Right ventricular remodelling induced by exercise training in competitive athletes

Flavio D’Ascenzi1*, Antonio Pelliccia2, Domenico Corrado3, Matteo Cameli1, Valeria Curci1, Federico Alvino1, Benedetta Maria Natali1, Marta Focardi1, Marco Bonifazi4, and Sergio Mondillo1

1Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Viale M. Bracci, 16, Siena 53100, Italy; 2Institute of Sports Medicine and Science, Rome, Italy; 3Division of Cardiology, Department of Cardiac, Thoracic and Vascular Sciences, University of Padua, Padua, Italy; and 4Department of Medicine, Surgery, and Neuroscience, University of Siena, Siena, Italy

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Aims

Conflicting evidence exists concerning right ventricular (RV) morphological and functional remodelling in trained athletes, with a very few longitudinal data prospectively investigating the RV changes. The aim of this study was to assess the morphological and functional RV changes occurring during the competitive season in young athletes engaged in the most popular team sports.

Methods and results

Twenty-nine top-level athletes (age: 20.9 ± 6.7 years), practicing basketball and volleyball, were evaluated at pre-season, mid-season, and end-season time-points, using tissue Doppler imaging and 2D speckle-tracking echocardiography. RV basal and mid-cavity end-diastolic diameters (EDDs; overall $P = 0.011$ and $P < 0.0001$, respectively), and RV diastolic area (overall $P < 0.0001$) increased during the season. Conversely, RV outflow tract did not vary (overall $P = 0.96$). During the season, no significant differences were observed in RV diastolic functional indexes and in RV fractional area change (overall $P = 0.35$). Global RV longitudinal strain did not significantly change (overall $P = 0.52$), although apical longitudinal strain significantly increased (overall $P = 0.017$). In association, left ventricular (LV) volume and mass increased during the season (overall $P = 0.007$). On multivariate analysis, LV mass was the only independent predictor of RVEDD at pre-season ($\beta = 0.69$, $P < 0.0001$) and at end-season ($\beta = 0.82$, $P < 0.0001$).

Conclusions

Right ventricular chamber size increases during the competitive season in top-level athletes, with no significant changes in the outflow tract. RV morphological adaptation in top-level athletes practicing team sports is not associated with a reduction in RV function or in myocardial deformation and occurs in close association with changes on the left ventricle, suggesting a physiological remodelling of the right ventricle.

Keywords

Athlete’s heart • Speckle-tracking echocardiography • Strain • Exercise conditioning • Arrhythmogenic right ventricular dysplasia

Introduction

Morphological and functional left ventricular (LV) and atrial remodelling induced by intensive exercise training have been extensively described.1–5 Conversely, relatively few reports have addressed the impact of exercise conditioning on the right ventricular (RV) morphology and function in young trained athletes.6–9 However, recently, concerns have been raised on the detrimental effects of strenuous and chronic exercise training on RV morphology and function in cohorts of endurance athletes, prompting the clinical question of an exercise-induced RV damage.10–12 Understanding the mechanisms and the potential reversibility of RV morphological and functional changes in highly trained athletes has become, therefore, a significant clinical issue, with relevant medical and legal implication in the context of pre-participation cardiovascular screening.

Although the echocardiographic assessment of the RV morphology and function is hampered by the complex RV geometry and the pronounced trabeculation of this cardiac chamber,13 2D speckle-tracking echocardiography (STE), being angle-independent and less load-sensitive than the other echocardiographic techniques,
is able to accurately analyse regional myocardial deformation,\textsuperscript{14,15} and may overcome some of the limitations of traditional echocardiography. This non-invasive technique has been already used to identify regional wall motion abnormalities in patients with arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D),\textsuperscript{16} and to quantify RV function in athletes.\textsuperscript{6—9,12}

At present, scarce longitudinal data are available regarding the occurrence of RV adaptation to the high volume, intensive endurance training, and no longitudinal studies have investigated RV adaptation to exercise in athletes engaged in the most widely practiced sports in Europe.\textsuperscript{17,18} Therefore, we planned this study with 2D STE to assess the RV morphology and function in a cohort of highly trained basketball and volleyball players. The aim of this longitudinal study was to investigate (i) to what extent morphological and functional RV changes occur red during the competitive season in highly trained athletes practicing team sports, and (ii) whether these changes were associated with any impairment of RV function.

**Methods**

**Study design**

Thirty-one top-level athletes, members of two elite sports teams and practicing basketball and volleyball, were enrolled in this study. Athletes, competing in an international level, participated in a supervised intensive training programme. Echocardiographic examination was performed at three different time-points: (i) at the beginning of the season, (ii) at mid-season, after 4 months of training, and (iii) at the end of the season, i.e. 8 months after the beginning of the study. Athletes were engaged in the intensive and closely supervised training programme during these periods. See Supplementary data for characterization of training periods.

Athletes were excluded from the study if they withdrew from the training programme >15 days due to musculoskeletal injuries. Accordingly, two athletes were excluded, and the final population consisted of 29 athletes. After the rationale and the study protocol were explained, the participants gave informed consent. Athletes underwent complete physical examination, ECG, echocardiography, and treadmill ECG test. All denied cardiac symptoms and reported negative family history for cardiac disease or sudden cardiac death. None showed structural cardiovascular abnormalities, hypertension, type I diabetes mellitus. The investigational protocol was approved by the local ethical committee.

**Echocardiographic measurements**

Echocardiographic examination was performed by one cardiologist using a high-quality echocardiograph (Vivid 9, GE, Milwaukee, WI, USA), equipped with an M4S 1.5–4.0 MHz transducer, and a one-lead ECG obtained during the echocardiographic examination. Echocardiographic examination was performed at the tips of tricuspid valve and at the tricuspid annulus, respectively.\textsuperscript{23} The following measurements of RV filling were considered: E peak and A peak velocities, E/A ratio, s’, e’, and a’ velocities. Pulsed-wave and tissue Doppler imaging were also performed for the LV: s’, e’, and a’ velocities were measured at the septal and lateral corners of the mitral annulus and an average value was calculated.\textsuperscript{23} Mitral and tricuspid E/e’ ratios were calculated and used as indexes of left and right cardiac filling pressures, respectively.\textsuperscript{13,22–25}

**Two-dimensional Speckle tracking echocardiography**

Two-dimensional STE analysis was performed on narrow-sector grayscale images of both RV and LV from an apical four-chamber view with temporal resolution of 60–90 frames/s. All images were optimized with gain, compression, and dynamic range to enhance myocardial definition with standardized depth, frequency, and insonation angle for all participants.\textsuperscript{26,27} Off-line analysis was performed by an experienced reader, blinded to the study time-point, using a commercially available semi-automated 2D strain software (EchoPAC PC, version 112, GE, USA). A region of interest was manually traced along the endocardial border from base to apex and width was set to match the wall thickness. If the automated 2D analysis appraisal of acceptable tracking quality indicated inappropriate tracking, retracking was performed until all segments were considered acceptable.\textsuperscript{28} RV systolic, early and late diastolic strain rates (SRs) were also obtained.

**Statistical analysis**

Normal distribution of all continuous variables was examined using the Shapiro–Wilks test. Comparison of data collected at different time-points was performed using Friedman test or repeated-measures ANOVA with Dunn or Bonferroni post hoc correction for multiple comparisons, as appropriate for data distribution. A P-value <0.05 was considered significant. Univariate correlation analysis was performed to find association between continuous variables using the Spearman and Pearson methods, as appropriate for data distribution. Consistencies of intra-observer measurements of RV basal and mid-cavity end-diastolic diameters, RV end-diastolic and ESAs, and RV strain were verified through the intra-class correlation coefficient (ICC). The outcomes of three consecutive examinations of pre-season echocardiographic measurements were collected for each parameter from a

\[ LVM = \frac{0.8 \times (1.04 \times (LVIDd + PWdTd + SWTd) - (LVIDd)^3)}{0.6} \text{ g}, \]

whereas LVIDd is LV internal dimension at end-diastole, PWdTd is posterior wall thickness at end-diastole, and SWTd is septal wall thickness at end-diastole. RV chamber size was assessed as recommended by current guidelines.\textsuperscript{13} Basal and mid-cavity end-diastolic diameters (EDDs), RV length, and the derived RV ejection fraction were obtained.\textsuperscript{13} According to the possible impact of gender on RV dimensions,\textsuperscript{21} data for RV cavity size were presented also separated between male and female athletes. RV outflow tract (RVOT) diameter was measured at the proximal level in the parasternal long-axis view, calculated from the anterior RV wall to the RV septum (RVOT-PLAX); at subvalvular level in the parasternal short axis at the level of the aortic valve, calculating the maximum distance between the anterior aortic wall and the RV free wall (RVOT-PSAX).\textsuperscript{13} RV end-diastolic and RV end-systolic areas (ESAs) were calculated by tracing around the endocardium from a modified apical 4-chamber view, and RV fractional area change (RVFAC) was obtained and expressed as percentage.\textsuperscript{13} Tricuspid annular plane systolic excursion was also calculated and considered as an index of RV longitudinal systolic function.\textsuperscript{13} The current revised diagnostic criteria for ARVC/D were used to assess whether athletes fulfilled the echocardiographic criteria for ARVC/D.\textsuperscript{12}
subsample of 15 randomly chosen subjects and were analysed by the same operator, blinded to patients’ data. The reliability was ranked as slight (0 ≤ ICC < 0.20), fair (0.20 < ICC < 0.40), moderate (0.40 < ICC < 0.60), substantial (0.60 < ICC ≤ 0.80), and almost perfect (0.80 < ICC ≤ 1). Besides the ICC, to evaluate the clinical utility of the measurements, the standard error of measurement was calculated, and the minimum detectable change was derived. Statistics were performed using SPSS version 20 (Statistical Package for the Social Sciences, Chicago, IL, USA).

**Results**

Mean age of the study population was 20.9 ± 6.7 years, and male athletes constitute 45% of overall population. BSA increased during the study period, reaching the highest value at the end of the season (2.2 ± 0.2 vs. 2.1 ± 0.2 m²,  

$P < 0.0001$ vs. pre-season data). Resting heart rate did not significantly change during the study period (from 65.5 ± 11.4 to 59.9 ± 8.3 bpm, overall $P$-value = 0.12). During the study period, all athletes remained in healthy condition, in the absence of cardiac or systemic symptoms requiring further investigations.

**Right ventricular morphological adaptation**

Right ventricular morphological changes observed during the season are reported in Table 1 and in Figure 1. Both RV basal and mid-cavity EDD increased during the season, reaching the highest value at mid-season time-point (overall $P$-value = 0.011 and <0.0001, respectively). Conversely, no changes were observed both in RVOT-PLAX and RVOT-PSAX diameters. Both RV end-diastolic area (EDA) and ESA significantly increased during the competitive season (overall $P$-value = 0.001 and <0.0001, respectively). Although male athletes showed greater absolute values of both RV end-diastolic and ESAs, the trend was similar between male and female athletes. The trend of RV EDA changes in females at pre-, mid-, and end-season time-points was: 19.0 ± 2.2, 22.4 ± 2.5, and 22.2 ± 2.0 cm², respectively, whereas in males, the trend of RV EDA was: 25.2 ± 3.1, 28.1 ± 4.7, and 29.5 ± 3.9 cm², respectively. The trend of RV ESA changes in females was 9.9 ± 1.7, 11.4 ± 1.7, and 11.4 ± 1.7, respectively, whereas the trend in males was 13.8 ± 1.3, 14.6 ± 2.6, and 16.4 ± 2.5, respectively.

Individual analysis showed that, at the study entry, substantial proportion of the athletes had a RVOT-PLAX diameter fulfilling the minor (46%, i.e. ≥ 29 and < 32 mm) and major (43%, i.e. ≥ 32 mm) diagnostic criteria for the echocardiographic diagnosis of ARVC/D; however, when RVOT-PLAX was indexed to BSA, only 11% of athletes fulfilled the minor criteria (i.e. ≥ 16 and < 19 mm/m²) and none the major criteria for ARVC/D (i.e. ≥ 19 mm/m²). During the season, when RVOT-PLAX/BSA increased, a further 10% of athletes fulfilled the minor criteria, whereas still no one attained the major criteria for ARVC/D.

At baseline analysis, 54% of athletes had a RVOT-PSAX diameter fulfilling the minor (i.e. ≥ 32 and < 36 mm) and 21% major criteria (i.e. ≥ 36 mm) for ARVC/D, respectively. When RVOT-PSAX was indexed to BSA, only 11% fulfilled the minor criteria (i.e. ≥ 18 and < 21 mm/m²) and none major criteria for ARVC/D.

**Table 1** Right ventricular morphological and functional echocardiographic parameters observed during the season in competitive athletes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-season</th>
<th>Mid-season</th>
<th>End-season</th>
<th>Overall $P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV basal ED diameter, mm</td>
<td>35.4 ± 5.7</td>
<td>39.5 ± 5.9*</td>
<td>37.8 ± 7.8***</td>
<td>0.011</td>
</tr>
<tr>
<td>RV mid-cavity ED diameter, mm</td>
<td>30.8 ± 5.0</td>
<td>35.2 ± 4.9***</td>
<td>32.0 ± 4.7***</td>
<td>0.000</td>
</tr>
<tr>
<td>RVOT-PLAX, mm</td>
<td>28.3 ± 4.2</td>
<td>28.3 ± 3.6</td>
<td>28.4 ± 4.6</td>
<td>0.96</td>
</tr>
<tr>
<td>RVOT-PSAX, mm</td>
<td>30.9 ± 5.3</td>
<td>31.1 ± 4.1</td>
<td>30.4 ± 5.4</td>
<td>0.052</td>
</tr>
<tr>
<td>RV sphericity index</td>
<td>0.41 ± 0.07</td>
<td>0.45 ± 0.06</td>
<td>0.41 ± 0.07</td>
<td>0.062</td>
</tr>
<tr>
<td>RVES area, cm²</td>
<td>21.2 ± 3.9</td>
<td>24.4 ± 4.3***</td>
<td>24.8 ± 4.5***</td>
<td>0.000</td>
</tr>
<tr>
<td>RVED area, cm²</td>
<td>11.3 ± 2.4</td>
<td>12.5 ± 2.5</td>
<td>13.2 ± 3.2***</td>
<td>0.001</td>
</tr>
<tr>
<td>RV FAC, %</td>
<td>47.0 ± 4.4</td>
<td>48.7 ± 4.6</td>
<td>47.4 ± 5.1</td>
<td>0.35</td>
</tr>
<tr>
<td>End-diastolic RV/LV diameter ratio</td>
<td>0.65 ± 0.12</td>
<td>0.72 ± 0.10***</td>
<td>0.65 ± 0.08</td>
<td>0.015</td>
</tr>
<tr>
<td>IVC diameter, mm</td>
<td>17.8 ± 4.6</td>
<td>21.1 ± 4.25</td>
<td>20.9 ± 3.6***</td>
<td>0.005</td>
</tr>
<tr>
<td>Tricuspid E/A ratio</td>
<td>2.1 ± 0.6</td>
<td>2.1 ± 0.6</td>
<td>2.4 ± 0.7</td>
<td>0.93</td>
</tr>
<tr>
<td>e’ peak value, m/s</td>
<td>0.16 ± 0.05</td>
<td>0.17 ± 0.06</td>
<td>0.19 ± 0.04</td>
<td>0.075</td>
</tr>
<tr>
<td>e’/a’ ratio</td>
<td>1.9 ± 0.6</td>
<td>1.9 ± 0.6</td>
<td>2.3 ± 1.2</td>
<td>0.66</td>
</tr>
<tr>
<td>El/e’ ratio</td>
<td>4.3 ± 0.9</td>
<td>4.0 ± 1.0</td>
<td>4.0 ± 0.9</td>
<td>0.15</td>
</tr>
<tr>
<td>TAPSE, mm</td>
<td>25.7 ± 5.0</td>
<td>27.4 ± 5.0</td>
<td>26.2 ± 3.7</td>
<td>0.15</td>
</tr>
<tr>
<td>SPAP, mmHg</td>
<td>20.0 ± 6.1</td>
<td>25.0 ± 5.0</td>
<td>28.0 ± 5.7</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD. RV free wall longitudinal strain represents the average value obtained from apical, mid, and basal walls. Global RV longitudinal strain includes the analysis of both RV free wall and interventricular septum.

RV, right ventricular; ED, end-diastolic; RVOT, right ventricular outflow tract; PLAX, parasternal long-axis view; PSAX, parasternal short-axis view; ES, end-systolic; FAC, fractional area change; IVC, inferior vena cava; LV, left ventricular; TAPSE, tricuspid annular plane systolic excursion; SPAP, systolic pulmonary artery pressure.

* $P < 0.05$ vs. pre-season; ** $P < 0.05$ vs. pre-season; ***$P < 0.05$ vs. mid-season.
During mid-season, a further 5% fulfilled the minor criteria, whereas none the major criteria for ARVC/D. In athletes, no regional RV wall motion abnormalities were detected neither at the beginning nor at the end of the study.

Right ventricular functional adaptation

The analysis of RV diastolic function is reported in Table 1. Neither pulsed-wave nor tissue Doppler-derived parameters showed significant changes during the competitive season.

Right ventricular fractional area change showed no changes during the study period (overall P-value = 0.35) and none of the athletes experienced a reduction of RVFAC (i.e. < 35%; Figure 2). Individual analysis showed that all the athletes had a RVFAC > 40%, and none fulfilled either minor or major diagnostic criteria for ARVC/D, neither at baseline nor during the season.

Right ventricular myocardial deformation analysis

Global and segmental RV longitudinal strain and strain rate values are reported in Table 2 and in Figure 2. The RV free wall longitudinal strain and the global RV longitudinal strain (including the interventricular septum) did not significantly change during the season (overall P-value = 0.56 and P = 0.14, respectively). However, regional differences were observed in the RV longitudinal strain: specifically, both mid and apical RV wall strain demonstrated a trend towards increase, with the latter being significantly higher at mid-season and end-season when compared with pre-season time-point (P < 0.005 and < 0.05, respectively; Figure 3). Conversely, basal RV strain showed a mild, non-significant decrease (overall P-value = 0.37).

The systolic RV free wall SR demonstrated a significant increase between pre- and mid-season to end-season time-point (overall P-value = 0.001). During mid-season, a further 5% fulfilled the minor criteria, whereas none the major criteria for ARVC/D. In athletes, no regional RV wall motion abnormalities were detected neither at the beginning nor at the end of the study.

(i.e. ≥ 21 mm/m²). During mid-season, a further 5% fulfilled the minor criteria, whereas none the major criteria for ARVC/D. In athletes, no regional RV wall motion abnormalities were detected neither at the beginning nor at the end of the study.
Right ventricular in-seasonal changes

Changes in LV parameters observed during the season are reported in the Supplementary data. A significant increase in LV end-diastolic volume and in LVM was observed, with the highest value reached at the end of the season ($P < 0.05$ and $P = 0.005$ vs. pre-season data, respectively). LV functional diastolic parameters and LV longitudinal strain did not significantly vary.

Correlation analysis

Right ventricular basal EDD was significantly correlated at each time-point with mid-cavity EDD ($R = 0.42$, $P < 0.05$; $R = 0.57$, $P < 0.005$; $R = 0.62$, $P < 0.001$, respectively), RV EDA ($R = 0.51$, $P < 0.01$; $R = 0.52$, $P < 0.01$; $R = 0.80$, $P < 0.0001$, respectively), and RV ESA ($R = 0.48$, $P < 0.05$; $R = 0.65$, $P < 0.001$; $R = 0.83$, $P < 0.0001$, respectively). At pre-season time-point, RV basal EDD was also significantly associated with IVC diameter ($R = 0.54$, $P < 0.005$), RV length ($R = 0.68$, $P < 0.0001$), and SV ($R = 0.56$, $P < 0.01$). At mid-season time-point, RV basal EDD was also associated with RV length ($R = 0.39$, $P < 0.05$), whereas at end-season time-point, RV basal EDD was associated with SV ($R = 0.59$, $P < 0.005$). While no significant associations were found for RV global longitudinal strain, end-season RV apical longitudinal strain was inversely associated with RV mid-cavity EDD ($R = -0.48$, $P < 0.05$), RV EDA ($R = -0.51$, $P < 0.05$), RV ESA ($R = -0.47$, $P < 0.05$), and SV ($R = -0.45$, $P < 0.05$). Neither RV $E'/e'$ ratio nor LV $E'/e'$ ratio were found to be associated with RV cavity dimensions.

For reproducibility of RV measurements, see Supplementary data.

Discussion

Dynamic changes in RV morphology

Highly trained athletes are known to present larger RV dimensions in comparison with sedentary subjects.7-9,31,32 RV cavity enlargement has been considered for a long time to represent the physiological consequence of exercise conditioning.31,32 However, these observations mostly rely on cross-sectional studies, which have the intrinsic limitation to be unable to prove a direct relationship between intensity and duration of exercise training and occurrence of RV morphological remodelling. Furthermore, most of the studies enrolled athletes engaged in endurance and/or ultra-endurance...
Disciplines that, although commonly practiced and with a growing number of participants, do not represent the vast majority of young adults athletes, commonly engaged in team sports.

For this reason, we planned this study that includes serially repeated measurements during the competitive season, offering novel information relative to occurrence and extent of RV morphological changes induced by intensive exercise training.

The first consideration is that significant RV dimensional changes do occur in athletes engaged in team sports during the competitive season, in response to the increased intensity of the training. Indeed, our study population included individuals not completely sedentary who had already been engaged in training programmes. Therefore, the RV has an extended potential for morphological adaptations in individuals already presenting some of the features of athlete’s heart.

Interestingly, while significant changes were found after training in global RV size, no significant changes occurred in the RVOT. The most likely explanation for these regional differences in RV adaptation to training-induced overload is their different embryological origin: the sinus or inlet portion of the RV has a distinct origin from that of the conus or outflow tract, and the main body of the RV connects these two regions, enclosed by the interventricular septum and RV free wall. Our findings are in agreement with a previous study investigating morphological differences and similarities between ARVC/D and athlete’s heart. In this cross-sectional study, Bauce et al. showed that both ARVC/D patients and athletes had greater RV dimensions when compared with controls; however, ARVC/D patients had larger RVOT diameters compared with athletes, confirming that RVOT dilatation represents primarily a pathological rather than a physiological finding. Thus, the evaluation of RVOT size could be a clinically relevant criterion to differentiate between ARVC/D and athlete’s heart, considering that a marked enlargement of the RVOT is uncommon in the athlete while it represents a peculiar pattern of pathological dilatation in ARVC/D patients.

Dynamic changes in RV function

At present, conflicting data exist concerning the evaluation of RV systolic and diastolic function in athletes. Recent reports have suggested that RV function may be profoundly affected by intensive and chronic endurance/ultra-endurance exercise. La Gerche et al. reported the incidence of an acute RV dysfunction immediately after a prolonged endurance race, with an impairment of systolic RV function (including RVFAC, RV global strain, and SR). However, no current data are available about exercise-induced RV adaptations in athletes engaged in the most widely practiced disciplines. In this study, we found that RV function in basketball and volleyball players does not deteriorate over the competitive season, despite a significant ventricular dilatation. Both systolic and diastolic indexes of RV function were within the normal range over the entire period of observation. Furthermore, E/e’ ratio did not vary during the season, and no correlations were found between this parameter and RV dimensions, suggesting that the increase in RV chamber size in trained athletes, as already described for the LV chamber, occurs as a consequence of predominantly volume overload.

RV myocardial deformation and regional differences

Scant and conflicting data are available concerning RV myocardial deformation in athletes, with some authors demonstrating a reduced deformation in athletes and some others reporting similar values between athletes and controls. This study provides new insights into the training-induced RV functional changes in athletes, demonstrating that RV enlargement is not associated with pathological reductions of RV myocardial deformation; conversely, RV strain demonstrates a (non-significant) trend towards increase.

Interestingly, regional differences in RV longitudinal deformation were detected: while RV apical wall strain increased, RV basal wall strain showed a non-significant trend towards decrease during the season. A modest reduction in systolic deformation at the inlet portion of the RV (i.e. the basal wall) has been also reported in a previous study in athletes and is known to occur in patients with open atrial septal defect, a pathological model of RV volume overload. Furthermore, RV is characterized by a sequential activation of the inflow, the body, and the outflow regions. This typical activation of RV myocardium and the contraction of the moderator band could influence the wall motion of the apical and basal free wall and could be a possible explanation of the observed regional differences in myocardial deformation. Considering that regional abnormalities have been detected by STE in asymptomatic patients carrier of genetic mutations for ARVC/D, knowing the influence of exercise conditioning on RV myocardial deformation could be relevant when the additional value of deformation myocardial imaging is used to distinguish between patients with ARVC/D and athlete’s heart.

Limitations

As per design in this study, we included athletes participating in the most practiced sports, such as volleyball and basketball, which incorporate a combination of endurance (dynamic), strength/resistance (static), and speed training and, therefore, the present data are not transferable to athletes practicing endurance and ultra-endurance sports. Thus, while a measure of caution still remains for the consequences of intensive and chronic exercise conditioning in athletes engaged in endurance disciplines, this study conveys a measure of reassurance and confidence regarding the clinical significance and consequences of the RV changes observed in top-level athletes engaged in the most practiced disciplines. The unavailability of 3D echocardiographic data on RV volumes represents a further limitation of this study.

Furthermore, future work is required to better understand whether exercise-induced RV changes might have clinical consequences over repeated, long-term bouts of competitive seasons.

Conclusions

Right ventricular chamber size increases during the competitive season in top-level athletes. However, no significant changes occur in the outflow tract. RV morphological adaptation in top-level athletes practicing team sports is not associated with an impaired function or myocardial deformation of the RV and occurs in close association with the adaptive changes on the left ventricle.
Supplementary data
Supplementary data are available at European Journal of Echocardiography online.

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Conflict of interest: none declared.

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