Comprehensive assessment of biventricular function and aortic stiffness in athletes with different forms of training by three-dimensional echocardiography and strain imaging†

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Aims

Previous studies have shown distinct models of cardiac adaptations to the training in master athletes and different effects of endurance and strength-training on cardiovascular function. We attempted to assess left-ventricular (LV) function, aortic (Ao) function, and right-ventricular (RV) function in athletes with different forms of training by using three-dimensional (3D) echocardiography, tissue Doppler imaging (TDI) and speckle-tracking imaging (STI).

Methods and results

We examined 35 male marathon runners (endurance-trained athletes, ETA), 35 powerlifting athletes (strength-trained athletes, STA), 35 martial arts athletes (mixed-trained athletes, MTA), and 35 sedentary untrained healthy men (controls, CTR). Two-dimensional and three-dimensional echocardiography were performed for the assessment of LV and RV systolic/diastolic function. LV and RV longitudinal strain (LS) and LV torsion (LVtor) were determined using STI (EchoPAC BT11, GE-Ultrasound). Maximum velocity of systolic wall expansion peaks (AoSvel) was determined using TDI. ETA experienced LV eccentric hypertrophy with increased 3D LV end-diastolic volume and mass and significant increase in peak systolic apical rotation and LVtor. In all groups of athletes, RV-LS was reduced at rest and improved after exercise. AoSvel was significantly increased in ETA and MTA and significantly decreased in STA compared with CTR. There were good correlations between LV remodelling and aortic stiffness values. Multivariate analysis showed aortic wall velocities to be independently related to LV mass index.

Conclusion

In strength-trained, endurance-trained, and mixed-trained athletes, ventricular and vascular response assessed by 3DE, TDI, and STI underlies different adaptations of LV, RV, and aortic indexes.

Keywords

Echocardiography • Athlete’s heart • Tissue Doppler imaging • Speckle tracking imaging • Three-dimensional echocardiography • Ventricular function • Aortic function

Introduction

The haemodynamic load caused by long-term training involves both cardiac ventricular chambers, inducing an increase in ventricular diameters, wall thickness, and mass. These changes are usually described as ‘athlete’s heart’. Adapations to exercise are dependent on the specific type of training performed. Previous studies have shown distinct models of cardiac adaptations to the training in master athletes and different effects of endurance and strength-training on cardiovascular function. Further reports pointed out that the classification of left-ventricular hypertrophy in athletes as eccentric or concentric is not an absolute or dichotomous concept but has to be considered a relative

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Three-dimensional echocardiography

Transthoracic examination was carried out with the patient lying in the left lateral recumbent position. The echocardiographic system was equipped with dedicated software for dynamic threedimensional acquisition. Standard parasternal, apical, and subcostal views were used. Three-dimensional images were saved in digital form on the hard disk of the ultrasound scanner and subsequently analysed using the EchoPAC view (BT11, GE Ultrasound). The left- and right-ventricular end-diastolic volumes (LVEDV, RVEDV) and ventricular end-systolic volumes (LVESV, RVESV) were measured from each three-dimensional echocardiographic data set. Left- and right-ventricular ejection fractions (LVEF and RVEF) were determined. The process of volume determination was done two times for each patient. Papillary muscles were not included in the volume estimation.

Speckle-tracking imaging

The general principles that underlie the STI modalities have been previously described. After tracing endocardial border at an end-systolic frame, the operator could validate the tracking quality and adjust the endocardial border or modify the width of the region of interest. Aortic valve opening and closure were selected on pulsed-wave Doppler tracings recorded from the LV outflow tract. Frame rate ranged from 60 to 100 frames/s, and three cardiac cycles were stored in cineloop format for offline analysis. Longitudinal LV strain was defined as the average of negative longitudinal strains of six segments of the septal and lateral walls in the apical four-chamber view (Figure 1A). Average radial and circumferential strain of six mid-LV segments was determined in the mid-short-axis view. The assessment of LV rotation by 2D speckle-tracking strain imaging required the acquisition of the LV short-axis at the basal and apical level. The basal level was defined as that which showed the mitral valve tip and the apical level as that which was just proximal to the level with LV cavity obliteration at end-systole. The time rotation curves were displayed along the cardiac cycle. Counterclockwise rotation was conventionally marked as positive value and clockwise rotation as negative value when viewed from the LV apex. Peak apical and basal rotation and peak LV torsion were obtained. Left-ventricular torsion or twist (LVtor) was defined as the net difference (in degrees) of apical and basal rotation at isochronal time points. Normalized torsion was torsion divided by LV ventricular diastolic longitudinal length between apex and mitral plane. Peak diastolic untwisting velocity and time-to-peak untwisting velocity were measured. Twisting rate (TR) was defined as (peak LVtor − LVtorEarlySystole)/(time difference between these two events). Untwisting rate (UTR) was defined as (peak LVtor − LVtorMVO)/(time difference between these two events), where MVO is mitral valve opening. The analysis of strain and rotation parameters was performed offline using customized computer software (EchoPAC BT11, GE Ultrasound). To assess regional and global RV systolic function in the longitudinal direction, we adopted a six-segment RV model (basal RV lateral wall, mid-RV lateral wall, apical RV wall, apical septum, mid septum, and basal septum). Peak systolic strain was recorded for the six RV myocardial segments and the entire RV myocardium. Global strain and strain rate were calculated by averaging local strains along the entire right ventricle using machine software.

Aortic function

To assess aortic distensibility, echocardiographic tracings were obtained using a two-dimensional guided M-mode evaluation of systolic and diastolic aortic diameter, 3 cm above the aortic valve.

Methods

Population

We examined 35 male marathon runners (endurance-trained athletes, ETA), 35 powerlifting athletes (strength-trained athletes, STA), and 35 martial arts athletes performing combined strength and endurance training (mixed-trained athletes, MTA). All of them were athletes participating in an organized team or individual sport in which regular training was a component. Each athlete trained for >15 h/week and had participated in competition for >6 years. Thirty-five sedentary untrained healthy age-matched male adults were selected as a control group. Both groups were free of heart and systemic diseases and took no medications. Subjects with echocardiographic recordings of inadequate quality were excluded. Ethical approval was obtained.

Echocardiography

All patients underwent transthoracic echocardiography with a commercially available cardiovascular ultrasound system (Vivid E9, GE, Horten, Norway). Measurements of cardiac chambers and aortic diameters were made according to established criteria. Left-ventricular ejection fraction by modified biplane Simpson method and mass index were estimated. Peak early (E) and late (A) diastolic velocities, deceleration time, left-ventricular isovolumic relaxation time, and myocardial performance index were obtained using standard Doppler practices. Right-ventricular systolic pressure (RVSP) was determined by continuous wave Doppler echocardiography. Two-dimensional measurements of aortic diameters were made at end-diastole in parasternal long-axis views at four levels: aortic annulus, sinuses of Valsalva, sinotubular junction, and proximal ascending aorta. Mitral and tricuspid annulus velocities (Sₐ, Eₐ, Aₐ) were measured by tissue Doppler imaging on the transthoracic four-chamber views.
The elastic properties of the aorta were indexed by calculation\(^\text{29–31}\) of aortic distensibility (D), stiffness index (SI), and pressure–strain elastic modulus (Ep) as
\[
D = \frac{2(A_s - A_d)}{A_d (P_s - P_d)}, \quad SI = \frac{\ln(P_s / P_d)}{(A_s - A_d)/A_d}, \quad \text{and} \quad Ep = \frac{(P_s - P_d)}{(A_s - A_d)/A_d},
\]
respectively, where \(A_s\), aortic diameter at end-systole; \(A_d\), aortic diameter at end-diastole; \(P_s\), systolic blood pressure; \(P_d\), diastolic blood pressure; and \(\ln\), natural logarithm.

Aortic wall TDI velocities have been obtained as previously described.\(^\text{32}\) By marking a region of interest on the 2D image in the anterior aspect of the ascending aorta at the same point as in M-mode measurements, velocities throughout the cardiac cycle for this area can be determined (Figure 1B). Offline analysis of the velocity data sets was performed using dedicated software (EchoPAC BT11, GE Ultrasound). Tissue Doppler imaging tracing displayed accelerated

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**Figure 1** Application of echocardiographic techniques in an athlete. (A). Speckle-tracking imaging (STI) and LV longitudinal strain. (B). Ascending aortic wall TDI velocity profile; aortic wall peak systolic radial strain is derived from velocity data. \(A_{Ao}\) = wall late diastolic velocity; \(E_{Ao}\) = wall early diastolic velocity; \(S_{Ao}\) = wall systolic velocity.
expansion of the aortic wall followed by a slow deceleration, a plateau, and then a rapid deceleration into diastole. This trace represents the mean of the instantaneous velocity spectrum. Systolic maximum wall expansion velocity (AoSvel, cm/s), wall contraction early diastolic velocity (AoEvel, cm/s), and wall peak systolic radial strain (AoS, %) were derived.

**Exercise stress-echo**

Exercise stress-echo test was undertaken by bicycle ergometry in the supine position, with initial workload set at 25 W and 25 W increments every 2 min. Heart rate and rhythm were continuously recorded by 12-lead ECG, and blood pressure was measured manually during the last 30 s of each stage by sphygmomanometry. The following functional and echocardiographic indexes were assessed at peak effort: maximal HR, maximal systolic blood pressure (SBP), maximal workload (number of Watts achieved by bicycle test), rate–pressure product (maximal HR x maximal SBP), LVEF, annular early diastolic velocity (Ea), left-ventricular longitudinal strain (LV LS), left-ventricular torsion (LVtor), aortic wall systolic velocity (AoSvel), RVEF, right-ventricular longitudinal strain (RV LS).

**Statistics**

Data are presented as mean value ± SD. Linear correlations, univariate and multivariate analysis were used for comparisons. Variables were compared between groups by Student’s t-test. Differences were considered statistically significant when the P-value was < 0.05. To analyse intraobserver variability, measurements of 2D strain parameters were made at multiple sites in different patients on two different occasions. For interobserver variability, a second investigator randomly made measurements at the above different sites without knowledge of the other echocardiographic parameters. The intraobserver and interobserver variabilities were determined as the difference between the two sets of observations divided by the mean of the observations and expressed as a percentage.

**Results**

Intraobserver variability was 4.6 ± 2.8% for 3D-LVEDV, 5.3 ± 4.9% for 3D-LVESV, 4.8 ± 3.6% for 3D-RVEDV, 5.7 ± 5.8% for 3D-RVESV, 7.9 ± 3.3% for STI longitudinal strain, 9.7 ± 5.4% for STI strain rate, and 4.7 ± 3.2% for AoSvel. Interobserver variability was 5.1 ± 3.3% for 3D-LVEDV, 6.2 ± 5.6% for 3D-LVESV, 5.4 ± 3.1% for 3D-RVEDV, 8.2 ± 5.6% for 3D-RVESV, 9.6 ± 4.1% for STI longitudinal strain, 15.8 ± 6.4% for STI strain rate, and 5.9 ± 4.2% for AoSvel.

With regard to the data obtained with exercise, intraobserver variability was 8.1 ± 3.8% for 3D-LVEDV, 10.9 ± 5.8% for 3D-LVESV, 8.6 ± 4.4% for 3D-RVEDV, 11.3 ± 6.1% for 3D-RVESV, 11.7 ± 3.6% for STI longitudinal strain, 16.3 ± 4.6% for STI strain rate, and 6.2 ± 4.2% for AoSvel. Interobserver variability was 9.3 ± 5.2% for 3D-LVEDV, 13.8 ± 6.5% for 3D-LVESV, 9.9 ± 4.5% for 3D-RVEDV, 14.9 ± 7.8% for 3D-RVESV, 13.3 ± 4.9% for STI longitudinal strain, 18.8 ± 6.7% for STI strain rate, and 7.7 ± 5.8% for AoSvel.

The overall feasibility of speckle-tracking echocardiography at rest and peak stress was 94% and overall feasibility of 3D echocardiography was 87%.

The baseline age, anthropometric, and clinical characteristics are given in Table 1. Mmode/2D findings are given in Table 2.

**3D volumetry in athletes and controls**

ETA and MTA experienced LV eccentric hypertrophy, characterized by increased LV end-diastolic volume and mass (Figure 2). Three-dimensional LVEDV, LVESV, RVEDV, and RVESV were enlarged in athletes compared with controls (Table 3). A close association between 3D LV and RV end-diastolic volume in the three groups of athletes was found (r = 0.72, P < 0.001). On multiple linear regression analysis, the body surface area, type and duration of training, and LV end-diastolic volume were the only independent determinants of RV end-diastolic volume (F = 73.2, P < 0.005).

**TDI–STI parameters in athletes and controls**

LV diastolic function measurements such as mitral Ea and tricuspid Ee were increased in athletes compared with the control group (Table 3).

| Table 1 | Age, anthropometric, and clinical characteristics of the study population |
|-----------------|-----------------|-----------------|-----------------|-----------------|------------------|
|                | Controls (n = 35) | ETA (n = 35)    | STA (n = 35)    | MTA (n = 35)    | P-value          |
| Age            | 28.3 ± 11.4     | 28.7 ± 10.7     | 30.3 ± 9.4      | 29.4 ± 9.8      | NS               |
| Body surface area (cm²) | 1.89 ± 0.23     | 1.91 ± 0.15     | 1.98 ± 0.18     | 1.93 ± 0.13     | NS               |
| Body mass index (kg/m²) | 22.4 ± 2.4     | 22.7 ± 2.6      | 23.4 ± 3.1      | 22.9 ± 2.5      | 0.05*            |
| Training experience (years) | /              | 9.6 ± 2.9       | 9.1 ± 3.2       | 9.7 ± 3.1       | NS               |
| HR (bpm)       | 69.5 ± 9.1      | 54.1 ± 5.6      | 65.8 ± 8.6      | 60.1 ± 7.4      | <0.005‡          |
| SBP (mmHg)     | 120.6 ± 7.2     | 121.9 ± 6.8     | 129.1 ± 7.4     | 123.2 ± 7.1     | 0.03‡            |
| DBP (mmHg)     | 75.2 ± 5.7      | 71.3 ± 5.4      | 76.3 ± 4.8      | 70.9 ± 4.6      | NS               |
| Double product (SBP x HR) | 7335 ± 1253 | 6731 ± 1249     | 8247 ± 1459     | 7121 ± 1231     | 0.01‡            |
| RVSP           | 21.6 ± 6.2      | 28.4 ± 7.3      | 23.4 ± 6.5      | 27.6 ± 7.6      | 0.05‡            |

DBP, diastolic blood pressure; ETA, endurance-trained athletes; HR, heart rate; MTA, mixed-trained athletes; NS, not significant; RVSP, right-ventricular systolic pressure; SBP, systolic blood pressure; STA, strength-trained athletes.

*STA vs. controls.
†ETA vs. controls.
‡STA vs. ETA, MTA, and controls.
§ETA and MTA vs. controls.
LV longitudinal strain increased from base to apex in all groups of athletes. There was a significant increase in peak systolic apical rotation and peak LV tor in ETA and MTA (Figure 2, Table 3). Global RV STI longitudinal strain was significantly lower than control group in all athletes and was especially reduced in basal segments (Table 3). Right-ventricular systolic pressure (RVSP) was obtained by tricuspid regurgitation in 79/105 athletes (Table 1). In these athletes, there was a close relation between RVSP and RV LS ($r = -0.76, P < 0.001$).

TDI-derived indexes of Ao function and correlation with LV function

Aortic diameters at all levels were significantly larger in STA than in ETA, MTA, and controls (Table 2). Aortic wall velocities and strain were significantly decreased in STA ($P < 0.001$) and significantly increased in MTA ($P < 0.05$) and ETA ($P = 0.003$) compared with controls (Figure 2, Table 3). By univariate analysis there was a good correlation between LV remodelling and aortic function.
Multivariate analysis showed aortic wall velocities and strain to be independently related to the body surface area, type and duration of training, and LV mass index ($F = 74.1$, $P < 0.001$).

**Bicycle ergometric test**

During maximal physical effort (Table 4), ETA and MTA showed a better functional capacity compared with STA, with greater maximal workload achieved with lower maximal heart rate and systolic blood pressure. The baseline increase in peak systolic apical rotation translated into a highly significant increase in peak systolic LV torsion after exercise. Right-ventricular strain was reduced at rest (Table 3) and improved after exercise (Table 4). Decreased AoSvel values persisted in STA during maximal physical effort (Table 4).

**Discussion**

To our knowledge, a study assessing comprehensively biventricular function and aortic distensibility in athletes with different forms of training with the use of 3DE and strain imaging has not been reported. Novel findings of this study in athletes were the assessment of 3D RV–LV volumes, the assessment of behaviour of RV–LV deformation and torsion parameters and assessment of TDI aortic wall properties. In athletes subjected to purer endurance-type training, we found eccentric hypertrophy characterized by increased LV end-diastolic volume and mass as well as LV torsion. Aortic wall velocities and strain were significantly increased in endurance athletes and significantly reduced in power-trained athletes compared with controls. The martial arts athletes performing combined strength and endurance training presented a significant increase in left-ventricular end-diastolic volume and torsion and aortic wall velocities and strain but with lower significance values when compared with endurance athletes. All athletes had increased LV longitudinal strain and decreased RV longitudinal strain. A good correlation was shown between left- and right-ventricular volumes and between LV function and aortic parameters.

**LV and RV volumes**

Marathon athletes, involved in a high dynamic component of aerobic training, developed an increase in the size of left- and right-ventricular chambers, with a proportional increase in LV thickness and volume caused by volume overload, thus showing eccentric LV hypertrophy. On the other hand, power athletes, involved in exercises with predominantly anaerobic component, showed an increase in LV wall thickness and relative wall thickness, with a geometric pattern of concentric hypertrophy due to the high systolic systemic blood pressure caused by this type of exercise. Recent studies7–9 considered that the classification of left-ventricular hypertrophy in athletes as eccentric or concentric is not an absolute or dichotomous concept but rather a relative concept. It should also be noted that genetic factors as well as the somatotype could play a significant role for the cardiac dimensions in addition to the body dimensions.33 In our study, athletes of martial arts with mixed training developed an intermediate pattern of adaptation with increased cardiac volumes albeit to a lesser extent than marathon runners. The use of 3DE helped to

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**Figure 2** Three-dimensional left-ventricular end-diastolic volume index (3D-LVEDVI) (A), LV mass index (LVMI) (B), LV torsion (LVtor) (C), and aortic wall velocity (AoSvel, D) in controls (CTR), endurance-trained athletes (ETA), strength-trained athletes (STA), and martial arts athletes (MTA). *$P < 0.005$, †$P = 0.01$, ‡$P = 0.02$, §$P = 0.05$, ¶$P = 0.01$, #$P = 0.02$, $**P < 0.005$ (all $P$-values vs. CTR).
### Table 3  3D, TDI, and STI values in athletes and controls

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controls (n = 35) mean ± SD</th>
<th>ETA (n = 35) mean ± SD</th>
<th>STA (n = 35) mean ± SD</th>
<th>MTA (n = 35) mean ± SD</th>
<th>P-value</th>
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<tbody>
<tr>
<td><strong>LV</strong></td>
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<tr>
<td>3D-LVEDV (mL)</td>
<td>126 ± 21</td>
<td>193 ± 26</td>
<td>154 ± 23</td>
<td>179 ± 27</td>
<td>&lt;0.005*, 0.03†</td>
</tr>
<tr>
<td>3D-LVEDVI (mL/m²)</td>
<td>64 ± 14</td>
<td>103 ± 11</td>
<td>82 ± 13</td>
<td>95 ± 10</td>
<td>&lt;0.005*, 0.01†</td>
</tr>
<tr>
<td>AoEvel (cm/s)</td>
<td>7.2 ± 0.8</td>
<td>10.1 ± 0.8</td>
<td>6.2 ± 0.7</td>
<td>6.5 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>AoSvel (cm/s)</td>
<td>6.2 ± 0.8</td>
<td>10.1 ± 0.8</td>
<td>6.2 ± 0.7</td>
<td>6.5 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>AoS (%)</td>
<td>8.5 ± 1.4</td>
<td>12.2 ± 2.3</td>
<td>7.2 ± 0.8</td>
<td>7.6 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>AoS (%)</td>
<td>8.5 ± 1.4</td>
<td>12.2 ± 2.3</td>
<td>7.2 ± 0.8</td>
<td>7.6 ± 0.8</td>
<td>NS</td>
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<tr>
<td><strong>TDI</strong></td>
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<tr>
<td>Global LS (%)</td>
<td>−20.3 ± 2.6</td>
<td>−21.7 ± 2.6</td>
<td>−22.5 ± 2.4</td>
<td>−21.6 ± 2.2</td>
<td>&lt;0.05†</td>
</tr>
<tr>
<td>Global RS (%)</td>
<td>48.9 ± 9.7</td>
<td>46.9 ± 9.4</td>
<td>49.6 ± 8.5</td>
<td>47.5 ± 8.7</td>
<td>NS</td>
</tr>
<tr>
<td>Global CS (%)</td>
<td>−24.7 ± 3.4</td>
<td>−22.9 ± 3.3</td>
<td>−24.1 ± 2.7</td>
<td>−22.6 ± 3.6</td>
<td>NS</td>
</tr>
<tr>
<td>Peak LVtor (degrees)</td>
<td>14.6 ± 4.3</td>
<td>21.5 ± 5.2</td>
<td>15.8 ± 4.5</td>
<td>20.8 ± 5.4</td>
<td>0.01*</td>
</tr>
<tr>
<td>Peak apical rotation (degrees)</td>
<td>10.1 ± 3.6</td>
<td>14.2 ± 4.3</td>
<td>10.8 ± 3.7</td>
<td>13.8 ± 3.9</td>
<td>0.05*</td>
</tr>
<tr>
<td>Peak basal rotation (degrees)</td>
<td>−6.7 ± 2.3</td>
<td>−7.7 ± 2.2</td>
<td>−6.8 ± 1.9</td>
<td>−7.6 ± 2.4</td>
<td>NS</td>
</tr>
<tr>
<td>Normalized peak LVtor (degrees/cm)</td>
<td>1.8 ± 0.4</td>
<td>2.5 ± 0.5</td>
<td>1.9 ± 0.8</td>
<td>2.4 ± 0.6</td>
<td>0.03*</td>
</tr>
<tr>
<td>Peak UTV (degrees/s)</td>
<td>78.9 ± 15</td>
<td>93.9 ± 21</td>
<td>83.1 ± 16</td>
<td>92.2 ± 22</td>
<td>NS</td>
</tr>
<tr>
<td>Normalized time to peak UTV (%)</td>
<td>16.5 ± 7.1</td>
<td>20.3 ± 8.3</td>
<td>17.5 ± 5.3</td>
<td>19.4 ± 6.7</td>
<td>NS</td>
</tr>
<tr>
<td>UTR (degrees/s)</td>
<td>61.7 ± 24</td>
<td>94.2 ± 29</td>
<td>64.2 ± 23</td>
<td>80.6 ± 31</td>
<td>0.03*</td>
</tr>
<tr>
<td><strong>Aorta</strong></td>
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<tr>
<td>AoSvel (cm/s)</td>
<td>6.2 ± 0.4</td>
<td>7.2 ± 0.6</td>
<td>4.7 ± 0.3</td>
<td>6.8 ± 0.5</td>
<td>0.04*, &lt;0.005†</td>
</tr>
<tr>
<td>AoEvel (cm/s)</td>
<td>7.2 ± 0.8</td>
<td>7.5 ± 0.8</td>
<td>4.3 ± 0.6</td>
<td>7.7 ± 0.4</td>
<td>0.04†</td>
</tr>
<tr>
<td>AoS (%)</td>
<td>8.5 ± 1.4</td>
<td>9.9 ± 1.6</td>
<td>5.5 ± 1.3</td>
<td>9.6 ± 1.3</td>
<td>0.02*, &lt;0.005†</td>
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<tr>
<td><strong>RV</strong></td>
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</tr>
<tr>
<td>3D-RVEDV (mL)</td>
<td>77 ± 19</td>
<td>115 ± 12</td>
<td>96 ± 12</td>
<td>112 ± 17</td>
<td>0.01*, 0.03†</td>
</tr>
<tr>
<td>3D-RVEDVI (mL/m²)</td>
<td>40 ± 8</td>
<td>63 ± 10</td>
<td>54 ± 10</td>
<td>59 ± 12</td>
<td>0.01*, 0.02†</td>
</tr>
<tr>
<td>3D-RVEF</td>
<td>56 ± 7</td>
<td>55 ± 4</td>
<td>52 ± 7</td>
<td>54 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td><strong>TDI</strong></td>
<td></td>
<td></td>
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<tr>
<td>TV E’ (cm/s)</td>
<td>11.6 ± 2.4</td>
<td>19.6 ± 4.7</td>
<td>15.5 ± 3.2</td>
<td>18.9 ± 4.3</td>
<td>0.02*, &lt;0.05†</td>
</tr>
<tr>
<td>TV E/Ea</td>
<td>4.6 ± 1.1</td>
<td>3.4 ± 0.7</td>
<td>2.4 ± 0.6</td>
<td>2.9 ± 1.1</td>
<td>0.04*, 0.01†</td>
</tr>
<tr>
<td><strong>STI</strong></td>
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<tr>
<td>RV Global LS (%)</td>
<td>−28.2 ± 3.8</td>
<td>−26.7 ± 2.9</td>
<td>−27.4 ± 3.4</td>
<td>−26.3 ± 3.2</td>
<td>0.03*, &lt;0.05†</td>
</tr>
<tr>
<td>RV FW Basal LS (%)</td>
<td>−25.7 ± 2.9</td>
<td>−21.2 ± 2.3</td>
<td>−22.3 ± 2.2</td>
<td>−21.5 ± 2.4</td>
<td>0.01*, 0.04†</td>
</tr>
<tr>
<td>RV FW Apical LS (%)</td>
<td>−29.8 ± 4.2</td>
<td>−29.3 ± 4.8</td>
<td>−29.8 ± 4.3</td>
<td>−28.9 ± 4.6</td>
<td>NS</td>
</tr>
</tbody>
</table>

AoEvel, aortic wall early diastolic velocity; AoS, aortic wall peak radial strain; AoSvel, aortic wall systolic velocity; CS, circumferential strain; E, mitral inflow early diastolic velocity; Ee, annular early diastolic velocity; EF, ejection fraction; FW, free wall; LS, longitudinal strain; LV, left ventricle; LVM, left-ventricular mass; LVMi, left-ventricular mass index; LVt, late-ventricular torsion; MV, mitral valve; NS, not significant; RS, radial strain; RV, right ventricle; time to peak UTV, time to peak untwisting velocity (expressed as percentage of duration of diastole); UTR, untwisting rate; UTV, untwisting diastolic velocity; TV, tricuspid valve; 3D-LVEDV, three-dimensional left-ventricular end-diastolic volume; 3D-LVEDVI, three-dimensional left-ventricular end-diastolic volume index; 3D-LVEF, three-dimensional left-ventricular ejection fraction; 3D-RVEDV, three-dimensional right-ventricular end-diastolic volume; RVEDVI, three-dimensional right-ventricular end-diastolic volume index; 3D-RVEF, three-dimensional right-ventricular ejection fraction.

*ETA and MTA vs. controls.
†STA vs. controls.
‡ETA, MTA, and STA vs. controls.
assess left- and right-ventricular volumes and better characterize different cardiovascular adaptations. This is in keeping with previous reports\textsuperscript{21} that showed that 3DE offers more detailed information on LV remodelling compared with 2DE since it takes into account differences in the length and shape of the LV chamber and provides data on LV geometry, function, and synchronicity of contraction, differently from patients with hypertrophic or dilated cardiomyopathy that do not show LV harmonic remodelling.

Our results have also highlighted a close association between LV and RV end-diastolic volumes in the three groups of athletes. Multivariate analysis has provided additional information about this association with adjustment for several confounding factors. These independent associations point to a mutual dependency between the two ventricles in the trained heart. Overload alterations may be highly obvious in athletes that represent a model of extreme physiological haemodynamic load with a large increase in training-induced LV stroke volume. Ventricular interaction is the expression of the close anatomical association between the two ventricles, which are surrounded by common muscle fibres, share a septal wall, and are enclosed within the pericardium. Left-ventricular volumetric expansion, changing the location and size of the ventricular septum, also causes deformation of RV walls as well as RV pressure and relaxation rate changes.

**LV and RV wall velocities and strain**

Tissue Doppler findings showed higher early diastolic myocardial velocity and E/E\textsubscript{a} ratio in LV and RV lateral walls in ETA and MTA compared with controls confirming previous reports\textsuperscript{14–16}. These observed differences in biventricular diastolic behaviour suggest a supernormal diastolic function of athlete's heart. Since peak velocity and E/E\textsubscript{a} ratio are the expression of abnormal atrio-ventricular pressure gradient and passive diastolic properties of ventricular walls, their increase in ETA and MTA suggests a training-induced improvement of left- and right-ventricular compliance.

LV longitudinal strain increased significantly from base to apex in all groups of athletes. Previous studies have used STI to characterize both athlete’s heart and pathological hypertrophy\textsuperscript{17,18}. Both global LS (cutoff point <−19%) and E/E\textsubscript{a} ratio (cutoff point ≤6.16) appeared to be accurate to discriminate between controls and hypertensive patients, with E/E\textsubscript{a} ratio being more sensitive and GLS more specific\textsuperscript{21}. It is known that the ‘physiological’ hypertrophy in athletes shows increased ventricular mass and normal organization of cardiac structure with no increase in collagen content, whereas hypertrophic cardiomyopathy is associated with structural myofibrillar changes, collagen accumulation, and potential systolic and diastolic dysfunction. In our athletes, longitudinal strain was higher compared with controls, whereas radial and circumferential strains were not significantly different. This is consistent with previous studies that compared various types of LV systolic deformation and found that the behaviour of longitudinal systolic function of subendocardial fibres allowed to distinguish trained athletes and patients with systemic hypertension\textsuperscript{35}.

Left-ventricular torsion, a key determinant in LV function in cardiac diseases, has recently been analysed in the setting of exercise\textsuperscript{18,36}. In addition to the systolic twist, the subsequent early diastolic untwisting is an important marker of LV filling. Differently from patients with pathological LV hypertrophy who do not experience torsion/untwisting augmentation during exercise, LV torsion and untwisting rate have been shown to increase during laboratory-based submaximal exercise in healthy persons\textsuperscript{18}. In athletes, some discordant data have been reported and both increased or decreased torsion after training have been described depending on study population, study design, and measurement technique\textsuperscript{36}. We found a different behaviour of LV torsion after exercise in endurance and strength athletes since in marathon and martial arts athletes we found a baseline increase in peak systolic apical rotation that translated into a highly significant increase in peak systolic LV torsion after exercise. The same increase was not shown in power athletes. The underlying mechanisms responsible for this heterogeneous response of LV torsion are not well understood. Left-ventricular torsion results from a complex arrangement of wall myocardial fibres and its progressive increase during effort could reflect a higher contribution of subepicardial vs. subendocardial layers blunted by local ischaemia. Another explanation could be based on the regular decrease in LV end-diastolic diameters observed with exercise intensity that could enhance subepicardial layers. The current uncertainty regarding
the significance of high resting LV torsion suggests that using these parameters to differentiate physiological and pathological LV remodelling is not feasible at the present time. The examination of LV torsion ‘reserve’ during exercise in athletes may provide further insight in the future.

RV function assessment in athletes is important due to the possible overlap between athlete’s heart and ARVD especially in the presence of ventricular arrhythmias. Long-lasting RV volume overload could be the mechanism contributing to the development of RV structural changes leading to functional abnormalities. A number of studies using 2D echocardiography have demonstrated transient RV systolic dysfunction in response to high-intensity sports activity such as marathon running.22,37,38 It has been suggested37 that the adaptation to volume and pressure stress might spatially differ within the RV as a result of differences in wall stress between the apical and the inflow tract and that the basal portion of the RV might be more vulnerable and prone to preferential dilatation and reduced strain, or both. Our results are partially in concordance with these findings. The greater regional heterogeneity in RV strain compared with controls was more pronounced in endurance vs. power athletes and this seems to be related to increased pulmonary artery pressure as well as to ventricular dilatation. However, there was an improvement of RV strain after exercise with a behaviour similar to control subjects. One can speculate that these ‘normal’ responses would differ from those seen in patients with RV disease (such as ARVD or pulmonary hypertension) but it remains to be demonstrated that RV dysfunction is accentuated with exercise using these new echocardiographic techniques.

The quantification of strain and strain rate as well as 3D ejection fraction during exercise has proved to be difficult in the present study as in previous reports.39–41 However, offline STI analysis was feasible in most subjects after strenuous levels of exercise. For two-dimensional parameters, the greatest limitation was the effective frame rate which compromised frame-to-frame tracking of the fast contracting myocardium while for Doppler indices cardiac translation due to respiration increased the noise-to-signal ratio due to several artefacts. Although the superior temporal resolution of Doppler modalities is an advantage for exercise measures allowing these techniques to be more sensitive to dynamic changes, it has been shown39 they have a greater variability in quantification of myocardial values at all levels of exercise compared with two-dimensional methods. As regards 3D echocardiography, the resolution of the images was not as high as that obtained with conventional scanners, due to the lower frame rates that limited its feasibility. Another strong limitation to feasibility and reproducibility was high heart rates reached at the peak of the stress test. However, in this study as in previous reports,40 quantifications of left-ventricular volumes were reproducible also when heart rate was high or resting images of sub-optimal quality.

**Ao wall velocities and strain**

Elastic properties of the aorta39 are important factors supporting LV function. The functions of the aorta are not only those of a blood conduit for tissues, but also an important modulator of the entire cardiovascular system, which hinder cardiac intermittent pulsatile flow to provide a constant flow in the capillary bed. By virtue of its elastic properties, the aorta influences left-ventricular function and coronary flow. Previous studies5 have shown an increase in the elastic properties of these endurance athletes and several cardiovascular adaptations in power athletes related to intermittent elevations of blood pressure during exercise in muscle strength. It was also suggested that in these athletes, the transient increase in cardiac output associated with high elevation of systemic blood pressure may cause a chronic increase in wall tension responsible for aortic dilatation and regurgitation. In a

<table>
<thead>
<tr>
<th>Bicycle ergometer</th>
<th>ETA (n = 35)</th>
<th>STA (n = 35)</th>
<th>MTA (n = 35)</th>
<th>CTR (n = 35)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal HR (bpm)</td>
<td>165.9 ± 15.4</td>
<td>180.2 ± 13.7</td>
<td>168.8 ± 12.9</td>
<td>177.4 ± 14.2</td>
<td>0.04*, &lt;0.05†</td>
</tr>
<tr>
<td>Maximal SBP (mmHg)</td>
<td>173.49 ± 24.7</td>
<td>195.11 ± 24.1</td>
<td>175.82 ± 23.1</td>
<td>165.16 ± 22.2</td>
<td>0.03*, 0.04‡</td>
</tr>
<tr>
<td>Rate–pressure product (bpm × mmHg × 10⁻³)</td>
<td>30.8 ± 3.1</td>
<td>34.6 ± 2.2</td>
<td>31.8 ± 3.3</td>
<td>28.4 ± 3.5</td>
<td>0.03*, &lt;0.05†</td>
</tr>
<tr>
<td>Maximal workload achieved (W)</td>
<td>207.42 ± 33.9</td>
<td>173.31 ± 30.8</td>
<td>197.18 ± 36.5</td>
<td>157.13 ± 35.3</td>
<td>0.02*, 0.03‡</td>
</tr>
<tr>
<td>3D-LVEF</td>
<td>68 ± 8</td>
<td>67 ± 5</td>
<td>67 ± 9</td>
<td>66 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>MV E (cm/s)</td>
<td>22.2 ± 3.7</td>
<td>16.1 ± 3.3</td>
<td>21.6 ± 3.8</td>
<td>15.2 ± 2.9</td>
<td>&lt;0.05‡</td>
</tr>
<tr>
<td>LV Global LS (%)</td>
<td>−24.2 ± 3.3</td>
<td>−25.1 ± 2.7</td>
<td>−26.5 ± 2.4</td>
<td>−24.9 ± 2.5</td>
<td>NS</td>
</tr>
<tr>
<td>Peak LVtorn (degrees)</td>
<td>23.8 ± 4.7</td>
<td>16.9 ± 4.6</td>
<td>23.6 ± 4.9</td>
<td>17.2 ± 4.5</td>
<td>&lt;0.05‡</td>
</tr>
<tr>
<td>Ao Svel (cm/s)</td>
<td>7.4 ± 0.5</td>
<td>5.3 ± 0.6</td>
<td>6.9 ± 0.7</td>
<td>6.7 ± 0.5</td>
<td>&lt;0.05‡</td>
</tr>
<tr>
<td>3D-RVEF</td>
<td>60 ± 9</td>
<td>58 ± 6</td>
<td>59 ± 8</td>
<td>59 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>TV E (cm/s)</td>
<td>22.1 ± 3.5</td>
<td>18.1 ± 4.3</td>
<td>21.7 ± 3.8</td>
<td>14.8 ± 4.4</td>
<td>&lt;0.05‡</td>
</tr>
<tr>
<td>RV Global LS (%)</td>
<td>−28.9 ± 2.9</td>
<td>−29.7 ± 2.6</td>
<td>−29.3 ± 2.7</td>
<td>−30.2 ± 2.5</td>
<td>NS</td>
</tr>
</tbody>
</table>

AoSvel, aortic wall systolic velocity; CTR, controls; E, annular early diastolic velocity; EF, ejection fraction; HR, heart rate; LS, longitudinal strain; LV, left ventricle; LVtorn, left-ventricular torsion; MV, mitral valve; NS, not significant; RV, right ventricle; SBP, systolic blood pressure; TV, tricuspid valve.

*ETA vs. STA. †MTA vs. STA. ‡ETA and MTA vs. CTR. § STA vs. CTR.
recent study, the aortic root diameter was significantly higher in power athletes than in endurance athletes matched for age and sex. However, a significant dilation of the ascending aorta and the presence of aortic regurgitation proved uncommon. Other studies have applied standard echocardiographic techniques to the assessment of elastic properties and diameters in top-level athletes and shown that aortic root diameters and stiffness were significantly greater in strength-trained athletes, while aortic distensibility was higher in endurance athletes compared with age- and sex-matched healthy controls.

We have studied aortic wall distensibility as well as wall velocity and strain with tissue Doppler techniques. The aortic wall velocities and strain were significantly increased in endurance and maximal athletes and significantly reduced in power athletes compared with controls. In power athletes, the decreased values persisted during maximal physical effort. These data can be interpreted as a result of a physiological ventricular–vascular coupling. The increase in aortic distensibility in marathon runners and athletes in aortic studies can cause an improvement in LV diastolic function as a factor of physiological adaptation. In power athletes, the greater aortic stiffness has a potential correlation with the ventricular wall stress. In response to increased afterload or increased blood pressure, LV hypertrophy reflects a compensatory mechanism to decrease wall stress and these changes may be a cardiovascular adaptation of the isometric exercise in harmony with a normalization of systolic and diastolic function. It has been speculated that changes in wall thickness and lumen diameter may reflect adaptations consistent with the Laplace of arterial and normalization of wall stress. The mechanism underlying the differences in arterial stiffness between endurance- and strength-trained athletes is not completely clear. It is possible that some changes in ventricular deformation and stiffness cannot be assessed by using conventional echocardiographic techniques. With regard to vascular function, we have previously shown that TDI values in the ascending aorta had a higher discriminating power in differentiating hypertensive patients from controls. Our present data in athletes show that TDI values in the ascending aorta, while using a faster methodology to be applied, have a higher significance in differentiating athletes from controls compared with stiffness index and distensibility assessed by M-mode echocardiography.

**Limitations**

Some limitations should be pointed out. First, technical limitations of strain Doppler echocardiography should be considered. A technical limitation is that speckle-tracking echocardiography is dependent both on frame rate and image resolution. In addition, the endocardial border tracing must be manually optimized. We found frame rates in the range of 60–100 Hz suitable for speckle-tracking analysis, that is a lower value than frame rate available with Doppler strain. However, STI may overcome some TDI limitations by assessing myocardial thickening in a manner less affected by passive translational motion or tethering. Also we have not directly estimated the exact distance between the two short-axis levels, which could have affected LV torsion measurements, nor have we selected reproducible anatomic landmarks for measuring apical rotation although we defined the apical level just proximal to the level with LV luminal obliteration at the end-systolic period. Furthermore, only one vessel bed (ascending aorta) was examined to assess aortic stiffness, whereas in different points the arterial wall distensibility may be different. Last, the accuracy of 3D volume and EF is related to the effects of endocardial trabeculation that play an important role in the modularity discorances in LV volume measurements, and could certainly be expected to affect RV measurements even more, since the RV is more heavily trabeculated. Moreover, the accuracy of 3D volume and EF is less certain in significantly dilated right ventricles since limited data are available in the literature.

**Conclusions**

The combined application of 3DE, TDI, and STI has shown that ventricular and vascular response underlie different adaptations of ventricular volumes and arterial stiffness in strength-, endurance-, and mixed-trained athletes. Future studies in this direction with a larger sample size and longitudinal study design will certainly increase our knowledge of cardiovascular adaptation of the athletes.

**Conflict of interest:** none declared.

**References**


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