# The Relationship between Left Ventricular Structure and Function in the Elite Rugby Football League Athlete as determined by Conventional Echocardiography and Myocardial Strain Imaging

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**Abstract**

**Aims**

The aims of this study were to establish the left ventricular (LV) phenotype in rugby football league (RFL) athletes and to mathematically model the association between LV size, strain (ε) and ejection fraction (EF).

**Methods and Results**

139 male athletes underwent echocardiographic LV evaluation including ε imaging. Non-athletic males were used for comparison. All absolute and scaled structural indices were significantly larger ($P < 0.05$) in athletes with a predominance for normal LV geometry. EF and global ε were similar between groups but strain rates (SR) were significantly lower ($P < 0.05$) in athletes. Lower apical rotation ($P = < 0.001$) and twist ($P = 0.010$) were exhibited in athletes.

**Conclusion**

Normal EF is explained by divergent effects of LV internal diastolic dimension (LVIDd) and mean wall thickness (MWT) on LV function. Reductions in SR and twist may be part of normal physiological LV adaptation in RFL athletes**.**

**Key Words:** Athletes' heart, left ventricle, echocardiography, strain

# 1. Introduction

Athletes' Heart (AH) describes the physiological adaptation from chronic exposure to exercise training[1]. The magnitude and type of adaptation is heterogeneous, being dependent on factors including age, body size, gender, ethnicity, training status and sporting discipline[2]. Recent studies have demonstrated changes in left ventricular (LV) geometry[3,4] alongside functional adaptation[5] across sporting disciplines. Pre-participation cardiac screening (PPS) in Rugby Football League (RFL) is mandatory for all male players competing in the professional RFL Super-League. Although Sudden Cardiac Death (SCD) in an athlete is rare[6], the impact is devastating for the family and the broader sporting community which often results with increased calls for more vigorous screening of athletes[7]. RFL is a high intensity sport[8], defined as moderate static (20-50% maximal voluntary contractions) and moderate dynamic (40-70% maximal oxygen uptake) activity and PPS aims to identify athletes at risk of SCD by detecting previously undiagnosed cardiac conditions. It is appropriate that screening strategies should be tailored to the population being screened[7] and it is therefore pertinent to establish the LV phenotype in RFL athletes. Echocardiography is routinely used in this setting with newer techniques, including strain ($\varepsilon$) and strain rate (SR) imaging now being implemented to describe chamber mechanics[9]. Previous data on LV mechanics is variable due to heterogeneous study design, methods and/or athlete populations with differentiation from inherited conditions often being based on a 'one size fits all' interpretation of echocardiographic derived measures and with little consideration of body size.

The relationships between LV geometry and ejection fraction (EF) have been extensively investigated in pathological hypertrophy[10,11] whilst the association in a physiological model, such as the AH, remains incompletely understood. Since the interrelationship between ventricular wall thickness, cavity dimension and EF is complicated, a better comprehension of the relationship between the thickness of the LV wall, EF and myocardial $\varepsilon$ has been aided using mathematical modelling[10,12]. Using intuition alone to assess the effects of multiple changes in structural and geometric may lead to incorrect interpretations. Mathematical modelling helps as it eliminates confounding factors and quantifies the individual effects of geometric and physiological changes. The understanding provided by modelling studies has now been applied to hypertensive hypertrophic ventricular disease[11]. It has been shown that using mathematical modelling[10] and confirmed observational clinical data, that increasing LV wall thickness and/or myocardial $\varepsilon$ independently leads to increased EF[11]. Similar findings have been seen in hypertrophic cardiomyopathy where the combination of reduced myocardial $\varepsilon$ and increased wall thickness results in a normal or even increased EF[13]. In contrast, athletes tend to have greater wall thickness and dimensions yet have similar EF compared with controls[14].

This study focusses on the LV to provide an in-depth assessment of the structural and functional characteristics of this chamber in the elite RFL athlete to aid PPS and differential diagnosis where the LV is implicated. The primary aims of this study are to (1) establish the LV phenotype in elite male RFL athletes using standard 2D, Doppler, tissue Doppler, $\varepsilon$ and SR speckle tracking echocardiography (STE), and (2) mathematically model the association between LV size, EF and $\varepsilon$ in a physiological model of hypertrophy.

**2. Methods**

**2.1 Study population and design**

Following ethical approval by the ethics committee of Liverpool John Moores University, 139 elite, 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 mandatory PPS. Athletes participated in more than 10 hours structured exercise training per week and healthy controls engaged in less than 3 hours recreational activity per week. Participants completed a medical questionnaire to document any cardiovascular symptoms, family history of SCD or other cardiovascular history and abstained from exercise training or recreational activity for at least 6 hours prior to the investigation. A cross-sectional study was employed and data 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. Further evaluation in cases of suspected pathology provided no evidence of cardiac disease, therefore all participants remained in the study.

**2.2 Procedures**

**2.2.1 Anthropometry**

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 described[15]. Blood Pressure (BP) was assessed with an automated sphygmomanometer (Dinamap 300, GE Medical systems, USA).

**2.2.2 Conventional 2D Echocardiography**

All echocardiographic images were acquired using a commercially available ultrasound system (Vivid Q, GE Medical, Horten, Norway) with a 1.5-4 MHz phased array transducer. Two experienced sonographers acquired the images with the participant lying in the left lateral decubitas position in adherence to American Society of Echocardiography (ASE) guidelines[16]. 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[16,17].

The internal LV cavity dimension was measured at end diastole (LVIDd) and end systole (LVISd) and its length calculated (LV length) from base to apex. LV end diastolic volume (LVEDV), LV end systolic volume (LVESV), stroke volume (SV) and EF were calculated using the Simpson's Biplane summation of discs method. In addition, a comprehensive assessment of LV wall thickness was employed. Essentially, four linear measurements (infero-septum, antero-septum, posterior wall and lateral wall) were made at both the basal and mid-levels in the parasternal short axis at end diastole[18]. The mean wall thickness (MWT) was calculated from the average of the 8 segments. The maximum wall thickness was also determined and relative wall thickness (RWT) was calculated to include the anterior septum (basal antero-septal and posterior wall thicknesses measured in diastole and dividing by LVIDd). LV mass was determined using the ASE corrected equation and a description of

LV geometry was provided based on a combination of LV mass index and RWT[16]. All structural indices were scaled allometrically to BSA based on the principle of geometric similarity[19]. Linear dimensions were scaled to $BSA^{0.5}$, areas directly to BSA and volumes to $BSA^{1.5}$ and LV mass scaled to $height^{2.7}$ [20] and BSA [16]. Transmitral Doppler allowed the assessment of early (E) and late (A) diastolic velocities and the ratio was calculated (E/A). TDI at the septum and lateral walls provided regional and average peak early (E'), late diastolic (A') and systolic S' myocardial velocities. To account for chamber size, average values were indexed for LV length (S' index, E' index and A' index) as previously recommended[21].

### 2.2.3 Myocardial ε Imaging - STE

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

LV Longitudinal ε and SR were assessed from the apical four-chamber, three-chamber and two- chamber images allowing for assessment of both regional and global values. Each apical image provided 6 segments (basal, mid and apical segments of each wall) from which longitudinal ε, time to peak ε, systolic strain rate (SRS), early diastolic strain rate (SRE) and late diastolic strain rate (SRA) were assessed. All regional values were recorded (Supplementary Material Figure S1a) and an average value of 18 segments was presented as a global parameter of LV longitudinal function.

LV radial and circumferential ε and SR were assessed from the LV parasternal short axis image at both basal and mid-levels. Both views provided 6 myocardial segments from which peak circumferential and radial ε, time to peak ε, SRS, SRE and SRA were assessed. This allowed regional circumferential and radial ε and SR to be recorded from 12 segments (Supplementary Material Figure S1b) and an average was calculated to provide global circumferential and radial ε and SR. LV basal and apical rotation were assessed from the basal and an apical parasternal image and twist was calculated as the net difference between peak basal and peak apical rotation[22]. Regional data across all the myocardial segments was assessed for variability by calculating the standard deviation (SD) of the 18 longitudinal segments and the 12 circumferential/radial segments.

### 2.2.4 Mathematical Model

In order to calculate the independent effects of LV cavity size, mural thickness and contractile ε on EF, a mathematical model of LV contraction was used as previously described[10,12]. The mathematical model has recently been validated using echocardiography[23]. The LV geometry was modelled using a two-layer with an ellipsoidal (prolate spheroidal) shape. The total mid-wall volume (intra-ventricular volume plus inner shell volume) was obtained and the volumes of the outer and inner shells were then calculated. The diastolic external and internal ventricular volumes were then obtained using the area-length method[24], and the total myocardial volume derived from the difference. The mid-wall short-axis diameter and LV length were reduced, so that myocardial longitudinal ε and mid-wall circumferential ε were the same, to simulate systole and the new mid-wall volume was derived. Myocardial volume was assumed to be conserved therefore allowing the internal end-systolic volume to be calculated by subtracting the total muscle volume from the

external end-systolic volume. The end-diastolic LV length was held constant and the end-diastolic MWT, end-diastolic diameter and myocardial ε were adjusted to include the range found in both the athletes and control groups. The systolic and diastolic left ventricular volumes were calculated as described above and EF calculated.

### 2.2.5 Statistical Analysis

Study data were collected and managed using REDCAP electronic data capture tools hosted at Liverpool John Moores University[25]. All echocardiographic data are presented as mean ± SD and ranges. Statistical analyses were performed using a commercially available software package (SPSS, Version 23.0 for Windows, Illinois, USA). Variables were analysed between athletes and controls using independent T-tests with a P value of <0.05 considered statistically significant.

Where significant differences in global ε, SR and TDI between groups were found, a bivariate Pearson's correlation was performed against appropriate structural measures and heart rate (HR). Where significant correlations were found multi–linear regression was undertaken to determine the relative contribution of each parameter on the dependent variable.

### 3. Results

Athletes were significantly older (P=0.001) than controls (24±4 and 22±3 years). Height (1.82±0.06 and 1.78±0.06 m), weight (96±11 and 78±9 kg) and BSA (2.20±0.15 and 1.96±0.13 kg/m$^2$) were all significantly (P<0.001) higher in the athlete group whilst HR was

significantly (P<0.001) lower in the athlete group (56±10 and 69±9 beats.min$^{-1}$). Blood pressure (BP) was 131/69 mmHg and 129/74 mmHg in the athlete and control groups respectively. There was no significant difference in systolic BP between groups but diastolic BP was significantly lower in athletes (P<0.001).

Conventional LV structural and functional indices are presented in table 1. All absolute and scaled LV structural indices were significantly larger (P<0.05) in the athlete compared to the control group. RWT was not significantly different between groups. LV geometry was assessed in all participants highlighting a predominance for normal geometry with 1.4 % and 0.7% of athletes having eccentric hypertrophy and concentric remodelling respectively. None of the athletes exhibited concentric hypertrophy. The entire control group presented with normal geometry (Supplementary Material Figure S2).

There was no significant difference in EF or septal S' between groups. However lateral S' and average S' were significantly lower in the athlete group (P<0.001 and =0.001 respectively). E wave velocity was similar between groups but A velocity was significantly lower (P<0.001) in athletes resulting in a higher E/A ratio (P=0.002). Septal E', A' and lateral A' were significantly lower in the athlete group (P=0.027, 0.003 and 0.016 respectively) and hence average E' and A' were also significantly lower (P=0.028 and 0.020). Indexed S', E' and A' were significantly lower (P<0.001) in the athlete group.

**Insert Table 1**

Global LV ε, SR and twist data are presented in table 2. There was no statistically significant difference between groups for global longitudinal, circumferential or radial peak ε. The respective time to peak ε (P<0.001) and was significantly increased in the athlete group across all planes of contraction. Longitudinal SRS, SRE, SRA (P=0.01, <0.001 and 0.011 respectively), circumferential SRS, SRE, SRA (P=0.08, <0.001 and 0.023 respectively) and radial SRS, SRE, SRA (P =<0.001, <0.001 and 0.019 respectively) were lower in the athlete group. Significant differences between groups were observed for LV rotational parameters with higher basal rotation (P=0.030), lower apical rotation (P<0.001) and lower twist (P = 0.010) exhibited in the athlete group compared to the control group.

**Insert Table 2**

There were significant correlations between HR, MWT, LVIDd, LV length and global SR parameters across both groups (Supplementary Material, Table S1). Increased HR correlated with higher SR, whilst increased structural indices correlated with lower SR's. Following multi-linear regression, HR (β=-0.003, P<0.001) **and** MWT (β=0.020, P=0.039) accounted for 16% of the variance in longitudinal SRS. HR (β=0.007, P=0.001) **and** MWT (β=-0.061, P=0.033) accounted for 11% of the variance in circumferential SRE, whilst HR (β=-0.013, P<0.001) **and** MWT (β=0.120, P=0.006) also accounted for 15% of the variance in radial SRE. HR (β =0.011, P<0.001) **and** LVIDd (β=-0.019, P = 0.001) accounted for 25% of the variance in radial SRS and HR (β=0.003, P=0.001) **and** LV length (β=-0.003, P=0.024) accounted for 15% of the variance in longitudinal SRA. MWT (β=-0.099, P<0.001) was a significant independent contributor to longitudinal SRE and apical rotation accounting for 19% and 10% of the variance respectively. MWT is also independently correlated to LV twist

(R=-0.170, P=0.021). HR also correlated with medial, lateral and average A' (R=0.311, P <0.001, R=0.349, P<0.001and R=0.390, P<0.001). There was no correlation between HR and TDI medial, lateral or average E'.

Regional LV longitudinal, circumferential and radial ε and SR data is presented in figure 1 (Supplementary Material, Tables S2-S4). Regional heterogeneity was most prominent within longitudinal SRS (P=0.049), circumferential SRE (P=0.008), circumferential SRA (P=0.011), radial SRS (P=0.009) and radial SRE (P=0.049).

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

The mathematical model demonstrated that increasing MWT from 7 to 18 mm predicted an increase in EF (Figure 2). Improving myocardial ε from -15 % to -19 % also predicted an increasing EF. As LVIDd was increased from 40 to 60 mm, however, the EF decreased. Furthermore, the combination of an increase in MWT combined with an elevated EDV, as seen in the athletes, led to a normalisation of EF.

**Insert Figure 2**

**4. Discussion**

The main findings of this study are: (1) Absolute and scaled values for LV chamber size and wall thickness are increased in RFL athletes whilst indexed TDI, SR, apical rotation and twist are lower in RFL athletes compared to sedentary controls, (2) EF is maintained which is likely due to the interaction of divergent effects of LVIDd and MWT on LV function.

Absolute and indexed LV structural parameters are increased in elite RFL athletes consistent with previous studies[2,4]. Utomi *et al* [14] described a predominance of normal LV geometry in both endurance and resistance trained athletes, a pattern seen in this study of RFL athletes who were engaged in structured training and competition and had a history of long-term chronic exposure to training. None of the athletes exhibited concentric LVH in contrast to a study by Finocchiaro *et al* [5] who reported that 12% of male athletes demonstrated concentric remodelling/LVH, rising to 15 % for males competing in dynamic sports. The natural progression of LV geometric changes are not completely understood within populations[26] however studies have shown that abnormal LV geometry can be detrimental and has been associated with increased morbidity and mortality risk[27] thereby supporting the inclusion of LV geometry assessment in athlete echocardiographic screening.

No significant differences in longitudinal, circumferential and radial ε were observed between groups similar to previous findings[9]. Previously, athletes with the most marked LV remodelling were found to have similar longitudinal ε patterns as those with normal LV dimensions[28] and in groups of untrained subjects assigned to either endurance or resistance training LV longitudinal ε did not change despite changes in LV mass and volumes[29]. During an 18 week intensive training programme in competitive athletes engaged in team sports, there was an increase in global longitudinal ε with an increase in LV cavity size, suggesting a

reduction in longitudinal ε is not associated with physiological adaptation[30]. Our data would suggest that a reduction in global longitudinal, circumferential and radial ε is not a normal, physiological training adaptation. Lower SR was observed in RFL athletes and has been observed previously in athletes'[28]. Regional heterogeneity was observed for both ε and SR, the latter demonstrating most variation, both within and between groups which suggests this may be a normal finding in adults possibly due to regional curvature and myocardial architecture differences[31] and/or a non-uniform contractile stress across the LV[13]. The decreased regional SR in athletes may be a normal physiological adaptation to exercise and likely reflects a combination of lower HR and larger LV dimensions. Speculatively, with increased MWT, the LV may reach the same required deformation or EF at a slower rate due to an increased number of myofibrils, or in other words, a similar wall tension and intraventricular pressure can be generated or released at a slower speed. An increase in MWT and a reduced contractile stress may result in the same contractile force[32].

Twist contributes to LV function by storing additional potential energy which is released to increase early diastolic suction, with the recoil inducing a rapid reduction of LV pressure leading to early diastolic filling[33]. Weiner *et al* [34] have previously highlighted that apical rotation is the primary determinant of peak systolic LV torsion. In the current study increased basal rotation and decreased apical rotation and twist in the athlete group is in part related to increased MWT and we can speculate that there may be some reduction in mechanical function or more simply this may be an adaptive training response to create a 'reserve' for the onset of exercise as previously suggested[35]. Zocalo *et al* [36] reported reduced twist in soccer players and Nottin *et al* [37] reported reduced twist in elite cyclists mainly driven by a reduction in apical rotation. Stöhr *et al* [38] also reported significantly lower LV apical rotation at rest and during submaximal exercise in individuals with high aerobic fitness, however this

could not be explained by LV wall thickness or HR. A phasic response to cardiac remodelling has been reported in competitive rowers where in the acute phase of exercise training (90 days) an increase in apical rotation and twist was reported; however follow up at 39 months following the chronic phase of adaptation revealed a regression in both apical rotation and twist[39]. It is possible that reduced apical rotation and twist is a normal physiological response to chronic exercise training. The LV base rotates in the opposite direction to that of the apex and is significantly lower in magnitude[40] with net twist explained on the basis of varying spiral myofibre architecture of these regions[40,41]. With high aerobic fitness, it has been previously speculated that lower apical rotation may be due to a change in LV microstructure with subsequent rearrangement of LV myofibres[38].

All participants in the current study exhibited normal indices of diastolic function. Indexed and absolute diastolic TDI measures were significantly lower in the RFL athletes compared to controls and were associated with a significantly increased, but normal E/A mitral inflow ratio. Importantly, unlike A', there was a lack of correlation between E' and HR demonstrating that a faster HR in the control population is not responsible for the differences observed. These data may be reflective of differences in cardiac mechanics between the two groups, in particular reduced apical rotation and twist. A reduction in LV twist would impact the subsequent diastolic recoil, which has implications for diastolic filling[42] and may help to explain the reduction in TDI.

LV remodelling in RFL athletes allows for preservation of EF within normal range possibly through an adaptive process involving a balance between the breakdown and rebuilding of myocardial tissue[43]. Longitudinal $\varepsilon$ is similar between groups but in the presence of a

significantly increased wall thickness, cavity size, and therefore, LV mass. No differences in EF between groups suggests a relationship exists between increased LVIDd and increased MWT to normalise EF for any given ε. EF is one of the most commonly used parameters to describe LV systolic function during serial athlete cardiac assessments. Our results are in agreement with Baggish *et al* [6] who concluded that EF alone was unable to account for geometric and functional changes, with lack of sensitivity to track LV function in the presence of significant changes in LV architecture.

**5. Limitations**

From this cross sectional study we cannot determine the timing of exercise induced changes in LV structure and function. The athletes were selected according to sporting discipline and whilst physiological adaptation of the nature observed in RFL athletes is likely similar to athletes of other sports of this type, further application of the model is warranted in athletes involved in a range of sporting disciplines. Genetic factors and seasonal variation should also be considered during cardiac evaluation.

**6. Conclusion**

Despite an increased LV size, there is a predominance for normal LV geometry in RFL athletes, who undertake mixed resistance and endurance based training. Despite normal EF and global ε, global SR is lower and there is significant regional ε and SR heterogeneity compared to controls. Apical rotation and twist are also significantly lower in and it is likely

that lower SR and twist mechanics are part of the normal physiological cardiac adaptation in RFL athletes. Normal EF and therefore ε, observed in these athletes, is explained by the increase in both MWT and LVIDd. This study suggests that the utilisation of myocardial mechanics in addition to standard functional indices may aid differential diagnosis during PPS. A normal or abnormal STE assessment in those RFL athletes presenting with standard LV parameters at or above/below the physiological limits or ranges considered normal for those parameters is likely to aid differential diagnosis.

**Supplementary Material**

Figures S1-S2 and Tables S3-S6

**Conflict of Interest**

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

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**Figure Legends**

Figure 1: Regional ε and SR in RFL athletes and controls (ε = strain; SR = strain rate; SRS = systolic strain rate; SRE = early diastolic strain rate; SRA = late diastolic strain rate).

a: Regional longitudinal, circumferential and radial ε

b: Regional longitudinal, circumferential and radial SRS

c: Regional longitudinal, circumferential and radial SRE

d: Regional longitudinal, circumferential and radial SRA

Figure 2: Mathematical modelling of left ventricular contraction. As ε decreases, ejection fraction decreases. The opposing effects of increased MWT and increased LVIDd results in a normalisation of ejection fraction. (MWT = mean wall thickness; LVIDd = left ventricular internal diastolic dimension; ε = strain).

Table 1: Echocardiographic parameters of the left ventricle

| | Athlete Mean±SD (Range) | Control Mean±SD (Range) | P value |
|---|---|---|---|
| LVIDd (mm) | 56±4 (47-63) | 50±4 (40-56) | <0.001* |
| LVIDd index (mm/(m$^2$)$^{0.5}$) | 37±2 (31-43) | 35±3 (30-40) | <0.001* |
| LVIDs (mm) | 38±3 (28-48) | 34±3 (28-40) | <0.001* |
| LVIDs index (mm/(m$^2$)$^{0.5}$) | 26±2 (19-31) | 25±2 (20-29) | 0.017* |
| Mean Wall Thickness (mm) | 9±1 (7-11) | 8±1 (6-9) | <0.001* |
| Maximum wall thickness (mm) | 10±1 (8-12) | 8±1 (7-10) | <0.001* |
| Relative Wall Thickness | 0.33±0.04 (0.24-0.42) | 0.32±0.04 (0.25-0.41) | 0.205 |
| LV Mass (g) | 191±31 (112-279) | 132±24 (81-187) | <0.001* |
| LV Mass index (g/(m$^2$)$^{2.7}$) | 38±7 (24-63) | 28±6 (15-39) | <0.001* |
| LV mass index (g/m2) | 87±13 (55-128) | 67±11 (42-86) | <0.001* |
| LV Length (mm) | 97±5 (84-111) | 87±6 (70-99) | <0.001* |
| LVEDV (ml) | 157±25 (105-228) | 105±20 (55-148) | <0.001* |
| LVEDV (ml/(m$^2$)$^{1.5}$)) | 48±7 (33-65) | 38±8 (22-51) | <0.001* |
| LVESV (ml) | 65±13 (40-108) | 43±9 (24-59) | <0.001* |
| LVESV (ml/(m$^2$)$^{1.5}$) | 20±4 (13-30) | 16±4 (9-23) | <0.001* |
| Stroke Volume (ml) | 92±16 (60-136) | 62±12 (30-90) | <0.001* |
| Ejection Fraction (%) | 59±4 (48-70) | 59±3 (54-68) | 0.466 |
| Transmitral E Velocity (m/s) | 0.79±0.15 (0.47-1.15) | 0.82±0.15 (0.49-1.19) | 0.307 |
| Transmitral A Velocity (m/s) | 0.41±0.10 (0.24-0.69) | 0.49±0.10 (0.31-0.81) | <0.001* |
| Transmitral E:A Ratio | 2.01±0.54 (0.84-3.83) | 1.75±0.47 (0.78-2.91) | 0.002* |
| Medial S' (cm/s) | 9±1 (8-13) | 9±1 (7-13) | 0.228 |
| Medial E' (cm/s) | 13±2 (9-18) | 13±3 (9-21) | 0.027* |
| Medial A' | 7±2 | 8±2 | 0.003* |

| | | | |
|---|---|---|---|
| **(cm/s)** | (4-12) | (5-12) | |
| **Lateral S'** | 11±2 | 13±3 | <0.001* |
| **(cm/s)** | (8-18) | (7-19) | |
| **Lateral E'** | 18±3 | 19±4 | 0.084 |
| **(cm/s)** | (11-27) | (8-28) | |
| **Lateral A'** | 7±2 | 8±2 | 0.016* |
| **(cm/s)** | (3-13) | (3-16) | |
| **Average S'** | 10±1 | 11±2 | 0.001* |
| **(cm/s)** | (8-15) | (8-16) | |
| **Average E'** | 16±2 | 16±3 | 0.028* |
| **(cm/s)** | (11-21) | (9-24) | |
| **Average A'** | 7±2 | 8±2 | 0.02* |
| **(cm/s)** | (4-12) | (6-11) | |
| **Average S'** | 1.06±0.15 | 1.28±0.24 | <0.001* |
| **index** | (0.73-1.61) | (0.87-1.79) | |
| **((cm/s)/cm)** | | | |
| **Average E'** | 1.61±0.24 | 1.89±0.33 | <0.001* |
| **Index** | (1.09-2.41) | (1.18-2.73) | |
| **((cm/s)/cm)** | | | |
| **Average A'** | 0.72±0.17 | 0.90±0.19 | <0.001* |
| **index** | (0.40-1.32) | (0.57-1.43) | |
| **((cm/s)/cm)** | | | |
| **Average E/E'** | 5.14±0.96 | 5.07±1.01 | 0.649 |
| | (3.03-9.33) | (3.29-7.50) | |

SD = standard deviation; * = Statistically significant (P < 0.05); LVIDd = left ventricular internal diastolic dimension; LVIDs = left ventricular internal systolic dimension; LVEDV = left ventricular end diastolic volume; LVESV = left ventricular end systolic volume; E = early diastolic velocity; A = late diastolic velocity; S' = systolic myocardial velocity; E' = early diastolic myocardial velocity; A' = late diastolic myocardial velocity.

Table 2:  Global Left ventricular ε, SR and Twist

| | Athlete mean ± SD (Range) | Control mean ± SD (Range) | P Value |
|---|---|---|---|
| **LV Longitudinal** | | | |
| **Global ε (%)** | -19.8±1.9 (-15.5- -24.5) | -19.4±1.8 (-15.8- -25.0) | 0.240 |
| **Time to Peak ε (s)** | 0.37±0.03 (0.30-0.44) | 0.35±0.03 (0.27-0.43) | <0.001* |
| **SRS (s⁻¹)** | -0.96±0.10 (-0.72- -1.31) | -1.02±0.15 (-0.81- - 1.41) | 0.01* |
| **SRE (s⁻¹)** | 1.41±0.23 (0.75-2.00) | 1.56±0.24 (1.02-2.15) | <0.001* |
| **SRA (s⁻¹)** | 0.61±0.13 (0.28-1.00) | 0.66±0.13 (0.40-0.99) | 0.011* |
| **LV Circumferential** | | | |
| **Global ε (%)** | -18.7±2.5 (-12.6- -24.9) | -19±2.4 (-13.9 - -25.0) | 0.458 |
| **Time to Peak ε (s)** | 0.37±0.03 (0.28-0.45) | 0.35±0.03 (0.28-0.43) | <0.001* |
| **SRS (s⁻¹)** | -1.06±0.15 (-0.72- -1.60) | -1.14±0.22 (-0.80- -1.72) | 0.008* |
| **SRE (s⁻¹)** | 1.51±0.33 (0.77-2.59) | 1.72±0.32 (1.09-2.54) | <0.001* |
| **SRA (s⁻¹)** | 0.42±0.13 (0.21-0.84) | 0.47±0.17 (0.22-1.11) | 0.023* |
| **LV Radial** | | | |
| **Global ε (%)** | 46.8±11.2 (25.1-72.7) | 50.1±9.0 (32.3-68.3) | 0.059 |
| **Time to Peak ε (s)** | 0.41±0.04 (0.26-0.52) | 0.38±0.04 (0.29-0.50) | <0.001* |
| **SRS (s⁻¹)** | 1.57±0.28 (1.03-2.38) | 1.90±0.43 (1.16-3.12) | <0.001* |
| **SRE (s⁻¹)** | -1.94±0.44 (-1.08- -4.08) | -2.39±0.61 (-1.59- -4.26) | <0.001* |
| **SRA (s⁻¹)** | -0.95±0.39 (-0.31- -2.76) | -1.12±0.54 (-0.30- -2.47) | 0.019* |
| **LV Rotation** | | | |
| **Basal rotation (º)** | -6.23±2.94 (-11.97-0) | -5.21±2.47 (-11.19-0) | 0.030* |
| **Apical rotation (º)** | 8.22±3.86 (0.87-22.75) | 11.22±4.59 (1.51-22.66) | <0.001* |
| **Twist (º)** | 14.0±4.7 | 16.1±4.9 | 0.010* |

| (3.0-28.1) | (6.9-26.5) |
| --- | --- |

SD = standard deviation; * = Statistically significant (P < 0.05); ε = strain; SRS = systolic strain rate; SRE = early diastolic strain rate; SRA = late diastolic strain rate.