

Upper Limb Muscle–Bone Asymmetries and Bone Adaptation in Elite Youth Tennis Players

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¹Institute for Biomedical Research into Human Movement and Health, Manchester Metropolitan University, Manchester, UNITED KINGDOM; ²Lawn Tennis Association, National Tennis Centre, London, UNITED KINGDOM; ³School of Graduate Entry Medicine and Health, University of Nottingham, Royal Derby Hospital, Derby, UNITED KINGDOM; and ⁴Institute of Aerospace Medicine, German Aerospace Centre, Cologne, GERMANY

ABSTRACT

IRELAND, A., T. MADEN-WILKINSON, J. MCPHEE, K. COOKE, M. NARICI, H. DEGENS, and J. RITTWEGER. Upper Limb Muscle–Bone Asymmetries and Bone Adaptation in Elite Youth Tennis Players. *Med. Sci. Sports Exerc.*, Vol. 45, No. 9, pp. 1749–1758, 2013. **Introduction:** The study of tennis players allows the nonracket arm to act as an internal control for the exercising racket arm. In addition, the study of the upper limbs removes the influence of gravitational loading, allowing the examination of the influence of muscular force on bone adaptation. **Methods:** The role of muscular action on bone, strength parameters of the radius, ulna (both at 4% and 60% distal–proximal ulnar length), and humerus (at 35% distal–proximal humerus length) as well as muscle size in both arms of 50 elite junior tennis players (mean \pm SD age = 13.5 \pm 1.9 yr) were measured with peripheral quantitative computed tomography (pQCT). **Results:** Strong relationships were found between muscle size and bone size in both arms (all correlations, $P < 0.001$, $R^2 = 0.73$ – 0.86). However, the muscle–bone ratio was significantly lower ($P < 0.001$) in the upper arm on the racket side (compared with the contralateral arm). In addition, material eccentricity analysis revealed that bone strength in bending and torsion increased more than strength in compression as the moment arms for these actions (bone length and width, respectively) increased (in all cases, $P > 0.001$, $R^2 = 0.06$ – 0.7) with relationships being stronger in torsion than in bending. Large side differences were found in bone strength parameters and muscle size in all investigated sites, with differences in distal radius total BMC (+37% \pm 21%) and humerus cortical cross-sectional area (+40% \pm 12%) being most pronounced (both $P < 0.001$). **Conclusions:** These results support a strong influence of muscular action on bone adaptation; however, interarm muscle–bone asymmetries suggest factors other than local muscle size that determine bone strength. The results also suggest that torsional loads provide the greatest stress experienced by the bone during a tennis stroke. **Key Words:** pQCT, EXERCISE, BMD, PHYSICAL ACTIVITY

Bone strength (as determined by its density, mineral mass, and micro- and macrostructure and geometry) is an important contributor to fracture risk. Genetic and nutritional factors certainly play a part in determining bone strength, as does mechanical loading. The mechanostat theory (13) suggests that bone adaptation is a homeostatic control system that aims to regulate the peak habitual strain experienced in bone by continual modeling and remodeling. In the case of low or reduced levels of loading (such as in bed rest, space flight, or spinal cord injury), bone strength de-

creases at rates of up to 2.5% per month (12,19,25). Conversely, regular exercise (particularly with a weight-bearing or high-impact element) leads to increases in bone strength parameters (21).

Although it was previously assumed that passive loading as a result of body-weight bearing was the major contributing load causing bone strains and hence adaptation, it was proposed less than 15 yr ago that muscle forces are a significant (and in some cases the primary) cause of bone strain and subsequent adaptation (32). Proponents of this hypothesis cite the short levers that muscles work against, requiring the internally generated forces imposed on bone to be up to 10 times that of external reaction forces. In addition, the strong relationship between muscle size (as a surrogate of force production capabilities) and bone strength (23,34) is also proffered as evidence of this link.

As much as spinal cord injury patients offer a model of long-term low habitual loading, athletes conversely offer an opportunity to study the effects of years or even decades (in the case of master athletes) of high-level loading. Indeed, athletes in several sports have been shown to have higher bone strength parameters than less active peers, with the effects more pronounced in sports requiring high impacts or

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rates of limb acceleration or deceleration (and hence higher peak muscle forces) (1,11,22). Associations of participation in these sports with higher bone strength have also been shown in junior (6) and veteran (39) sportspeople.

However, it is not clear whether these group differences are attributable to the effects of exercise, or whether an element of self-selection affects the results—critics of these studies cite possible genetic, nutritional, and environmental factors that could also contribute. Therefore, some recent studies have looked at bone asymmetries in sports where one arm or leg is loaded more than the other, such as tennis (2,5,7,14), baseball (38), cricket (35), gymnastics (18,40), or jumping events (18)—allowing the less active limb to act as an internal control. Such studies have found significant and (in the case of tennis players) very large differences in volumetric bone density, size, and geometry between the dominant and the nondominant limb, whereas in controls, these differences were not evident or much reduced (0.5%–5.2% side difference in bone size or bone mass dependent on site [14,38]).

The study of asymmetries in the upper limbs also allows experimenters to control for gravitational loading as the upper limbs are not weight bearing. This offers an opportunity to study the effects of muscular action on bone adaptation, as it is certainly the primary (if not the only) cause of strain on these bones. Despite these benefits, the role of muscle in the development of these asymmetries has not been thoroughly explored—although bone and muscle asymmetries in tennis players have been studied separately (5,7,10), only one study (that the authors are aware of) has looked at muscle–bone relationships in the two arms (7). In this study, it was concluded that although muscle contributed to bone side differences, it only played a minor role. However, this study combined magnetic resonance imaging assessment of average bone size throughout a 30% bone length diaphyseal section and dual-energy x-ray absorptiometry assessment of BMC in this section. Therefore, it cannot be deduced from that study whether the differences observed were related to variation in density and distribution and how strongly bone strength was affected. Moreover, the participants were sub-elite players only, generally playing tennis less than 10 h·wk⁻¹, and so their training volume and hence exercise stimulus to the bone would be less than that of an elite cohort.

The authors believe that a study on muscle–bone side differences in elite tennis players will provide useful information on the role of muscular action in bone adaptation. Peripheral quantitative computed tomography (QCT) also allows the analysis of bone's resistance to compressive, bending, and torsional strains (26), which may give some evidence as to the primary loading modality straining the bone during tennis. An elite tennis-playing population will likely have larger side differences in muscle and bone than athletes studied previous as training volume affects the magnitude of interarm asymmetries in tennis (29). It is hypothesized that muscle size will be shown to be the primary determinant of bone stiffness

and strength in the upper limbs, even when potentially confounding allometric factors (e.g., bone length) are controlled for.

METHODS

Participants. Fifty junior tennis players (30 males, 20 females; mean ± SD age = 13.5 ± 1.9 yr) competing at the British Junior Tennis Championships in Bournemouth, England, in 2011 and in Nottingham, England, in 2012 were recruited for this study. Participants were included when they reported to be in good health and had had no leg or arm fractures within the preceding 24 months. The study conformed to the Declaration of Helsinki guidelines and was approved by Manchester Metropolitan University's Ethics Committee before the start of the study. Written informed consent was obtained from all subjects before their participation.

Information on training history in tennis and other sports was collected from each participant during a structured interview with the main author. They were then asked whether they regularly played any other sports and if so for how long—particular note was taken if the sport involved favoring one arm over the other (such as in golf, javelin throw, or cricket). Participants were also asked at what age they started to play tennis regularly and how many hours they played per week as well as details of any other tennis-related training (fitness work, strength and conditioning sessions, etc.). Participants were asked for their preferred racket hand and whether they played single- or double-handed backhand and forehand. Finally, they were asked if they had suffered an injury that forced them to stop playing within the last 2 yr or whether for any other reason (lack of interest, schoolwork pressures, etc.) they had ceased playing regularly. In either of these cases, further details of the cause for absence, timing, and length of absence were recorded. For females, the date of menarche (if applicable) was also recorded.

Bone measurements. Scans were taken with a Stratec XCT-3000 (six participants) or XCT-2000 (remainder of cohort) pQCT scanner (Stratec Medizintechnik GmbH, Pforzheim, Germany). In all cases, all scans for a particular participant were taken with the same scanner, and scanners were cross-calibrated with the European forearm phantom. Voxel size of 0.5 mm was used, with scan speed of 50 mm·s⁻¹. Scans were taken at two sites of the left and right forearm, corresponding to 4% and 60% distal–proximal ulnar length (measured between the olecranon and the ulnar styloid process). Muscle cross-sectional area (CSA) measurements were also examined at the 60% site to examine the muscle–bone relationship. Where evidence of an open growth plate was seen in the scout view, the first scan was initiated at the most distal point of the growth plate rather than the end of the bone; the same measurements were then taken from this as a reference point. Scans of the left and right humerus at 35% proximal–distal humerus length (measured between the olecranon and lateral border of the acromion) as well as muscle

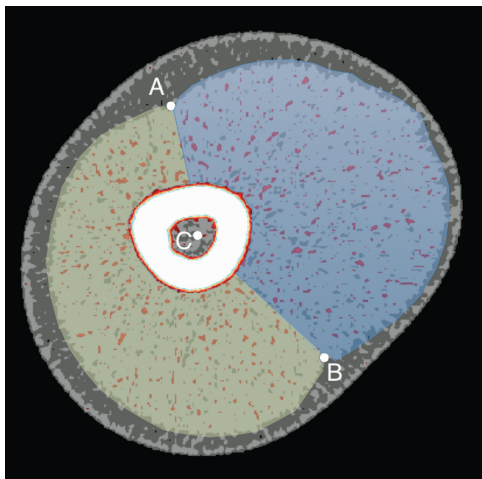


FIGURE 1—pQCT image of 35% humerus site showing points used to separate flexor and extensor muscles. (A and B) Indentations indicating separation of flexors and extensors. (C) Center of bone mass. Light blue area represents area designated as flexors; light green area represents extensors.

CSA at this site were examined. Measurements were then exported using the Automated Analysis Tools in Version 6.00 of the software supplied with the machine. A peeling threshold of $650 \text{ mg}\cdot\text{cm}^{-3}$ was set for diaphyseal sections of bone, with a threshold of $180 \text{ mg}\cdot\text{cm}^{-3}$ set for the epiphyseal 4% slice. Only the inner 45% of bone was selected for the analysis of the trabecular bone in the epiphysis, using contour mode 1 and peeling mode 1 included in the machine software.

Given the lack of a standard nomenclature for pQCT results, the suggestions for reporting high-resolution CT results (<http://www.asbmr.org/StandardizationofBoneStructureandDensityNomenclature.aspx>) and those of a recent publication (25) have been followed. The parameters examined in the 4% epiphyseal slice were total bone area (Ar.tot, mm^2), total bone mineral content (vBMC.tot, $\text{mg}\cdot\text{mm}^{-1}$), and trabecular bone mineral density (vBMD.tb, $\text{mg}\cdot\text{cm}^{-3}$). In diaphyseal sites, Ar.tot, vBMC.tot, cortical area (Ar.ct, mm^2), and cortical density (vBMD.ct, $\text{mg}\cdot\text{cm}^{-3}$) were examined, with adjustments made to the cortical density values (due to partial volume effect) by equations established in an earlier publication (28). In addition, at the diaphyseal sites, moments of inertia indicating bone's stiffness in bending perpendicular to the line of elbow flexion/extension, in line with elbow flexion/extension, and torsion, respectively, (I_x , I_y , and I_p), cortical thickness, (Ct.Th_{der}, mm), periosteal circumference (PsC, mm), and endosteal circumference (EcC, mm) derived from a circular ring model were measured. In addition, gross muscle CSA (MuscA, mm^2 , as a surrogate for maximal force production) in the 60% slice was obtained using a threshold of $35 \text{ mg}\cdot\text{cm}^{-3}$. In the upper arm, the flexor and extensor muscles were measured separately—indentations marking the separation of these muscles were located (points A and B on Fig. 1) and lines drawn from these points to the center of the bone's mass (point C on Fig. 1, whose position is given in the loop output from the XCT software). These lines were then used to separate the flexors and extensors.

Finally, material eccentricity (ME) was also examined. ME is the ratio between R_x , R_y , and R_p (or moments of resistance in x -axis, y -axis, and polar plane, respectively, representing bone's strength in x -axis bending, y -axis bending, and torsion) and Ar.ct (which indicates bone's strength in compression). ME thereby gives an indication of bone's relative strength in bending/torsion compared with compression (26). A greater bone length and bone circumference means a greater moment arm for bending and torsional moments, respectively. Therefore, the relationships of ME with bone length and bone circumference were examined to establish any link between these moments and bone geometry. *In vivo* precision of the laboratory's pQCT measurements has been reported elsewhere (26)—precision is $<0.5\%$ for vBMC.tot, Ar.tot, and Ar.ct, 1.15% for MuscA, and up to 5.1% for derived values.

Force platform data. A press-up was performed on a force platform (Leonardo, Novotec, Pforzheim, Germany)—participants were asked to assume a straight-armed press-up position with one hand on each force plate equidistant from the center of the platform. They then were instructed to bend the arms and push-up as powerfully as possible with the aim of leaving the ground. Total peak power and force were recorded as well as left and right arm peak force and power. Grip strength was also measured using a Jamar+ hand grip dynamometer (Sammons Preston Inc., Bolingbrook, IL). Participants completed three measures in each hand, with the arm down by the side but not touching the hip, and the highest force value on each side was recorded.

Statistical analysis. Data were examined using PASW Statistics 18.0 (SPSS Inc., Chicago, IL). To test for significant differences between the dominant and the nondominant arm and effects of sex, repeated-measures ANOVA with bone or muscles parameters in racket and nonracket arm as within-subject factor and sex as a between-subject factor was used. Linear regression analysis was used to examine the relationship between MuscA (independent variable) and bone CSA and maximal force/velocity/power/height values (dependent values) in both the dominant and the nondominant arm. ANCOVA analysis of ME (polar or x/y -axis SSI divided by bone area, serving as a measure of how bone is adapted to resist torsion and x/y -axis bending relative to compression) was also carried out using univariate ANCOVA with ME as dependent variable, side (racket/nonracket arm) as fixed factor, and bone length as covariate. A custom model was used, examining the main effect of bone length, the main effect of side, and the interaction between bone length and side. In the case of a side difference or bone length–side interaction, parameter estimates were used to assess differences in

TABLE 1. Cohort characteristics and training habits.

Parameter	Group Mean SD	
	Male	Female
Age (yr)	13.5 ± 1.9	13.5 ± 2.0
Tennis start age (yr)	6.5 ± 2.3	7.4 ± 2.4
Tennis training ($\text{h}\cdot\text{wk}^{-1}$)	10.8 ± 3.7	11.3 ± 3.5
Height (m)	1.64 ± 0.15	1.61 ± 0.10
Body mass (kg)	52.8 ± 13.6	50.5 ± 10.4

TABLE 2. Results of pOCT scans of dominant and nondominant arms in males and females.

Site	Bone/Muscle	Measured Variable ^a	Males				Females				Cohort		
			Racket Arm	Nonracket Arm	Side Difference (%)		Racket Arm	Nonracket Arm	Side Difference (%)		Side ^b	Interactions	
					Mean	95% CI			Mean	95% CI			
4% ulna	Radius	vBMC.tot (mg/mm)	159 ± 40	112 ± 29	44.6	36.1 to 53	129 ± 26	102 ± 19	26.5	19.4 to 33.6	***	* **	
		Ar.tot (mm ²)	290 ± 62	277 ± 40	28.2	20.1 to 36.3	253 ± 44	222 ± 34	14.9	6.1 to 23.7	***	***	
	Ulna	vBMD.tb (mg·mm ⁻³)	463 ± 98	377 ± 85	23.9	18.7 to 29	389 ± 61	346 ± 49	12.5	8.2 to 16.8	***	*	
		vBMC.tot (mg/mm)	78 ± 18	66 ± 19	25.8	11.7 to 39.9	70 ± 15	59 ± 14	20.0	9.5 to 30.5	***		
	60% ulna	Radius	Ar.tot (mm ²)	377 ± 51	332 ± 57	16.6	6.6 to 26.6	351 ± 59	319 ± 65	12.1	4.1 to 20.2	**	*
			vBMD.tb (mg·mm ⁻³)	207 ± 42	192 ± 50	12.8	2.1 to 23.4	191 ± 34	168 ± 30	15.3	5.2 to 25.5	***	
		vBMC.tot (mg/mm)	95 ± 22	80 ± 19	19.2	16.2 to 22.2	94 ± 20	82 ± 18	15.5	11.5 to 19.4	***		
		Ar.tot (mm ²)	122 ± 25	101 ± 20	20.3	17 to 23.5	114 ± 13	99 ± 12	14.9	10.4 to 19.5	***	*	
		Ar.ct (mm ²)	83 ± 18.3	70 ± 15	18.7	15.6 to 21.8	80 ± 15	70 ± 13	14.6	11.3 to 17.9	***		
		vBMD.ct (mg·mm ⁻³)	1180 ± 45	1182 ± 41	-0.2	-0.8 to 0.4	1196 ± 38	1192 ± 42	0.4	-0.1 to 0.9	***		
35% humerus	Ulna	Ct.Thier (mm)	2.72 ± 0.45	2.53 ± 0.45	7.8	4.7 to 10.9	2.78 ± 0.59	2.60 ± 0.56	6.9	4.6 to 9.3	***	*	
		PSc (mm)	39 ± 4.1	35.5 ± 3.4	9.6	8.1 to 11.1	37.7 ± 2.2	35.2 ± 2.1	7.1	5 to 9.2	***		
	Humerus	Esc (mm)	21.9 ± 3.6	19.6 ± 3.1	11.8	7.9 to 15.7	20.3 ± 3.5	18.9 ± 3.2	7.6	2.9 to 12.4	***		
		I _x (mm ⁴)	973 ± 388	631 ± 249	54.2	46 to 62.5	870 ± 186	633 ± 124	38.2	29.2 to 47.2	***	*	
	Humerus	I _y (mm ⁴)	1323 ± 603	970 ± 419	36.2	29.6 to 42.8	1080 ± 365	890 ± 260	21.1	13.9 to 28.3	***	*	
		I _z (mm ⁴)	2296 ± 957	1601 ± 643	43.3	36.5 to 50.1	1950 ± 517	1523 ± 358	28.3	21.2 to 35.4	***	*	
	Humerus	vBMC.tot (mg/mm)	113 ± 28	99 ± 24	14.2	11.1 to 17.3	113 ± 27	100 ± 23	12.0	9 to 15.1	***		
		Ar.tot (mm ²)	135 ± 29	120 ± 22	12.2	8.4 to 16	127 ± 21	117 ± 19	8.8	5.7 to 11.9	***		
	Humerus	Ar.ct (mm ²)	99 ± 24	87 ± 19	15	11.7 to 18.3	95 ± 22	85 ± 18	12.3	8.6 to 16	***		
		vBMD.ct (mg·mm ⁻³)	1155 ± 42	1169 ± 46	-1.2	-1.9 to 0.4	1185 ± 50	1188 ± 45	-0.2	-1.2 to 0.7	*		
35% humerus	Humerus	Ct.Thier (mm)	3.2 ± 0.55	2.93 ± 0.52	9.8	7.4 to 12.1	3.19 ± 0.66	2.92 ± 0.58	9.3	6.5 to 12	***		
		PSc (mm)	41.0 ± 4.5	38.7 ± 3.5	5.8	4 to 7.6	39.8 ± 3.3	38.2 ± 3	4.3	2.8 to 5.8	***		
	Humerus	Esc (mm)	20.8 ± 3.1	20.3 ± 2.9	3	-0.6 to 6.5	19.8 ± 2.9	19.9 ± 3.1	-0.2	-2.1 to 1.8	***	*	
		I _x (mm ⁴)	1539 ± 676	1158 ± 444	30.7	22.4 to 39	1306 ± 466	1095 ± 483	21.8	14 to 29.6	***		
	Humerus	I _y (mm ⁴)	1459 ± 671	1134 ± 477	28.6	19.5 to 37.8	1333 ± 549	1069 ± 348	23.4	14.1 to 32.7	***		
		I _z (mm ⁴)	2999 ± 1298	2292 ± 874	29.2	21.3 to 37.1	2639 ± 984	2164 ± 776	21.9	15.3 to 28.5	***		
	Humerus	Forearm MuscaA (mm ²)	3452 ± 782)	2861 ± 680	21.1	17.9 to 24.4	3142 ± 561	2629 ± 444	19.4	17.2 to 21.6	***		
		Hand grip force (N)	301 ± 88)	252 ± 80	19.6	12.6 to 26.7	297 ± 92	251 ± 92	20.6	15.6 to 25.6	***		
	Humerus	vBMC.tot (mg/mm)	236 ± 65	166 ± 41	41.6	36.7 to 46.5	228 ± 49	172 ± 36	32.7	28.9 to 36.5	***		
		Ar.tot (mm ²)	262 ± 69	209 ± 40	24.7	20.8 to 28.7	249 ± 37	209 ± 28	18.9	15.7 to 22.2	***	*	
Humerus	Ar.ct (mm ²)	203 ± 55	140 ± 33	44.4	39.1 to 49.8	189 ± 38	142 ± 28	33.9	30 to 37.8	***			
	vBMD.ct (mg·mm ⁻³)	1195 ± 49	1195 ± 41	0	-1.9 to 1.9	1196 ± 46	1198 ± 44	-0.1	-2.2 to 2.1	***	**		
Humerus	Ct.Thier (mm)	4.78 ± 0.95	3.46 ± 0.64	38.1	33.6 to 42.7	4.54 ± 0.78	3.53 ± 0.66	29.3	25.1 to 33.5	***	*		
	PSc (mm)	57 ± 6.6	51.1 ± 5	11.6	9.8 to 13.4	55.7 ± 4.2	51.1 ± 3.4	9.0	7.5 to 10.5	***			
Humerus	Esc (mm)	27 ± 3.8	29.3 ± 4	-7.9	-10 to 5.9	27.2 ± 2.7	28.9 ± 3	-5.8	-8.3 to 3.4	***			
	I _x (mm ⁴)	5406 ± 2459	3194 ± 1250	66.4	55.4 to 77.3	4781 ± 1484	3199 ± 987	50.0	43 to 56.9	***	*		
Humerus	I _y (mm ⁴)	5747 ± 2660	3413 ± 1432	66.5	56.5 to 76.5	4918 ± 1629	3311 ± 998	48.6	37.8 to 59.5	***	*		
	I _z (mm ⁴)	11152 ± 5100	6607 ± 2643	65.9	56.3 to 75.6	9700 ± 3095	6510 ± 1944	48.7	41.4 to 55.9	***	*		
Humerus	Upper arm MuscaA (mm ²)	2339 ± 626)	2159 ± 574	8.4	5.8 to 10.9	2298 ± 501	2084 ± 434	10.2	7.7 to 12.6	***			
	Press-up force (N)	266 ± 85)	255 ± 77	5.0	1.7 to 8.3	220 ± 40	210 ± 38	4.8	1.5 to 8.0	**			
Humerus	Press-up power (W)	326 ± 150)	285 ± 131	14.7	10.2 to 19.2	253 ± 104	231 ± 92	9.8	4.9 to 14.6	***			

Data are presented as mean ± SD.

^aSee Methods section for abbreviations.

^bSignificant result indicates significant side difference in favor of racket arm.

Asterisks denote significant side difference or significant effect on the magnitude of side difference of age or sex: *P < 0.05, **P < 0.01, ***P < 0.001.

intercepts and gradients. Differences were considered significant at $P < 0.05$. Data are shown as mean \pm SD.

RESULTS

Cohort characteristics. Cohort characteristics and training habits are displayed in Table 1. There were no significant sex differences in participant age, training hours, tennis starting age, height, or mass.

Athletic history. Eighteen players regularly participated in other sports on a weekly basis—these sports included swimming, association football, running, netball, basketball, and ballet. Six players also regularly played another sport that favored one arm—hockey, table tennis, badminton, and cricket. In all cases, the player played this additional sport with the same favored arm as in tennis, and in no case did they train more than 2 h·wk⁻¹ in the secondary sport.

Muscle differences. Muscles in the racket forearm and upper arm were 9%–20% larger than those in the nonracket arm (Table 2; all $P < 0.001$). All but six players were able to perform the power press-up and higher press-up maximal

force ($4.9\% \pm 6.9\%$, $P < 0.01$), maximal power ($13.0\% \pm 11.4\%$), and grip strength ($20.0\% \pm 15.7\%$; both $P < 0.001$) were recorded in the racket arm. Maximal press-up force and power were positively correlated with MuscA in both upper and lower arms—with correlation coefficients of press-up maximal force ($R^2 = 0.76$ racket arm, 0.68 nonracket arm) being higher than those of press-up maximal power ($R^2 = 0.61$ racket arm, 0.47 nonracket arm) and maximal handgrip force ($R^2 = 0.49$ racket arm, 0.56 nonracket arm).

Bone differences. In all cases, bone strength parameters (except radius and humerus vBMD.ct, and humerus and ulna EcC) had significantly higher values in the racket than that in the nonracket arm (Table 2). At the 4% forearm slice, vBMC.tot differences ($37.2\% \pm 21.4\%$ radius, $23.5\% \pm 32.4\%$ ulna) were due to greater Ar.tot and vBMD.tb in the racket arm (all $P < 0.001$, except ulnar Ar.tot $P < 0.01$). In contrast, vBMC.tot differences at the 60% forearm slice and 35% upper arm slice were a result of 17%–40% greater Ar.ct (all $P < 0.001$) and not vBMD.ct differences. Compared with age-matched reference data (24), radius Ar.tot was $19.7\% \pm 17.0\%$ and MuscA was $17.1\% \pm 17.8\%$ greater

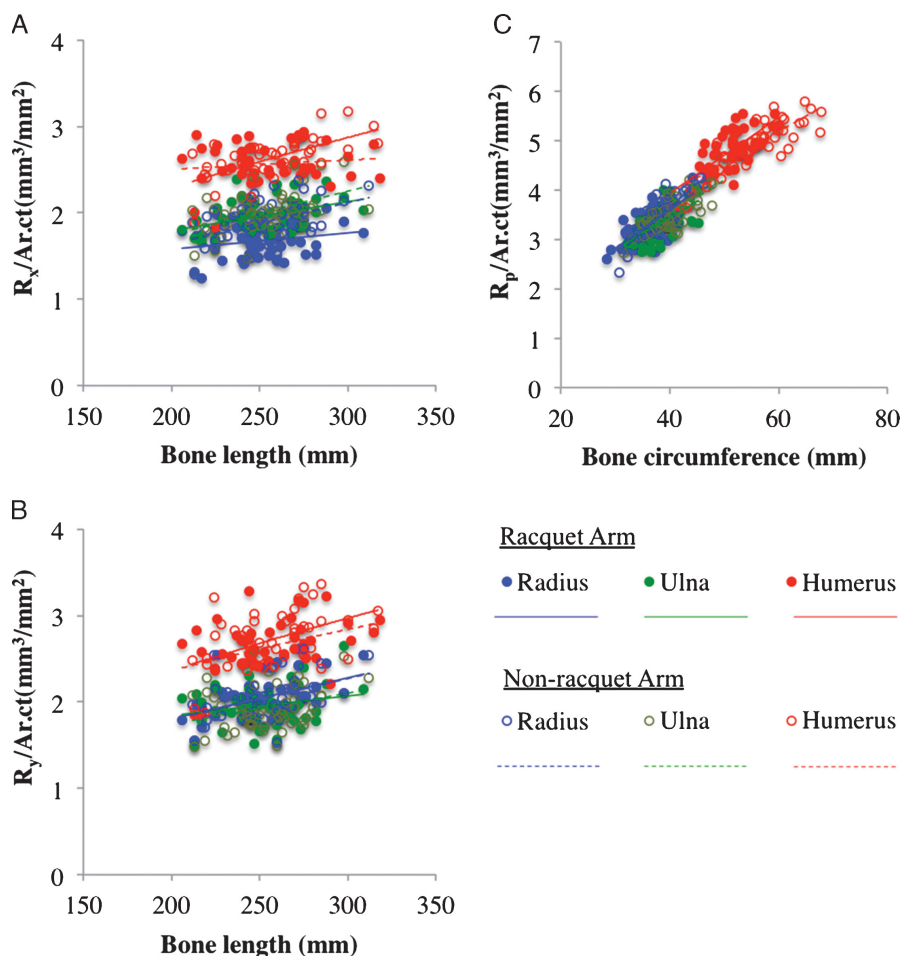


FIGURE 2—ME in radius ulna at 60% distal–proximal ulna length and humerus at 35% distal–proximal length of both arms in anteroposterior bending (A) and mediolateral bending (B) with respect to bone length and torsion with respect to bone’s outer circumference (C). All regressions $P < 0.001$, $R^2 = 0.06$ –0.7, with exception of anteroposterior bending in nondominant humerus ($P > 0.05$).

than average in the racket arm (both $P < 0.01$); nonracket arm values were not significantly different. In both arms, vBMD.tb was $5.2\% \pm 3.0\%$ greater in this cohort than that in the reference population ($P < 0.001$).

Bone geometry. At diaphyseal locations, Ct.Th_{der} and PsC were all greater in the racket arm (all $P < 0.001$). However, EcC side differences differed greatly in the three bones—racket arm radius EcC was $11.2\% \pm 10.3\%$ greater, humerus EcC was $7.1\% \pm 5.1\%$ smaller (both $P < 0.001$), and ulna EcC was no different compared with the nonracket arm. As a result of these size and shape differences, I_x , I_y , and I_p were 26%–59% greater in all three bones—all $P < 0.001$.

Diaphyseal radius and humerus (but not ulna) in both arms were stiffer in bending in the y -axis (along the line of elbow flexion/extension) than that in the x -axis (all $P < 0.001$). ME was also examined (Fig. 2). There was a significant effect of bone length on x -axis and y -axis ME and of PsC on polar ME in both arms in radius, ulna, and humerus (all $P < 0.001$)—except x -axis ME in the nondominant humerus. Coefficients of determination of these regressions were much higher for polar ME and PsC ($R^2 = 0.4$ – 0.7), compared with x - or y -axis ME and bone length ($R^2 = 0.06$ – 0.4). This shows that (except the nondominant humerus) longer bones and bones with a larger PsC are more than proportionally stronger in response to bending and torsional strains (compared with compressive strain), respectively, as opposed to a shorter bone or one with a smaller circumference.

Effects of sex. Side differences in distal radius vBMC.tot, Ar.tot, and vBMD.tb; proximal radius Ar.tot, PsC, I_x , I_y , and I_p ; proximal ulna I_x ; and humerus Ar.ct, Ct.Th_{der}, PsC, I_x , and I_y were more pronounced in males compared with females ($P < 0.05$; except distal radius vBMC.tot and humerus Ar.ct, $P < 0.01$, and distal radius Ar.tot, $P < 0.001$).

Effects of menarche. Of the female players, 10 had not reached menarche by the time of testing, and 10 were postmenarcheal—the mean time because menarche for postmenarcheal players was 1.83 ± 1.14 yr. Premenarcheal and postmenarcheal players had similar training volumes (11.7 ± 1.9 and 11.0 ± 4.3 h-wk⁻¹, respectively) and had been playing for a similar time (5.9 ± 0.8 and 6.5 ± 3.0 yr), although premenarcheal players were younger (12.4 ± 1.7 yr compared with 14.5 ± 1.8 yr). However, when age was controlled for (by inclusion as a covariate in ANOVA), there were no significant differences in muscle, bone, or force parameters (or the magnitude of side difference) between the two groups.

Muscle–bone relationship. There were strong correlations between MuscA and Ar.ct in both forearms and upper arms ($R^2 = 0.79$ – 0.86 , all $P < 0.001$), with the correlation coefficients in the dominant arm being higher in all cases (Fig. 3). Similarly, there were strong correlations between humerus Ar.ct and maximal press-up force (dominant arm $R^2 = 0.86$, nondominant arm $R^2 = 0.72$) and power (dominant arm $R^2 = 0.71$, nondominant arm $R^2 = 0.57$) and maximal hand grip force and racket and ulna Ar.ct ($R^2 = 0.51$ – 0.60),

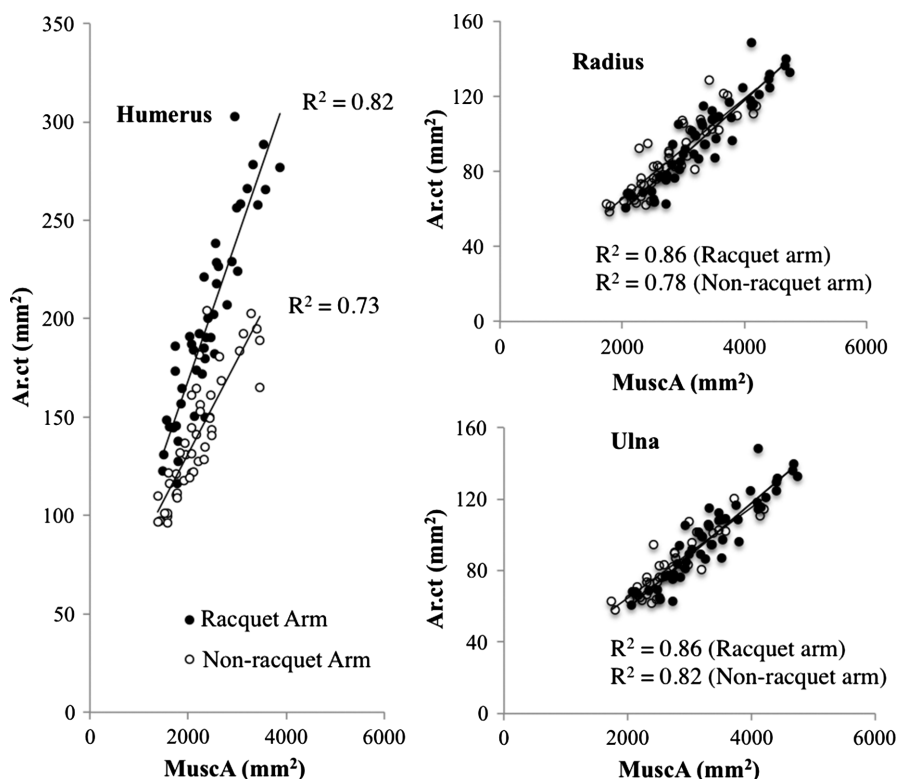


FIGURE 3—Linear regressions showing relationship between muscle CSA (MuscA) and cortical bone CSA (Ar.ct) at midshaft radius, ulna, and humerus sites in racket and nonracket arms.

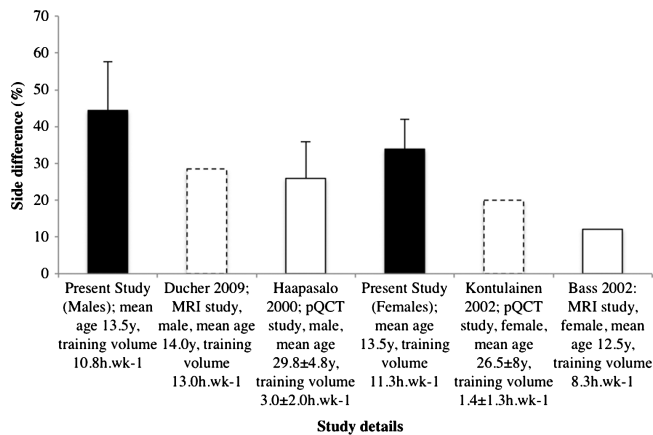


FIGURE 4—Mean side differences in humerus cortical area and details of cohort age, sex, and training volume data in this and similar studies. Error bars indicate SD; dashed bars indicate studies where cohort dispersion value (e.g., SD) was unfortunately not reported.

all $P < 0.001$. These strong relationships remained (albeit with lower coefficients) even when bone length was accounted for (partial correlation). Muscle–bone ratio was higher in the racket arm in the proximal ulna ($4.0\% \pm 9.9\%$, $P < 0.05$) and radius ($6.1\% \pm 7.8\%$, $P < 0.001$) and lower in humerus ($20.8\% \pm 7.5\%$, $P < 0.001$).

DISCUSSION

The main observations of this study are the close correlations of muscle size and bone size, in addition to a significantly lower muscle–bone ratio in the upper arm on the racket side compared with the other arm. Also, strong relationships between bone strength in bending and torsion and bone length and width (the moment arms for these actions respectively) were found. The results also showed large side differences in bone strength indicators, muscle size, and force/power output in favor of the racket arm.

Muscle differences. In line with previous observations, it is shown here that regular participation in tennis during youth is associated with large, site-specific differences in bone strength and muscle size between the racket and the nonracket arm (2,5,7,14). The results qualitatively conform with the literature in that muscle size was found to be larger on the racket side (7) and that the increased upper arm muscle size is due primarily to an increase in the triceps brachii rather than that in the elbow flexors in youth players (30) although not in adults. This suggests that the triceps have a larger role in playing tennis than the flexors and as such their action may more markedly stress the bone. Perhaps then, looking at gross muscle CSA in a limb segment as a surrogate for the force experienced by the bone is too simplistic. This could be more apparent in the upper limbs, where muscles play very different roles as flexors, extensors, rotators, and so on. In addition to muscle side differences, this study also demonstrates functional superiority on the racket side in grip strength—again in agreement with previous studies (9)—but also in a powerful push-up whereby even in this bilateral activity the

racket arm contributes significantly more force and power to the movement.

Bone differences. The bone side differences in this study are comparable with previous similar studies on a qualitative level. Side differences were site specific with differences in diaphyseal sites being almost entirely due to CSA differences, whereas greater bone mass in the distal radius and ulna of the racket arm was a result of a combination of CSA and bone density differences.

On a quantitative level, the bone side differences seen in this study are larger than those in previous similar studies (2,10,14,20) (Fig. 4). However, compared with players in the first three of those studies, players in the present study had ~270%, ~700%, and ~35% higher training volume, respectively, which has been shown to result in greater side differences (29). In addition, the players in this study had been playing for approximately 20% longer than participants in the Bass study, and several participants in the Haapasalo study had not started to play tennis regularly until adulthood—previous studies have seen greater side differences in those who start playing tennis before the onset of puberty (20). The two studies recording the smallest side differences in bone included solely female participants. We have seen in this study that side differences in females were smaller than those in males. The authors believe that these factors largely explain why the side differences in this study are much greater than those previously found. Although the Ducher study participants were of a similar age with a similar training volume and length of time played to the cohort in this study, they were not reported to be elite-level players. Perhaps the increased demands of elite-level training and competition are responsible for the differences found; however, the Ducher cohort side differences were most similar to those found in this study.

Bone geometry. A previous study on female youth tennis players found that humerus periosteal and endocortical radii were greater in the racket arm (except in postpubertal players where there was no significant difference) (2). In this study, in both sexes, the racket arm humerus had a larger periosteal and smaller endocortical radius (although it is unclear whether this was as a result of increased endosteal apposition or retention of bone mass on this surface). In comparison, in racket arm proximal radius and ulna, both periosteal and endosteal circumferences were greater (although not significantly in the case of ulna endocortical circumference). Perhaps the larger adaptation in the humerus in this group (when combined with the large skeletal changes already happening in an adolescent group) means that the bone is unable to remodel by bone drift and so only completes periosteal apposition during this period—although only a longitudinal study could clarify this.

The results also showed that longer bones and bones with a larger outer circumference are more than proportionally stronger in bending and torsion than compression compared with a shorter bone or a bone with a smaller circumference. This is likely due to an increase in the moment arm for bending and torsional moments that accompany an increase in bone length or width—this is supported by the linear

nature of these relationships. The relationships with bone width and strength in torsion are stronger (and the increase in torsional strength with increased bone width greater) than those of bone length and strength in bending, suggesting that torsional strains may be the dominating influence on bone adaptation as a result of regular tennis. The one anomaly is the lack of an association of bone length and resistance to bending in the nondominant humerus, the reason for this is currently unclear.

Muscle–bone relationships. The most important finding of this study is that the muscle–bone relationship is quantitatively different in both upper arms; the racket arm has a much lower muscle–bone ratio than the nonracket arm. In previous studies, side differences in muscle and bone in the arms of tennis players were found to be similar (5,7), although side differences in arm muscles in golfers were larger than those in bone (8). Although different muscle–bone relationships have been found in males and females and in groups of differing age or developmental status (particularly during puberty [7,33] and old age [36]), in groups of similar age and sex, muscle–bone relationships have followed the same regression line. This is true even in the extreme case of spinal cord injury (3). The sole exception the authors are aware of is young women with anorexia nervosa, in which case muscle–bone ratio increases as the disease progresses (4). Clearly, none of these explanations can account for the observed discrepancy in muscle–bone slope in the two arms. The observation of differing muscle–bone relationships in the two upper arms also rules out, on a profane level, that the strong muscle–bone relationships often observed are a mere consequence of our genetic-anatomical makeup. By contrast, and if we follow the idea that muscular forces constitute the most influential (if not the only) source of mechanical stimulation to the arm bones and that this guides their adaptation, then it seems that muscle cross-section (as well as muscle function as per our testing) is modulated by another influence.

It is well known from basic muscle physiology that muscles require resistance to work against in order to generate force (17). One interpretation would therefore be that the way the arm muscles are used during tennis enable them to produce greater forces than the habitual usage most of us follow—which seems a reasonable assumption. Muscle activity during the tennis stroke peaks around time of ball impact (15)—this may be a direct response to the high-impact nature of the tennis stroke, whereby a large impulse is imparted to the ball over a short contact time requiring a high level of force. Athletes competing in high impact sports have greater bone strength than those in low-impact sports and, in turn, controls (1,11,22). Alternatively, this peak in muscle activity could be a response to the large impact-induced vibrations transferred to the arm during the tennis stroke (16). Although the effects of vibration on bone strength in the lower limbs have been studied in several populations (37), the effects on upper limb bone remain unexplored. Another possibility would be that certain muscles have particular influence on the forearm bones and that these muscles were

not able to be fully identified by our pQCT approach—an interpretation that is equally attractive to us.

In the radius and humerus, axial moment of inertia was much greater along the line of action of the elbow flexor and extensors than in the orthogonal direction, suggesting that their action is a major cause of strain within these bones in the areas scanned. However, that side differences in bending strength in this axis and the perpendicular axis were similar and those in the racket arm the difference in bending strength between the two axes were greatly reduced suggests that torsional stress may be the greatest stress experienced by the bone during a tennis stroke. In addition, the stronger relationships seen between polar ME and bone width also support the idea that torsion is the primary stressor of bone.

Although the muscle–bone relationships in both limbs were strong, side differences in bone CSA only had a weak correlation with side difference in muscle size (similar to the results in the only previous article to examine muscle–bone relationships in the playing and nonplaying arms of tennis players [7]). However, there is a certain measurement error in pQCT scans (~1%); as there are two measurements (muscle area and cortical area), there are two sets of measurement errors within each muscle–bone relationship. Because only the smaller side difference values are taken, the measurement error becomes proportionally larger. For instance, the mean side difference in the humerus muscle area was 9.2%, and previous error measurement by one of the authors has revealed muscle area measurement error to be 1.2% (27). Therefore, the SE is 13% of the side difference value. It may follow that this is a significant reason for the weak correlation in side differences in muscle and bone. Further to this, there are several other factors (tendon stiffness, muscle specific tension, motor unit recruitment and rate of force development, muscle architecture, etc.), which may be different between the two limbs, that affect the muscle forces produced (and hence strains experienced by the bone); hence, correlating side differences in muscle and bone introduces the influence of these factors. Although side differences in most of these factors have not yet been explored, fiber-type distribution has been shown not to differ significantly between the two arms in tennis players (31). Finally, the gross muscle cross-sections obtained by pQCT do not allow for the full separation of individual flexors, extensors, rotators, and so on. As we have seen in this and a previous study (30), there are differential adaptations between muscle groups in tennis players, and hence (as discussed earlier), analyzing the gross muscle CSA may not provide the most accurate representation of the strains experienced by the bone.

Limitations. There are several limitations to this study. First, no sedentary controls were examined. However, we have seen in previous studies (14,18,30,40) that, in arms and legs of controls side, differences in bone strength and muscle size are nonexistent or minimal (a maximum of 5.2% in bone mass or area and 5.9% in muscle size) when compared with those completing regular asymmetric exercise. In addition, the comparison of the player's muscle and bone size

relative to reference data (24) revealed that regular participation in tennis and even the use of the nonracket arm in double-backhand strokes were not sufficient to confer significant size increases in muscle or bone on this limb. This supports the use of the youth tennis player model in order to obtain control and exercise data within a single participant, hence largely discounting any of the intersubject factors listed in the Introduction section. Another potentially confounding factor is the use of pre-, peri-, and postpubertal children; however, in previous studies of male (10) and female (2) youth tennis player cohorts, no effects of pubertal status on side differences were found. In addition, the comparison of premenarcheal and postmenarcheal females revealed no effect on menarche on the magnitude of side difference. That the muscle–bone relationships found are so strong despite the different maturation levels and sex of the participants adds further weight to the idea of dominating influence of muscular force on bone adaptation.

CONCLUSIONS

In summary, this study provides further evidence of the large osteogenic potential of regular tennis participation and

demonstrates a strong relationship between muscle and bone size. In addition, differing muscle–bone relationships in the two upper arms suggest that other factors aside from local muscle size dictate exercise-induced adaptations in bone. There is also some evidence that the predominant stress experienced by the diaphyseal radius and humerus is torsional. Finally, these adaptations are site specific, with differences between endocortical and periosteal adaptation as well as adaptation in the upper and lower arm—possibly due to either the dominating force at play or the ability of the body to adapt to a large exercise stimulus.

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