

The Right Heart of the Elite Senior Rugby Football League Athlete

Lynsey Forsythe^a, John Somauroo^a, Keith George^a, Michael Papadakis^b, Benjamin Brown^a,
Mohammad Qasem^a, and David Oxborough^a

^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

Abstract

Background: Right heart enlargement is common in the athletes' heart phenotype however few data exist regarding interpretation of normal athletic adaptation during Pre-participation Cardiac Screening (PCS) of Rugby Football League (RFL) athletes. Echocardiography is utilised during PCS and thus the primary aim of this study was to establish the normal right ventricular (RV) phenotype in elite 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 (ϵ) and strain rate (SR) imaging using speckle tracking echocardiography (STE). Non-athletic males were used for comparison and allometric scaling was applied 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 ϵ were observed between groups. Tissue Doppler imaging (TDI) indexed parameters and global strain rate (SR) were lower ($P<0.05$) in athletes with HR and weight found to have covariance with SR. 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 body mass 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 PCS of RFL athletes.

Keywords: Athletes' heart, right heart, 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-analyses^{1,2} with the impact of remodelling on the right ventricle (RV) and right atrium (RA) having received less attention^{3,4,5,6,7,8}. RV enlargement is a common phenotype in AH but is also one of the diagnostic criteria for Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) a condition linked to sudden cardiac death (SCD)^{9,10}. Echocardiography is utilised in the assessment of the RV during athletic pre-participation cardiac screening (PCS) but this can be challenging in the athlete due to the potential for physiological and pathological overlap. There are currently no comprehensive, sport-specific, echocardiographic data regarding the interpretation of normal RV athletic adaptation compared to non-athletic controls.

Novel, functional, echocardiographic techniques including strain (ϵ) and strain rate (SR) imaging can assist clinical differentiation between normal physiologic RV adaptation and inherited pathological conditions such as ARVC^{11,12}. There is, however, conflicting data defining the magnitude of RV ϵ values in athletes with some studies reporting reduced RV ϵ ^{13,14,15} whilst others have reported normal values^{16,17}. Normative, sport-specific RV ϵ and SR data alongside conventional 2D echocardiographic parameters may improve the utility of echocardiography in PCS.

Only a few studies have investigated the RA phenotype in athletes^{5,6,7,8} 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 arrhythmias^{20,21,22} and cardiomyopathy²³ and therefore the ability to define normal RA physiology in the athletic population is also clinically relevant.

Rugby Football League (RFL) is a high intensity, intermittent sport with moderate static (10-20%) and moderate dynamic (50-75%) components²⁴. 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 normative echocardiographic parameters of the right heart in this specific group of athletes. This may lead to increased sensitivity, specificity and diagnostic capability of echocardiography during PCS of RFL athletes.

The primary aim of this study was to establish the RV phenotype in elite male RFL athletes using standard 2D, Doppler, tissue Doppler, ϵ 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 decubitus 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 guidelines^{21,26}.

Right Ventricle

The RV outflow tract (RVOT) was measured at three locations ($RVOT_{\text{plax}}$, $RVOT_1$ and $RVOT_2$) in the parasternal orientation. The RV inflow was measured from a modified (RV focused) apical four chamber orientation at end diastole at basal (RVD_1) and mid (RVD_2) levels and RV length (RVD_3) measured from apex to base at the level of the tricuspid annulus (Figure 1). The $RVOT_1:RVD_1$ 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 RVD_3 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

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 $BSA^{0.5}$, areas directly to BSA and volumes to $BSA^{1.5}$.

Speckle Tracking Echocardiography (STE)

Images for the assessment of myocardial ϵ and SR were acquired at frame rates between 40 and 90 frames per second with settings adjusted to provide optimal endocardial delineation. ϵ 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 ϵ 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. $RV\epsilon$, time to peak $RV\epsilon$, 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 ϵ gradient was calculated as the

percentage difference between apical and basal ϵ . A previous study from our laboratory demonstrated excellent intra-observer reproducibility for RV ϵ 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.

Standardisation of STE Data

The data was processed using cubic spline interpolation. All raw data was exported to allow a standardised, temporal assessment throughout the cardiac cycle. To account for inter or intra individual differences in heart rate, the data were normalised to the percentage of systolic and diastolic duration. RV ϵ and SR data were defined by 5% increments across systole and diastole and the peak value was reported.

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 \pm SD and ranges. Statistical analyses were performed using the commercially available software package SPSS (SPSS, Version 23.0 for Windows, Illinois, USA). The Kolmogorov-Smirnov test highlighted normal distribution across all parameters and therefore variables were analysed between athletes and controls using independent T-tests with $P < 0.05$ considered statistically significant. Where parameters met statistical significance an ANCOVA was performed with subsequent Bonferonni post-hoc tests to establish the impact of any co-variance from Age, HR, height and weight on functional RV STE parameters. A $P < 0.05$ was considered statistically significant.

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 RVD_2 . 88% of athletes and 38% of controls met $RVOT_{plax}$ dimension criteria for ARVC¹⁰. 78% of athletes and 29% of controls met $RVOT_1$ dimension criteria for ARVC¹⁰. None of the controls met major ARVC criteria for $RVOT_1$ compared to 46% of athletes (Figure 2). The $RVOT_1:RVD_1$ 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 ϵ , although lower in athletes, was not statistically significant between groups (Table 3, Figure 3). Time to peak ϵ 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 (Figure 3).

Insert Table 3 and Figure 3

There were no significant differences between groups for RV regional longitudinal ϵ (Table 3) and both groups exhibited similar apex to base ϵ 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).

T-tests showed significant differences only in SR between groups. Following ANCOVA age was not found to alter statistical significance within SR between groups. HR did alter statistical significance and had an effect particularly on the S and A waves rendering the changes non-significant (Global RV SRS became non-significant ($P = 0.061$), basal and mid RV SRS and basal, mid and apical RV SRA ($P > 0.05$)). It is important to note that Eta squared i.e. estimated effect size were all less than 1-2% contribution. Height had no impact but weight altered significance for some regional parameters, rendering changes non-significant (mid RV SRS, basal and apical RV SRE, mid and apical RV SRA ($P > 0.05$)).

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 4) however con:boo was not different between groups ($P = 0.557$).

Insert Table 4

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 RVD_2 . There are no differences in the functional parameters RVFAC and RV longitudinal ϵ 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 dimensions^{6,16} 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 athletes^{16,33,34} 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 RVOT_{plax} 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.

ϵ imaging is advocated in the assessment of RV function²¹ and it has been reported that STE ϵ parameters are superior to conventional echocardiographic parameters in aiding the identification of ARVC¹¹. No difference in longitudinal global RV ϵ 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 $RV\epsilon$ 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 ϵ ¹⁴. In the current study there was no difference in regional $RV\epsilon$ between groups and both RFL athletes and controls exhibit an $RV\epsilon$ 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 ϵ 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 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. HR has been identified as a co-variance for global and regional SR parameters, predominantly RV SRS and RV SRA. These effects however are small as the estimated effect size were all less than 1-2%. Weight was also found to be a co-variance for some regional RV SR parameters. An increased body mass in athletes was expected and likely attributable to an increased fat free mass leading to increases in RV chamber size. Similarly, Forsythe *et al* (2018)³⁶ reported a correlation between LV size and SR, where SR was also found to be lower in RFL 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 ϵ , 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 myofibrils³⁷ 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 understood^{22,38}, the RA is likely to receive more attention in the future.

Limitations

There is a significant difference in age between athletes and controls in this study however all athletes fit within an age decile that we utilise for normative echocardiographic parameters. It is expected to observe differences in height and weight between elite athletes and non-athletic controls and hence we have allometrically scaled all structural indices and performed ANCOVA to establish any effect on STE data. By inference an athlete will have a slower HR and thus we have provided temporally indexed peak strain data. The athletes were selected according to sporting discipline and these findings may not therefore be representative of all athletes. The athletes were male and data cannot be applied to female athletes. As a cross sectional study, the timing and development of exercise induced structural and functional adaptation cannot be determined and it is possible that seasonal variation in training may also affect 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. Lower SR in RFL athletes is in a small part related to HR and body size but these cannot fully explain the variance, particularly in the global SR parameters. 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.

Acknowledgements

This research received funding from the charitable organisation Cardiac Risk in the Young (CRY).

Conflict of Interest

The authors report no relationships that could be construed as a conflict of interest.

References

1. Pluim BM, Zwinderman AH, van der Laarse A, et al. The Athlete's Heart : A Meta-Analysis of Cardiac Structure and Function. *Circulation* 2000; 101: 336–344.
2. Utomi V, Oxborough D, Whyte GP, et al. Systematic review and meta-analysis of training mode, imaging modality and body size influences on the morphology and function of the male athlete's heart. *Heart* 2013; 99: 1727–1733.
3. Zaidi A, Ghani S, Sharma R, et al. Physiological right ventricular adaptation in elite athletes of African and Afro-Caribbean origin. *Circulation* 2013; 127: 1783–1792.
4. D'Ascenzi F, Pisicchio C, Caselli S, et al. RV Remodeling in Olympic Athletes. *JACC Cardiovasc Imaging* 2017; 10: 385–393.
5. D'Ascenzi F, Cameli M, Padeletti M, et al. Characterization of right atrial function and dimension in top-level athletes: A speckle tracking study. *Int J Cardiovasc Imaging* 2013; 29: 87–94.
6. D'Andrea A, Riegler L, Golia E, et al. Range of right heart measurements in top-level athletes: The training impact. *Int J Cardiol* 2013; 164: 48–57.
7. Pagourelias ED, Kouidi E, Efthimiadis GK, et al. Right atrial and ventricular adaptations to training in male caucasian athletes: An echocardiographic study. *J Am Soc Echocardiogr* 2013; 26: 1344–1352.
8. McClean G, George K, Lord R, et al. Chronic adaptation of atrial structure and function in elite male athletes. *Eur Heart J Cardiovasc Imaging* 2015; 16: 417–422.
9. D'Ascenzi F, Pelliccia A, Solari M, et al. Normative Reference Values of Right Heart in Competitive Athletes: A Systematic Review and Meta-Analysis. *J Am Soc*

Echocardiogr 2017; 30: 845–858.

10. Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/Dysplasia: Proposed modification of the task force criteria. *Circulation* 2010; 121: 1533–1541.
11. Teske AJ, Cox MG, De Boeck BW, et al. Echocardiographic Tissue Deformation Imaging Quantifies Abnormal Regional Right Ventricular Function in Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy. *J Am Soc Echocardiogr* 2009; 22: 920–927.
12. D’Ascenzi F, Caselli S, Solari M, et al. Novel echocardiographic techniques for the evaluation of athletes’ heart: A focus on speckle-tracking echocardiography. *Eur J Prev Cardiol* 2016; 23: 437–446.
13. Teske AJ, Prakken NH, De Boeck BW, et al. Echocardiographic tissue deformation imaging of right ventricular systolic function in endurance athletes. *Eur Heart J* 2009; 30: 969–977.
14. La Gerche A, Burns AT, D’Hooge J, et al. Exercise strain rate imaging demonstrates normal right ventricular contractile reserve and clarifies ambiguous resting measures in endurance athletes. *J Am Soc Echocardiogr* 2012; 25: 253–262.
15. King G, Almuntaser I, Murphy RT, et al. Reduced right ventricular myocardial strain in the elite athlete may not be a consequence of myocardial damage. “cream masquerades as skimmed milk.” *Echocardiography* 2013; 30: 929–935.
16. Oxborough D, Sharma S, Shave R, et al. The right ventricle of the endurance athlete: The relationship between morphology and deformation. *J Am Soc Echocardiogr* 2012; 25: 263–271.

17. Utomi V, Oxborough D, Ashley E, et al. The impact of chronic endurance and resistance training upon the right ventricular phenotype in male athletes. *Eur J Appl Physiol* 2015; 115: 1673-1682.
18. Baggish AL and Wood MJ. Athlete's heart and cardiovascular care of the athlete: Scientific and clinical update. *Circulation* 2011; 123: 2723–2735.
19. Roca GQ, Campbell P, Claggett B, et al. Right Atrial Function in Pulmonary Arterial Hypertension. *Circ Cardiovasc Imaging* 2015; 8: e003521.
20. Müller H, Burri H, Lerch R. Evaluation of right atrial size in patients with atrial arrhythmias: Comparison of 2D versus real time 3D echocardiography. *Echocardiography* 2008; 25: 617–623.
21. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2010; 23: 685–713.
22. Calvo N, Brugada J, Sitges M, et al. Atrial fibrillation and atrial flutter in athletes. *Br J Sports Med* 2012; 46: i37–43.
23. D'Andrea A, Scarafile R, Riegler L, et al. Right atrial size and deformation in patients with dilated cardiomyopathy undergoing cardiac resynchronization therapy. *Eur J Heart Fail* 2009; 11: 1169–1177.
24. Levine BD, Baggish AL, Kovacs RJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task force 1: Classification of sports: Dynamic, static and impact. *J Am Coll Cardiol* 2015; 66: 2350-2355.
25. Mosteller, RD. Simplified calculation of body surface area. *N Engl J Med* 1987; 317:

- 1098.
26. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015; 28 :1–39.
 27. Popović ZB, Sun JP, Yamada H, et al. Differences in left ventricular long-axis function from mice to humans follow allometric scaling to ventricular size. *J Physiol* 2005; 568: 255–265.
 28. Batterham AM, George KP, Whyte G, et al. Scaling cardiac structural data by body dimensions: a review of theory, practice and problems. *Int J Sports Med* 1999; 20: 495-502.
 29. Mor-Avi V, Lang RM, Badano LP, et al. Current and evolving echocardiographic techniques for the quantitative evaluation of cardiac mechanics: ASE/EAE consensus statement on methodology and indications endorsed by the Japanese Society of Echocardiography. *J Am Soc Echocardiogr* 2011; 24: 277–313.
 30. Oxborough D, George K, Birch K. Intraobserver reliability of two dimensional ultrasound derived strain imaging in the assessment of the left ventricle, right ventricle and left atrium of healthy human hearts. *Echocardiography* 2012; 29: 793-802.
 31. Harris PA, Taylor R, Thielke R, et al. Research electronic data capture (REDCap)-A metadata-driven methodology and workflow process for providing translational research informatics support. *J Biomed Inform* 2009; 42: 377–381.
 32. Spence AL, Naylor LH, Carter HH, et al. A prospective randomised longitudinal MRI study of left ventricular adaptation to endurance and resistance exercise training in

- humans. *J Physiol* 2011; 589: 5443–5452.
33. Arbab-Zadeh A, Perhonen M, Howden E, et al. Cardiac remodeling in response to 1 year of intensive endurance training. *Circulation* 2014; 130: 2152–2161.
 34. La Gerche A, Heidbüchel H, Burns AT, et al. Disproportionate exercise load and remodeling of the athlete's right ventricle. *Med Sci Sports Exerc* 2011; 43: 974–981.
 35. Qasem M, Utomi V, George K et al. A Meta-Analysis for the Echocardiographic Assessment of Right Ventricular Structure and Function in ARVC. *Echo Res Pract* 2016; 3: 95-104
 36. Forsythe, L., MacIver, D. H., Johnson, C et al. The relationship between left ventricular structure and function in the elite rugby football league athlete as determined by conventional echocardiography and myocardial strain imaging. *Int J Cardiol* 2018; 261: 211-217.
 37. MacIver DH and Townsend M. A novel mechanism of heart failure with normal ejection fraction. *Heart* 2008; 94: 446–449.
 38. Turagam MK, Velagapudi P and Kocheril AG. Atrial Fibrillation In Athletes. *Am J Cardiol* 2012; 109: 296-302.

Table Legends

Table 1: Demographics

Table 2: Echocardiographic parameters of the right ventricle

Table 3: Global and regional right ventricular ϵ and SR

Table 4: Echocardiographic parameters of the right atrium

Figure 1: Measurement of RV structural dimensions. a, RVOT_{plax}; 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

Figure 3: Measurement of RV ϵ and SR. a, Athlete RV ϵ ; b, Athlete RV SR; c, Control RV ϵ ; d, Control RV SR.

Table 1: Demographics

	Athlete Mean±SD (Range)	Control Mean±SD (Range)	P value
Age (Years)	24±4 (19-34)	22±3 (20-35)	0.001*
Height (m)	1.82±0.06 (1.62-1.98)	1.78±0.06 (1.65-1.91)	<0.001*
Weight (Kg)	96±11 (75-132)	78±9 (60-107)	<0.001*
BSA (m²)	2.20±0.15 (1.91-2.66)	1.96±0.13 (1.66-2.38)	<0.001*
HR (Beats.min⁻¹)	56±10 (39-83)	69±9 (50-95)	<0.001*
Systolic BP	131±9 (107-155)	129±10 (113-151)	0.413
Diastolic BP	69±7 (53-89)	74±7 (63-90)	<0.001

*denotes statistical significance at the level $P < 0.05$

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

Table 2: Echocardiographic parameters of the right ventricle

	Athlete Mean±SD (Range)	Control Mean±SD (Range)	P Value
RVOT_{PLAX} (mm)	34±4 (21-47)	28±4 (20-36)	<0.001*
RVOT₁ (mm)	36±5 (22-49)	29±4 (19-35)	<0.001*
RVOT₂ (mm)	27±3 (19-35)	23±2 (18-28)	<0.001*
RVD₁ (mm)	44±5 (33-60)	39±4 (31-47)	<0.001*
RVD₂ (mm)	33±4 (22-44)	30±5 (17-42)	<0.001*
RVD₃ (mm)	91±8 (72-111)	82±7 (71-98)	<0.001*
RVD_a (cm²)	30±4 (21-41)	22±3 (15-29)	<0.001*
RVS_a (cm²)	16±3 (10-23)	12±2 (6-18)	<0.001*
RV WT (mm)	4±1 (2-7)	4±1 (3-5)	<0.001*
TAPSE (mm)	24±4 (16-33)	23±3 (17-32)	0.144
RVOT₁:RVD₁ Ratio	0.81±0.14 (0.52-1.23)	0.76±0.11 (0.44-0.97)	0.012*
RV:LV Ratio	0.91±0.10 (0.70-1.20)	0.82±0.07 (0.66-1.01)	<0.001*
RVFAC (%)	46±6 (34-61)	47±7 (38-64)	0.442
RVOT_{PLAX} (mm/(m²)^{0.5})	23±3 (15-30)	20±2 (15-25)	<0.001*
RVOT₁ (mm/(m²)^{0.5})	24±3 (15-32)	21±3 (14-26)	<0.001*
RVOT₂ (mm/(m²)^{0.5})	18±2 (13-24)	17±2 (13-20)	<0.001*
RVD₁ (mm/(m²)^{0.5})	30±3 (22-38)	28±2 (23-34)	<0.001*
RVD₂ (mm/(m²)^{0.5})	22±3 (15-30)	21±3 (12-29)	0.174
RVD₃ (mm/(m²)^{0.5})	61±5 (47-75)	59±5 (49-70)	0.04*
RVD_a Index (cm²/m²)	14±2 (9-18)	11±2 (7-15)	<0.001*
RVS_a Index (cm²/m²)	7±1 (4-11)	6±1 (3-8)	<0.001*
RVS' (cm/s)	15±2	14±2	0.581

	(6-23)	(10-18)	
RVE' (cm/s)	15±3 (7-24)	15±3 (9-21)	0.502
RVA' (cm/s)	10±3 (5-17)	11±3 (6-18)	0.852
RV E'/A' (cm/s)	1.54±0.44 (0.71-3.00)	1.56±0.49 (0.81-2.71)	0.775
RVS' index ((cm/s)/cm)	1.61±0.29 (0.61-2.44)	1.75±0.26 (1.06-2.34)	0.002*
RVE' index ((cm/s)/cm)	1.65±0.32 (0.84-2.76)	1.89±0.39 (0.98-2.76)	<0.001*
RVA' index ((cm/s)/cm)	1.15±0.33 (0.56-2.05)	1.28±0.34 (0.74-2.05)	0.015*

*denotes statistical significance at the level $P < 0.05$

RVOT_{PLAX}, 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 ϵ and SR

	Athlete mean \pm SD (Range)	Control mean \pm SD (Range)	P Value
Global RVϵ (%)	-27.2 \pm 3.4 (-18.4 to -40.7)	-28.4 \pm 4.2 (-19.1 to -41.2)	0.053
Time to Peak RV ϵ (s)	0.38 \pm 0.03 (0.31-0.46)	0.36 \pm 0.03 (0.31-0.44)	<0.001*
RVSRS (s⁻¹)	-1.32 \pm 0.22 (-0.77 to -2.19)	-1.48 \pm 0.28 (-0.97 to -2.34)	<0.001*
RVSRE (s⁻¹)	1.59 \pm 0.33 (0.79-2.67)	1.92 \pm 0.50 (1.11-3.26)	<0.001*
RVSRA (s⁻¹)	0.89 \pm 0.27 (0.34-1.77)	1.09 \pm 0.28 (0.39-1.85)	<0.001*
Basal RVϵ (%)	-24.9 \pm 5.4	-26.3 \pm 4.9	0.105
Mid RVϵ (%)	-27.2 \pm 4.1	-28.4 \pm 4.8	0.096
Apical RVϵ (%)	-30.0 \pm 4.3	-31.1 \pm 4.6	0.146
Apex to Base RVϵ gradient (%)	-5.2 \pm 6.9	-4.8 \pm 5.0	0.743
Basal RVSRS (s⁻¹)	-1.50 \pm 0.41	-1.74 \pm 0.41	<0.001*
Mid RVSRS (s⁻¹)	-1.37 \pm 0.27	-1.50 \pm 0.31	0.004*
Apical RVSRS (s⁻¹)	-1.58 \pm 0.31	-1.86 \pm 0.43	<0.001*
Basal RVSRE (s⁻¹)	2.10 \pm 0.67	2.50 \pm 0.84	0.001*
Mid RVSRE (s⁻¹)	1.68 \pm 0.40	2.02 \pm 0.58	<0.001*
Apical RVSRE (s⁻¹)	2.07 \pm 0.55	2.22 \pm 0.54	0.081
Basal RVSRA (s⁻¹)	1.06 \pm 0.34	1.26 \pm 0.38	0.001*
Mid RVSRA (s⁻¹)	0.98 \pm 0.33	1.13 \pm 0.37	0.007*
Apical RVSRA (s⁻¹)	1.25 \pm 0.40	1.46 \pm 0.44	0.003*

*denotes statistical significance at the level $P < 0.05$

RV ϵ , 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: Echocardiographic parameters of the Right Atrium

	Athlete Mean±SD (Range)	Control Mean±SD (Range)	P Value
RAa (cm2)	22±4 (13-29)	15±2 (10-20)	<0.001*
RAa Index (cm²/m²)	10±1 (6-13)	8±1 (5-10)	<0.001*
RAVOLes (ml)	73±18 (33-121)	44±10 (25-63)	<0.001*
RAVOLes Index (ml/(m²)^{1.5})	22±5 (11-35)	16±4 (10-24)	<0.001*
RAVOLpreA (ml)	49±13 (25-92)	28±7 (14-45)	<0.001*
RAVOLpreA Index (ml/(m²)^{1.5})	15±4 (6-27)	10±2 (6-16)	<0.001*
RAVOLed (ml)	35±10 (15-75)	18±5 (7-32)	<0.001*
RAVOLed Index (ml/(m²)^{1.5})	10±3 (5-22)	6±2 (3-10)	<0.001*
RAVOLres (ml)	39±11 (13-77)	26±7 (13-44)	<0.001*
RAVOLcon (ml)	53±17 (5-96)	36±12 (12-65)	<0.001*
RAVOLboo (ml)	14±5 (5-30)	10±3 (4-19)	<0.001*
Con : Boo Ratio	4.32±2.65 (0.17-16.2)	4.07±2.27 (1.2-12.75)	0.557

*denotes statistical significance at the level P<0.05

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.