ELSEVIER

Bone Vol. 27, No. 3
September 2000:351–357

Exercise-induced Bone Gain Is Due to Enlargement in Bone Size Without a Change in Volumetric Bone Density: A Peripheral Quantitative Computed Tomography Study of the Upper Arms of Male Tennis Players

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Bilateral bone characteristics of the humerus (proximal, shaft, and distal sites) and radius (shaft and distal sites) in 12 former Finnish national-level male tennis players (mean age 30 years) and their 12 age-, height-, and weight-matched controls were measured with peripheral quantitative computed tomography (pQCT). The pQCT variables analyzed were bone mineral content (BMC), total cross-sectional area of bone (Tot.Ar), cross-sectional area of the marrow cavity (M.Cav.Ar), cortical bone (Co.Ar) and trabecular bone (Tr.Ar), volumetric density of cortical (Co.Dn) and trabecular (Tr.Dn) bone, cortical wall thickness (Co.Wi.Th), bone strength index (BSI), and principal moments of inertia (I_{min} and I_{max}). In the players, significant side-to-side differences, in favor of the dominant (playing) arm, were found in BMC (ranging 14%–27%), Tot.Ar (16%–21%), Co.Ar (12%–32%), BSI (23%–37%), I_{min} (33%–61%), and I_{max} (27%–67%) at all measured bone sites, and in Co.Wi.Th. (5%–25%) at the humeral and radial shafts, and distal humerus. The side-to-side M.Cav.Ar difference was significant at the proximal humerus (19%) and radial shaft (29%). Concerning the players' Co.Dn and Tr.Dn, the only significant side-to-side difference was found in the Co.Dn of the distal humerus, with the playing arm showing a slightly smaller Co.Dn than the nonplaying arm (−2%). In controls, significant dominant-to-nondominant side differences were also found, but with the majority of the differences being rather small, and significantly lower than those of the players. In conclusion, despite the large side-to-side differences in BMC, the volumetric bone density (Co.Dn, Tr.Dn) was almost identical in the dominant and nondominant arms of the players and controls. Thus, the players' high playing-arm BMC was due to increases in the Tot.Ar, M.Cav.Ar, Co.Ar, and CW.Th. In other words, the playing arm's extra bone mineral, and thus increased bone strength, was mainly due to increased bone size and not due to a change in volumetric bone density. These upper arm results may not be generalized to the entire skeleton, but the finding may give new insight into conventional dual-energy X-ray absorptiometry (DXA)-based bone density measurements when interpreting the effects of exercise on bone. (Bone 27:351–357; 2000)

Key Words: Osteoporosis; Physical activity; Bone density; Bone geometry; Peripheral quantitative computed tomography (pQCT); Tennis.

Introduction

Previous studies of tennis players[10,18,19,23,24,31] have shown large side-to-side differences in bone mineral content (BMC) and density (BMD) between the dominant and nondominant arms, but the potential changes in bone size and geometry have remained largely unclear. In some studies, bone mineral "apparent" density (BMAD, in grams per cubic centimeter), bone geometry (bone width, cortical wall thickness [CW.Th]), and strength (cross-sectional moment of inertia [CSMI]) have been estimated from dual-energy X-ray absorptiometry (DXA) data by applying simple engineering formulas and assuming the shape of the particular bone.[6,7,19,25,40] These studies have provided tentative evidence that exercise and loading may not only promote bone mineral acquisition but also changes in bone geometry, especially if the activity is started during childhood and adolescence.[6,19] Because final bone strength is also closely related to bone size, these changes in size, if permanent, could reduce fracture risk in later life.

DXA constitutes the basis for the evaluation of bone mineral status of the skeleton and is currently considered the method of choice in both the clinical practice and bone research.[13] Despite the indisputable benefits of DXA (excellent precision, accuracy, short examination time, low radiation exposure, ability to predict fracture risk), the measurements are somewhat restricted by their planar nature. The primary variable of the DXA measurement is the areal BMD (in grams per square centimeter). However, this variable is neither the true material nor volumetric density of the bone, but rather represents a combination of bone size and volumetric density.[29,37] Moreover, DXA is unable to separate cortical bone from trabecular bone, components that are known to respond differently to aging, diseases, loading, and medication.[5,33,34] Augat et al.[3] recently suggested that the fracture load of the distal radius and the

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8756-3282/00/$20.00
PII S8756-3282(00)00331-8

Table 1. Characteristics of subjects [mean (SD)][a]

Characteristic	Players (N = 12)	Controls (N = 12)
Age (yr)	29.8 (4.8)	29.8 (5.2)
Height (cm)	181.0 (4.4)	180.7 (7.7)
Weight (kg)	72.2 (11.8)	76.1 (5.6)
Isometric grip strength (kg)		
Dominant	38.4 (4.3)	38.8 (7.8)
Nondominant	32.3 (4.6)	34.5 (5.7)
Isometric forearm flexion (kg)		
Dominant	29.8 (5.3)	30.7 (8.2)
Nondominant	28.2 (4.8)	30.1 (6.5)
Isometric forearm extension (kg)		
Dominant	20.8 (3.3)	20.7 (4.2)
Nondominant	19.7 (2.8)	20.7 (3.4)
Training history		
Years of playing (yr)	19.6 (5.3)	—
Starting age of playing (yr)	9.8 (3.0)	—
Training sessions per week, previous (1992)	5.3 (3.8)	—
Training sessions per week, current	2.7 (1.8)	—
Duration of each session (min), previous (1992)	81.7 (18.5)	—
Duration of each session (min), current	67.5 (26.0)	

[a]No significant differences between players and controls.

proximal femur could best be predicted by a measurement of the geometric properties of the cortical shell.

Due to the aforementioned methodological constraints, assessment of bone size and geometry from the planar DXA data is not possible and thus the DXA-based estimates of bone strength are only indicative.[38] On the other hand, it clear that, in most cases, there is a strong association between BMC (grams) and ultimate bone strength. This association probably arises from the fact that, if the bone is healthy and grown and loaded in an appropriate manner during the growth of the body, the building material (BMC, grams) is likely to become distributed in an ideal way to build a large, strong bone. Concern about bone size and geometry, however, becomes relevant with patients of unusual size (either small or large), especially in situations where the bone size or geometry may change during the study, such as in studies of growing children or in those evaluating the effects of different exercise regimens on bone.[6,7,14,15–17,19,25,39,40]

The purpose of this study is to evaluate the characteristics of the upper extremity bones in male tennis players and their sedentary controls using peripheral quantitative computed tomography (pQCT).[38] We were especially interested in determining whether there were differences in bone size and geometry between the loaded and nonloaded arms of the players.

Materials and Methods

Subjects

Twelve former Finnish national top-level male tennis players and 12 corresponding controls participated in the study (**Table 1**). All subjects were clinically healthy nonsmokers and without disease or medication known to affect bone metabolism, and none of them had past upper extremity fractures. The mean age (±SD) of players was 30 ± 5 years. The players had started their playing careers during childhood (mean starting age 10 ± 3 years) and had been playing tennis for >19 years, on average. At the time of the pQCT measurements, all players had been retired from top-level tennis for 1.5–3 years, but were still playing recreationally 2.7 ± 1.8 times per week (Table 1). The controls were age-, height-, and weight-matched healthy volunteers who were not involved in physical activity or work affecting dominant extremity only. All subjects were informed of the study procedure, purposes, and known risks, and all gave their informed consent. The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the ethics committee on human research of our institute.

Study Protocol

Information about training and medical history of each subject was obtained by response to a mailed questionnaire that included questions about lifetime history of physical activity, possible special diets, vitamin or mineral supplementation, medication, consumption of alcohol, known diseases, and past injuries. The questionnaire was reviewed and checked with an interview at the time of the measurements. Height and weight were measured with all subjects wearing indoor clothing, without shoes.

The bone characteristics at three different sites of the humerus and two sites of the radius were measured with a Norland/Stratec XCT-3000 pQCT scanner (Stratec Medizintechnik GmbH, Pforzheim, Germany) according to our standard procedures.[38] In short, the tomographic slices (2.5 mm) of humerus were taken at 80% (proximal humerus), 50% (humeral shaft), and 20% (distal humerus) of the approximate humeral length (0.186 × subject height), proximal to the proximal endplate of the radius. The radial slices (2.5 mm) were taken at 4% (distal radius) and 30% (radial shaft) of the approximated radial length (0.146 × subject height), proximal to the distal endplate of the ulna.[38]

The bone variables used for the analyses were the bone mineral content of the given bone section (BMC, mg); volumetric trabecular and cortical density (Tr.Dn and Co.Dn, mg/mm^3); cross-sectional areas of the total bone (Tot.Ar, mm^2), cortical bone (Co.Ar, mm^2), trabecular bone (Tr.Ar, mm^2), and marrow cavity (M.Cav.Ar, mm^2); (CW.Th, mm); density-weighed polar section modulus as a bone strength index (BSI, mm^3); and principal moments of inertia (I_{min} and I_{max}, mm^4). The BSI is defined as the density-weighed polar section modulus of given bone cross section.[38] Given a low proportion of trabecular bone at the other measurement sites, and therefore poor precision of the trabecular measurements (Sievänen 1999, personal communication), trabecular bone variables (i.e., Tr.Ar and Tr.Dn) were assessed from the distal radius only. In our laboratory, precision (root-mean-square coefficient of variation, CVrms) of the applied pQCT measurements are shown to range from 1.1% to 7.5% at the proximal humerus, 0.5% to 5.6% at the

humeral shaft, 1.0% to 2.4% at the distal humerus, 0.8% to 4.3% at the radial shaft, and 2.2% to 7.6% at the distal radius.[38]

Statistical Analysis

The data were analyzed using version 4.53 of the StatView statistical package (Abacus Concepts Inc., Berkeley, CA). The side-to-side evaluation between the dominant and nondominant arms was performed with a paired *t*-test. In the comparison of the absolute bone values, relative side-to-side differences, and the background characteristics between the players and controls, a nonpaired *t*-test was used. In the comparison of the absolute bone values between players and controls, body weight was used as a covariate. In all tests, an α-level of $<5\%$ was considered significant.

Results

Characteristics of the study subjects are given in Table 1. There were no significant intergroup differences in age, height, weight, or isometric strength measurements.

The crude pQCT data in players and controls are shown in **Table 2**, and the percentage side-to-side differences between the players and controls are presented in **Table 3**. In the players, significant side-to-side differences, in favor of the dominant arm, were found in BMC (14.2% to 27.3%), Tot.Ar (15.8% to 21.3%), Co.Ar (11.5% to 31.9%), BSI (22.5% to 36.9%), I_{min} (33.4% to 60.8%), and I_{max} (27.1% to 67.0%) at all measured bone sites, and in CW.Th (4.5% to 24.6%) at the humeral and radial shafts and distal humerus. The side-to-side difference in M.Cav.Ar was significant at the proximal humerus (18.5%) and radial shaft (28.6%). Regarding the players' volumetric density, the only significant but marginal side-to-side difference was found in the Co.Dn of the distal humerus (-2.1%). In controls, significant side-to-side differences were only found at the humeral shaft (M.Cav.Ar 8.7%, Co.Ar 3.4%, BSI 9.9%, and I_{max} 16.1%); distal humerus (BMC 5.2%, Co.Ar 6.2%, CW.Th 4.6%, BSI 11.2%, and I_{min} 16.3%); radial shaft (I_{max} 6.7%); and distal radius (BMC 5.1%, Co.Ar 6.3%) (Table 2). These controls' percentage side-to-side differences were, however, significantly smaller than those of the players among the majority of the humeral (proximal, shaft, distal) and radial shaft variables (Table 3). In the distal radius, the interindividual variability in the side-to-side differences of the geometric variables was large, and therefore, despite the large differences in the mean data of the players and controls, a statistically significant intergroup difference was reached in BMC only (Table 3).

The geometric adaptation of bones in the dominant arm of the players seemed to be site-specific. At all measured sites, the total cross-sectional area was larger on the dominant side (Table 2), whereas the proportions of cortical and marrow cavity areas varied substantially according to the measured bone site (**Figures 1** and **2**): At the proximal humerus and radial shaft, the side-to-side differences were significant in the total, cortical, and marrow cavity area—a finding indicating that, at these sites, the bone of the dominant arm had expanded, also increasing the area of the marrow cavity, whereas the cortical wall thickness changed only slightly, if at all. At the humeral shaft and distal humerus, in turn, the size of the marrow cavity was almost similar in both arms, whereas the cortical bone area and cortical wall thickness were increased significantly (Figures 1 and 2).

Discussion

Given the evidence from numerous animal and human studies, there is no doubt that mechanical loading can have positive effects on bone by increasing the BMC and areal BMD[9,11,12] and the failure load of the loaded bones.[21,32] However, the effects of loading on the true volumetric density of bone as well as bone geometry have remained largely unknown because of the lack of reliable and safe measurement techniques. Recently, several pQCT devices have been introduced for examining peripheral bone sites with a significantly lower radiation dose than with conventional CT equipment.[13,26,28,38] In contrast to the commonly used DXA method, pQCT allows discrimination between the trabecular and cortical components of bone, and gives precise information on the actual cross-sectional geometry of the bone.

In this pQCT study of male tennis players, known to have large side-to-side differences in BMC between dominant and nondominant arms,[10,19,22–24,31] we showed that the additional bone mineral in the dominant arm was mainly used for increasing the bone size, not the volumetric density of the cortical or trabecular bone. In fact, when the absolute values of the cortical and trabecular densities were compared, they seemed to be virtually constant between players and controls, and across the bone sites, except the distal radius. This finding is similar to other CT studies where the volumetric density of cortical and trabecular bone has been shown to be fairly similar between children at different ages, and between men and women,[2,15–17,36] and where the higher areal bone mineral density and ultimate bone strength in older children and men were due to larger bone size, not volumetric density of bone. In contrast to our study, Rico et al.[33] found a significantly larger total and cortical volumetric density, but not trabecular density of the dominant distal radius, compared with the nondominant counterpart of sedentary right-handed adult subjects. Despite the fact that pQCT was also used in their study, unfortunately only one bone site and no variables other than volumetric bone density was reported.

The importance of examining bone dimensions arises from recent findings suggesting that the effects of mechanical loading on bone may be accentuated during growth, with these effects manifesting as not only increased peak bone mass but also changes in skeletal geometry.[6,19,27,35] In his preliminary study of 14 healthy children, Schönau[35] suggested that the volumetric bone density of trabecular and cortical bone in the nondominant distal radius was unrelated to age and grip strength, and adaptation with age (also with natural increase in grip strength) occurred as changes in bone geometry (cross-sectional area, cortical area). The effects of physical activity on bone geometry were not investigated in Schönau's work, but the study provided preliminary evidence that, during childhood, the most important adaptation mechanism to biomechanical usage might be the change in bone geometry. Because the failure load of bone depends largely on bone size, adaptations in bone geometry during growth, if permanent, could have a significant impact on mechanical competence of bone in later life. Parfitt[30] postulated that the period of longitudinal growth is the only time in life when bone may be added substantially on both the inner (endosteal) and outer (periosteal) sides of bone, with endosteal accumulation fading away thereafter. As a result, a bone with more mineral and thicker walls develops, which, in turn, significantly increases bone strength and provides protection against cortical bone loss, which usually takes place at the endocortical surface of the bone (e.g., due to disuse atrophy or age-related bone loss).[4,8]

In our study, there was a rather consistent pattern of bone values between arms of the subjects. At all skeletal sites, the bone values seemed to be greatest at the dominant arm of the players, followed by the dominant arm of the controls, the nondominant arm of the controls, and finally the nondominant arm of the players, which seemed to be "the weakest of all" (Table 2). One explanation for this may be that, among these four groups of upper extremities, the players' nondominant arm was the least used, and thus the weakest, as can be seen from the isometric strength measurements (Table 1). Most of the players studied had not been involved in any physical work, or any sports other than tennis since they started playing at

Table 2. Results of the peripheral quantitative computed tomography (pQCT) measurements from the dominant and nondominant arms of male tennis players and their sedentary controls [mean (SD)]

	Players (N = 12)			Controls (N = 12)		
Measured variable[a]	Dominant	Nondominant	Side-to-side difference (%)[b]	Dominant	Nondominant	Side-to-side difference (%)[b]
Proximal humerus						
BMC (mg)	265.3 (25.0)[c]	218.8 (21.4)[c]	21.7 (9.4)[f]	250.1 (30.7)	247.6 (28.0)	0.9 (3.1)
Tot.Ar (mm^2)	558.0 (81.5)	490.5 (78.5)	15.8 (12.4)[e]	502.4 (100.5)	500.1 (90.6)	0.5 (6.7)
M.Cav.Ar (mm^2)	371.7 (93.0)	320.3 (83.9)	18.5 (21.8)[d]	300.9 (115.8)	306.5 (103.3)	−1.6 (14.3)
Co.Ar (mm^2)	185.6 (32.1)	167.0 (20.1)	11.7 (13.2)[d]	195.2 (32.4)	187.3 (29.0)	4.4 (9.3)
Co.Dn (mg/cm^3)	1004.6 (30.0)	1017.1 (23.3)	−1.2 (1.9)	1017.9 (34.5)	1014.7 (22.4)	0.3 (2.8)
CW.Th (mm)	2.5 (0.5)	2.4 (0.4)	4.1 (14.9)	2.9 (0.7)	2.7 (0.6)	5.4 (13.9)
BSI (mm^3)	1428.0 (268.3)	1172.0 (187.0)	22.5 (17.4)[e]	1378.6 (159.8)	1292.2 (207.2)	8.2 (14.5)
I_{min} (mm^4)	10,635.0 (3019.6)	8580.2 (2893.9)	33.4 (37.6)[d]	9017.7 (2997.3)	8771.3 (3214.7)	6.6 (25.8)
I_{max} (mm^4)	14,672.3 (2247.6)[c]	11,780.7 (2059.5)	27.1 (23.0)[e]	14,118.6 (2032.2)	13,392.8 (2452.4)	6.4 (10.8)
Humeral shaft						
BMC (mg)	348.7 (31.7)[c]	281.0 (31.0)	24.6 (8.5)[f]	313.8 (35.4)	305.1 (26.9)	2.7 (4.7)
Tot.Ar (mm^2)	417.5 (51.6)	356.3 (44.1)	17.4 (7.6)[f]	391.4 (40.2)	373.9 (30.8)	4.6 (6.1)
M.Cav.Ar (mm^2)	125.7 (41.6)	124.2 (29.1)	0.8 (16.1)	126.1 (28.4)	117.1 (29.4)	8.7 (10.2)[d]
Co.Ar (mm^2)	291.9 (27.7)[c]	232.2 (26.1)	26.3 (9.8)[f]	263.3 (33.1)	254.2 (25.0)	3.4 (1.4)[d]
Co.Dr (mg/cm^3)	1093.7 (17.2)	1096.1 (31.1)	−0.2 (2.2)	1100.0 (21.4)	1103.7 (25.0)	−0.3 (1.1)
Co.Wi.Th (mm)	5.3 (0.5)	4.4 (0.4)	20.2 (10.5)[f]	4.8 (0.6)	4.8 (0.6)	0.4 (3.1)
BSI (mm^3)	1649.7 (270.6)[c]	1247.3 (232.7)	33.7 (16.4)[f]	1506.9 (214.7)	1372.5 (166.0)	9.9 (10.8)[e]
I_{min} (mm^4)	8984.6 (3652.7)	6323.9 (2460.6)	46.4 (36.9)[f]	7506.2 (2800.7)	7120.6 (2231.8)	5.2 (31.2)
I_{max} (mm^4)	14,968.1 (2278.3)	10,954.2 (2312.2)	39.0 (17.4)[f]	13,646.2 (1847.5)	11,782.0 (1350.8)	16.1 (12.4)[e]
Distal humerus						
BMC (mg)	349.0 (32.1)[c]	275.0 (2.62)[c]	27.3 (10.1)[f]	321.0 (31.2)	304.8 (22.8)	5.2 (4.3)[e]
Tot.A (mm^2)	379.5 (39.6)[c]	313.9 (34.7)[c]	21.3 (9.7)[f]	361.0 (34.8)	345.1 (25.3)	4.6 (7.0)
M.Cav.A (mm^2)	88.2 (19.5)	92.3 (22.6)	−3.3 (12.7)	95.6 (21.0)	95.9 (25.1)	1.0 (13.2)
Co.Ar (mm^2)	291.3 (29.1)[c]	222.2 (23.2)[c]	31.9 (11.6)[f]	265.1 (29.8)	249.6 (21.7)	6.2 (5.9)[e]
Co.Dn (mg/cm^3)	1106.1 (25.8)	1130.5 (24.5)	−2.1 (1.7)[e]	1112.2 (22.5)	1118.1 (24.2)	−0.5 (1.5)
CW.Th (mm)	5.7 (0.4)[c]	4.6 (0.4)	24.6 (8.8)[f]	5.2 (0.6)	5.0 (0.6)	4.6 (4.3)[e]
BSI (mm^3)	1279.4 (181.3)	943.0 (159.3)[c]	36.9 (14.2)[f]	1216.1 (146.2)	1095.4 (116.1)	11.2 (9.4)[e]
I_{min} (mm^4)	6827.7 (1156.1)	5049.9 (1025.1)	37.4 (21.8)[f]	6844.1 (1362.7)	5944.6 (1194.9)	16.3 (13.9)[e]
I_{max} (mm^4)	15,563.1 (3210.5)	9650.0 (2592.1)	67.0 (37.8)[f]	12,687.9 (3645.0)	11910.4 (2282.0)	5.7 (18.0)
Radial shaft						
BMC (mg)	145.2 (17.3)	127.5 (16.0)	14.2 (7.6)[f]	137.5 (10.7)	136.1 (12.6)	1.2 (4.3)
Tot.Ar (mm^2)	155.6 (20.1)	132.6 (19.5)	17.8 (8.3)[f]	147.4 (11.9)	144.0 (11.9)	2.5 (3.9)
M.Cav.A (mm^2)	42.3 (8.8)	33.6 (9.3)	28.6 (19.0)[f]	39.7 (6.1)	37.5 (4.1)	5.9 (14.0)
Co.Ar (mm^2)	113.4 (13.5)	99.0 (12.0)	14.8 (8.2)[f]	108.0 (9.0)	106.6 (10.7)	1.5 (4.5)
Co.Dn (mg/cm^3)	1174.2 (20.9)	1176.2 (22.6)	−0.2 (1.2)	1167.4 (25.1)	1172.6 (16.9)	−0.4 (1.1)
CW.Th (mm)	3.4 (0.2)	3.2 (0.2)	4.5 (6.5)[d]	3.3 (0.2)	3.3 (0.3)	−0.1 (5.6)
BSI (mm^3)	367.1 (80.3)[c]	303.4 (71.3)	22.1 (16.2)[f]	327.2 (37.4)	341.1 (49.2)	−3.5 (6.4)
I_{min} (mm^4)	1306.9 (505.9)	895.1 (418.6)	60.8 (46.6)[f]	1145.2 (355.3)	1100.7 (354.7)	5.3 (14.5)
I_{max} (mm^4)	2030.1 (437.5)	1531.7 (351.3)	34.6 (20.9)[f]	1875.0 (207.7)	1771.3 (266.5)	6.7 (8.6)[d]
Distal radius						
BMC (mg)	409.4 (63.4)	358.9 (50.8)	14.6 (12.7)[f]	388.0 (51.3)	371.0 (55.5)	5.1 (8.2)[d]
Tot.Ar (mm^2)	446.8 (115.1)	384.4 (99.7)	19.4 (27.8)[d]	417.4 (89.1)	401.3 (60.1)	3.9 (14.0)
Co.Ar (mm^2)	136.1 (50.5)	120.8 (32.0)	11.5 (36.3)[d]	143.8 (55.6)	134.7 (47.6)	6.3 (8.8)[d]
Co.Dn (mg/cm^3)	670.8 (135.8)	681.1 (147.6)	0.2 (19.2)	661.7 (149.0)	656.6 (158.5)	4.2 (29.4)
Tr.Ar (mm^2)	310.3 (95.8)	263.5 (96.5)	28.1 (49.0)	273.6 (45.7)	266.5 (42.8)	4.4 (19.0)
Tr.Dn (mg/cm^3)	241.5 (28.5)	230.7 (29.7)	5.1 (8.0)	245.9 (34.6)	241.2 (35.5)	2.1 (4.1)
CW.Th (mm)	2.0 (0.6)	2.0 (0.6)	3.6 (18.8)	2.2 (0.7)	2.1 (0.7)	5.2 (13.1)
BSI (mm^3)	515.9 (107.9)	441.0 (119.7)	22.6 (28.5)[d]	501.3 (134.5)	486.3 (122.7)	4.0 (18.9)

[a]See *Materials and Methods* for abbreviations.
[b]Side-to-side difference (%) = (dominant − nondominant)/nondominant ∗ 100%.
[c]Significantly different when compared with the corresponding (dominant or nondominant) arm of controls ($p < 0.05$).
[d]$p < 0.05$; [e]$p < 0.01$; [f]$p < 0.001$.

about age 10, whereas most of the controls had previously been involved in various sports (although none consisting of unilateral activities), which could explain the differences in arm-strength measurements. On the other hand, in our previous adult-player study,[19] an investigation in which bone dimensions were examined with DXA in players who had started playing either during childhood or clearly after puberty, the aforementioned phenomenon was seen in "young starters" only. Also, in our earlier study with junior players,[18] this was seen in the Tanner III-starter group only, which corresponds to the period of rapid growth of the body. In light of the aforementioned findings it is thus possible that, during the rapid phase of growth, the period when most of the bone mass accumulates, the "less used" nondominant arm of the tennis players stays somewhat behind developmentally, because the bone mineral is needed to strengthen the more vigorously loaded sites of the skeleton. Although this "steal phenomenon" is entirely possible, does

Table 3. Comparison of relative side-to-side arm differences in bone variables between controls and players [mean (SD)]

Measured variable[a]	Side-to-side difference (%)		Player-to-control difference
	Players (N = 12)	Controls (N = 12)	
Proximal humerus			
BMC (mg)	21.7 (9.4)	0.9 (3.1)	20.8[d]
Tot.Ar (mm^2)	15.8 (12.4)	0.5 (6.7)	15.3[c]
M.Cav.Ar (mm^2)	18.5 (21.8)	−1.6 (14.3)	20.1[b]
Co.Ar (mm^2)	11.7 (13.2)	4.4 (9.3)	7.3
Co.Dn (mg/cm^3)	−1.2 (1.9)	0.3 (2.8)	−1.5
CW.Th (mm)	4.1 (14.9)	5.4 (13.9)	−1.3
BSI (mm^3)	22.5 (17.4)	8.2 (14.5)	14.3[b]
I_{min} (mm^4)	33.4 (37.6)	6.6 (25.8)	26.8
I_{max} (mm^4)	27.1 (23.0)	6.4 (10.8)	20.7[b]
Humeral shaft			
BMC (mg)	24.6 (8.5)	2.7 (4.7)	21.9[d]
Tot.Ar (mm^2)	17.4 (7.6)	4.6 (6.1)	12.8[d]
M.Cav.A (mm^2)	0.8 (16.1)	8.7 (10.2)	−7.9
Co.Ar (mm^2)	26.3 (9.8)	3.4 (1.4)	22.9[d]
Co.Dn (mg/cm^3)	−0.2 (2.2)	−0.3 (1.1)	0.1
CW.Th (mm)	20.2 (10.5)	0.4 (3.1)	19.8[d]
BSI (mm^3)	33.7 (16.4)	9.9 (10.8)	23.8[d]
I_{min} (mm^4)	46.4 (36.9)	5.2 (31.2)	41.2[c]
I_{max} (mm^4)	39.0 (17.4)	16.1 (12.4)	22.9[c]
Distal humerus			
BMC (mg)	27.3 (10.1)	5.2 (4.3)	22.1[d]
Tot.Ar (mm^2)	21.3 (9.7)	4.6 (7.0)	16.7[d]
M.Cav.Ar (mm^2)	−3.3 (12.7)	1.0 (13.2)	−4.3
Co.Ar (mm^2)	31.9 (11.6)	6.2 (5.9)	25.7[d]
Co.Dn (mg/cm^3)	−2.1 (1.7)	−0.5 (1.5)	−1.6[b]
CW.Th (mm)	24.6 (8.8)	4.6 (4.3)	20.0[d]
BSI (mm^3)	36.9 (14.2)	11.2 (9.4)	25.7[d]
I_{min} (mm^4)	37.4 (21.8)	16.3 (13.9)	21.1[c]
I_{max} (mm^4)	67.0 (37.8)	5.7 (18.0)	61.3[d]
Radial shaft			
BMC (mg)	14.2 (7.6)	1.2 (4.3)	13.0[d]
Tot.Ar (mm^2)	17.8 (8.3)	2.5 (3.9)	15.3[d]
M.Cav.A (mm^2)	28.6 (19.0)	5.9 (14.0)	22.7[c]
Co.Ar (mm^2)	14.8 (8.2)	1.5 (4.5)	13.3[d]
Co.Dn (mg/cm^3)	−0.2 (1.2)	−0.4 (1.1)	0.2
CW.Th (mm)	4.5 (6.5)	−0.1 (5.6)	4.6
BSI (mm^3)	22.1 (16.2)	−3.5 (6.4)	25.6[d]
I_{min} (mm^4)	60.8 (46.6)	5.3 (14.5)	55.5[d]
I_{max} (mm^4)	34.6 (20.9)	6.7 (8.6)	27.9[d]
Distal radius			
BMC (mg)	14.6 (12.7)	5.1 (8.2)	9.5[b]
Tot.Ar (mm^2)	19.4 (27.8)	3.9 (14.0)	15.5
Co.Ar (mm^2)	11.5 (36.3)	6.3 (8.8)	5.2
Co.Dn (mg/cm^3)	0.2 (19.2)	4.2 (29.4)	−4.0
Tr.Ar (mm^2)	28.1 (49.0)	4.4 (19.0)	23.7
Tr.Dn (mg/cm^3)	5.1 (8.0)	2.1 (4.1)	3.0
CW.Th (mm)	3.6 (18.8)	5.2 (13.1)	−1.6
BSI (mm^3)	22.6 (28.5)	4.0 (18.9)	18.6

[a]See *Materials and Methods* for abbreviations.
[b]$p < 0.05$; [c]$p < 0.01$; [d]$p < 0.001$.

not receive support from cross-sectional studies of athletes in which the bone densities in the nonloaded skeletal sites of athletes and their sedentary controls were generally similar.[20] Clearly, the issue of the intraskeletal steal phenomenon of bone mineral during long-term physical activity should be studied in a prospective exercise trial of growing children.

In the present study, all our players started their playing careers during childhood and significant enlargements in bone geometry, without changes in volumetric density, were observed. Interestingly, in a recent pQCT study of the radius of tennis players, Ashizawa et al.[2] showed that three players who had not started playing until the age of 16 seemed to have higher trabecular bone density, but similar cross-sectional area of the distal radius. This may suggest a compensatory mechanism of mature bone in response to mechanical loading. In other words, if mechanical load is applied to a bone site that cannot grow in size, the apparent density of bone may increase to adapt the bone to withstand the increased load. This idea is supported by our previous DXA study[19] in which the players who had started training in adulthood still seemed capable of increasing humeral bone mass, but not width. A redistribution of the mineral of a bone without changes in the total amount of bone mineral or bone's outer geometry, has also been shown to be possible.[1,8]

Earlier studies of tennis players[10,22,31] suggested that the outer dimensions of the playing-arm bones are larger than those of their

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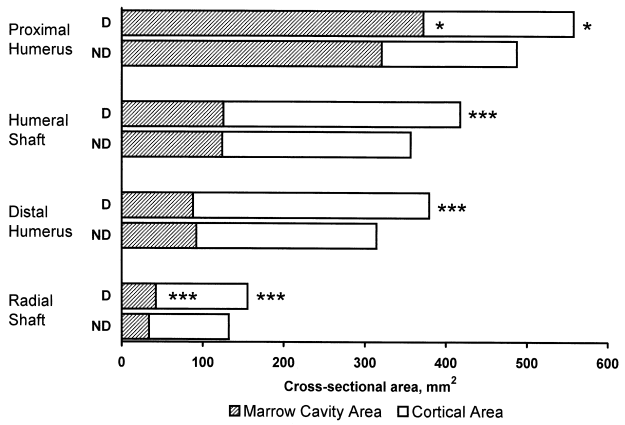

Figure 1. Distribution of total cross-sectional area (Tot.CSA) of bone to the marrow cavity area (Cav.CSA) and cortical cross-sectional area (Co.CSA) at the proximal humerus, humeral shaft, distal humerus, and radial shaft of the 12 tennis players studied. D, dominant arm; ND, nondominant arm. The side-to-side difference in Tot.Ar was significant ($p < 0.05$) at every bone site. The significant side-to-side difference in M.Cav.Ar and Co.Ar is shown with an asterisk (*) beside the bar of the dominant arm (*$p < 0.05$, ***$p < 0.001$).

nonactive counterparts. In addition, in the CT study by Dalen et al.,[10] the marrow cavity area of the humeral shaft of seven professional tennis players was examined. They found a +21% side-to-side difference in total cross-sectional area, a +43% difference in cortical cross-sectional area, and a −4% (nonsignificant) difference in marrow cavity area between the dominant and nondominant humeral shafts of the players. However, in their study, the variation in the side-to-side difference in marrow cavity area was large (−20% to +64%), thus preventing any reliable conclusions on the effects of "tennis-loading" on the marrow cavity of bone. In the present study, at every measured bone site, the total cross-sectional area was significantly larger in the playing than in the nonplaying arm, but the proportion of cortical area and marrow cavity area varied according to bone site (Figures 1 and 2). At the proximal humerus, for example, the marrow cavity area (side-to-side difference +18.5%) contributed to most of the increase in total cross-sectional area, whereas, at the shaft and distal part of this bone, the marrow cavity area was almost equal in both arms (side-to-side differences +0.8% and −3.3%, respectively); thus, at these sites, the increase in total cross-sectional area was attributable to the increase in cortical area. In the radial shaft, both the cortical (side-to-side difference +14.8%) and marrow cavity areas (side-to-side difference +28.6%) were significantly increased. It is likely that these site-to-site differences were due to different loading conditions at different skeletal sites. In particular, in resisting bending and torsional forces, a hollow, cylindrical bone (where the bone mineral mass is distributed away from the center of gravity) is a desirable structure, whereas a large cross-sectional area is more appropriate for resisting axial, compressive forces.

Overall, this study has shown that the extra bone mineral gained in the playing-arm bones of the male tennis players (all of whom had started playing already in childhood) was, in large part, used for increasing the bone dimensions (total cross-sectional area, cortical wall thickness), not volumetric cortical or trabecular bone density. The changes in bone geometry seemed to be site-specific, most probably due to the differences in bone composition and loading conditions at the given skeletal sites. According to current knowledge, it is likely that these loading-induced adaptations had developed already during skeletal growth of the players, although our study cannot give a definite answer to this, because we did not evaluate players who started training in adulthood. However, previous studies have suggested that a mature bone does not have as much capacity as a growing bone to increase its BMC and areal BMD via mechanical loading.[12,19,28,32] The new measurement techniques, such as pQCT, will provide means to examine whether mature bone is still responsive to loading by changing its geometry, increasing and redistributing its mineral mass, or both, and thus able to increase its ultimate strength and reduce the risk of fracture.

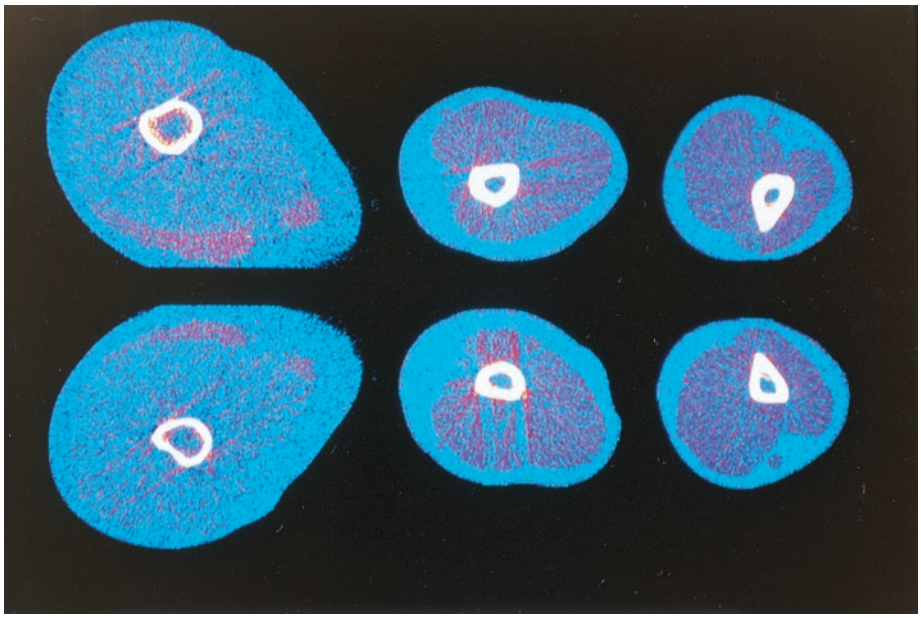

Figure 2. Humeral pQCT scan of a 26-year-old male tennis player. Paired slices, from left to right, are the proximal humerus, humeral shaft, and distal humerus. Upper panel: dominant arm; lower panel: nondominant arm. From left to right, the side-to-side differences were: 29.9%, 38.5%, and 46.5% in BMC; 26.3%, 24.7%, and 34.5% in Tot.Ar; 27.1%, −7.3%, and −1.7% in M.Cav.Ar; 24.4%, 43.4%, and 52.9% in Co.Ar; −2.9%, 0.8%, and −1.3% in Co.Dn; 10.5%, 35.5%, and 39.4% in CW.Th; and 51.1%, 56.9%, and 65.5% in BSI, respectively.

Acknowledgments: This study was supported by grants from the Medical Research Fund of Tampere University Hospital, the Academy of Finland, and the Finnish Cultural Foundation. The authors thank Virpi Koskue and Anne Rauhio for the skillful pQCT measurements.

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Date Received: December 30, 1999
Date Revised: April 13, 2000
Date Accepted: April 26, 2000