**The Right Heart of the Elite Senior Rugby Football League Athlete**

Lynsey Forsythea, John Somaurooa, Keith Georgea, Michael Papadakisb, Benjamin Browna, Mohammad Qasema, and David Oxborougha

a Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, United Kingdom.

b Cardiovascular Sciences Research Centre, St Georges University of London, London, United Kingdom.

**Address for Correspondence:**

Dr David Oxborough,

Reader in Cardiovascular Physiology

Research Institute for Sport and Exercise Sciences

Tom Reilly Building

Liverpool John Moores University

Liverpool

L3 3AF

**Email:** d.l.oxborough@ljmu.ac.uk **Tel:** 44 151 904 623

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**Abstract**

**Background:** Right heart enlargement is common in the athletes’ heart (AH) phenotype however right ventricular (RV) enlargement is also one of the diagnostic criteria for Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC), a condition linked to sudden cardiac death (SCD). The primary aim of this study was to establish the normal RV phenotype in elite rugby football league (RFL) athletes using standard 2D echocardiography and myocardial mechanics. The secondary aim was to describe right atrial (RA) structure and function using 2D echocardiography.

**Methods:** 139 male RFL athletes underwent echocardiographic evaluation of the right heart including RV strain (ɛ) and strain rate (SR) imaging using speckle tracking echocardiography (STE). Non-athletic males were used for comparison and allometric scaling was utilised for conventional echocardiographic parameters.

**Results:** Scaled RV dimensions were larger in athletes (P<0.05) with the exception of the mid cavity. No differences (P>0.05) in RV fractional area change (FAC) and RV longitudinal ɛ were observed between groups. Tissue Doppler imaging (TDI) indexed parameters and global strain rate (SR) were lower (P<0.05) in athletes with significant correlations to RV dimensions and heart rate (HR). The RA was larger in athletes (P<0.001) with no functional difference (P>0.05) observed by volume assessment.

**Conclusions:** Reduction in SR and indexed TDI are partly associated with lower HR and increased RV chamber size and are likely to represent normal physiological adaptation in RFL athletes. RA enlargement appears proportional to RV enlargement. These data may aid interpretation of normal athletic adaptation during pre-participation screening of RFL athletes.

**Abstract Word count: 248**

**Keywords:** Athletes’ Heart, right ventricle, right atrium, echocardiography, strain

**Introduction**

Physiological cardiac adaptation that occurs in response to intense physical athletic training is known as the Athletes Heart (AH)1,2. Although the AH phenotype involves all cardiac chambers, the left ventricle (LV) has been the most extensively studied and reported in meta-analyses1,2 with the impact of remodelling on the right ventricle (RV) and right atrium (RA) having received less attention3,4,5,6,7,8. RV enlargement is a common phenotype in AH but is also one of the diagnostic criteria for Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)9, a condition linked to sudden cardiac death (SCD), thereby creating a diagnostic challenge. Current Task Force criteria for the diagnosis of ARVC have been developed10 where data was obtained from a patient population with established ARVC, however this criteria demonstrates poor specificity when applied to a lower risk population, such as athletes3.

Echocardiography is utilised in the assessment of the RV during pre-participation cardiac screening (PCS) and novel functional assessment techniques including strain (ɛ) and strain rate (SR) imaging can assist clinical differentiation between normal physiologic RV adaptation and inherited pathological conditions such as ARVC11,12. There is, however, conflicting data defining the magnitude of RVɛ values in athletes with some studies reporting reduced RVɛ13,14,15 whilst others have reported normal values16,17.

Only a few studies have investigated the RA phenotype in athletes5,6,7,8 with the consensus being an enlargement reflective of the physiological change in haemodynamic loading conditions5. Whilst RA enlargement is a recognised manifestation of the AH18 it also occurs in patients with increased filling pressures secondary to RV anomalies and cardiovascular disease19. Enlargement can also be associated with atrial arrhythmias20,21,22 and cardiomyopathy23 and therefore the ability to define normal RA physiology in the athletic population is clinically relevant.

Rugby Football League (RFL) is a high intensity, intermittent sport with moderate static (10-20%) and moderate dynamic (50-75%) components24. RFL athletes therefore provide an ideal model for right heart assessment. With recent high profile cases of SCD in RFL athletes and PCS being made mandatory for all male athletes competing in the professional RFL Super-League, it is pertinent to establish the nature of the AH phenotype in this specific group of athletes.

The primary aim of this study was to establish the RV phenotype in elite male RFL athletes using standard 2D, Doppler, tissue Doppler, ɛ and SR imaging. The secondary aim was to describe RA structure and function using 2D echocardiography.

**Methods**

**Study population and design**

Following approval from the Ethics Committee of Liverpool John Moores University, 139 elite senior RFL Super-league athletes aged 24±4 years (range 19-34) and 52 sedentary control subjects 22±3 years (range 20–35) provided written informed consent to participate in the study. Athlete data was collected as part of their mandatory annual PCS. All athletes participated in more than 10 hours structured exercise training per week and controls were healthy individuals who were not involved in structured sport related training, engaging in less than 3 hours recreational activity per week. After a detailed explanation of the testing protocol participants completed a medical questionnaire to document any cardiovascular symptoms, family history of SCD or other cardiovascular history. All participants abstained from exercise training or recreational activity for at least 6 hours prior to the investigation. They were allowed to take food and water *ad libitum* but were restricted from alcohol consumption 24 hours prior. A cross-sectional study was employed and data was acquired in a resting state at a single testing session. Screening results were reported by a sports cardiologist with clinical referrals made for any participant requiring further cardiac evaluation. On further evaluation no cardiac disease was present in any of the athletes or controls, allowing for all participants to be included in the study.

**Procedures**

**Anthropometry**

A routine standard anthropometric assessment included height (Seca 217, Hannover, Germany) and body mass (Seca supra 719, Hannover, Germany) measurements with body surface area (BSA) calculated as previously described25. Resting blood pressure (BP) was assessed with an automated sphygmomanometer (Dinamap 300, GE Medical systems, USA).

**Conventional 2D Echocardiography**

All echocardiographic images were acquired using a commercially available ultrasound system (Vivid Q, GE Medical, Horten, Norway) with a 1.5-4 MHz phased array transducer. Two experienced sonographers acquired the images with the participant lying in the left lateral decubitas position in adherence to American Society of Echocardiography (ASE) guidelines26. Images were stored as a raw digital imaging and communications in medicine (DICOM) format and exported to an offline workstation (Echopac, Version 110.0.2, GE Healthcare, Horten, Norway) for subsequent analysis. Data was analysed by a single experienced sonographer and standard 2D, Doppler and pulsed wave tissue Doppler (TDI) measurements of chamber structure and function were made in accordance with ASE guidelines21,26.

**Right Ventricle**

The RV outflow tract (RVOT) was measured at three locations (RVOTplax, RVOT1 and RVOT2) in the parasternal orientation. The RV inflow was measured from a modified (RV focused) apical four chamber orientation at end diastole at basal (RVD1) and mid (RVD2) levels and RV length (RVD3) measured from apex to base at the level of the tricuspid annulus (Figure 1). The RVOT1:RVD1 ratio was calculated to establish the relative outflow and inflow dimensions. RV diastolic area (RVDa) and RV systolic area (RVSa) were measured from the same orientation and fractional area change calculated (RVFAC). RV free wall thickness was measured from the subcostal image with write-zoom applied to the RV mid wall taking care to avoid papillary muscle and trabeculation. Tricuspid annular plane systolic excursion (TAPSE) was measured and TDI allowed measurement of peak RV lateral systolic (RVS’), and early (RVE’) and late (RVA’) diastolic myocardial velocities. Peak RV TDI data was indexed for RV length by dividing by RVD3 to provide TDI index (RVS’index, RVE’index and RVA’index) as per recommendations for the LV27. The RV:LV ratio was subsequently determined from the measurement of the diameter of the base of both ventricles at end diastole from a standard apical 4 chamber image.

**Insert Figure 1 (a-d)**

**Right Atrium**

RA area (RAa) and volume (RAVOL) was measured from the apical 4 chamber image by tracing the RA and by method of discs respectively21. Volumes were calculated at end ventricular systole (RAVOLes), pre atrial contraction (RAVOLpreA) and end ventricular diastole (RAVOLed). These static volumes allowed the derivation of RA functional data and were assessed to provide reservoir (RAVOLres), conduit (RAVOLcon) and booster (RAVOLboo) volumes. RAVOLres was calculated as the difference between RAVOLes and RAVOLed. RAVOLcon was determined by difference between LV stroke volume (as this should equal RV stroke volume) (from Simpsons Biplane method) and RAVOLres. RAVOLboo was determined by difference between RAVOLpreA and RAVOLed8. The conduit to booster ratio (con:boo) was derived as a measure of relative contributions to diastolic filling.

All structural indices were scaled allometrically to BSA based on the principle of geometric similarity28. Linear dimensions were scaled to BSA0.5, areas directly to BSA and volumes to BSA1.5.

**Speckle Tracking Echocardiography (STE)**

Images for the assessment of myocardial ɛ and SR were acquired at frame rates between 40 and 90 frames per second with settings adjusted to provide optimal endocardial delineation. ɛ and SR were analysed by STE using an offline software package (Echopac, Version 110.0.2, GE Healthcare, Horten, Norway).

The assessment of global and regional longitudinal ɛ and SR was achieved using the RV focused, modified apical four chamber image to track the lateral wall only. The region of interest (ROI) was placed from basal to apical wall encompassing three regional segments basal, mid and apical. RVɛ, time to peak RVɛ, systolic SR (RVSRS), early diastolic SR (RVSRE) and late diastolic SR (RVSRA) were assessed and an average was calculated to provide peak global longitudinal values29. Base to apex ɛ gradient was calculated as the percentage difference between apical and basal ɛ. A previous study from our laboratory demonstrated excellent intra-observer reproducibility for RV ɛ with an intra-class correlation coefficient (ICC) of 0.834 and coefficient of variation (CoV) of 7%. RV SR was more variable but with an acceptable ICC of >0.6 and CoV of <15%30.

**Statistical Analysis**

Study data were collected and managed using REDCAP electronic data capture tools hosted at Liverpool John Moores University31. All echocardiographic data were presented as mean ± SD and ranges. Statistical analyses were performed using the commercially available software package SPSS (SPSS, Version 23.0 for Windows, Illinois, USA). Variables were analysed between athletes and controls using independent T-tests with P<0.05 was considered statistically significant.

Where group differences were found for RV functional parameters, a bivariate Pearson’s correlation was performed to establish the association to appropriate structural measures and heart rate (HR). A P<0.05 was considered statistically significant. When multiple significant correlations were found with ɛ and SR multi–linear regression was undertaken to determine the relative contribution of each parameter on the dependent variable.

**Results**

Athletes were slightly older than controls (P=0.001) but within the same age range. Height, weight and BSA were all greater (P<0.001) whilst HR was lower (P<0.001) in the athlete group. There was no difference in systolic BP (P=0.413) but diastolic BP was lower in athletes (P<0.001) (Table 1).

**Insert Table 1**

**Right Ventricular Structure and Function**

RV standard structural and functional indices are presented in Table 2. All absolute measures of RV size including RVWT and the RV:LV ratio were larger (P<0.01) in the athlete compared to the control group. All parameters remained statistically significant following allometric scaling with exception of RVD2.  88% of athletes and 38% of controls met RVOTplax dimension criteria for ARVC10.78% of athletes and 29% of controls met RVOT1 dimension criteria for ARVC10. None of the controls met major ARVC criteria for RVOT1 compared to 46% of athletes (Figure 2). The RVOT1:RVD1 ratio was increased in athletes (P=0.012). TAPSE and RVFAC were not significantly different between groups. Absolute RV systolic and diastolic TDI values - RVS’, RVE’, RVA’ and RV E’/A’ ratio were not different between groups however the associated indexed values for RVS’, RVE’ and RVA’ were lower in the athlete group (P=0.002, <0.001 and 0.015 respectively).

**Insert Table 2 and Figure 2**

Global RV longitudinal ɛ, although lower in athletes, was not statistically significant between groups (Table 3). Time to peak ɛ was higher (P<0.001) in the athlete group whilst RVSRS, RVSRE and RVSRA were all lower (P<0.001) in the athlete compared to the control group.

**Insert Table 3**

There were no significant differences between groups for RV regional longitudinal ɛ (Table 3) and both groups exhibited similar apex to base ɛ gradients (-5%, P=0.743). In the athlete group, all 3 RV wall segments demonstrated lower RVSRS (basal P<0.001, mid P=0.004 and apical P<0.001) and RVSRA (Basal P=0.001, mid P=0.007 and apical P=0.003). Basal (P=0.001) and mid segments also demonstrated lower RVSRE (P<0.001) (Table 3).

There were significant correlations between SR and HR, RVD1 and RVOT1 and between TDI index and RVD1, RVD3 and HR (with exception of RVE’ index) across both groups (Table 4). Following multi-linear regression, HR (β= -0.006, P<0.001) **and** RVD1 (β= 0.008, P=0.014) accounted for 15% of the variation in RVSRS. HR (β= -0.013, P<0.001) **and** RVD1 (β= 0.016, P=0.030) accounted for 14% of the variation in RVSRE. HR (β= -0.018, P<0.001) **and** RVOT1 (β= - 0.014, P=0.012) accounted for 19% of the variation in RVSRA.

**Insert Table 4**

**RA Structure and functional volumes**

Absolute RAa, RAVOLes, RAVOLpreA and RAVOLed and their respective indexed values were larger in the athlete group compared to controls (P<0.001). RAVOLres, RAVOLcon and RAVOLboo were larger in the athlete group (P<0.001) (Table 5) however con:boo was not different between groups (P=0.557).

**Insert Table 5**

**Discussion**

The main findings of this study are 1) absolute measures for RV chamber size and wall thickness are greater in RFL athletes compared to sedentary controls. This finding remains following allometric scaling with the exception of RVD2. There are no differences in the functional parameters RVFAC and RV longitudinal ɛ between groups but TDI index and SR are lower in athletes, which are, in part, associated with lower HR and increased RV chamber size, and 2) all absolute and indexed structural RA parameters are greater in athletes. Whilst functional RA volumes are increased in athletes there is no difference in the relative contribution to diastolic filling.

**RV Structure**

Larger RV cavities in endurance athletes have been previously demonstrated with increases in both inflow and outflow dimensions6,16 however, there are few studies that have assessed the RV in resistance athletes or those involved in mixed training2. A 6 month resistance exercise training study demonstrated no increase in RV cavity dimensions32 and in a study by D’Andrea *et al* (2013)6 RV chamber size in resistance athletes were similar to sedentary controls. The results of the current study in athletes with mixed endurance and resistance components would suggest that RV structure in the RFL athlete is more akin to that of the endurance athlete with an observed increased RV inflow and outflow dimensions and an increased RV:LV ratio compared to controls. Unequal remodelling and increased RV:LV ratio has been reported previously in endurance athletes16,33,34 attributable to disproportionate loading on the RV during exercise34. The increased RVOT1:RVD1 ratio in athletes suggests a lack of proportional enlargement of RV outflow and inflow as RVOT1 appears to dilate to a greater extent. Differentiation of physiological RV enlargement from ARVC in RFL athletes is pertinent given that 88% and 78% of these meet ARVC criteria10 at RVOTplax and RVOT1 respectively.

Little attention has been paid to appropriate scaling of RV structural parameters but it is likely to aid interpretation of the RV in AH17. With appropriate scaling for body size in this study all structural parameters were significantly greater in athletes compared to controls, with the exception of RVD2. This would suggest that body size alone does not account for the enlarged RV in a RFL athlete.

**RV Function**

In addition to structural assessment, functional assessment is key when attempting to differentiate physiological RV remodelling from ARVC10. It is considered best practice to apply a multifactorial approach to functional assessment including the use of TAPSE, RVFAC and RV TDI35. The current study reports no difference in TAPSE, RVFAC or standard indices of TDI between RFL athletes and controls and therefore the presence of abnormal values should prompt further investigation.

ɛ imaging is advocated in the assessment of RV function21 and it has been reported that STE ɛ parameters are superior to conventional echocardiographic parameters in aiding the identification of ARVC11. No difference in longitudinal global RVɛ was noted between RFL athletes and controls, providing additional support that a reduction in function is not a normal physiological response in RFL athletes. Lower global RVɛ values have been previously reported in elite endurance athletes due to reduction in basal function13, a finding that was reproduced in a subsequent study but with the additional finding of increased function in the apical segment ɛ14. In the current study there was no difference in regional RVɛ between groups and both RFL athletes and controls exhibit an RVɛ gradient of 5% from base to apex, suggesting a normal pattern of deformation even with increased RV size in RFL athletes. Other recent studies reported no difference in resting ɛ parameters16 and no differences in global or regional RV deformation in athletes compared to controls17. Similarly a study involving both endurance and resistance athletes found few meaningful differences in deformation parameters of the right heart irrespective of sporting discipline, training volume and physiological remodelling7.

SR and TDI index are largely related to HR and RV dimensions. TDI index is reduced in athletes and despite regional SR showing similar distribution in both RFL athletes and controls, both global and regional SR is lower in athletes. In an endurance training study by Teske *et al.* (2009)13 SR values were found to be reduced in basal and mid segments in athletes with marked RV dilatation, whereas athletes without RV dilatation showed no significant difference compared to controls. It was reported that this should be interpreted as a normal finding when evaluating athletes suspected for RV pathology13. Lower SR in athletes in the current study is also likely to represent normal physiological adaptation to training in the RFL athlete given that ɛ, TDI, RVFAC and TAPSE were not different compared to controls. It has been previously reported that during brief maximal exercise the RV has the capacity to increase contractility to compensate for disproportionate increases in work34 and it is reasonable to speculate that reduced SR (aligned to chamber size and HR) may be an adaptation of myocardial contractility to support contractile reserve during exercise. The increased size of the RV would suggest an increased RV mass and number of myofibrils and it is plausible that a greater number of myofibrils36 may reach the same required deformation at a slower rate, or in other words, a similar wall tension and intraventricular pressure can be generated or released at a slower speed. An increase in RV free wall thickness and a reduced contractile stress may result in the same contractile force.

**RA Structure and Function**

Increased RA area, volume and indexed volume has been reported in athletes with changes in the RA proportional to those of the RV5. McClean et al (2015)8 reported that RA size is consistently larger throughout the cardiac cycle, in athletes with high dynamic training. These data of both studies are supported by the current study. The RA assists RV filling by 1) acting as a reservoir for venous return, 2) acting as a passive conduit in early diastole and 3) acting as an active conduit (booster) in late diastole during atrial contraction21. During all 3 phases of RV filling, functional volumes, RAVOLres, RAVOLcon and RAVOLboo were greater in RFL athletes. This does not infer a functional RA improvement in RFL athletes as no difference in the con:boo volume ratio was found between groups. Although atrial enlargement appears to be a normal physiological response to dynamic training there is increasing evidence of an association between an AH phenotype and autonomic alterations with atrial arrhythmia22. As mechanisms of atrial arrhythmia in the athlete are not clearly understood22,37, the RA is likely to receive more attention in the future.

**Limitations**

This is a cross sectional study and hence the timing and development of exercise induced structural and functional adaptation cannot be determined. The athletes were selected according to sporting discipline and these findings may not therefore be representative of all athletes. Genetic factors and seasonal variation should also be considered during cardiac evaluation.

**Conclusions**

This study provides a novel and comprehensive assessment of the right heart in the RFL athlete. RV dimensions are larger in athletes independent of body size, whilst reduced SR and indexed TDI is likely a normal physiological phenomenon in the elite RFL athletes. Despite RA enlargement in RFL athletes we cannot infer a functional RA/RV improvement compared to controls. These data may be used to aid differentiation between physiology and pathology during PCS of these athletes.

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**Table Legends**

**Table 1:** Demographics

**Table 2:** Echocardiographic parameters of the right ventricle

**Table 3:** Global and regional right ventricular ɛ and SR

**Table 4:** Right ventricular bivariate correlation

**Table 5:** Echocardiographic parameters of the right atrium

**Figure 1:** Measurement of RV structural dimensions. a, RVOTplax; b, RVOT1; c, RVOT2; d, RVD1, RVD2 and RVD3

**Figure 2:** Percentage of athletes and controls meeting minor and major criteria for ARVC according to Marcus *et al*10

|  |  |  |
| --- | --- | --- |
|  | AthleteMean±SD (Range) | ControlMean±SD (Range) |
| Age (Years) | 24±4\*\*(19-34) | 22±3 (20-35) |
| Height (m) | 1.82±0.06\*\*\*(1.62-1.98) | 1.78±0.06 (1.65-1.91) |
| Weight (Kg) | 96±11\*\*\*(75-132) | 78±9 (60-107) |
| BSA (m2) | 2.20±0.15\*\*\*(1.91-2.66) | 1.96±0.13 (1.66-2.38) |
| HR (Beats.min-1) | 56±10 (39-83) | 69±9\*\*\*(50-95) |
| Systolic BP | 131±9(107-155) | 129±10(113-151) |
| Diastolic BP | 69±7(53-89) | 74±7\*\*\*(63-90) |

**Table 1: Demographics**

\*P<0.05, \*\*P<0.01, \*\*\*P<0.001

BSA, Body Surface Area; HR, Heart Rate; BP, Blood Pressure

**Table 2: Echocardiographic parameters of the right ventricle**

|  |  |  |
| --- | --- | --- |
|  | AthleteMean±SD (Range) | ControlMean±SD (Range) |
| RVOTPLAX (mm) | 34±4\*\*\*(21-47) | 28±4 (20-36) |
| RVOT1 (mm) | 36±5\*\*\* (22-49) | 29±4 (19-35) |
| RVOT2 (mm) | 27±3\*\*\* (19-35) | 23±2 (18-28) |
| RVD1 (mm) | 44±5\*\*\* (33-60) | 39±4 (31-47) |
| RVD2 (mm) | 33±4\*\*\*(22-44) | 30±5 (17-42) |
| RVD3 (mm) | 91±8\*\*\*(72-111) | 82±7 (71-98) |
| RVDa (cm2) | 30±4\*\*\* (21-41) | 22±3 (15-29) |
| RVSa (cm2) | 16±3\*\*\* (10-23) | 12±2 (6-18) |
| RV Free Wall Thickness (mm) | 4±1\*\*\* (2-7) | 4±1 (3-5) |
| TAPSE (mm) | 24±4 (16-33) | 23±3 (17-32) |
| RVOT1:RVD1 Ratio | 0.81±0.14\*(0.52-1.23) | 0.76±0.11(0.44-0.97) |
| RV:LV Ratio | 0.91±0.10\*\*\* (0.70-1.20) | 0.82±0.07 (0.66-1.01) |
| RVFAC (%) | 46±6 (34-61) | 47±7 (38-64) |
| RVOTPLAX (mm/(m2)0.5)) | 23±3\*\*\* (15-30) | 20±2 (15-25) |
| RVOT1 (mm/(m2)0.5)) | 24±3\*\*\* (15-32) | 21±3 (14-26) |
| RVOT2 (mm/(m2)0.5)) | 18±2\*\*\* (13-24) | 17±2 (13-20) |
| RVD1 (mm/(m2)0.5)) | 30±3\*\*\* (22-38) | 28±2 (23-34) |
| RVD2 (mm/(m2)0.5)) | 22±3 (15-30) | 21±3 (12-29) |
| RVD3 (mm/(m2)0.5)) | 61±5\* (47-75) | 59±5 (49-70) |
| RVDa Index (cm2/m2) | 14±2\*\*\* (9-18) | 11±2 (7-15) |
| RVSa Index (cm2/m2) | 7±1\*\*\*(4-11) | 6±1 (3-8) |
| RVS’ (cm/s) | 15±2 (6-23) | 14±2 (10-18) |
| RVE’ (cm/s) | 15±3 (7-24) | 15±3 (9-21) |
| RVA’ (cm/s) | 10±3 (5-17) | 11±3 (6-18) |
| RV E’/A’ (cm/s) | 1.54±0.44 (0.71-3.00) | 1.56±0.49 (0.81-2.71) |
| RVS’ index ((cm/s)/cm) | 1.61±0.29 (0.61-2.44) | 1.75±0.26\*\* (1.06-2.34) |
| RVE’ index ((cm/s)/cm) | 1.65±0.32 (0.84-2.76) | 1.89±0.39\*\*\* (0.98-2.76) |
| RVA’ index ((cm/s)/cm) | 1.15±0.33 (0.56-2.05) | 1.28±0.34\* (0.74-2.05) |

\*P<0.05, \*\*P<0.01, \*\*\*P<0.001

RVOTPLAX**,** Right ventricular outflow tract at parasternal long axis; RVOT1, Right ventricular outflow tract (proximal);RVOT2,Right ventricular outflow tract (distal); RVD1, Right ventricular dimension (basal); RVD2, Right ventricular dimension (mid); RVD3, Right ventricular length; RVDa, Right ventricular diastolic area; RVSa, Right ventricular systolic area; TAPSE, Tricuspid annular plane systolic excursion; RV:LV ratio, Right ventricular to left ventricular ratio; RVFAC, Right ventricular fractional area change; RVS’, RV TDI lateral systolic myocardial velocity; RVE’, RV TDI early diastolic myocardial velocity; RVA’, RV TDI late diastolic myocardial velocity.

**Table 3: Global and regional right ventricular ɛ and SR**

|  |  |  |
| --- | --- | --- |
|  | Athletemean ± SD(Range) | Controlmean ± SD(Range) |
| Global RVɛ (%) | -27.2±3.4 (-18.4- -40.7) | -28.4±4.2 (-19.1- -41.2) |
| Time to Peak RV ɛ (s) | 0.38±0.03\*\*\* (0.31-0.46) | 0.36±0.03 (0.31-0.44) |
| RVSRS (s-1) | -1.32±0.22 (-0.77- -2.19) | -1.48±0.28\*\*\* (-0.97- -2.34) |
| RVSRE (s-1) | 1.59±0.33 (0.79-2.67) | 1.92±0.50\*\*\* (1.11-3.26) |
| RVSRA (s-1) | 0.89±0.27 (0.34-1.77) | 1.09±0.28\*\*\* (0.39-1.85) |
| Basal RVɛ (%) | -24.9±5.4 | -26.3±4.9 |
| Mid RVɛ (%) | -27.2±4.1 | -28.4±4.8 |
| Apical RVɛ (%) | -30.0±4.3 | -31.1±4.6 |
| Apex to Base RVɛ gradient (%) | -5.2±6.9 | -4.8±5.0 |
| Basal RVSRS (s-1) | -1.50±0.41 | -1.74±0.41\*\*\* |
| Mid RVSRS (s-1) | -1.37±0.27 | -1.50±0.31\*\* |
| Apical RVSRS ( s-1) | -1.58±0.31 | -1.86±0.43\*\*\* |
| Basal RVSRE (s-1) | 2.10±0.67 | 2.50±0.84\*\* |
| Mid RVSRE (s-1) | 1.68±0.40 | 2.02±0.58\*\*\* |
| Apical RVSRE (s-1) | 2.07±0.55 | 2.22±0.54 |
| Basal RVSRA (s-1) | 1.06±0.34 | 1.26±0.38\*\*\* |
| Mid RVSRA (s-1) | 0.98±0.33 | 1.13±0.37\*\* |
| Apical RVSRA (s-1) | 1.25±0.40 | 1.46±0.44\*\* |

\*P<0.05, \*\*P<0.01, \*\*\*P<0.001

RVɛ, Right ventricular longitudinal strain; RVSRS, Right ventricular systolic strain rate, RVSRE, Right ventricular early diastolic strain rate; RVSRA, Right ventricular late diastolic strain rate.

**Table 4: Right ventricular bivariate correlation**

|  |  |  |
| --- | --- | --- |
| Functional Parameter | Parameters correlated | R value |
| RV Time to Peak ɛ | HRRVOT1RVD1RVD3 | -0.583\*\*\*0.278\*\*\*0.303\*\*\*0.235\*\* |
| RVSRS | HRRVOT1RVD1RVD3 | -0.347\*\*\*0.220\*\*0.279\*\*\*0.256\*\*\* |
| RVSRE | HRRVOT1RVD1RVD3 | 0.203\*\*-0.233\*\*-0.332\*\*\*-0.330\*\*\* |
| RVSRA | HRRVOT1RVD1RVD3 | 0.193\*\*-0.244\*\*-0.172\*-0.178\* |
| RVS’ index | HRRVOT1RVD1RVD3 | 0.159\*-0.013-0.212\*\*-0.538\*\*\* |
| RVE’ index | HRRVOT1RVD1RVD3 | 0.112-0.137-0.163\*-0.366\*\*\* |
| RVA’ index | HRRVOT1RVD1RVD3 | 0.227\*\*-0.113-0.160\*-0.256\*\*\* |

\*P<0.05, \*\*P<0.01, \*\*\*P<0.001

ɛ, Strain; HR, Heart Rate; RVOT1, Right ventricular outflow tract (proximal); RVD1**,** Right ventricular dimension (basal); RVD3, Right ventricular length; RVSRS, Right ventricular systolic strain rate, RVSRE, Right ventricular early diastolic strain rate; RVSRA, Right ventricular late diastolic strain rate; RVS’, RV TDI lateral systolic myocardial velocity; RVE’, RV TDI early diastolic myocardial velocity; RVA’, RV TDI late diastolic myocardial velocity.

**Table 5: Echocardiographic parameters of the Right Atrium**

|  |  |  |
| --- | --- | --- |
|  | AthleteMean±SD (Range) | ControlMean±SD (Range) |
| RAa (cm2) | 22±4\*\*\* (13-29) | 15±2 (10-20) |
| RAa Index (cm2/m2) | 10±1\*\*\* (6-13) | 8±1 (5-10) |
| RAVOLes (ml) | 73±18\*\*\* (33-121) | 44±10 (25-63) |
| RAVOLes Index (ml/(m2)1.5)) | 22±5\*\*\* (11-35) | 16±4 (10-24) |
| RAVOLpreA (ml) | 49±13\*\*\* (25-92) | 28±7 (14-45) |
| RAVOLpreA Index (ml/(m2)1.5)) | 15±4\*\*\* (6-27) | 10±2 (6-16) |
| RAVOLed (ml) | 35±10\*\*\* (15-75) | 18±5 (7-32) |
| RAVOLed Index (ml/(m2)1.5)) | 10±3\*\*\* (5-22) | 6±2 (3-10) |
| RAVOLres (ml) | 39±11\*\*\* (13-77) | 26±7 (13-44) |
| RAVOLcon (ml) | 53±17\*\*\* (5-96) | 36±12 (12-65) |
| RAVOLboo (ml) | 14±5\*\*\* (5-30) | 10±3 (4-19) |
| Con : Boo Ratio | 4.32±2.65(0.17-16.2) | 4.07±2.27(1.2-12.75) |

\*P<0.05, \*\*P<0.01, \*\*\*P<0.001

RAa, Right atrial area, RAVOLes, Right atrial end systolic volume; RAVOLpreA, Right atrial volume pre-atrial contraction; RAVOLed, Right atrial end diastolic volume; RAVOLres, Right atrial reservoir volume; RAVOLcon, Right atrial conduit volume; RAVOLboo, Right atrial booster volume; Con:Boo ratio, Conduit to booster volume ratio.