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ORIGINAL ARTICLE

Increased active phase atrial contraction is related to marathon runner performance

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Abstract

Purpose Left atrial (LA) contraction is essential for left ventricular (LV) filling during exertion. We sought to evaluate the relationship of LA contraction and exercise capacity in trained athletes.

Methods Sixteen male marathon runners were recruited and allocated into two groups according to their previous training status ($\geq$ or $<$ 100 km peer week). All subjects underwent a baseline cardiopulmonary test to evaluate maximal aerobic capacity and a transthoracic echocardiography previous and immediate post-marathon. LA contractile function evaluation was accomplished by measuring the negative deformation of the post P wave strain curve (LASa). LASa change was defined as LASa pre-marathon minus LASa immediate post-marathon.

Results Mean age was 39 ± 6 years. LA volume index (39 ± 13 vs. 31 ± 5 mL/m^2, $p = 0.04$), LV mass index (91 ± 21 vs. 73 ± 12 g/m^2, $p = 0.04$), VO_2 max (59 ± 3 vs. 50 ± 8 mL/kg/min, $p = 0.036$) were higher in more intensive trained group and marathon time was lower (185 ± 14 vs. 219 ± 24 min, $p = 0.017$). An increase in LASa after immediate post-marathon was observed in both groups, which was significantly greater in the highly trained group (18.9 ± 5.8 vs. $6.3 \pm 3.5\%$, $p < 0.003$). Maximum VO_2 measured previous to the marathon was inversely related to marathon time and directly correlated to LASa change (rho = 0.744, $p = 0.001$, rho = 0.546, $p = 0.028$, respectively).

Conclusions Athletes with more intensive training load have larger LV mass and LA size. An increase in LA contraction was seen post-marathon, which was significantly greater in the highly trained group. This increase in the LA contraction was related to the maximum VO_2 measured previous to the marathon and to performance in a highly demanding test.

Keywords Running performance · Trained runners · Left atrium · Functional cardiac capacity

Abbreviations

BP	Blood pressure
cTnT	Cardiac troponin T
GFR	Glomerular filtration rate
hs-CRP	High-sensitivity C-reactive protein
LA	Left atrium
LASa	Negative deformation of the post P wave strain curve
LAVI	Left atrial volume index
LV	Left ventricle

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NT-proBNP	N-terminal prohormone of brain natriuretic peptide
SD	Standard deviation
TTE	Transthoracic echocardiography
VO_2 max	Maximum oxygen consumption

Introduction

Moderate exercise is essential to maintain superior cardiovascular health (Morris et al. 1953). However, with increasing frequency, a growing group of athletes is performing 20 or more hours of intense training per week, thus presenting various morphological and functional cardiac adaptations, a condition termed "athlete's heart" (Haskell et al. 2007; O'Donovan et al. 2010). This remodeling is an adaptation to increased overload and is often reversible. It also implies a greater cardiac reserve (Pelliccia et al. 2010) with increases in the left ventricular (LV) wall thickness and volume (Makan et al. 2005; Pelliccia et al. 1991), along with left atrium (LA) enlargement and functional changes (Wright et al. 2015). Remarkably LA enlargement is not related to augmented atrial filling volume, hence athletes have a normal resting diastolic function (Wright et al. 2015). On the other hand, in some subjects, long-duration and high-intense exercise induces changes beyond to those expected by adaptation to exercise, a scenario with uncertain long-term consequences (Gabrielli et al. 2016, 2014; Krol et al. 2016; Nemes et al. 2017). Some evidence has shown that intense endurance exercise might generate cardiac chamber remodeling associated with normal or enhanced myocardial function (Carbone et al. 2017). This last work proposed that there may be some overlap between exercise-induced physiological and clinical conditions associated with pathological cardiac remodeling, such that a modest amount of cardiac fibrosis associated with life-long endurance training can act as a substrate for arrhythmias (Carbone et al. 2017). Moreover, Trivax and McCullough (2012) proposed that long-duration and high-intense exercise, such as marathon running, generates multiple cardiac abnormalities (fibrosis, atrial arrhythmias, ventricular arrhythmias, and sudden death), a condition denominated "Phidippides cardiomyopathy". In some athletes, long-duration and high-intense exercise may not have a net mortality benefit (O'Keefe and Lavie 2013). Supporting this hypothesis, chronic extreme exercise seems to be related to the elevation of biomarkers related to myocardial injury creatine kinase, hsCRP, cardiac troponin T (cTnI), and N-terminal prohormone of brain natriuretic peptide (NT-proBNP) (Yoon et al. 2016). Evidence poses that significant atrial remodeling exists in highly trained athletes and that LA function during active contraction undergoes substantial changes (Gabrielli et al. 2012; Gjerdalen et al. 2015; Pelliccia et al. 2005). As a consequence of the shortening of the cardiac cycle during effort, LA contraction becomes determinant in LV filling. By other side, LA function during active contraction and its relation to exercise capacity in high-performance athletes have not been fully explored. Therefore, we evaluate LA contraction mechanics in athletes with different training levels, their response following a long-duration and high-intense exercise (marathon) and its association to exercise capacity.

Methods

Subjects

The study was prospective, single-blind cohort, including 16 male Caucasians marathon runners aged between 18 and 50 years, who had at least complete three marathons within the last 5 years. They were divided into two groups according to their previous training level during the pre-marathon period (defined as the 16 weeks before the day of the marathon). Group 1: highly trained, with weekly training ≥ 100 km and Group 2: less trained, with weekly training < 100 km. The cut-off point of 100 km per week was based on protocols from our institution where high performance amateur athletes and professionals are suggested to train at least 100 km a week in the period prior to a marathon run. The study design is shown in Fig. 1. Exclusion criteria were: high blood pressure (BP) (resting BP > 140/90 mmHg on two instances), dyslipidemia (defined as total cholesterol > 200 mg/dL, LDL > 100 mg/dL, HDL < 40 mg/dL, triglycerides > 150 mg/dL), diabetes, smoking, insulin resistance (defined as homeostasis model assessment-estimated insulin resistance: HOMA > 2.5), previous cerebrovascular

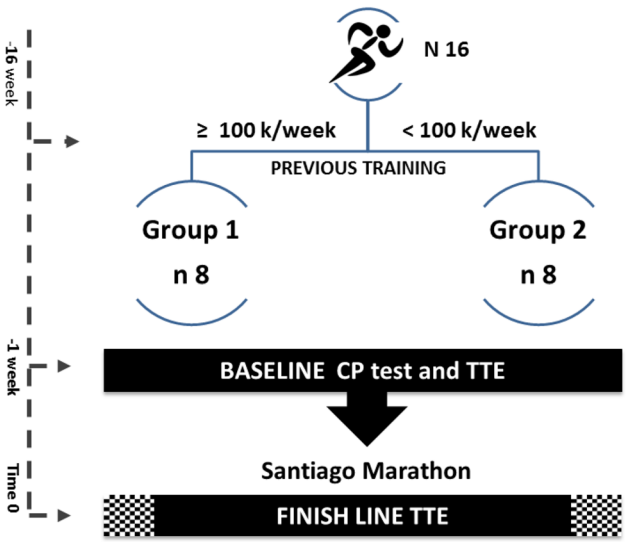

Fig. 1 Study design

disease, drug and alcohol abuse, use of nutritional supplements (to exclude possible confounders in the evaluation of athlete cardiac remodeling process), chronic kidney disease (defined as glomerular filtration rate, GFR < 60 mL/min/m^2), family history of sudden death, liver disease, autoimmune disease, active neoplasm, chronic obstructive pulmonary disease, chronic diseases or use of medications (antidepressants, diuretics, beta-blockers, antihypertensives, anorexigens and antibiotics). The protocol obtained ethics committee approval and was in accordance with the Helsinki declaration concerning experimental research on humans.

Marathon

All athletes ran the Marathon of Santiago, Chile, carried out on April 2, 2017. The weather during the marathon was cloudy, average temperature 9 °C, 87% relative humidity, wind speed 3.7 km/h. The marathon had available 12 hydration points located at kilometer 5, 10, 15, 18, 21, 24, 27, 30, 33, 36, 39 and 42. All participants were hydrated freely using all 12 hydration points. Group 1 drunk: 1318 ± 231 mL vs. group 2: 1338 ± 340 mL; $p = 0.89$ (data obtained by self-report), with no differences between groups.

Cardiopulmonary test

A cardiopulmonary test was performed one week previous to the marathon. Maximum oxygen consumption was expressed as an absolute (mL/min) and relative value, adjusted by weight (mL/kg/min). All participants accomplished the maximum oxygen consumption test (VO_2 max) on a treadmill (HP Cosmos®, Traunstein, Germany). The protocol consisted of a 3-min resting stage, a 5-min warm-up stage (8 km/h), and after that a 2 km/h increase every 3 min until reaching maximum effort. Test termination criteria were by the American College of Sports Medicine (ACSM) normative. Each participant was motivated to give maximum effort respiratory quotient ≥ 1.2, preferably, or heart rate close to the theoretical maximum value [220—age (years)]. The RS800cx® model was used as a portable heart rate monitor to record the electrocardiographic RR interval (Polar Electro OY, Kempele, Finland). Exhaled gases were measured continuously by the breath-by-breath method using a metabolic detector (Masterscreen-CPX, Jaeger, Hoechberg, Germany) equipped with O_2 and CO_2 analyzers. Recorded data correspond 20-s average. Anaerobic threshold was determined by the V-slope method.

Echocardiography

A baseline transthoracic echocardiography (TTE) (the week before the marathon) and an immediate post exercise TTE (at the finish line) was obtained for each participant (acquisitions were made during the first 5 min of the end of the marathon in all subjects). TTE was performed using a Vivid I portable equipment (GE, Healthcare, Horton, Norway), with a 1.5/3.5 MHz cardiac transducer, according to the American Society of Echocardiography guidelines (Lang et al. 2015). LV mass was calculated using the linear method (Lang et al. 2015). Image quality was optimized to obtain at least 60 frames per sec and digitally stored for later analysis using the EchoPAC BT 12 software (GE Healthcare, Horton, Norway). Biatrial and biventricular structure and function were analyzed with standard methods (Simpson biplane method, biplanar disk summation volume) and by bidimensional speckle tracking strain. Trained echocardiographers performing the analysis were blind to patient allocation and data. The atrial contractile function was evaluated by means of bidimensional speckle tracking by quantifying the negative deformation of the strain curve post P wave (LASa), using for reference the surface electrocardiograph (Fig. 2). In addition, the positive peak wave during ventricular systole (LASs) was obtained to evaluate LA reservoir function (Fig. 2). Special care was taken in the correct tracking of the atrial borders, to further clarify the tracking method: the software divides the atrial wall into six segments, giving each segment a unique value, for our analysis, we considered the average strain value of the six segments. LASa change was calculated as the difference of LASa pre-marathon minus LASa immediately post-marathon (in this way LASa increase post-marathon results in a positive number).

Statistical analysis

Each variable was assessed for normal distribution by the Kolmogorov–Smirnov test and non-parametric test were used as detailed below. Continuous variables were expressed as the mean ± standard deviation (SD). Categorical variables were expressed as total number (percentages) and compared between groups using Chi square or Fisher's test when appropriate. Continuous variables were tested with Mann–Whitney *U* test (unpaired data) and with Wilcoxon text (paired data). We use a dichotomic variable (LASa change > 10% or LASa change ≤ 10%; 8 subjects by group) to perform a univariate analysis. Spearman's methods were used to analyze the correlation between continuous variables when suitable. Intra-observer and inter-observer reproducibility for LASa at baseline and post effort were assessed offline in eight randomly selected

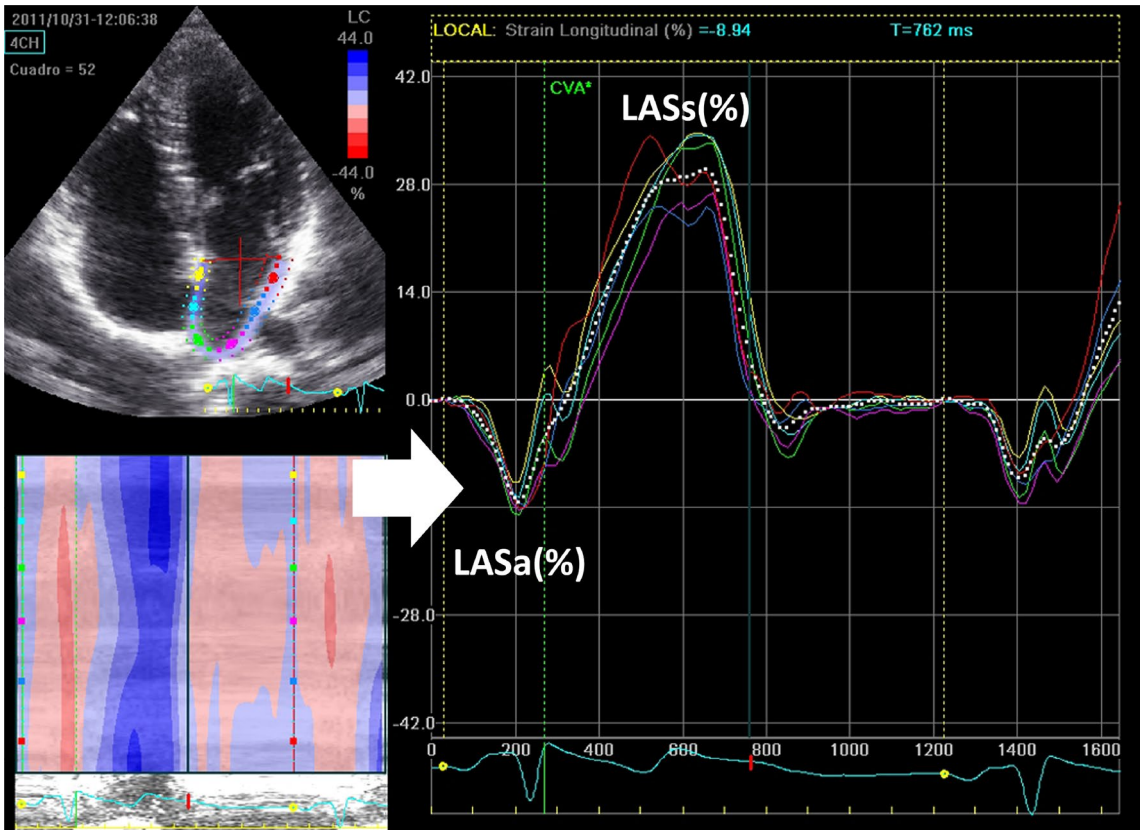

Fig. 2 Left atrial strain curves obtained by 2D STE (speckle tracking). White arrow corresponds to the negative deformation of the post-P wave strain curve (LASa) and LASs corresponds to the peak positive wave

subjects using Bland–Altman analysis. SPSS software version 15.0 (SPSS, Inc., Chicago, IL, USA) was used. Type I error rate was set to 0.05 for all analyses.

Results

Population characteristics

The group was composed of 16 male marathon runners, average age was 39 ± 6 years. Athletes were divided into two groups as previously described in the "Methods" section: Group 1: highly trained athletes and group 2: less trained athletes. Both groups were composed of 8 subjects. Group 1 had a training record of 8.2 ± 4.4 years and group 2 of 7.1 ± 5.7 years ($p = 0.66$). Both groups were similar regarding demographic characteristics (Table 1).

Table 1 Demographic characteristics

Variable	Group 1 (≥ 100 km/week)	Group 2 (< 100 km/week)	*p* value
Age (years)	38 ± 7	40 ± 6	0.55
Weight (kg)	66 ± 9	70 ± 5	0.26
Height (m)	1.71 ± 0.08	1.72 ± 0.04	0.44
Body surface (m^2)	1.77 ± 0.16	1.81 ± 0.08	0.28

Effect of long duration and high-intense exercise on atrial contractile function and functional capacity

Table 2 summarizes pre- and post-marathon hemodynamic and echocardiographic characteristics. In both groups systolic and diastolic function were similar. Post-marathon, both groups showed a significant increment in transmitral A wave. Group 1 showed a significantly higher absolute and relative VO_2 max as compared to group 2

Table 2 Hemodynamic and echocardiographic characteristics of the subjects previous and post-marathon

Variable	Group 1 (≥ 100 km/week)		Group 2 (< 100 km/week)	
	Pre	Post	Pre	Post
HR (bpm)	54 ± 9(2)	105 ± 10	57 ± 5(2)	101 ± 11
SAP (mmHg)	110 ± 25(2)	134 ± 42	112 ± 20(2)	136 ± 39
DAP (mmHg)	65 ± 10	67 ± 15	68 ± 12	67 ± 15
EDD (mm)	48.1 ± 4.2	47.1 ± 4	46.2 ± 3.5	45.0 ± 3.2
ESD (mm)	29.5 ± 4.2	28.3 ± 3.8	28.3 ± 3.3	26.1 ± 3.6
IVS (mm)	9.1 ± 1.1	8.8 ± 1.2	8.5 ± 1.2	8.5 ± 1.0
PW (mm)	8.5 ± 1.1	8.8 ± 1.1	8.5 ± 1.5	8.2 ± 0.8
LVEDV (mL)	123 ± 25(2)	114 ± 11	107 ± 18(2)	91 ± 16
LVESV (mL)	54 ± 11(2)	37 ± 6	48 ± 10(2)	33 ± 6
LVEF (%)	55.8 ± 3.3	57.6 ± 5.5	58.6 ± 6.7	65 ± 4.8
GLS (%)	− 20.6 ± 1.8	− 20.4 ± 1.7	− 20.5 ± 1.9	− 21.4 ± 2.0
LV mass (g/m)	91 ± 21(1)	–	73 ± 12	–
Mitral E wave (cm/s)	81.7 ± 9.8	84.4 ± 12.6	88.0 ± 14.2	68.9 ± 12.8
Mitral A wave cm/s)	51.5 ± 14.3(2)	73.9 ± 12.9	53.4 ± 12.5(2)	75.26 ± 18.6
LAVI (ml/m^2)	39.4 ± 12.6(1)	39.5 ± 12.2	30.6 ± 4.6	31.1 ± 4.7
LASa (%)	− 10.3 ± 3.0(2)	− 25.1 ± 5.5(3)	− 12.10 ± 3.6(2)	− 18.6 ± 2.7
LASs (%)	32.9 ± 4.3(2)	26.3 ± 3.6	35.9 ± 10.6(2)	24.6 ± 3.7

HR heart rate, *SAP* systolic arterial pressure, *DAP* diastolic arterial pressure, *EDD* end-diastolic diameter, *ESD* End-systolic diameter, *IVS* interventricular septum, *PW* posterior wall, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *LVEF* Left ventricular ejection fraction, *GLS* Global longitudinal strain of the left ventricle, *LV mass* left ventricular mass indexed by body surface area, *Transmitral E Wave velocity* transmitral filling E wave, *Transmitral A Wave velocity* transmitral filling A wave, *LAVI* left atrial volume index by body surface area, *LASa* left atrial strain (negative deformation of the post-P wave strain curve; contractile function), *LASs* left atrial strain (positive deformation during ventricular systole; reservoir function)

(1) $p < 0.05$ group 1 pre-marathon vs. group 2 pre-marathon

(2) $p < 0.01$ pre-marathon vs. immediate post-marathon

(3) $p < 0.009$ group 1 immediate post-marathon vs. group 2 immediate post-marathon

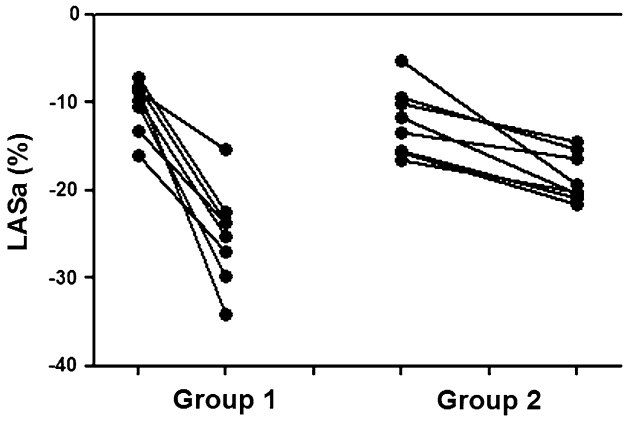

Fig. 3 LASa individual changes in both groups

(4055 ± 237 vs. 3675 ± 549 mL/min, $p = 0.046$ and 59 ± 3 vs. 50 ± 8 mL/kg/min, $p = 0.036$, respectively) and a substantially shorter marathon completion time (185 ± 14 vs. 219 ± 24 min, $p = 0.017$).

Effect of long-duration and high-intense exercise on atrial deformation

Left atrial volume index by body surface area (LAVI) size was significantly higher in group 1, also reaching more significant deformation during the active phase of the atrial contraction immediate post-marathon (− 25.1 ± 5.5 vs. − 18.6 ± 2.7%, $p = 0.009$). Both groups evidence an increase in LASa immediately post-marathon compared with their resting strain values, being significantly higher in group 1 (18.9 ± 5.8 vs. 6.3 ± 3.5%, $p = 0.003$). Figure 3 shows individual changes in both groups. Regarding LA reservoir function, both groups showed a decrease in LASs post exercise without difference between them (Table 2). VO2 max, evaluated previous to marathon run, correlated inversely with marathon completion time and directly with the increase in atrial deformation during active contraction (rho = − 0.744, p = 0.001, rho = 0.546, $p = 0.028$, respectively). Change

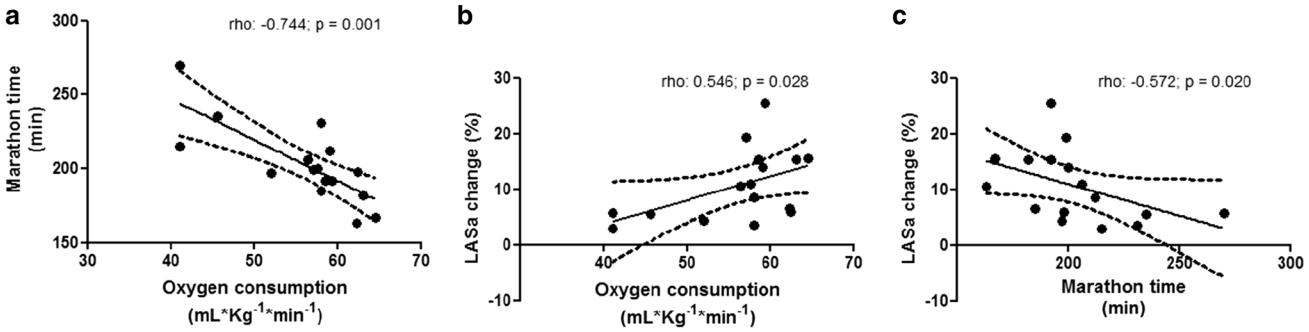

Fig. 4 Relations between: **a** maximum basal oxygen consumption and marathon finishing time; **b** left atrial deformation during the immediate post-marathon contraction phase (LASa change) and oxygen consumption; **c** LASa change and marathon time

in LASa also correlated with marathon completion time (rho = −0.572, $p = 0.020$) (Fig. 4).

Variables related to left atrial deformation post-marathon run

Significant variables (p value) including in the univariate analysis for LASa change > 10% post-marathon race were: amount of km per week in the training marathon period (0.018); marathon official time (0.02) and VO_2 max in mL/kg/min in cardiopulmonary test previous to marathon run (0.015). Non significant variables were: age (0.114); weight (0.957); height (0.866); systolic arterial pressure (0.754); years of training (0.55); pre-marathon LV ejection fraction (0.574); pre-marathon LAVI (0.064); LV longitudinal global strain (0.055); pre-marathon right ventricular fractional area change (0.825); LV mass index (0.564); and hydration during the race in mL (0.928).

LA strain agreement analysis

The mean differences for the intra-observer agreement were at baseline 1.28% (95% CI −0.12–1.89%) and post effort 1.45% (95% CI −0.18–1.95%). The mean differences for the inter-observer agreement were at baseline 1.44% (95% CI −0.21–1.98%) and post-effort 2.1% (95% CI −0.41–3.5%).

Discussion

Highly trained marathon runners, at baseline, had significant higher LV mass and LAVI compared to the less trained group. Regarding LA deformation during active contraction immediately post-marathon, both groups displayed an increase in this parameter, being significantly greater in the highly trained group, and correlating directly to VO_2 max and inversely to marathon completion time.

As mentioned before, athletes exhibit variable degrees of LA enlargement in the setting of normal filling pressures that is probably related to volume overload (D'Ascenzi et al. 2015a). D'Ascenzi et al. (2015b) showed that athletes subjected to successive load increments in a given period exhibited LA enlargement with normal contractile function. Regarding LA mechanics, in patients without heart disease, peak oxygen consumption (pVO2) has been associated independently with the LA duct and reservoir phases (SRe and SRs) (Leite et al. 2017). Consequently, we observed a positive correlation between LASa and VO_2 max.

Sanchis et al. (2017) reported that athletes (men and women) exhibit significant larger atriums than the general population. The LA response to the training stimulus is dynamic, and the extent of LA adaptation in athletes changes during the training period (D'Ascenzi et al. 2012). Moreover, the dynamic remodeling process of the LA, confirmed that this cardiac chamber rapidly adapts to different training loads, and in most cases can be reversed after a detraining or deconditioning period (D'Ascenzi et al. 2012). It is worth mentioning that exercise-induced cardiac remodeling does not affect the left heart exclusively, thus hemodynamic changes induced by long-duration and high-intense exercise typically involve both left and right chambers in a global process (D'Andrea et al. 2013). Moreover, the most recent evidence shows more accentuated changes in right cardiac cavities (La Gerche et al. 2012; Sanz-de la Garza et al. 2017).

The highly trained group presented larger LA volumes associated with a faster mechanical LA performance, in simple terms: less deformation is required to maintain a similar expulsive volume. In Gabrielli et al. (2014), this structural change was associated to a sustained increase in LA parietal stress that, in the long-term, could produce structural damage to the atrial wall. We hypothesize that these changes in LA structure and function could lead to a better performance during effort, but some athletes may suffer an extreme remodeling process

that could trigger the appearance of atrial fibrillation or atrial flutter (Calvo et al. 2012).

Oxborough et al. (2010) showed an impairment in LA reservoir function assessed by strain post exercise and an increment in LA booster function assessed by strain rate and volume. These observation agrees with our present findings.

It could be hypothesized that the changes observed in LA contractile function with exercise could be conditioned by changes in the effective blood volume. However, Sanz-de la Garza et al. (2016) showed no correlation between LV filling parameters, atrial function and dehydration parameters in a study with runners in different distances. They showed that most athletes increased LA contractile function in distances up to 35 km (in agreement with our observation) and a small group showed a continuous increase in LA contractility with greater distances but with great variability between them; globally an impairment post 56 km in LA contractile and LA reservoir function were observed. These observations suggest that athletes develop different cardiac adaptations to effort and the underlying mechanism for these differences should be clarified.

LA function contributes profoundly to cardiovascular performance by modulating pulmonary venous return and ventricular filling pressures. Atrial size solely is insufficient to provide mechanistic information about the atrium, and an increase in atrial size is not intrinsically an expression of atrial dysfunction (D'Ascenzi et al. 2018). Moreover, in patients with heart failure and preserved ejection fraction, LA function is related to exercise capacity and is a good predictor of VO_2 max (von Roeder et al. 2017). Although a comprehensive evaluation of the LA integrate morphological and functional parameters, future studies should also include the LA response to exercise.

In summary, this is a novel description of LA behavior in marathon runners. The highly trained group showed a greater LA functional and structural remodeling process as compared to less trained group. Furthermore, post-marathon increment in LA contractile function is significantly superior in highly trained athletes and is related to the VO_2 max measured previous to the marathon. To our knowledge, this is the first study that shows a positive relationship between the increase in the active phase of atrial contraction and sports performance in marathon runners.

Further uncertainties to clarify include: to what extent this phenomenon can predispose to arrhythmias, which subjects are predisposed to suffer them and which biomarkers could aid in identifying them, topics that are currently under investigation.

Study limitations

The primary study limitations were: (a) although all athletes were hydrated similarly during the marathon, we cannot discard the possibility that changes in blood volume occurred during the race. However, due to the mild weather conditions during the marathon and the similar hydration between both groups, the obtained results could reflect the effects of a long-duration and high-intense exercise on atrial contraction. (b) The small sample size is a limitation owing it to the challenging patient enrolment process and the technical complexity of performing an instant finish line TTE. (c) Other limitations are related to the lack of long-term follow-up, the absence of female athletes (which may present a different LA adaptation process), and that right heart chambers response to exercise was not analogously evaluated. Finally, our data are only valid in male, Caucasians, marathon athletes and cannot be extrapolated to other sport disciplines.

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Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest.

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