Does training type result in a dichotomous athletic heart phenotype in the right ventricle of male athletes

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Abstract

Aims: This study evaluated; A) global right ventricular (RV) phenotype in endurance vs. resistance trained male athletes; B) the impact of scaling for body size on the interpretation of RV structural data, and c) regional RV function using modern imaging technologies. **Methods:** A prospective cross-sectional design assessed the RV in 19 elite endurance-trained (ET), 21 elite resistance-trained (RT) and 21 sedentary control (CT) participants. Standard 2D, tissue-Doppler imaging (TDI) and speckle tracking echocardiography (STE) assessed RV structure and function. Indexing of RV structural parameters to body surface area (BSA) was undertaken using allometric scaling. **Results:** A higher absolute RV diastolic area was observed in ET (mean ± SD: 27 ± 4 cm^2) compared to CT (22 ± 4 cm^2; $P<0.05$) that was maintained after scaling. There was no difference between CT and RT (24 ± 4 cm^2; $P>0.05$). Whilst absolute RV longitudinal dimension was greater in ET (88 ± 9 mm) than CT (81 ± 10 mm; $P<0.05$) this difference was removed after scaling. There were no between group differences in global or regional RV function. **Conclusion:** ET athletes presented with some structural characteristics of an athletic heart but the RV in RT athletes was not different to CT. Scaling can impact structural data interpretation in the RV of athletes. Global and regional RV function was not altered in athletes. These data should be taken into account when discriminating the athlete's heart from pathology during screening.

Abstract 236 (Max 250)

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Tables 5, Reference 27 (Max 30)

INTRODUCTION

Regular intense sporting activity can lead to physiological hypertrophy of the left ventricle (LV) that was initially proposed to be training dependent (Morganroth et al. 1975). The training-dependent dichotomous athletic heart phenotype was linked to differential haemodynamic loading during acute exercise (George et al. 1991) resulting in eccentric hypertrophy (balanced increase in chamber and wall dimension) with endurance training (ET) versus concentric hypertrophy (disproportionate increase in wall thickness) with resistance training (RT). Recent evidence from individual studies and meta-analysis supports the existence of an athletic heart in ET but casts doubt over the degree of adaptation in RT (Haykowsky et al. 2000, Utomi et al. 2013, Utomi et al. 2014).

Whether the right ventricle (RV) demonstrates a training-dependent athletic heart phenotype has received less attention (Utomi et al. 2013). This may be partially due to the complexity of non-invasive imaging of the RV but technological advancements have resulted in the establishment of standardised assessment protocols for the RV (Rudski et al. 2010). This has prompted renewed interest in the RV of athletes and the ability to differentiate RV physiological hypertrophy from pathological changes associated with hereditary diseases such as arrhythmogenic RV cardiomyopathy (ARVC) that may increase the risk of sudden cardiac death (Maron et al. 2007).

A small number of studies have described greater RV structural parameters in ET athletes compared to controls (Baggish et al. 2008, Teske et al. 2009b, D'Andrea et al. 2013) but limited evidence has assessed the impact of different training focus on the RV (Koc et al 2007, D'Andrea et al. 2013, Pagourelias et al. 2013, King et al. 2013). In addition it is well established that taking into account differences in body size and composition can alter the

interpretation of LV athletic heart data (Dewey et al. 2008) but little attention has been given to the scaling of RV dimensions (Oxborough et al. 2012). This is, however, important in any RV data interpretation and could have implications for improving the sensitivity and specificity of existing upper normal limits for RV dimensions (Marcus et al. 2010).

Finally, available global and regional functional data in the RV of athletes is also limited. RV ε data in athletes are controversial with reduced resting deformation in some (Teske et al. 2009a, King et al. 2013, La Gerche et al. 2012) but not all studies (Oxborough et al. 2012, Pagourelias et al. 2013). Given the potential value of deformation parameters to improve the discrimination of normal and pathologic adaptation (Teske et al. 2009a, Marcus et al. 2010) further evaluation in a different athlete groups will be unique.

This study tests the hypotheses that; 1) global RV adaptation to exercise is mediated by training type; 2) the scaling of RV structural data will alter data interpretation, and 3) global and regional RV function will be training-type dependent.

METHODS

Study population

After the approval of the institutional ethics committee, 61 males provided written informed consent to participate in the study. Participants were recruited by direct contact with individuals, athletes, coaches and event organizers. Inclusion and exclusion criteria were as follows. We recruited elite athletes (competitive at a national or international level). Specifically, 19 were elite endurance (ET) athletes recruited from an international field at the Western States 100 miles endurance run (California, USA), 21 were elite

resistance trained (RT) athletes recruited from the British national weightlifting and Aikido squad. The ET and RT were matched for accumulated mean training years, training hours per week and training days per week of (ET: 11 yr, 12 hours/wk and 6 days/wk; RT: 12 yr, 11 hours/wk, 6 days/wk, respectively). All athletes reported a minimum of 6 years continuous national and international level participation and were competing at the highest competitive sporting level in their respective specialties. In addition, 21 sedentary controls (CT) were recruited from the university student population in a similar age range. The CT were healthy individuals who were not engaged in systematic sport-related training and engaged in less than 3 hours recreational activity per week. All participants self-reported as healthy, free from known cardiovascular disease and were not currently taking any form of prescribed medication. All participants were Caucasian, non-smokers, between the ages 18-45 and reported no early family history of cardiovascular disease. The study was guided by the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies (see checklist) (von Elm E et al. 2008) and conformed to the standards set by the Declaration of Helsinki. The demographic characteristics of all participants are presented in Table 1.

Study Design and Procedures

A prospective cross-sectional study design was employed with data acquired in a resting state at a single testing session. All subjects were advised to abstain from exercise training, caffeine and alcohol consumption at least 3 hours prior to the investigation. After a detailed explanation of the test protocol, subjects were initially issued with a medical questionnaire and then assessed for basic anthropometrics. Height and body mass were assessed using a stadiometer and digital weighing machine (SECA 764, Birmingham, UK.)

and body surface area (BSA) was calculated. After 5 minutes of supine rest, brachial artery systolic and diastolic blood pressures as well as resting heart rate were assessed with an automated sphygmomanometer (DINAMAP 300, GE Medical Systems, Milwaukee, Wisconsin).

Echocardiographic Assessment

Standard echocardiographic investigation was performed using a Vivid Q ultrasound machine (GE Medical System, Horten, Norway) with a 2.5-5 MHz transducer. All acquisitions were made with the subject lying in the left lateral decubitus position by the same experienced echocardiographer (DO) using a standard echo-protocol in accordance with the American Society of Echocardiography (ASE) (Lang et al. 2005). Standard 2D echocardiographic parameters were obtained from parasternal and modified apical acoustic windows (Rudski et al. 2010). Images were recorded to DVD in a raw Digital Imaging and Communications in Medicine (DICOM) format. Further offline analysis was performed using commercially available software (EchoPAC Version 7.0; GE Vingmed Ultrasound, Horten, Norway).

Conventional 2D and Doppler / Tissue Doppler

In accordance with ASE guidelines, RV size was measured at end-diastole from the proximal out flow tract (RVOT) using a parasternal short-axis (PSAX) orientation at the level of the aortic valve and at the basal RV inflow (RV1) from a modified apical 4-chamber orientation. RV length (RVL) was also measured from the RV apex to the tricuspid annulus (RVD3). RV diastolic and systolic areas were calculated by tracing around the endocardium from a modified apical 4-chamber orientation, and RV fractional area change was calculated

(Rudski et al. 2010). All RV structural variables were allometrically scaled for individual differences in BSA according to laws of geometric similarity (Dewey et al. 2008).Colour TDI was used to assess RV myocardial velocities. Standard colour Doppler tissue imaging acquisition was undertaken with a temporal resolution >150 framesec^{-1} . Offline analysis allowed a 6 x 6mm sample volume to be placed in the tricuspid annulus of the RV lateral wall and peak velocities in systole (RVS') and early diastole (RVE') and late diastole (RVA') to be measured. Tissue velocity data were scaled to RV length (Oxborough et al. 2012).

Speckle Tracking echocardiography (STE)

The apical 4-chamber orientation was used for the assessment of longitudinal RV ε and SR. During off-line analysis, the region of interest was placed around the RV lateral wall from the base to apex. Global peak values were obtained as the average of the base, mid and apical wall segments to determine a basal-to-apex gradient. Indices obtained included peak RV ε and SR during ventricular systole (SRS) and during early and late ventricular diastole (SRE and SRA). Intra-observer variation for RV structural and functional data were analysed in our laboratory on 20 subjects with two separate acquisitions to achieve a within-participant, within day design (Oxborough et al. 2012). No systematic bias was demonstrated for peak ε or indices of SR ($P < 0.05$). The ICCs and CoVs for peak ε and peak SR parameters were 0.834 and 0.610 and 7% and 13% respectively.

Data Analysis and Statistics

All the analysis were performed using SPSS, version 20.0, for Windows (SPSS, Chicago, IL, USA) and the critical alpha was set at $p \leq 0.05$. Absolute data were extracted from DICOM file format. Data are presented as mean ± SD, and were analysed between

groups using one-way ANOVA and Bonferoni post hoc test for multiple comparison to estimate differences between groups. A sample size ≥ 15 from each target population (CT, ET and RT) was prospectively determined to achieve 80% statistical power, accommodate data variability in the study population and detect a 3 mm differences in LV chamber dimension between groups.

RESULTS

Demographic characteristics of the study population

The three groups were comparable for body mass, height, BSA and resting blood pressure (Table 1). ET athletes were older than CT as well as having a lower resting HR compared to RT.

Insert Table 1.

2D-Echocardiography: RV structure

RV structural data (absolute and allometrically scaled) are contained in Table 2. Higher absolute values for RVD1, RVD3, RV area (diastolic and systolic) and RV free wall thickness were observed in the ET compared to CT. Absolute RVD1 was also higher in ET than RT. When these variables were scaled allometrically, all remained significant except RVD3 (between ET and CT) and RVD1 (between ET and RT). There were no differences in absolute or scaled RV structural data between RT and CT. Table 3 shows the % of subjects in each group that fulfilled the major and minor ASE Taskforce Criteria for the diagnosis of ARVC.

Insert Table 2 and 3

Global and Regional RV Function

Global RV functional data are presented in Table 4. There was no statistically significant difference ($p<0.05$) in standard RV functional parameters between all groups. Global RV ε, SRS as well as segmental ε did not significantly differ between the three groups. The base-to-apex gradient for ε was not different between groups (Table 5).

Insert Table 4 and 5.

DISCUSSION

The key findings from this study were; 1) evidence that RV chamber and wall thickness were greater in ET compared to CT but with no differences noted between RT and CT, 2) that the scaling or indexation of RV structural data for individual differences in BSA can significantly alter data interpretation, and 3) both global and regional RV function were similar in all groups.

RV Morphology

This study provides additional evidence that physiological remodelling can occur in the RV of ET athletes. Overall, the higher RV diameters and areas in ET athletes support previous studies (Oxborough et al. 2012, D'Andrea et al. 2013). D' Andrea et al (2013) reported an RVD1 (38.1±5.3 [32–45]) mm in ET which compares favourably to the current data (45 ± 5 [39:57]). Cross-sectional athlete data were supported by a prospective study of ET athletes (Baggish et al. 2008), where a progressive increase in training load over 12 weeks resulted in evidence of RV dilatation. A greater RV wall thickness was also observed in the ET compared to CT (Koç et al. 2007, King et al. 2013). The putative mechanism(s)

involved in RV adaptation to ET remain controversial and poorly understood. Haemodynamic theories of adaptation previously applied to the LV have been purported to play a similar role in the RV such that ET activity places a prolonged but intermittent haemodynamic volume overload that initiates structural adaptation to normalise wall stress (Grossman et al. 1975). Recently, La Gerche et al (2011) demonstrated that a disproportionate haemodynamic afterload is placed on the RV, compared with the LV, during intense ET due to a greater relative increase in pulmonary artery systolic pressure (PASP) (LaGerche et al. 2011). This may partially explain the increase RV wall thickness data in the ET athletes.

Of note in the current study was the lack of difference in RV structural data between RT and CT, especially in relation to wall thickness. This lack of adaptation mirrors recent reports (D'Andrea et al. 2013, King et al. 2013, Pagourelias et al. 2013) and athlete-control cross-sectional examination of the LV (Utomi et al. 2014). The lacks of RV adaptation in RT athletes agrees with prospective data collected before and after a progressive increase in RT load (Baggish et al. 2008). Potential reasons for the lack of adaptation in the RV of elite RT athletes may relate to the limited time exposure to any haemodynamic overload, compared to the long-term steady state haemodynamic conditions associated with endurance training and/or the lack of any significant haemodynamic pressure overload stimulus in the presence of a Valsalva manoeuvre (Haykowsky et al. 2001).

The present study also demonstrated that when absolute structural data for the RV are indexed or scaled for individual differences in body size (here BSA) alterations in between group comparisons can occur which fundamentally change data interpretation. Whilst not a consistent affect across all variables, this should be noted carefully as it may well impact clinical data interpretation. The importance of body size on cardiac dimension

has been demonstrated in empirical studies (George et al. 1998, Utomi et al. 2014) and has been highlighted in review articles (Dewey et al. 2008) and a recent meta-analysis (Utomi et al. 2013). The current data add to this knowledge base specifically with respect to the interpretation of RV data in athletes but further work should produce normative data for large athlete groups using this allometric approach.

Global and Regional RV Function

Similar to study by King et al (2013), RV FAC and TAPSE as well as a range of other global parameters were not different between ET, RT and CT. Conversely, Teske et al (2009) reported higher TAPSE in ET (26.0 ± 4.7 mm) than CT (24.1 ± 2.6 mm). Heterogeneity between studies may be due to differences in age, training levels, echocardiographic window and non-uniformity of echo vendor algorithms. As with global systolic function we observed no between group differences in RV diastolic Doppler-flow parameters which confirms some past work (Prakken et al. 2010, Oxborough et al. 2012, Pagourelias et al. 2013) but disagrees with Teske et al (2009). Other studies have suggested some differences between ET and CT (Teske et al. 2009b). Overall there seems to be little evidence to support training induced changes in global RV systolic and diastolic function, including prospective data from Baggish et al (2008), and this would suggest that at rest cardiac function remains normal in athletes.

In addition to global function we observed no between group differences in regional RV function using tissue-Doppler and STE. This confirms previous TDI data in athlete-control comparisons (Pela et al. 2004, Krol et al. 2011, D'Andrea et al. 2013). Of note global and regional longitudinal RV ε and SR were not different between groups and were also not diminished in any athletes meeting the ARVC ASE revised Task Force criteria (Marcus et al.

2010). This observation contrasts the report by Teske et al. (2009) who demonstrated that Ɛ and SR of the RV free wall were reduced in 63 highly trained athletes relative to controls, particularly in the basal segments of athletes with RV dilatation. The authors proposed that this reflected normal physiological adaptation that resulted in a base-to-apex gradient in RV deformation (Teske et al. 2009b). Whilst we observed a base-to-apex gradient in deformation, this was not different between all groups. It is speculated that because RV volume is greatest at the base, a smaller degree of deformation may be required to generate the same stroke volume, if contractility is preserved which consequently produces a base-to-apex gradient (La Gerche et al. 2012). It is also possible that any functional adaptation in the athletic heart is not apparent at rest, but may be noticeable during exercise (Utomi et al. 2014). Further assessment of global and regional RV function at peak exercise could provide further insight to allow for distinction between athletes, control participants and those with RV pathology.

Clinical Implications: The ASE revised criteria for the diagnosis of ARVC

We applied the revised Task force diagnostic criteria for ARVC (Marcus et al. 2010) to RV data in all groups. Major and minor criteria were observed in all groups but generally the higher percentages occurred in ET athletes. The highest prevalence of criteria reached was for RVOT dimension measured in the PLAX, with lower expression in the PSAX suggesting that imaging window should be taken into account in any data interpretation. In 50% of ET, absolute PLAX RVOT fulfilled the major echocardiographic diagnostic criteria (≥ 32 mm) and 16% met the minor criteria (≥ 29 mm but ≤ 32 mm) for ARVC/D, although when this data were scaled (ratio) to BSA the prevalence dropped markedly and again reflects the

importance of scaling RV structural data. These findings are consistent with previous reports by our group (Oxborough et al 2012b) and others (Teske et al. 2009b, Prakken et al. 2010). The important point, from a clinical perspective is that no individual participant met either the major or minor ARVC criteria of poor RV function. Given that in the early concealed phase of ARVC, individuals are often asymptomatic but may nonetheless be at risk of sudden cardiac death (Maron and Pelliccia 2006 , Marcus et al. 2010), continued research in this area is suggested.

Limitations and Future Research

This was a cross-sectional study in small but highly trained cohorts of male Caucasian athletes. Further work should expand the participant base as well as development of a prospective cohort designs. Although STE is independent of angle of isonation, issues with reverberation and drop out artefact can result in low signal-to-noise ratios and uninterpretable data. Future studies should describe the upper limits of atrial structure and function in elite athlete groups as well as develop data on the acute exercise response of cardiac function in the athletic heart.

Conclusion

This study observed increased RV dimensions in elite ET athletes compared to CT with no evidence of physiological RV hypertrophy in RT athletes. Most of the ET met at least one major or minor criteria for ARVC but this data, as well as statistical between group comparisons, were ameliorated by scaling or indexing of absolute data. Global and regional RV function was not different between both athlete group and CT.

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Table 1: Demographic data of male endurance-trained (ET), resistance-trained (RT) and sedentary control (CT) subjects.

Parameter	ET	RT	CT
Sample Size	19	21	21
Age (years)	34 ± 5 [23:41]†	29 ± 8 18:44]	27 ± 8 [20:43]
Body Mass (kg)	74 ± 9 [59:87]	83 ± 14 [61:111]	76 ± 10 [61:91]
Height (m)	1.8 ± 0.1 [1.5:1.9]	1.8 ± 0.1 [1.7:1.9]	1.8 ± 0.1 [1.6:1.9]
BSA (kg/m^2)	2.1 ± 0.2 [1.7:2.4]	2.3 ± 0.3 [1.8:2.9]	2.1 ± 0.2 [1.7:2.5]
Resting Heart Rate (beats/min)	56 ± 11 [40:80]‡	70 ± 11 [56:98]	63 ± 10 [53:84]
Resting Systolic BP (mmHg)	130 ± 10 [110:140]	131 ± 9 [110:141]	128 ± 12 [105:140]
Resting Diastolic BP (mmHg)	78 ± 11 [58:90]	76 ± 7 [59:89]	75 ± 8 [59:89]

Data are mean ± SD with [range].

BSA – Body surface area; † $P<0.05$ versus CT and ‡ $p< 0.05$ versus RT.

Table 2: Absolute and allometrically scaled right ventricular structural data in male endurance-trained (ET), resistance-trained (RT) and sedentary control (CT) subjects.

Parameter	ET	RT	CT
RVOT PLAX (mm)	31 ± 4 [23:37]	29 ± 4 [22:35]	30 ± 4 [23:38]
RVOT PLAX (mm/$[m^2]^{0.5}$)	21 ± 3 [17:26]	20 ± 3 [16:24]	21 ± 2 [17:25]
RVOT1 (mm)	32 ± 5 [24:40]	31 ± 5 [22:42]	32 ± 3 [23:36]
RVOT1 (mm/$[m^2]^{0.5}$)	22 ± 3 [18:27]	21 ± 3 [16:25]	22 ± 3 [17:27]
RVOT2 (mm)	27 ± 5 [22:39]	25 ± 3 [20:32]	26 ± 3 [20:31]
RVOT2 (mm/$[m^2]^{0.5}$)	19 ± 3 [17:20]	17 ± 2 [16:18]	18 ± 2 [17:20]
RVD1 (mm)	45 ± 5 [39:57]†‡	40 ± 5 [32:51]	39 ± 4 [31:45]
RVD1 (mm/$[m^2]^{0.5}$)	31 ± 4 [26:42]†	28 ± 4 [20:33]	27 ± 3 [22:33]
RVD2 (mm)	30 ± 3 [25:35]	30 ± 4 [24:38]	29 ± 3 [20:32]
RVD2 (mm/$[m^2]^{0.5}$)	21 ± 2 [20:22]	20 ± 3 [19:21]	20 ± 2 [19:21]
RVD3 (mm)	88 ± 9 [72:106]†	84 ± 10 [69:102]	81 ± 10 [64:98]
RVD3 (mm/$[m^2]^{0.5}$)	61 ± 5 [58:64]	56 ± 7 [54:59]	56 ± 7 [53:60]
RV diastolic area (cm^2)	27 ± 4 [23:35]†	24 ± 5 [17:36]	22 ± 4 [15:29]
RV diastolic area (cm^2/m^2)	19 ± 3 [16:26]†	16 ± 4 [11:24]	15 ± 3 [10:20]
RV systolic area (cm^2)	14 ± 2 [10:18]†	13 ± 3 [8:18]	11 ± 3 [7:18]
RV Systolic area (cm^2/m^2)	9 ± 2 [7:13]†	8 ± 2 [5:13]	7 ± 2 [5:13]
RV wall thickness (mm)	4 ± 1 [3:5]†	4 ± 1 [3:5]	3 ± 1 [2:4]
RV wall thickness (mm/$[m^2]^{0.5}$)	2.8 ± 0.4 [2.1:3.2]†	2.3 ± 0.4 [1.1:3.1]	2.1 ± 0.5 [1:3]

Data are mean ± SD with [range].

RV-Right ventricle, RVOT PLAX-RV outflow tract dimension parasternal long axis anterior portion, RVOT1-basal parasternal short axis, RVOT2-distal parasternal short axis at pulmonary bifurcation, RVD1-RV basal dimension, RVD2-mid cavity dimension, RVD3-RV longitudinal dimension, RV diastolic area-RV area at end-diastole, † $p<0.05$ versus CT and ‡ $P<0.05$ versus RT.

Table 3: Percentage of participants in each group who meet specific ASE revised task force criteria for diagnosis of ARVC (Marcus et al 2010).

Parameters		**ET**	**RT**	**CT**
Major	PLAX RVOT ≥ 32 mm	50	31	23
	PLAX RVOT/BSA ≥ 19mm	6	0	0
	PSAX RVOT ≥ 36 mm	27	5	28
	PSAX RVOT/BSA ≥ 21 mm/m^2	0	0	0
	FAC ≤ 33%	0	0	0
Minor	PLAX RVOT ≥ 29 mm but < 32 mm	16	23	19
	PLAX RVOT/BSA ≥ 16 but < 19 mm/m^2	28	13	19
	PSAX RVOT ≥ 36 mm but < 36 mm	22	43	24
	PSAX RVOT/BSA ≥ 18 but < 21 mm	28	14	19
	FAC ≤ 40% but > 33%	0	0	0

ASE-American Society of Echocardiography, PSAX-parasternal short axis, BSA-body surface area and FAC-fractional area change.

Table 4: Right ventricular functional data in male endurance-trained (ET), resistance-trained (RT) and sedentary control (CT) subjects.

Parameter	ET	RT	CT
RVFAC (%)	50 ± 10 [40:62]	50 ± 10 [42:60]	50 ± 10 [40:58]
TAPSE	24 ± 3 [21:29]	24 ± 3 [22:26]	25 ± 2 [23:26]
RVOT VTI	18 ± 3 [15:24]	20 ± 2 [19:21]	15 ± 3 [10:20]
RVSV (ml)	104 ± 53 [78:164]	92 ± 33 [62:144]	99 ± 33 [42:149]
RVSV (ml/[m^2]$^{1.5}$)	34 ± 15 [26:63]	29 ± 10 [21:49]	32 ± 11 [22:51]
RVS' cm/s	15 ± 1 [13:17]	15 ± 2 [13:18]	14 ± 2 [11:17]
RVS' ([cm/s]/cm)	1.7 ± 0.3 [1.1:2.3]	1.8 ± 0.3 [1.4:2.2]	1.7 ± 0.3 [1.3:2.3]
RVE' cm/s	15 ± 2 [13:19]	16 ± 3 [14:19]	14 ± 3 [9:17]
RVE' ([cm/s]/cm)	1.7 ± 0.3 [1.2:2.4]	1.7 ± 0.4 [0.5:2.8]	1.7 ± 0.4 [1.0:2.5]
RVA' cm/s	12 ± 2 [10:17]	12 ± 1 [9:14]	12 ± 2 [9:14]
RVA' ([cm/s]/cm)	1.5 ± 0.4 [0.9:2.4]	1.3 ± 0.4 [0.7:2.4]	1.4 ± 0.3 [0.7:2.0]

Data are mean ± SD with [range].

TAPSE-tricuspid annular plane systolic excursion, RVOTVTI-RV out flow tract velocity time integral, RVS- RVS'-RV lateral wall peak systolic tissue velocity, RVE'-RV lateral wall peak early diastolic tissue velocity, RVA'-RV lateral wall peak late diastolic tissue velocity. RVS', RV E' and RVA' were Scaled to RV length (RVS'/RVD3) ([cm/s]/ [cm]).

Table 5: STE-derived RV longitudinal ε and SR.

RV Parameters	ET	RT	CT
Peak global RV ε (%)	-26.9 ± 4.3 [-34.7:-21.3]	-27.4 ± 4.9 [-36.6:-21.8])	-28.4 ± 2.7 [-37.0:-25.0]
Peak basal wall ε (%)	-26.2 ± 3.4 [-32.9:-20.7]	-26.5 ± 3.2 [-32.6:-20.7]	-28.2 ± 3.0 [-34.8:-24.5]
Peak mid wall ε (%)	-26.4 ± 5.2 [-33.2:-20.4]	-27.5 ±2.5 [-33.6:-23.6]	-28.4 ± 4.6 [-37.9:-22.0]
Peak apical wall ε (%)	-30.7 ± 4.1 [-38.8: -23.5]	-31.3 ± 3.3 [-36.6:-26.2]	-32.6 ± 3.0 [-38.8:-23.5]
Base to apex ε gradient (%)	-4.6 ± 2.5 [-9.4: -0.8]	-4.7 ± 2.5 [-8.3:-1.0]	-4.4 ±2.9 [-9.1:-1.9]
Peak global RV SRS (s^{-1})	-1.4 ± 0.2 [-2.0:-1.1]	-1.5 ± 0.3 [-2.1:-1.0]	-1.5 ± 0.2 [-2.0:-1.2]
Peak global RV SRE (s^{-1})	1.2 ± 0.5 [1.1:2.8]	1.8 ± 0.5 [1.2:2.8]	1.9 ± 0.5 [1.2:2.8]
Peak global RV SRA (s^{-1})	1.1 ± 0.2 [0.8:1.6]	1.1 ± 0.3 [0.5:1.6]	1.0 ± 0.3 [0.5:1.4]

Data are mean ± SD with [range].

ε-strain, SRS -strain rates in systole, SRE- strain rate in early diastole, SRA- strain rate in late diastole