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ORIGINAL ARTICLE

Left ventricular morphology and geometry in élite athletes characterised by extreme anthropometry

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ABSTRACT

OBJECTIVE The aim of the study was to explore the individual impact of BMI and height on LV size and geometry in a cohort of healthy athletes.

METHODS From a total cohort of 1857 healthy élite athletes (21 ± 5 years, males 70%) investigated with ECG and echocardiogram, we considered three groups: Group 1 n = 50: BMI ≥ 30 and height < 1.90 m; Group 2 n = 87: height ≥ 1.95 m and BMI < 30; control Group 3 n = 243: height < 1.90 m and BMI = 20–29.

RESULTS BSA was ≤2.3 m^2 in 52% of athletes in group 1 and 47% of athletes in group 2. Athletes in group 1 and in group 2 showed an enlarged LV end-diastolic diameter (LVEDD) (57 ± 6 vs 57 ± 4 vs 53 ± 4 mm in Group 3); 50% of athletes in group 1 and 38% of athletes in group 2 exhibited a LVEDD > 57 mm (p = 0.23). LV wall thickness was higher in group 1 (11 ± 1 vs 10 ± 2 mm in Group 2, p = 0.001). Concentric hypertrophy or concentric remodelling was found in 20% of athletes in group 1 vs 7% of athletes in group 2 (p = 0.04). Athletes of group 1 with BSA ≤ 2.3 m^2 showed lower LVEDD (53 ± 5 vs 60 ± 5 mm, p < 0.001), similar LV wall thickness (10 ± 1 vs 11 ± 1 mm, p = 0.128) and higher prevalence of concentric hypertrophy or concentric remodelling (31% vs 8%, p = 0.04) compared to those with BSA > 2.3 m^2.

CONCLUSION Athletes with high BMI have similar LV dimensions but greater wall thickness and higher prevalence of concentric remodelling compared to very tall athletes. Athletes with high BMI and large BSA have the widest LV dimensions. (Hellenic Journal of Cardiology 2025;86:111-119) © 2024 Hellenic Society of Cardiology. Publishing services by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. INTRODUCTION

Long-term exercise results in cardiac electrical and morphologic adaptation.[1] The most common finding in highly trained sports participants is an enlargement of all cardiac chambers and increased left ventricular (LV) mass.[1,2] In some cases, marked morphologic changes pose a *dilemma* in distinguishing physiologic cardiac adaptation to exercise from potentially fatal cardiac conditions such as cardiomyopathies.[3–7] The challenges in the differential diagnosis are particularly critical in assessing athletes with a large body size.[8] However, a limited number of studies involving athletes with extreme body anthropometry are currently available, and there is a lack of reference values for normality in this subset of individuals. Moreover, most

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data are inherent to athletes with large body surface area (BSA >2.3 m^2), not considering other parameters such as body mass index (BMI), which is an index of corporeal density. Recent studies provided normative data for basketball players.[9] Although these studies shed some light on athletes with above-average height, the effect of other indices of extreme body anthropometry, such as high BMI, on cardiac dimensions in athletes is largely unknown. In addition, the normal cardiac values in athletes with above-average height engaged in sports other than basketball are unclear. The aim of this study was to explore if there is any individual impact of BMI and height on LV size and geometry in a cohort of healthy athletes.

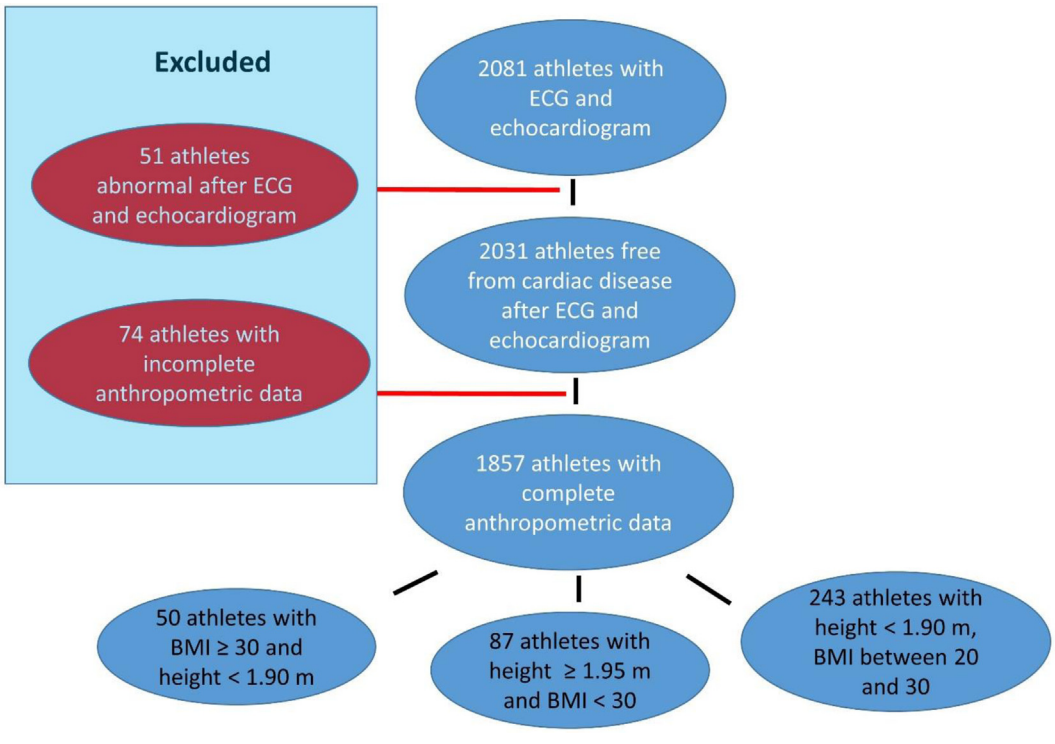
2. MATERIAL AND METHODS

The United Kingdom does not support a state-sponsored cardiac screening programme for athletes. However, the charitable organisation Cardiac Risk in the Young (www.c-ry.org.uk) has an established cardiac screening programme for young individuals that also serves many professional sporting organizations in the United Kingdom. Details of the cardiac screening programme have been previously described.[10,11]

Between 2007 and 2014, 2081 consecutive young, highly trained athletes were evaluated with a health questionnaire, electrocardiogram (ECG) and echocardiogram for screening purposes. Of these, 2031 were not affected by cardiomyopathy or a major valvular/congenital/electrical disease after an ECG and echocardiogram. Complete anthropometric data were available in a total of 1857 athletes, which constituted the study cohort (study protocol is summarised in Fig. 1).

In the attempt to investigate the different contributions of BMI and height in cardiac remodelling in athletes, we focused mainly on three subgroups of athletes. The first (group 1, n = 50, 3%) constituted of athletes with high BMI and normal height (BMI ≥ 30 and height < 1.90 m); the second (group 2, n = 87, 5%) included athletes who were particularly tall but had a normal BMI (height ≥ 1.95 m and BMI < 30); and the third (group 3, n = 243, 13%) served for comparison and included age-matched athletes characterised by height < 1.90 m and normal BMI (between 20 and 29). A BMI value of 30 was used as a cut-off because it is an established threshold to define obesity. The threshold of 1.95 m of height was arbitrarily chosen as the value equated to mean plus 2 standard deviations in our cohort. BMI and BSA were calculated as previously described.[12,13] We used the previously utilised threshold of BSA > 2.3 m^2 to define extreme anthropometry.[8]

FIGURE 1 **Study protocol**



Two-dimensional echocardiography was performed using either a GE Vivid I (Tirat, Israel), Philips Sonos 7500, Philips iE33 or Philips CPX50 (Bothell, Washington). The echocardiographic protocol consisted of parasternal long-axis views of the ventricles, long-axis view of the aortic root and ascending aorta, basal short-axis view of the origin of the coronary arteries, mid papillary short-axis view of the left ventricle, apical four-chamber, and two-chamber views of the left ventricle and pulsed tissue Doppler of the lateral mitral annulus. Digitised images of two beats were stored. Digitised images were analysed offline according to the European Society of Echocardiography guidelines by cardiologists and expert sonographers. LV mass was automatically calculated by the machine using the Devereux formula (0.8 x 1.04 x $[(DTd + SIV + PP)^3 - DTd^3] + 0.6$ gr). Isometric scaling of LV mass to BSA was chosen, as per the American Society of Echocardiography and European Society of Echocardiography recommendations.[14] LV internal diameter, septal wall thickness, posterior wall (PW) thickness, and left atrial diameter were measured from two-dimensional images in the parasternal long-axis view at both end-diastole and end-systole. Relative wall thickness (RWT) was calculated as the ratio of the sum of the interventricular septum (IVS) and PW thickness in end-diastole to the left ventricular end-diastolic diameter (LVEDD); care was taken to exclude right ventricular septal bands and PW chordae when measuring IVS and PW thickness, respectively. According to international guidelines, the RWT upper threshold of normality was 0.42.[14] Calculation of RWT permits categorisation of an increase in LV mass as either concentric (RWT >0.42) or eccentric (RWT < 0.42) hypertrophy and allows identification of concentric remodelling (normal mass with RWT >0.42) vs normal geometry (normal mass and RWT < 0.42).

Finally, systolic function was measured using the biplane Simpson's rule from the apical four- and two-chamber views, fractional shortening, and visual assessment. LV ejection fraction was considered to be normal when > 50%.

Ethical approval was granted by the National Research Ethics Service, Essex 2 Research Ethics Committee in the United Kingdom. Written consent was obtained from individuals ≥16 years and from a parent/guardian for those <16 years.

Statistical analysis was performed using the PASW software (PASW 18.0 Inc, Chicago, IL). Results are expressed as mean ± standard deviation for continuous variables or as number of cases and percentage for categoric variables. Comparison between continuous variables among groups was performed with the analysis of variance—using the Brown-Forsythe statistic when the assumption of equal variances did not hold—whereas the proportions were compared by means of the chi-square test, using Fisher's exact test when necessary. Statistical analysis was performed using PASW software (PASW 18.0 Inc, Chicago, IL).

3. RESULTS

Baseline demographic features are listed in **Table 1**. The mean age of athletes was 25 ± 7, 24 ± 5, and 25 ± 1 years in groups 1, 2, and 3, respectively. Athletes were predominantly males (n = 45, 90% in group 1, n = 86, 99% in group 2, and n = 219, 90% in group 3). The average hours of exercise per week were 17 ± 6 in group 1, 22 ± 7 in group 2, and 19 ± 7 in group 3 ($p < 0.001$ between groups 2 and 3). BSA was similar in groups 1 and 2 (2.3 ± 0.2 m^2 in group 1 vs 2.3 ± 0.1 m^2 in group 2) but was significantly lower in group 3 (2.0 ± 0.1 m^2, $p < 0.001$ between groups 1 and 3 and between groups 2 and 3). BSA was ≤ 2.3 m^2 in 52% of cases in group 1, 47% in group 2, and 90% in group 3.

In athletes of group 1, the top five sports were rugby (n = 40, 80%), shooting (n = 3, 6%), athletics (n = 3, 6%), cricket (n = 1, 2%), and swimming (n = 1, 2%). In athletes of group 2, the top five sports were rowing (n = 19, 22%), volleyball (n = 11, 13%), cricket (n = 11, 13%), football (n = 10, 11%), and rugby (n = 10, 11%).

Table 1 lists the echocardiographic characteristics of the study population. Both athletes of groups 1 and 2 showed an enlarged LVEDD compared with those of group 3 (57 ± 6 mm in group 1 vs 57 ± 4 mm in group 2 vs 53 ± 4 mm in group 3, $p < 0.05$ between groups 1 and 3 and groups 2 and 3, p non-significant between groups 1 and 2). Twenty-five (50%) athletes of group 1 vs 33 (38%) of group 2 and 31 (13%) of group 3 exhibited an LVEDD > 57 mm ($p < 0.001$ between groups 1 and 3, $p = 0.23$ between groups 1 and 2). **Fig. 2** shows the relationship between LVEDD and BMI and height.

LV maximum wall thickness (MWT) was significantly higher in athletes of group 1 (11 ± 1 mm vs 10 ± 2 mm in group 2, $p = 0.001$, vs 9 ± 1 mm in group 3, $p < 0.001$). Similarly, RWT was significantly higher in athletes of group 1 (0.37 ± 0.05) vs athletes in groups 2 (0.35 ± 0.05) and 3 (0.34 ± 0.05) ($p < 0.05$ between groups 1 and 2, and between groups 1 and 3).

Twelve (24%) athletes in group 1 vs 19 (21%) in group 2 and 16 (6%) in group 3 exhibited an MWT > 11 mm ($p < 0.001$ between groups 1 and 3, $p = 0.85$ between groups 1 and 2, as shown in **Fig. 3**).

Concentric hypertrophy or concentric remodelling was found in 10 athletes of group 1 (20%), 6 athletes

TABLE 1 Demographic and echocardiographic characteristics

	Group 1 (n = 50)	Group 2 (n = 87)	Group 3 (n = 243)
Age (years)	25 ± 7	24 ± 5	25 ± 1
Males n (%)	45 (90)	86 (99)	219 (90)
Caucasian n (%)	39 (78)[a]	80 (92)	217 (89)
Height (m)	1.81 ± 0.09[a,b,c]	1.98 ± 0.03	1.79 ± 0.12
Weight (kg)	109 ± 13[a,b,c]	96 ± 7	77 ± 8
BSA (m^2)	2.3 ± 0.2[b,c]	2.3 ± 0.1	2.0 ± 0.1
BSA ≤ 2.3 m^2 n (%)	26 (52)[b,c]	41 (47)	220 (90)
Hours per week	17 ± 6[c]	22 ± 7	19 ± 7
LVEDD (mm)	57 ± 6[b,c]	57 ± 4	53 ± 4
LVEDD > 57 mm n (%)	25 (50)[b,c]	33 (38)	31 (13)
LVEDD > 65 mm n (%)	4 (2)	1 (1)	3 (1)
LVESD (mm)	37 ± 6	37 ± 4	36 ± 4
IVS WT (mm)	11 ± 1[a,b,c]	10 ± 2	9 ± 1
PW WT (mm)	10 ± 1[b,c]	10 ± 2	9 ± 1
MWT > 11 mm n (%)	12 (24)[b,c]	19 (21)	16 (6)
RWT	0.37 ± 0.05[a,b]	0.35 ± 0.05	0.34 ± 0.05
Normal geometry n (%)	29 (58)[b]	63 (73)	190 (78)
Concentric hypertrophy/remodelling RWT > 0.42 n (%)	10 (20)[b,c]	12 (14)	15 (6)
Eccentric hypertrophy n (%)	11 (22)	11 (13)	38 (16)

Legends: BSA: body surface area; IVS: interventricular septum; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; PW: posterior wall; RWT: relative wall thickness; MWT: maximal wall thickness; WT: wall thickness. Group 1: athletes with BMI ≥ 30 and height < 1.90 m. Group 2: athletes with height ≥ 1.95 m and BMI < 30. Group 3: athletes with BMI between 20 and 29 and height < 1.90 m. [a]$p < 0.05$ between groups 1 and 2. [b]$p < 0.05$ between groups 1 and 3. [c]$p < 0.05$ between groups 2 and 3.

of group 2 (7%) ($p = 0.04$ between groups 1 and 2), and 16 (6%) athletes of group 3 (Fig. 4). On the other hand, 11 athletes (22%) in group 1 vs 11 (13%) in group 2 and 38 (16%) in group 3 showed eccentric hypertrophy (p non-significant between the three groups). Normal geometry was found in 29 athletes (58%) in group 1 vs 63 (73%) in group 2 and 190 (78%) in group 3 ($p = 0.07$ between groups 1 and 2, $p = 0.003$ between groups 1 and 3, and $p = 0.344$ between groups 2 and 3).

We found that 26 (52%) of athletes of group 1 and 41 (47%) of group 2 had a BSA ≤ 2.3 m^2. We compared LVEDD, MWT, and the prevalence of concentric remodelling or hypertrophy in athletes of group 1 with BSA > 2.3 m^2 and athletes from the same group with BSA ≤ 2.3 m^2. Athletes with BSA ≤ 2.3 m^2 showed smaller LVEDD (53 ± 5 vs 60 ± 5 mm, $p < 0.001$), similar wall thickness (10 ± 1 mm vs 11 ± 1 mm, $p = 0.128$), higher RWT (0.39 ± 0.05 vs 0.35 ± 0.04, $p = 0.006$), and a higher prevalence of concentric hypertrophy or remodelling (31% vs 8%, $p = 0.04$).

Most athletes were further followed up (with ECG and echocardiogram) as per sports organizations' screening protocols, and none of them developed pathologic findings.

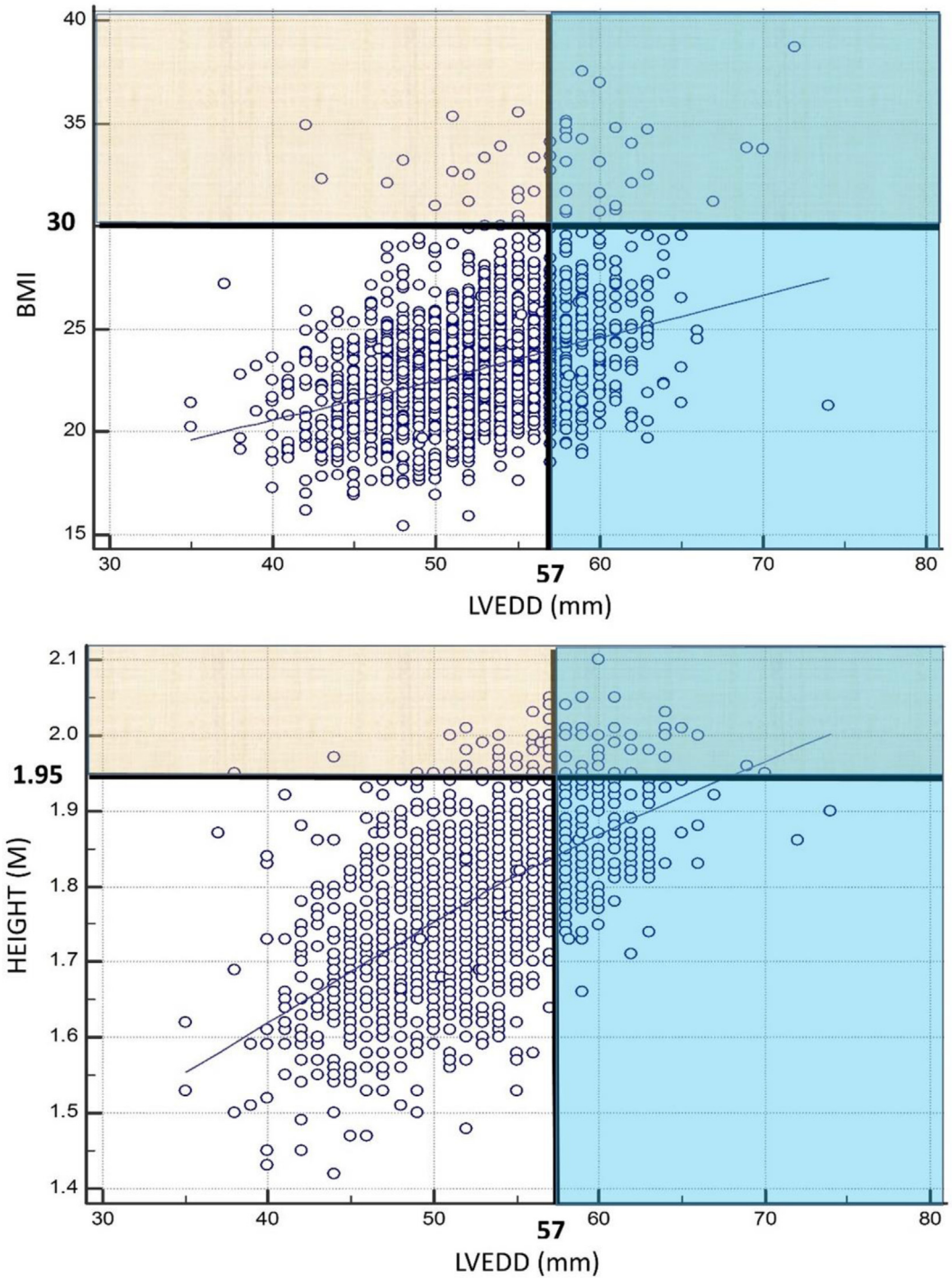
The average difference between the two independent readers (inter-observer variability based on 80 echocardiograms) was 1.8 ± 0.4 mm for LVEDD (Kappa inter-observer coefficient of 0.86), 0.6 ± 0.2 mm for interventricular septal thickness (Kappa inter-observer coefficient of 0.79), and 0.5 ± 0.2 mm for PW thickness (Kappa inter-observer coefficient of 0.79).

4. DISCUSSION

This study provides data on LV dimensions and geometry in a cohort of young *élite* athletes with extreme body anthropometry defined as either high BMI or above-average height. Although previous studies considered BSA as the only marker of large body size, we sought to establish the contribution of both increased BMI and height to LV dimensions and geometry in athletes.

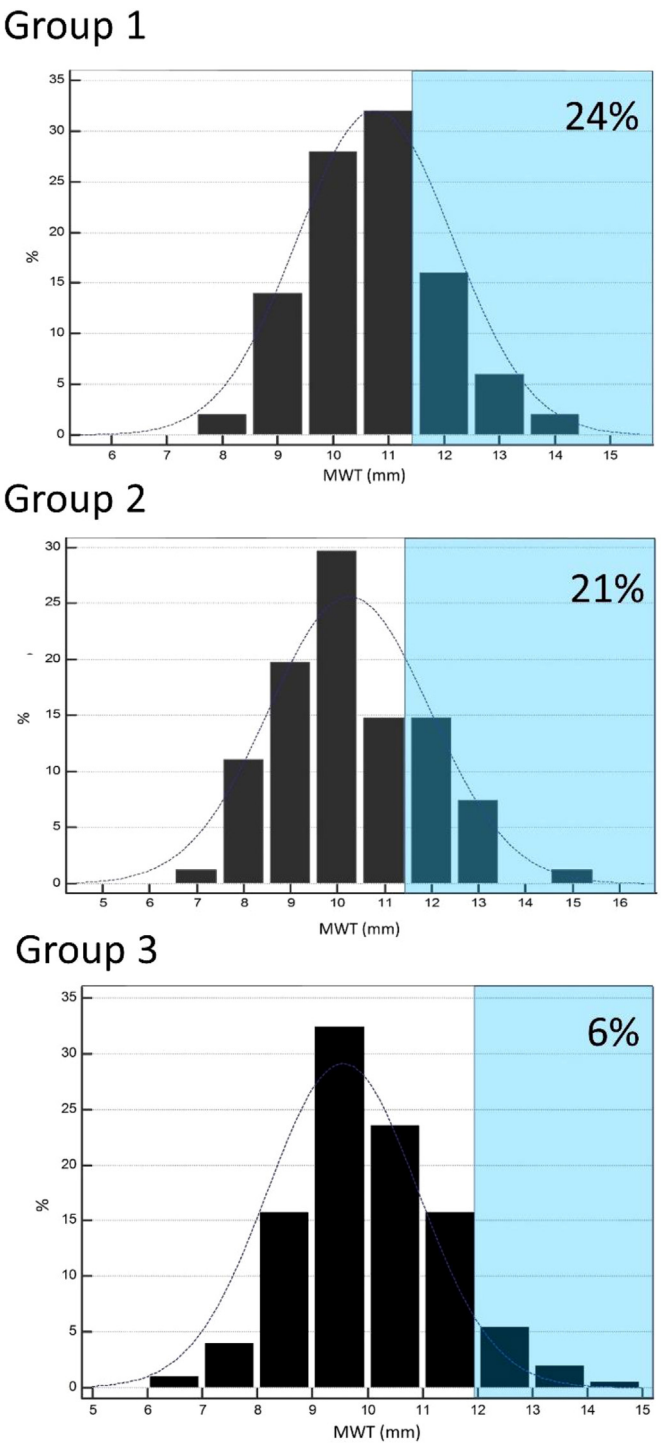
The main findings of our study are: a) both athletes with high BMI/normal height and athletes with normal BMI/above-average height exhibit larger LV dimension and thicker walls than those with normal BMI/normal height, meaning that both BMI and above-average height have an impact on LV size and geometry; b) half of the athletes with high BMI/normal height have a dilated LV (LVEDD >57 mm), and nearly a quarter have an MWT >11 mm; similarly, more than a third of athletes with normal BMI/above-average height have a dilated LV and 20% of them have an MWT >11 mm; c) athletes with high BMI/normal height exhibit a thicker IVS (11 mm) and

FIGURE 2 **Relationship of LV size with BMI and height. Upper panel: a BMI cut-off of 30 was used as per methods; lower panel: a height cut-off of 1.90 was used as per methods**



Abbreviations: LVEDD: left ventricular end-diastolic diameter.

FIGURE 3 **Maximal left ventricular wall thickness in the three groups and percentages of athletes exceeding normal values. A cut-off of 11 mm was considered to define increased wall thickness. Group 1: athletes with BMI ≥ 30 and height <1.90; group 2: athletes with height ≥1.90 m and BMI <30; and group 3: athletes with BMI 20-29 and height < 1.90 m**



Abbreviations: MWT: maximal wall thickness.

higher RWT (0.37) and more frequently have concentric hypertrophy/remodelling than those with normal BMI/above-average height, while they share similar values of LV dimensions; and d) within the group of athletes with high BMI/normal height, those with BSA <2.3 mq have similar wall thickness but smaller diameters, greater RWT, and a higher prevalence of concentric hypertrophy/remodelling compared with those with BSA >2.3 mq.

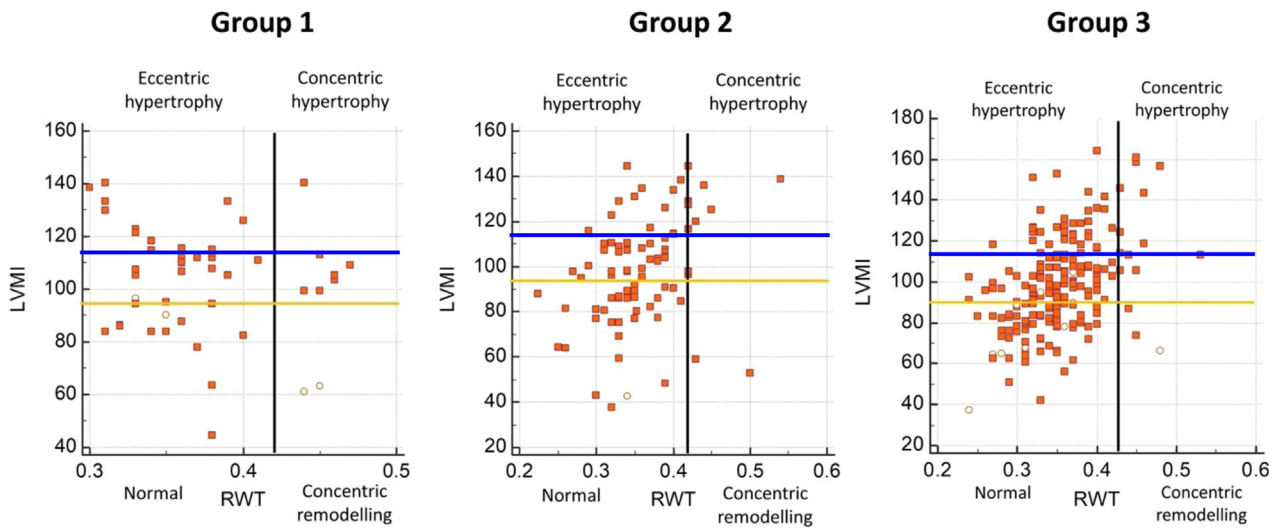
Regular physical exercise results in physiologic electrical, functional, and structural cardiac adaptations.[15] These changes may overlap with pathologic conditions, such as cardiomyopathies, where strenuous exercise is associated with an increased risk of adverse events including sudden cardiac death.[16-19] It is well established that *élite* athletes exhibit, on average, a 10-20% increase in LV wall thickness and LVEDD.[1] Electrical and structural changes are determined by a variety of factors, including age, sex, type of sport, ethnicity, and body size.[8,10,20-23]

The relationship between body size, long-term exercise, and cardiac dimensions is complex. Extreme body anthropometry is supposed to be a key determinant of cardiac adaptation to exercise, but few studies have attempted to address this specific issue.

In the largest study on athletes with extreme anthropometry to date, Riding et al.[8] showed that established upper limits for physiologic cardiac hypertrophy of 14 mm for MWT and 65 mm for LVEDD are clinically appropriate for all athletes (including the ones with BSA > 2.3 m^2). These results have important clinical implications, especially in terms of screening, because they define the physiologic upper limits of exercise-induced cardiac adaptation in athletes with large BSA, even though the 2.3 m^2 cut-off for the definition of extreme anthropometry was arbitrarily established. In our study, 52% of athletes with BMI >30 and 47% of extremely tall athletes (height ≥ 1.95 m) had a BSA ≤ 2.3 m^2. Although most American and European guidelines suggest reference values normalised per BSA, some caution should be exercised when considering BSA as the only measure of anthropometry. The use of allometric scaling as opposed to simple ratio scaling has been suggested by several studies, supporting the thesis that the latter does not allow for relationships between body size and cardiovascular variables that accommodate different relative geometries.[24,25]

Our study on a cohort constituted predominantly of male Caucasian *élite* athletes shows that high BMI is associated with dilated LV, increased LV wall thickness, and concentric hypertrophy/remodelling in 50%, 24%, and 20% of the cases, respectively.

FIGURE 4 **Prevalence of geometric patterns in the three groups. The yellow horizontal line identifies LV mass index upper cut-offs in women, whereas the blue horizontal line identifies the same cut-offs in men. The vertical blue line identifies the values of 0.42**



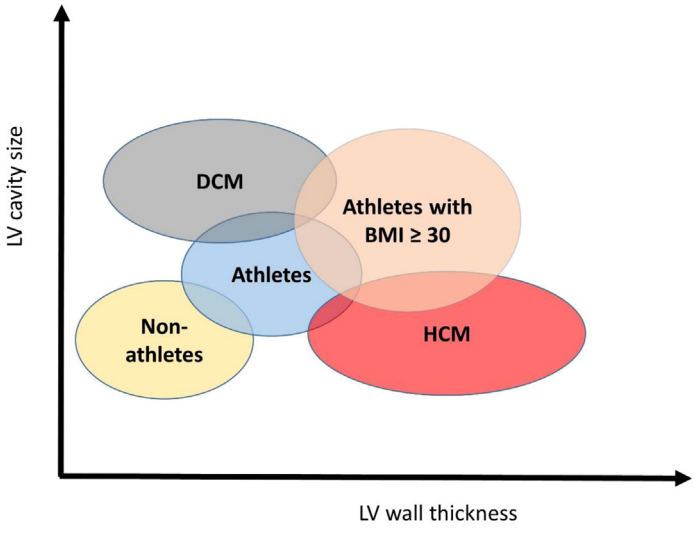
Abbreviations: LVMI: left ventricular mass index, RWT: relative wall thickness.

These data have useful implications, for example, in the assessment of rugby players, whose body *habitus* is almost invariably defined by elevated BMI (greater body weight and standard height). We found that, particularly in these individuals, the echocardiographic findings pose a diagnostic *dilemma*, and several athletes fall in the diagnostic overlap with cardiomyopathies, such as dilated cardiomyopathy and hypertrophic cardiomyopathy (Fig. 5). In our series, the upper limits of normality were 72 mm for LVEDD and 14 mm for MWT.

The gaps in knowledge are not limited to athletes with high BMI but also to athletes with above-average height. In this subset of individuals, data on basketball players are the only available. Cardiac adaptation is determined by a mixture of factors, and the type of sport is likely to represent one of them. Therefore, data limited to only one sport may not apply to the whole spectrum of sporting disciplines. In our cohort, just a minority of athletes with height $\geq$ 1.95 m and BMI $<$ 30 were a basketball player. Our data confirm that these athletes may develop significant dilatation and physiologic hypertrophy, even though RWT was rarely above thresholds of normality, in keeping with a harmonic increase in LV size and wall thickness. The degree of cardiac morphologic changes was similar to the one described by Engel et al.[9] in a cohort of National Basketball Association professional athletes with comparable average height and BSA.

Our data confirms that no direct linear relationship exists between both high BMI and above-average height and LV size and wall thickness. From our results, speculations can be made about high BMI

FIGURE 5 **Diagnostic overlap between cardiomyopathies and physiologic cardiac adaptation**



Abbreviations: DCM: dilated cardiomyopathy; HCM: hypertrophic cardiomyopathy.

(intended as weight prevailing over height) having an impact on both LV linear dimensions (LVEDD) and LV wall thickness, resulting in more frequent LV dilatation and increased MWT and higher prevalence of LV hypertrophy/remodelling, whereas above-average height and BSA influencing more LV dimensions rather than wall thickness, generating a more harmonic adaptation to exercise. This is further corroborated by the fact that in those athletes with BMI > 30 and BSA > 2.3, LVEDD is larger than those with BSA < 2.3 and that these athletes have similar values of RWT and less concentric hypertrophy, suggesting that height might primarily influence LV size.

Nevertheless, BMI is a measure used to determine a person's degree of overweight, whereas BSA measures the total surface area of the body. When the height value is fixed, a strong correlation exists between BSA and BMI, as only weight values vary. However, when the two parameters change, no correlation exists between BSA and BMI.

Our study has some limitations. Nearly all athletes in our cohort are males. This issue has been raised and discussed in previous studies and reflects mainly the social and cultural aspects of sports participation. We considered only young *élite* athletes of Caucasian ethnicity. Therefore, these results may not be applicable to recreational or veteran athletes, and further series, including athletes of other ethnicities, may show different results in terms of LV geometric remodelling and cut-offs for LV size and physiologic hypertrophy. Moreover, we acknowledge that our control group was not matched for the type of sports/exercise, and there is the possibility that LV remodelling was determined by the sporting discipline rather than anthropometry solely. Finally, this analysis is cross-sectional; thus, we cannot derive any inference about changes in cardiac structure during follow-up.

5. CONCLUSIONS

Athletes with high BMI and above-average height exhibit more pronounced LV physiologic adaptive changes compared with those with normal BMI/normal height. In particular, athletes with high BMI have similar LV dimensions but greater wall thickness and a higher prevalence of concentric remodelling compared with very tall athletes. Athletes with high BMI and large BSA have the widest LV dimensions. Global LV remodelling in response to exercise is complex and multifactorial, and no single body-size parameter can predict LV morphology.

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