International Journal of Cardiology 289 (2019) 125–130

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Hemodynamic and ECG responses to stress test in early adolescent athletes explain ethnicity-related cardiac differences☆

Pierluigi Demola [a], Antonio Crocamo [a], Laura Ceriello [a], Andrea Botti [a], Isabella Cremonini [a], Paolo Pattoneri [b], Domenico Corradi [a], Francesco Visioli [c,d], Matteo Goldoni [a], Giovanna Pelà [a,*]

[a] Department of Medicine and Surgery, University Medical School and University Hospital of Parma, Italy
[b] AUSL Cardiology Unit, Parma, Italy
[c] Department of Molecular Medicine, University of Padova, Italy
[d] IMDEA-Food, CEI UAM+CSIC, Madrid, Spain

ARTICLE INFO

Article history:
Received 14 January 2019
Received in revised form 10 April 2019
Accepted 26 April 2019
Available online 28 April 2019

Keywords:
Athlete's heart
Cardiac remodelling
Left ventricular geometry
QT dispersion
Black-white differences in exercise blood pressure
Echocardiography

ABSTRACT

Background: Ethnicity is an important determinant of athletes' cardiovascular adaptation. Black adolescent and adult athletes exhibit a left ventricular (LV) hypertrophy with a concentric remodelling higher than their Caucasian counterparts. Scant data, however, are available on race-related differences in hemodynamic response of adolescent athletes to exercise and its relation with heart remodelling. We evaluated if race-specific, sport-related structural and electrical remodelling in adolescent athletes of Caucasian and African ethnicity exclusively depends on race itself rather than on different cardiovascular responses to physical exercise.
Methods: We examined 90 adolescent athletes, 60 Caucasian (WA) and 30 Black (BA). All participants underwent thorough clinical, echocardiographic and stress test evaluations.
Results: BA had greater indexed LV mass (LVM/BSA) with increased relative wall thickness (RWT) implying a concentric remodelling. BA showed higher systolic blood pressure (SBP) compared to WA during the whole exercise test. ECG data showed that BA vs WA had a significant shorter QRS duration in each step considered with a significant greater QT dispersion. BA reached a higher relative pressure peak as compared to WA. RWT was strongly influenced by ethnicity and less by SBP at peak of exercise (PE), although LVM/BSA was significantly related to SBP at PE and just marginally to age and not significantly to race.
Conclusions: Black adolescent athletes showed higher SBP during all steps of exercise associated to a different trend. Ethnicity was the main determinant of RWT, suggesting that LV geometry is principally race-related rather than influenced by a different hemodynamic profile to physical activity.

1. Introduction

Africans have higher left ventricular (LV) mass compared to Caucasians [1]. This holds true for both the general population and different situations triggered by physiological and pathological stimuli such as physical exercise and hypertension [1–3]. The exaggerated left ventricular hypertrophy, quantified by echocardiography as LV mass (LVM) and thicknesses, sometimes mirrors the classical features of hypertrophic cardiomyopathy (HCM). This is an issue in pre-participation cardiovascular screening process of sports eligibility.

We recently demonstrated that LV wall thicknesses and mass are higher in adolescent amateur black versus white soccer players [4]. This has been previously demonstrated in adult athletes, despite their much lower level of sport training and activity [4]. At difference with white athletes, such changes are associated with a concentric pattern of left ventricular remodelling independent of the type of exercise; hence, concentric heart adaptation to sport activity is, likely, a peculiar phenotype of black ethnicity [4]. Yet, whether such structural ethnic differences could be intrinsically related to race or, rather, to a different hemodynamic response to exercise is still an unresolved issue.

Further, excessive elevation in systolic blood pressure during physical exercise is associated to an increased left ventricular mass in prehypertensive individuals [5].

Recently an independent relationship between exercise BP and cardiovascular structure has been demonstrated in a large cohort of apparently healthy adolescents [6]. These findings suggest that the systolic BP at exercise is an important determinant of cardiac structure and would be involved in the different remodelling of left ventricle in the two ethnicities. Epidemiological studies showed that African ethnicity correlates with a higher risk of sudden death (SD) as compared with

☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

* Corresponding author at: Department of Medicine and Surgery, University Medical School and University Hospital of Parma, via Gramsci n.14, 43100 Parma, Italy.
E-mail address: giovanna.pela@unipr.it (G. Pelà).

https://doi.org/10.1016/j.ijcard.2019.04.084
0167-5273/

Caucasian, with greater incidence in adolescence [7,8]. Moreover, HCM (the first cause of sudden death in competitive athletes in the USA, mostly affecting black athletes) is more difficult to diagnose in this population, not only for its overlapping with physiological hypertrophy, but also because some typical features of this disease, e.g. familiarity for HCM, subvalvular obstruction, and systolic anterior motion of mitral valve, are often absent [9,10].

Therefore, we need to define the physiological LV adaptation to exercise in African athletes and the mechanisms through which LV hypertrophy develops, to differentiate it from its pathological analogue.

We undertook this study to address the aforementioned issues and to assess a possible relationship between peculiar LV remodelling (more concentric hypertrophy) and hemodynamic response to physical exercise (e.g. greater blood pressure response) in adolescent athletes of African ethnicity compared to counterpart of Caucasians.

2. Methods

2.1. Study population

The local ethics' committee approved this study. Written informed consent was obtained from the athletes' parents.

We enrolled 90 amateur level, male adolescent athletes, 60 of which were white (WA) and 30 black (BA), practicing different endurance disciplines. All BA were coming from the Central/West Africa, namely Burkina Faso, Cameroon, Ghana, Ivory Coast, Nigeria, and Senegal. All participants underwent physical examinations with 12 lead resting ECG, ECHO and ergometer stress test. S1 + R5 in precordial leads were calculated using the Sokolow-Lyon voltage criteria to assess the presence of ECG-based LVH [11]. The analysis of ECG also includes the innovative parameter recently introduced for the stratification of arrhythmic risk, e.g. QT dispersion (maximum QT interval minus minimum QT interval, QTd). The latter is an index of the spatial dispersion of ventricular recovery times and, therefore, is an index of inhomogeneity potentially involved in the genesis of arrhythmias.

We assessed blood pressure (BP) and heart rate (HR) (OMRON 705 IT) during the clinical evaluation, with three consecutive measurements whose data were averaged (see Supplementary Methods for details).

2.2. Echocardiography

M-mode, two-dimensional, and Doppler ECHO were performed by an ultrasonography-experienced cardiologist, using a commercially available, multi-hertz sector, 2–4 MHz probe-equipped machine (Vivid S5, GE Healthcare, USA). The interventricular septal (SWT) and posterior wall (PWT) thicknesses, systolic (ESD) and diastolic (EDD) LV diameters, absolute left ventricular mass (LVM) and indexed to body surface area (LVM/BSA) were calculated as previously described [4]. LVM was also normalized to height $^{2.7}$, an estimate of lean body mass. RWT was calculated as: (SWT + PWT)/EDD. According to the ASE guidelines, we calculate LV remodelling categories (normal, concentric remodelling, concentric and eccentric hypertrophy) in the two groups [12], based on LVM and RWT. Cut-off values were based on 95th percentiles for this population (see Supplementary methods). Simpson's biplane rule-based end-diastolic (EDV) and systolic (ESV) volumes and ejection fraction (EF) were calculated, while Fractional Shortening (FS) was: [(EDV − ESV)/EDV] × 100.

Mitral inflow pattern was analysed from apical 4-chamber view and E and A waves and their ratio were considered as peak flow velocity and time velocity integral, in order to evaluate the conventional diastolic function. From the same projection, DTE analysis was performed at lateral site and postero-septum of mitral annulus. All echo measurements were undertaken at rest.

2.3. Stress test

All participants performed a cycle ergometer stress test (Cubestress Cardioline S.p.A., Italy) until volitional exhaustion, via 30 watts resistance increases every 2 min, during which the ECG was recorded continuously. We calculated heart rates from the ECGs. Systolic (SBP) and diastolic (DBP) blood pressures were measured by trained and certified technicians either using manual sphygmomanometers or an automated system (with small blood pressure cuffs when appropriate).

The average of both pressure values was computed at six points: basal time (BT); 2nd (2E), 4th (4E) min and peak of exercise (PE); 2nd (2R) and 5th (5R) minute during the recovery time (RT). The test was stopped when at least 80% of predicted HR was attained or when the participant was exhausted.

HR x BP (DP) was considered in each of the six points and some ECG parameters such as P wave duration, PR interval, QRS, QT, and QTd in four of them (BT, 2E, PE, 2R) were assessed off-line, after the stress test, using the magnification system of the cycle ergometer software. The QTd was calculated as the difference between the maximum and the minimum QT interval in the 12 lead ECG.

2.4. Statistical analysis

We report data as means ± SD and we compared, after testing for normality (Kolmogorov-Smirnoff), groups using two-tailed Student's *t*-test (SPSS 20.0 software package, IBM, Armonk, New York, USA). To consider the effect size taking into account the differences between groups' sample sizes, was used the values of Hedges' g. The interpretation is the same as Cohen's d: Small effect (cannot be discerned by the naked eye) = 0.2; Medium Effect = 0.5; Large Effect (can be seen by the naked eye) = 0.8. Our sample size was generally sufficient to see medium effects (significance with g values between 0.4 and 0.5). Univariate analysis was performed to analyse the relationship of cardiac structural parameters, such as RWT and LVM/BSA and BP at BT and PE including also relative pressure peak (ΔSBP: difference between SBP at PE and at BT). Of note, we report BPs taken before stress tests.

Mixed factorial ANOVA was used to test the effect of time and ethnicity (inter-subject factor) on quantitative variables, assessing a different trend by the significance of the interaction race × time.

Multiple regression analysis was used to analyse the relationship between RWT and LVM/BSA to ethnicity with age, HR, body surface area (BSA, not used for LVM/BSA), and BP as the covariates. In the analysis BP, from time to time, was included both to the BT, PE and ΔSBP.

We performed univariate analyses to assess the relationship between ECG-derived variables (such as QRS duration and QTd) and structural cardiac parameters in the two groups. A 2-tailed p value <0.05 was considered as statistically significant.

3. Results

3.1. Baseline characteristics

All athletes were engaged in organized amateur-level training for approximately 7 h/week (Table 1). The two groups had similar age, height, weight, BSA, and body mass index. SBP and DBP were higher and HR was lower in BA than WA (Table 1). BA exhibited higher S1 + R5 voltages (38 ± 10 vs 30 ± 10 in WA; $p < 0.001$) with greater prevalence of ECG-based LVH (19/30, 63% vs 18/60, 30% in WA; $p < 0.001$) and ST elevation (28/30, 93% vs 32/60, 53% in WA; $p < 0.001$) and that of partial right- bundle branch block was lower (7/30, 23% vs 30/60, 50% in WA; $p < 0.001$) compared with WA. We did not find differences in inverted T waves.

3.2. Echocardiographic results

SWT, PWT and LVM were significantly greater in BA compared with WA with an increased RWT suggesting a concentric remodelling despite mainly endurance training (Table 1).

This fits with our previous report of a peculiar geometry due to black ethnicity [4]. By using the LVM and RWT cut-off values to define the distribution of LV remodelling in the two groups, none of the WA was above the cut-off value for LVM and 5% was above the cut-off value for RWT. On the contrary, the percentages for BA were 23.3% and 13.3%, respectively. Therefore, the distribution of adolescents in the two higher quadrants was significantly higher in BA ($p < 0.001$) (see Supplementary Fig. 2).

LV diameters and volumes were similar in the two groups. LV systolic and diastolic functions, assessed by conventional and Doppler tissue imaging, were also similar between groups (data not shown).

3.3. Stress test

At BT of the stress test and during the whole exercise test, BA had a significantly higher SBP than WA, consistent with their clinical evaluation (Table 2). At PE, such differences increased then returned to basal values during the recovery time (Fig. 1, on the left).

No significant differences were observed in DBP, except at BT and at 5R, while HR and SBP contrastingly behaved during the effort as the former increased less in BA than in WA; this difference reached statistical significance at 4E and PE (Table 2 and Fig. 1, on the right). DP did not change at any step.

During ECG monitoring, BA showed significant shorter QRS durations than WA at each point (BT: 93 ± 12 vs 85 ± 10 ms, $p < 0.01$; 2E: 93 ± 12 vs 86 ± 12, $p = 0.01$; PE: 94 ± 17 vs 84 ± 11 ms, $p = 0.01$;

Table 1
Clinical and echocardiographic data in adolescent white and black, amateur-level athletes.

Parameter	WA n = 60	BA n = 30	*p* value	Hedges' g Size effect
Age (years)	13.3 ± 1.5	13.8 ± 1.6	0.17	0.33
Training (hours/w)	6.3 ± 2.0	7.0 ± 1.8	0.11	0.36
Height (cm)	1.64 ± 0.12	1.68 ± 0.08	0.11	0.37
Weight (kg)	53.1 ± 13.5	56.8 ± 10.9	0.27	0.29
BMI (kg/m^2)	19.4 ± 3.0	19.9 ± 2.3	0.61	0.18
BSA (m^2)	1.56 ± 0.25	1.64 ± 0.19	0.18	0.34
SBP (mmHg)	105 ± 12	113 ± 13	0.018	0.65
DBP (mmHg)	64 ± 9	71 ± 8	0.006	0.81
HR (beats-per-minute)	76 ± 11	70 ± 8	0.005	0.59
EDD (mm)	46.4 ± 4.3	46.1 ± 4.2	0.63	0.07
ESD (mm)	28.3 ± 3.6	28.5 ± 4.0	0.98	0.05
EDV (ml)	97 ± 26	100 ± 25	0.67	0.12
ESV (ml)	32 ± 11	32 ± 9	0.77	<0.01
SWT (mm)	7.8 ± 1.1	8.9 ± 1.1	<0.0001	1.00
PWT (mm)	7.9 ± 1.4	9.0 ± 1.3	<0.0001	0.80
RWT	0.34 ± 0.04	0.39 ± 0.04	<0.0001	1.25
LVM (g)	149 ± 46	175 ± 47	0.009	0.56
LVM/BSA (g/m^2)	94 ± 18	106 ± 22	0.005	0.62
LVM/h $^{2.7}$ (g/m$^{2.7}$)	38 ± 7	43 ± 9	0.021	0.65

Data are means ± standard deviation. BA, black athletes; BMI, Body mass index; BSA, Body surface area; DBP, Diastolic blood pressure; EDD, End-diastolic diameter; EDV, End-diastolic volume; ESD, End-systolic diameter; ESV, End-systolic volume; HR, Heart rate; h, Height; LVM, absolute left ventricle mass; LVM/BSA, indexed left ventricle mass; MWT Maximal wall thickness; PWT Posterior wall thickness; RWT, relative wall thickness; SBP, Systolic blood pressure; SWT, Septal wall thickness; WA, white athletes.

2R: 93 ± 11 vs 84 ± 12 ms, $p = 0.002$) with a consistently significant greater QTd (Table 2). The other ECG parameters, i.e. P wave duration, PR interval and QT, did not differ between BA and WA.

3.4. Statistical analyses

Concerning SBP, mixed factorial ANOVA showed that time and ethnicity were significantly different ($p < 0.001$ and $p = 0.002$ respectively). Their interaction was also significantly different ($p = 0.013$). Specifically, BA reached a higher ΔSBP as compared to WA. As regards HR, time and ethnicity were significantly different ($p < 0.001$ and $p = 0.039$, respectively), but their interaction was not significant different. Finally, concerning DBP, only time was significant ($p < 0.001$).

After univariate analysis, the highest correlation coefficient was found between LVM/BSA and SBP at PE for both BA and WA (SBP at PE- WA: $r = 0.45$, $p < 0.001$; BA: $r = 0.38$, $p < 0.05$; SBP at BT - WA: $r = 0.27$, $p < 0.05$; BA: $r = 0.28$, $p =$ ns; ΔSBP WA: $r = 0.40$, $p < 0.001$; BA: $r = 0.30$, $p =$ ns) while RWT correlated with SBP at PE and ΔSBP only in WA (SBP at PE- $r = 0.35$, $p < 0.01$; ΔSBP- $r = 0.29$, $p < 0.05$).

Still after multivariate data analysis that included ethnicity, age, HR, and SBP at BT or at PE as covariates, RWT was strongly influenced by ethnicity ($p < 0.001$) and less by SBP at PE ($p = 0.02$).

SBP at PE, but not at BT, was significantly related to LVM/BSA ($p = 0.003$) suggesting that the under hemodynamic stress load rather that at baseline influences the weight of LV, which was also marginally related to age ($p < 0.05$), but not significantly to race ($p = 0.061$).

Among the ECG-parameters we analysed, a significant relationship between LVM/BSA and QTd was found at 2R ($r = 0.44$; $p = 0.014$) in BA, suggesting that inhomogeneity of ventricular recovery time is related to LVM in blacks (Supplementary Fig. 1, A). No correlations between LVM (Supplementary Fig. 1, B) and QTd were seen in Caucasians. QRS was not related to structural characteristics of LV in either group.

4. Discussion

This is the first work that targets the different response to effort stress test between adolescent athletes of two different ethnicities. The main goal was to clarify the physiological mechanisms that underlie the structural and electrical heart differences of the African vs. Caucasian races, in the context of sport medicine.

Table 2
Exercise test in adolescent white and black, amateur-level athletes: Hemodynamic and ECG data.

	WA		BA		*p* value	Hedges' g Size effect
SBP						
Basal	108	±13	116	±13	0.007	0.62
2° min	117	±11	124	±13	0.008	0.60
4° min	129	±13	136	±16	0.029	0.50
Peak exercise	162	±22	178	±27	0.004	0.67
2° min rec	138	±17	152	±22	0.001	0.74
5° min rec	115	±13	123	±16	0.016	0.57
DBP						
Basal	66	±9	70	±8	0.027	0.58
2° min	64	±8	67	±8	0.069	0.38
4° min	65	±8	68	±10	0.14	0.34
Peak exercise	72	±11	73	±12	0.76	0.09
2° min rec	58	±9	60	±11	0.43	0.021
5° min rec	58	±7	61	±9	0.046	0.39
HR						
Basal	83	±12	80	±14	0.43	0.24
2° min	105	±14	100	±10	0.078	0.39
4° min	121	±18	113	±12	0.027	0.49
Peak exercise	179	±10	172	±11	0.005	0.68
2° min rec	133	±13	130	±15	0.23	0.22
5° min rec	103	±19	101	±13	0.60	0.12
QRS duration						
Basal	93	±12	85	±10	0.004	0.70
2° min	93	±12	86	±12	0.01	0.58
Peak exercise	94	±17	84	±11	0.012	0.65
2° min rec	93	±11	84	±12	0.002	0.79
QT dispersion						
Basal	27	±8	37	±13	<0.001	1.01
2° min	28	±12	36	±17	0.006	0.58
Peak exercise	24	±10	29	±9	0.019	0.52
2° min rec	31	±23	51	±19	0.001	0.92

Data are means ± standard deviations. BA, black athletes; DBP, diastolic blood pressure; HR, heart rate; min, minute; SBP, systolic blood pressure; rec, recovery time; WA, white athletes.

We previously showed that pubertal African athletes have higher LVM compared to their Caucasian counterparts, which is associated to a peculiar concentric, ethnicity-determined remodelling, resulting in a distinct LV phenotype [4]. Moreover, more pronounced ECG sport-related adaptations, such as LV hypertrophy and early repolarisation, were observed in Black vs Caucasian adolescent athletes [4]. Here, we report different hemodynamic and ECG changes occurring in the two groups during stress tests. Namely, BA vs WA exhibited higher SBP at rest, both during clinical examinations and at T_0 of a cycle-ergometer test. This difference was maintained during the whole exercise and actually increased at its peak. The converse was detected for HR, which was lower in BA vs WA with significant differences at 4E and at PE. Therefore, the double product was unchanged in the two groups in all steps. A significantly lower HR was also observed during clinical evaluation (yet not at base time of the ergometer test), which we suggest is due to the different clinical context, the different pressure measurement mode (an automated blood pressure system rather than ECG), and a likely greater autonomic response in the Caucasian race as already as demonstrated by other authors [13].

As regards DBP, the effort test confirmed lower values in WA (in agreement with what we observed during the clinical visit), although this difference disappeared during the effort to reappear at R5.

Population studies indicate that both adult and young Africans have higher blood pressure values; whether this phenomenon is due to different genetics or to social-economic factors is yet to be elucidated [14]. Indeed, clinical studies confirmed these findings, but accumulated data are still controversial [15,16].

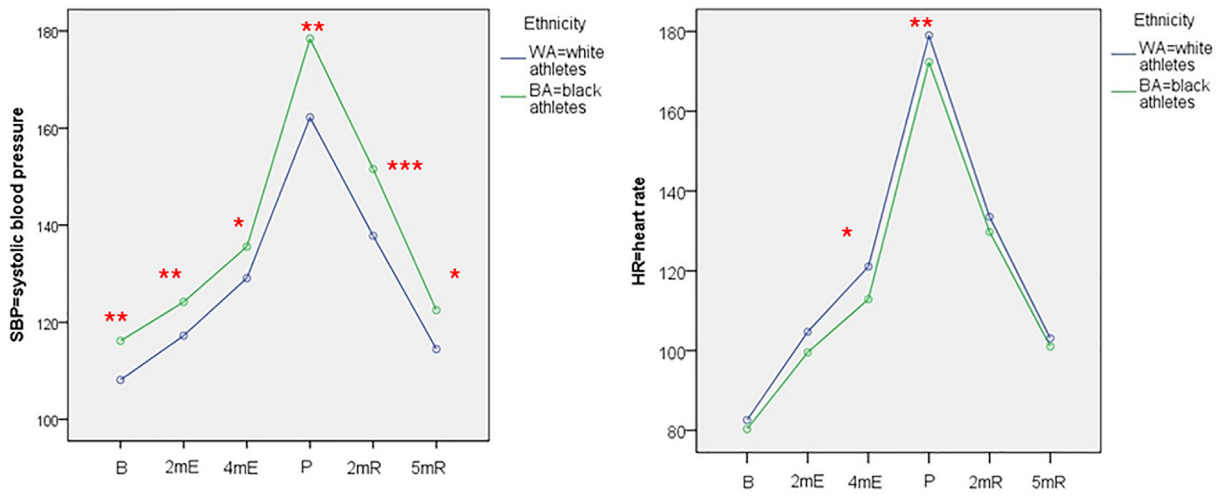

Fig. 1. Trend of systolic blood pressure (on the left) and heart rate (on the right) during exercise stress test in the two groups of athletes. $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.

A higher increase in SBP during treadmill exercise tests was observed in adult black men (from 35 to 40 years) compared to white ones, suggesting that the exaggerated vascular reactivity to physical exercise of the former might be responsible for their major incidence of hypertension [17]. Similar observations were done in pediatric subjects: children from Caucasian and African ethnicities - between 6 and 15 years - were submitted to an effort test and their pressure response was compared [18]. In all age ranges, except for the younger group, a significant higher SBP at peak was detected in black subjects, even though, at base time, their SBP was comparable to that of their Caucasian counterparts [18]. Of note, other investigators did not confirm these results [19,20].

In a longitudinal study that lasted over 15-years, ethnic differences in ambulatory BP (ABPM) have been reported: African-Americans compared to European-Americans have higher systolic BP both during daytime and nighttime, with a greater increase with age [21]. Using ABPM, researchers showed that black adolescents had higher SBP than white ones during sleep, but not during daytime [22].

We confirm that black adolescent athletes have higher SPB compared to their Caucasian counterparts and that such difference is more evident during the exercise test. Our results were obtained by using a dual method for BP evaluation, manual and automatic systems integrated to the cycle-ergometer. This allows overcoming the well-known technical difficulties of BP's assessment during exercise and increases the diagnostic accuracy.

From an anatomical viewpoint, we confirm the close relationship previously found between cardiac geometry and race [4,23]. The multivariate analysis showed that ethnicity was strictly related to RWT. The latter weakly correlated with SBP at PE, but not with SBP at BT and age.

No correlation between LVM and SBP at rest was found, suggesting that altered BP is not representative of the global pressure profile determining left ventricular remodelling/LV mass. Rather, SBP at PE is the strongest predictor of LVM demonstrating that BP measured during the stress test is more stable (being less influenced by neuroendocrine factors) and is a more accurate estimation of cardiac workload. These results are in agreement with Schultz MG et al. who recently demonstrated the association between exercise blood pressure, measured immediately after submaximal step-test, and cardiovascular structure (LVM in particular) in a large non-selected cohort of adolescents of both sexes [6]. The association between LVM and SBP developed during physical exercise has been reported also both in healthy subjects and in hypertensive patients [24,25].

In summary, the main determinants of LVM and RWT are different: SBP at PE for the former and race for the latter. Africans conceivably develop higher LVM as a compensation mechanism for pressure overload (as detected by the effort test), whereas their geometry appears to be primarily related to genetic traits rather than SBP (Fig. 2).

Race-related differences in cardiac structure become evident during development when more pronounced changes in myocardial anatomy could be measured [26,27]. In a prospective study of 687, 8–27 years old subjects, followed for more than ten years, ethnicity was a strong predictor of LVM [26]. This feature was noticeable from puberty and was independent of socioeconomic status, anthropometric characteristics, and hemodynamic stimuli. Expanding on a previous publication, we suggest that the younger, i.e. six months age of athletes explains the lower extent of cardiac morphology differences [26,27].

From a mechanistic viewpoint, we suggest that a larger increase of myocytes' size (and not their number) in Africans compared to Caucasians partially explains the cardiac structure differences in the transpubertal age [28]. Further, black adolescent athletes might exhibit increased LVM because of a different hemodynamic load.

Finally, genome-wide association studies recently identified a single nucleotide polymorphism in intron 16 of the cardiac sodium channel gene SCN5A linked with QRS duration among African Americans that can be related to shorter duration of this interval [29]. Polymorphisms of *SCN5A* could be related to an overall predominance of transient outward epicardial currents determining also the early repolarization, more frequent in Africans [30].

LVM also significantly influenced QTd in BA, demonstrating that only the inhomogeneity of the ventricular repolarization in the African race is related to cardiac structure. Other studies did not report associations between athlete's heart and ventricular repolarization heterogeneity compared with healthy sedentary controls, despite physiological and structural changes. Thus, an increase in left ventricular mass, in the frame of athlete's heart, does not influence QTd [31]. Our results indicate differences in the spread of the electric stimulus between the two ethnicities and suggest that the higher QTd of Africans could be employed to assess the risk for arrhythmia in this setting, helping the prognostic stratification of adolescent black athletes. The higher QTd of Africans might also be a marker of a “midway” condition between physiological and pathological hypertrophy. In summary, ECG measurements differ depending on ethnicity, having Africans shorter QRS duration and increased QTd; and such differences did not fluctuate with heart rate variations during stress tests.

4.1. Limitations

The main limitation of our study is the small number of BA we enrolled. However, that sample was homogeneous for gender, age, ethnic/geographic origin (all from West-African descent), time spent in

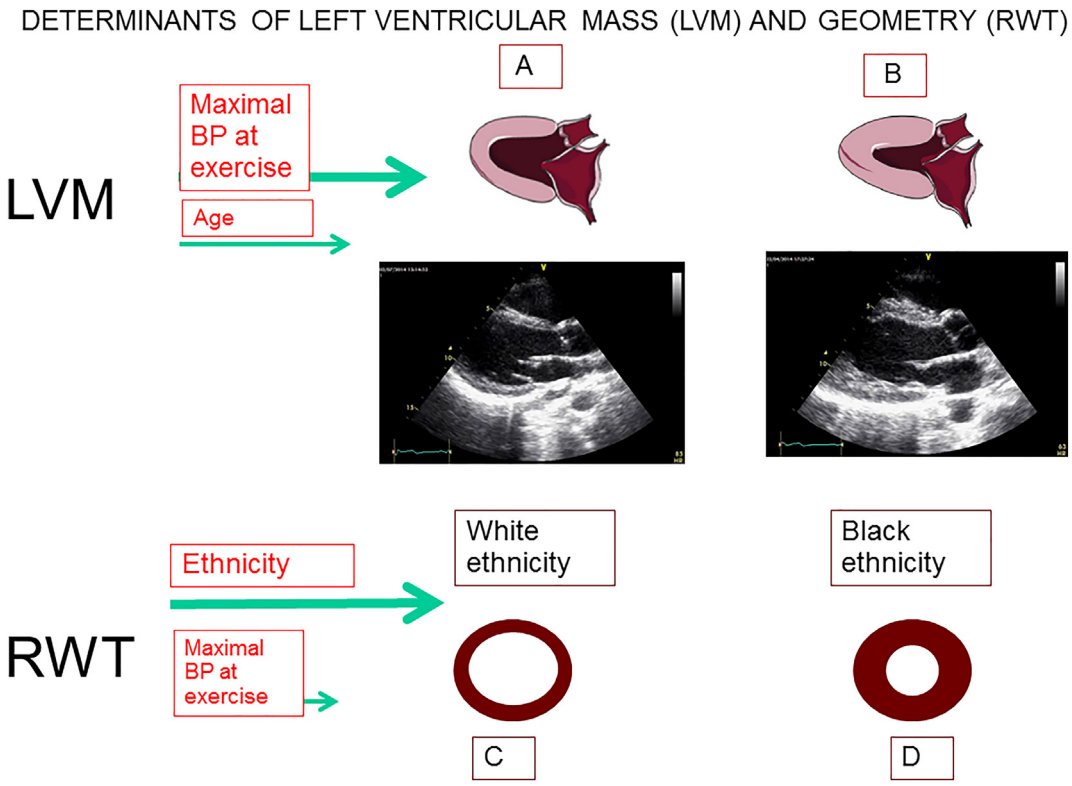

Fig. 2. Determinants of structural characteristics of left ventricle in the two groups of adolescent athletes. The systolic blood pressure at peak of exercise, is the main determinant of left ventricular mass (LVM) (shift from model A to B with growing LVM). On the contrary, Geometry, in terms of Relative wall thickness (RWT), is strictly related to ethnicity, with eccentric geometry (C) being peculiar in Caucasians, while concentric geometry (D) in Africans.

Italy, socio-economic environment, lifestyle, and food habits. All Africans except one practiced football, the most prevalent sport in Caucasians. However, no differences were observed between football players and athletes of other disciplines in the Caucasian group.

The main strength of our work is the accurate assessment of blood pressure during stress tests, which we performed by two methods, i.e. manual sphygmomanometer and an automated system.

5. Conclusion

Black adolescent athletes exhibit higher SBP when they exercise; their SBP at PE is the strongest predictor of LVM/BSA. These results suggest that the more pronounced left ventricular remodelling of black athletes could be related to a potentially higher hemodynamic afterload during sport exercise. Conversely, ethnicity is the main determinant of RWT, suggesting that LV geometry is principally race-related rather than influenced by a different hemodynamic response to physical activity. African adolescent athletes display greater inhomogeneity of ventricular recovery time (as evaluated through QTd), which could reveal different risk for arrhythmia, worth further appropriate investigations.

An integrated and multi-parametric evaluation of cardiovascular adaptation in black athletes could reduce the grey zone between physiological heart remodelling associated with exercise and the phenotype aspects of hypertrophic cardiomyopathy, which is the first cause of sudden death in the black population.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.04.084.

Disclosures

No conflicts of interest to declare. The authors received no financial support for the research, authorship, and/or publication of this article. All authors have materially participated in the research and/or article preparation. All authors have approved the final article.

References

[1] M.H. Drazner, D.L. Dries, R.M. Peshock, et al., Left ventricular hypertrophy is more prevalent in blacks than in whites in the general population: the Dallas Heart Study, Hypertension 46 (2005) 124–129.
[2] S. Basavarajaiah, A. Boraita, G. Whyte, et al., Ethnic differences in left ventricular remodeling in highly-trained athletes relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy, J. Am. Coll. Cardiol. 51 (2008) 2256–2262.
[3] J.R. Kizer, D.K. Arnett, J.N. Bella, et al., Differences in left ventricular structure between black and white hypertensive adults. The Hypertension Genetic Epidemiology Network study, Hypertension 43 (2004) 1182–1188.
[4] G. Pelà, M. Li Calzi, A. Crocamo, et al., Ethnicity-related variations of left ventricular remodeling in adolescent amateur football players, Scand. J. Med. Sci. Sports 25 (2015) 382–389.
[5] P. Kokkinos, A. Pittaras, P. Narayan, C. Faselis, S. Singh, A. Manoplis, Exercise capacity and blood pressure associations with left ventricular mass in prehypertensive individuals, Hypertension 49 (2007) 55–61.M.G.
[6] C. Schultz, A. Fraser Park, et al., Submaximal exercise blood pressure and cardiovascular structure in adolescence, Int. J. Cardiol. 275 (2019) 152–157.
[7] B.J. Maron, K.P. Carney, H.M. Lever, et al., Relationship of race to sudden cardiac death in competitive athletes with hypertrophic cardiomyopathy, J. Am. Coll. Cardiol. 41 (2003) 974–980.
[8] G. Finocchiaro, M. Papadakis, J.-L. Robertus, et al., Etiology of sudden death in sports. Insights from a United Kingdom Regional Registry, J. Am. Coll. Cardiol. 67 (2016) 2108–2115.
[9] N. Sheikh, M. Papadakis, V.F. Panoulas, et al., Comparison of hypertrophic cardiomyopathy in Afro-Caribbean versus white patients in UK, Heart 102 (2016) 1797–1804.
[10] L.L. Sorensen, A. Pinheiro, V.L. Dimaano, et al., Comparison of clinical features in blacks versus whites with hypertrophic cardiomyopathy, Am. J. Cardiol. 117 (2016) 1815–1820.
[11] D. Corrado, A. Pelliccia, H. Heidbuchel, et al., Recommendations for interpretation of 12-lead electrocardiogram in the athlete, Eur. Heart J. 31 (2010) 243–259.
[12] R.M. Lang, M. Bierig, R.B. Devereux, et al., Recommendations for chamber quantification, Eur. J. Echocardiogr. 7 (2006) 79–108.
[13] M.J. Berry, T.J. Zehnder, C.B. Berry, S.E. Davis, S.K. Anderson, Cardiovascular responses in black and white males during exercise, J. Appl. Physiol. 74 (1993) 755–760.
[14] S. Harding, M. Whitrow, E. Lenguerrand, et al., Emergence of ethnic differences in blood pressure in adolescence. The determinants of adolescent social well-being and health study, Hypertension 55 (2010) 1063–1069.
[15] F.A. Traiber, L. Musante, W.B. Strong, M. Levy, Racial differences in young children's blood pressure, AJDC 143 (1989) 720–723.
[16] C. Thomas, C.M. Nightingale, A.S. Donin, et al., Ethnic and socioeconomic influences on childhood blood pressure: the Child Heart and Health Study in England, J. Hypertens. 39 (2012) 2090–2097.

[17] L.-G. Ekelund, C.M. Suchindran, J.M. Karon, R.P. McMahon, H.A. Tyroler, Black-white differences in exercise blood pressure, Circulation 31 (1990) 1568–1574.
[18] B.S. Alpert, N.L. Flood, W.B. Strong, et al., Responses to ergometer exercise in a healthy biracial population of children, J. Pediatr. 101 (1982) 538–545.
[19] R.R. Pate, C. Matthews, B.S. Alpert, W.B. Strong, R.H. DuRant, Systolic blood pressure response to exercise in black and white preadolescent and early adolescent boys, Arch. Pediatr. Adolesc. Med. 148 (1994) 1027–1031.
[20] F.W. Arensman, F.A. Traiber, M.P. Gruber, W.B. Strong, Exercise-induced differences in cardiac output, blood pressure, and systemic vascular resistance in a healthy biracial population of 10-year-old boys, AJDC 143 (1989) 212–216.
[21] X.W. Wang, J.C. Poole, F.A. Treiber, G.A. Harshfield, C.D. Hanevold, H. Snieder, Ethnic and gender differences in ambulatory blood pressure trajectories: results from a 15-year longitudinal study in youth and young adults, Circulation 114 (2006) 2780–2787.
[22] G.A. Harshfield, B.S. Alpert, E.S. Willey, G.W. Somes, J.K. Murphy, L.M. Dupaul, Race and gender influence ambulatory pressure patterns of adolescents, Hypertension 14 (1989) 598–603.
[23] F. Haddad, S. Peter, O. Hulme, et al., Race differences in ventricular remodeling and function among college football players, Am. J. Cardiol. 112 (2013) 128–134.
[24] S. Michelsen, K.M.K. Nutsen, M. Stugaard, J.E. Otterstad, Is left ventricular mass in apparently healthy, normotensive men correlated to maximal blood pressure during exercise? Eur. Heart J. 11 (1990) 241–248.
[25] J.-F. Ren, A.-H. Hakki, M.N. Kotler, A.S. Iskandrian, Exercise systolic blood pressure: a powerful determinant of increased left ventricular mass in patients with hypertension, J. Am. Coll. Cardiol. 5 (1985) 1224–1231.
[26] C. Dekkers, F.A. Treiber, G. Kapuku, E.J.C.G. van den Oord, H. Snieder, Growth of left ventricular mass in African American and European American youth, Hypertension. 39 (2002) 943–951.
[27] G. De Simone, R.B. Devereux, T.R. Kimball, et al., Interaction between body size and cardiac workload. Influence on left ventricular mass during body growth and adulthood, Hypertension 31 (1998) 1077–1082.
[28] G. Olivetti, G. Giordano, D. Corradi, et al., Gender differences and aging: effects on the human heart, J. Am. Coll. Cardiol. 26 (1995) 1068–1079.
[29] K. Hnatkova, P. Smetana, O. Toman, G. Schmidt, Sex and race differences in QRS duration, Europace 18 (2016) 1842–1849.
[30] D.S. Evans, C.L. Avery, M.A. Nalls, et al., Fine-mapping, novel loci identification, and SNP association transferability in a genome-wide association study of QRS duration in African Americans, Hum. Mol. Genet. 25 (2016) 4350–4368.
[31] M. Turkmen, B.I. Aructu, A.M. Esen, et al., Assessment of QT interval duration and dispersion in athlete's heart, J. Int. Med. Res. 32 (2004) 626–632.