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## Differential atrial performance at rest and exercise in athletes: potential trigger for developing atrial dysfunction?

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## Abstract

**Background:** Highly trained athletes show an increased risk of atrial arrhythmias. Little is known about atrial volumes and function during exercise in this population. Our aim was to analyze atrial size and function during exercise.

**Methods:** 50 endurance athletes with  $11\pm 8$  hours of training per week and 30 sedentary control subjects were included. Echocardiography was performed at baseline and during exercise. Left (LA) and right atrial (RA) size and function were assessed by two-dimensional echocardiography. Peak negative strain (Sa) during atrial contraction and active atrial emptying volume (AEV) were measured.

**Results:** Athletes and control subjects showed a significant increment of deformation and AEV of both atria with exercise ( $p < 0.01$  vs baseline for LA and RA). Among athletes, a subgroup with significant LA ( $n=8$ )/RA ( $n=15$ ) dilatation ( $\geq 40\text{mL/m}^2$ ) showed a significantly lower increment in AEV with exercise (LA $\Delta$ AEV:  $1.4\pm 1.1\text{mL/m}^2$  vs.  $2.1\pm 0.9\text{mL/m}^2$ ,  $p=0.04$ ; RA $\Delta$ AEV:  $0.9\pm 0.8\text{mL/m}^2$  vs.  $2.3\pm 1.1\text{mL/m}^2$ ,  $p < 0.01$ ) and lower increment in deformation versus other athletes (LA $\Delta$ Sa:  $-3.2\pm 2.9\%$  vs.  $-9.5\pm 4.4\%$ ,  $p < 0.01$ ; RA $\Delta$ Sa:  $-2.5\pm 3.3\%$  vs.  $-9.8\pm 3.3\%$ ,  $p < 0.01$ ).

**Conclusion:** During exercise, active atrial strain increases, but less in athletes compared to controls, but due to larger atrial volumes, they reached similar increases in atrial emptying volume. However, this overall lesser deformation increases from a subgroup with significant atrial dilatation showing impairment in atrial contractile [reserve](#).

**Key words:** atrial function, athletes, imaging stress test, atrial arrhythmia.

## INTRODUCTION

Highly trained athletes show an increased prevalence of atrial fibrillation (AF) as compared to case-matched controls from the general population (Molina, 2008; Wilhelm, 2012a). Indeed, a meta-analysis of several observational studies reported a five-fold increase in the overall risk of AF in athletes (Abdulla, 2009). The etiology of the higher risk of arrhythmia in this particular group of subjects is still poorly understood (Wilhelm, 2012b; Wilhelm, 2012c). Atrial remodeling is actually a highly frequent finding among endurance athletes (Pelliccia, 2005). [In this population, left ventricular filling occurs at early diastole at rest \(D'Ascenzi, 2011\), but active atrial function is particularly stressed and is a fundamental contributor to cardiac function during exercise \(Gabrielli, 2014; Kusunose 2012\).](#) Accordingly, atrial function and volume are closely related to functional capacity during exercise (Naoum 2012; Vaturi 2010) and impairment of its contribution leads to a reduced exercise capacity (D'Andrea 2009). Atrial myocardial deformation as depicted by atrial strain and strain-rate analysis by two-dimensional speckle tracking has emerged as a novel method to evaluate left atrium (LA) (Saraiva, 2010) and right atrium (RA) function (Padeletti, 2012). These deformation imaging tools have been useful in different clinical scenarios such as pulmonary vein ablation (Schneider, 2008), coronary artery by-pass grafting (Gabrielli, 2011) and athlete's heart (Gabrielli, 2012). Moreover, simple computational models showed an inverse relationship between myocardial deformation and chamber size at a determined contractile performance (Bijnens, 2012).

The performance of the atria during exercise may provide insights into the mechanism underlying the development of atrial remodeling and this elevated risk of atrial arrhythmias. We have previously shown, in a subgroup of highly trained athletes at rest, significantly higher atrial volumes and lower deformation during active atrial contraction (Gabrielli, 2014). These particular working conditions potentially provide more atrial contractile reserve, but at the cost of higher atrial wall stress. However, little is known about atrial size and function during exercise in highly trained athletes. Especially the question, whether individuals with large atria and little active deformation during rest, are able to exploit their potential

contractile reserve (Gabrielli, 2014) during exercise, is key to understand whether excessive atrial dilatation is beneficial or potentially detrimental in highly trained individuals.

According to this, the objective of the present study was to analyze the performance of both atria at rest and during exercise in a group of highly trained athletes, as quantified by relating myocardial deformation and size with a special focus on the performance of individuals with the most dilated atria.

## **METHODS**

### **Study design and population**

This was a cross-sectional study comparing two groups: 50 athletes and 30 sedentary healthy controls with similar age (mid thirties), who were recruited among hospital staff and patients' relatives with a strictly normal medical record. All participants were male, in sinus rhythm, normotensive, did not have any other cardiovascular risk factor and had normal echocardiography findings. The athletes group consisted of competitive marathon runners and triathletes. All of them had been training an average of 12 hours/week during the past year, and in particular they had performed swimming, long distance running and cycling at workloads of 70% to 90% of theoretical maximal heart rate. All the subjects in the control group did not participate in any routine competitive or recreational sports. The ethics committee of our institution approved the study and informed consent was obtained from all individuals.

### **Echocardiography**

A two-dimensional echocardiographic study using a commercially available ultrasound scanner (Vivid-Q, GE Healthcare, Milwaukee, WI) with a 2.5-MHz phased array transducer (M4S) was performed in all subjects at baseline and during exercise. Standard echocardiographic views were obtained, including parasternal long and short axis, as well as apical two and four chamber views. Particular care in the acquisition of atrial images was taken. Frame rates were at least 60 Hz for two dimensional images. Stress echocardiography

was performed with an ergometric supine bicycle and images were acquired at maximum exercise (previous to fatigue). The maximum exercise tolerance was expressed in Watts and estimated by metabolic equivalents (METs) and O<sub>2</sub> consumption (mL/Kg/min).

Images were analyzed offline with commercially available software (EchoPac version 108.1.6, GE Healthcare, Milwaukee, WI). LV and right ventricle (RV) size measurements were done according to the recommendations of the American Society of Echocardiography (Lang, 2006). LA volume was calculated from apical four and two-chamber views using the biplane method summation of discs (MacClean, 2014) and RA volume was measured from the apical four chamber view. LA and RA volumes were measured at three instants: (i) just before the opening of the mitral valve (maximum volume), (ii) at pre-atrial (A) contraction volume, obtained at mid-diastole at the beginning of the P wave on surface electrocardiogram., and (iii) at the minimum volume just at the closure of the mitral valve. Atrial active emptying volume (AEV) was estimated as the difference between the pre A-wave volume and the minimum atrial volume and was used as a surrogate for stroke volume produced by atrial contraction. (Atrial active emptying fraction) AEF was calculated using the formula: (pre-A wave volume – minimum volume)/pre A-wave volume. Absolute differences between rest and exercise parameters were also calculated. Cardiac chamber size measurements were indexed by the body surface area. All data were analyzed by blinded, experienced sonographers. All measurements were done at baseline and during exercise. Significant LA dilatation was defined using a cut-off volume of 40 ml/m<sup>2</sup> (Lang, 2006) and arbitrarily the same value for RA.

#### **Atrial strain and strain rate**

Strain and strain rate of both atria were analyzed offline with a commercially available software package (2Dstrain, EchoPac version 108.1.6) from images acquired in the four-chamber view. The endocardial border was manually traced using a point-and-click technique. For speckle tracking analysis, we selected images with at least 60 frames/sec (Gabrielli, 2014). LA and RA active strains (LA negative peak strain during atrial contraction (LASa) and RA negative peak strain during atrial contraction (RASa), respectively)

were calculated with the reference point set at the onset of the P-wave of the surface ECG (Rimbaş, 2014), which allowed identifying the peak negative strain (active shortening) during atrial contraction (Figure 1). Similarly, in the strain-rate curve we identified the peak negative strain rate (active shortening) during atrial contraction and the peak positive strain rate (stretching) during ventricular contraction in each atrium (LA negative peak strain rate during atrial contraction (LASRa) and RA negative peak strain rate during atrial contraction (RASRa), respectively). The software divided the atrial wall into 6 segments and the average was taken for analysis. Segments in which inadequate tracking was observed were excluded from further analysis and the remaining segments were averaged. Analysis was done at rest and during exercise (Figure 1).

### **Statistical methods**

Continuous baseline variables were expressed as mean  $\pm$  standard deviation (SD) values or median (interquartile range), after checking for normal distribution as assessed by the Kolmogorov-Smirnov test. Categorical variables were expressed as total number (percentages) and compared between groups using Chi-square or Fisher's test when appropriate. Depending on the normality of the distribution, continuous variables were tested by unpaired T-test or Mann-Whitney U-test (unpaired data) and by paired T-test or Wilcoxon analysis (paired data). **Complementarily, statistical differences in exercise response were assessed by mixed factorial ANOVA, where time (rest/exercise) is the within-subject factor, and group (control/athlete) is the between-subject factor. Box's test was used to control the homogeneity of variances.** Pearson or Spearman methods were used to analyze the correlation between continuous variables when suitable. Intra-and inter-observer reproducibility was assessed using Bland-Altman analysis, on 10 different sequences/subjects for each strain and strain rate parameter by two different observers, respectively. Statistical significance was established at  $p < 0.05$ . All data were analyzed using the SPSS version 15.0 (SPSS, Inc., Chicago, IL).

## RESULTS

### Population characteristics

All subjects were male, and the two populations had similar age ( $37 \pm 6$  [athletes] vs.  $35 \pm 4$  [controls] year old,  $p =$  non significant [NS]) and body surface areas ( $1.9 \pm 0.1 \text{ m}^2$  [athletes] vs.  $1.9 \pm 0.2 \text{ m}^2$  [controls],  $p =$  NS). The average athletes' training story was of  $12 \pm 7$  years at the moment of the study.

Hemodynamic parameters at rest and during exercise in both groups are summarized together with ventricular performance in Table 1. As previously anticipated, athletes showed significantly higher oxygen consumption and reached higher METs and exercise load as compared to controls.

LV and RV dimensions were significantly larger in athletes as compared to controls at baseline (rest). During exercise, both groups showed a significant decrease in LV and RV volumes. LV ejection fraction and RV fractional area change were similar at rest and during exercise in both groups.

### LA and RA dimensions and function at rest and during exercise.

Table 2 summarizes all the data regarding LA and RA dimensions and function at rest and during exercise in both groups. Athletes showed significantly higher LA volumes, including active emptying volume, at rest and during exercise as compared to controls. Additionally, both athlete and control groups showed a significant increment in LA active emptying volume and a significant decrease in LA volumes with exercise. Regarding LA deformation parameters, LASa and LASRa were similar among athletes and controls at rest, but significantly lower in athletes during exercise. A similar behavior regarding RA volumes and deformation parameters was observed among athletes and controls.

Figure 2 (A-D) depicts the interplay between atrial volume and deformation both for the LA and RA, through individual changes with exercise. Athletes globally showed a lower increment of atrial deformation parameters with exercise, ( $\Delta\text{LASa} = -8.4 \pm 4.8\%$  [athletes] vs.  $-12.2 \pm 4.5\%$  [controls],  $p < 0.01$ ;  $\Delta\text{LASRa} = -1.28 \pm 1.03\text{s}^{-1}$  [athletes] vs.  $-1.76 \pm 0.74\text{s}^{-1}$  [controls],  $p = 0.03$ ;  $\Delta\text{RASa} = -7.2 \pm 4.8\%$  [athletes] vs.  $-10.3 \pm 3.9\%$  [controls],  $p = 0.01$ ; and  $\Delta\text{RASRa} = -0.79 \pm 0.69\text{s}^{-1}$  [athletes] vs.  $-1.11 \pm 0.72\text{s}^{-1}$  [controls],  $p = 0.07$ ). [In terms of](#)



group\*exercise interaction, significant differences were observed for both LASa and LASRa ( $p=0.001$  and  $p=0.037$ , respectively). Tests could not be performed on RASa as equality of variances cannot be assumed ( $p<0.001$  in Box's test), and RASRa returned non-significant differences. However, when athletes with significant LA dilatation i.e. LA volume  $\geq 40$  mL/m<sup>2</sup> (right part of Figure 2A and 2C) were excluded from the analysis ( $n= 8$ ), no significant differences were observed between athletes and controls regarding the increase in LASa and LASRa during exercise ( $p=$  NS, for both). The same observation was obtained excluding athletes with RA volume  $\geq 40$  mL/m<sup>2</sup> ( $n= 15$ ) (right part of Figure 2B and 2D): no significant differences were observed between athletes and controls in the increase of RASa and RASRa during exercise ( $p=$  NS, for both).

#### **Subgroup of athletes with significant LA/RA atrial dilatation**

Figures 3, A-F show changes in atrial active deformation and atrial active emptying volume under the effect of exercise, in athletes with and without significant atrial dilatation. The subgroup of athletes with severely enlarged atria showed significantly less atrial deformation at rest and during exercise and a significantly lower increment during exercise, as compared to athletes without significant atrial dilatation [Figures 3A and 3C for LA (8 subjects with significant LA dilatation); Figures 3B and 3D show the same data for the RA (15 subjects with significant RA dilatation)]. This subgroup also showed a significantly lower increment of atrial active emptying volume during exercise as compared to athletes with smaller atria (Figures 3E and 3F). All subjects with significant LA dilatation ( $n=8$ ) showed also significant RA dilatation ( $n=15$ ).

Finally, a modest, but statistically significant correlation between reached METs during the exercise test and LA active emptying volume at rest and during exercise among the studied population were found (Figure 4)

#### **Quality of the speckle tracking procedure.**

Appropriate wall tracking (corresponding to low drift artifacts, as indicated by the software interface) was obtained in 97.5% of the segments evaluated at rest (LA and RA), in 93.7% of the LA and in 92.2% of the RA

segments during exercise. Animated sequences of the speckle tracking output at rest and during exercise, illustrate the tracking quality on a concrete example are available as Supplementary data.

Reproducibility of the measurements (LA and RA, strain and strain rate, at rest and during exercise) is represented in Figure 5 (intra-observer agreement) and Figure 6 (inter-observer agreement) as Bland-Altman plots. In both we showed a good agreement.

## **DISCUSSION**

The present study showed that: (i) at rest, highly trained athletes had more dilated atria (both LA and RA), showed similar active atrial deformation parameters but an increased atrial active ejection volume, as compared to control subjects; (ii) athletes and controls showed a significant increment in atrial deformation parameters and active emptying volume during active atrial contraction with exercise; (iii) a subgroup of athletes, with significant atrial dilatation, showed lower increment in deformation parameters with exercise together with a significant lower increment in active emptying volume as compared to athletes with smaller atria. Moreover, the significant correlation found between atrial active emptying volume and exercise performance, supports the importance of atrial function and its response during exercise. These findings are in agreement with those of Kusunose (2012), who showed an independent association of LA function with exercise capacity in patients with preserved ejection fraction.

### **Atrial remodeling**

While more data has been reported on the LV performance of athletes, atrial dimensions and function are scarcely studied in this population (Pluim, 2000). [Previous echocardiographic reports, including endurance and non-endurance athletes have showed that LA dilatation \(assessed with determination of the anteroposterior diameter of the LA\) is common among athletes, but no relationship with supraventricular arrhythmia development was found \(Pelliccia, 2005\), in fact atrial mechanical function at rest is preserved](#)

in this population (Brugger, 2014). However, more recent experimental and clinical reports including endurance athletes have indeed shown an association between atrial remodeling and atrial arrhythmia development in the long-term follow-up (Benito, 2011; Mont, 2009; Van Buuren, 2012).

The atrial remodeling observed in athletes is an adaptive mechanism to the exposed volume overload (D'Andrea 2010; Voeller, 2011) with the objective of supporting the increased demand in cardiac output, and probably a mild or moderate remodeling process represents a physiologic and benign response in the vast majority of subjects (D'Andrea, 2010). However, this leads to different atrial working conditions at rest and during exercise, with less deformation and larger volumes, which potentially provides a better functional reserve but also results in an increased atrial wall tension, particularly in the subgroup with larger atrial volumes (Gabrielli, 2014). We have previously demonstrated this increased atrial wall stress with computer models that assumed that deformation increased with exercise (Gabrielli, 2014); the present work further supports our previous findings in confirming that atrial deformation actually increases during exercise both in controls and athletic subjects. In fact, elevated wall stress is an important risk factor for the initiation/worsening of myocardial damage (Marciniak, 2007; Di Martino, 2011). On the longer run, this atrial dysfunction derived from excessive atrial adaptation (remodeling) might result in increased vulnerability to atrial arrhythmias, through increased wall stress triggering atrial myocardial stretching and fibrosis (Margreet, 2010; Tsai, 2011). These findings are also consistent with observations on experimental animal models. In a rat model of long-term intensive exercise training, atrial fibrosis was demonstrated to be associated with an increased inducibility of atrial arrhythmia (Benito, 2011). However, further experiments on a dedicated study that include contractility would be necessary for a better assessment of atrial wall stress.

#### **Significant atrial dilatation: atria already in trouble?**

Additionally, we also identified a subgroup of endurance athletes with a significantly more advanced atrial remodeling process and evidence of contractile dysfunction. In our study population, using biplane indexed atrial volume we found that 30% of mid-thirties marathon runners showed significant RA dilatation and

16% significant LA dilatation, as defined by a reference cut-off point of 40 ml/ m<sup>2</sup> (Lang, 2006); moreover a subgroup of these subjects had severe atrial dilatation according to recently published guidelines (Lang, 2015). Our study showed a different active atrial deformation pattern and exercise response among these athletes. These observations add relevant information about the atrial remodeling process that is more pronounced in this subgroup of athletes and could potentially translate into a substrate for future atrial arrhythmias, and not only a physiological adaptation mechanism of LV filling process. In order to confirm this hypothesis, a long-term follow-up study is needed, especially in this particular subgroup of athletes. The reason why other athletes, with similar training loads, showed only modest atrial remodeling warrants further investigation. These individuals may probably have their own threshold for potential atrial myocardial damage in a dose-dependent (exercise load) fashion (O'Keefe, 2012).

Another important issue concerns the higher remodeling observed for the right atrial chamber with respect to the left one. This is in agreement with other observations of more pronounced structural remodeling of right-sided cardiac chambers in endurance athletes (La Gerche, 2012; D'Ascenzi, 2013).

### **Limitations**

One limitation of our study was the lack of a strict homogenization of athletes' population regarding their training intensity and performance, which could help in the identification of the subjects with the biggest risk of atrial remodeling. The impact of different body sizes was normalized by indexing cardiac dimensions by body surface area; however, it is well recognized that this may not fully counteract the effect of absolute atrial myocardial mass, a factor that has been also related to the occurrence of atrial fibrillation (Mont, 2008). Moreover, the changes in atrial performance that we observed could not be extrapolated to other forms of cardiac loading rather than supine bicycle and the maximal oxygen consumption reached could be underestimated due to discomfort in doing exercise in the supine bicycle.

Another limitation, inherent from the use of echocardiographic parameters, is the reproducibility of atrial strain measurements particularly during exercise. Additionally, current speckle tracking tools may perform worse when applied to the right side of the heart and the atria, and during exercise, specially due to the

lower temporal resolution, the lower quality of the images, and possible a-priori design of the algorithms for the left ventricle; however we believe it is based in principles that can be applied to atrial function assessment (Behar, 2004). Also we paid special attention to the correct tracking of the images and we have also reported the feasibility and reproducibility of our measurements, with adequate intra- and inter-observer variability. Consequently, we believe that these measurements are reliable provided the observer takes the time to verify frame-by-frame that the proposed tracking actually follows the wall seen in the echocardiographic image, and that the deformation values actually reflect what is seen in the image. Animated figures showing a concrete example of the tracking quality at rest and during exercise are available as Supplementary data. We used optimized views to perform our reproducibility study, and, despite the same loops were used to repeat measurements, all endocardial traces were re-traced from scratch, blinded to the previously done segmentations in a different frame image. [A reliability study that includes sequences taken at different times could add more confidence to the method, and perhaps the concomitant use of tissue Doppler imaging.](#)

The assessment of atrial myocardial deformation was only performed from images in the four-chamber apical view; potentially, assessing also LA segments from the two-chamber apical view would have provided more comprehensive information for atrial function characterization, and also the analysis of reservoir and conduit function. Additionally, speckle tracking derived values of strain and strain rate may be slightly underestimated because of the elevated heart rate at higher exercise intensities.

[Another consideration to interpret our data is that reservoir and conduit function during exercise were not analyzed. These concepts might help for a better understanding of atrial changes in this population. However, they do not consist in the most predominant change observed during effort, which was the active phase.](#)

[Finally, our population of athletes was small in comparison with other studies in the field \(D'Andrea, 2010\); this issue might partially influence the conclusions from the subgroup analysis, but is strong enough for raising our primary hypothesis.](#)

### **Perspective**

To our knowledge this is the first study reporting atrial performance during exercise in athletes, demonstrating the expected increase in atrial contractile function that contributes to the increased cardiac output demand and to functional capacity. Importantly, we identified a subgroup of athletes with potential atrial dysfunction and adverse remodeling that **might identify a subgroup with a different atrial behavior**. Moderate aerobic sport is essential for cardiovascular health (Biswas,2015), but an excessive program training for a single subject potentially could lead to an adverse cardiac remodeling process.

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### **Competing interests**

Authors have nothing to disclose

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**Table 1.** Hemodynamic and ventricular characteristics of athletes and controls

	Baseline			Peak Exercise		
	Athletes (n=50)	Controls (n=30)	p	Athletes (n=50)	Controls (n=30)	P
Heart Rate (bpm)	59 ± 9	69 ± 12	< 0.01	156 ± 13 ¶	156 ± 9 ‡	NS
Systolic BP (mmHg)	125 ± 9	119 ± 6	< 0.01	198 ± 17 ¶	191 ± 20 ‡	NS
Diastolic BP (mmHg)	79 ± 4	77 ± 4	NS	81 ± 3 ¶	80 ± 5 ∆	NS
Maximum load (Watts)	-	-	-	224 ± 38	169 ± 26	< 0.01
METs	-	-	-	11.4 ± 1.6	8.8 ± 1.3	< 0.01
Estimated O <sub>2</sub> consumption (mL/kg/min)	-	-	-	40 ± 5.6	31 ± 4.6	< 0.01
LV Cardiac Index (l/min/m <sup>2</sup> )	2.52 ± 0.63	2.53 ± 0.44	NS	8.33 ± 3.18 ¶	7.04 ± 1.24 ‡	0.02
LVEF (%)	61 ± 5	61 ± 5	NS	68 ± 5 ¶	66 ± 3 ‡	NS
LV end-diastolic volume index (mL/m <sup>2</sup> )	60.9 ± 12.2	45.9 ± 6.9	< 0.01	51.3 ± 10.7 ¶	38.9 ± 7.4 ‡	< 0.01
LV end-systolic volume index (mL/m <sup>2</sup> )	24.0 ± 4.3	19.0 ± 4.9	< 0.01	16.0 ± 4.2 ¶	12.8 ± 3.1 ‡	< 0.01
RV end-diastolic area (cm <sup>2</sup> )	26.7 ± 4.7	20.2 ± 4.4	< 0.01	23.4 ± 4.4 ¶	17.1 ± 4.6 ‡	< 0.01
RV end-systolic area (cm <sup>2</sup> )	13.3 ± 2.8	10.1 ± 2.2	< 0.01	11.1 ± 2.4 ¶	8.3 ± 3.0 ‡	< 0.01
RV fractional area change (%)	49 ± 6	49 ± 7	NS	52 ± 6 ¶	51 ± 7	NS

¶ p < 0.05 versus athletes at baseline; ‡ p < 0.05 versus controls at baseline.

Abbreviations: BP: blood pressure; LV, left ventricle; LVEF: LV ejection fraction; RV, right ventricle; NS, not significant (p ≥ 0.05)

**Table 2.** Atrial size and function at baseline and during exercise.

	Baseline			Peak Exercise		
	Athletes (n=50)	Controls (n=30)	p	Athletes (n=50)	Controls (n=30)	p
<b>Left atrium</b>						
Area (cm <sup>2</sup> )	18.0 ± 3.6	14.5 ± 2.3	< 0.01	15.7 ± 3.7 ¶	12.7 ± 2.5 ‡	< 0.01
Maximum Volume (mL)	55.9 ± 17.5	36.1 ± 8.8	< 0.01	43.3 ± 18.7 ¶	29.4 ± 8.3 ‡	< 0.01
BSA Indexed Maximum Volume (mL/m <sup>2</sup> )	29.4 ± 9.2	18.6 ± 4.1	< 0.01	22.7 ± 10.2 ¶	15.0 ± 4.0 ‡	< 0.01
Pre- P wave volume (mL)	28.5 ± 10.5	19.1 ± 5.0	< 0.01	23.4 ± 8.7 ¶	15.6 ± 4.2 ‡	< 0.01
Minimum volume (mL)	19.4 ± 8.8	11.9 ± 4.6	< 0.01	10.7 ± 7.6 ¶	5.6 ± 2.8 ‡	< 0.01
Active emptying volume (mL)	9.0 ± 1.2	7.1 ± 2.8	< 0.01	12.6 ± 2.7 ¶	9.9 ± 2.1 ‡	< 0.01
Active emptying volume (mL/m <sup>2</sup> )	4.7 ± 1.4	3.7 ± 0.7	< 0.01	6.6 ± 1.3 ¶	5.1 ± 1.0 ‡	< 0.01
Active emptying fraction	0.34 ± 0.09	0.39 ± 0.09	0.01	0.59 ± 0.14	0.65 ± 0.09	0.02
LASa (%)	-12.7 ± 1.9	-11.7 ± 2.5	NS	-21.4 ± 5.3 ¶	-24.0 ± 4.6 ‡	0.04
LASRa (s <sup>-1</sup> )	-1.57 ± 0.42	-1.58 ± 0.51	NS	-2.85 ± 1.86 ¶	-3.34 ± 0.85 ‡	0.04
<b>Right atrium</b>						
Area (cm <sup>2</sup> )	18.9 ± 4.0	15.2 ± 2.2	< 0.01	17.2 ± 3.8 ¶	13.5 ± 2.3 ‡	< 0.01
Maximum Volume (mL)	65.1 ± 20.7	43.8 ± 10.2	< 0.01	54.7 ± 20.9 ¶	34.5 ± 9.3 ‡	< 0.01
BSA Indexed maximum Volume (mL/m <sup>2</sup> )	34.1 ± 10.9	22.6 ± 4.7	< 0.01	28.6 ± 10.7 ¶	17.8 ± 4.6 ‡	< 0.01
Pre-P wave volume (mL)	43.2 ± 14.5	27.6 ± 8.0	< 0.01	32.5 ± 12.2 ¶	22.0 ± 7.8 ‡	< 0.01
Minimum volume (mL)	34.8 ± 14.2	20.3 ± 7.7	< 0.01	20.6 ± 12.8 ¶	10.6 ± 5.8 ‡	< 0.01
Active emptying volume (mL)	8.4 ± 1.9	7.3 ± 1.4	0.01	11.9 ± 2.6 ¶	11.4 ± 3.2 ‡	NS
Active emptying volume (mL/m <sup>2</sup> )	4.4 ± 0.9	3.8 ± 0.7	< 0.01	6.2 ± 1.4 ¶	5.9 ± 1.6 ‡	NS
Active emptying fraction	0.22 ± 0.07	0.28 ± 0.08	< 0.01	0.41 ± 0.1 ¶	0.53 ± 0.1 ‡	< 0.01
RASa (%)	-11.9 ± 1.7	-12.4 ± 2.7	NS	-19.2 ± 5.8 ¶	-22.5 ± 3.4 ‡	0.01
RASRa (s <sup>-1</sup> )	-1.52 ± 0.44	-1.57 ± 0.55	NS	-2.13 ± 1.09 ¶	-2.51 ± 1.08 ‡	NS

¶ p < 0.01 versus athletes at baseline; ‡ p < 0.01 versus controls at baseline.

Abbreviations: BSA: body surface area; LASa, LA late diastolic (atrial contraction) peak negative strain; LASRa: LA late diastolic (atrial contraction) peak negative strain rate; RASa, RA late diastolic (atrial contraction) peak negative strain; RASRa, RA late diastolic (atrial contraction) peak negative strain rate.

## Figure Legends

**Figure 1. Left atrial strain curves of a given subject at rest (Panel A) and during exercise (Panel B).** Arrows indicate the peak negative left atrial strain during active atrial.

**Figure 2. Scatterplot of individual changes in the active LA/RA deformation with exercise in athletes (blue) and controls (red).** Each line links baseline (dot symbol) and exercise (cross symbol) values for a given individual. The subgroup with significant atrial dilatation according to the indexed atrial volume is also indicated. LASa: LA late diastolic (atrial contraction) peak negative strain; LASRa: LA late diastolic (atrial contraction) peak negative strain rate; RASa, RA late diastolic (atrial contraction) peak negative strain; RASRa, RA late diastolic (atrial contraction) peak negative strain rate.

**Figure 3. Changes in atrial contractile function with exercise in athletes with significant (atrial volume  $\geq 40 \text{ mL/m}^2$ ) or athletes with non-significant atrial dilatation (atrial volume  $< 40 \text{ mL/m}^2$ ).** Left and right panels correspond to the LA and RA measurements, respectively. LASa: LA late diastolic (atrial contraction) peak negative strain; LASRa: LA late diastolic (atrial contraction) peak negative strain rate; RASa, RA late diastolic (atrial contraction) peak negative strain; RASRa, RA late diastolic (atrial contraction) peak negative strain rate.

**Figure 4. Contribution of LA contractile function to exercise capacity.** Scatterplots of the relationship between LA rest and exercise active emptying volume and metabolic equivalents (METS) reached in the stress test.

**Figure 5. Bland-Altman plots for the intra-observer reproducibility of Sa and SRa measurements of the LA and RA, at rest (top) and during exercise (bottom).** Values are expressed as mean differences and 95% confidence intervals (% for Sa and  $\text{s}^{-1}$  for SRa).

**Figure 6. Bland-Altman plots for the inter-observer reproducibility of Sa and SRa measurements of the LA and RA, at rest (top) and during exercise (bottom).** Values are expressed as mean differences and 95% confidence intervals (% for Sa and  $s^{-1}$  for SRa).

Figure 1

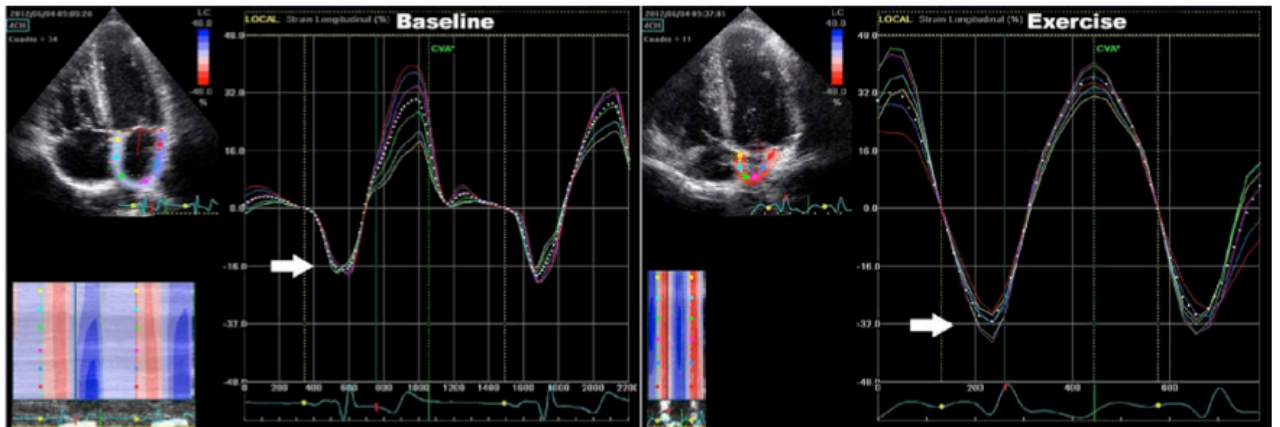




Figure 2

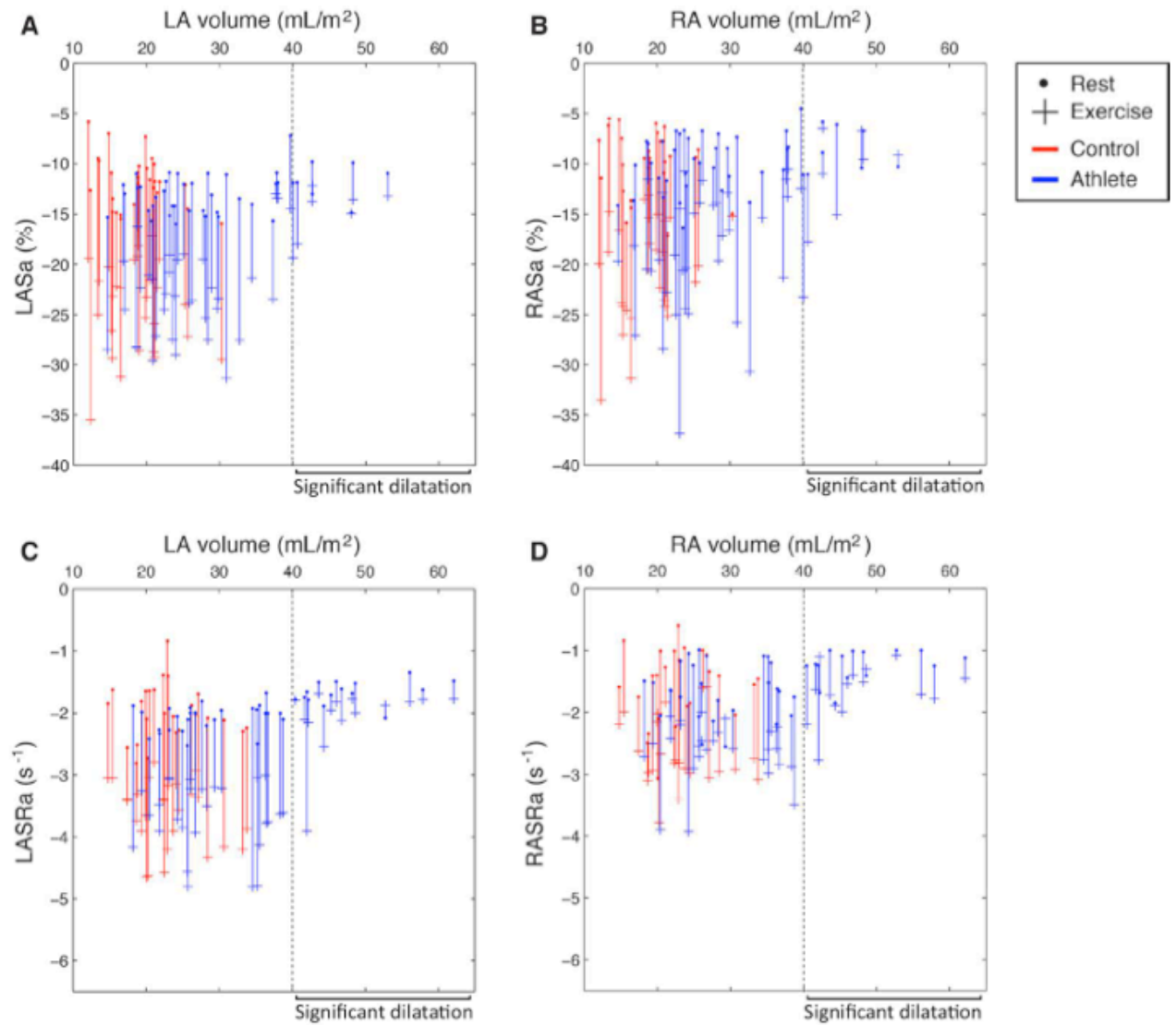


Figure 3

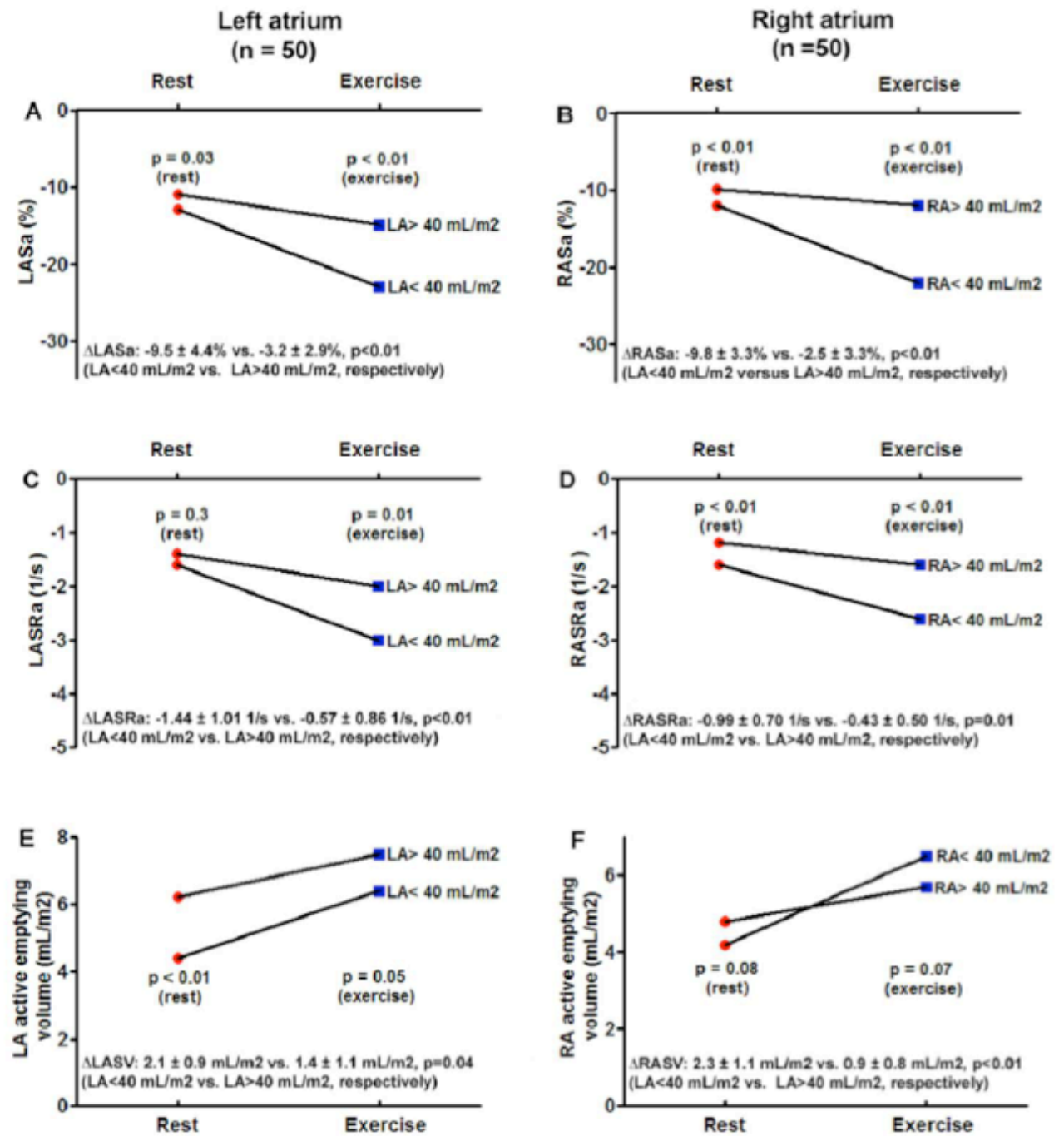


Figure 4

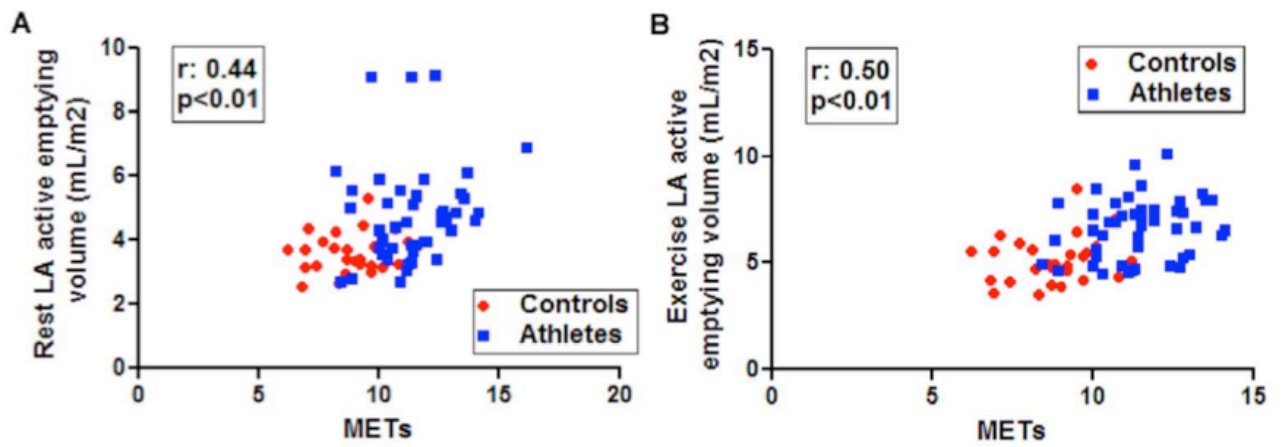


Figure 5

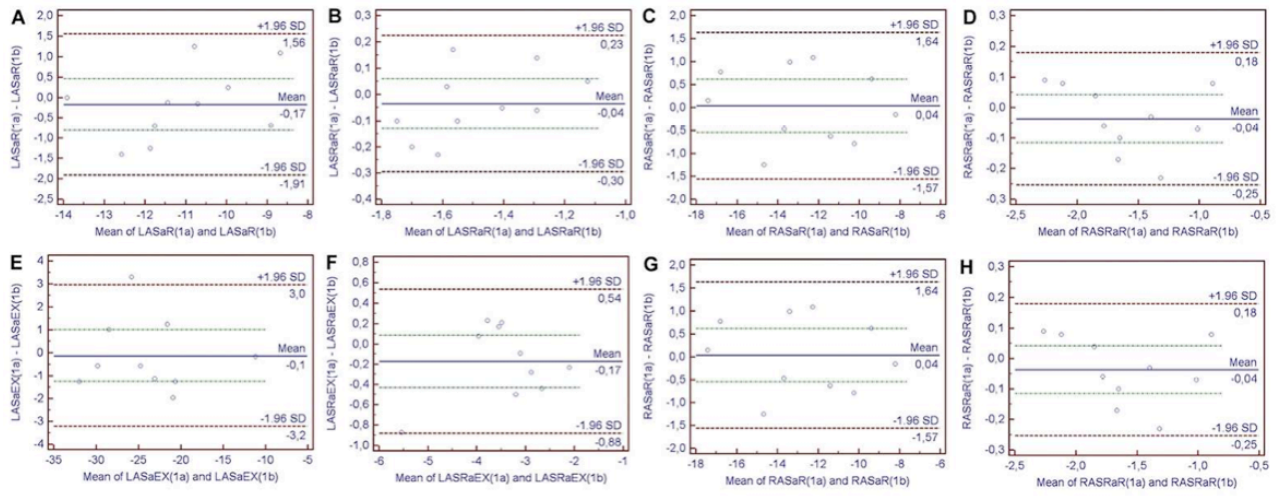


Figure 6

