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**Citation** (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

**McClea, G, George, KP, Lord, R, Utomi, V, Jones, N, Somauroo, J, Fletcher, S and Oxborough, D (2015) Chronic adaptation of atrial structure and function in elite male athletes. EUROPEAN HEART JOURNAL-CARDIOVASCULAR IMAGING. 16 (4). pp. 417-422. ISSN 2047-2404**

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## **Chronic Adaptation of Atrial Structure and Function in Elite Male Athletes**

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## ABSTRACT

**Aims:** To establish the degree of structural and functional adaptations in the left (LA) and right atria (RA) in elite male athletes engaged in “high dynamic:high static” (HDHS) and “low dynamic:high static” (LDHS) sporting disciplines compared to sedentary controls.

**Methods and Results:** 18 male, elite HDHS athletes (13 boxers and 7 triathletes), 18 male, elite LDHS athletes (8 weightlifters and 10 Akido) and 20 male, age-matched sedentary controls were assessed using conventional 2D and myocardial speckle tracking (MST) echocardiography. Absolute LA and RA volumes (end systole (VOLes), Pre A (VOLpreA) and end diastole (VOLed)) as well as the functional indices of reservoir (RESvol), conduit (CONvol) and booster volumes (BOOvol) were defined. MST allowed the assessment of atrial strain ( $\epsilon$ ) during the reservoir (RES $\epsilon$ ), conduit (CON $\epsilon$ ) and booster (BOO $\epsilon$ ) phases of the cardiac cycle. Both LA and RA size were significantly larger in HDHS compared to LDHS and controls ( $P < 0.05$ ) across all structural and functional volume parameters with no significant difference between LDHS and controls (LAVOLes  $35 \pm 8$  ml/m<sup>2</sup>,  $26 \pm 10$  ml/m<sup>2</sup> and  $23 \pm 5$  ml/m<sup>2</sup>; RAVOLes  $37 \pm 10$  ml/m<sup>2</sup>,  $26 \pm 9$  ml/m<sup>2</sup> and  $23 \pm 5$  ml/m<sup>2</sup>, LARESvol  $35 \pm 9$  ml,  $25 \pm 11$  ml and  $23 \pm 7$  ml, RARESvol  $41 \pm 11$  ml,  $34 \pm 11$  ml and  $28 \pm 7$  ml for HDHS, LDHS and controls respectively). RA:LA ratios were greater than 1 in all groups due to a comparatively larger RA volume (RAVOLes : LAVOLes  $1.05 \pm 0.26$ ,  $1.12 \pm 0.55$  and  $1.04 \pm 0.28$  for HDHS, LDHS and controls ( $P > 0.05$ )). There was no significant between group differences for any  $\epsilon$  parameter.

**Conclusion:** Bi-atrial hypertrophy is demonstrated in HDHS athletes and not LDHS athletes suggesting that the dynamic component to training is the primary driver for both LA and RA adaptation. Although functional data derived from volume shifts suggest augmented function in HDHS athletes, MST imaging demonstrated no difference in intrinsic atrial  $\epsilon$  in any of the groups.

**Key Words:** Athletes Heart; Left Atrium; Right Atrium; Strain Imaging; Echocardiography

## INTRODUCTION

The athlete's heart (AH) has been relatively well described with particular attention to the structure and function of both the left (LV) and right ventricles (RV)<sup>1</sup>. The predominant adaptation appears to be one of chamber enlargement (eccentric hypertrophy) affecting endurance trained athletes to a greater extent than those athletes predominantly involved in resistance training<sup>2,3,4</sup>. Whilst data on the ventricles has been forthcoming, there is limited comprehensive structural and functional data available on the left (LA) and right atrium (RA) of elite male athletes of varying training types<sup>5,6,7</sup>. In addition, the RV has previously been shown in endurance athletes to adapt disproportionately when compared to the LV<sup>4</sup>, however the relationship of RA to LA size has not been explored.

Previous studies that have assessed cardiac adaptation in athletes have described training type as either endurance or resistance<sup>1,9</sup>, however the definitions of these groups are often ill-defined. In reality, sports exist within various forms, and as such classification as "endurance" or "resistance" may be overly simplistic, ineffectively reflecting the haemodynamic volume load of training and competition in any sport. The task force classification of the American College of Cardiology (ACC)<sup>10</sup>, developed a more complex classification system based on the exposure to acute dynamic (isotonic) and/or static (isometric) muscle component. Dynamic exercise is a whole body exercise causing a marked increase in oxygen consumption, with a moderate increase in blood pressure whilst static exercise results in a smaller increase in oxygen consumption, with significant increases in blood pressure. In this way athletes can be differentiated by both the dynamic and static components of their training (for example a boxer is defined as high dynamic/high static [HDHS; group CIII] whilst a weightlifter is defined as low dynamic:high static [LDHS; Group AIII]) It is therefore believed that athletes from these contrasting training groups pose the ideal model of comparison for structural and functional adaptations within the atria.

When compared to the non-athletic population, athletes have been documented to be at a higher risk of developing atrial fibrillation (AF)<sup>11</sup>. The specific mechanisms have not been fully determined, however atrial size and function may be a contributing factor. It is therefore clear that a greater understanding of atrial physiology in a well-defined training specific athletic population may provide some insight into those that are at a higher risk of AF development.

In view of this, the study aims to establish LA (RA) structure and function in HDHS and LDHS athletes and sedentary controls. This broad aim leads to three specific hypotheses.

- 1) HDHS athletes will have larger atrial volumes during ventricular systole and therefore greater functional volumes than LDHS athletes and sedentary controls
- 2) HDHS athletes will have superior atrial function when compared to LDHS athletes and sedentary controls
- 3) Relative RA to LA ratio will be greater in HDHS compared to LDHS athletes and sedentary controls

## **METHODS**

### ***Study design and Population***

This study utilised a cross-sectional design consisting of two groups of elite athletes as classified by the task force classification of the ACC<sup>11</sup>. Following an *a priori* sample size power calculation aimed at discerning a 5% difference in indexed atrial volume and atrial strain ( $\epsilon$ ), 18 male HDHS athletes (CIII) ( $> 70\% \text{ VO}_{2\text{max}}$ ,  $> 50\%$  maximum voluntary contraction (MVC)) included 11 boxers and 7 triathletes; (mean age,  $28 \pm 8$  years; range, 16 - 41 years) and 18 male LDHS athletes (AIII) ( $< 40\% \text{ VO}_{2\text{max}}$ ,  $> 50\%$  MVC included 8 weightlifters and 10 aikido athletes; (mean age,  $26 \pm 7$  years; 17 - 40 range years) were prospectively recruited. Average

weekly training hours per week were  $13 \pm 5$  hrs/week and  $10 \pm 3$  hrs/week for HDHS and LDHS athletes respectively. The number of competitive training years were  $10 \pm 7$  years and  $11 \pm 7$  years for HDHS and LDHS athletes respectively. In addition, 20 male age matched sedentary controls (CON), (defined as  $< 3$  hours exercise per week) were recruited (mean age  $27 \pm 8$  years; range 20 - 43 years). All subjects were healthy and free from known cardiovascular disease and not taking any form of prescribed medication. All subjects provided written informed consent to participate, and ethics approval was granted by the Liverpool John Moores University Ethics Committee.

### ***Procedures***

After a full explanation of procedures weight (Seca 217, Hannover, Germany) and height (Seca Supra 719, Hannover, Germany) were recorded. Following 5 minutes of seated rest, left brachial artery blood pressure (BP) was obtained (GE Dinamap Pro 300 V2 Vital Signs Monitor, USA). A resting 12-lead electrocardiogram (ECG) was performed (CardioExpress SL6, Spacelab Healthcare Washington, US) followed by an echocardiographic examination. All echocardiographic images were acquired by a single experienced sonographer using a commercially available ultrasound system (Vivid Q; GE Healthcare, Horten, Norway) with a 1.5-MHz to 4-MHz phased-array transducer and heart rate (HR) was acquired from the ECG inherent to the ultrasound system.. All images were acquired with the subject lying in the left lateral decubitus position and recorded to DVD in a raw Digital Imaging and Communications in Medicine format (DICOM). All data were analysed offline by a single experienced operator using commercially available software (EchoPAC version 6.0, GE Healthcare, Horten, Norway).

### ***Conventional 2D Echocardiography***

Standard 2D echocardiographic parameters were obtained from parasternal and apical acoustic windows. All settings were optimised to obtain maximum signal-to-noise ratio and optimal endocardial delineation. LA volumes were obtained using the acoustic windows of apical 4 and 2-chambers, with the biplane Simpsons method, according to the American Society of Echocardiography guidelines<sup>12</sup> whilst RA volumes were acquired using the 4-chamber orientation with a monoplane Simpsons method. For both chambers, volumes were calculated at end ventricular systole (LA(RA)VOLes), pre-atrial contraction (LA(RA)VOLpreA) and at end ventricular diastole (LA(RA)VOLed). Volumes permitted calculation of atrial reservoir volume (LA(RA)RESvol) defined as the difference between LA(RA)VOLes and LA(RA)VOLed, atrial conduit volume (LA(RA)CONvol) defined as the difference between LV stroke volume (measured using a biplane Simpsons method) and LA(RA)RESvol and atrial booster pump volume (LA(RA)BOOvol) defined as the difference between LA(RA)VOLpreA and LA(RA)VOLed as previously described<sup>13</sup>. LA linear dimension (LAd) was measured from the parasternal long axis orientation. To obtain accurate values for chamber structural size, all volumes and dimensions were indexed for body surface area (BSA)<sup>14</sup>. Relative ratio of RA to LA (RA:LA) was established from the volumes of LA(RA)es, LA(RA)preA and LA(RA)ed.

### ***Myocardial Speckle Tracking Echocardiography***

Myocardial Speckle Tracking (MST) software was used for the assessment of atrial  $\epsilon$  data. For acquisition an apical 4-chamber orientation was used with frame rates maintained between 40-90 frames per second (FPS). The focal point was positioned at the mid atrial level and all images were optimised to ensure optimal endocardial delineation. Using dedicated software (EchoPAC, version 6.0, GE Healthcare, Horten, Norway), a region of interest (ROI) for both RA and LA was created by tracing around the endocardial surface of the atrial lateral wall, superior wall, and atrial septum using a manually traced point-and-click technique. Tracking quality was determined by both the software and the operator and if any segments were considered unacceptable the participant was excluded from the study. Global LA(RA)  $\epsilon$

was reported as an average of 6 myocardial segments allowing assessment during the reservoir phase (defined as the peak positive value during ventricle systole) (LA(RA)RES $\epsilon$ ), the conduit phase (the difference between peak positive  $\epsilon$  and the starting point of diastasis) (LA(RA)CON $\epsilon$ ) and the booster phase (the difference between terminal diastolic strain and end diastole (immediately following the P wave on the ECG) (LA(RA)BOO $\epsilon$ ) (see Figure 1).

### ***Data Analysis***

Following assessment for normal distribution using a Kolmogorov-Smirnov test, demographic and echocardiographic data from the 3 groups were analysed using a one-way between-subjects ANOVA with an alpha value set to  $p=0.05$ . In order to establish the impact of training longevity on atrial remodelling a Pearson's bivariate correlation was undertaken. All data was analysed using the Statistical Package for Social Sciences software program (SPSS) (version 20). Previous data collected in our laboratory demonstrated excellent intra-observer reliability for peak atrial  $\epsilon$  with a coefficient of variation (CoV) = 6% and intra-class correlation coefficient (ICC) = 0.969<sup>15</sup>.

### **RESULTS**

All participant demographics are presented in Table 1. All resting 12-lead ECG's were considered normal as defined by the European Society of Cardiology<sup>16</sup>. There was no difference between any of the groups for age, systolic and diastolic BP and BSA whilst there was no difference in training years between the athlete groups. HDHS athletes had a significantly lower heart rate (HR) than both LDHS athletes and controls ( $50 \pm 9$ ,  $72 \pm 18$  and  $63 \pm 9$  beats.min<sup>-1</sup>, respectively) whilst training hours per week were higher in HDHS athletes when compared to LDHS athletes ( $13 \pm 5$ , and  $10 \pm 3$  hrs/wk, respectively). There was no significant correlation of training years to any parameter of atrial structure.

### ***Conventional Echocardiography***

All atrial structural data are presented in Table 2. HDHS athletes had higher indexed LAd, LA(RA)VOLes, LA(RA)VOLpreA, LA(RA)VOLed when compared to both LDHS athletes and controls ( $P < 0.05$ ). There were no significant differences for any of the parameters of atrial structure between LDHS and controls. RA:LA ratios were greater than 1 for all parameters of size but not significantly different between any of the groups.

Functional volume data are presented in Table 2. HDHS athletes had a significantly larger LA(RA)RESvol than LDHS athletes, as well as significantly larger LA(RA)CONvol LABOOVol and RARESvol compared to controls. There were no significant differences for any of the functional volume parameters between LDHS athletes and sedentary controls.

### ***Myocardial Speckle Tracking***

Atrial  $\epsilon$  values are presented in Table 3. There were no statistically significant differences for any of the  $\epsilon$  indices between the three groups.

## **DISCUSSION**

The main findings of this study were 1) HDHS athletes have larger atrial dimensions and volumes than both LDHS athletes and controls, which subsequently provided this group with larger functional volumes, 2) there are no significant differences in atrial  $\epsilon$  as determined by MST between any of the groups and 3) although RA:LA structural ratio data were greater than 1 for all variables none of these were different between athlete groups or sedentary controls.

## ***Atrial Structure***

LA enlargement has previously been documented in athletes engaged in high-dynamic training<sup>17</sup> and the current study confirms this, with the additional value of demonstrating that LA size is consistently larger throughout the cardiac cycle. In addition, novel data from the current study highlighted that the RA adapts in a similar fashion, supporting the concept that chronic dynamic training contributes to a “bi-atrial” hypertrophy of the myocardium<sup>1</sup>. Atrial enlargement is likely related to the sustained elevation in preload experienced during dynamic training that causes a repetitive volume challenge<sup>18</sup>. This enlargement permits an increased capacity to meet the high-intensity workload through an amplified atrial ejection volume to the simultaneously dilating ventricle<sup>19</sup>. Enlargement may be further compounded by increased expression levels of the B-myosin heavy chain isoform (fundamental to chamber enlargement) as evidenced from chronic dynamic training within animal studies<sup>20</sup>.

In contrast, structural remodelling was not observed in LDHS athletes. This could be explained by the limited elevation in preload during static training, due to its intermittent nature of repetitions with sets and work-to-rest ratios. Additionally, a Valsalva manoeuvre may be integrated into a static exercise which would have the impact of increasing intra-thoracic pressures and thereby concomitantly reducing atrial preload<sup>21</sup>. Our data suggest that for an athlete to undergo physiological structural remodelling of the atrium, a chronic sustained elevation of preload must be present.

In contrast to our original hypothesis an increased RA:LA ratio for all structural variables was demonstrated across all groups. This suggests that the RA is larger than the LA in both conditioned and sedentary individuals throughout the cardiac cycle. The consistency of RA:LA ratios across athletic and sedentary populations is at odds with some data observed in the ventricles<sup>4</sup>. Disproportionate RV remodelling in response to high-dynamic training is thought

to be related to the divergent wall stress that the ventricles are exposed to during exercise<sup>22</sup>. In view of our findings we can speculate that dilatation of the RV during prolonged exercise<sup>23,24</sup> may protect the RA and venous system from any relative elevation in afterload. It is clear that future studies aimed at assessing RA structure and pressure during exercise are important in determining the mechanisms involved in this process.

There is limited data pertaining to RA size in healthy individuals however a previous small study suggests that absolute volumes derived from 2D echocardiography are similar between both the RA and LA<sup>25</sup>. Our findings of a larger RA than LA in all groups are of interest and raise clinical / diagnostic issues which require further studies to establish normal RA volumes in a large heterogeneous population.

### ***Atrial Function***

Data from the current study demonstrates that chronic HD training contributes to increased functional volumes of the LA and the RA. HDHS athletes exhibited higher passive and active emptying volumes compared to LDHS athletes and controls, whilst also demonstrating a larger reservoir for pulmonary venous return during LV contraction and isovolumetric relaxation. This improved volumetric flow may be a consequence of increased flexibility and compliance of the ventricular muscle and increased myocardial distensibility at end diastole<sup>3</sup>. In turn it is likely that this would improve atrial function through its dependence on myocardial compliance, preload, and descent of the ventricular base<sup>26</sup>. That aside the increased volumes are likely due to a greater initial starting volume and may not fully reflect superior intrinsic functional capacity. In view of this,  $\epsilon$  imaging was undertaken in order to establish a less load dependent measurement of atrial myocardial function.

We observed no difference in myocardial  $\epsilon$  during any of the phases of the cardiac cycle. This is at odds with previous studies assessing LA  $\epsilon$  in highly trained female athletes have demonstrated reduced values at rest when compared to controls<sup>27</sup> whilst others have demonstrated increased LA diastolic  $\epsilon$  in elite soccer players when compared to controls<sup>6</sup>. The disparity with these studies is difficult to explain but may be partially related to gender, sample size and training type and volume. Here we have utilised a male population specifically defined by the ACC task force criteria as HDHS and LDHS whereas soccer players are defined as HDLS. It may well be a combination of the HS and HD components that create a balanced volume challenge on the atria that maintains intrinsic function. It is clear that further work in this area is required. It is also important to note that  $\epsilon$  has previously been reported to be less dependent on volume load<sup>28</sup> and related to a greater extent to true intrinsic myocardial function and is very likely to explain, in part, normal  $\epsilon$  in the presence of larger atrial functional volumes.

### ***Clinical Implications***

Structural remodelling of the LA and RA has been identified as the main contributor for initiation and persistence of AF<sup>29</sup> and there is strong evidence of an increased prevalence in 'masters' endurance athletes<sup>11</sup>. In view of bi-atrial enlargement being specific to HDHS athletes, it is therefore pertinent to speculate that athletes involved in high dynamic training may be more susceptible to AF and hence additional longitudinal research in the masters HDHS athlete groups would add value to the current evidence base. Interestingly our data was unable to highlight any association between training years and the magnitude of atrial remodelling and thus provides some assurance that training longevity is not the primary driver for atrial enlargement.

Atrial enlargement is also an indicator of underlying pathology secondary to raised ventricular filling pressures in conditions such as hypertrophic cardiomyopathy (HCM) and

arrhythmogenic right ventricular cardiomyopathy (ARVC)<sup>31</sup>. In view of these conditions accounting for 35% and 8% of sudden cardiac death in athletes<sup>19</sup>, it is important to ensure that any atrial enlargement in athletes involved in HDHS activity is physiological in nature. This can be achieved by ensuring that reservoir and conduit functional volumes are equally enlarged, RA:LA ratios are only mildly increased above 1 and atrial  $\epsilon$  is within normal limits. Equally any atrial enlargement in athletes engaged in LDLS activity should be interpreted with caution and corroborative investigations may be warranted.

### ***Limitations***

There are some specific limitations to the study. This study is constrained to a relatively small population of male athletes and therefore in order to fully establish differences in atrial structure and function a much larger sample size in a more diverse athletic population would be required. The model used for MST, provides only an approximation of the global characteristics of the atrial wall, despite the fact that RA and LA structure is complex with a non-contractile atrial septum. The use of a global  $\epsilon$  value was utilised to ensure parity with other studies in this area, however, it could be argued that the assessment of individual segments may be beneficial. Another important limitation relates to the use of linear scaling to BSA as an index of structure, when in reality biologic relationships rarely conform to such linearity<sup>1,32</sup>. We chose to undertake linear ratio scaling in order to conform with clinical guidelines, however an allometric approach may provide added value. The use of BSA as a scaling variable is also problematic in that the body mass component, if predominantly based on fat, rarely influences cardiac size<sup>31</sup>. It would be more accurate to utilise fat-free mass, however the challenges in obtaining the measurement often deem it impossible in the clinical setting.

### **CONCLUSION**

To our knowledge this is the first study that has assessed both LA and RA structure and function, in combination with using novel  $\epsilon$  imaging in specific athletes groups described within the ACC task force classification<sup>10</sup>. The novel findings from this study include bi-atrial hypertrophy throughout the cardiac cycle as well as increased functional volumes in a well-controlled model of high dynamic exercise. The lack of this finding in LDHS athletes suggests that the dynamic component to training is the primary driver for atrial adaptation. Although volumetric function was increased throughout the cardiac cycle in HDHS athletes,  $\epsilon$  imaging demonstrated further novel findings with no significant reduction in intrinsic atrial function in any of the groups. This data may aid pre-participation screening of the athlete in upper normal limits for physiological atrial adaptation and furthermore highlights the potential for a higher risk of AF development in athletes engaged in high-dynamic training loads.

## **ACKNOWLEDGMENTS**

We would like to thank Mr William Dedes, Mr Daniel Green, Mr Punit Mistry and Mr Matthew Smith for their help in recruiting participants.

## **CONFLICTS OF INTEREST**

None declared.

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## FIGURE AND TABLE LEGENDS

**Table 1** - Demographic variables for HDHS athletes, LDHS athletes and sedentary controls.

**Table 2** – Left and right atrial structure and functional volumes for HDHS athletes, LDHS athletes and controls.

**Table 3** - Left and right atrial strain ( $\epsilon$ ) for HDHS athletes, LDHS athletes and sedentary controls

**Figure 1** - Myocardial strain ( $\epsilon$ ) for a single participant of both the left (A) and right atrium (B)

**Table 1** - Demographic variables for HDHS athletes, LDHS athletes and sedentary controls.

<b>Parameter</b>	<b>HDHS Athletes (mean±SD)</b>	<b>LDHS Athletes (mean±SD)</b>	<b>Controls (mean±SD)</b>
<b>Age (years)</b>	28 ± 8	26 ± 7	27 ± 8
<b>Height (m)</b>	1.76 ± 0.07	1.78 ± 0.08	1.76 ± 0.06
<b>Body mass (kg)</b>	73 ± 12 <sup>†</sup>	84 ± 14	80 ± 9
<b>BSA (m<sup>2</sup>)</b>	1.78 ± 0.47	2.02 ± 0.2	1.96 ± 0.13
<b>Systolic BP (mmHg)</b>	131 ± 7	134 ± 9	129 ± 18
<b>Diastolic BP (mmHg)</b>	74 ± 7	74 ± 8	81 ± 14
<b>HR (bpm)</b>	50 ± 9 <sup>*†</sup>	72 ± 18	63 ± 9
<b>Training Hours (hrs/wk)</b>	13 ± 5 <sup>*†</sup>	10 ± 3 <sup>#</sup>	0 ± 0
<b>Training Years (years)</b>	10 ± 7	11 ± 7	0 ± 0

BSA – body surface area, BP – blood pressure, HR – heart rate

\* P<0.05 HDHS versus controls; # P<0.05 LDHS versus controls and † P<0.05 HDHS versus LDHS.

**Table 2 – Left and Right Atrial Structure and Functional Volumes for HDHS Athletes, LDHS Athletes and Controls**

<b>Parameter</b>	<b>HDHS Athletes (mean±SD)</b>	<b>LDHS Athletes (mean±SD)</b>	<b>Controls (mean±SD)</b>
<b>LAD (mm/m<sup>2</sup>)</b>	20 ± 2*†	17 ± 2	17 ± 2
<b>LAVOLes (ml/m<sup>2</sup>)</b>	35 ± 8*†	26 ± 10	23 ± 5
<b>LAVOLpreA (ml/m<sup>2</sup>)</b>	21 ± 6*†	16 ± 6	13 ± 3
<b>LAVOLed (ml/m<sup>2</sup>)</b>	14 ± 4*†	10 ± 5	8 ± 2
<b>RAVOLes (ml/m<sup>2</sup>)</b>	37 ± 10*†	26 ± 9	23 ± 5
<b>RAVOLpreA (ml/m<sup>2</sup>)</b>	24 ± 9*†	18 ± 5	15 ± 4
<b>RAVOLed (ml/m<sup>2</sup>)</b>	18 ± 9*	13 ± 5	11 ± 3
<b>RAVOLes : LAVOLes ratio</b>	1.05 ± 0.26	1.12 ± 0.55	1.04 ± 0.28
<b>RAVOLpreA : LAVOLpreA ratio</b>	1.13 ± 0.31	1.41 ± 0.88	1.26 ± 0.36
<b>RAVOLed : LAVOLed ratio</b>	1.31 ± 0.48	1.67 ± 0.98	1.40 ± 0.39
<b>LARESvol (ml)</b>	35 ± 9*	25 ± 11	23 ± 7
<b>LACONvol (ml)</b>	44 ± 18*	41 ± 18	30 ± 11
<b>LABOOvol (ml)</b>	11 ± 7*	10 ± 7	9 ± 4
<b>RARESvol (ml)</b>	41 ± 11*†	34 ± 11	28 ± 7
<b>RACONvol (ml)</b>	38 ± 14*	32 ± 18	24 ± 11
<b>RABOOvol (ml)</b>	14 ± 6	12 ± 4	10 ± 3

LAD – left atrial diameter, LA(RA)VOLes – Left (right) atrial volume at end systole, LA(RA)VOLpreA – left (right) atrial volume at pre A, LA(RA)VOLed – left (right) atrial volume at end diastole, LA(RA)RESvol – left (right) atrial reservoir volume, LA(RA)CONvol – left (right) atrial conduit volume, LA(RA)BOOvol – left (right) atrial booster volume

\* P<0.05 HDHS versus controls; # P<0.05 LDHS versus controls and † P<0.05 HDHS versus LDHS.

**Table 3** – Left and right atrial strain ( $\epsilon$ ) for HDHS athletes, LDHS athletes and sedentary controls

<b>Parameter</b>	<b>HDHS Athletes (mean<math>\pm</math>SD)</b>	<b>LDHS Athletes (mean<math>\pm</math>SD)</b>	<b>Controls Athletes (mean<math>\pm</math>SD)</b>
<b>LARE<math>\epsilon</math> (%)</b>	36 $\pm$ 7	34 $\pm$ 7	32 $\pm$ 6
<b>LACON<math>\epsilon</math> (%)</b>	26 $\pm$ 6	24 $\pm$ 7	22 $\pm$ 6
<b>LABOO<math>\epsilon</math> (%)</b>	11 $\pm$ 3	13 $\pm$ 5	11 $\pm$ 4
<b>RARE<math>\epsilon</math> (%)</b>	33 $\pm$ 9	37 $\pm$ 10	32 $\pm$ 8
<b>RACON<math>\epsilon</math> (%)</b>	22 $\pm$ 8	22 $\pm$ 8	24 $\pm$ 9
<b>RABOO<math>\epsilon</math> (%)</b>	12 $\pm$ 6	13 $\pm$ 8	10 $\pm$ 5

LA(RA)RES  $\epsilon$  – left (right) atrial reservoir strain, LA(RA)CON  $\epsilon$ - left (right) atrial conduit strain,  
 LA(RA)BOO  $\epsilon$  – left (right) atrial booster strain



