Physiological upper limits of left ventricular dimensions in highly trained junior tennis players

Sandeep Basavarajaiah, Mathew Wilson, Reza Naghavi, Gregory Whyte, Mike Turner, Sanjay Sharma

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See end of article for authors' affiliations

Correspondence to: Sanjay Sharma, University Hospital Lewisham, London, UK; ssharma21@hotmail.com

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Background: The differentiation between physiological cardiac enlargement and cardiomyopathy is crucial, considering that most young non-traumatic deaths in sport are due to cardiomyopathy. Currently, there are few data relating to cardiac dimensions in junior elite tennis players. The aim of this study was to define the upper limits of left ventricular dimensions in a large cohort of national adolescent tennis players.

Methods: Between 1996 and 2003, 259 adolescent tennis players (152 males), mean (SD) age 14.8 (1.4) years (range 13–19) and 86 healthy age, gender and body surface matched sedentary controls underwent 12-lead ECG and 2D-transthoracic echocardiography.

Results: Inter-ventricular septal end diastolic dimension (IVSd), left ventricular end diastolic dimension (LVEDd) and left ventricular end diastolic posterior wall dimension (LVPWd) in tennis players were significantly higher than in controls (8.9 mm vs 8.3 mm $p < 0.001$, 48.9 mm vs 47.9 mm $p < 0.05$ and 9 mm vs 8.3 mm $p < 0.001$ respectively), however in absolute terms, the difference did not exceed 7%. None of the tennis players had a wall thickness exceeding 12 mm or a left ventricular cavity size exceeding 60 mm.

Conclusions: Tennis players exhibit modest increases in cardiac dimensions, which do not resemble those seen in individuals with cardiomyopathy affecting the left ventricle.

The differentiation between physiological cardiac adaptation (athlete's heart) and cardiomyopathy is crucial, as up to 40% of all non-traumatic sudden cardiac deaths in young athletes are either due to hypertrophic or dilated cardiomyopathy.^{1,2} The vast majority of echocardiographic studies have evaluated only adult athletes, however, there are few data assessing physiological adaptation in adolescent athletes in whom sudden death from cardiomyopathy is most prevalent.³ The steady trickle of sudden cardiac deaths in high profile athletes including tennis players^{4,5} has prompted some sporting bodies to implement compulsory cardiovascular evaluation of all junior recruits specifically aimed at excluding cardiomyopathy prior to acceptance for competition.

Tennis is a popular sport attracting millions of players and fans worldwide.⁶ The British Lawn Tennis association was the first elite sporting organisation in the UK to adopt cardiovascular screening of all their national junior athletes for conditions predisposing to sudden cardiac death. Our group has been responsible for performing cardiovascular evaluation on all junior national tennis players since 1996. The aim of this study was to identify physiological upper limits of cardiac enlargement in junior tennis players to help facilitate the differentiation between physiological and pathological cardiac enlargement should other countries decide to follow suit.

METHODS

Athletes

Between August 1996 and May 2003, 259 elite, adolescent tennis players from the British Lawn Tennis Association underwent 12-lead ECG and 2-dimensional echocardiography preceded by a full cardiovascular evaluation during the peak competitive season. All athletes competed at regional level and trained for an average (SD) of 10.1 (3.8) h/week (range 5–24 h). The athletes were aged 13–19 years (mean (SD) 14.8 (1.4) years). A total of 152 athletes (59%) were male and 107 (41%) were female. Mean (SD) body surface area (BSA) among the athletes was 1.6 (0.2) m² (range 1.2 to 2.2 m²). None of the

athletes had symptoms of underlying cardiovascular disease or a family history of premature sudden death (aged <50 years) from heart disease. Written consent for cardiovascular evaluation was obtained from subjects aged ≥ 16 years and from a parent/guardian in those aged <16 years old. All subjects were judged to be peri-pubertal based upon the onset of menstruation in female and deepening of voice or the presence of obvious secondary sexual characteristics (facial and body hair) in males.

Ethical approval for the study was sought from the Harrow Research Ethics Committee by the Cardiac Risk in the Young (CRY), Centre of Sports Cardiology.

Controls

The control population comprised of 86 healthy adolescent volunteers who were students at two large secondary education boarding schools. All individuals selected had a relatively sedentary lifestyle, defined as <2 h of organised physical activity a week. The controls were matched with athletes with respect to age (15.7 (1.4) years), gender (66% male) and BSA (1.7 (0.2) m²).

Echocardiography

Two-dimensional echocardiography was performed using an Acuson Computed Sonograph 128XP/10c (San Jose, California, USA) with 3 MHz transducer. Images of the heart were obtained in the standard parasternal long axis, short axis and apical four chamber planes. The LV wall thickness measurements were made in end-diastole from 2-dimensional short axis views at the level of the mitral valve and papillary muscle level. Measurements of the interventricular septum and the posterior wall were used to assess left ventricular wall thickness and the greatest measurement was defined as the maximal wall thickness. The M-mode

Abbreviations: BSA, body surface area; IVSd, inter-ventricular septal end diastolic dimension; LVEDd, left ventricular end diastolic dimension; LVH, left ventricular hypertrophy; LVPWd, left ventricular end diastolic posterior wall dimension

Table 1 Characteristics of tennis players and normal controls

	Athletes	Controls	p Value
Age (years)	14.8 (1.4) (13–19)	15.7 (1.4) (13–18)	NS
BSA (m ²)	1.6 (0.2) (1–2.2)	1.6 (0.2) (1.2–2.3)	NS
Heart Rate (bpm)	54 (6) (40–78)	71 (11) (48–96)	<0.001
Systolic Blood Pressure (mmHg)	108 (11) (70–150)	113 (9) (90–126)	<0.001
IVSd (mm)	8.9 (1.3) (6–12)	8.3 (1.3) (6–11)	<0.001
LVPWd (mm)	9 (1.3) (6–12)	8.3 (1.3) (5–11)	<0.001
LVEDd (mm)	48.9 (3.9) (32–59)	47.9 (3.5) (37–54)	<0.05
LVESd (mm)	31.6 (4.1) (22–42)	31.9 (4.2) (22–46)	NS
LVM (g)	207 (62) (101–323)	155 (50) (50–248)	<0.001
FS (%)	33.1 (4) (31–40)	31.9 (4) (30–39)	NS
EF (%)	72 (13) (56–100)	72 (11.8) (55–92)	NS
E-wave (m/s)	0.9 (0.2) (0.5–1.7)	0.9 (0.2) (0.6–1.8)	NS
A-wave (m/s)	0.4 (0.1) (0.2–0.9)	0.5 (0.2) (0.2–0.8)	=0.005
E/A ratio	2.3 (0.7) (1.0–5.5)	2.1 (0.2) (1.2–4.5)	NS

Values are mean (SD) and (range).

BSA, body surface area; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; IVSd, interventricular septal end diastolic dimension; LVPWd, left ventricular end diastolic posterior wall dimension; LVEDd, left ventricular end diastolic dimension; LVESd, left ventricular end systolic dimension; LVM, left ventricular mass; FS, Fractional shortening.

echocardiograms derived from 2D images in the parasternal long axis were used for the measurement left ventricular end-diastolic and systolic dimensions, left atrial diameter and aortic root according to American Society of Echocardiography standards.^{7,8} Fractional shortening and ejection fraction of the left ventricle were considered as an index of systolic function. Pulsed Doppler recordings performed at the distal margins of mitral valve leaflets provided index of diastolic function. The predicted upper limits for left ventricular wall thickness and cavity size were derived from the control population. A value exceeding 2 SD from the mean was regarded above the physiological upper limit. Based upon these a left ventricular end-diastolic wall thickness >11 mm and a left ventricular end-diastolic cavity size >54 mm were considered to exceed predicted upper limits and represent left ventricular hypertrophy (LVH) and left ventricular cavity dilatation respectively.

Electrocardiogram

A standard 12-lead ECG examination was performed during quiet respiration in a supine position and analysed using a Marquette Hellige (Milwaukee, Wisconsin, USA) ECG recorder. The electrodes were placed carefully to ensure consistency of the precordial lead locations and ECGs were recorded at a paper speed of 25 mm/s. Left ventricular hypertrophy was defined by the Sokolow–Lyon voltage amplitude of (SV1 + RV5 or SV1 + RV6 ≥35 mV)

Statistical analysis

Data are expressed as the mean (SD) (range). Statistical analyses comparing athletes and non-athletes were performed using unpaired Student t test, univariate analysis of variance test with post hoc (Bonferroni) and χ^2 test. p Value of <0.05 was considered statistically significant. In the overall population of 259 athletes, a multivariable linear model was used to assess the relation between LV wall thickness and LV cavity size

as a dependent variable and body surface area, age, gender as independent variables.

RESULTS

The demographics and cardiac dimensions of tennis players and normal controls are shown in table 1. Tennis players had lower heart rate, systolic and diastolic blood pressure readings compared to controls.

LV wall thickness

Athletes had significantly greater interventricular septal and LV-posterior wall thickness compared with controls. However, in terms of absolute measurements, the difference in septal wall thickness and LV posterior wall thickness between tennis players and controls was only (7%) and within resolution limits. The absolute wall thickness measurements in tennis players ranged from 6–12 mm. The distribution of wall thickness measurements in tennis players is shown in fig 1. Male tennis players had greater wall thickness measurements compared with female tennis players (table 2). None of the control group or female tennis players had a wall thickness >11 mm (6–11 mm). A wall thickness >11 mm was considered to represent LVH (see methodology). Only three male athletes (1.2%), all aged >15 years, had a LV wall thickness >11 mm (table 3). The pattern of LVH observed in the 3 athletes was symmetrical, with no athlete showing a difference of >2 mm in left ventricular wall thickness measurements between contiguous segments of the wall. All three athletes had an enlarged left ventricular cavity and normal indices diastolic function on Doppler studies implying the hypertrophy was physiological rather than hypertrophic cardiomyopathy.

LV cavity size

Athletes had a significantly greater end diastolic LV cavity size as compared with the control group (48.9 vs 47.9 p<0.05). The

Figure 1 Distribution of left ventricular wall thickness in elite junior players.

distribution of the LV cavity size in adolescent tennis players is as shown in fig 2. None of the control group had LV cavity size >54 mm, However 6 (3.9%) male and 2 (1.9%) female athletes had a LV cavity exceeding 54 mm. All 8 athletes with increased LV cavity size had preserved systolic function.

Distribution of LV cavity size in male and female tennis players divided according to different age group are shown in table 4. No athlete with aged <14 years had a LV cavity dimension >54 mm. A multivariable linear model that was used to assess the relation between LV dimensions and other demographic variables (age, gender and body surface area) demonstrated an independent association between the two.

Electrocardiography

The Sokolow–Lyon voltage criterion for LVH was present in 117 (45%) of athletes and 20 (23%) of non-athletes. Of the 117 athletes with Sokolow–Lyon voltage criteria for LVH, only 3 (2.5%) had echocardiographic criteria for LVH. None of the non-athletes had echocardiographic evidence of LVH. The voltage criterion for LVH was positive in all individuals with echocardiographically proven LVH. No athletes had minor or deep T-wave inversions or ST-segment depression in the lateral leads (V5–V6, I or aVL), to indicate pathological LVH. There was a poor correlation between LV mass and the amplitude of R-wave on the 12-lead ECG.

DISCUSSION

Regular participation in systematic physical training is associated with a modest increase in cardiac dimensions.^{9–12} A small proportion of adult athletes develop substantial LVH or cavity enlargement simulating the diagnosis of hypertrophic or dilated cardiomyopathy respectively.¹³ There are few data evaluating upper limits of cardiac dimensions in adolescent tennis players but data derived from adult athletes cannot be extrapolated to younger athletes who are physically less mature and trained for shorter periods.

Figure 2 Distribution of left ventricular cavity size in elite junior players.

Our study revealed that tennis players have modestly increased cardiac dimensions compared with non-athletes. These findings are not dissimilar to those identified in adolescent football players but are slightly smaller when compared with adolescent rowers.^{10–12} In absolute terms, however, almost all tennis players had cardiac dimensions within normal limits for the general population. Only 3 athletes were considered to have LVH, defined as a left ventricular wall thickness >11 mm, however none of the 3 athletes had wall thickness >12 mm. All 3 athletes were male and aged >15 years indicating that gender and age were important determinants of cardiac dimensions in adolescent athletes. Therefore, the magnitude of hypertrophy was significantly less than that usually observed in individuals with HCM. Furthermore the 3 athletes with LVH had symmetric hypertrophy, enlarged left ventricular size, and normal indices of diastolic function supporting physiological (athlete’s heart) hypertrophy as opposed to HCM, which is characterised by a non-dilated left ventricular cavity and impaired myocardial relaxation.

A significant proportion of our male athletes fulfilled Sokolow–Lyon voltage criteria for LVH, a finding that is common in young male athletes and has poor correlation with echocardiographic LVH. The most probable explanation for the high QRS voltages in males is the thin chest walls resulting in reduced distance between the myocardium and the chest wall surface. None of our athletes exhibited pathological Q waves, ST segment depression or deep T wave inversions to indicate cardiac pathology

A slightly higher proportion of tennis players had an enlarged left ventricular cavity, which has also been reported in

Table 2 Comparison between male and female adolescent tennis players

	Male	Female	p Value
IVSd (mm)	9.4 (1.2) (6–12)	8.3 (1.0) (6–11)	<0.001
LVPWd (mm)	9 (1.3) (6–12)	8.5 (1.1) (6–11)	<0.001
LVEDd (mm)	49.9 (3.8) (40–59)	47.5 (3.5) (32–58)	<0.001
LVESd (mm)	32.5 (4.2) (22–40)	30.4 (3.7) (22–42)	<0.001
LVM (g)	226.6 (69) (141–323)	176.2 (51) (101–260)	<0.001

Values are mean (SD) and (range).
HR, heart rate; IVSd, interventricular septal end diastolic dimension; LVPWd, left ventricular end diastolic posterior wall dimension; LVEDd, left ventricular end diastolic dimension; LVESd, left ventricular end systolic dimension.

Table 3 Distribution of LV wall thickness in different age groups of male and female tennis players

Age (years)	No of players	Male	Female	LVWTd (male)	LVWTd (female)
13	53	28	25	8.5 (1.3) (7–11)	7.9 (1.1) (7–10)
14	66	36	30	9.2 (1.2) (8–10)	8.2 (0.9) (7–10)
15	66	34	32	9 (1.4) (6–11)	8 (1.1) (7–10)
16	42	33	9	9.6 (1) (8–12)	7.9 (0.7) (7–9)
17	19	13	6	10 (0.7) (9–11)	8.8 (0.9) (8–10)
18	11	9	2	10 (0.6) (9–12)	9 (0.8) (9–11)
19	2	1	1	–	–

Table 4 Distribution of LV cavity size in different age groups of male and female tennis players

Age (years)	No of players	Male	Female	LVIDd (male)	LVIDd (female)
13	53	28	25	45.4 (3.6) (41.4–52.7)	45.3 (3.3) (39–51)
14	66	36	30	48.8 (3.5) (40–59)	47.9 (2.4) (43.8–54)
15	66	34	32	50 (4.1) (40.5–56.7)	47.7 (3.8) (32–55)
16	42	33	9	51.08 (3.2) (44–59)	48.6 (4.7) (45–58)
17	19	13	6	53.1 (2.5) (49–59)	49.2 (2) (45–51)
18	11	9	2	53 (2.2) (49–55)	50 (2.8) (48–52)
19	2	1	1	–	–

adolescent football players, swimmers, rowers and rugby players and probably represents a large isotonic component to the sporting discipline. A total of 3% of our athletes were

considered to have an enlarged LV cavity size but none had a left ventricular cavity exceeding 60 mm or associated impairment of systolic function to indicate the diagnosis of dilated cardiomyopathy.

We relied on absolute measurements of LV dimensions (rather than values normalised to body surface area) so that our observations could be placed directly in the context of clinical cardiovascular diagnosis. Nevertheless, our multivariable analysis defined body surface area, age and gender to be independent determinants of LV dimensions.

CONCLUSIONS

Junior elite tennis players exhibit modest increases left ventricular wall thickness and cavity size. Absolute values rarely exceed predicted normal upper limits and do not generally resemble those seen in individuals with cardiomyopathy affecting the left ventricle.

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Authors' affiliations

Sandeep Basavarajiah, Reza Naghavi, Sanjay Sharma, University Hospital Lewisham, London, UK

Mathew Wilson, Gregory Whyte, Olympic Medical Institute, London, UK

Mike Turner, British Lawn Tennis Association, London, UK

Competing interests: None declared.

What is already known on this topic

Adult athletes demonstrate a modest increase in the left ventricular (LV) dimensions secondary to exercise as compared to non-athletes but there is limited data on upper limits of LV dimensions in highly trained adolescent athletes.

What this study adds

- Adolescent tennis players exhibit modest increases in the LV dimensions. Absolute values of LV dimensions rarely exceed predicted normal upper limits and do not generally resemble those seen in individuals with cardiomyopathy.
- The upper limits of LV wall thickness and cavity size in adolescent tennis players in our study is 12 mm and 60 mm respectively.

REFERENCES

- 1 **Maron BJ**. Sudden death in young athletes. *N Engl J Med* 2003;**349**:1064–75.
- 2 **Maron BJ**, Epstein SE, Roberts WC. Causes of death in competitive athletes. *J Am Coll Cardiol* 1986;**7**:204–14.
- 3 **Maron BJ**, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes: clinical, demographic, and pathological profiles. *JAMA* 1996;**276**:199–204.
- 4 **Fornes P**, Lecomte D. Sudden death and physical activity and sports. *Rev Prat* 2001;**30**:S31–5.
- 5 **Eichner ER**. Sudden death in racquet sports. *Clin Sports Med* 1988;**7**:245–52.
- 6 **BM Pluim**, Turner M. Fuzzy balls: *Br J Sports Med* 2006;**40**:378.
- 7 **Henry WL**, DeMaria A, Gramiak R, et al. Report of the American Society of Echocardiography Committee on Nomenclature and Standards in Two-dimensional Echocardiography. *Circulation* 1980;**62**:212–7.
- 8 **Sahn DJ**, DeMaria A, Kisslo J, et al. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation* 1978;**58**:1072–83.
- 9 **Pelliccia A**, Maron BJ, Spataro A, et al. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N Engl J Med* 1991;**324**:295–301.
- 10 **Sharma S**, Maron BJ, Whyte G, et al. The physiologic limits of left ventricular hypertrophy in elite junior athletes. *J Am Coll Cardiol* 2002;**40**:1431–6.
- 11 **Pelliccia A**, Culasso F, Di Paolo FM, et al. Physiologic left ventricular cavity dilation in elite athletes. *Ann Intern Med* 1999;**130**:23–31.
- 12 **Makan J**, Sharma S, Firoozi S, et al. The physiologic limits of ventricular cavity size in highly trained adolescent athletes. *Heart* 2005;**91**:495–9.
- 13 **Maron BJ**, Pelliccia A, Spirito P. Cardiac disease in young trained athletes. Insights into methods for distinguishing athletes heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. *Circulation* 1995;**91**:1596–1601.

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