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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.

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 (PCS) 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, with moderate static (10–20%) and moderate dynamic (50–75%) components [8] and PCS 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 (ε) 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 ε has been aided using mathematical modelling [10,12]. Using intuition alone to assess the effects of multiple changes in structure and geometry 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

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myocardial ε independently leads to increased EF [11]. Similar findings have been seen in hypertrophic cardiomyopathy where the combination of reduced myocardial ε 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 PCS 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, ε and SR speckle tracking echocardiography (STE), and (2) mathematically model the association between LV size, EF and ε 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 PCS. Athletes participated in >10 h structured exercise training per week and healthy controls engaged in <3 h 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 h 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 Fig. 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 Fig. 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 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 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 Fig. 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