Left ventricular systolic performance is improved in elite athletes

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Aims

We sought to investigate the systolic time interval (STI) and efficiency of left ventricular (LV) contraction comparatively in elite athletes and healthy sedentary controls by means of three-dimensional echocardiography (3DE).

Methods and results

Four hundred and twenty-nine elite athletes, involved in skill (n = 41), power (n = 63), mixed (n = 167), and endurance (n = 158) disciplines and 98 sedentary controls, matched for age, underwent 3DE. By off-line analysis, we measured the absolute and relative (normalized by the R-R interval) timing of LV systolic emptying (STI and STI%) and the systolic flow velocity (SFV = stroke volume/STI). Both STI and STI% were shorter in athletes, regardless of the sport discipline, compared with controls (respectively, 324 ± 36 vs. 345 ± 33 ms, P < 0.001; 30 ± 4 vs. 40 ± 4%; P < 0.001). Regression analysis showed that heart rate was the most important determinant of STI (R² = 0.38; P < 0.001), while age, body surface area, blood pressure, LV volumes, and mass had no significant association. After removing the effects of heart rate and gender, athletes showed a significant reduction (by 50.4 ms; 95% confidence interval, from 57.7 to 43.1) in STI compared with untrained subjects. Finally, higher SFV were identified in skill (256 ± 60 mL/s; P < 0.001), strength (297 ± 78 mL/s; P < 0.001), mixed (308 ± 67 mL/s; P < 0.001), and endurance (334 ± 74 mL/s; P < 0.001) athletes compared with controls (204 ± 50 mL/s).

Conclusion

Elite athletes show a significant shortening of the systolic time duration in comparison with sedentary controls, in association with a significant increase in LV emptying velocity. This pattern characterizes the physiological LV adaptation of the athletes and may potentially be useful in differential diagnosis of the ‘athlete heart’.

Keywords

Athletes • Left ventricle • Myocardial contraction • 3D echocardiography • Systole

Introduction

Morphological remodelling of the left ventricle (LV) has been extensively described in highly trained athletes, including increased cavity size, wall thickness, and mass, which represents the physiological response to the haemodynamic loading conditions induced by chronic exercise. Although several reports have focused on the characteristics and limits of morphological LV adaptations, less is known regarding the occurrence of changes in LV performance as a consequence of athletic conditioning. Namely, it is not clear whether morphological changes in trained athletes are associated with enhanced LV systolic performance in order to sustain the increased haemodynamic load associated with chronic exercise.

In recent years, three-dimensional echocardiography (3DE) has been increasingly used to gather quantitative information on heart morphology and geometry and has proved to accurately assess LV volumes and mass in trained athletes. This technology allows a dynamic analysis of LV volume changes over the heart cycle, thereby identifying systolic and diastolic time intervals with accuracy.

Therefore, in the present analysis, we sought to comparatively assess the LV systolic time duration and mechanical efficiency in a large population of elite athletes and sedentary controls, by using 3DE.

Methods

Study population

The Institute of Sports Medicine and Science is the medical division of the Italian National Olympic Committee, where all national team members undergo periodical medical evaluation before taking part in

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LV systolic performance in elite athletes

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major international competitions. Within this programme, in the period from March to June 2008, we evaluated 434 consecutive athletes, selected for participation in the 2008 Beijing Olympic Games.

Cardiovascular evaluation consisted of taking medical history, a physical examination, a resting and exercise 12-leads electrocardiogram, and two-dimensional and Doppler echocardiography. Five subjects were excluded because of evidence of cardiac abnormalities (n = 2), complex ventricular arrhythmias (n = 2), and systemic hypertension (n = 1). The remaining 429 athletes underwent a 3DE examination.

All the athletes have been involved in vigorous training protocols for ≥ 3 years. Mean age was 26 ± 5 years (range, 15–45 years), and 337 were male (78%). Athletes were engaged in a broad spectrum of 27 different disciplines, which were arbitrarily grouped in four major categories according to a modified Mitchell’s classification,12 which considered the predominant characteristics of training: e.g. skill (n = 41), power (n = 63), mixed (n = 167), and endurance (n = 158).

Ninety-eight healthy controls—matched for age (27 ± 4 years, range 18–40 years)—69% males, were also enrolled to take part in the study; they were volunteers with ≤ 3 h of regular exercise practice per week and not involved in sports competitions. Untrained controls underwent medical history, physical examination, a resting and exercise 12-leads electrocardiogram, and two- and 3DE examination.

All subjects agreed to take part in the study and signed an informed consent; the protocol was approved by the institutional review board.

Echocardiography

Echocardiographic examination was performed by using ‘ie33’, a commercially available instrument (Philips, Andover, MA, USA) equipped with an S3 probe (2–4 MHz) for two-dimensional and Doppler measurements and with X3-1 matrix-array transducer (1.9–3.8 MHz) for three-dimensional examination. All the acquisitions were performed by expert cardiologists (F.D.P. and C.P.).

Two-dimensional measurements of LV cavity diameters, wall thickness, left atrium, and aortic root diameters were performed as previously reported.13

Transthoracic harmonic real-time 3DE was performed from the apical four-chamber view by using the full-volume technique. Wide angle acquisition, taking care to include all LV segments, was performed. A high frame rate (31 ± 4 Hz) was obtained by using the 7 beats acquisition protocol, in which seven consecutive wedge-shaped subvolumes are consecutively sampled with a trigger to the R-wave of the electrocardiogram.

The data were then transferred to a separate workstation for off-line analysis by commercially available software (QLab version 5.0, Philips, Andover, MA, USA). LV volumes and mass were obtained by a semi-automatic border-detection technique as previously reported.10 End-diastolic and end-systolic volumes, stroke volume, and ejection fraction were measured. A volume/time curve was generated and the time interval from the beginning of the QRS (at the electrocardiogram) to the minimum systolic volume was considered to be the absolute mechanical systolic time interval (STI; Figure 1). Systolic time interval was also normalized to the duration of the cardiac cycle and expressed as a percentage (STI%). The STI values as here defined were the expression of the overall electro-mechanical systole, including isovolumic contraction and ejection time. The systolic flow velocity (SFV) was calculated as the ratio between stroke volume and STI; it represented a measure of the velocity of LV systolic emptying and an index of LV mechanical performance.

Acquisition and off-line analysis were feasible for all controls and athletes. All the off-line analysis was performed by an operator with specific training on 3DE who was blinded from patients’ data (S.C.).

Reproducibility

In order to assess reproducibility, STI measurements were repeated in a random sample of 30 subjects, by the first investigator (intra-observer variability) at least 1 week after the previous measurement and by a second investigator (inter-observer variability). Investigators were blinded from each other results and patients’ data. Inter- and intra-observer variability were calculated as the difference between the two measurements as a percentage of their mean.

Statistical analysis

Continuous data are expressed as mean ± standard deviation; categorical data are expressed as frequencies. A two-tailed P-value < 0.05 was considered statistically significant. Differences between mean values for continuous variables were assessed by unpaired Student’s t-test or Mann–Whitney’s for variables with non-normal distribution. Difference of male proportions in athletes and controls was assessed by χ² test.14

Stepwise regression analysis was used to assess the impact that several variables [age, body surface area (BSA), systolic and diastolic blood pressure, heart rate, LV volumes, and mass] had on STI those with statistically significant correlation were subsequently incorporated in the covariance analysis together with gender and participation in sports. These two categorical variables were coded using (n – 1) binary dummy variables, and their impact on STI was assessed after removing the effect of continuous covariates. The significance of each variable in the covariance analysis was assessed by the Wald test.14,15

Differences among sport discipline groups in terms of STI% and SFV were calculated using one-way ANOVA with post hoc Bonferroni test.14 Statistical analysis was performed using SPSS software (Version 15.0).
Results

Demographic and echocardiographic characteristics

Demographic and morphological cardiac characteristics of the athletes and untrained controls are reported in Table 1. No significant differences existed in terms of age and systolic and diastolic blood pressure, but BSA was larger in athletes than in controls. Athletes had a larger LV cavity and maximum wall thickness than controls; 3DE-assessed LV end-diastolic and end-systolic volumes, stroke volume, and mass were also larger in athletes, even when indexed by BSA; instead, ejection fraction was not different in the two groups.

Demographic and morphological cardiac features of athletes in relation to the type of sport are shown in Table 2. Endurance athletes showed the largest end-diastolic and end-systolic LV volumes, mass, and stroke volume compared with athletes engaged in mixed, power, and skill disciplines. No difference was found for ejection fraction among the different groups.

Systolic time interval

Both STI and STI% were shorter in athletes compared with those in sedentary subjects (respectively, 324 ± 36 vs. 345 ± 33 ms, P < 0.001; 30 ± 4 vs. 40 ± 4; P < 0.001).

Subgroup analysis of STI and STI% in athletes, according to the sport participated, brought out no significant differences among sport discipline groups (Table 2 and Figure 2).

No gender-related differences were detected in terms of STI (343 ± 29 ms for male and 339 ± 35 for female; P = 0.183) and STI% (40 ± 5% for male and 41 ± 5% for female; P = 0.104) among sedentary controls, whereas male athletes had a mild but significantly shorter STI and STI% as opposed to female athletes (respectively, 322 ± 35 vs. 333 ± 34 ms, P = 0.010; 30 ± 4 vs. 32 ± 4%; P = 0.001).

Regression analysis showed that heart rate was the most important determinant of STI (R² = 0.38; P < 0.001), whereas age, BSA, blood pressure, LV volumes, and mass were not significantly associated with STI.

Analysis of covariance was then performed to derive the regression coefficients and 95% confidence intervals (CIs) that described the association between STI and heart rate in athletes and controls according to gender. The regression coefficients were: 501 (95% CI: 481 to 520) for the constant term (the heart rate value, the expected STI was significantly shorter in sedentary subjects (respectively, 324 ± 36 versus 333 ± 34 ms, P = 0.010; 30 ± 4 vs. 40 ± 4; P = 0.001).

Eventually, these differences among SFV values remained significant on STA the Wald test confirmed that the impact of these variables on STA was significant (P < 0.001).

We also derived the regression lines describing the association between STI values and heart rate separately in athletes (R² = 0.30; P < 0.001) and controls (R² = 0.44; P < 0.001). As shown in Figure 3, the regression lines were different in the two groups with a downward displacement in athletes, meaning that for each heart rate value, the expected STI was significantly shorter in athletes.

Systolic flow velocity

Athletes as a group had a higher SFV compared with controls (310 ± 75 vs. 204 ± 50 mL/s; P < 0.001) corresponding to a 52% increase.

Subgroups analysis within the athlete population, according to different sport disciplines, showed increasing SFV value from skill (256 ± 60 mL/s), to power (278 ± 76 mL/s), to mixed (312 ± 68 mL/s), and to endurance (334 ± 74 mL/s) athletes (Figure 4). In particular, endurance athletes had the fastest SFV, which was significantly higher than for those engaged in both skill and power (P < 0.001) but not in mixed disciplines; on the other hand, athletes engaged in skill disciplines did not differ significantly from untrained subjects.

Eventually, these differences among SFV values remained significant even when the stroke volume in the formula was indexed by
Table 2  Demographic and echocardiographic characteristics in the athlete population according to the type of sport participated

<table>
<thead>
<tr>
<th></th>
<th>Skill</th>
<th>Power</th>
<th>Mixed</th>
<th>Endurance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=41</td>
<td>n=63</td>
<td>n=167</td>
<td>n=158</td>
</tr>
<tr>
<td>Age</td>
<td>27 ± 6</td>
<td>26 ± 4</td>
<td>26 ± 6</td>
<td>26 ± 5</td>
</tr>
<tr>
<td>Sex (male, %)</td>
<td>70</td>
<td>75</td>
<td>78</td>
<td>82</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.81 ± 0.20(E,M)</td>
<td>1.90 ± 0.22(P)</td>
<td>2.02 ± 0.20(S,P)</td>
<td>1.94 ± 0.20(S)</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>60 ± 12(E)</td>
<td>57 ± 12(E)</td>
<td>56 ± 9(E)</td>
<td>52 ± 10(S,P,E)</td>
</tr>
<tr>
<td>LV end-diastolic diameter (mm)</td>
<td>50 ± 5(E)</td>
<td>52 ± 4(E)</td>
<td>54 ± 4(E)</td>
<td>57 ± 4(S,P,M)</td>
</tr>
<tr>
<td>Maximum wall thickness (mm)</td>
<td>9 ± 1(E)</td>
<td>10 ± 1(E)</td>
<td>10 ± 1(S,E)</td>
<td>11 ± 1(S,P,M)</td>
</tr>
<tr>
<td>LV end-diastolic volume (mL)</td>
<td>127 ± 30(E,M)</td>
<td>140 ± 31(M,E)</td>
<td>160 ± 31(S,P,E)</td>
<td>178 ± 32(S,P,M)</td>
</tr>
<tr>
<td>LV end-systolic volume (mL)</td>
<td>47 ± 15(E)</td>
<td>52 ± 14(E)</td>
<td>61 ± 16(E)</td>
<td>68 ± 17(S,P,M)</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>79 ± 18(M,E)</td>
<td>88 ± 20(M,E)</td>
<td>99 ± 19(S,P,E)</td>
<td>110 ± 20(S,P,M)</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>125 ± 28(M,E)</td>
<td>141 ± 31(M,E)</td>
<td>162 ± 30(S,P)</td>
<td>176 ± 34(S,P,M)</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>63 ± 5</td>
<td>64 ± 10</td>
<td>62 ± 5</td>
<td>62 ± 5</td>
</tr>
<tr>
<td>STI (ms)</td>
<td>313 ± 29</td>
<td>321 ± 30</td>
<td>320 ± 36</td>
<td>333 ± 37</td>
</tr>
<tr>
<td>STI (%)</td>
<td>31 ± 4</td>
<td>31 ± 4</td>
<td>31 ± 4</td>
<td>29 ± 4</td>
</tr>
<tr>
<td>Systolic flow velocity (mL/s)</td>
<td>256 ± 60(M,E)</td>
<td>278 ± 76(E)</td>
<td>312 ± 68(S)</td>
<td>334 ± 74(S,P)</td>
</tr>
</tbody>
</table>

Skill disciplines include golf, table tennis, equestrian, artistic gymnastic, shooting, fencing, karate, taekwondo, dancing, and sailing. Power disciplines include weightlifting, wrestling, and short-distance running (100–200 m). Mixed disciplines include soccer, basketball, tennis, volleyball, handball, water polo, swimming, and fencing. Endurance disciplines include rowing, long-distance running (800–3000 m), marathon, cycling, triathlon, and pentathlon.

BSA: body surface area; LV, left ventricle; STI, systolic time interval; S, P-value < 0.01 vs. skill athletes; P, P-value < 0.01 vs. power athletes; M, P-value < 0.01 vs. mixed athletes; E, P-value < 0.01 vs. endurance athletes.
Intra-observer variability was 14 ms. This was graphically expressed (Figure 3) by a downward displacement of the regression line describing the relationship between STI and heart rate in the two population groups, meaning that for the overall spectrum of the heart rates, the expected STI was shorter in athletes compared with untrained subjects. In detail, regression analysis showed that athletes had a significant reduction in terms of STI (by 50 ms, as average) compared with untrained subjects of the same gender and with the same heart rate. Moreover, although no gender-related differences were detected among untrained controls, the shortening of STI was more evident in male athletes. This finding may in part be explained by differences in adaptive changes in male compared with female athletes.16,17

This mild shortening of the mechanical systole (and the consequent lengthening of diastole) in athletes may eventually represent an additional means to improve LV filling and emptying, particularly during exercise, when highest heart rates are achieved. However, we believe that the advantage to mechanical performance of this mild shortening of the systolic time is relatively modest. This consideration is also supported by the lack of significant differences in STI in athletes in relation to the type of sport participated, despite the substantial differences in LV remodelling among the different athletic disciplines.3

In association with a mild shortening of systolic time duration, athletes also showed a significantly faster emptying velocity compared with controls, implying that a larger blood volume is ejected in a shorter time. Indeed, intra-group analysis showed that systolic emptying velocity displayed a greater increase in athletes with more substantial LV remodelling, such as those engaged in endurance disciplines (Figure 4). Moreover, these changes were more substantial in male compared with female subjects, in accord with larger morphologic remodelling described in male athletes.16,17

Therefore, our data depict the typical pattern of LV systolic mechanics in trained athletes as characterized by a significant decrease in STI and by a more substantial increase in SFV, particularly in athletes with a more marked LV remodelling.

Mechanisms leading to a shortening of the STI and to enhanced SFV in the athletes were not addressed by the present study, and remain to be further investigated. Animal studies suggest the occurrence of molecular alterations in trained hearts involving the excitation–contraction process, which result in improved pump efficiency.18–23 That is, exercise training is associated with an over-expression of the isoform α of the myosin-heavy-chain, which is responsible for enhanced heart contractility;18 besides, the expression of mRNA for the α myosin-heavy-chain is an early event in the adaptation to chronic exercise and occurs before significant cardiac growth.19 Conversely, pathological remodelling is characterized by the expression of myosin β-heavy-chains, associated with impaired systolic and diastolic function.20 Moreover, other studies suggest that improved LV function in trained animals is also related to an alteration of the Ca2+ regulatory systems involved in the excitation–contraction coupling and relaxation processes.21–23 However, no direct evidence for such changes occurring in trained humans has been demonstrated and the spectrum of pathophysiological mechanisms leading to improvement of LV mechanics in the human trained heart remains to be further investigated.

A methodological limitation regarding our investigation is worth mentioning. Even if the frame rate of three-dimensional acquisitions was the best possible by the use of the 7-beats protocol, it is still relatively low, and when the minimum systolic time is reached between two frames, it can be attributed to either the previous or the next frame with an error margin ranging from −15 to +15 ms, which limits the accuracy of systolic time definition. However, this problem is partially overcome by the high number of subjects analysed.
In conclusion, our study demonstrates that highly trained athletes show a significant shortening of the systolic time duration, associated with a substantial increase of the systolic velocity emptying, enabling the ejection of larger blood volume in a shorter time. This pattern characterizes the physiological LV remodelling of highly trained individuals and may potentially be useful in differential diagnosis of the ‘athlete’s heart’.

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Conflict of interest: none declared.

References