Cardiac and Metabolic Characteristics of Long Distance Runners of the Sport and Exercise Cardiology Outpatient Facility of a Tertiary Hospital

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Objective: To characterize electrocardiographic and functional cardiac parameters and cardiopulmonary responses to exercise in long-distance Brazilian runners monitored at the Sport and Exercise Cardiology Outpatient Facility of a tertiary care hospital.

Methods: Of an initial population of 443 male and female athletes of different sport modalities, we assessed 162 (37%) long-distance male runners, aged from 14 to 67. Electrocardiographic (12 leads) and echocardiographic (M-mode and two-dimensional) parameters were recorded at rest. Cardiopulmonary responses were evaluated on a treadmill with a ramp protocol.

Results: Metabolic alterations and cardiovascular diseases were diagnosed in 17% and 9% of the runners, respectively. Sinus bradycardia and left ventricular hypertrophy were observed in 62% and 33% of the runners, respectively. Structural alterations such as ventricular cavity > 55mm, relative wall thickness > 0.44, and ventricular mass index > 134g/m2 were found in 15%, 11% and 7% of the runners, respectively. Ejection fraction < 55% was observed in 4% of the runners. Peak oxygen uptake (VO2peak) decreased as of the age of 41, although the anaerobic threshold relative to the VO2peak remained unchanged with age.

Conclusion: Resting bradycardia and left ventricular hypertrophy are the most frequent cardiovascular adaptations in Brazilian long distance runners monitored by the Sport and Exercise Cardiology Outpatient Facility. Although VO2peak decreases after the age of 41, the relative oxygen uptake at the anaerobic threshold of these runners remained unchanged.

Key words: Electrocardiography, heart rate, hypertrophy, left ventricular, oxygen consumption, anaerobic threshold.

Intense physical training carried out by athletes to improve their performance in sports exposes the heart to intense overload in the course of months or years. This frequent exposure to overload results in alterations in heart automaticity, such as bradycardia at rest, and alterations in atrioventricular conduction, ventricular depolarization and repolarization1,2. Structural adjustments of the heart are also considerable and may lead to increases of up to 85% in the mass of the left ventricle3. Despite the fact that these functional and structural alterations have been duly documented, their limits of normality and their long-term consequences remain unknown.

The clinical and cardiological assessment of athletes is a very important medical procedure that enables: A) the identification of the degree of cardiac alterations as a result of intense physical training and B) the diagnosis of pre-existing heart diseases4. Ruling out pre-existing heart diseases is paramount to prevent fatal events in athletes. Although the risk of sudden death in athletes is low (one death per three hundred thousand athletes per year)5,6, the presence of a hidden cardiovascular disease increases one-hundred fold the risk of a fatal cardiac event. Therefore, being able to differentiate alterations triggered by physical training from pre-existing pathological alterations is the first challenge for sport cardiology practitioners4.

In Brazil, data on the health conditions and on the characteristics of the cardiovascular systems of athletes are scarce. Adding to this is the lack of statistics on the most frequent conditions and on the rate of sudden death for these athletes. In the United States, hypertrophic cardiomyopathy ranks first among the conditions that account for the sudden death of athletes, whereas in Italy, arrhythmogenic right ventricle dysplasia7,8 is the most frequently found condition. These results evidence the fact that cardiovascular characteristics of high level athletes may vary across countries.

In an attempt to broaden our knowledge on the cardiovascular characteristics of Brazilian athletes, this study aimed at characterizing: 1) the structure and functioning of the heart of Brazilian long-distance runners, registered at the Sport and Exercise Cardiology Outpatient Facility of a tertiary hospital in the City of São Paulo; and 2) the cardiopulmonary capacity and the metabolic profile during maximum progressive physical exercise in these athletes.

Methods

Of the records of 443 athletes (342 men and 101 women) registered at the Sport and Exercise Cardiology Outpatient Facility of a tertiary hospital of the City of São Paulo, Brazil,

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distributed in nineteen sport modalities as presented in figure 1, we selected 162 long distance male runners (> 3,000 m), with ages between fourteen and 67 years. The athletes' mean time of training for competition was 11.2 ± 0.7 years. They had from three to seven training sessions per week, with a volume that varied from 30 to 160 km per week, with moderate (heart rates between the anaerobic threshold and the respiratory compensation point) and high intensity (heart rate above the respiratory compensation point). Most athletes included in the study were amateurs.

Electrocardiographic assessment - The electrocardiogram was performed in the supine position, after five minutes at rest, with twelve standard leads, speed of 25 mm/s and voltage of 10 mm/mV (Hewlett Packard, HP 708), as part of the routine examination for registration at the Sport and Exercise Cardiology Outpatient Facility. Sinus bradycardia at rest was defined in two heart rate ranges: < 60bpm and < 50bpm. We used the Sokolow-Lyon[9] index to verify left ventricle hypertrophy.

Morphological and functional assessment of the heart - The morphology and function of the left ventricle were assessed on two-dimensional echocardiogram (Sonos 5500, Hewlett Packard, Massachusetts, USA). Left ventricular cavity diameters were obtained on the M-mode, guided by the two-dimensional mode, according to the guidelines of the American Society of Echocardiography[10]. Left ventricular mass was calculated with the Cube formula11: 0.8[1.04(LVDD + PWT + IVST)3 – LVDD3] + 0.6g, and was expressed as a ratio of the body surface area (left ventricular mass index, g/m2). The relative wall thickness (RWT) was calculated as: (ventricular septum + posterior wall)/left ventricular diastolic diameter, with a distinction between eccentric (RWT < 0.44) and concentric hypertrophy (RWT ≥ 0.44). Ventricular volumes were calculated using Teichholz's formula: [7/(2.4 + LVDD) x LVDD3] and were used to calculate left ventricular ejection fraction (LVEF - %). Left ventricular hypertrophy was defined as ventricular septum and/or left ventricle posterior wall values >13 mm and left ventricular mass index > 134g/m2. Abnormal dilatation of the left ventricular cavity was defined as left ventricular diastolic diameter (LVDD) > 60 mm[12-14].

Cardiopulmonary functional analysis - The assessment of maximum functional capacity of athletes was carried out by employing a cardiopulmonary test on a treadmill (Quinton Q65, model 645, Quinton Instruments Co., Washington, USA), with a ramp protocol until exhaustion. Load increments were added every minute and defined according to the physical capacity of each athlete, so that the total exercise time varied between eight and seventeen minutes, as previously described[15]. The athletes were advised as to the diet prior to the test and were told not to exercise within 24 hours prior to the test.

Oxygen uptake (VO2) and the production of carbon dioxide (VCO2) were determined through the mean of respiratory gas exchange by using a computerized system (Vmax, series 229 - SensorMedics Corporation, California, USA). Peak oxygen uptake (VO2peak) was defined as the maximum VO2 measured at the end of the exercise bout, when the athlete was no longer able to maintain the load imposed by the ergometer. The anaerobic threshold was determined by the loss of linearity between VCO2 and VO2, the point where the ventilatory equivalent of oxygen (VE/VO2) or the end-tidal oxygen pressure (PetO2) reached its lowest level before starting to increase during the test. The respiratory compensation point was determined at the point where the ventilatory equivalent of carbon dioxide (VE/VCO2) reached its lowest level before starting to increase and where the end-tidal carbon dioxide pressure (PetCO2) reached its maximum value before decreasing[16].

Heart rate was monitored throughout the test using a 12-lead electrocardiogram (Marquette Medical Systems, InC. CardioSoft, Wisconsin, USA). The blood pressure was measured using a mercury-column sphygmomanometer before the beginning of the test and during the exercise bout.

Statistical analysis - The data are presented as mean ± standard error of mean. In order to identify the possible differences between age groups we used the one-way analysis of variance (one-way ANOVA – Statistica Software), followed by Scheffé's post hoc test. We adopted $p < 0.05$ for statistical significance.

Results

Clinical and cardiological parameters - Of the 162 runners assessed, 27 (17%) presented metabolic alterations (six had iron deficiency anemia; one had hypothyroidism; seventeen had dyslipidemia; three had elevated glycemia) and fifteen (9%) presented cardiovascular diseases (thirteen had systemic arterial hypertension and two had Chagas disease, with one athlete having its undetermined form and the other having myocardiopathy). Only the athlete with Chagas' myocardiopathy was excluded from the sample and declared unfit for the practice of competitive sports.

Physical and hemodynamic characteristics of the different age groups at rest are presented in Table 1. The percentage distribution of athletes across the different age groups was as follows: until 30 years (26%), 31 to 40 years (32%), 41 to 50 years (24%), 51 to 60 years (14%) and 61 to 70 years (4%). The group aged up to 30 years presented a body mass index (BMI) significantly lower that the other groups from 31 to 40 years, 41 to 50 years, 51 to 60 years, and 61 to 70 years (21 ± 0.3 versus 22 ± 0.3; 23 ± 0.4; 24 ± 0.5; 24 ± 1,0; $p = 0,0001$), respectively. Systolic and diastolic arterial pressure and heart rate values were not different across athletes of different age groups. We verified that 86% of the athletes presented heart rate < 60 bpm (mean of 49 ± 0.5 bpm), but when we consider heart rates < 50 bpm (mean of 44 ± 0.1 bpm), this percentage decreases to 62%. Left ventricular hypertrophy was verified in 33% of the runners and early repolarization was observed in 20% (fig. 2). The prevalence of these alterations remained unchanged when the electrocardiogram analysis was carried out per age group (fig. 3).

Echocardiographic parameters did not show significant differences across groups of runners on different age groups, with the exception of relative wall thickness (RWT), which was significantly higher in the 51 to 60 age group, as compared with the aged up to -30 group (0.41 ± 0.02 versus 0.36 ±

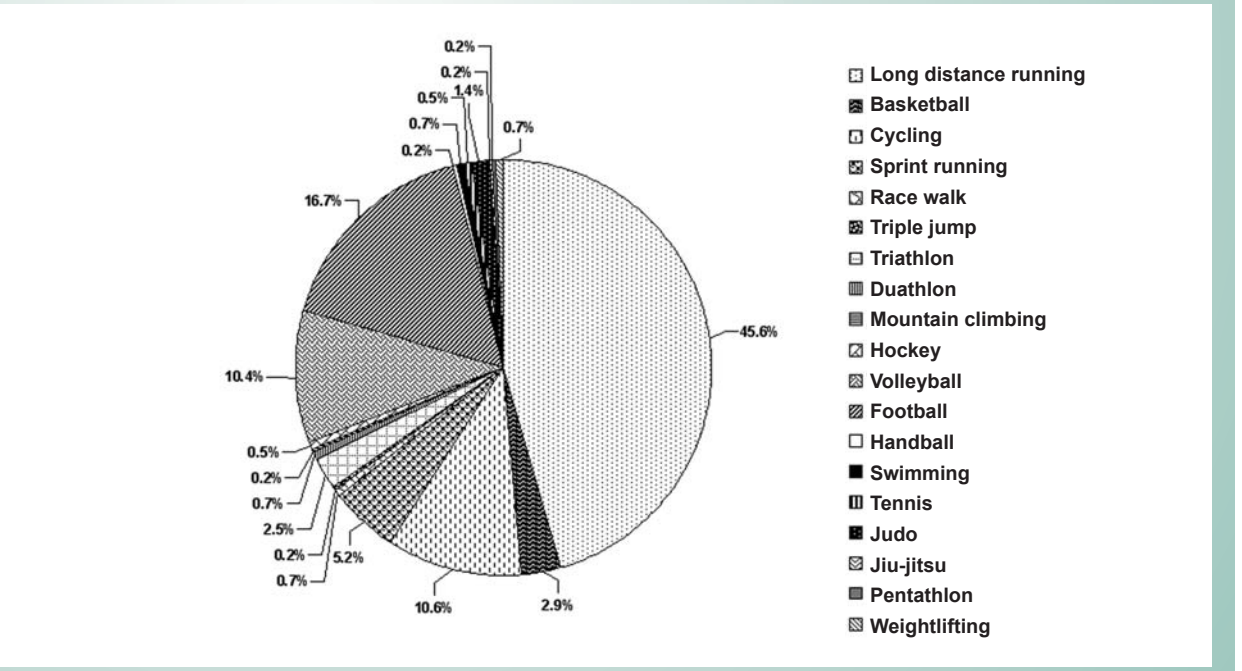

Fig. 1 - *Distribution of male (n = 342) and female (n = 101) athletes, by sport modality.*

0.01; p = 0.04) (table. 2). Eight long-distance runners (11%) presented relative wall thickness values (RWT) ≥ 0.44, characterizing a concentric hypertrophy pattern17. Of these eight runners, six (75%) were hypertensive.

In 64 athletes (85%), the size of the cavity of the left ventricle (left ventricular diastolic diameter) was within normal values (≤ 54 mm). However, eleven long-distance runners (15%) presented values above the upper normal limit (≥ 55 mm)14,17. The highest measures of ventricular septum and posterior wall of the left ventricle did not exceed the upper normal limit (13 mm)14. As regards the left ventricular mass index, five long-distance runners (7%) exceeded the normal limit (> 134 g/m2)18. Left ventricular ejection fraction was below the normal limit (< 55%)14,18 in only three athletes (4%), of whom one was in the 31 to 40 age group, one was in the 51 to 60 age group and one was in the 61 to 70 age group.

Cardiovascular and metabolic parameters during cardiopulmonary functional assessment - The values of heart rate and oxygen uptake at the anaerobic threshold, at the respiratory compensation point and at peak exertion for the different age groups are shown on Tables 3 and 4, respectively. Heart rates at the anaerobic threshold for the different age groups were similar in terms of absolute values (147 ± 2; 142 ± 2; 137 ± 3; 140 ± 3; 131 ± 6, p > 0.05) as well as in terms of relative values (percentage of peak heart rate). Absolute heart rate at the respiratory compensation point was significantly lower in the age groups from 31 to 40 years, 41 to 50 years, 51 to 60 years, and 61 to 70 years, as compared with the group aged up to 30 years (167 ± 2; 165 ± 2; 161 ± 3; 158 ± 5 versus 175 ± 2; p < 0.05), respectively. However, when the heart rate was expressed relative to the percentage of peak heart rate, no significant difference was observed across age groups. Peak heart rate decreased significantly in the groups from 31 to 40 years, 41 to 50 years, 51 to 60 years, and 61 to 70 years, as compared with the group aged up to 30 years (176 ± 1; 175 ± 2; 171 ± 3; 167 ± 5 versus 184 ± 2; p < 0.05), respectively. When peak heart rate was presented as a percentage value of the maximum heart rate predicted for their age19, athletes of the groups from 41 to 50 years, 51 to 60 years and 61 to 70 years presented significantly higher values than the group aged up to 30 years and the group from 31 to 40 years (100 ± 1; 103 ± 1; 103 ± 3 versus 94 ± 1 and 95 ± 1; p < 0.05), respectively.

Absolute oxygen consumption at the anaerobic threshold was significantly lower in the group from 51 to 60 years as compared with the group aged up to 30 years (33 ± 1 versus 40 ± 1; p < 0.05). However, when oxygen consumption was expressed relative to peak oxygen consumption, this difference was no longer observed. Absolute oxygen consumption at the respiratory compensation point decreased significantly in the groups from 41 to 50 years, 51 to 60 years and 61 to 70 years as compared with the group aged up to 30 years (46 ± 1; 44 ± 1; 42 ± 3 versus 54 ± 1; p < 0.05), respectively. However, when these values were expressed relative to peak oxygen consumption, these differences were no longer observed. Peak oxygen consumption decreased significantly in the groups from 41 to 50 years, 51 to 60 years and 61 to 70 years as compared with the group aged up to 30 years (52 ± 0.2; 48 ± 0.4; 46 ± 1.1 versus 60 ± 0.2; p < 0.05), respectively. However, athletes aged from 41 to 50 reached maximum oxygen consumption values predicted for their age which were significantly higher than those of athletes aged up to 30 years (150 ± 1 versus 128 ± 0.5; p < 0.05). Athletes in the groups from 51 to 60 years and 61 to 70 years presented higher maximum oxygen consumption values predicted for

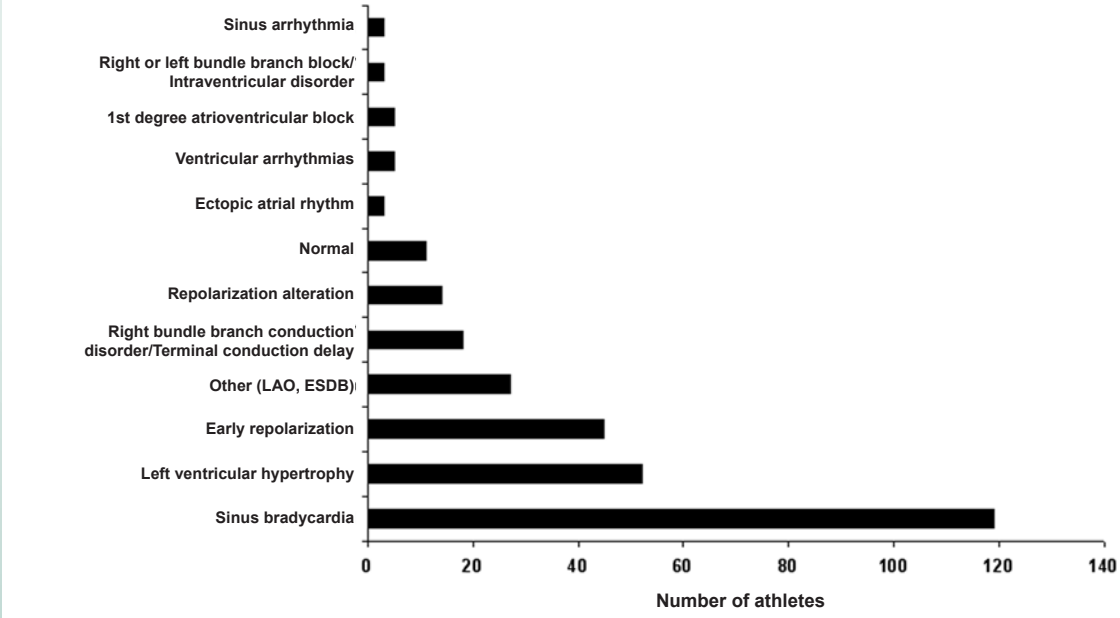

Fig. 2 - Prevalence of electrocardiographic alterations in long-distance male runners (n = 155). LAO – left atrial overload; ASDB – antero-superior division block.

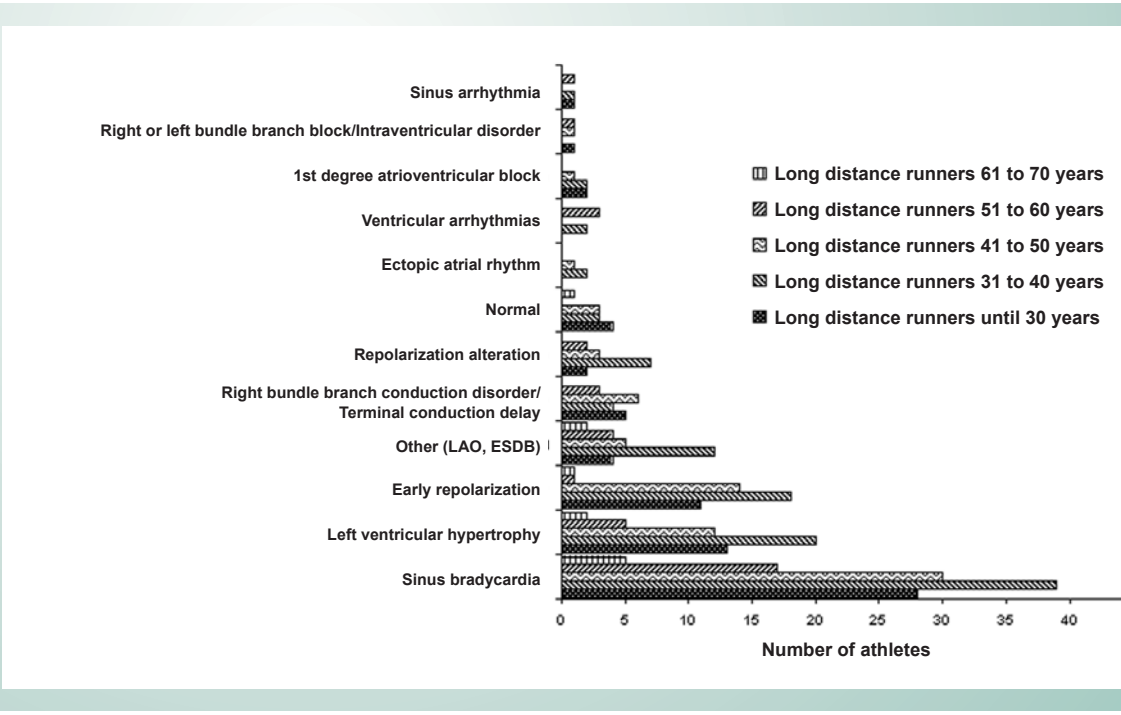

Fig. 3 - Prevalence of electrocardiographic alterations in long-distance male runners, distributed by age groups (n = 155). LAO - left atrial overload; ASDB – antero-superior division block.

their age as compared with the group aged between 31 to 40 (158 ± 1; 172 ± 3 versus 135 ± 0.4; $p < 0.05$).

Discussion

One of the echocardiographic criteria used to differentiate pathological ventricular hypertrophy from physiological hypertrophy is the increase in relative wall thickness (RWT)30. In our study, 11% of the runners presented increased RWT values, which suggests concentric remodeling, i.e., increased wall thickness relative to the left ventricular diastolic diameter.

	up to 30 years (n = 42)	31 to 40 years (n = 51)	41 to 50 years (n = 39)	51 to 60 years (n = 24)	61 to 70 years (n = 6)	Mean
Age (years)	25 ± 0.8	36 ± 0.4	46 ± 0.5	55 ± 0.5	63 ± 0.9	39 ± 0.9
BMI (kg/m2)	21 ± 0.3*	22 ± 0.3	23 ± 0.4	24 ± 0.5	24 ± 1.0	22 ± 0.2
SAP (mmHg)	123 ± 3.1	127 ± 2.1	126 ± 2.3	136 ± 4.0	134 ± 8.2	127 ± 1.4
DAP (mmHg)	82 ± 1.4	85 ± 1.6	82 ± 1.3	86 ± 1.9	88 ± 2.5	84 ± 0.8
HR (<60bpm)	49 ± 1.1	48 ± 0.8	49 ± 1.3	51 ± 1.1	52 ± 1.2	49 ± 0.5
HR (< 50 bpm)	43 ± 1.0	45 ± 0.7	42 ± 1.4	46 ± 0.4		44 ± 0.1

*BMI – body mass index; SAP – systolic arterial pressure; DAP – diastolic arterial pressure; HR – heart rate. Data are presented as mean ± standard error. *Significant difference relative to the other groups, p = 0.0001.*

Table 1 - Physical and hemodynamic characteristics at rest of long distance runners in different age groups

	up to 30 years (n = 16)	31 to 40 years (n = 23)	41 to 50 years (n = 18)	51 to 60 years (n = 13)	61 to 70 years (n = 5)
BSA(m2)	1,72 ± 0,02 (1,57-1,83)	1,78 ± 0,02 (1,66-1,97)	1,72 ± 0,03 (1,47-1,96)	1,74 ± 0,04 (1,51-2,00)	1,68 ± 0,04 (1,60-1,79)
LVDD (mm)	51 ± 0,6 (46-56)	51 ± 0,7 (45-59)	51 ± 0,9 (46-60)	49 ± 1 (42-55)	50 ± 0,6 (46-55)
LVSD (mm)	33 ± 0,7 (26-37)	33 ± 0,7 (28-39)	33 ± 0,8 (27-40)	32 ± 0,9 (28-37)	33 ± 1,8 (29-38)
EDV (ml)	122 ± 4 (97-154)	124 ± 4 (92-173)	128 ± 5 (97-180)	114 ± 6 (79-147)	121 ± 9 (97-147)
ESV (ml)	44 ± 2 (25-58)	44 ± 2 (29-66)	45 ± 3 (27-70)	43 ± 3 (29-58)	44 ± 6 (32-62)
SV (ml)	78 ± 2 (63-99)	79 ± 2 (59-107)	83 ± 3 (56-110)	71 ± 4 (49-96)	77± 6 (64-97)
VS (mm)	9,2 ± 0,2 (8-10)	10 ± 0,2 (8-12)	9,5 ± 0,3 (7-12)	10 ± 0,3 (9-12)	9,4 ± 0,4 (8-10)
PW (mm)	8,9 ± 0,2 (8-10)	9,5 ± 0,2 (8-11)	9,3 ± 0,3 (7-12)	10 ± 0,3 (9-12)	9,4 ± 0,4 (8-10)
RWT(mm)	0,36 ± 0,01 (0,33–0,40)	0,38 ± 0,01 (0,31-0,47)	0,36 ± 0,01 (0,28–0,44)	0,41 ± 0,02* (0,34-0,55)	0,38 ± 0,03 (0,30-0,43)
LA (mm)	34 ± 1 (27-38)	35 ± 1 (30-43)	36 ± 1 (30-42)	37 ± 1 (33-42)	38 ± 2 (32-43)
LVM (g)	167 ± 6 (135-225)	184 ± 7 (126-255)	185 ± 14 (119-321)	182 ± 10 (136-253)	173 ± 7 (155-191)
VMI (g/m2)	97 ± 3 (81-127)	104 ± 4 (70-146)	107 ± 7 (70–184)	104 ± 5 (80-137)	104 ± 5 (86-118)
LVEF (%)	64 ± 1 (57-75)	64 ± 1 (54-74)	65 ± 1 (58-76)	63 ± 1 (51-70)	64 ± 3 (54-72)

BSA – body surface area; LVDD – left ventricular diastolic diameter; LVSD – left ventricular systolic diameter; EDV – end diastolic volume; ESV – end systolic volume; SV – systolic volume; VS –ventricular septum; PW – posterior wall; RWT –relative wall thickness; LA – left atrium; VM –ventricular mass; VMI –ventricular mass index; LVEF – left ventricular ejection fraction. Data are presented as mean ± standard error and maximum and minimum values.
** Significant difference relative to the group up to 30, p = 0.04.*

Table 2 - Body surface area and left ventricle morphological and functional values for long-distance runners, in different age groups

It is unlikely that the type of physical training should have influenced this cardiac adaptation, once long-distance running is a stimulus to excentric cardiac remodeling3. It is more likely that concentric hypertrophy may be due to the fact that 75% of the runners with increased RWT are hypertensive. Vinereanu et al.30, in a comparative study between pathological and physiological hypertrophy, demonstrated significant differences in RWT between athletes and normal control subjects (0.46 ± 0.06 versus 0.38 ± 0.05, p < 0.05, respectively), as well as between athletes, hypertensive

		up to 30 years (n = 34)	31 to 40 years (n = 43)	41 to 50 years (n = 33)	51 to 60 years (n = 20)	61 to 70 years (n = 6)
AT	HR (bpm)	147 ± 2	142 ± 2	137 ± 3	140 ± 3	131 ± 6
	HR (% maximum HR)	79 ± 1	80 ± 1	79 ± 1	82 ± 1	79 ± 3
RCP	HR (bpm)	175 ± 2	167 ± 2*	165 ± 2*	161 ± 3*	158 ± 5*
	HR (% maximum HR)	95 ± 1	93 ± 1	95 ± 0,5	93 ± 1	96 ± 2
PEAK	HR (bpm)	184 ± 2	176 ± 1*	175 ± 2*	171 ± 3*	167 ± 5*
	(% HR predicted for the age)	94 ± 1	95 ± 1	100 ± 1*+	103 ± 1*+	103 ± 3*+

AT – anaerobic threshold, RCP – respiratory compensation point, HR – heart rate.
** Significant difference relative to the group up to 30, p < 0.05.*
+ Significant difference relative to the group from 31 to 40 years, p < 0.05.

Table 3 - Values of heart rate at the anaerobic threshold, respiratory compensation point and peak of exertion for long-distance runners, in different age groups.

		up to 30 years (n = 34)	31 to 40 years (n = 43)	41 to 50 years (n = 33)	51 to 60 years (n = 20)	61 to 70 years (n = 6)
AT	VO2 (ml/kg/min)	40 ± 1	36 ± 1	34 ± 1	33 ± 1*	31 ± 2
	VO2 (% maximum VO2)	66 ± 2	67 ± 2	66 ± 2	69 ± 3	66 ± 3
RCP	VO2 (ml/kg/min)	54 ± 1	49 ± 1	46 ± 1*	44 ± 1*	42 ± 3*
	VO2 (% maximum VO2)	89 ± 2	90 ± 1	88 ± 1	88 ± 1	92 ± 2
PEAK	VO2 (ml/kg/min)	60 ± 0,2	55 ± 0,2	52 ± 0,2*	48 ± 0,4*†	46 ± 1,1*
	(% VO2 predicted for the age)	128 ± 0,5	135 ± 0,4	150 ± 1*	158 ± 1*†	172 ± 3*†

AT – anaerobic threshold, RCP – respiratory compensation point, VO2 – oxygen uptake.
** Significant difference relative to the group up to 30, p < 0.05.*
† Significant difference relative to the group from 31 to 40 years, p < 0.05.

Table 4 - Values of oxygen uptake at the anaerobic threshold, respiratory compensation point and peak of exercise for long-distance runners, in different age groups.

patients and patients with hypertrophic cardiomyopathy (0.46 ± 0.06 versus 0.56 ± 0.09 versus 0.72 ± 0.13, p < 0.01, respectively). These results demonstrate that RWT in pathological conditions is much more accentuated than the RWT observed in athletes. It is worth noticing that, although the electrocardiographic analysis has evidenced left ventricular hypertrophy in 33% of the athletes, only 7% had this diagnosis confirmed by left ventricular mass index values > 134 g/m2. These results suggest higher sensitivity, but low specificity of electrocardiogram to determine ventricular hypertrophy in athletes, which makes the echocardiogram a fundamental test to allow better stratification of cardiac adaptations caused by physical training. In fact, although the electrocardiogram is considered a reference standard for the noninvasive diagnosis of arrhythmias and conduction disorders, its specificity is low for the detection of structural alterations, and may generate false-positive results and overestimate the incidence of left ventricular hypertrophy in athletes.

As regards the ventricular systolic function, it is known that there is no change triggered by physical training[13,28] of moderate and high intensity. However, in three athletes we found ejection fraction values below the lower normal limit, and we verified cardiovascular disease in two of these cases. One athlete had dilated myocardiopathy of Chagasic etiology and another had myocardiopathy secondary to arterial hypertension. It is important to stress that none of them was aware of the disease before the assessment. Although there was a low percentage of athletes (4%) with ejection fraction below the lower normal limit, this result stresses the need of clinical assessment of athletes before they are deemed fit for competitive sports. The presence of ventricular dysfunction, in these cases, did not prevent these athletes from showing good physical performance in training and competitions to that point.

The decrease in chronotropic response to more intense exertion after the age of 31 suggests that beta-adrenergic responsivity decrease with age[31]. This alteration has been explained by the impairment of post-receptor adrenergic signaling with age32. Curiously enough, the athletes presented a higher percentage value of predicted heart rate for the age as of the age of 41. These results confirm that heart rate estimation for age based on the formula 220 – age[19] underestimates the maximum absolute heart rate of runners above 41 years of age[33].

Despite the significant decrease of absolute oxygen consumption at the anaerobic threshold in the group from 51 to 60 years, the percentage values of oxygen consumption

remain unchanged in older runners. These results suggest a delay in the beginning of metabolic acidosis and an increase in oxidation efficiency in these athletes[16,34-36]. The fact that we did not observe significant difference in absolute oxygen consumption at the anaerobic threshold in runners aged between 61 and 70 years is probably due to the small number of athletes in this age group. Similar results were observed at the respiratory compensation point. As of the age of 41, we observed a decrease in the absolute value of oxygen consumption, while the percentage values of oxygen consumption relative to the peak of exertion remain unchanged. These results are additional evidence of the effects of physical training on the maintenance of oxidation capacity even with aging[16,37].

Additionally, results of previous studies show that aerobic physical training causes an increase in the specific activity of oxidizing enzymes of skeletal muscles[38] and greater efficacy in the use of free fatty acids through beta-oxidation. Additionally, it causes an increase in the distribution of oxidative muscle fibers (Typo I)[39,40].

Our runners did not present extremely high values of peak oxygen consumption, unlike those described by other authors[35,41,42]. In previous studies, highly trained athletes[35] and especially long distance runners (marathonists)[42] were assessed, which favors the development of greater aerobic capacity. Additionally, Fagard et al[41] described a volume of training of runners that ranged from 69 km/week to 167 km/week, a higher level of training of these athletes as compared to our athletes. Therefore, this difference between our study and others may be explained by the period of physical training that our athletes were engaged in, since not all of them were in a competitive period, this being so because only 16% of our sample were comprised of world-class runners, or else because of genetic differences[40,43]. Although peak oxygen consumption decreases significantly after the age of 41, the percentage value predicted for the age was significantly higher in older age groups. This demonstrates that the effects of physical training on maximum functional capacity are independent of age[31,44-46].

Limitations of the study - We recognize that our study has some limitations including the fact that the sample relates to athletes of a specific outpatient facility, and that our data cannot be extrapolated to other populations of athletes. Additionally, the period of physical training of the athletes was not controlled, and the athletes with metabolic or cardiovascular alterations were not excluded from the study. Although we recognize that these decisions may have influenced our results, our aim was to describe the population of athletes registered at the Sport and Exercise Cardiology Outpatient Facility of a tertiary hospital of the City of São Paulo and, with this, provide cardiologists and health practitioners with the characteristics of long-distance runners who seek these cardiology outpatient facilities. The number of athletes per age group is still small, but is enough to guide other studies in the area.

Future outlook - These results, although preliminary, may help cardiologists gain knowledge on the normal values for the morphology and functioning of the heart of Brazilian long-distance runners in different age groups. Additionally, these results may anticipate values of oxygen consumption and heart rate in different phases of maximum progressive exercise in these athletes.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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