An Echocardiographic Study of Heart in a Group of Male Adult Elite Athletes

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Abstract

Background: Severe and prolonged physical training is associated with morphological and physiological cardiac changes, often termed as the “athlete’s heart”. Echocardiographic features peculiar to elite Iranian athletes have not been previously described. The aim was to examine the echocardiographic characteristics of highly trained Iranian athletes involved in three different sports.

Methods: We studied cardiac morphology and function as assessed by rest echocardiography in 50 elite adult male athletes referring to a university hospital in Tehran between February 2001 and March 2006. Resting ejection fraction, interventricular septal wall thickness (IVSWT), left ventricular posterior wall thickness (LVPWT), left ventricular internal end diastolic dimension (LVEdD), left ventricular internal systolic dimension (LVIsD), left ventricular (LV) mass, and relative wall thickness (RWT) were measured. The control group consisted of 50 age- and weight-matched normal healthy men.

Results: Of the athletes, 38 were engaged in predominantly dynamic (running and soccer) and 12 in predominantly static (weightlifting) sports. The overall mean LVEdD (51.06±5.49mm) and IVSWT (10.24±1.43mm) were higher in the athletes than those in the normal subjects. The mean of IVSWT in the 38 endurance-trained athletes was significantly more than that of the 12 strength-trained athletes (11.1 mm vs. 10.3 mm, P<0.05). LVEdD was also greater in the endurance-trained athletes, but the difference was not statistically significant (51.2 mm vs. 50.6 mm).

Conclusion: Our results of higher LVEdD and IVSWT in Iranian male athletes are in line with previous reports. To generalize the results, we require more studies with larger sample sizes (with female athletes included).

Introduction

Cardiac enlargement in athletes was initially described at the end of the 16th century by Henschen.1 Regular physical training in athletes leads to cardiac enlargement through a...
combination of left ventricular (LV) cavity enlargement (dilatation) and increased wall thickness (hypertrophy). Athlete’s heart is a condition characterized by changes in cardiac function and morphology associated with intense physical training. Physiological changes from training include an increased stroke volume and decreased heart rate, whereas morphological changes include increased LV cavity dimension, wall thickness, and LV mass. Standard Doppler echocardiography has been widely used to identify the athlete’s heart and to distinguish it from LV pathologies. Morganroth et al. were the first to postulate that two different morphological forms of athlete’s heart can be distinguished: strength-trained heart and endurance-trained heart. According to their theory, aerobic isotonic sports with a high dynamic component (e.g. running) stimulate LV hypertrophy and cavity dilatation (eccentric), while resistance or isometric training (e.g. weight lifting) stimulates hypertrophy with normal cavity dimensions (concentric). Endurance-trained athletes are presumed to demonstrate eccentric LV hypertrophy, characterized by an unchanged relationship between LV wall thickness and LV radius (i.e. ratio of wall thickness to radius), whereas strength-trained athletes are presumed to demonstrate concentric LV hypertrophy, which is characterized by an increased ratio of wall thickness to radius.

Although the morphology of athlete’s heart and the impact of various sports on cardiac structure have been recently investigated by several authors, few data are presently available about the possible impact of racial differences in the response of the heart to certain physical training, as part of the normal physiological adaptation process.

On these grounds, we were prompted to utilize echocardiography to evaluate the LV function at rest in a group of highly trained Iranian athletes.

**Methods**

Between February 2001 and March 2006, 50 elite adult male athletes (mainly competing at an international level and national title holders) in three different sports, namely running, weightlifting, and soccer, were included in the study. All the athletes, who had a minimum average of 10 hours per week exercise activity, underwent a routine physical examination and electrocardiography as part of their evaluation for selection into the national teams. At the time of the present investigation, the athletes had been engaged in systematic training for at least the previous 2 months in national training camps or had been examined up to one week after the end of national competitions to avoid reconditioning effects. Exclusion criteria were a history of coronary artery disease, arterial hypertension, and valvular disease. The study group consisted of 22 soccer players, 16 runners, and 12 weightlifters. We categorized the athletes as 38 predominantly endurance-trained (sum of runners and soccer players) and 12 predominantly strength-trained (weightlifters) athletes. The control group consisted of 50 pair-matched for age, and weight normal healthy men, none of whom exercised over 2 hours per week. Informed consent was obtained from the athletes and the control group.

The echocardiographic studies were performed with a Contron Sigma Iris (Contron Medical, Pans France Instrument), equipped with a 2.5 MHz transducer. Standard transthoracic, i.e. two-dimensional guided and M mode examinations were performed, with the participants positioned at 30º left lateral position. Measurements were taken from “frozen” M mode tracings obtained using two-dimensional guiding in the long axis parasternal view, with the ultrasound beam perpendicular to the ventricular walls. Internal LV diameter, interventricular septal wall thickness (IVSWT), and left ventricular posterior wall thickness (LVPWT) were measured at end diastole and end systole as recommended by the American Society of Echocardiography. Relative wall thickness was calculated by dividing the sum of the IVSWT and LVPWT by left ventricular internal end diastolic dimension (LVEdD), and ejection fraction was calculated by cross-sectional echocardiography. LV mass was measured by both the American Society of Echocardiography (ASE) criteria and the Cornell-Penn convention:

<table>
<thead>
<tr>
<th>LV mass (Penn formula)</th>
<th>1.04</th>
</tr>
</thead>
<tbody>
<tr>
<td>([LVEdD+LVPWT+IVSWT]-[LVEdD])-13.6 g</td>
<td></td>
</tr>
<tr>
<td>LV mass (ASE method)</td>
<td>0.8 (1.04)</td>
</tr>
<tr>
<td>([LVEdD+LVPWT+IVSWT]-[LVEdD])+0.6 g</td>
<td></td>
</tr>
</tbody>
</table>

Left atrial (LA) diameter was measured at the end of atrial diastole at the parasternal long axis view by M-mode. A single cardiologist blind to the participant’s training group took three measurements of each parameter, and the average was calculated.

The continuous data are expressed as mean±SD, and the discrete variables are shown as percentages. The statistical analyses were performed using the independent sample t-test, univariate analysis of variance (ANOVA) test with post hoc, and Chi-square test. A P value<0.05 was considered statistically significant.

**Results**

The characteristics of the athletes and control cases at rest are depicted in Table 1. The athletes and control groups were matched in terms of mean age, sex, and weight.

Table 1. Athletes and control groups characteristics*  
<table>
<thead>
<tr>
<th>Variable</th>
<th>Athletes (n=50)</th>
<th>Control (n=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>27±5.5(20-43)</td>
<td>26.0±3.0(21-37)</td>
<td>0.26</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.6±8.4(58-69)</td>
<td>71.0±9.0(55-88)</td>
<td>0.36</td>
</tr>
<tr>
<td>LOT (y)</td>
<td>9.8±5.5(2-24)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Data are presented as mean±SD (min-max)
LOT, Length of training

Table 2 lists the results of the echocardiographic data regarding the cardiac structure in the predominantly
endurance-trained athletes including runners and soccer players in comparison to the strength-trained athletes (weightlifters) and the control group. LVpWT, IVSWT, LVEDD, and atrial dimension were significantly larger in the athletes group.

Table 2. Echocardiographic measurements in endurance-trained athletes, strained athletes, and normal matched control subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Endurance-trained (n=38)</th>
<th>Strength-trained (n=12)</th>
<th>Control subjects (n=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA diameter(mm)</td>
<td>34.5±3.5</td>
<td>32.6±2.6</td>
<td>30.1±0.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>51.2±5.7</td>
<td>50.6±5.0</td>
<td>47.0±4.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVlsD (mm)</td>
<td>33.5±4.6</td>
<td>31.3±2.2</td>
<td>30.7±4.5</td>
<td>NS</td>
</tr>
<tr>
<td>LVPWT (mm)</td>
<td>11.1±1.5</td>
<td>10.3±1.2</td>
<td>9.25±1.22</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>RWT (mm)</td>
<td>0.40±0.08</td>
<td>0.39±0.06</td>
<td>0.34±0.03</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LV mass(g) [Penn formula]</td>
<td>255.4±55.2</td>
<td>230.6±45.7</td>
<td>148.1±6.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV mass(g) [ASE method]</td>
<td>204.3±44.2</td>
<td>184.5±36.5</td>
<td>143.1±6.2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>68.8±6.3</td>
<td>70.1±4.2</td>
<td>67.2±4.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

'Data are presented as mean±SD

LA, Left atrium; LVEDD, Left ventricular end diastolic dimension; LVlsD, Left ventricular internal systolic dimension; LVPWT, Left ventricular posterior wall thickness; IVSWT, Interventricular septal wall thickness; RWT, Relative wall thickness; LV, Left ventricle; ASE, American society of echocardiography

The maximal value of LVEDD in the athletes was 64.5 mm. Nine of the athletes showed LVEDD above 58 mm, of whom 7 (14%) athletes were in the endurance-trained group and the rest (2.4%) were in the strength-trained group. Ejection fractions were similar in the athletes and control group, but there was a significant increase in relative wall thickness (RWT) in the athletes compared to the controls.

There was a significant difference between the 2 groups of athletes and the control subjects with respect to IVSWT (P<0.05); however, the endurance-trained athletes and strength–trained athletes did not show a significant difference regarding LVEDD (P=0738).

There was a significant difference with respect to IVSWT between the control and athletes groups (9.25±1.25mm vs. 10.94±1.43mm, P<0.05) and also between the endurance–trained athletes and strength–trained athletes (11.1±1.5mm vs. 10.3±1.2mm, P<0.05). Septal or posterior wall thickness above 12 mm were seen in 4 (8%) of the athletes, in all of whom, except one, the septal wall to posterior wall ratio remained lower than 1.3.

In one case, the ratio was 1.41. For this reason, all of the individual’s first-degree relatives were investigated echocardiographically with respect to hypertrophic cardiomyopathy (HCM). No evidence of HCM was found; nevertheless, the athlete was advised to return for further investigation after a period of avoiding severe physical activities (reconditioning). There was no significant relationship between LVEDD and RWT and age of the athletes or length of training.

Discussion

Physiological hypertrophy is a common feature of the “athlete’s heart”. We found a 7.91% increase in LVEDD and 15% increase in IVSWT in our athletes as compared to the control subjects. The cardiac morphological changes seen in our study in terms of LVEDD and IVSWT are similar to values reported by Maron et al.,16 who found a 10% and 15-20% increase in LVEDD and RWT, respectively, when comparing highly-trained Olympic athletes to control subjects.

The septal-to-LVPWT ratios in prior studies13,16 have almost always been found to be below 1.3, and rarely have markedly increased ratios (1.3-1.5) in some athletes been reported. Fagard17 in a meta-analysis suggested that a wall thickness more than 16 mm was very unusual in a healthy athlete and that most of the cases would be less than 13 mm.

Early echocardiographic studies showed that intense isometric (anaerobic, strength/power) exercise training led to a more concentric LV hypertrophy, which was characterized by an increase in LV mass with an augmented the ratio of wall thickness to the LV diameter,18 whereas extensive isotonic (aerobic, endurance) exercise training beot a more dominant enlargement of LV diameter.19,20 Recent studies, however, have been unable to prove such a dichotomy of the cardiac structural adaptation patterns in athletes.21-23 Some investigators have demonstrated that even very intense strength training sports will not necessarily lead to cardiac wall thickening18,24-27 or the resultant myocardial structure in strength athletes will not differ from that of endurance athletes.26-28 In our study, the group comprised of runners and soccer players did not have exceptionally thicker LV walls, and nor did the group composed of weightlifters differ from the other group of athletes with respect to the LVEDD parameter. Similar absence of difference in myocardial wall thickness between strength-trained and endurance-trained athletes has also been documented by other echocardiographic studies.27,29 Possible explanation for the discrepancy between the results of different studies might be related to the variations in training regimes (season, specificity) and usually limited statistical power to detect subtle differences.30

In the present study, we found an increased LVEDD as well as a significant increase in wall thickness and RWT in the athletes compared to the control subjects. These findings are consistent with other previous studies,5,15,31 in which the authors demonstrated a combination of LV cavity dilatation and increase in IVSWT in elite athletes. In addition, studies in other countries with different populations in regard to ethnicity or race have shown a raised mean IVSWT and LVEDD in competitive athletes compared to control groups, which is in line with our study.5,15,32,33
Results of several studies have demonstrated that in dynamic sports, ventricular dilatation is predominantly observed rather than an increase in IVSWT. Other studies have demonstrated that in static sports, in contrast, an increase in IVSWT predominates the LV dilation. However, in the present study we found that LV dilatation and IVSWT in endurance-trained athletes were greater than those of strength-trained athletes. Therefore, we concluded that elite sports, associated with more increase in LVEdD, also resulted in a more increase in IVSWT. This finding is in line with several recent studies. Be that as it may, the results of various similar studies are controversial. The likely reason for conflicting results regarding the type of sport and pattern of hypertrophy may be related to differences in training methods, duration, and severity of champion sports in various studies. In one study, cardiac morphology changes in the “athlete’s heart” were attributed to determinants such as type of sport, gender, and possible inherited genetic factors. In our investigation, only 4 athletes had IVSWT above 12 mm, but in a recent study on African handball players, none of them showed IVSWT>12mm.

Conclusion

The present study demonstrated that highly trained Iranian athletes had increased LV mass, LVEdD, and IVSWT, which chimes in with the results of previous studies with different populations. Contrary to common belief, ejection fraction is not supra-normal in athletes. However, because our study population was limited in size and also restricted to the male gender, caution should be exercised in extrapolating our results to the whole population of Iranian athletes.

Acknowledgement

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