Effects of different training protocols on left ventricular myocardial function in competitive athletes: a Doppler tissue imaging study

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Athlete's heart; Doppler tissue imaging; Endurance; Left ventricular hypertrophy; Strength; Training.

Background. **The aim of the study was to analyze the differences in myocardial function in case of left ventricular (LV) hypertrophy induced by either endurance or strength training in top-level athletes.**

Methods. **Standard Doppler echo and pulsed Doppler tissue imaging (DTI) of the interventricular septum and of the LV inferior wall were performed in 26 competitive endurance athletes (long-distance swimmers) (group A) and in 20 strength-trained athletes (short-distance swimmers) (group B). By means of DTI, the following parameters of myocardial function were assessed: the systolic peak velocities (S_m), the pre-contraction time, the contraction time, the early (E_m) and late (A_m) diastolic velocities, the E_m/A_m ratio and the relaxation time.**

Results. **The two groups were comparable for age and sex, but at rest group B showed a higher heart rate, systolic blood pressure and body surface area. The LV mass index and fractional shortening did not significantly differ between the two groups. However, group B showed an increased sum of the wall thicknesses (septum + posterior wall) ($p < 0.001$) and of the relative wall thickness, while the LV stroke volume and LV end-diastolic diameter ($p < 0.001$) were greater in group A. All transmitral Doppler indexes were higher in group A, with an increased E/A ratio. DTI analysis showed, in group A, a higher E_m and E_m/A_m ratio as well as a longer relaxation time both at the septal and at the inferior wall levels, with comparable S_m, pre-contraction and contraction times. Distinct multiple linear regression models revealed an independent positive association between the inferior peak E_m velocity and the LV end-diastolic diameter ($p < 0.001$) in group A, and an independent direct correlation of the inferior peak S_m velocity with the sum of the wall thicknesses ($p < 0.001$) and with the end-systolic stress in group B.**

Conclusions. **The early diastolic myocardial function is positively influenced by the preload increase in group A, while an increased afterload and LV wall thickness in group B mainly seem to induce an enhancement of the regional myocardial systolic function.**

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Introduction

The athlete's heart is a cardiac adaptation to long-term, intensive training which includes changes of both ventricles such as an increased cavity diameter, wall thickness and mass index produced with a degree consistent with the intensity and duration of sports activity and exercise programs[1-10]. Standard Doppler echocardiography has been widely used to evaluate the athlete's heart and to distinguish it from left ventricular (LV) pathologies[6-10]. A recent large meta-analysis on this topic confirmed how the combination of endurance- and strength-training may determine both extreme volume and pressure loads, thus explaining the increase in LV internal dimensions and wall thickness observed in athletes[8]. However, standard Doppler echocardiography allows only the assessment of the global LV function, while it might be useful to obtain information regarding the changes in myocardial regional function occurring under these circumstances.

Pulsed Doppler tissue imaging (DTI) extends Doppler applications beyond the analysis of cardiac blood flows to the evaluation of myocardial wall motion[11-17]. Our previous reports have documented the usefulness of DTI in identifying the influence of endurance training on LV myocardial longitudinal function[16] as well as the relation between DTI parameters at rest and cardiac performance during effort[17]. A recent report has also analyzed, by this tool,

the difference of myocardial function in different kinds of LV hypertrophy including the athlete's heart, systemic arterial hypertension, and hypertrophic cardiomyopathy[18]. However, few data about the possible effects of strength-training on LV regional function are available at present[18].

On these grounds, the present study was designed to extend the analysis of myocardial regional function to competitive swimmers undergoing either endurance- or strength-training protocols. This issue may be critical to the better understanding of the myocardial mechanisms underlying the LV supernormal systolic and diastolic functions observed in athletes at rest and during physical effort[9,10].

Methods

Study population. After the approval of the Institutional Ethics Committee, 26 top-level endurance-trained (long-distance swimmers, group A) and 20 strength-trained (short-distance swimmers, group B) athletes, both having evidence of LV hypertrophy (LV mass index > 50 g/m$^{2.7}$ according to the Cornell criteria) were included in the study after having obtained their informed consent. Exclusion criteria were: coronary artery disease, valvular and congenital heart disease, congestive heart failure, cardiomyopathies, diabetes mellitus, sinus tachycardia, and echocardiograms of inadequate quality.

Training protocols. All the subjects had been submitted to intensive training (15-20 hours/week for > 4 years). Group A was submitted to intensive aerobic isotonic dynamic exercise at incremental workloads at 70-90% of the maximal heart rate. In particular, they performed 3 hours/day of incremental long-distance swimming (7000 m/day divided into series of 400-800 m), 3 hours/week of long-distance running, and only 2 hours/week of weight-lifting at low workloads.

On the other hand, group B underwent anaerobic isometric static exercise at incremental workloads performed at 40-60% of the maximal heart rate. In particular, their training protocol included both 3 hours/day of short-distance swimming (3000 m/day divided into series of 25-50-100 m) and 1 hour/day of weight-lifting at high workloads.

Procedures. Standard Doppler echocardiography and DTI were performed using an Acuson Sequoia machine (Mountain View, CA, USA) equipped with DTI capabilities. A variable frequency phased-array transducer (2.5-3.5-4.0 MHz) was used for echo-Doppler and DTI imaging. The cuff blood pressure (mean of three measurements) was estimated by a physician blinded to the examination. All the measurements were analyzed by two experienced readers on the basis of the average of 3-5 cardiac cycles. M-mode measurements were performed in the parasternal long-axis view and using the criteria of the American Society of Echocardiography[19]. The LV mass was calculated according to the recommendations of the Penn convention[20], and indexed for height$^{2.7}$ (Cornell adjustment)[21]. The relative diastolic wall thickness was determined as the ratio between the sum of the septal and posterior wall thickness and the LV end-diastolic diameter. The endocardial fractional shortening was calculated as the percentage change in LV internal dimensions between systole and diastole. The circumferential end-systolic stress (ESSc) was considered as a measurement of LV afterload and calculated using a cylindrical model according to the following formula[22]:

$$\text{ESSc (g/cm}^2) = \frac{\text{SBP} \times {}^1/_2\,\text{Ds}^2\,\{1 + [({}^1/_2\,\text{Ds} + \text{Ps})^2 / ({}^1/_2\,\text{Ds} + {}^1/_2\,\text{Ps})^2]\}}{({}^1/_2\,\text{Ds} + \text{Ps})^2 - {}^1/_2\,\text{Ds}^2}$$

where SBP = systolic blood pressure, Ds = end-systolic diameter, and Ps = posterior wall thickness in systole. The stroke volume was obtained using the LV outflow Doppler method and calculated as the product of the outflow tract area and LV output velocity integral[23].

The standard Doppler-derived LV diastolic inflow was recorded in the apical 4-chamber view by placing the sample volume at the tips level[24]. The following LV diastolic measurements were evaluated: the E and A peak velocities (m/s) and their ratio, the E wave deceleration time (ms) and the isovolumic relaxation time (ms, taken as the time interval elapsing between the end of the systolic output flow and the onset of the transmitral E wave as determined by placing the pulsed Doppler sample volume between the outflow tract and the mitral valve)[25]. Our methods and the reproducibility of the Doppler diastolic indexes were previously reported[26].

Pulsed DTI was performed adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15-20 cm/s, and using the minimal optimal gain. In the apical 4- and 2-chamber views, a 5 mm pulsed Doppler sample volume was placed at the level of both the basal septum and the LV inferior wall. The apical view was chosen to obtain a quantitative assessment of the regional wall motion almost simultaneously to the Doppler LV inflow and outflow and to minimize the incidence angle between the Doppler beam and the LV longitudinal wall motion. Pulsed DTI of both the basal septal wall and of the inferior wall is characterized by a myocardial systolic wave (S_m) and two diastolic waves – early (E_m) and atrial (A_m)[11,12]. The myocardial peak velocity of the S_m (m/s), the myocardial pre-contraction time (from the onset of the ECG QRS complex to the beginning of the S_m) and the contraction time (from the beginning to the end of the S_m wave) (all in ms) were calculated as systolic indexes. The E_m and A_m peak velocities (m/s), the E_m/A_m ratio and the regional relaxation time (ms) – considered as the time interval elapsing between the end of the S_m and the onset of the E_m – were

determined as diastolic measurements. Our DTI methods and reproducibility were previously described[13].

Statistical analysis. All the analyses were performed using SPSS for Windows release 9.0 (Chicago, IL, USA). Variables are presented as mean ± SD. The Student's t-test was used for paired data estimated between the two groups. Linear regression analyses and the partial correlation test by Pearson's method were used to assess univariate relations. Stepwise forwards, multiple regression analyses were performed to weigh the independent effects of potential determinants not obviously related to each other (e.g., LV internal end-diastolic diameter and LV mass) on a dependent variable. Differences were significant at $p < 0.05$.

Results

Clinical characteristics of the study population (Table I). As expected, group B showed a higher heart rate, body surface area and systolic blood pressure than group A.

Table I. Characteristics of the study population.

Variable	Group A (n=26)	Group B (n=20)	p
Age (years)	23.2 ± 2.3	22.8 ± 3.4	NS
Sex (male)	16	13	NS
BSA (m^2)	1.88 ± 0.5	1.96 ± 0.6	< 0.05
HR (b/min)	53.1 ± 3.4	68.9 ± 9.9	< 0.001
SBP (mmHg)	116.8 ± 9.1	137.9 ± 7.1	< 0.001
DBP (mmHg)	72.7 ± 4.9	78.9 ± 4.0	NS

BSA = body surface area; DBP = diastolic blood pressure; HR = heart rate; SBP = systolic blood pressure.

Standard Doppler echocardiographic analysis (Table II). The LV mass index and fractional shortening did not significantly differ between the two groups. However, group B showed an increased sum of wall thicknesses (septum + LV posterior wall), ESSc and relative wall thickness, while the LV stroke volume and LV end-diastolic diameter were greater in group A. All transmitral Doppler indexes were higher in group A, with an increased E/A ratio.

Pulsed Doppler tissue imaging analysis (Table III). DTI analysis showed, in group A, a higher E_m and E_m/A_m ratio as well as a longer relaxation time both at the septal and inferior wall levels, with comparable S_m, pre-contraction and contraction times. The differences in systolic and diastolic myocardial peak velocities remained significant even after adjusting for age, heart rate, LV mass index and ESSc, while the prolongation of the relaxation time appeared to be mostly related to the reduced heart rate in group A. Figure 1 shows the DTI pattern of the LV inferior wall in both groups: the E_m peak velocity is very high in group A.

Relations of Doppler tissue imaging indexes. By univariate analysis, the E_m peak velocity of the inferior wall was positively related to the LV end-diastolic diameter in group A ($r = 0.77$, $p < 0.001$; Fig. 2). On the other hand, the peak S_m velocity was directly associated with the sum of the wall thicknesses ($r = 0.68$, $p < 0.001$) in group B (Fig. 3).

These relations were tested by separate multiple linear regression analyses after adjusting for potential confounders such as age, body surface area, heart rate, systolic blood pressure, ESSc and LV mass index. These models showed the independent association of the peak E_m velocity with the LV end-diastolic diame-

Table II. Standard Doppler echocardiographic quantitative analysis.

Variable	Group A (n=26)	Group B (n=20)	p
M-mode echocardiography			
Septal wall thickness (mm)	9.8 ± 2.1	12.3 ± 2.3	< 0.01
Posterior wall thickness (mm)	9.4 ± 2.1	11.8 ± 1.4	< 0.05
Sum of wall thicknesses (mm)	18.6 ± 4.2	23.6 ± 2.8	< 0.001
LV end-diastolic diameter (mm)	55.4 ± 4.7	48.2 ± 3.5	< 0.001
LV end-systolic diameter (mm)	28.3 ± 2.9	26.8 ± 4.1	NS
Relative diastolic wall thickness	0.37 ± 0.04	0.45 ± 0.06	< 0.001
Endocardial fractional shortening (%)	46.7 ± 4.7	45.1 ± 2.8	NS
LV mass index ($g/m^{2.7}$)	62.4 ± 9.9	64.2 ± 8.7	NS
LV ESSc (g/cm^2)	90.1 ± 15.2	168.6 ± 19.5	< 0.001
Standard Doppler analysis			
LV stroke volume (ml)	90.1 ± 6.2	73.4 ± 4.3	< 0.01
Peak E velocity (m/s)	0.96 ± 0.13	0.78 ± 0.16	< 0.01
Peak A velocity (m/s)	0.45 ± 0.12	0.47 ± 0.14	NS
Peak E/A ratio	2.13 ± 0.5	1.6 ± 0.8	< 0.001
E wave deceleration time (ms)	166.6 ± 16.3	158 ± 17.7	NS
Isovolumic relaxation time (ms)	80.8 ± 20.7	68.3 ± 16.6	NS

ESSc = circumferential end-systolic stress; LV = left ventricular.

Table III. Doppler tissue imaging assessment of the basal septal and left ventricular inferior walls.

Variable	Group A (n=26)	Group B (n=20)	p
Basal posterior septal wall			
S_m peak (m/s)	0.17 ± 0.03	0.18 ± 0.04	NS
PCT_m (ms)	104.2 ± 19.7	101.6 ± 18.4	NS
CT_m (ms)	265.7 ± 13.4	257.8 ± 37.2	NS
E_m peak (m/s)	0.21 ± 0.04	0.17 ± 0.02	< 0.01
A_m peak (m/s)	0.09 ± 0.02	0.10 ± 0.02	NS
E_m/A_m ratio	2.3 ± 0.3	1.7 ± 0.4	< 0.001
RT_m (ms)	82.3 ± 12.2	70.2 ± 21.2	< 0.05
Basal inferior wall			
S_m peak (m/s)	0.18 ± 0.02	0.19 ± 0.03	NS
PCT_m (ms)	105.6 ± 18.2	102.5 ± 22.1	NS
CT_m (ms)	280.4 ± 16.1	277.3 ± 25	NS
E_m peak (m/s)	0.24 ± 0.05	0.18 ± 0.3	< 0.001
A_m peak (m/s)	0.08 ± 0.02	0.09 ± 0.02	NS
E_m/A_m ratio	2.9 ± 0.9	1.9 ± 0.8	< 0.0001
RT_m (ms)	86.2 ± 13.1	73.7 ± 15.1	< 0.01

A_m = myocardial atrial diastolic wave; CT_m = myocardial contraction time; E_m = myocardial early diastolic wave; PCT_m = myocardial pre-contraction time; RT_m = myocardial relaxation time; S_m = myocardial systolic peak velocity.

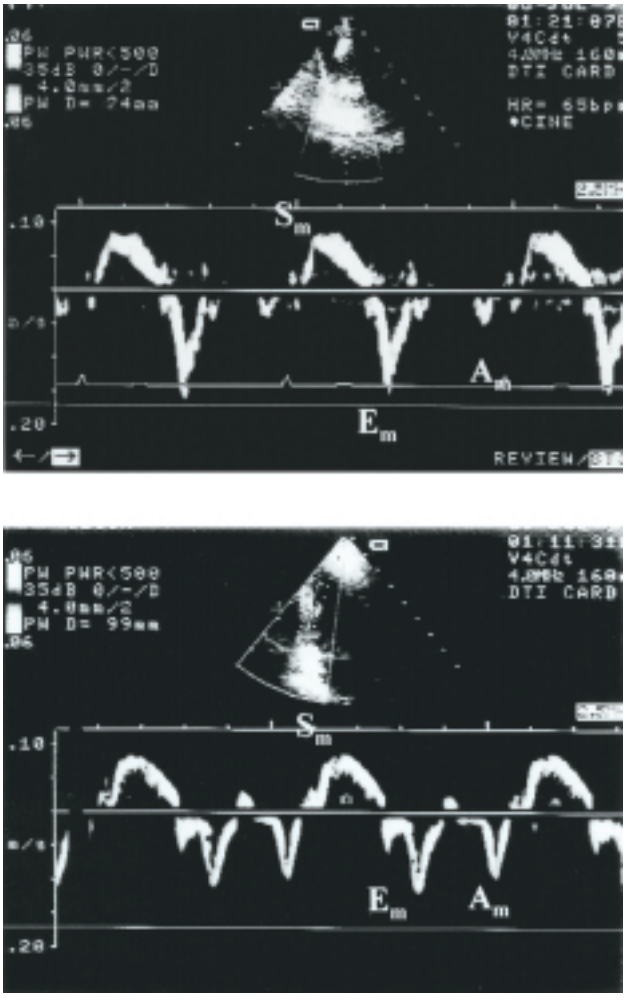

Figure 1. *Pulsed wave Doppler tissue imaging pattern with the sample volume placed at the level of the left ventricular inferior wall in an endurance-* (upper panel) *and in a strength-trained athlete* (lower panel). *A_m = myocardial atrial diastolic wave; E_m = myocardial early diastolic wave; S_m = myocardial systolic wave.*

ter (β coefficient 0.69, $p < 0.001$) and heart rate (β = -0.38, $p < 0.01$) (cumulative $r^2 = 0.76$; SE 0.03 m/s; $p < 0.001$) in group A. Conversely, an independent direct correlation of the peak S_m velocity with the sum of the wall thicknesses (β = 0.64, $p < 0.001$) and ESSc (β = 0.32, $p < 0.01$) (cumulative $r^2 = 0.72$, SE 0.01 m/s; $p < 0.0001$) was observed in group B.

Discussion

This study underscores the usefulness of DTI in the assessment, at a regional level, of the differences between the effects of endurance- and strength-training on LV hypertrophy. Our findings suggest that, in the absence of any difference in LV mass index, 1) group A presents higher E_m peak velocities and an increased E_m/A_m ratio as well as prolonged diastolic time intervals than group B; 2) the E_m peak velocity of the LV inferior wall is independently and positively associated with the LV end-diastolic diameter in group A; 3) in group B the most powerful determinant of the S_m peak velocity is the sum of wall thicknesses.

Left ventricular global structure and function. According to Cornell indexing (very sensitive for increased body size)[21], all the athletes had an increased LV mass. However, two distinct models of cardiac adaptations to the training were highlighted. Endurance athletes, involved in training with high dynamic aerobic components, developed predominantly an increased LV chamber size, with a proportional increase in wall thickness and LV stroke volume caused by volume overload. Thus, they presented with eccentric LV hypertrophy. On the other hand, strength-trained athletes, involved mainly in static isometric

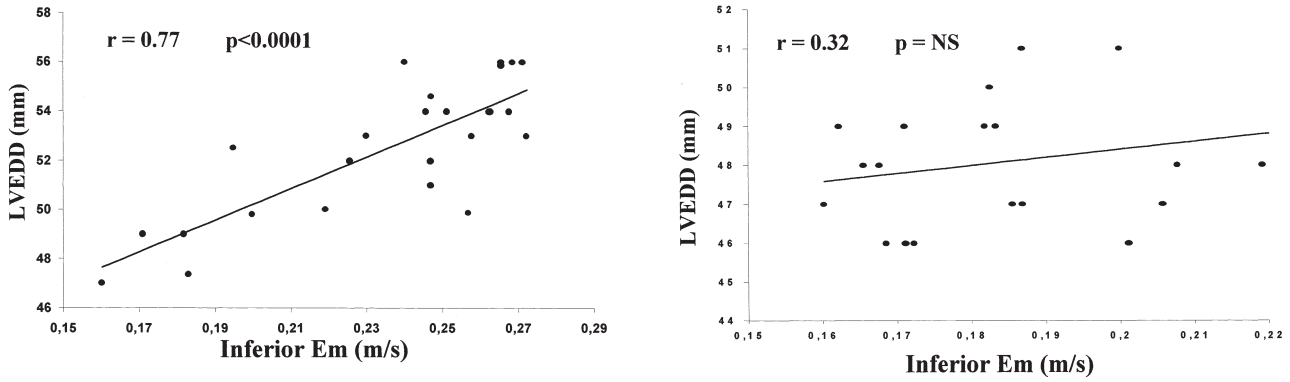

Figure 2. *Relation of the myocardial early diastolic wave (E_m) peak velocity of the left ventricular inferior wall with the left ventricular end-diastolic diameter (LVEDD) in the endurance-trained* (left panel) *and strength-trained* (right panel) *athletes.*

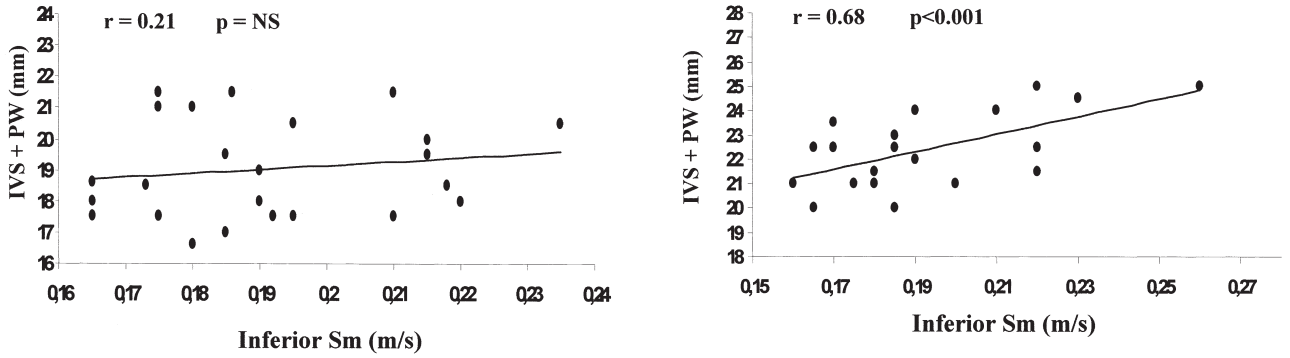

Figure 3. *Relation between the myocardial systolic wave (S_m) peak velocity of the left ventricular inferior wall and the sum of the wall thicknesses in the endurance-trained* (left panel) *and strength-trained* (right panel) *group. IVS = interventricular septum; PW = posterior wall.*

anaerobic exercise, showed an increased LV sum of wall thicknesses and an increased relative wall thickness, with a pattern of LV concentric geometry caused by a high systemic systolic arterial pressure determined by this kind of effort[6-9].

Our findings are consistent with the results of a recent large meta-analysis including 59 echocardiographic studies on 1451 athletes[8], which confirmed the hypothesis of divergent cardiac adaptations in dynamic and static sports.

Global and regional diastolic function. Standard Doppler transmitral analysis showed, in group A, a greater contribution to LV filling by the early-diastolic phase, with a higher E wave and an increased E/A ratio with respect to group B. These differences, observed in the global LV diastolic function, might be expected according to previous studies showing a supernormal diastolic performance of the endurance athlete's heart[10]. Furthermore, in accordance with standard Doppler findings, even the myocardial early-diastolic velocities and E_m/A_m ratios at the level of both the septal and inferior wall were higher in group A than in group B. Since the E_m peak velocity and the E_m/A_m ratio are expressions of the changes in the left atrial to LV pressure gradient as well as of the passive diastolic properties of the LV walls[24,25], their increase in group A indicates a training-induced improvement in LV myocardial compliance.

Relation between the left ventricular preload and myocardial diastolic function in endurance-trained athletes. In accordance with our previous reports[16,17], the present study confirms the close positive relation between the E_m peak velocity, the thickness of the inferior wall and the LV end-diastolic diameter in group A. This relation may be considered a reliable marker of an increased LV preload in the trained heart, and remained significant even after adjustment for several demographic variables, explaining, together with the heart rate, more than 76% of the variability in the inferior E_m peak. The adjustment for age, body surface area, heart rate and systolic blood pressure was needed because of their recognized influence on diastolic indexes[27,28]. The association between the LV end-diastolic diameter and the E_m peak velocity underscores the beneficial effect of a training-increased preload on LV myocardial relaxation. It is theoretically plausible that a greater LV end-diastolic volume might induce earlier and better early diastolic stretching of myocardial fibers (i.e., E_m peak), which, in turn, is probably able to induce an enhanced effort systolic perfor-

mance (i.e., LV stroke volume) by a better utilization of the Frank-Starling mechanism[29]. A preload independence of DTI diastolic measurements was previously demonstrated by placing the DTI sample volume at the level of the mitral annulus[30] but it has to be borne in mind that the DTI measurements of the present study were determined at the level of LV inferior wall. In addition, the load independence of DTI measurements seems to be evident in patients with an impaired LV systolic function while it disappears in those with a normal performance[31,32].

Relation of afterload and myocardial systolic function in strength-trained athletes. Our findings highlighted, in group B, a close positive relation of the S_m peak velocity of the inferior wall with both the sum of the wall thicknesses as well as with the LV ESSc, the latter representing a reliable marker of LV afterload[22]. In a multivariable model, the sum of the wall thicknesses and the LV ESSc were the only independent contributors to the S_m peak velocity, explaining more than 72% of its variability. This association underscores the impact of pressure overload on LV myocardial systolic function in group B. Of interest, in a previous analysis of pathological hypertensive LV hypertrophy we found that the degree of LV hypertrophy was related to the extent of myocardial diastolic impairment, a strong negative association being observed between the septal thickness and the myocardial early diastolic velocities[13]. Conversely, in the healthy population of the present study, rapid increases of the arterial systolic blood pressure during heavy-resistance power training, and the consequent increase in LV afterload, determined the development of a physiological concentric LV hypertrophy, with normal global and regional systolic and diastolic LV functions. Such an increase of the LV wall thickness and of the ESSc may therefore represent the basis to explain the predominant increase in systolic myocardial function in group B[6,7,9] which, in turn, is probably able to better sustain the rapid increases in systolic blood pressure during static isometric exercise.

Conclusions. The present study underscores the usefulness of DTI in identifying the myocardial mechanisms underlying different kinds of physiological LV hypertrophy. The early diastolic myocardial function is positively influenced by the preload increase in group A, while the increased LV wall thickness and end-systolic stress in group B seem mainly to induce an enhancement of regional myocardial systolic function. Therefore, the combined use of pulsed DTI and standard Doppler echocardiography should be taken into consideration when it is necessary to distinguish the different cardiac adaptations in case of endurance or strength sports activities, and eventually to quantify the degree of LV adaptation to long-term training.

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