

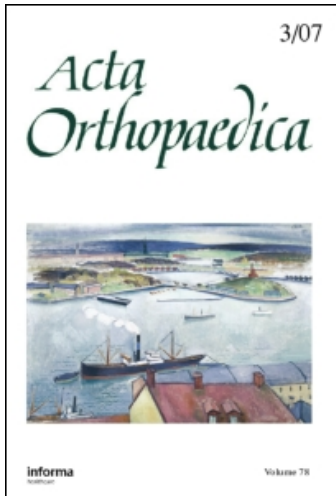
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Microfractures in coxarthrosis

Barbara Koszyca, Nicola L. Fazzalari and Barrie Vernon-Roberts

We examined the subchondral bone architecture of the femoral head in relation to trabecular microfracture. Three groups of femoral head specimens were studied. Twenty-eight specimens taken during hip replacement had grade III or IV arthrosis (70 ± 8 years). From autopsy, 40 femoral heads were obtained, 18 in a group greater than 50 years of age (72 ± 10 years) and 22 in a group less than 50 years of age (25 ± 11 years). None of these 40 heads had worse than grade II arthrosis. Coronal slices of the femoral heads were macerated and examined under a dissecting microscope to count trabecular microfractures. For bone histomorphometry, blocks were taken from the subchondral principal compressive and tensile trabeculae. The bone volume, trabecular thickness, and marrow space were quantified. In the subchondral principal compressive region, the arthrotic group had more bone volume, thicker trabeculae, similar trabecular space, and trabecular microfractures when compared with the group greater than 50 years old. In the tensile region, there were no differences except for decreased trabecular microfracture number in the arthrotic group. With the thinnest trabeculae in the compressive region occurring in the greater than 50 years old group, the trabeculae of the younger age group have thinned with age, but with the onset of arthrosis, the thinning is overtaken by pathologic thickening of trabeculae.

Detailed studies of the spatial structure of the cancellous bone in the hip joint have been performed in osteoarthritis and controls (Fazzalari et al. 1983, 1985). The bone was found to be structurally remodeled in arthrosis such that the trabecular patterns may not be transformed back to normal. Moreover, Fazzalari et al. (1987) have suggested that trabecular microfracture may maintain joint normality, and that the reduction in trabecular microfracture numbers seen in arthrosis contributes to joint deterioration.

We have studied the possible role of trabecular microfracture in the pathogenesis of arthrosis (Radin et al. 1972). The spatial structure of the subchondral bone and trabecular microfracture incidence in the femoral head were studied in arthrosis and in normal young and old controls.

Material and methods

The surgical and autopsy femoral heads were macroscopically graded for arthrosis according to Collins (1949).

Grade I arthrosis has destruction of the superficial cartilage confined to areas of greatest pressure and movement. Early fibrillation of the articular cartilage is detected and no hyperplasia of bone or cartilage is evident. The articular contour is normal.

Grade II arthrosis has more extensive destruction of cartilage, but is still confined to areas of greatest pressure and movement. Deeper fibrillation of areas affected in Grade I is present. There are no osteophytes and no marked remodeling of the articular contour.

Grade III arthrosis has total loss of cartilage on one or more pressure areas with exposure and usually eburnation of bone. Widespread fibrillation is evident, though regions of unaffected cartilage often survive in parts least subject to pressure and movement. Marked bone changes are present, such as osteophytes at the joint margins, alterations of the articular contour, and sclerosis of the subchondral bone.

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Grade IV arthrosis has complete loss of hyaline cartilage from large areas of the joint surface and eburnation of exposed bone. The articular surface has gross unevenness from surviving islets of cartilage, epiarticular osteophytes, and remodeling of the articular contour. Prominent marginal osteophytes are also a feature.

The arthrosis group consisted of 28 consecutive heads of femur, with primary arthrosis Grade III or IV, taken during hip replacement from 16 females and 12 males (aged 70 ± 8 years, mean \pm SD).

From autopsy, 40 femoral heads from 22 males and 18 females (aged 50 ± 25 years) were obtained; 19 cases (aged 37 ± 24 years) had died from accident, and 21 (aged 62 ± 20 years) had succumbed from a variety of conditions, including diabetes, myocardial infarction, and tumors. None of the femoral heads had worse than Grade II arthrosis.

The wide disparity in age between the control and the arthrosis groups, coupled with the knowledge of the exponential relationship between age and the number of healing trabecular microfractures (Fazzalari et al. 1987), led to the distinction between femoral heads over and under 50 years of age (n 18, aged 72 ± 10 years and n 22, aged 25 ± 11 years).

All the femoral heads were sliced coronally. The slices were macerated except for the end slices, which consisted mainly of cortical bone. The macerated slices were examined using a binocular dissecting microscope at $25\times$ magnification, and the trabecular microfractures in the principal compressive and tensile subchondral areas were marked on an overlying sheet of clear acetate. Trabecular microfractures were considered to exist where callus formation was evident with well-rounded, discrete nodules up to 0.5 mm in diameter or smooth swellings surrounding the trabecular (Koszyca et al. 1989).

The central coronal slice was taken for histologic processing of selected blocks. A contact radiograph of the 5-mm coronal slice was taken using a faxitron system (Hewlett-Packard Co. McMinnville, OR, USA). Thus, from the coronal radiograph, we were able to obtain autopsy and surgical femoral blocks from reproducible locations. The principal compressive block was taken proximal to the fovea and the principal tensile block distal to the fovea. Both blocks extended to the edge of the leash trabecular region where the compressive and tensile trabeculae overlap (Figure 1).

Thin $10\text{-}\mu\text{m}$ sections for histoquantitation were impregnated with silver by the von Kossa technique to render calcified bone black and counterstained by van Gieson's technique. Epiarticular osteophytes

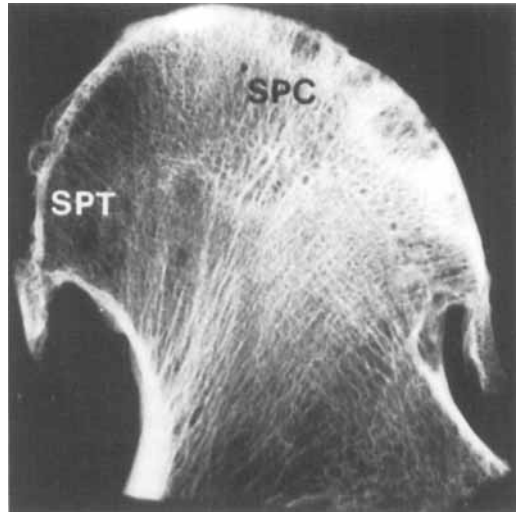


Figure 1. Radiograph of the central coronal slice of the femoral head showing the subchondral principal compressive (SPC) and tensile (SPT) zone examined by quantitative histomorphometry. These zones also represent the areas in adjacent slices examined for the presence of trabecular microfractures

that may have been present in blocks taken from the principal tensile region were not quantified. This was possible because the original articular contour and subchondral bone plate were identifiable in these sections. The parameters studied were percentage mineralized bone volume, trabecular thickness, and spacing. The slides were analyzed by a Quantimet Image Analysing computer (Cambridge Instruments Ltd, UK) as described by Fazzalari et al. (1983).

Where the sample numbers differ from the number of collected specimens, this occurs because of technical failures in tissue processing and/or specimen cutting.

The nonparametric Mann-Whitney test was used for comparison of unpaired data, and the Wilcoxon signed rank test for paired data.

Results

In all the groups the bone volume and trabecular thickness were higher in the principal compressive region compared with the principal tensile region, and trabecular spacing in the compressive region

was less in all the groups (Table 1). Trabecular microfractures were more numerous in the compressive region compared with the tensile region in the young controls, but not in arthrosis and old controls (Table 2).

In the principal compressive region, the arthrosis group had more bone volume, thicker trabeculae, similar trabecular spacing, and microfractures when compared with old controls. Compared with arthrosis and the old controls, the young controls had similar bone volume and trabecular spacing. The young controls had thicker trabeculae and less trabecular microfractures than the old controls, whereas the arthrosis group had thicker trabeculae than the young controls (Table 2).

In the principal tensile region, the arthrosis group has fewer trabecular microfractures, but no other difference from the old controls despite the trend towards more widely spaced trabeculae in arthrosis. On the other hand, the young had a higher bone volume and thicker and more closely spaced trabecular architecture than the old (Table 2).

Discussion

A possible role for trabecular microfractures in the pathogenesis of osteoarthritis was first outlined by Radin et al. (1972). They hypothesized that repetitive impulse loading of a joint results in trabecular microfractures and subsequent subchondral bone remodeling. This remodeled bone is stiffer and thus reduces the protection of the cartilage to impulse loading and leads to the ultimate failure of the articular cartilage.

The problem with this hypothesis is that it does not necessarily follow that the remodeling bone will be less compliant. It is possible, but unlikely, that the presence of substantial amounts of microcallus on healing trabecular microfractures could be sufficient to increase bone stiffness. Radin and his colleagues (Radin et al. 1970, 1972, 1973, Radin 1982) do not distinguish between increased bone stiffness due to the presence of microcallus on trabecular microfractures or trabeculae that have been remodeled as an end result of microfractures. The study of Radin et al. (1973), which correlated trabecular microfractures with increased bone stiffness, argues against bone remodeling as the cause of increased bone stiffness, because the identification of healing microfractures indicates that the trabeculae are still in their original location. This assumes that bone, still in the process of being remodeled, is already less compliant.

Table 1. Histoquantitative and trabecular microfracture (F) data for subchondral bone in the compressive and tensile regions of the femoral head

Variable	Group	n	Min	25th percentile	Median	75th percentile	Max
Subchondral principal compressive region							
M	L	20	20	25	27	31	39
	G	18	9.2	20	23	30	36
	A	28	18	25	29	36	59
T	L	20	0.01	0.09	0.11	0.13	0.15
	G	18	0.05	0.08	0.09	0.10	0.16
	A	28	0.07	0.11	0.13	0.14	0.21
S	L	20	0.21	0.26	0.30	0.33	0.49
	G	18	0.21	0.26	0.30	0.37	0.54
	A	28	0.15	0.22	0.31	0.36	0.45
F	L	20	0.0	0.0	0.5	1.5	3.0
	G	18	0.0	1.0	2.5	6.0	33.0
	A	28	0.0	0.0	2.0	5.5	14.0
Subchondral principal tensile region							
M	L	22	10	17	20	23	29
	G	18	5.2	8.4	13	17	29
	A	23	3.0	7.7	11	13	32
T	L	22	0.06	0.07	0.09	0.11	0.14
	G	18	0.04	0.06	0.07	0.08	0.10
	A	23	0.05	0.06	0.07	0.08	0.14
S	L	22	0.27	0.33	0.38	0.42	0.52
	G	18	0.25	0.37	0.58	0.62	1.22
	A	23	0.26	0.44	0.55	0.80	3.00
F	L	22	0.0	0.0	0.0	0.0	3.0
	G	18	0.0	2.0	2.5	6.0	25.0
	A	23	0.0	0.0	1.0	4.0	9.0

M mineralized bone volume (percentage)
 T trabecular thickness (mm)
 S trabecular spacing (mm)
 F trabecular microfracture
 L less than aged 50 years
 G greater than aged 50 years
 A arthrosis

Table 2. Comparison of histoquantitative parameters between femoral subchondral regions and study groups

	Group	M	T	S	F
SC vs ST	L	SC > ST ^a	SC > ST ^b	SC < ST ^a	SC > ST ^b
	G	SC > ST ^a	SC > ST ^a	SC < ST ^a	NS
	A	SC > ST ^a	SC > ST ^a	SC < ST ^a	NS
A vs L	SC	NS	A > L ^c	NS	A > L ^a
	ST	A < L ^a	A < L ^a	A > L ^a	A > L ^a
A vs G	SC	A > G ^b	A > G ^a	NS	NS
	ST	NS	NS	NS	A < G ^c
L vs G	SC	NS	L > G ^c	NS	L < G ^b
	ST	L > G ^a	L > G ^a	L < G ^a	L < G ^a

^a $P > 0.001$, ^b $P < 0.01$, ^c $P < 0.05$

M, T, S, F, L, G, and A see Table 1

SC subchondral principal compressive region

ST subchondral principal tensile region

The nature of the relationship between cartilage state and trabecular microfracture is complex. A number of studies have investigated the role of bone stiffness in relation to cartilage damage (Pedley and Meachim 1979, Pugh et al. 1974, Radin et al. 1970, 1972, 1973, Radin 1982); and it would appear that in the results of Radin (1982), the bone underlying degenerate cartilage is associated with a relative scarcity of trabecular microfractures. The bone underlying cartilage in good condition contains a large proportion of the total number of trabecular microfractures (Fazzalari et al. 1987). Predictably, the bone underlying healthy cartilage consists of a network of thinner trabeculae more susceptible to fatigue fracture. Stiffer bone consists of highly interconnected trabeculae providing a mechanically strong network resistant to deforming force and microfracture (Pugh et al. 1974).

In the principal compressive region, the young controls had trabeculae that were thicker and had fewer microfractures. This suggests that under the age of 50 years, the bone is more resistant to deforming forces with reduced numbers of trabecular microfractures. The number of trabecular microfractures for the arthrosis and old groups were not different, though the former has more bone volume and thicker trabeculae. This suggests that the spatial disposition of the trabecular bone elements is an important factor in determining bone stiffness and trabecular fracture.

Our results suggest that trabecular microfractures do not contribute to the onset or progression of arthrosis (Fazzalari et al. 1987). The largest number of trabecular microfractures occurred in the control groups with minimal primary arthrosis, only a small proportion of which seem to progress to the late stage and more severe forms of the disease (Collins 1949, Byers et al. 1970).

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