ORIGINAL ARTICLE

Echocardiographic evaluation of left ventricular morphology and function in young male football players and runners

Piraye Kervancioglu and E. Savas Hatipoglu

Department of Anatomy, Faculty of Medicine, Dicle University, Diyarbakir, Turkey

Abstract

Background: *Long-term physical exercise leads to left ventricular morphological adaptations, which vary with the kind of sport and the intensity and extent of the training. Although the echocardiographic changes related to exercise in athletes participating in various sports have been well described, changes associated with football players have been less well documented. The aim of this study was to investigate the morphological adaptations of the heart to athletic training and to compare by means of echocardiography the left ventricular dimensions, masses and systolic functions of runners and football players.*

Methods: *A total of 40 male football players, 18 male long and middle-distance runners and 25 sedentary males were enrolled to the study and M-mode and two-dimensional echocardiograms with Doppler analysis were performed in all subjects.*

Results: *The runners and football players were shown to have significantly greater left ventricular end-diastolic internal dimensions, left ventricular mass (corrected for body mass, height and body surface area), end-diastolic interventricular septum thicknesses and ejection fraction values than a control group. The present results show no significant difference between the left ventricular adaptations of runners and football players except when the left ventricular mass is indexed with body mass, the runners being thinner.*

Conclusions: *The present results show that the echocardiographic findings of left ventricular adaptations in runners and football players are quite similar and lead to physiological enlargement of the heart.* (Cardiol J 2007; 14: 37–43)

Key words: echocardiography, left ventricle, runner, football player, athlete

Introduction

The enlarged heart of the athlete was initially described in the 19th century by Henschen [1]. Using the percussion of the chest technique in cross-country skiers he observed that all parts of the heart were enlarged. Since that time the physiological effects of chronic training on the heart have been investigated by invasive and non-invasive methods. With the development of echocardiography wall thicknesses, internal dimensions and functions could be studied in detail and estimations of left ventricular mass became possible [2, 3].

Long-term athletic training produces morphological changes in the cardiac dimensions which are frequently referred to as "athlete's heart". It has been suggested that these morphological changes

may differ among athletes participating and training in different sports. For example, athletes training for endurance (isotonic) sports such as distance running have been described as having a disproportionate increase in cavity dimensions in relation to wall thickness. In contrast, athletes participating in strength training, including weight-lifting and throwing events, have been described as having an increase in wall thickness compared with that of non-athletic control subjects (possibly due to pressure overload), which is disproportionate to any alterations in cavity dimensions. It has also been inferred from these observations that left ventricular wall thicknesses are often increased in absolute terms in power athletes. The activities involved in most ball sports are combinations of several types of training [2, 4, 5]. Few studies have focused on the cardiac adaptation of athletes engaged in ball sports.

In the present echocardiographic study we investigated the left ventricular cavity dimensions, wall thicknesses, masses and systolic functions of runners and football players. These were compared with each other and with non-athletic controls in order to investigate the influence of discipline-specific factors on left ventricle adaptation and to produce echocardiographic reference values for clinicians.

Methods

Subjects

The study group consisted of 40 male football players aged 19–26 years (mean age 22.4 ± 1.9 years) and 18 male middle-distance runners also aged 19–26 years (mean age 21.5 ± 2.2 years). The control subjects were 25 age and sex-matched normal sedentary volunteers without historical or echocardiographic evidence of heart disease aged 18–27 years (mean age 21.5 ± 2.5 years).

The football players were from three different teams of clubs in the Turkish Third Football League and all the players were professionally licensed. All played in football matches and trained for at least 2 h per day, four days per week.

The middle-distance runners performed a training programme of at least 1.5 h of daily activity for 5 days per week, the activity consisting of running 15–20 km. All were competing in regional competitions and 13 were competing at national level. All the runners had been training regularly for years.

Standing height was measured without shoes. Body weight was measured with the same weighing machine. Heart rate (HR) was assessed with an echocardiography machine. A representative value of arterial blood pressure, evaluated by means of a cuff sphygmomanometer, was taken at the time of the echocardiographic study.

Echocardiographic studies

M-mode and two-dimensional echocardiograms with Doppler analysis were performed, in all subjects using a commercially available machine (Hewlett-Packard Sonos 1000, with 2.5 and 3.5 MHz transducers). During the echocardiography basal examination all subjects were in semisupine left lateral decubitus. To avoid including trabeculations in the wall thickness measurements, an integrated M-mode and two-dimensional study was performed to determine interventricular septum thickness (IVS), left ventricular posterior wall thickness (LVPW) and left ventricular internal (cavity) dimensions (LVID) both in systole and diastole. Two-dimensionally targeted M-mode images were obtained in the parasternal long-axis and short-axis views and in apical four- and two-chamber views using the standard transducer position. All the echocardiographic measurements were made by the same observer and obtained directly from the screen monitor with the aid of callipers and an instrument trackball.

Left ventricular mass (LVM) was calculated from the end-diastolic left ventricular internal dimension (LVIDD), end-diastolic left ventricular posterior wall thickness (LVPWD) and end-diastolic interventricular septum (IVSD) by Devereux and Reichek formula [6]. Left ventricular systolic function was determined by measurements of fractional shortening (FS) and ejection fraction (EF) percentages. These values were calculated according to the Teicholz et al. formula [7].

Statistical analysis

Values were expressed as mean and standard deviation. The results were analysed using the Mann-Whitney U-test. Differences were considered to be statistically significant if the P-value was less than 0.05.

Results

The football players (22.4 ± 1.9 years), runners (21.5 ± 2.2 years) and sedentary subjects (21.5 ± ± 2.5 years) were similar in age, and height (173.54 ± ± 5.24 cm, 171.64 ± 4.27 cm, 171.88 ± 4.76 cm). There were no significant differences between the body masses of football players and the sedentary group (71.31 ± 6.46 kg and 71.46 ± 5.36 kg, p = 0.91). Because the runners were thinner than the others

Table 1. Demographic and echocardiographic parameters of football players, runners and the control group. Values are expressed as mean ± standard deviation and (min–max)

	Football players (min–max)	Runners (min–max)	Control group (min–max)
Age (years)	22.40 ± 1.91 (19–26)	21.50 ± 2.20 (19–26)	21.56 ± 2.52 (18–27)
Weight [kg]	71.31 ± 6.46 (58.5–85.0)	62.25 ± 7.68 (52.0–81.5)	71.46 ± 5.36 (62.0–88.0)
Height [cm]	173.54 ± 5.24 (163–184.5)	171.64 ± 4.27 (165.5–178)	171.88 ± 4.76 (164–183)
Heart rate [/min]	60.48 ± 6.71 (42–72)	59 ± 7.78 (48–74)	73.2 ± 3.44 (66–78)
Thicknesses of the end-diastolic interventricular septum [cm]	0.98 ± 0.13 (0.71–1.36)	1.01 ± 0.20 (0.62–1.48)	0.85 ± 0.09 (0.74–1.10)
End-diastolic left ventricle posterior wall thickness [cm]	0.85 ± 0.09 (0.73–1.13)	0.84 ± 0.15 (0.62–1.2)	0.81 ± 0.08 (0.68–1.06)
End-diastolic left ventricular internal diameter [cm]	5.37 ± 0.38 (4.69–6.39)	5.44 ± 0.38 (4.7–6.1)	4.63 ± 0.32 (3.87–5.2)
Left ventricular mass [g]	228.81 ± 37.36 (164.7–307.9)	237.4 ± 52.14 (179.8–343.6)	155.79 ± 22.17 (111.5–211.7)
Left ventricular mass corrected for body mass	3.23 ± 0.57 (2.04–4.40)	3.86 ± 0.97 (2.97–6.14)	2.20 ± 0.41 (1.62–3.36)
Left ventricular mass corrected for body surface area	126.73 ± 20.76 (86.3–167.2)	142.04 ± 32.55 (109.2–217.2)	86.28 ± 13.99 (63.4–125.1)
Left ventricular mass corrected for height	1.32 ± 0.22 (0.93–1.76)	1.38 ± 0.30 (1.06–1.94)	0.91 ± 0.13 (0.68–1.28)
Ejection fraction (%)	69.42 ± 4.50 (60.10–78.80)	70.89 ± 3.76 (64.60–77.90)	66.28 ± 4.54 (57.00–76.00)
Fractional shortening (%)	39.74 ± 4.12 (32.40–49.60)	40.69 ± 3.09 (35.70–46.30)	36.83 ± 3.40 (30.10–40.50)

there were significant difference between the body masses of runners (62.25 ± 7.68 kg) and those of the football players and the sedentary group (p < 0.001, p < 0.001). The general characteristics of the subjects studied can be seen in Table 1.

No subject had pathological values of systolic and diastolic arterial pressure as defined by the OMS criteria. Heart rates were between 42 and 72/min (60.48 ± 6.71 min) in football players, 48 and 74/min (59.00 ± 7.78 min) in runners and 66 and 78/min (73.20 ± 3.44 min) in the control group. There was no significant difference between the HR of football players and that of runners (p = 0.39) but there were significant differences between football players and controls (p < 0.001) and runners and controls (p < 0.001).

The echocardiographic data from left ventricular cavity and wall measurements and measurements corrected for body surface area (BSA), height and body mass of the left ventricular cavity and wall dimensions are shown on Table 1.

The runners and football players were shown to have significantly higher values than the control
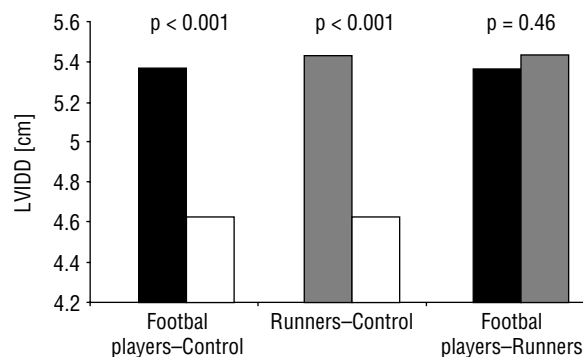
Figure 1. Distribution of end-diastolic left ventricular internal diameter (LVIDD) values in runners (grey bars), football players (black bars) and controls (white bars).

group for LVIDD, IVSD, LVM and LVM corrected for body mass (LVM/BM), height (LVM/H) and body surface area (LVM/BSA) and EF (p < 0.001) (Fig. 1–3). There was no significant difference between the LVPWD of the runners, football players and control group (runners–controls: p = 0.48,
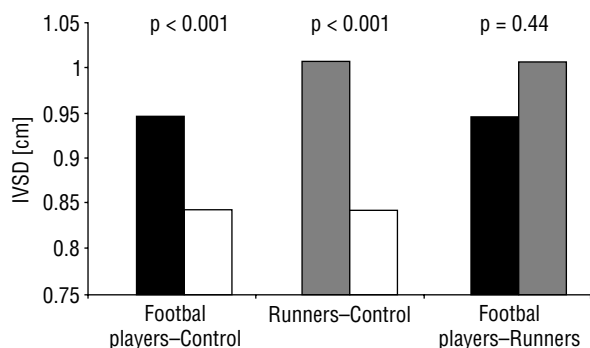
Figure 2. Distribution of end-diastolic interventricular septum thickness (IVSD) values in runners (grey bars), football players (black bars) and controls (white bars).
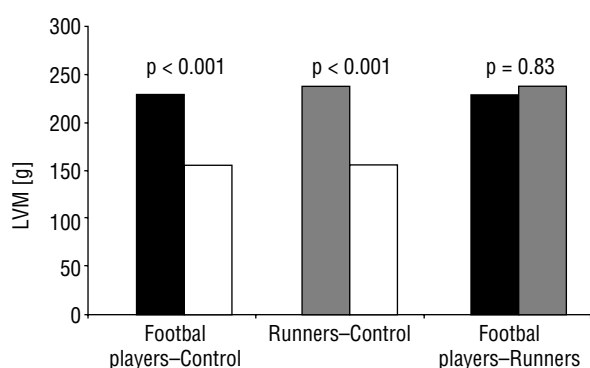

Figure 3. Distribution of left ventricular mass (LVM) values in runners (grey bars), football players (black bars) and controls (white bars).
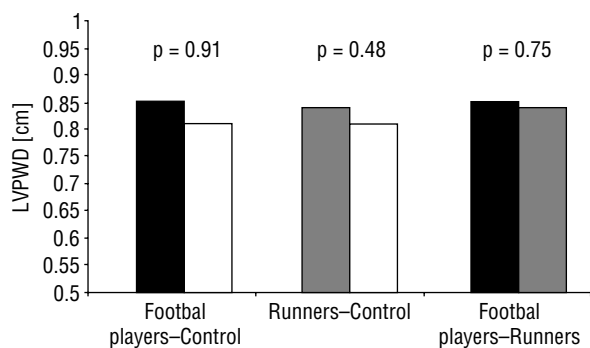

Figure 4. Distribution of end-diastolic left ventricular posterior wall thickness (LVPWD) values in runners (grey bars), football players (black bars) and controls (white bars).

football players–controls: $p = 0.91$, runners–football players: $p = 0.75$) (Fig. 4). The differences between the FS parameters of the runners and the control group were significant statistically. However, the differences between the parameters of runners and football players were not significant, except for the difference in LVM/BM ($p < 0.02$).

Discussion

Since the first echocardiographic study in 1975 by Morganroth et al. [8], who were the first to describe the greater septal and posterior wall thicknesses that contribute to an increased left ventricular mass in male wrestlers and shot putters, an extensive literature has developed on the cardiac morphological response to training.

Exercise training causes a number of well-known physiological changes in the heart: an increase in LVIDD and left ventricular wall thicknesses (IVS and LVPW) lead to increased LVM, stroke volume is increased and heart rate is decreased during resting conditions [2, 9]. The values obtained by echocardiography for cardiac dimensions and wall thickness for athletes do not provide a distinct data set or a bimodal distribution and are usually within the ranges accepted as normal. Although such values are usually significantly different from the norm in statistical terms, the reports on echocardiographic findings in athletes are somewhat contradictory, possibly because of varying methodology [2, 10].

A consistently demonstrated feature in endurance trained athletes compared with normal sedentary age-matched and sex-matched controls is an increase in LVIDD. This increase in LVIDD accounts in a large part for the elevated LVM in athletes. The elevated LVIDD reflects a true increase in cavity size and is present even when corrections are made for body surface area and weight. The degree of increase is usually within the normal range (5.7 cm). A few studies, however, show values greater than this. The greatest values appear to occur in cyclists, with a measurement of 7 cm being reported [2]. Measurements above 6 cm, however, are rare and primarily have to be clarified [11]. Pelliccia et al. [12] showed that only 38 (4.01%) of 947 athletes and Henriksen et al. [13] showed that 6 (4.6%) of 127 athletes had an LVIDD greater than 6 cm. In this study an LVIDD exceeding 6 cm was found in 2 (5%) of 40 football players and 1 (5.5%) of 18 runners, a result similar to those of other studies. Urhausen et al. [11] found that 69% of male rowers had an LVIDD above 5.5 cm. In this study the LVIDD measured was above the upper clinical limit of 5.5 cm in 20% of football players and in 38.8% of runners.

The accumulated data from the many echocardiographic studies of athletes in training shows that as an average LVIDD is increased by approximately 10% compared with matched sedentary control subjects [5]. In this study the runners and football

players were shown to have significantly higher LVIDD values than the control group, but there was no significant difference between runners and football players.

In different studies the left ventricular wall thickness (septum and posterior wall) values obtained are only mildly increased from normal or may lie within accepted normal ranges (0.8–1.2 cm). However, in athletes younger than 35 years hypertrophic cardiomyopathy is the most common cardiac condition associated with sudden death. Average values for accumulated data show an increase in septal thickness to 1.04 cm (14% greater than normal matched controls) and in the posterior wall to 1.06 cm (19% greater than normal). Left ventricular wall thicknesses up to 1.9 cm have been described in professional cyclists [3, 11]. Sharma et al. [14] studied 720 adolescent athletes and reported that athletes showed a range in left ventricle wall thicknesses of up to 1.4 cm; in just 4% of the overall group it was > 1.1 cm and in 0.5% it was 1.3–1.4 cm. Pelliccia et al. [12] found an LVPWD above 1.2 cm (the generally accepted upper limit of the norm) in 1.7% out of 947 athletes and only one athlete had an LVPWD 1.5 cm or more. In another study the LVPWD measured above 1.3 cm in 13% of 127 athletes [15]. Pelliccia et al. [4] examined 100 athletes, and reported that none of them had an LVPWD of over 1.1 cm. In this study, out of 18 runners examined, the largest LVPWD was measured at 1.2 cm (5.5%) and there was no significant difference between the LVPWD of the runners, football players and control group.

The development of septal hypertrophy is a characteristic feature of hypertrophic cardiomyopathy. Some athletes, however, develop septal hypertrophy as part of their physiological development. The average increase in IVS for pooled data is 1.04 cm, which is only 14% greater than normal. Values for IVS, however, have been reported up to 1.6 cm [3]. Pelliccia et al. [4] reported that of 100 athletes 12 (12%) had an IVS thickness of 1.1 cm or 1.2 cm. Urhausen et al. [11] reported an average IVS of 1.1 cm in 64 male rowers. In another study the average IVS value was reported as 1.08 cm in 56 weightlifters [16]. In this study one of the runners had a 1.48 cm IVS thickness. Runners and football players were shown to have significantly higher IVS values than the control group but there was no significant difference between runners and football players.

Echocardiographic estimates of left ventricular mass by various formulae use end-diastolic dimension and myocardial thickness. Although the increases in cavity size and wall thickness are small

and often within the normal range, because of the nature of the calculation of mass large increases may be observed. Pooled data suggest an average increase of 45% compared with control subjects [3]. For left ventricular hypertrophy, M-mode echocardiographic studies determined an upper clinical limit for LVM and an indexed mass from 215 g to 259 g and 125 g/m^2 to 131 g/m^2 respectively, which causes an increase in cardiovascular mortality if exceeded. In autopsy studies upper limits from 184 g to 204 g were determined. In recent MRI studies 238 g and 113 g/m^2 were reported by Lorenz et al. [17] and 200 g and 107 g/m^2 were reported by Scharhag et al. [18]. The average LVM value of male rowers reported by Urhausen et al. [11] was 256 g, for runners and wrestlers as reported by Abinader et al. [19] it was 289 g and 280 g respectively, for runners as reported by Karjalainen et al. [20] 263 g, for athletes as reported by Douglas et al. [21] 249 g, for athletes as reported by Pelliccia et al. [4] 193 g and for runners and triathletes as reported by Hoogsteen et al. [22] 253 g and 322 g respectively. In this study the average LVM was 228 g in football players and 237 g in runners. The calculated LVM index reported by Urhausen et al. [5] was 120 g/m^2, that reported by Karjalainen et al. [20] was 141 g/m^2, that by Abinader et al. [19] 166 g/m^2, that by Douglas et al. [21] 131 g/m^2 and that reported by Pelliccia et al. [4] 96 g/m^2. Enlarged left ventricular muscle mass exceeding 130 g/m^2 was described as a significant risk factor for cardiovascular complications in hypertensive patients [23]. In this study the average LVM index was found to be 126 g/m^2 in football players and 142 g/m^2 in runners. Runners and football players were shown to have significantly higher LVM, LVM/BM, LVM/H and LVM/BSA values than the control group. Although the LVMs of runners and football players were similar, when LVM was corrected with BM the difference became significant, as the runners were thinner.

The study by Urhausen et al. [5] was the first to compare ball game (football) players with runners. Their results revealed a significantly larger LVIDD and heart volume in the football players in comparison to the runners. Muir et al. [24] reported that elite footballers have significantly increased cardiac dimensions compared with healthy controls. In our study football players had larger LVIDD than controls but had similar LVIDD to runners. Reindell et al. [25] reported that during the short breaks between the various half-time exercises, especially those typical for ball games, the oxygen pulse and stroke volume are apparently increased, which would be an especially effective stimulus for the

enlargement of the heart in accordance with the concept of regulative dilatation of the athlete's heart. Abernethy et al. [26] reported that the (American) football players in their series showed cardiac changes similar to those of other athletes who placed an emphasis on strength training, with a relatively larger increase in LV wall thickness and a slight increase in LVID. However, they noted that this would not be surprising, as most players attached importance to weight lifting during their training, an activity that causes large transient increases in blood pressure. In our study football players had similar echocardiographic findings to runners classified as endurance athletes. The type of training programme undergone by athletes is important, as is the type of sporting discipline.

In some studies left ventricular systolic functions (EF, FS) remain unchanged in athletes' hearts [10, 27, 28]. In this study the runners and football players were shown to have significantly higher EF values than the control group and the FS parameters of runners were higher than those of the controls. The evaluation of contractility in humans is complicated, however, by the fact that contractile indices are influenced by afterload, preload and heart rate [2]. The increase in the EF of athletes and in the FS of runners was a consequence of lower heart rates.

Conclusions

In conclusion, both the runners and football players in our series were shown to have significantly higher LVIDD, LVM (corrected for body mass, height and body surface area), IVSD and EF values than controls. The present results show no significant difference in the left ventricular adaptations of runners and football players except when left ventricular mass is indexed with body mass, the runners being thinner. We consider that our data will contribute to the body of literature and will aid clinicians in the evaluation of athletes' hearts.

Acknowledgements

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