

Tennis Service Stroke Benefits Humerus Bone - is Torsion the Cause?

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Mini Abstract: Tennis appears highly effective in improving bone strength throughout the upper limb. A player with an unusual two-armed playing style had substantially greater bone strength in the mid-shaft of his service-arm humerus and ground-strokes arm ulna - suggesting these strokes are the agents of tennis' osteogenic potential.

ABSTRACT

Purpose: Regular tennis play is associated with impressive asymmetries in bone strength in favour of the racquet arm, particularly in the humerus. However, the relative effects of service and ground strokes are not known. Serendipitously, we encountered a 46-year old regular tennis player who has played service and ground strokes with different arms for over 30 years, and thus allowed differentiation of stroke effects.

Methods: Grip strength, and pQCT scans of both arms of radius at 4% distal-proximal ulna length, radius and ulna at 60% distal-proximal ulna length, and at distal (35% length) humerus were analysed in this player, and 12 male veteran players of similar age, height and mass who played a conventional, single-sided style. Confidence intervals (95%) were calculated for asymmetries and bone, muscle and force parameters in the control players – values in the case study player were compared to these intervals.

Results: Sizeable differences in bone strength in favour of the serving arm humerus were observed in this player - comparable to those found in the control players. Whilst asymmetries in favour of the ground stroke arm ulna were also evident, no sizeable asymmetry was found in proximal or distal radius, forearm or upper arm muscle size or hand grip force.

Conclusions: These results suggest that the service stroke is responsible for the humeral hypertrophy observed in tennis players, and that ulna adaptation may be attributable to the ground strokes.

The osteogenic potential of the service stroke may be related to the large torsional stresses it produces.

Keywords: Bone, pQCT, Exercise, Humerus, Tennis.

INTRODUCTION

Bone fractures are highly prevalent throughout life, with annual incidence around 5% in adolescent males and older females. Bone strength indicators *e.g.* bone mass predict fracture incidence [1], therefore understanding factors influencing bone strength is important in reducing fracture risk. Bone is responsive to the habitual loading it experiences [2] - chronic disuse is associated with substantial bone loss, *e.g.* 50% bone mass loss in spinal cord injury (SCI) patients [3]. Conversely, bone strength in athletes can be 25-30% greater than sedentary peers even in master athletes [4, 5] - suggesting a lifelong osteogenic potential for exercise. However, the most marked exercise effects on bone in humans are observed in tennis and baseball players.

The impressive humeral hypertrophy associated with tennis was brought to attention by Jones *et al* [6]. Well-known to radiologists, and highly-cited Jones' study found 35% greater cortical thickness in the racquet arm humerus of male players (compared to the non-racquet arm). Recent peripheral quantitative computed tomography (pQCT) studies found over 40% greater total bone mass in the racquet and throwing arm humeri of youth tennis players and adult baseball players respectively [7]. These asymmetries are ten times greater than those observed in the lower limbs [8] and occur throughout the bone's length [9, 10], including proximal humerus (a common fracture site in elderly). However, whilst the effects of baseball play are limited to the humerus [11] tennis also results in substantial forearm bone asymmetry (*e.g.* 40% greater racquet arm

bone mass at distal radius [7] - the most common fracture site in elderly).

Tennis play consists of the serve, backhand and forehand strokes. However, the contribution of each stroke to bone adaptation is unknown. During study of side-asymmetries in veteran players, a participant presented himself with the unusual quirk of serving with one arm and playing ground-strokes with the other. Given his bilateral playing style, he was excluded from the previous publication [4]. However, this case provides an ideal model to distinguish effects of ground and service strokes, albeit at the level of a case study. Therefore, we report upper limb bone, muscle and force values and side-asymmetries observed in this player, and age-matched players from the same study.

CASE REPORT

<Table 1 about here>

The case study participant (referred to as VTP) began playing tennis in adolescence, and had played regularly ever since – using his right (dominant/writing) arm to serve, and the left arm to play ground-strokes. He had not suffered any significant injuries, or engaged regularly in other upper limb sports for any period of time during his life. VTP worked in a retail environment requiring some heavy two-armed lifting. The control cohort were 12 male players who had played tennis regularly (>3 hours per week) since childhood (start age 7-15 years) – all played with a conventional single-sided style. VTP was of similar age, mass and height to controls, and had similar tennis training volume - whilst he had played tennis for a similar time period, he had started playing at a later age (Table 1). Peripheral quantitative computed tomography (pQCT) scans of the radius at 4% distal-proximal ulna length, radius and ulna at 60% distal-

proximal ulna length and humerus at 35% distal-proximal length were taken in both arms as previously described [4] and grip strength (F_{\max}) was measured using a dynamometer.

Humeral bone strength indicators were greater in the serving arm of VTP (Figure 1) – these side-asymmetries were similar to those observed in the single-sided control players. VTP's serving racquet arm values were similar to cohort racquet arm values, and greater than cohort non-racquet arm values (Table 1). Conversely, VTP's ground-strokes arm values were lower than control racquet arm and similar to non-racquet arm values.

Bone measures in VTP's ground-strokes arm ulna were greater than those in the serving arm, and side-asymmetries were similar to those observed in the control player group. Ground-strokes arm values were similar to both cohort racquet and non-racquet arm values, except polar moment of resistance in the ground strokes arm which was lower than control player racquet arm values. Serving arm values were lower than control player racquet arm values but similar to non-racquet arm values.

Radius bone, muscle and hand grip asymmetries in VTP were generally smaller than those in control players. Values in both VTP's arms were smaller than cohort racquet arm values and similar to cohort non-racquet arm values.

<Figure 1 about here>

DISCUSSION

This case allows examination of the effects of service and ground-strokes on upper limb adaptation to tennis. VTP had greater bone strength in the serving arm humerus and non-serving arm ulna. Most

pronounced were the 22-27% humeral side-asymmetries in bone mass, total and cortical bone CSA and 47% greater torsional stiffness (indicated by polar moment of inertia). Humerus cortical area asymmetries were over four and seven times greater than those observed in sedentary controls at 50% and 20% humerus length respectively [9]. VTP's serving arm values were similar to control players' racquet arm values and greater than non-racquet arm values. These results suggest that observed differences were the result of adaptation to physical activity and not merely usual arm dominance. When compared to control players, VTP's humeral bone side-asymmetries were similar qualitatively although slightly less pronounced. This may be in part explained by VTP's late tennis start age compared to the control group – alternatively, it is possible that ground-strokes have some minor effect on humeral bone.

Ulnar side-asymmetries in favour of the ground-strokes arm were also observed, similar to those in control players. With the exception of cortical thickness (which was similar), side-asymmetries were 70-170% larger than those observed at 50% humerus length in non-tennis players [12]. As VTP was habitually right-handed, we would expect some asymmetry in favour of the serving forearm. That the non-dominant, ground-strokes left arm was stronger is evidence that observed asymmetries are not a result of daily arm activities. However, that ground-strokes arm values were similar to racquet and non-racquet arm values of control players suggest caution in interpreting these observations. Asymmetries in radius bone, forearm muscle and grip strength were smaller than in control players and within the usual range of habitual side-asymmetries in non-tennis players [9].

These results suggest that the service stroke is the effective agent of the impressive humeral bone strength side-asymmetries in tennis players. Whilst tennis' osteogenic potential was previously

explained by the impact element of the sport, a recent article suggested that the high level of torsional loading osteogenic potential of service stroke could relate to [13]. There is increasing evidence that torsional stresses are important in bone mechanoadaptation. Torsional strains are more effective in attenuating disuse-related bone loss than axial loading in mice [14]. Similarly, turning movements (likely to engender torsional stresses) result in greater bone strength in mice than linear locomotion [15]. Substantial torsion occurs in the tibia even during walking/running [16], and when loaded via the patellar tendon *ex vivo* [17]. However, there are few habitual movements likely to load the upper limbs substantially in torsion. Common movements (*e.g.* opening doors/drawers, eating, drinking, *etc.*) produce peak shoulder rotation torques of 5-10Nm [18] – shoulder internal rotation torques during tennis serves can reach 70Nm+ and accompany similar elbow varus torques [19]. These peak torques occur around peak external shoulder rotation where substantial elbow flexion ($>90^\circ$) produces a long torsional moment arm - at impact peak torques and flexion angle are much smaller [19, 20]. Indeed, static modelling of tennis service shows large pectoralis major and latissimus dorsi forces at peak rotation acting orthogonally to the humerus [13]. In contrast, the large deltoid force at impact acts almost in alignment with the bone. Therefore it is unsurprising that modelled humeral hypertrophy and density increase is greater at peak external shoulder rotation than ball impact [13]. Evidence of humeral geometrical adaptation to torsion has been observed in tennis [4, 7] and baseball players [11], who also experience substantial shoulder rotation and elbow varus torques [21]. Baseball adaptations occur in the absence of any distinct impact event, supporting the osteogenic potential of torsional stresses. Similarly, if impact were an important factor in tennis' osteogenic potential one would expect that radius and ulna adaptation would be greater than humeral adaptation (being more

proximal to the impact site). However, radius and ulna side asymmetries in VTP and controls were much less pronounced.

In the forehand stroke, whilst similar elbow varus torques to those experienced in the serve occur, elbow pronation/supination and shoulder rotation torques and muscle activity are lower [22, 23]. There is little analysis of the backhand, although elbow flexion angle is small at impact limiting torsion. Therefore, the relative effects of tennis strokes on the forearm are less easily distinguished. Muscular activation varies between strokes – however current studies have only focused on a few muscles, preventing further current investigation.

Upper limb fractures are as prevalent as lower limb fractures in the elderly. Hence there is a clear clinical need for strategies to improve upper limb bone. The upper limbs do not use body mass as a resistor and thus do not experience the large muscular forces (e.g. four-five times bodyweight during walking [24]) that the legs are exposed to. Therefore they could be considered to be in a comparative state of disuse. This is supported by the lack of pronounced bone loss in the upper compared to lower limbs following disuse e.g. bed rest [25]. Along that line of reasoning the upper limbs would be more responsive to exercise than the lower limbs because exercising represents a greater departure from habitual loading.

Given this - and the potential of tennis and baseball to improve upper limb bone strength - it is surprising that no interventional studies involving these sports have yet been completed. Whilst frail, osteoporotic patients may not be able to take part in regular tennis matches, this case report constitutes the first step in identifying and subsequent isolating the osteogenic components of tennis play. In addition, the effectiveness of exercise for bone is not limited to interventions following a diagnosis of osteoporosis. Warden and

colleagues have recently shown that exercise benefits to bone size and strength accrued in early life persist (with some attenuation) even decades after cessation of exercise [10]. The most effective exercise interventions for bone (in terms of bone mass/density increase) are those completed in childhood [26, 27]. Hence, tennis-based interventions in children (likely better tolerated than in frail elderly) may provide a lifelong benefit to upper limb bone.

Conflicts of Interest: Alex Ireland, Hans Degens, Nicola Maffulli, and Jörn Rittweger declare that they have no conflict of interest.

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Basic Characteristics							
Variable		VTP	Conventional Players				
			Mean		95% CI		
Age (y)		46.8	46.7		42.6 - 50.8		
Height (m)		1.76	1.79		1.76 - 1.82		
Mass (kg)		78	79.5		73.2 - 85.8		
Start age (y)		13	10.8		9.2 - 12.3		
Training years ¹		34	35.9		31.5 - 40.3		
Current tennis training (h.wk ⁻¹)		6	8		4.8 - 11.2		
Childhood tennis training (h.wk ⁻¹)		10	10.3		6.6 - 14.0		
pQCT Measures							
Site	Variable	Case Study Player		Conventional Players			
		Service Arm	Ground-Stroke s Arm	Racquet Arm		Non-Racquet Arm	
				Mean	95% CI	Mean	95% CI
35% Hu me rus	Total BMC (mg.mm ⁻¹)	346.5	279.6	376.3	353 - 399.7	281.1	257.2 - 304.9
	Total CSA (mm ²)	361	297.3	383	358.7 - 407.4	303.5	278.2 - 328.7
	Cortical BMD (mg.mm ⁻³)	1217	1239	1186	1171 - 1201	1199	1182 - 1215
	Cortical CSA(mm ²)	276	218	309	288.6 - 329.4	226.9	206.4 - 247.3
	Cortical Thickness (mm)	5.52	4.71	6.23	5.83 - 6.64	4.91	4.56 - 5.27
	Periosteal Circumference (mm)	67.4	61.1	69.3	67.1 - 71.5	61.6	59.1 - 64.1
	Endocortical Circumference (mm)	32.7	31.6	30.1	27.2 - 33.0	30.7	28.3 - 33.2
	Polar Moment of Resistance (mm ³)	1655	1205	1816	1641 - 1991	1255	1085 - 1425
	Polar Moment of Inertia (mm ⁴)	20018	13592	23131	20208 - 26055	14315	11891 - 16738
60% Ul na	Total BMC (mg.mm ⁻¹)	153.6	165.4	182.6	170.7 - 194.5	162	150.4 - 173.5
	Total CSA (mm ²)	151.3	167.5	180.4	166.5 - 194.3	163	149.9 - 176.2

	Cortical BMD (mg.mm ⁻³)	1217	1202	1195	1182 - 1208	1195	1181 - 1210
	Cortical CSA(mm ²)	123	134.3	148.1	138.1 - 158.1	130.9	121.7 - 140.1
	Cortical Thickness (mm)	3.94	4.05	4.39	4.20 - 4.58	4.03	3.84 - 4.22
	Periosteal Circumference (mm)	43.6	45.9	47.5	45.7 - 49.4	45.2	43.3 - 47.0
	Endocortical Circumference (mm)	18.8	20.4	19.9	18.1 - 21.7	19.8	18.0 - 21.7
	Polar Moment of Resistance (mm ³)	418	500	553	490 - 616	481	423 - 538
	Polar Moment of Inertia (mm ⁴)	3942	4470	5751	4966 - 6536	4454	3764 - 5143
60% Radius	Total BMC (mg.mm ⁻¹)	136.4	132.7	146.5	138.2 - 154.7	129.1	121.5 - 136.8
	Total CSA (mm ²)	133.8	132.8	161.1	152.8 - 169.5	140.1	130.3 - 149.9
	Cortical BMD (mg.mm ⁻³)	1232	1233	1170	1157 - 1183	1188	1177 - 1198
	Cortical CSA(mm ²)	108	104	120.4	113.6 - 127.2	104.5	98.3 - 110.7
	Cortical Thickness (mm)	3.66	3.48	3.58	3.37 - 3.78	3.33	3.17 - 3.48
	Periosteal Circumference (mm)	41.0	40.8	45.0	43.8-46.1	41.9	40.4-43.4
	Endocortical Circumference (mm)	18.0	19.0	22.5	21.0 - 24.0	21.0	19.4 - 22.6
	Polar Moment of Resistance (mm ³)	344	365	439	417 - 460	377	344 - 410
	Polar Moment of Inertia (mm ⁴)	2940	2833	4182	3699 - 4665	3125	2708 - 3543
4% Radius	Total BMC (mg.mm ⁻¹)	157.4	152.4	187.3	174.6 - 200.0	165.2	151.5 - 178.8
	Trabecular BMD (mg.mm ⁻³)	183.4	175.5	238.2	211.6 - 264.9	233.4	209.7 - 257.1
	Total Bone CSA (mm ²)	438.3	468	497.2	467.2 - 527.1	449.9	417.2 - 482.5
Muscle/Force Side Differences (%)							
	Hand grip force (N)	378	354	519	469 - 568	431	384 - 478
	Forearm Muscle CSA (mm ²)	2021	2078	2351	2126-2576	2039	1841-2238
	Upper Arm Muscle CSA (mm ²)	3680	3855	4114	3728-4500	3712	3294-4129

Table 1. Participant (VTP) and conventional control player characteristics and pQCT scan results. ¹Calculated as (current age – start age), rounded to nearest integer.

