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# Diastolic filling in hypertrophied hearts of elite runners: an Echo-Doppler study

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*Abstract.* – **The aim of this study was to establish if the physiologic adaptations following a prolonged physical training could influence the diastolic function in a professional Olympic male runner group.**

**From February to December 1999 we studied 25 athletes (Group I) during the period of maximal training compared with 18 healthy sedentary subjects of matched age and sex (Group II). We used mono and bidimensional Echocardiography to assess left ventricular structure and systolic function. The diastolic function was evaluated by Doppler method assessing transmitral and venous pulmonary flow.**

**From the comparison between the two groups, we found great differences in the interventricular septum and the posterior wall thickness; the analysis of the systolic function demonstrated a significant increase in ejection fraction, stroke volume, left ventricular mass, and end-diastolic volume in the athletes' population. Fluximetric study of transmitral and pulmonary venous flow showed that ventricular diastolic function is not influenced by hypertrophy.**

**Our data indicate that diastolic function remains normal or improves in some cases after physical training; left ventricular hypertrophy and concentric remodeling do not involve diastolic changes like hypertrophic and hypertensive heart diseases.**

*Key Words:*

Cardiac hypertrophy, Diastolic function, Echocardiography, Concentric remodeling.

## Introduction

The athlete's heart is a physiological adaptation due to intensity and duration of training. The long term agonistic activity can produce a cardiac enlargement and hypertrophy with modification of wall thickness, left ventricular cavity dimension and cardiac mass. Functional responses to long term training include also an increase of the cardiac output and the ejection fraction, and a decrease of the heart rate, the blood pressure and the peripheral oxygen extraction[1]. Such anatomical and structural changes may produce other modifications of the diastolic phase[2].

The best method for non-invasive evaluation is the echocardiographic study: this technique allows physicians to obtain many information about structural and functional adaptation in the healthy hearts[3,4].

The aim of our work was to establish if the physiological hypertrophy of the left ventricle found in runners could modify the diastolic function evaluated by Doppler method.

## Materials and Methods

25 male professional runners, aged 26 to 32 years (mean age 26 ± 3 years) were recruited during a period of maximal training (group I). Control group (group II) consisted of 18 healthy subjects of the same age (mean age 28 ± 4 years). All athletes were in active high level agonistic training, they competed about the distance of 1500 meters; runners trained an average of 5 hours/day including run, cycle and sprint; subjects from control group conducted common daily living activities, including sporadic physical exercise. No subject had history of hypertension or cardiovascular disease. All subjects had similar body surface: Group I: 1.88 ± 0.1 $m^2$, Group II: 1.89 ± 0,1 $m^2$.

Blood pressure and heart rate were recorded for all subjects. Both groups underwent Doppler Echocardiography. An Esaote

Biomedica SPR 8000 ultrasound system with 2.5 MHz probe was used to perform resting supine end-expiratory M-mode and B-mode measurements, together with ECG recordings. All examinations were recorded on a video-tape and subsequently analyzed by two different echocardiographists. All results are the mean of three different measurements.

Left ventricular (LV) diameters, LV volumes, posterior wall and septum thickness were assessed in mono and two dimensional, long and short axis views. All measurements were performed "leading edge to leading edge", following the recommendations of the American Society of Echocardiography. Ejection fraction, myocardial mass (following Devereux formula) and cardiac output were assessed[5,6].

Septal and posterior thickness were considered abnormal if > 11 mm, LV End-telediastolic diameters were considered abnormal if > 56 mm.

Relative wall thickness was calculated as the addition between diastolic interventricular sept and diastolic posterior wall divided by diastolic cavity diameter, and was considered to be elevated if > 0.42 (concentric remodeling) and reduced if < 0.30 (eccentric remodeling)[7].

Mitral flow velocity was assessed by pulsed-wave Doppler Echocardiography in the apical four-chamber view with the sample volume positioned adjacent to the tip of the mitral leaflet in diastole. The following measurements were made with the sample volume positioned between mitral afflux and aortic efflux: maximal early diastolic velocity (E wave), maximal late diastolic velocity (A wave), and their ratio (E/A); the deceleration time of E wave (DT) and the time of isovolumetric relaxation were measured from flow to flow (IVRT).

Venous pulmonary flow was assessed by pulsed-wave Doppler Echocardiography in the right superior pulmonary vein in the apical four-chamber view in its three components (S: systolic wave, D: diastolic wave, Ar: retrograde wave following atrial contraction)[8-10].

The data, expressed by means ± SD, were analyzed for statically significant differences by two-tailed t-test for unpaired data using the SPSS/PC + 4 statistical package (SPSS Inc., Chicago, IL).

## Results

Heart rate was significantly lower in group I (I: 50 ± 6 vs II: 66 ± 8 beats; p < 0,001). Systolic and diastolic blood pressure did not differ between the two groups (systolic pressure: group I: 118 ± 10 mmHg, group II: 120 ± 8 mmHg; diastolic pressure group I: 78 ± 5, group II: 80 ± 7 mmHg).

Table I shows mono and bidimensional echocardiographic parameters studied: our analysis did not reveal a significant difference of left ventricular diameters.

LV Interventricular septum was significantly thicker in group I during both the diastolic and the systolic phase, LV Posterior wall thickness behaved in the same way .

LV End-diastolic volume was significantly higher in group I, instead LV End-systolic

***Table I.*** Analysis of morphological aspects in athletes and control group.

| *Morphological aspects* | *Group I (n = 25)* | *Group II (n = 18)* | *P* |
|---|---|---|---|
| End-systolic diameter (mm) | 34.2 ± 4.1 | 33.7 ± 4.4 | NS |
| End-diastolic diameter (mm) | 53.3 ± 3.7 | 52.6 ± 4.1 | NS |
| Interventricular septum | | | |
| Diastolic thickness (mm) | 12.6 ± 1.0 | 8.7 ± 0.7 | < 0.001 |
| Systolic thickness (mm) | 16.4 ± 1.3 | 13.3 ± 1.5 | < 0.001 |
| Posterior wall | | | |
| Diastolic thickness (mm) | 11.2 ± 1.3 | 7.7 ± 0.7 | < 0.001 |
| Systolic thickness (mm) | 16.5 ± 1.3 | 13.7 ± 1.2 | < 0.001 |
| End-systolic index volume (ml/m$^2$) | 23.0 ± 5.4 | 22.2 ± 3.1 | NS |
| End-diastolic index volume (ml/m$^2$) | 67.0 ± 8.8 | 55.3 ± 5.4 | < 0.001 |
| Myocardial index mass (g/m$^2$) | 176.9 ± 22.2 | 110.0 ± 25.4 | < 0.001 |
| Relative wall thickness | 0.45 ± 0.01 | 0.30 ± 0.01 | < 0.001 |

volume did not demonstrate any significant difference. Discordance between End-diastolic diameters and volumes in athletes was probably due to more LV sphericity at apical segments.

Regarding the parameters of systolic function the following differences were observed: (1) Fractional shortening was not different between groups; (2) Ejection fraction and systolic output was significantly higher in group I, (3) Myocardial mass was greater in group I, and (4) Relative wall thickness revealed a concentric hypertrophy in group I whereas control group was in the normality range.

Table II shows the differences regarding transmitral and pulmonary veins flow: the parameters of diastolic function revealed a mild higher velocity of the E wave in runners group; A wave and E/A ratio were not significantly different between two groups. Isovolumetric relaxation time (IVRT) was similar; instead Deceleration time (DT) was significantly lower in the group I.

Doppler evaluation of the venous pulmonary flow evidenced an higher S wave in group I group; D wave was similar between the two groups; whereas Retrograde atrial contraction wave (Ar) was significantly higher in group I.

## Discussion

Echocardiography is the most reliable non-invasive methodology in order to study the functional and structural characteristics of the healthy heart. It is particularly useful in the athletes' heart to assess if the hypertrophy is due to physiological adaptation to long term training or to pathology.

Few studies demonstrated an enlargement of the left ventricular cavity and of all others cardiac chambers. However, this does not involve a functional impairment[2,3,11].

These modifications are not only due to the type, the duration and the strength of the physical activity but to the intrinsic characteristics of the subject: indeed, not all the athletes who practice the same kind of sport have the same morphological aspects[12].

These cardiovascular changes are probably produced by a complex interaction of central and peripheral mechanisms operating at the structural, neurohumoral and metabolic level.

In our study, we demonstrated an increase of wall thickness, End-diastolic volume and left ventricular mass in runners in comparison to control subjects. The thickening of the septum and posterior wall has already been found in previous studies; it is considered to be normal below 14 mm[1,11,13].

In contrast with others studies[11,13], we have found a left ventricular concentric hypertrophy: thus may be due to the training model, to the type of effort or finally to the agonist activity that require frequent isometric strength. Similar results were previously observed by Urhausen and Whyte[14,15].

The indexes of systolic function were normal in runners, confirming that all others morphological modifications should be considered as due to physical activity more than pathology[16].

Doppler study showed that ventricular diastolic function is not influenced by hypertrophy, and in some cases it might also be improved in athletes: indeed, the Doppler analysis of the transmitral flow showed a bigger velocity of the E wave in group I. Similarly, when we assessed pulmonary venous flow, we found faster systolic S wave and retrograde Ar wave. This suggests that diastolic filling is earlier and faster in runners, possibly due to a lower intracavitary pressure during protodiastolic phase and to a greater atrial contraction comparing to normal subjects (Table III).

Furthermore, Doppler analysis seems useful in differential diagnosis: indeed, it is well known that many patients with hypertension

***Table II.*** Behavior of systolic function parameters in the two groups.

| *Systolic function index* | *Group I (n = 25)* | *Group II (n = 18)* | *P* |
|---|---|---|---|
| Ejection fraction (%) | 68.0 ± 2.4 | 60.7 ± 1.7 | < 0.001 |
| Stroke volume (ml/m$^2$) | 71.8 ± 6.2 | 43.5 ± 2.6 | < 0.001 |
| Shortening fraction %) | 39.1 ± 3.9 | 38.7 ± 3.0 | NS |

***Table III.*** Doppler analysis results of diastolic function in the two groups.

| *Diastolic function index* | *Group I (n = 25)* | *Group II (n = 18)* | *P* |
|---|---|---|---|
| E (cm/s) | 72.7 ± 7.1 | 58.4 ± 12.8 | < 0.001 |
| A (cm/s) | 35.2 ± 5.8 | 34.8 ± 5.3 | NS |
| E/A | 2.0 ± 0.3 | 1.8 ± 0.4 | NS |
| Isovolumetric relaxation time (msec) | 97.7 ± 15.5 | 107.1 ± 11.7 | NS |
| Deceleration time (msec) | 176 ± 21 | 205 ± 24 | < 0.005 |
| Systolic wave (cm/s) | 33.5 ± 6.3 | 31.7 ± 5.0 | < 0.01 |
| Diastolic wave (cm/s) | 46.8 ± 4.8 | 46.0 ± 7.2 | NS |
| Reverse atrial wave (cm/s) | 21.8 ± 2.6 | 18.0 ± 1.3 | < 0.005 |

and left ventricular hypertrophy have abnormalities of transmitral and pulmonary venous flows.

Therefore concentric hypertrophy of the athletes heart has a different behavior from hypertensive patients where this type of remodeling influences negatively the left ventricular filling profile. For this reason a fluximetric evaluation should be performed in all patients with a diagnosis of left ventricular hypertrophy[7,17].

Echocardiography is therefore useful in the diagnosis of hypertrophic cardiomyopathy, which is the main cause of death in young athletes[18]. This pathology is characterized by inappropriate thickening of the cardiac walls and more often to the hypertrophy of the anterior septum[19]. This can lead to obstruction of the ventricular efflux at the beginning during physical stress and only after at rest. In any case, wall thickness cannot be considered the only parameter for the diagnosis of hypertrophic car-

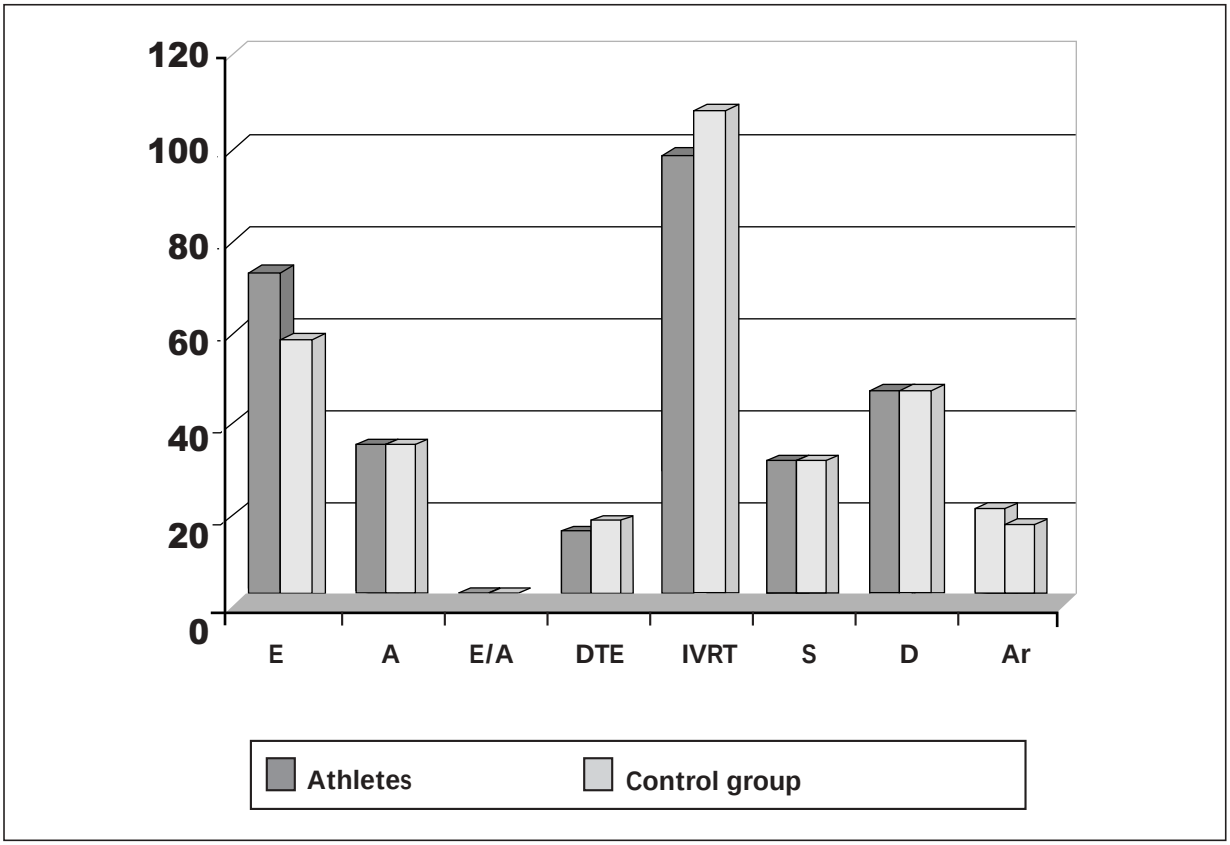


***Figure 1.*** Graphic comparison of diastolic functions index in athletes and control group.

diomyopathy. It is often observed an impairment of the diastolic phase due to the stiffness of the cardiac walls and to the reduced compliance of the cardiac chamber[20,21]. An accurate Eco-Doppler study, after a clinical examination and electrocardiogram, can be considered the best resource to perform an early diagnosis.

Morphological changes of the athlete's heart are the result of physiological and functional adaptation to the physical training performed. Left ventricular concentric remodelling does not impair diastolic profile, which seems to be improved.

Echocardiography appears to be the best technique in order to assess structural and functional alterations in young people: fluximetric Doppler study is particularly useful to emphasize the abnormalities of diastolic phase which accompany some diseases like hypertensive and hypertrophic cardiopathies.

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