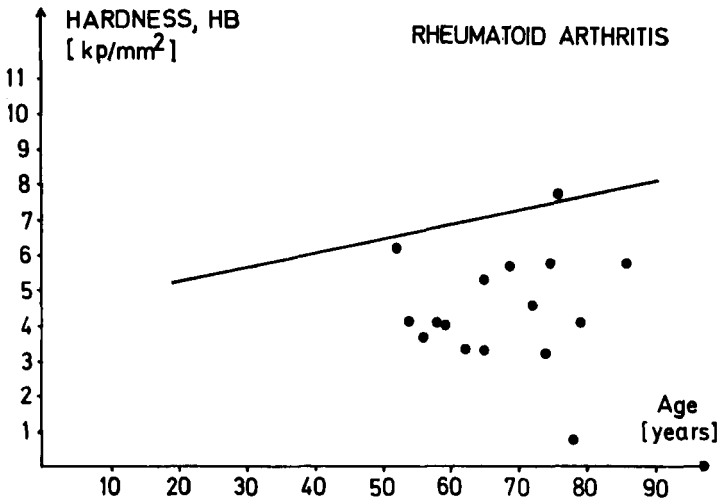


Figure 7. Hardness of subchondral bone of medial condyle of tibia in rheumatoid arthritis. no. = 16. Regression line represents normal bone (cf. Figure 5).

In the rheumatoid arthritis material no difference in hardness was observed between the steroid treated and non-steroid treated groups. The mean value for both groups remained the same and was 4.5 with $SD = 1.7$.

The duration of rheumatoid disease did not appear to have any influence on the hardness (Figure 9). In two cases, however, the duration could not be determined.

DISCUSSION

Hardness of bone is its capacity to resist the impact of a penetrating agent (Weaver 1966). According to Currey (1970) the hardness of bone varies with the strength, modulus of elasticity and the plastic flow

Table 3. Mean difference in hardness values (HB) for normal bone, and that in osteoarthritis and rheumatoid arthritis.

	Age	Normal	Osteoarthritis	Rheumatoid arthritis
HB	50-89 years	7.6 ± 2.2		4.5 ± 1.6
	60-91 years	7.6 ± 2.3	3.9 ± 2.5	

Difference at 99.9 per cent level between normal and rheumatoid arthritis and normal and osteoarthritis.

No statistical difference between osteoarthritis and rheumatoid arthritis.

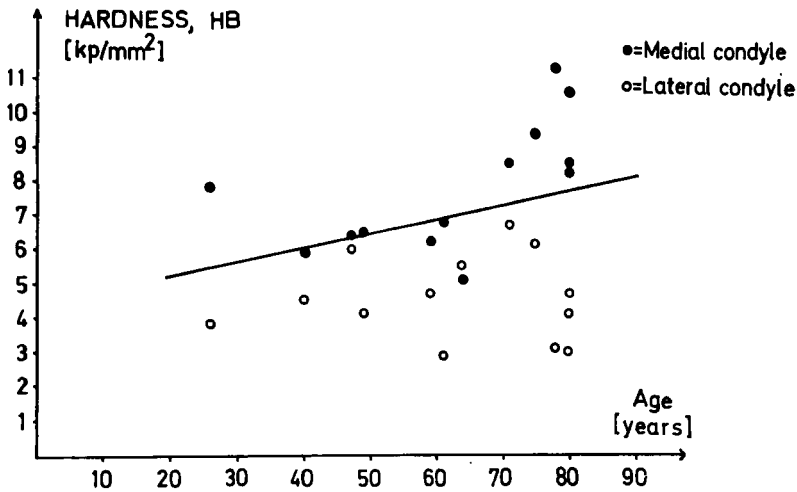


Figure 8. Comparison of hardness of normal subchondral bone in medial and lateral tibial condyles. Regression line represents the medial condyle (cf. Figure 5).

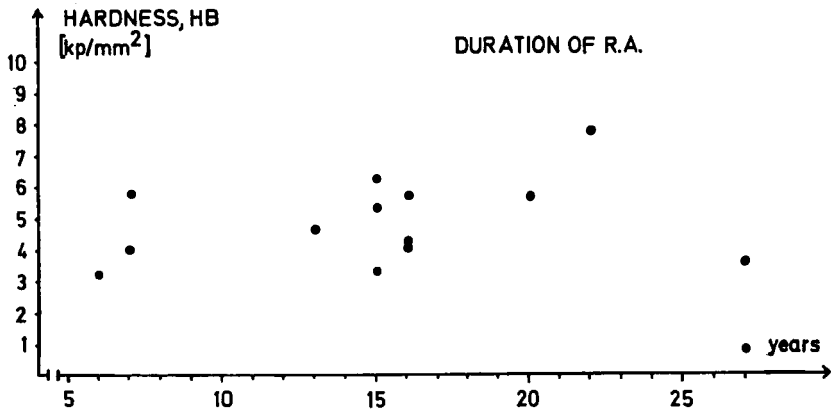


Figure 9. Hardness of subchondral tibial bone in relation to duration of rheumatoid process.

that bone can undergo. In 1954 Carlström published observations on microhardness of single Haversian systems. These showed a varying degree of mineralization (previously demonstrated by Amprini & Engström 1952), and the hardness of each Haversian system was directly related to the degree of mineralization. This was later confirmed by Amprino (1958). Weaver (1966) also determined the hardness of individual trabeculae of cancellous in comparison to cortical bone.

Besides the direct correlation between mineralization and hardness he also demonstrated that cancellous bone was slightly more pliable than cortical bone.

A factor influencing hardness is the number of osteons. Evans & Vincentelli (1969) have shown a strong positive correlation between hardness and the number of osteons/mm² in cortical bone. The same applies to trabecular bone (Weaver & Chalmers 1966).

Hardness decreases with age in cancellous bone (Evans 1961, Weaver & Chalmers 1966). No great difference exists between the two sexes below the age of fifty (Weaver & Chalmers 1966); over the age of fifty hardness and mineral content are significantly lower in women (Weaver & Chalmers 1966, Rockoff et al. 1969). The cancellous bone loses its hardness sooner than reduction of bone tissue occurs (Evans & King 1957, Bell et al. 1967).

The influence that disease has on bone hardness has been investigated by Weaver (1966). In *Paget's disease* the hardness differed at various sites and a good correlation was obtained with the histologic findings, i.e. severe changes—soft bone. In *osteogenesis imperfecta* the bone was so soft that no measurements could be made, and in *osteoporosis* hardness was not significantly affected. In *renal rickets* hardness was decreased in general, and most in the trabecular bone. The latter might be due to the finding of De Luca (1973) that the kidney acts as an endocrine organ for the production of the actual calcium regulating hormone (1,25 dihydroxy-vitamin D₃).

The difficulty connected with the testing of physical properties in cancellous bone is due to the lack of homogeneity in the trabecular structure, which e.g. aggravates testing conditions for tensile strength as gripping the cancellous structure is almost impossible without crushing it. Likewise difficulties arise in indentation tests as the removal of cartilage often results in serious intervention with the trabecular network. The subchondral bone of the tibial condyles enables the removal of the over-lying cartilage without interference with the trabeculae. Other reasons for choosing the medial tibial condyle was *that* it belongs to one of the more loaded joint systems of the body, *that* it most often becomes the site of osteoarthritis affecting the knee joint and *that*, according to theoretical calculations, the compression forces are about ten times larger medially than laterally. It moreover lends itself without too great technical difficulties to indentation tests and for these reasons the tibial condyles have been used in this investigation.

In our series the hardness increased in the normal material with advancing age. This is in opposition to previously published observations (Evans 1961, Weaver & Chalmers 1966) which pointed to a decrease with age. We have not been able to find a rational explanation of this difference. However, there is a difference in the technique: other authors have been testing the microhardness in single Haversian systems and single trabeculae which have a rather homogenous structure, while we were testing a network of trabeculae. Nevertheless, if one accepts the dependency of hardness on the degree of mineralization some investigations report in favour of hardness of bone increasing with age. In 1957 Robinson & Elliot could demonstrate that an increase of the mineral content occurred with age at the expense of the water content, leaving the content of organic material per volume unaltered. Later this was verified by Strandh & Nörlén (1965) who showed that the calcium content per unit weight or volume increased with age up to the 50th to 70th year and paralleled density. Furthermore Bergström & Bell (1954) demonstrated that the carbonate content of bone increased with age. Thus, there is evidence that mineralization can augment with advancing age and consequently the hardness of bone should increase as the data in our normal material suggest. In our further studies of this material, consideration will be taken of the degree of mineralization.

There is a difference between the normal group and the osteoarthritis and rheumatoid arthritis groups. The diminished hardness of the trabecular bone in the latter groups might be ascribed to the patho-morphologic features. In osteoarthritis it is assumed that one of the first changes in the subchondral bone is microfractures (Trueta 1963), which might alter the peak transmission force and also the absorption of energy leading to an increased demand on the cartilage, which ultimately loses its capacity to handle the forces placed on it and becomes destroyed (Radin et al. 1970, Radin 1972).

With the osteolyses of the subchondral bone in rheumatoid arthritis a situation analogous to the osteoarthritis process might arise thus contributing to the cartilage loss, which also can be caused by enzymatic or other chemical reactions. It is interesting to note that neither age of patient nor duration of disease seems to influence the hardness. To further complete this investigation a histological analysis is under way of the material used for the hardness tests.

The tests made on the lateral condyles showed lower hardness in comparison to the medial condyles in the normal group (Figure 8). This is in accordance with the estimations by Kettelkamp & Chao

(1972) that the compression forces on the tibial plateaus are about ten times greater on the medial condyle than on the lateral.

SUMMARY

A comparison of the hardness of subchondral, trabecular bone has been carried out in normal bone, and that in osteoarthritis and rheumatoid arthritis. It was found that with increasing age the hardness of subchondral bone increased in the normal group irrespective of sex. The increase is statistically significant.

The hardness of subchondral bone in osteoarthritis and rheumatoid arthritis was significantly lower than in normal bone.

There was no difference between osteoarthritis and rheumatoid arthritis.

In the normal group the hardness values for the medial condyles were significantly higher than for the lateral condyles.

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