Analysis of Regional Right Ventricular Function by Tissue Doppler Imaging and Three-Dimensional Echocardiography in Highly Trained Athletes

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Abstract

Background: Regional right ventricular (RV) function has not yet been characterized in highly trained athletes, and the effects of increased RV volumes on resting changes of regional RV deformation are unknown. Purpose: The aim of the study was to analyze global and regional RV function by a multisegemental approach using tissue Doppler imaging (TDI) and to determine whether higher RV volumes evaluated by three-dimensional echocardiography (3DE) had an impact on the RV mechanics assessed by resting regional TDI parameters. Methods: We enrolled prospectively 25 professional soccer players and 25 age- and sex-matched nonathletic controls. Transthoracic echocardiography including additional views of the RV was performed. The TDI sample volume was placed in the basal region of the anterior, inferior, and RV free wall to assess the following parameters: isovolumic contraction time (IVCT\textsubscript{RV}), isovolumic relaxation time (IVRT\textsubscript{RV}), ejection time (ET\textsubscript{RV}), and myocardial performance index (MPI\textsubscript{RV}). Furthermore, conventional left ventricular (LV) and RV parameters at two-dimensional (2D) and 3DE were determined. Results: In athletes, LV mass index/body surface area (BSA), left atrial volume index, 2D LV volumes/BSA were significantly increased in comparison with controls. Moreover, athletes had higher 2D LV and RV stroke volume (SV), lower values for A wave and E/e’ ratio, higher basal RV diameter, and right atrial area ("P < 0.0001). Moreover, athletes showed significantly increased LV and RV volumes and SV indexed for BSA ("P < 0.0001) evaluated at 3DE. In athletes, ET\textsubscript{RV anterior}, ET\textsubscript{RV inferior}, IVCT\textsubscript{RV anterior}, IVCT\textsubscript{RV inferior} and IVRT\textsubscript{RV anterior} were statistically increased ("P < 0.0001). Conversely, IVRT\textsubscript{RV inferior} was reduced in comparison with controls. A significant positive correlation between IVRT\textsubscript{RV anterior} and three-dimensional (3D) RV end-diastolic volume (EDV), end-systolic volume, and SV was observed in athletes. Finally, a good positive correlation was observed between 3D RV EDV and 3D LV SV indexed for BSA. Conclusions: In athletes, the higher 3D RV volumes are proportionally related to an increase of IVRT\textsubscript{RV anterior} and 3D LV SV. Further studies on the resting changes of regional RV deformation for screening and follow-up in these participants are needed.

Keywords: Athletes, right ventricular function, three-dimensional echocardiography, tissue Doppler imaging

INTRODUCTION

Intensive exercise in competitive highly trained athletes induces morphological and functional changes both of the left and right sides of the heart in terms of dimensions and wall thickness associated with high hemodynamic load.\[1\]

Whereas a great amount of studies has been published on left ventricular (LV) remodeling induced by intensive training, little is still known about right ventricular (RV) morphology and function of the “athlete’s heart.” This is due to the fact that for many decades, the right ventricle has been neglected, but also because the characterization of RV structure and mechanics is often challenging.

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and intensity of training in addition to individual factors are determinant factors in RV remodeling.\[^{[3]}\]

It has been described that RV is disproportionately affected compared with LV if exposed to repeated bouts of intensive and prolonged exercise. Healthy training results in physiological adaptation in order to better tolerate exercise loads. Conversely, excessive training, especially in genetically predisposed individuals, may lead to injury and proarrhythmic remodeling.\[^{[4]}\] Particularly in athletes, the RV should normally be well investigated and special attention should be devoted to professional athletes or in cases of clinically suspected RV diseases.

A crucial point is that chronic cardiac remodeling is expected in elite athletes, but the differentiation between “healthy” athletic RV remodeling and pathological changes that predispose to an increased risk of sudden cardiac death (SCD) may be extremely difficult.\[^{[5]}\]

Echocardiography plays an important role in RV assessment, and the addition of noninvasive imaging techniques such as tissue Doppler imaging (TDI) and three-dimensional echocardiography (3DE) may represent an important tool for the evaluation of global and regional indices of RV mechanics at rest and for the differentiation of physiological from pathological RV adaptation in athletes.

The methodological problems of RV analysis at echocardiography are mainly related to the peculiar RV architecture. The pyramidal shape of the RV makes it difficult to standardize RV inflow tract (RVIT) for fractional area change measurements. Furthermore, lung artifacts, especially in the ventral RV regions, limit in some cases the complete visualization of RV walls, three-dimensional (3D) RV volume acquisition, and speckle-tracking analysis, particularly in these regions.

Tissue Doppler is a robust technique for analyzing myocardial velocities. Especially for RV relaxation times analysis, high-frame-rate color-coded TDI Doppler sequences and TDI PW Doppler with high temporal resolution are suitable for RV deformation analysis.

Döbel et al. have proposed an extended standardized RV imaging protocol that includes the documentation of the anterior, lateral, and inferior RV wall, by additional apical RV views by tilting the apical LV long-axis view as well as the apical two-chamber view, and TDI analysis of regional myocardial RV velocities in three points at the basal free RV wall.

The object was to focus the attention on global and regional RV function and on the assessment of RV volume changes in athletes for screening and follow-up investigations.\[^{[6]}\]

To date, regional RV function has not yet been well characterized in highly trained athletes, and the effects of increased RV volumes on resting changes of regional RV deformation are unknown.

The aim of the study was to analyze global and regional RV function in professional athletes by a multisegmental approach using TDI and to determine the impact of higher RV volumes evaluated by 3DE on resting regional TDI parameters.

### Methods

#### Study population

Fifty individuals admitted to the echocardiography laboratory of Universitätsklinikum in Leipzig (Germany), without evidence of cardiovascular diseases and/or risk factors, with completely normal clinical examination and normal structural and functional findings on ECG and echocardiography were prospectively enrolled. The study population was divided into two groups: Group I \((n = 25)\) included highly trained top-level professional athletes and Group II \((n = 25)\) – age- and sex-matched nonathletic controls. All athletes were members of the “Bundesliga” soccer team of Leipzig, had trained intensively 15–20 h/week for more than 4 years, and were closely monitored during periodic screening visits. All denied symptoms, family history of SCD, and the use of illicit abuse substances were never tested positive for doping. All measurements were performed during the in-season competition phase and at least 24 h after the last athletic training. All participants were in sinus rhythm, and none received any medication. The following exclusion criteria were considered: poor image quality of echocardiographic examination, incomplete echocardiographic data sets, frequent supraventricular or ventricular extrasystoles, right bundle branch block and left bundle branch block, arrhythmias or other electrocardiographic conduction abnormalities, cardiomyopathies, congenital or acquired coronary artery diseases that had an impact on LV and RV ventricular function, concomitant moderate or severe valvular defects, lung disease or pulmonary hypertension, pericardial effusion, use of anabolic steroids, or other illicit drugs. However, none of the athletes were excluded due to arrhythmia or structural heart diseases.

#### Echocardiography

Standardized transthoracic echocardiography (TTE) and TDI were performed according to the current guidelines of the American Society of Echocardiography and European Society of Cardiology/European Association of Cardiovascular Imaging.\[^{[7]}\] All TTE examinations were performed with a Vivid E95 system with a MSS phased array probe (GE Healthcare Vingmed Ultrasound AS, Horten, Norway), and all parameters were analyzed offline by two expert operators using the EchoPac software (version 12.0.1; GE Healthcare Vingmed Ultrasound AS, Horten, Norway). M-mode echocardiography was used to determine LV diameters and LV wall thickness. LV mass was indexed for body surface area (BSA) and measured as LV mass \((g) = 0.8 \times (1.04 \times ([LVIDd + IVSd + LVPWd]^{3} – LVIDd^{3}) + 0.6)\), where LVIDd is LV end-diastolic diameter, IVSd is interventricular septum thickness at end-diastole, and LVPWd is posterior wall thickness at end-diastole. LV systolic function was reported by LV volumes and LV EF analysis applying the
modified Simpson’s rule on the apical two- and four-chamber views. Left atrial (LA) volume was assessed by biplane LA planimetry in the apical two- and four-chamber view. LV diastolic function was evaluated by the peak E-wave velocity, peak A-wave velocity, mitral valve deceleration time, and E/e’ ratio. To assess early diastolic filling (E), the pulsed-wave (PW) Doppler sample volume was positioned in the apical long-axis view at the tip of the tenting area of the mitral valve. The mean e’ was assessed in the basal inferoseptal and lateral LV region in the apical four-chamber view using TDI.[8] LV ventricular outflow tract (LVOT) diameter was measured in the long-axis view in systole, and LVOT velocities were assessed by PW Doppler echocardiography. LV stroke volume (SV) was calculated as the product of the LVOT area and outflow tract time-velocity integral.

RV ventricular outflow tract (RVOT) diameter was assessed at nearest to the region of the pulmonary valve in the parasternal short-axis view and RV SV calculated as the product of the RVOT area and RV outflow tract time-velocity integral. By integration of 3D analysis of the LV and RV, the comparison of LV and RV volume analysis was used as a plausibility check of the respective measurements [Figure 1].

The echocardiographic transducer was adjusted to the level of the RV chamber, with the goal of optimizing the RV chamber size. The following RV parameters were assessed: basal and mid-RV diameters in the apical four-chamber view at the end-diastole, tricuspid annular plane systolic excursion (TAPSE), systolic pulmonary artery pressure (sPAP), and right atrium (RA) area in the apical four-chamber view. Beyond conventional standardized RV views, reported by Rudski et al.,[10] additional RV views were recorded that permitted a regional assessment of the different RV walls. The RVIT was centralized within the sector by tilting the apical long-axis view of the LV into the RVIT to document the anterior RV regions. By 60° clockwise rotation, the inferior RV regions were documented, followed by a documentation of the free RV wall in the conventional four-chamber view, which is often labeled as lateral RV wall [Figure 2]. Using PW Doppler TDI, Doppler sample volume was placed at the anterior, inferior, and lateral RV myocardium near the tricuspid annulus.[11] RV isovolumic contraction time (IVCT) and relaxation time (IVRT) and RV ejection time (ET) were assessed from each region (lateral, anterior, and inferior). RV myocardial performance index (MPI) was calculated by the following: \( \frac{IVCT_{RV} + IVRT_{RV}}{ET_{RV}} \).[12]

Finally, in all participants, 3DE was performed and RV and LV full-volume data sets were acquired using the 4V probe.

Figure 1: Verifiable documentation in assessing LV and RV volumes. The figures in the top line illustrate the LV SV assessment by Doppler echocardiography resulting in a LV SV of 92 ml. The figures in the midline illustrate the conventional biplane LV volume assessment resulting in a LV EDV of 156 ml and a LV SV of 90 ml. The figures in the bottom line illustrate the three-dimensional volume assessment of the LV and RV resulting in a LV and RV EDV of 152 and 213 ml as well as in a LV and RV SV of 94 and 98 ml, respectively. The comparison of LV and RV volume analysis confirmed the plausibility of the measurements. LV = Left ventricle, RV = Right ventricle
To improve visualization of the RV, acquisitions were obtained from the four-chamber apical view adapted with the patient in the left decubitus position. For each patient, a full volume was obtained from six consecutive cardiac cycles during a single breath-hold to avoid translational motion. Echocardiographic data sets were stored digitally. Postprocessing analysis and 3D reconstruction were all performed offline. Designed software was used for the quantification of global RV and LV function. The following landmarks were manually defined: (1) LV and RV long axes at end-diastole in apical four- and two-chamber views, (2) LVOT in apical three-chamber view, (3) the anterior and posterior junction points of the RV free wall with the interventricular septum, and (4) the longest dimension of the RV cavity between the septum and the free wall in short-axis view. From these landmarks, the software automatically extracted the RV-focused four-chamber view and serial short-axis views from base to apex for both end-systole and end-diastole, from which a 3D model of the right ventricle was generated. Manual adjustments of tracking were made if necessary. The 3D RV endocardial surface was then tracked, and volumes and EF calculated offline using the method of summation of discs.

**Statistical analysis**

All statistical analyses were performed using standard statistical software (SPSS version 25.0, Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were expressed as numbers and percentages. Categorical variables were compared with Chi-square statistics, whereas variables with a nonparametric distribution between the groups were compared by Mann–Whitney U-test. Kruskal–Wallis test was performed to compare RV TDI parameters calculated in correspondence with the anterior, inferior, and lateral RV walls in Groups I and II. Correlations were examined by Spearman’s correlation model for nonparametric data. Values were considered statistically significant at $P < 0.05$.

**Results**

In Table 1, the baseline characteristics of the study population are presented. No differences in terms of age and sex were recognized between the two groups. In athletes, diastolic and systolic LVID (5.7 ± 0.4 vs. 4.8 ± 0.3 and 3.6 ± 0.5 vs. 3 ± 0.3; $P < 0.0001$) and LV mass/BSA (132.3 ± 22.7 vs. 114.9 ± 30.1; $P = 0.017$) were significantly higher in comparison with the control group. Furthermore, athletes showed higher two-dimensional (2D) LV end-diastolic volume (EDV)/BSA (69.3 ± 10.5 vs. 58.7 ± 10; $P = 0.001$), 2D LV ESV/BSA (25.1 ± 4.7 vs. 20.5 ± 4.1; $P = 0.001$), and LA volume index (24.7 ± 4.9 vs. 20.2 ± 3.5; $P = 0.047$), whereas MV A peak (0.4 ± 0.1 vs. 0.5 ± 0.1; $P = 0.001$) and E/e’ ratio (5.8 ± 1.3 vs. 7.2 ± 1.3; $P = 0.001$) were reduced in comparison with nonathletic. Noteworthy, LV and RV SV (LV: 93.8 ± 15 vs. 76.3 ± 13.3 and RV: 99.4 ± 16.7 vs. 79.1 ± 13.6; $P < 0.0001$, respectively) were significantly higher in athletes, as well as basal RVD (3.9 ± 0.4 vs. 3.3 ± 0.3; $P < 0.0001$) and RA area (20.4 ± 3.3 vs. 15.6 ± 2.6; $P < 0.0001$) were significantly increased in comparison with controls [Table 1]. No difference was found between the two groups in terms of EF, TAPSE, and sPAP.

In athletes, ET measured at lateral (282.4 ± 31.2 vs. 255.6 ± 35.5; $P = 0.008$) and anterior (287.6 ± 21.9 vs. 262.2 ± 30; $P = 0.002$) tricuspid annulus was statistically increased, as well as IVCT was longer if calculated in correspondence with inferior (81.7 ± 12.7 vs. 71.3 ± 16.7; $P = 0.01$) and anterior (79.4 ± 12.4 vs. 68.5 ± 9.6; $P = 0.002$) RV walls. Conversely, the anterior RV

**Figure 2:** On the left, apical views of RV using TDI in addition to the conventional standardized RV views were recorded. The sample volume of PW TDI was placed on the RV lateral, anterior, and inferior wall at the lateral, anterior, and inferior tricuspid annulus (in green, yellow, and blue, respectively). Tissue velocities were evaluated from the three RV walls, respectively. In the middle, triplane imaging of the RV was obtained focusing on RV by the apical four-chamber view in order to visualize the RV lateral, anterior, and inferior wall, respectively. On the right, a representative case of three-dimensional RV quantification after tracing the endocardial border at end-diastole and end-systole was reported. Below, a model of the right ventricle was automatically constructed in short-axis view. RV = Right ventricular, Diast = diastole, Syst = systole.
Table 1: Demographic and echocardiographic data of the study population

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| General characteristics |                |                |     |
| Age (years)         | 31±9           | 26±4           | 0.068 |
| Male                | 22 (88%)       | 25 (100%)      | 0.074 |
| BSA (m²)            | 1.9±0.1        | 2.1±0.1        | 0.0001 |
| M mode              |                |                |     |
| IVSd (cm)           | 1±0.2          | 1±1.1          | 0.568 |
| LVIDd (cm)          | 4.8±0.3        | 5.7±0.4        | <0.0001 |
| LVFWd (cm)          | 1±0.2          | 0.9±0.1        | 0.113 |
| IVSs (cm)           | 1.4±0.2        | 1.4±0.2        | 0.984 |
| LVIDs (cm)          | 3±0.3          | 3.6±0.5        | <0.0001 |
| LVPWs (cm)          | 1.6±0.2        | 1.7±0.2        | 0.157 |
| LV mass/BSA (g/m²)  | 114.9±30.1     | 132.3±22.7     | 0.017 |

2D left chamber measurements

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| LV EDV/BSA (ml/m²)  | 58.7±10        | 69.3±10.5      | 0.001 |
| LV ESV/BSA (ml/m²)  | 20.5±4.1       | 25.1±4.7       | 0.001 |
| LV EF (%)           | 64.9±3.7       | 63.8±2.8       | 0.347 |
| LA volume index (ml/m²) | 20.2±3.5    | 24.7±4.9       | 0.047 |

Diastolic function

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| MV E max (m/s)      | 0.8±0.1        | 0.7±0.1        | 0.607 |
| MV dec time (ms)    | 175.3±29.8     | 174.6±54.8     | 0.634 |
| MV A max (m/s)      | 0.5±0.1        | 0.4±0.1        | 0.001 |
| E/A                 | 1.5±0.6        | 1.9±0.4        | 0.006 |
| E'/e' ratio         | 7.2±1.3        | 5.8±1.3        | 0.001 |

LVOT measurements

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| LVOT diameter (cm)  | 2±0.1          | 2.2±0.1        | <0.0001 |
| LVOT SV (ml)        | 76.3±13.3      | 93.8±15        | <0.0001 |

RVOT measurements

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| RVOT diameter (cm)  | 2.2±0.2        | 2.3±0.2        | 0.122 |
| RVOT SV (ml)        | 79.1±13.6      | 99.4±16.7      | <0.0001 |

RV parameters

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| Basal RVd (cm)      | 3.3±0.3        | 3.9±0.4        | <0.0001 |
| Mid RVd (cm)        | 2.5±0.3        | 2.7±0.4        | 0.091 |
| RA area (cm²)       | 15.6±2.6       | 20.4±3.3       | <0.0001 |
| TAPSE (mm)          | 25.1±1.9       | 26.2±2.2       | 0.051 |
| sPAP (mmHg)         | 28.5±4.1       | 26.4±2.9       | 0.121 |

3DE=Three dimensional, BSA=Body surface area, LV=Left ventricular, RV=Right ventricular, LV EDV=LV end-diastolic volume, LV ESV=LV end-systolic volume, LV SV=LV stroke volume, RV EDV=RV end-diastolic volume, RV ESV=RV end-systolic volume, RV SV=RV stroke volume, BSA=Body surface area, IVSd=Interventricular septum thickness at end-diastole, LVIDd=LV end-systolic internal dimension at end-diastole, LVPWd=LV posterior wall thickness at end-diastole, IVSs=Interventricular septum thickness at end-systole, LVIDs=LV internal dimension at end-systole, TAPSE=Tricuspid annular plane systolic excursion, sPAP=Systolic pulmonary artery pressure

Table 2: TDI parameters measured at lateral, inferior, and anterior right ventricular wall

| Parameters          | Controls (n=25) | Athletes (n=25) | P   |
|---------------------|----------------|----------------|-----|
| Lateral RV wall     |                |                |     |
| IVCT (ms)           | 71±11.8        | 76.2±13.5      | 0.285 |
| IVRT (ms)           | 58.6±14.9      | 55.1±11.5      | 0.331 |
| ET (ms)             | 255.6±35.5     | 282.4±31.2     | 0.008 |
| MPI                 | 0.5±0.1        | 0.4±0.1        | 0.135 |
| Inferior RV wall    |                |                |     |
| IVCT (ms)           | 71.3±16.7      | 81.7±12.7      | 0.01 |
| IVRT (ms)           | 53.4±13.2      | 55.4±10.7      | 0.770 |
| ET (ms)             | 264.5±28.9     | 279±26         | 0.06 |
| MPI                 | 0.4±0.1        | 0.4±0.1        | 0.620 |
| Anterior RV wall    |                |                |     |
| IVCT (ms)           | 68.5±9.6       | 79.4±12.4      | 0.002 |
| IVRT (ms)           | 78.9±16.8      | 68.5±15.8      | 0.025 |
| ET (ms)             | 262.2±30       | 287.6±21.9     | 0.002 |
| MPI                 | 0.5±0.1        | 0.5±0.1        | 0.068 |

IVCT=Isovolumic contraction time, IVRT=Isovolumetric relaxation time, ET=Ejection time, MPI=Myocardial performance index, RV=Right ventricular

± 9.1 vs. 40.7 ± 9.3, respectively) and end-systolic (LV: 22.5 ± 3.1 vs. 17.3 ± 3.1 and RV: 17 ± 4.9 vs. 11.5 ± 3.8) volumes indexed for BSA [Table 3] as well as 3D LV and RV SV/BSA (LV: 42.5 ± 3.7 vs. 33.9 ± 5.9 and RV: 38.9 ± 5.2 vs. 29.2 ± 6.4) in comparison with nonathletic (P < 0.0001) [Figure 3]. Conversely, 3D LV and RV EF were not different between the two groups.

Finally, a significant positive correlation between IVRT and 3D RV end-diastolic (r = 0.310, P = 0.03), end-systolic (r = 0.332, P = 0.02), and stroke (r = 0.284, P = 0.048) volumes indexed for BSA was observed in athletes. Of note, 3D RV EDV/BSA was significantly associated with the increased 3D LV SV/BSA (r = 0.697, P < 0.0001).

Discussion

The key findings from this study are as follows: (1) highly trained athletes show generally LV and RV remodeling in terms of increased ventricular volumes and SV than nonathletic controls; (2) regional RV function in athletes evaluated by a multisegmental TDI approach is different for lateral, anterior, and inferior RV wall; (3) the higher RV volumes assessed by 3DE are proportionally correlated to an increase of IVRT and (4) greater exercise-induced RV EDV leads to greater LV SV.

To the best of our knowledge, this is the first study that evaluates regional RV function in selected athletes and the impact of RV volumes evaluated by 3DE on resting regional RV TDI parameters.

In the last decades, many studies have been published about cardiac remodeling induced by intensive training.[15]

It is well known that in athletes, morphofunctional changes do not involve only the LV but all the heart chambers. Prolonged...
exercise may cause an increase of LV wall thickening\textsuperscript{[16]} and LV dilatation,\textsuperscript{[17]} as well as a mild increase of LA and RA diameters and volume is considered a physiological adaptation to exercise.\textsuperscript{[18,19]} Specifically, regarding the RV, Prakken \textit{et al.} have demonstrated significantly larger RV volumes in elite athletes as compared with recreational athletes and nonathletes.\textsuperscript{[20]} In agreement with the previous studies, also in our cardiac remodeling characterized by LV hypertrophy, left and right chamber dilatation was observed.

However, a problematic issue is that in the majority of athletes, there is a high prevalence of dilatation and reduced functional measures of RV that in most cases are not signs of pathology. Therefore, it is often difficult to distinguish physiological from pathological resting changes of RV function, and there is a potential risk to confuse arrhythmogenic RV cardiomyopathies (ARVCs) from athletic physiological cardiac remodeling.\textsuperscript{[21]} In literature, it is documented that ARVCs are the substrate of 3\%–10\% of all SCD in athletes.\textsuperscript{[22]} In this regard, crucial is the differentiation of RV physiological adaptation from RV diseases, particularly in preparticipation screening programs. Accordingly, how should the athletes be correctly evaluated in relation to RV remodeling? These are questions that are yet to be answered, and clinical diagnostic algorithms are needed. An extended comprehensive echocardiographic protocol for RV, including also 3DE and TDI, in athletes has been proposed.\textsuperscript{[6]}

Certainly, in these, a full and careful evaluation of right-sided heart chamber morphology and function is necessary. TTE is generally the most commonly used tool for routine RV assessment. However, various limitations that impair its accuracy still exist: the inadequate visualization of the RV for its geometric complexity and poorly reliable 2D measurements of morphology and function such as RV EF.\textsuperscript{[23]}

In the last years, various imaging techniques for RV evaluation have been employed and overcome the limitations of 2D imaging, especially real-time 3DE and cardiac magnetic resonance (CMR) imaging, able to provide high-resolution reconstructions in multislice of RV, LV longitudinal strain imaging, and TDI that represent useful additional imaging modalities in the comprehensive assessment of RV function.\textsuperscript{[24]}

It is known that RV architecture is characterized by a lack of circular midventricular RV fibers, as well as inteventricular parietal and septal myocardial fibers are responsible for about 30\%–60\% of the RV contractility due to their oblique

Table 3: Three dimensional left ventricular and right ventricular data of the study population

| Parameters | Controls \((n=25)\) | Athletes \((n=25)\) | \(P\) |
|------------|------------------|----------------|-----|
| 3D LV measurements | | | |
| LV EDV/BSA (mL/m\(^2\)) | 51.4±8.6 | 65.1±6.1 | 0.0001 |
| LV ESV/BSA (mL/m\(^2\)) | 17.3±3.1 | 22.5±3.1 | <0.0001 |
| LV SV/BSA (mL/m\(^2\)) | 33.9±5.9 | 42.5±3.7 | <0.0001 |
| LV EF (%) | 66.1±2.1 | 65.3±2.4 | 0.327 |
| 3D RV measurements | | | |
| RV EDV/BSA (mL/m\(^2\)) | 40.7±9.3 | 55.9±9.1 | <0.0001 |
| RV ESV/BSA (mL/m\(^2\)) | 11.5±3.8 | 17±4.9 | <0.0001 |
| RV SV/BSA (mL/m\(^2\)) | 29.2±6.4 | 38.9±5.2 | <0.0001 |
| RV EF (%) | 71.9±5 | 70.1±4.7 | 0.194 |

3D=Three dimensional, BSA=body surface area, LV=Left ventricular, RV=Right ventricular, LV EDV=LV end-diastolic volume, LV ESV=LV end-systolic volume, LV SV=LV stroke volume, LV EF=LV ejection fraction, RV EDV=RV end-diastolic volume, RV ESV=RV end-systolic volume, RV SV=RV stroke volume, RV EF=RV ejection fraction
infiltration into the free RV wall. Accordingly, RV function is mainly determined by the coupling between intrinsic contractility and RV afterload. Therefore, the rationale of our study was to analyze regional differences in RV function related to the peculiar anatomical distribution of myocardial RV fibers and, using TDI imaging, to focus on the radial component of the RV contraction, less influenced by the LV contraction in comparison with the longitudinal deformation.

The peculiarity of the present work is the choice of a combined RV evaluation using multisegmental TDI and 3DE, which allowed a more complete and accurate assessment of global and regional RV morphology and function. Our data suggest that in athletes, ET, IVCT, and IVRT evaluated by TDI were different for lateral, anterior, and inferior RV wall, whereas MPI was not different between the two groups. In highly trained athletes, the increased RV load in association with increased ventricular volumes may explain the different regional patterns of contraction and relaxation at resting. Furthermore, despite some studies suggest that RV MPI of athletes was lower than controls, our results are in agreement with the study of Alsafi et al., in which no significant difference in MPI was found.

In addition to TDI, we used 3DE, a robust and reproducible approach to quantify reliably LV and RV volumes and EF. As expected, athletes showed significantly greater EDV, ESV, and SV of both ventricles in comparison with the controls, indicating an exercise-induced cardiac remodeling, although no difference in terms of EF was found.

Interestingly, we found that the higher 3D RV volumes indexed for BSA were proportionally correlated to an increase of IVRT_{RV-inferior}. This finding extends the knowledge on RV physiology of the super-trained heart, suggesting that greater RV volumes and SV have a determinant impact on relaxation and contraction times of RV inferior. Furthermore, in agreement with the findings of D’andrea et al., in our study, greater exercise-induced RV EDV leads to higher LV EDV and, subsequently, better stretching of myocardial fibers able to enhance LV SV by Frank–Starling mechanism. This suggests the interdependence between two ventricles also in the context of athletic cardiac remodeling, in order to insure an adequate systolic output and a high cardiac performance.

The current studies suggest that modern echocardiographic tools should be implemented in clinical routine to assess RV mechanics in athletes and also to identify potential clues of subtle RV dysfunction.

Although not available in our study population, speckle-tracking echocardiography (STE) has been applied to the assessment of RV function at rest and during exercise and has been proposed as a promising noninvasive means of identifying intrinsic RV dysfunction that sometimes is not apparent at rest. Lower values of RV longitudinal $\varepsilon$ (RV $\varepsilon$) have been reported in elite endurance athletes compared to controls, specifically in those with an associated dilated RV cavity, largely due to lower basal strain. La Gerche et al. have attributed this finding to a normal physiological response of the RV to exercise, likely due to an enhanced cardiac reserve of the basal segment. Noteworthy, the major limitation of the clinical application STE is the lack of universally accepted RV $\varepsilon$ values and protocols, particularly in the functional assessment of RV.

On the basis of our findings, offline analysis of RV TDI and 3DE parameters may be useful on all examinations of elite athletes as an integration to screening program. We do not know the significance of resting changes of regional RV TDI parameters, their clinical impact, whether these could be different in other subsets of athletes, and whether they could have a role in identifying selected individuals to candidate to further diagnostic examinations. We might hypothesize that higher RV volumes in athletes affect RV mechanics and, subsequently, regional RV TDI parameters as physiological response to exercise.

Further studies on the resting changes of regional RV deformation both for screening and follow-up in competitive athletes are needed and may represent an important area of research.

**Limitations**

We recognize as limitations applied to our study the fact that the sample size is limited, as well as that our population has been enrolled only in one institution. Thus, our findings suffer the problems related to a single-center analysis, and the generalizability of our observations is not possible. Undoubtedly, we enrolled a highly selected population, including only individuals with optimal images and excellent echocardiographic windows. Furthermore, the data of the present study may not be extended to the overall population of athletes but only to top-level professional soccer players with the characteristics of training cited in Methods. Another study limitation is that ours is a pilot study that did not include RV longitudinal strain data, currently not available. In addition to CMR data, also speckle-tracking analysis might have provided additional information about RV morphology and function. Finally, in order to attribute a clinical meaning to our findings, the comparison with patients with known RV cardiomyopathies would have been useful.

**Conclusions**

The present study emphasizes the evaluation of global and regional RV function in athletes. TDI and 3DE represent promising and feasible noninvasive techniques for a more comprehensive assessment of RV remodeling. The higher 3D RV volumes are proportionally related to an increase of IVRT_{RV-inferior}, as well as a greater exercise-induced RV EDV leads to higher LV SV.

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**Conflicts of interest**

There are no conflicts of interest.
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