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Right Ventricular Structure and Function in Senior and Academy Elite Footballers

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Abstract

Aims

Right ventricular (RV) adaptation is a common finding in the athlete's heart. The aim of this study was to establish the extent of RV structural and functional adaptation in elite and academy professional footballers compared to age-matched controls.

Methods and Results

100 senior and 100 academy elite footballers, 20 senior and 19 academy age-matched controls were recruited. All participants underwent 2D, Doppler, tissue Doppler and strain (ε) echocardiography of the right heart. Structural indices were derived and indexed allometrically for individual differences in body surface area. Standard RV function was assessed alongside peak RV ε and strain rate (SR). Senior football players had larger scaled RV structural parameters than players for the RV outflow ($RVOT_{plax}$) (32.7±4.2 and 29.5±4.0 $mm(m^2)^{0.326}$, P<0.001), the proximal RV outflow ($RVOT_1$) (26.6±3.5 and 24.7±3.9 $mm(m^2)^{0.335}$, P<0.001), the basal RV inflow (RVD_1) (33.1±4.1 and 30.7±3.2 $mm(m^2)^{0.404}$, P=0.020), RV length (RVD_3) (66.5±6.1 and 62.9±5.1 $mm(m^2)^{0.431}$, P<0.001) and RV diastolic area (RVD_{area}) (16.9±2.6 and 15.7±2.6 $mm(m^2)^{0.735}$, P<0.001). Both academy and senior football players demonstrated larger scaled structural RV parameters in comparison to age matched controls. Systolic SR (SRS) was lower in the senior players compared to academy players in the mid (-1.52±0.49 and -1.41±0.34 l/s, P=0.019) and apical (-1.97±0.74 and -1.72±0.42 l/s, P=0.025) wall regions, respectively.

Conclusion

RV structural adaptation occurs in both senior and academy football players with senior players having larger RV dimensions. Although senior players have slightly lower peak SRS than academy players, all global ε and SR are within normal ranges.

Key Words: Football; Strain Imaging; Echocardiography; Right Ventricle; Age

Introduction

It is well established that the athlete's heart (AH) undergoes physiological remodelling in response to chronic exercise training with the right ventricle (RV) demonstrating both structural and functional adaptation[1-4]. Much of the work in this field has utilised either endurance or strength trained athletes[3,4]. Football involves high dynamic components to training and competition placing specific haemodynamic loading on the heart. The American College of Cardiology Task Force[5] categorized football in group AIII (high dynamic [> 70% max O_2] and low static [<20% maximal voluntary contractions]) such that this sporting discipline serves as an ideal model to assess the impact of dynamic training on RV structure and function.

Football is one of the world's most popular sports, with its world governing body Federation Internationale de Football Association (FIFA) requiring all players to have a resting transthoracic echocardiogram, including a full assessment of the RV as part of their cardiovascular screening. The echocardiographic assessment of the RV aims to differentiate physiological adaptation from

pathological changes associated with arrhythmogenic right ventricular cardiomyopathy (ARVC). Evidence suggests that ARVC accounts for between 3 and 10% of sudden cardiac death in the athletic population[6]. FIFA guidelines suggest that tricuspid plane systolic excursion (TAPSE) and RV fractional area change (RV_{FAC}) are undertaken to assess RV function despite the known limitations of these indices[6]. Myocardial strain (ε) imaging (MSI) and tissue Doppler imaging (TDI) provide alternative measures of RV function that may overcome some of the limitations of traditional 2D imaging[7] but have yet to be fully explored in the footballing population. With this in mind, it is clear that a comprehensive understanding of RV structure and function in this sporting discipline is important to on-going screening programmes worldwide.

Pre-participation cardiovascular screening is not constrained to the senior footballers with many elite, professional clubs providing a screening service to the academy players between the ages of 14 and 18. There is also a national mandate in England from the Football Association to screen any player over the age of 15 years. The impact of accumulate training volume with increasing age has received minimal attention in elite footballers. Whilst some data demonstrated a lower magnitude of left ventricular (LV) adaptation in adolescent athletes compared to their senior counterparts[8,9] there is no available data for the RV.

In view of this, the study was primary aimed to examine football-specific variation of RV structural and functional parameters in elite senior and academy professional players who were compared to age-matched controls. The findings from this study will impact upon cardiovascular pre-participation screening.

Materials and Methods

Study Design and Population

Two hundred male elite football players were prospectively recruited into the study during pre-participation screening. Of these, one hundred were professional senior players and one hundred were professional academy players. Average training hours were 20 hours/week for senior players and 15 hours/week for academy players. Thirty-nine participants were recruited as sedentary (undertaking less than 3 hours structured exercise per week) controls; twenty of which were age matched to senior players and nineteen age matched to the academy players.

The screening process occurred during the preseason period and all players refrained from any exercise within 6 hours of the examinations. Participants completed a pre-participation screening questionnaire prior to examination which demonstrated all to be free of known cardiovascular disease, diabetes and renal disease. There was no evidence of a family history of sudden cardiac death or unexplained death under the age of 40 years. A 12-lead electrocardiogram (ECG) was undertaken to exclude a range of "silent" inherited cardiovascular diseases. If any participant was found to have abnormal training-related ECG recordings, categorised via published criteria[10], they were referred for a full range of cardiac investigations and all were subsequently confirmed to have no cardiovascular disease. Height and body mass were measured using standard equipment and body surface area (BSA) was calculated using a standardised formula[11].

Informed consent was provided by the individual or the legal guardian (in the case of minors) and the study was granted ethics approval by the Ethics Committee of Liverpool John Moores University or St Georges University Hospital.

Transthoracic Echocardiography

All echocardiographic examinations were performed with the subject in the left lateral decubitas position using a commercially available ultrasound system (Vivid-Q, GE Healthcare, Horton, Norway) by two experienced sonographers, to maximise quality control. A complete echocardiographic study was performed with an additional focus of the right heart and all images were acquired in accordance with the American Society of Echocardiography (ASE)[12,13]. Images were stored in a raw Digital Imaging and Communications in Medicine (DICOM) format and exported to an offline analysis system (EchoPac, GE Healthcare, Horton, Norway). Subsequent data analysis was performed by a single experienced sonographer using an average of 3 cardiac cycles for all measurements.

Standard Conventional 2D Doppler & Tissue Doppler. All RV measurements were made in accordance with ASE guidelines[13]. The parasternal long and short axis orientations were used to establish RV outflow tract dimensions at the proximal level from a parasternal long axis ($RVOT_{plax)}$ and parasternal short axis ($RVOT_{1)}$ as well as distal level ($RVOT_2$). A modified apical four chamber orientation was used to obtain measurements from the main body of the RV and included dimensions at the RV base (RVD_1), mid-cavity (RVD_2) and RV length (RVD_3). In addition, RV area was calculated in diastole (RVD_{area}) and systole (RVS_{area}) allowing the calculation of fractional area change (RV_{FAC}). RV wall thickness (RVT) was measured in a subcostal orientation. TAPSE was

measured using M-mode echocardiography with the cursor positioned through the lateral aspect of the tricuspid valve annulus. A 4 mm pulsed wave Doppler sample volume was placed sub-pulmonary valve in the RV outflow tract allowing the assessment of the velocity time interval ($RVOT_{VTI}$). Pulsed wave TDI was used to interrogate the RV lateral wall with a 2 mm sample volume positioned within the tricuspid annulus. Peak systolic (S'), early diastolic (E') and late diastolic (A') myocardial velocities were measured.

Myocardial Speckle Tracking. A modified apical four chamber with RV focus was utilised for assessment of myocardial ε and strain rate (SR). Images were optimised using depth, gain, compression and sector width to provide optimal endocardial delineation. The focal point was positioned mid cavity to reduce the impact of beam divergence and frame rates were set between 80 and 90 frames per second. Offline analysis involved placing a region of interest along the RV lateral wall from base to apex. The software automatically tracked the 3 segments (base, mid and apex) and provided an interpretation on tracking quality. Furthermore, the operator provided a subjective assessment of tracking quality and segments were excluded if deemed unacceptable. The peak values for ε and SR during ventricular systole (SRS') and early and late ventricular diastole (SRE' and SRA' respectively) were reported for the 3 wall segments[13].

Statistical Analysis

In order to establish body size independent indices for RV structural parameters we adopted sample-specific allometric exponents for the relationship between RV data and BSA. This involved an iterative, non-linear protocol using age as a co-variate with the model y = a:xb*exp(c*age) providing *b* exponents and coefficient C for each structural parameter. The coefficient C was close to zero for all parameters suggesting that the derived *b* exponent could be applied to all players irrespective of age. Finally, a correlation analysis was undertaken for each allometrically scaled parameter to BSA to establish true size independence. We defined the smallest worthwhile effect for the allometric relationships as a correlation coefficient of r = 0.30, a moderate effect size in Cohen's terms.

All absolute and scaled RV structural and functional parameters are presented in terms of mean ± SD. To compare between the senior and academy populations, normal distribution was confirmed using a Kolmogorov-Smirnov test followed by a two way between group ANOVA to compare means. Where a significant interaction was identified a post-hoc pairwise comparison with Bonferroni correction was undertaken to determine the nature and significance of the interaction. All statistical data was analysed using dedicated software (Statistical Package for the Social Sciences (SPSS) version 23.0,

Chicago IL). To inform and support clinical pre-participation screening, absolute and scaled RV upper limit (cut –off) data for academy and senior players were established using 2 standard deviations upwards from the group mean. These values were compared to published data for the non-athletic population[13], the endurance athletic population[1] and ARVC population[14].

Results

As would be expected the senior players and controls (25 ± 5 years and 25 ± 4 years) were older than academy players and academy controls (16 ± 1 years and 16 ± 1 years), respectively. There was a significant main effect for age for BSA with senior players and senior controls having a larger BSA (2.0 ± 0.1 m^2 and 2.0 ± 0.1 m^2) than academy players and academy controls (1.9 ± 0.1 m^2 and 1.8 ± 0.1 m^2). There was a main effect for playing status on resting heart rate with academy (60 ± 11 bpm) and senior (58 ± 11 bpm) football players having lower heart rates than age-matched controls (academy: 69 ± 11 bpm and senior: 70 ± 8 bpm).

All RV structural parameters are presented in table 1. There was a significant main effect for both age (P<0.05) and playing status (P<0.05) on absolute and indexed $RVOT_{plax}$, $RVOT_1$, $RVOT_2$, RVD_1, RVD_3 and RVDarea highlighting higher values with age and elite playing status. These differences remained after allometric scaling for BSA, except for $RVOT_2$. Absolute and scaled RVD_2 and RVWT had a significant main effect for playing status, with senior players and academy players having larger parameters than their respective age matched controls. There was also a significant interaction between age and playing status for absolute and scaled RVDarea and RVD_3 (see Figure 1) with the between group difference demonstrating with age; these measures of RV size decline in size whilst athletes demonstrate a converse pattern with enlargement of the RV during the ageing process.

INSERT TABLE 1 AND FIGURE 1

All conventional functional data are presented in table 2. There was a significant main effect for playing status on TAPSE with senior and academy players having higher values than their respective age matched sedentary controls. There was a significant main effect of age on RVOT VTI and TDI RVA' with senior players and senior controls having higher values than academy players and controls.

Regional RV peak ε and SR data tended to be more similar between groups. Both peak SRS basal and peak SRE basal (see table 3) presented with a significant main effect of age (P<0.05) where senior players and controls had higher values than academy players and controls. There was a significant interaction effect for age and playing status on peak basal SRE basal with the difference between senior players and controls being smaller than those between academy player and controls (see Figure 1).

Table 4 presents absolute RV upper limit (cut-off) data for academy and senior players, compared to published data for the non-athletic population, the endurance athletic population and ARVC population. Both academy and senior football groups had players whose data fell above the normal upper limits for $RVOT_1$ (13% and 30%), RVD_1 (20 and 57%) RVD_3 (29% and 66%) and RVD_{area} (44% and 70%), respectively. Senior players also displayed data that fell above published values in endurance athletes for $RVOT_1$ (1%), RVD_1 (2%) and RVD_{area} (3%). Both academy and senior football players demonstrated data above cut off values for an ARVC population for $RVOT_{PLAX}$ (19% and 52%) and $RVOT_1$ (10% and 22%), respectively.

Discussion

To the best of our knowledge, this is the first study of its type to assess RV adaptation in elite senior and academy football players. The main findings from this study are; 1) when appropriately indexed for body size, senior football players have significantly larger right ventricles than their academy counterparts, with the exception of $RVOT_2$, RVD_2 and RVWT and 2) Regional and global SR is lower in senior players than academy players, with mid and apical values reaching statistical significance for SRS and basal level for SRE.

Right Ventricular Structure

RV structural adaptation to chronic exercise is a physiological process and has been linked to training volume and exercise type[15,16]. Previous work has demonstrated larger RV volumes and dimensions in endurance trained athletes compared to those athletes predominantly based in strength disciplines[3]. Our data builds on this and highlights that elite athletes with minimal static / strength component to their training still develop significant RV enlargement compared to the non-athletic population. This is likely based on the significant volume challenges associated with dynamic, high intensity exercise. As well as the increased preload required to augment cardiac stroke volume, there is the relative

disproportionate wall stress that the RV is exposed to[17], both of which are likely to act as acute stimuli for chronic adaptation[18].

To date, RV structural adaptation and its relationship to an athlete's experience and training longevity has not been assessed. A single study assessed LV adaptation in 900 adolescent national level athletes from ten different sporting disciplines and 250 age and sex matched sedentary controls[8]. They observed increased echocardiographic dimensions compared to controls, however, when compared to reported values of senior athletes, the adaptation was of a lower magnitude. Our data reflects this finding in that all the allometrically scaled dimensions, apart from $RVOT_2$, RVD_2 and RVWT, are statistically larger in football groups and to a greater magnitude in senior players than academy. This is further highlighted when comparing individual values to normative ranges with both academy and senior players demonstrating average data above the established cut-offs. The lack of difference in some indices reflects the nature of physiological RV adaptation i.e. primarily affecting the basal inflow and proximal outflow. Our data is also in agreement to others as to the limited effect on RV wall thickness[3] and is likely due to the thin walled RV not having the muscular integrity to overcome elevated wall stress through a process of concentric hypertrophy. This suggests that the RV may undergo greater and variable adaptation with repeated exposures to an exercise stimulus and is therefore dependent on training longevity. The lack of statistical significance between senior and academy sedentary controls re-confirms that this is a training-related rather than an age-related adaptation. The RVOT is an extremely important measurement when excluding ARVC[14] and therefore we should be aware that the $RVOT_{plax}$ and $RVOT_1$ may be larger in football players, and to a greater extent in senior players. This clearly has implications for pre-participation screening and hence justifies the rationale to develop a set of age related normal values.

The use of scaling when assessing cardiac size aids diagnostic decision making and is advocated in both clinical and research settings[19]. The impact of body size on cardiac morphology is a well distinguished relationship and routine ratio indexing of chamber size to BSA is recommended in guidelines[13]. However, ratio indexing is based on an assumption of a linear relationship which does not conform to biological associations which often occur in a non-linear fashion[20]. It has therefore been encouraged in recent literature to use allometric indexing where possible[1]. Published cut off values for RV size as defined by the ASE[12] and adopted by the British Society of Echocardiography (BSE) are reported as absolute and unscaled. This therefore becomes problematic when attempting to compare groups of various body sizes. Our data highlights that values for both academy and senior football players are above these normal values and hence body size may in part, explain the observed difference. However, until a substantive indexed normal data set is available, it is unreasonable to

draw any firm conclusions. The study by Oxborough et al. (2012) used elite endurance athletes, including top level triathletes and cyclists, to produce endurance athlete data. Allometrically indexed RV structural values fall below those of the endurance athlete population, with the exception of $RVD_{3,}$ which can be justified by the task force continuum[5] where football requires the same volume of energy (10.0 METS) as an endurance pursuit but with a lower static component.

Right Ventricular Function

Conventional assessment of the RV is challenging due to its complex structure, location and thin myocardial wall[6], and hence the recent interest and application of MSI. Previous literature has demonstrated that global ε is lower in the athletic population because of regional lower values in the basal segment[2,21]. The mechanisms behind this are unclear, with Teske et al. (2009) suggesting normal physiologic adaptation (representative of myocardial reserve) or RV damage due to chronic endurance training. Previous work by our group[1] has not managed to reproduce these findings and the current data presented here highlights normal ε and SR values for all participants. Although all ε and SR values were within normal ranges we did observe a significantly lower global and regional SRS in the senior footballers compared to those from the academy. We could speculate that the reduced rate of systolic deformation could be a consequence of the increased RV volume requiring less rate of contractility to generate an adequate resting stroke volume i.e. smaller change for any given volume. This is important from a physiological perspective and highlights the direct relationship between structure and function which further supports the work of Teske et al.[2] and La Gerche et al.[17]. Interestingly, we performed a post-analysis bivariate correlation between RV structural indices and regional SRS which failed to demonstrate any relationship in both senior and academy players. This lack of any clear correlation may provide evidence to refute this hypothesis however this finding may also be a consequence of limitations related to isolated and individual linear dimensions not being able to fully represent the volumetric geometry of the RV. It is apparent that if this technique was to be taken on board for routine assessment of the athlete then specific demographics need to be considered when interpreting the absolute values. It is equally important to note that other indices of RV function in systole and diastole (including TDI), apart from TAPSE and TDI RVA', were not different between the two groups and therefore this finding highlights the increased sensitivity of MSI in detecting discrete changes in RV function. Basal RV diastolic function as determined by SRE' demonstrated a statistical interaction which highlights a lower value in athletes compared to controls which normalizes with age. This demonstrates a reduction in regional SRE' through normal ageing which is not seen to the same magnitude in the athletic population. We speculate that the enlargement of the RV in this region in the football players probably accounts for the initial lower values but with

the additional evidence to suggest that a lack of structured exercise may cause an ageing decline even in this relatively young population.

Limitations

To compare the cardiac impact of training longevity, the study was conducted on a population of predominantly Caucasian, male footballers. Caution must therefore be taken when trying to extrapolate this data to athletes of other sporting disciplines, gender or ethnicity. Screening occurred at variable points in the competitive season and although all athletes were participating in national and international standard football, it would be valuable to establish whether we would observe similar findings pre-and post-season[22]. In addition, the unbalanced sample sizes for each group may impact on statistical significance.

Conclusions

RV structural adaptation occurs in senior and academy football players, however training longevity and experience appear to impact directly on the size of the RV inflow and outflow tract. Although senior players have slightly lower peak SRS and SRE than academy players, global ε and SR are within normal ranges in both groups and therefore this technique, in conjunction, with other variables may provide added value in the pre-participation screening environment. The proposed cut off values may also provide useful ranges in pre-participation screening. Allometric scaling for body size is important and future guidelines should aim to incorporate this into their recommendations.

Perspectives

In a young athletic population, it is thought that 3% to 10% of all cardiac sudden deaths are associated with ARVC[6]. With this is mind, the differential diagnosis of physiological adaptation from ARVC is of utmost importance in the pre-participation environment. The current literature provides guidelines for a normal population[12,13], an endurance athletic population[1] and an ARVC population[14], yet guidelines for an age specific football population are currently not available. This study presents absolute and scaled RV cut off values for both academy and senior football populations. It is important to highlight that if the RV chamber size falls within these proposed cut-offs in the presence of normal RV conventional indices of function and myocardial ε then physiological adaptation is likely. That aside, it is important to consider these findings in tandem with clinical findings, family history and the 12-lead ECG. Where absolute values are elevated, consider scaling allometrically and

recheck. If the value still falls above the proposed cut-offs, then corroborative investigations are recommended. It is also important to note that although a relatively large specific population was utilized within this study, further large cohort studies should be undertaken in order to build on the current data.

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Figure and table legends

Table 1 – Absolute and Scaled RV Structural Parameters

Table 2 – RV Conventional Functional Parameters

Table 3 - RV Global Strain (ϵ) and Strain Rate variables

Table 4 – Absolute and indexed cut-off values for screening purpose

Figure 1 – Significant Interactions for RVDarea, RVD_3 and Basal SRE'

Table 1 – Absolute and scaled RV structural parameters (data are mean ± SD)

	Academy Football	**Academy Control**	**Senior Football**	**Senior Control**
Age (years)	16 ± 1	16 ± 1	25 ± 5	25 ± 4
$RVOT_{plax}$ (mm) *Δ	30 ± 4	29 ± 3	33 ± 4	30 ± 3
$RVOT_{plax}$ (index) ($mm(m^2)^{0.326}$) *Δ	24 ± 3	24 ± 3	26 ± 3	24 ± 3
$RVOT_1$ (mm) *Δ	30 ± 5	29 ± 3	34 ± 5	31 ± 3
$RVOT_1$ (index) ($mm(m^2)^{0.335}$) *Δ	25 ± 4	24 ± 3	27 ± 4	25 ± 2
$RVOT_2$ (mm) *Δ	24 ± 3	22 ± 3	25 ± 4	24 ± 3
$RVOT_2$ (index) ($mm(m^2)^{0.296}$) *	20 ± 3	19 ± 2	21 ± 3	19 ± 3
RVD_1 (mm) *Δ	40 ± 4	37 ± 4	44 ± 6	39 ± 4
RVD_1 (index) ($mm(m^2)^{0.404}$) *Δ	31 ± 3	30 ± 3	33 ± 4	30 ± 3
RVD_2 (mm) *	30 ± 5	27 ± 4	31 ± 4	27 ± 4
RVD_2 (index) ($mm(m^2)^{0.64}$) *	20 ± 3	18 ± 3	20 ± 3	17 ± 3
RVD_3 (mm) *Δ○	82 ± 8	80 ± 9	90 ± 8	80 ± 9

RVD_3 (index) *○ **$(mm(m^2)^{0.431})$**	63 ± 5	63 ± 6	67 ± 6^+	59 ± 7
RVD_{area} (cm^2) *○	25 ± 4	22 ± 3	28 ± 5^+	22 ± 4
RVD_{area} (index) $(cm^2(m^2)^{0.735})$ *○	16 ± 3	14 ± 2	17 ± 3	13 ± 3
RVWT (mm) *Δ	3.9 ± 0.7	2.7 ± 0.6	3.9 ± 0.9	3.6 ± 0.5
RVWT (index) $((mm/m^2)^{-0.111})$ *Δ	4.2 ± 0.8	2.9 ± 0.6	4.3 ± 1.0	3.9 ± 0.5

** Main effect of playing status (P<0.05)*

Δ Main effect of age (P<0.05)

○Interaction (P<0.05)

^ Post Hoc Pairwise Significance between Senior Players and Academy Players

+ Post Hoc Pairwise Significance between Senior Players and Senior Controls

Table 2 – RV conventional functional parameters (data are mean ± SD)

	Academy Football	**Academy Control**	**Senior Football**	**Senior Control**
RV_{FAC} (%)	51 ± 10	49 ± 7	48 ± 7	51 ± 8
TAPSE (mm) *	23 ± 4	22 ± 3	25 ± 4	22 ± 2
RVOT VTI (cm) Δ	21 ± 3	19 ± 2	20 ± 3	23 ± 2
TDI RVS' (cm/s)	14 ± 2	15 ± 2	14 ± 3	14 ± 2
TDI RVE' (cm/s)	15 ± 3	16 ± 3	15 ± 3	14 ± 4
TDI RVA' (cm/s) Δ	8 ± 3	9 ± 3	10 ± 3	10 ± 2

** Main effect of playing status (P<0.05)*

Δ Main effect of age (P<0.05)

Table 3 - RV Strain (ϵ) and Strain Rate variables (data are mean ± SD)

		Academy Football	Academy Control	Senior Football	Senior Control
PEAK ϵ (%)	**Basal**	-24.00 ± 4.87	-26.72 ± 5.61	-23.87 ± 4.38	-23.90 ± 4.01
	Mid	-27.81 ± 4.39	-28.43 ± 4.64	-26.56 ± 6.98	-28.01 ± 5.32
	Apical	-29.64 ± 6.35	-31.46 ± 4.54	-30.80 ± 4.79	-31.27 ± 5.59
PEAK SRS (l/s)	**Basal** *Δ	-1.52 ± 0.49	-1.52 ± 0.44	-1.41 ± 0.34	-1.81 ± 0.53
	Mid	-1.55 ± 0.41	-1.54 ± 0.40	-1.36 ± 0.44	-1.54 ± 0.47
	Apical	-1.97 ± 0.74	-2.01 ± 0.43	-1.72 ± 0.42	-1.77 ± 0.43
PEAK SR E (l/s)	**Basal** *Δ○	1.80 ± 0.51^	2.74 ± 0.82	1.97 ± 0.55	2.01 ± 0.83
	Mid	1.90 ± 0.44	2.13 ± 0.54	1.76 ± 0.41	1.83 ± 0.64
	Apical	2.34 ± 0.81	2.43 ± 0.52	2.24 ± 0.61	2.24 ± 0.67
PEAK SRA (l/s)	**Basal**	0.93 ± 0.39	1.14 ± 0.46	1.01 ± 0.36	1.04 ± 0.52
	Mid	0.91 ± 0.35	1.13 ± 0.45	0.99 ± 0.34	1.05 ± 0.43
	Apical	1.14 ± 0.44	1.40 ± 0.62	1.26 ± 0.38	1.34 ± 0.60

** Main effect of playing status (P<0.05)*

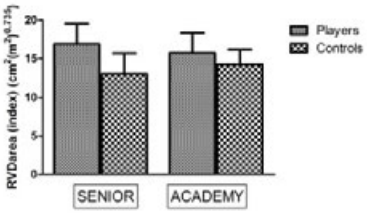
Δ Main effect of age (P<0.05)

○Interaction (P<0.05)

^ Post Hoc Pairwise Significance between Senior Players and Senior Controls

Table 4 – Absolute and indexed cut-off values for screening purposes

	Football Cut off values		**Published Cut off values**				
RV Parameter	**Academy (Mean ± 2 SD)**	**Senior (Mean ± 2 SD)**	**ASE Guidelines (Rudski *et al*., 2010)**	**Endurance Athletes (Oxborough *et al*., 2012)**	**ARVC Marcus (2010)**	**% academy players above upper limits**	**% senior players above upper limits**
$RVOT_{plax}$ (mm)	38	41			32	19>ARVC	52>ARVC
$RVOT_1$ (mm)	40	43	35	44	36	13>ASE 0>Endurance 10>ARVC	30>ASE 1>Endurance 22>ARVC
$RVOT_2$ (mm)	30	32					
RVD_1 (mm)	48	55	42	54		20>ASE 0>Endurance	57>ASE 2>Endurance
RVD_2 (mm)	39	40					
RVD_3 (mm)	98	107	86	110		29>ASE 0>Endurance	66>ASE 0>Endurance
RVD_{area} (cm^2)	34	37	25	36		44>ASE 1>Endurance	70>ASE 3>Endurance



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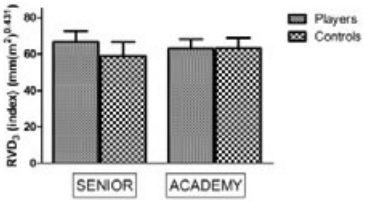

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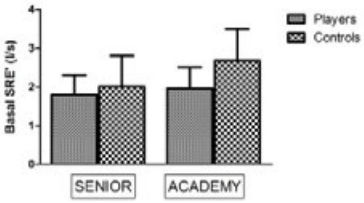

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