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William J. McKenna

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Left Ventricular Hypertrophy in Athletes

Physiologic Limits of Left Ventricular Hypertrophy in Elite Junior Athletes: Relevance to Differential Diagnosis of Athlete's Heart and Hypertrophic Cardiomyopathy

Sanjay Sharma, MD,*† Barry J. Maron, MD,‡ Greg Whyte, PhD,§ Sami Firoozi, MD,†
Perry M. Elliott, MD,† William J. McKenna, MD†

London and Walsall, United Kingdom; and Minneapolis, Minnesota

OBJECTIVES	The present study was undertaken to define physiologic limits of left ventricular hypertrophy in elite adolescent athletes.
BACKGROUND	Systematic sports training may cause increased left ventricular wall thickness (LVWT), creating uncertainty regarding the differential diagnosis of athlete's heart from hypertrophic cardiomyopathy (HCM). This distinction is crucial because HCM is responsible for about one-third of all sudden deaths in young athletes. Echocardiographic data defining athlete's heart are limited largely to adults, with little information specifically in adolescent athletes (14 to 18 years old), for whom the risk of sudden death from HCM is highest.
METHODS	Seven hundred and twenty elite adolescent athletes (75% male) aged 15.7 ± 1.4 years participating in ball, racket, and endurance sports and 250 healthy sedentary controls of similar age, gender, and body surface area underwent echocardiography.
RESULTS	Compared with controls, athletes had greater absolute LVWT (9.5 ± 1.7 mm vs. 8.4 ± 1.4 mm; $p < 0.0001$). Maximal LVWT exceeded predicted upper limits in 38 athletes (5%); however, no female athlete had a LVWT >11 mm and only three trained male athletes had absolute LVWT >12 mm (0.4%). Each of the 38 athletes with a LVWT exceeding predicted limits also showed enlarged left ventricular cavity dimension (54.4 ± 2.1 mm; range 52 to 60 mm).
CONCLUSIONS	Trained adolescent athletes demonstrated greater absolute LVWT compared with nonathletes. Only a small proportion of athletes exhibited a LVWT exceeding upper limits, very rarely >12 mm, and then always with chamber enlargement. Hypertrophic cardiomyopathy should be considered strongly in any trained adolescent male athlete with LVWT >12 mm (females >11 mm) and nondilated left ventricle. (J Am Coll Cardiol 2002;40:1431-6) © 2002 by the American College of Cardiology Foundation

Regular intensive physical training in some endurance sports is associated with a physiologic increase in left ventricular wall thickness (LVWT), cavity size, and mass (1-7). In a small proportion of trained adult athletes, the magnitude of LVWT may be comparable to that encountered in some patients with mild morphologic expression of hypertrophic cardiomyopathy (HCM) (8-10). The clinical distinction between physiologic increases in LVWT in athletes (i.e., athlete's heart) and HCM is crucial because the latter accounts for about one-third of all exercise-related sudden cardiac deaths in trained athletes aged <35 years old (11-13), and disqualification from intense competitive sports is recommended (14).

Most exercise-related sudden cardiac deaths in athletes

from HCM occur during adolescence (14 to 18 years old) (15). However, paradoxically, definition of the normal upper limits of physiologic hypertrophy comprising part of the athlete's heart syndrome has largely been based on echocardiographic studies performed in adult athletes (8,16,17). Reference values for LVWT derived from adult athletes (2,8,9,18) cannot be explicitly extrapolated to younger athletes, who are less physically mature and are exposed to shorter periods of intense training, for differentiating physiologic LVH from HCM (19). Therefore, the present study was undertaken to define the physiologic limits of LVWT in elite adolescent athletes.

METHODS

Selection of athletes. Between August 1996 and November 2000, 720 elite adolescent athletes (age 15.7 ± 1.4 years; range 14 to 18 years old) underwent two-dimensional (2D) echocardiography during the peak competitive season. Written consent for cardiovascular evaluation was obtained from individuals aged ≥ 16 years and from a parent/guardian of those <16 years old. Five hundred and forty

From the *Department of Cardiology, University Hospital Lewisham, London, United Kingdom; †Department of Cardiological Sciences, St George's Hospital Medical School, Cranmer Terrace, London, United Kingdom; ‡Minneapolis Heart Institute Foundation, Minneapolis, Minnesota; and §University of Wolverhampton, Division of Sports Studies, Walsall Campus, Walsall, United Kingdom. Sanjay Sharma and Sami Firoozi were supported by a grant from the British Heart Foundation.

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Abbreviations and Acronyms

HCM	= hypertrophic cardiomyopathy
h/R	= relative wall thickness
LV	= left ventricular
LVH	= left ventricular hypertrophy
LVWT	= left ventricular wall thickness
2D	= two-dimensional

athletes were male (75%), 702 (98%) were Caucasian, and 2% were black. Body surface area was $1.74 \pm 0.17 \text{ m}_2$ (range 1.09 to 2.24 m_2).

Ten sporting disciplines predominantly made up the study group: boxing, cycling, field hockey, karate, rowing, rugby, soccer, swimming, tennis, and triathlon. Tennis and soccer were the most commonly studied sports, and between them accounted for 333 (46%) athletes (Table 1). Tennis players were recruited from the British Lawn Tennis Association, soccer players from youth teams at clubs in the British Premier Soccer League, boxers from prominent amateur boxing association clubs, rugby players from British Rugby League clubs, cyclists from large county cycling squads, triathletes from top-10 finishers at the national U.K. championships in 1997 and 1998, and rowers, swimmers, and hockey and karate players from the U.K. Junior National team. An additional 15 athletes (2%) had participated at the national level in squash (n = 3), fencing (n = 2), and track and field events (n = 10).

All athletes had competed at the regional level for 4.3 ± 1.5 years (range 1 to 10 years) and 50% were competing at the national level at the time of this study. The number of hours of intensive training (assessed by questionnaire) averaged 9.8 ± 3.6 h/week (range 5 to 27 h). No athlete had symptoms of underlying cardiovascular disease or a family history of premature death from heart disease; none had blood pressure >125 mm Hg systolic or >85 mm Hg diastolic.

Controls. The control group comprised 250 healthy adolescent volunteers who were students at a large secondary education boarding school. All individuals led a relatively

Table 1. Demographic Characteristics of 720 Elite Adolescent Athletes Within Each Sporting Discipline

Sport	Total	%	M	F	Age	BSA
Tennis	175	24	105	70	15.4 ± 1.2	1.7 ± 0.8
Soccer	158	22	158	0	16.0 ± 1.2	1.8 ± 0.5
Swimming	83	12	38	45	15.5 ± 0.9	1.7 ± 0.2
Boxing	60	8	60	0	16.1 ± 0.8	1.7 ± 0.3
Rowing	60	8	40	20	16.3 ± 0.9	1.9 ± 0.3
Rugby	60	8	60	0	15.8 ± 0.7	1.7 ± 0.1
Cycling	40	6	40	0	15.6 ± 1.2	1.7 ± 0.2
Triathlon	29	4	14	15	16.6 ± 1.1	1.7 ± 0.2
Field hockey	22	3	0	20	15.8 ± 1.1	1.7 ± 0.3
Karate	20	3	20	0	15.6 ± 0.9	1.7 ± 0.3
Others*	15	2	5	10	15.8 ± 0.7	1.7 ± 0.2

*Track and field events (n = 10), squash (n = 3), fencing (n = 2)
BSA = body surface area.

Table 2. Predicted Upper Limits of LVWT Derived From 250 Healthy Non-Athletic Controls

Age (yrs)	Males		Females	
	Mean \pm SD (mm)	Upper Limit (mm)	Mean \pm SD (mm)	Upper Limit (mm)
14	8.4 ± 1.4	11	7.0 ± 1	9
15	9.0 ± 1.0	11	7.5 ± 1.3	10
16	9.0 ± 1.0	11	8.0 ± 1.2	10
17	9.4 ± 1.2	12	8.7 ± 1.2	11
18	10.2 ± 0.9	12	8.5 ± 1.3	11

LVWT = left ventricular wall thickness.

sedentary lifestyle, defined as <2 h of organized physical activity per week. Controls were of similar age, gender, and body surface area as the trained athletes (15.5 ± 1.2 years [range 14 to 18]; 70% male; $1.70 \pm 0.20 \text{ m}_2$ [range 1.17 to 2.24] respectively; NS).

Echocardiography. Two-dimensional echocardiography was performed by two experienced technicians, with the subjects resting in a left lateral decubitus position, using an Acuson Computed Sonograph 128XP/10c (San Jose, California) with 3 MHz transducer. Images of the heart were obtained in the standard parasternal long-axis and short-axis and apical four-chamber planes, as previously described (20). The LVWT was measured from 2D short-axis views at end-diastole, with the greatest measurement within the left ventricular (LV) wall defined as the maximal wall thickness.

M-mode echocardiograms derived from 2D images in the parasternal long axis were used for the measurement of LV end-diastolic and systolic dimensions, left atrial diameter, and aortic root according to American Society of Echocardiography standards (21). Three to five consecutive measures were made and the average was taken by a single experienced sonographer (S.S.) blind to the identity of the subjects.

Percent LV percentage shortening fraction was calculated as an index of systolic function. Pulsed Doppler recordings were performed at the distal margins of mitral valve leaflets to provide an index of diastolic function (22). Relative wall thickness (h/R) was calculated by dividing the sum of the septal and posterior wall thickness at end-diastole (h) by the LV end-diastolic diameter (R) (23). Left ventricular mass was calculated from the LV cavity size and wall thickness in end diastole by the formula of Devereux (24).

Predicted upper limits for normal values for the LVWT were derived from the control group. The mean LVWT measurements were calculated separately for males and females in each age group. The predicted upper values were defined as two or more standard deviations from the mean (Table 2).

We relied largely on absolute LVWT measurements. However, for selective comparisons of gender, age, and different sporting disciplines, wall thickness and mass were corrected for differences in body surface area (25,26).

Table 3. Demographic and Echocardiographic Parameters in 720 Adolescent Athletes Compared to 250 Non-Athletic Controls*

	Athletes	Controls	% Difference	p Value
Age (yrs)	15.7 ± 1.4 (14-18)	15.5 ± 1.2 (14-18)	1	NS
BSA (m ⁻²)	1.74 ± 0.17 (1.09-2.23)	1.70 ± 0.2 (1.17-2.24)	2	NS
VS (mm)	9.4 ± 1.3 (6-14)	8.2 ± 1.5 (6-11)	13	< 0.0001
LVPW (mm)	9.3 ± 1.3 (6-13)	8.3 ± 1.3 (5-11)	11	< 0.0001
LVWT (mm)	9.6 ± 1.3 (6-14)	8.5 ± 1.3 (6-11)	13	< 0.0001
h/R ratio	0.36 ± 0.04 (0.26-0.5)	0.35 ± 0.05 (0.15-0.48)	3	NS
LVEDD (mm)	50.8 ± 3.7 (40-60)	47.9 ± 3.5 (37-54)	6	< 0.0001
LVMI (gm ⁻²)	113 ± 33.3 (29-232)	86.3 ± 24.7 (24-165)	31	< 0.0001
LA (mm)	32.7 ± 4.8 (19-45)	30.9 ± 5 (20-40)	5	0.0002
Ao (mm)	28.7 ± 3.5 (17-40)	27.1 ± 3.5 (19-33)	6	NS
LVSF (%)	33.1 ± 3.5 (31-40)	31.9 ± 3.9 (30-39)	4	NS
E-wave (m/s)	0.88 ± 0.16 (0.5-1.7)	0.87 ± 0.2 (0.6-1.8)	1	NS
A-wave (m/s)	0.41 ± 0.1 (0.2-0.9)	0.45-0.16 (0.2-0.8)	-9	0.005
E/A ratio	2.25 ± 0.7 (1.0-5.5)	2.14 ± 0.16 (1.2-4.5)	5	NS

*Expressed as mean ± SD with p values reflecting unpaired Student *t* test.

Ao = aortic diameter; BSA = body surface area; h/R = relative wall thickness; LA = left atrial diameter; LVEDD = left ventricular end-diastolic dimension; LVMI = left ventricular mass index; LVSF = left ventricular shortening fraction; LVPW = left ventricular posterior wall thickness; LVWT = left ventricular wall thickness; NS = not specified; VS = ventricular septal thickness.

Electrocardiography. Electrocardiograms (ECGs) were recorded on all athletes at the time of echocardiography with a Marquette Hellige recorder (Milwaukee, Wisconsin) (27). From the ECGs, Sokolow-Lyon voltage criteria (sum of the S-wave in V₁ and R-wave in V₅ >3.5 mV) (28) and the Romhilt and Estes point score system (≥5 points) (29) were used to identify LVH.

Statistical analysis. Data are expressed as mean ± SD. Statistical analyses were performed using unpaired Student *t* test, univariate analysis of variance test with post hoc (Bonferroni) and chi-square test. In the overall population of 720 athletes, a multivariable linear model was used to assess the relation between LVWT as a dependent variable and body surface area, age, gender, and type of sport as independent variables. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

Cardiac dimensions in elite adolescent athletes. Athletes had a significantly greater maximal LVWT, end-diastolic cavity size, and mass as well as left atrial diameter, when compared with nonathletic controls (Table 3). There was no difference in h/R between the two groups (Table 3). Percentage differences in LVWT, cavity size, and atrial diameter were 13%, 11%, 6%, and 5%, respectively. Absolute LVWT in athletes ranged from 6 to 14 mm (Fig. 1). In the athlete group, males had greater dimensions than females even after correction for body size (Table 4).

Athletes with wall thickness exceeding predicted limits.
DEMOGRAPHICS. Of the 720 elite athletes, 38 (5%) had LVWT that exceeded predicted upper limits, including 6% of male and 5% of female athletes. The 38 athletes with a LVWT greater than predicted were represented by each of the sports in the study group, but most commonly rowing (n = 9), soccer (n = 6), and swimming (n = 5).

In the 38 athletes with a greater than predicted LVWT,

the absolute LVWT measurements ranged from 11 mm to 14 mm. Of these 38 athletes, 35 (4% of all 720 athletes) had a LVWT ≥12 mm but only three (0.4% of the 720 athletes) had LVWT >12 mm in absolute terms, and consistent with HCM. Each of these three were male; two were engaged in rowing and one in tennis. All female athletes had a LVWT of ≤11 mm.

CARDIAC MORPHOLOGY. In those 38 athletes with a greater than predicted LVWT, the pattern of LVH was concentric (symmetric), with no athlete showing >2 mm difference in LVWT measurements between contiguous segments of the wall. Each of these athletes also had a greater than predicted LV end-diastolic cavity size (i.e., 2 standard deviations from the mean calculated from the control population) (54.4 ± 2.1 mm; range 52 mm to 60 mm). Also each athlete had normal mitral inflow velocity patterns (Table 3).

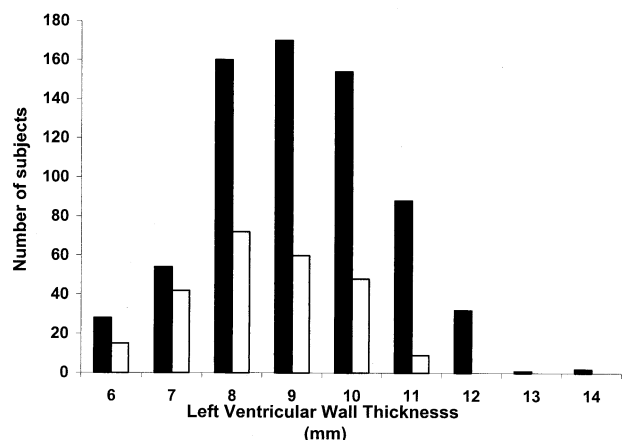


Figure 1. Distribution of left ventricular wall thicknesses in 720 junior elite athletes (black bars) and 250 controls (white bars).

Table 4. Gender Differences in Cardiac Dimensions for 720 Elite Adolescent Athletes

	Males	Females	p Value
Age	15.6 ± 1.2	15.4 ± 1.12	NS
BSA (m ⁻²)	1.77 ± 0.16 (1.34-2.23)	1.65 ± 0.13 (1.33-1.97)	< 0.001
VS (mm)	9.5 ± 1.3 (6-14)	8.3 ± 1.1 (6-11)	< 0.001
LVPW (mm)	9.5 ± 1.2 (6-13)	8.6 ± 1.2 (6-11)	< 0.001
LVWT (mm)	9.8 ± 1.2 (7-14)	8.4 ± 1.1 (6-11)	< 0.001
LVWTI (mm/m ⁻²)	7.1 ± 0.8 (4.5-9.9)	6.8 ± 0.8 (4.6-8.5)	< 0.001
LVEDD (mm)	51.6 ± 3.3 (42-60)	47.7 ± 3.3 (41-55)	< 0.001
LVM (g)	211 ± 65 (42-465)	160 ± 50 (54-268)	< 0.001
LVMi (gm ⁻²)	90 ± 25 (24-165)	75 ± 21 (26-117)	< 0.001
LA (mm)	33.3 ± 3.8 (19-45)	31.1 ± 4.5 (20-42)	< 0.001

BSA = body surface area; LA = left atrial diameter; LVPW = left ventricular wall thickness; LVWT = maximal left ventricular wall thickness; LVWTI = maximal left ventricular wall thickness index (wall thickness/BSA^{1/2}); LVEDD = left ventricular end-diastolic dimension; NS = not specified; VS = ventricular septal thickness.

ELECTROCARDIOGRAPHY. Of the 38 athletes with increased LVWT, 31 (82%) fulfilled either Sokolow-Lyon voltage criteria (28) or Romhilt-Estes points score (29) for LVH. Two male athletes had minor T-wave inversions (<0.2 mV) in the inferior leads. Another 236 athletes (33%) also showed Sokolow voltage criteria for LVH but had normal criteria wall thickness. Deep T-wave inversions, pathologic Q-waves (>0.04 s in duration or >25% of the height of the ensuing R-wave) and ST segment depression were absent in all 720 athletes.

Determinants of LVWT. A multivariable linear model was used to assess the relation between LVWT and several demographic variables: body surface area, age, gender, type of sport, duration of training, and athletic achievement. After multivariable adjustment, an independent association was found between LVWT and body surface area, age, male gender, and type of sport (such as rowing in male athletes) (p < 0.05) (Table 5).

DISCUSSION

The differential diagnosis between athlete's heart and HCM represents a vital clinical dilemma because at least 10% of adolescent patients with HCM may be at unacceptably high risk for sudden cardiac death (30,31). For example, a false positive diagnosis of HCM would result in unnecessary

disqualification from sport (14), thereby potentially depriving the athlete of the many physical, psychologic, and financial benefits of athletic competition. Conversely, an erroneous (false negative) diagnosis of athlete's heart could jeopardize a young life. Indeed, given the therapeutic strategies now available to prevent sudden death, such as the implantable cardioverter-defibrillator (31), the necessity for earlier identification of high-risk HCM patients is now magnified. Furthermore, accurate diagnosis of HCM offers the opportunity to disqualify such individuals from intense competitive sports in order to reduce their risk (14).

In this regard, the present study of more than 700 elite adolescent athletes shows that an important minority have an absolute increase in maximum LVWT. Whereas our athletes showed a range in LVWT of up to 14 mm, just 4% of the overall group was >11 mm, and only < 0.5% were 13 mm or 14 mm (all male). Given these observations and the fact that none of the nonathlete controls demonstrated LVWT >11 mm, we consider any trained adolescent athlete with a LVWT >11 mm to probably have LVH, justifying consideration for the diagnosis of HCM. Furthermore, because none of the 720 subjects studied had a LVWT >14 mm, it is also reasonable to infer that a LVWT of 15 mm or more in a highly trained adolescent athlete probably represents HCM until proven otherwise.

Table 5. LVWT and LVMi Within Each Sporting Discipline for 720 Elite Adolescent Athletes

Sport	LVWT (mm)		LVWTI (mm/m ²)		LVMi (g/m ²)	
	M	F	M	F	M	F
Rowing	11 ± 1.0*	9.3 ± 1.0*	7.8 ± 0.7*	7.1 ± 0.8	138 ± 39*	94 ± 31
Triathlon	10 ± 1.0	8.0 ± 1.0	7.3 ± 0.7	6.7 ± 0.9	117 ± 16	99 ± 29
Swimming	9.2 ± 1.0	8.0 ± 1.0	7.1 ± 0.9	6.8 ± 0.9	118 ± 31	101 ± 31
Boxing	9.1 ± 0.8	—	7.2 ± 0.7	—	118 ± 36	—
Cycling	9.0 ± 1.0	—	7.3 ± 0.6	—	118 ± 32	—
Soccer	9.0 ± 1.0	—	7.3 ± 0.7	—	119 ± 32	—
Rugby	9.0 ± 1.0	—	7.3 ± 0.7	—	—	—
Tennis	9.0 ± 1.0	8.3 ± 0.7	7.4 ± 0.7	6.8 ± 0.8	115 ± 33	—
Karate	8.8 ± 0.8	—	7.0 ± 0.7	—	110 ± 28	—
Field hockey	—	8.0 ± 1.0	—	—	—	98 ± 30

*Significantly greater than in other athletes (p analysis of variance < 0.001).

LVWT = maximal left ventricular wall thickness; LVWTI = left ventricular wall thickness index (wall thickness/body surface area^{1/2}); LVMi = left ventricular mass index (mass/body surface area); — = no data available.

We relied on absolute LVWT (rather than values normalized 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 as well as several other demographic variables to be independent determinants of LVWT.

Differentiation of physiologic LVH from HCM. Our 38 athletes with greater than predicted LVWT showed several echocardiographic features permitting differentiation from HCM. First, LV cavity dimension exceeded the predicted upper normal limits ranging from 52 to 60 mm. In contrast, adolescents with HCM show small or normal-sized LV chamber size; our experience with 70 adolescents with HCM showed that none had a LV end-diastolic cavity dimension >48 mm (32). Enlarged LV cavity dimension occasionally encountered in adults with HCM is usually associated with marked progressive symptoms and systolic dysfunction and reduction in functional capacity (33). Furthermore, our athletes with increased LVWT showed normal mitral inflow velocities suggesting normal diastolic function, whereas the vast majority of patients with HCM have abnormal LV filling patterns because of impaired myocardial relaxation (34).

Gender differences also proved pertinent to the differential diagnosis of athlete's heart and HCM (35). Because no female athlete had LVWT >11 mm (which in this respect resembled the control group), a LVWT \geq 12 mm in trained females with a nondilated LV in the adolescent age group should raise the suspicion of HCM.

Finally, the precise age of the athlete may be relevant when considering the diagnosis of HCM, in that young affected individuals typically begin to show evidence of the HCM phenotype by echocardiography at 13 to 14 years of age (36). Although we cannot exclude with certainty that an occasional young adolescent with HCM may not have the diagnosis recognized with echocardiography, the absence of ECG abnormalities such as ST segment depression, pathologic Q-waves or deep T-wave inversions in all 720 athletes suggest that it is highly unlikely that any athlete in this study group carried an HCM gene without evidence of the phenotype, because abnormal ECG pattern may be evident several years before onset of LVH in HCM (37).

Wall thickness in adult and adolescent athletes. Adolescent athletes who are the subject of this study demonstrated several similarities with previously reported populations of older athletes (9,10). Similar to adult athletes, only a small proportion of our junior athletes had a LVWT exceeding predicted upper limits, and then usually associated with an enlarged LV cavity dimension. Also, gender-related differences were evident in that adolescent male athletes showed greater wall thickness and cavity dimensions than females (Table 4). In terms of specific sports, and consistent with reports in Italian athletes (17), male rowers had the greatest LVWT measurements (Table 5), suggesting that the combined stresses of intensive isometric and isotonic training within this sporting discipline are a particularly potent

stimulus for LVH. However, most importantly, adolescent and adult athletes clearly differed with respect to the range of LVWT measurements, as a manifestation of physiologic LVH. The LVWT ranged to 16 mm in adult athletes (17), but to only 14 mm in our adolescent athletes regarded to have physiologic LVH. This relative shift in wall thickness between the two age groups of trained athletes is also evident in the proportion of athletes with a LVWT >12 mm: about 2% in adults but only <0.5% in adolescents.

Such data showing important differences between adolescent and adult athletes with regard to the upper limits of physiologic LV hypertrophy underline the need for developing separate normal values for LVWT in younger athletes. Furthermore, it is our aspiration that defining the physiologic limits for LVH in the present study cohort will facilitate differentiation of athlete's heart from HCM in the important subgroup of adolescent athletes, thereby enhancing the efficiency of preparticipation screening and ultimately avoiding sudden deaths due to HCM. However, because our study population was largely Caucasian, caution should be exercised in extrapolating our findings to other ethnic and racial athletic populations. Also, some sports more common in the U.S., such as basketball and American football, are not represented.

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Reprint requests and correspondence: Dr. Sanjay Sharma, Department of Cardiology, University Hospital Lewisham, Lewisham High Street, London SE13 6LH, United Kingdom.

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William J. McKenna

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