ORIGINAL ARTICLE

Concentric myocardial hypertrophy after one year of increased training volume in experienced distance runners

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Br J Sports Med 2006;40:706–709. doi: 10.1136/bjsm.2006.027813

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Accepted 16 May 2006
Published Online First
24 May 2006

Objectives: As evidence on the predominant type of cardiac hypertrophy due to endurance running training is inconsistent, the aim of this study was to investigate the effect of increased training volume on echocardiographic variables of distance runners.

Methods: Twenty three adult, experienced, male distance runners underwent standard two dimensionally guided M mode and Doppler echocardiography before and after a one year period during which they were randomly allocated to either control (n = 11) or intervention (n = 12) groups. The intervention group increased their training volume from (mean (SD)) 8.0 (3.0) to 12.5 (3.9) hours/week without increasing the intensity, and the controls changed neither training parameter.

Results: In the intervention group, training induced an increase in left ventricular (LV) mass (from 240.4 (53.8) to 279.5 (60.6) g, $p < 0.001$) and LV mass index (from 126.7 (28.2) to 147.6 (32.3) g/m², $p < 0.001$) mainly due to an increase in end diastolic interventricular septum (from 10.4 (1.8) to 11.5 (1.7) mm, $p < 0.01$) and LV posterior wall thickness (from 10.4 (1.6) to 11.5 (1.6) mm, $p < 0.001$). No significant changes in LV internal diameter or measured indices of LV function occurred ($p > 0.05$). The sum of the right ventricular diameter and wall thickness was greater after the increased volume training ($p < 0.05$). None of the variables changed significantly in the control group ($p > 0.05$).

Conclusions: In experienced, subelite distance runners, further increasing the training volume results in concentric cardiac hypertrophy.

Myocardial hypertrophy due to thickening of the left ventricular (LV) walls and dilation of its cavity is usually observed in endurance athletes.^{1–4} The magnitude of these alterations and the ratio between the extent of each of the two—that is, the type of myocardial hypertrophy, the hypertrophic index, or the relative LV wall thickness—are probably influenced by the athlete's training regimen in line with constitutional and genetic factors.^{1–6} However, these changes vary not only between athletes of different endurance sports,^{2,3,7} but also between athletes from the same kind of sport.^{1,7}

Although moderate changes in LV dimensions are known to be common within weeks of initiation of endurance training of previously untrained subjects^{8–11} as well as in response to detraining or seasonal variations of the training regimen in endurance trained athletes,^{11–17} no data on the alterations in cardiac geometry caused by a prolonged increase in training volume in experienced athletes are available.

Discrepancy exists over the predominant type of LV hypertrophy in myocardial remodelling during adaptation to large volumes of training such as performed by distance runners.^{4,18} In our previous cross sectional study, we reported a positive correlation between the training volume of distance runners and the degree of LV concentric hypertrophy.¹⁹ We hypothesised that, in experienced endurance runners, a prolonged increase in training load due to expansion of its volume and thus augmentation of training induced haemodynamic overload would result in further concentric myocardial hypertrophy. Accordingly, this investigation was performed to evaluate the changes in myocardial geometry in trained endurance runners resulting from a one year increase in training volume. These changes were also interpreted in relation to indices of aerobic capacity and LV lusitropy.

METHODS

Subjects and training intervention

The regional committee of bioethics approved the research protocol. A total of 23 experienced, white, male distance runners were enrolled in this longitudinal study during their active training period. The athletes were introduced to the experimental design, and, after their written, informed consent had been obtained, they were randomly assigned to either a control (n = 11) or intervention (n = 12) group. Each group included four 1500 m runners, and the remainder were specialists in longer distances.

All athletes completed questionnaires about their age, training experience, and weekly training volume averaged over the preceding four weeks (only time devoted to running sessions, including periods of rest between separate bouts of intervals if present, was asked for). All the runners denied use of illicit substances. The training volume as well as other data on the training regimen were obtained from the athletes' diaries. Table 1 presents the characteristics of the subjects of both groups at the initial evaluation.

The runners in the intervention group increased their training volume during the first two months until comfortable, but not by more than 150%, and then continued with the increased training volume for the next 10 months. They were instructed to run at least 90% of the total weekly distance below or at their individual lactate threshold determined on the treadmill before the intervention. Otherwise, no precise control of intensity and amount of running above lactate threshold was implemented. The athletes participated in competitions no more than once every two months. Runners were allowed to alternate the mileage of training sessions as desired. The increase in the

Abbreviations: IVS, interventricular septal; LV, left ventricular

Table 1 Clinical and training characteristics of the runners at baseline

Variable	Intervention (n = 12)	Control (n = 11)
Age (years)	24.7 (7.2)	22.8 (6.0)
Height (cm)	182.0 (6.1)	180.3 (7.0)
Body surface area (m ²)	1.90 (0.10)	1.86 (0.12)
Training experience (years)	10.3 (7.0)	9.9 (7.5)
Training volume (h/week)	8.0 (3.0)	8.3 (3.2)

Data are mean (SD). None of the differences was statistically significant ($p > 0.05$).

number of training sessions was limited to no more than two a week to avoid a possible effect of training frequency.

Athletes in both groups were examined twice: before and after the one year training period. Subjects in the intervention group were tested for a second time within one week of discontinuation of the increased training volume. The training regimen (frequency, duration, and intensity of workouts) did not change substantially during the year of investigation in the subjects in the control group compared with that of the previous year. Non-running activity was negligible in both groups.

Determination of the lactate threshold

Each subject performed an increasing intensity interval treadmill test on a motorised treadmill (h/p/Cosmos Mercury 4.0; Cosmos, Nussdorf-Traunstein, Germany). The test consisted of four-minute running bouts interspaced with four-minute passive rest spans in a seated position. The initial speed was 60% of the subject's average running velocity achieved during the last 5000 m race. Then the speed was increased by 0.7 km/h each bout. Subjects would run on a treadmill the minimum number of times required to increase the blood lactate concentration by no less than 1 mmol/l after two consecutive exercise bouts. A micropipette was used to draw 0.1 ml blood samples from warmed fingertips within one minute of cessation of each bout and immediately analysed by an enzymatic membrane technique.²⁰ Before blood analyses, the analyser was calibrated with standard solutions. The lactate threshold was defined as the point at which blood lactate concentration began to increase systematically above resting values. It was determined using computer aided iterative calculation as the point of intersection of two regression lines, obtained within the range from the first to the last lactate concentration measured during the test, and was expressed as running speed (in km/h). Runners did not exercise for at least 12 hours before the treadmill test and did not eat for at least two hours before the test.

Echocardiography

Echocardiography was performed within one week of treadmill testing. The subjects were asked to refrain from intense exercise for at least 10 hours before echocardiography and not to eat for at least two hours before. Standard transthoracic, two dimensional guided M mode and Doppler echocardiographic examinations were performed, with the subjects recumbent in a left lateral position. An ultrasound scanner (AU3 Partner; Esaote Biomedica, Genoa, Italy) with a 2.5 MHz transducer was used. Measurements were taken from "frozen" M mode tracings obtained using two dimensional guiding in long axis parasternal view, with the ultrasound beam perpendicular to the ventricular walls. Internal LV diameter, interventricular septal (IVS), and LV posterior wall thicknesses were measured at end diastole and end systole as recommended by the American Society of Echocardiography.²¹ Right ventricular diameter and free wall

thickness at end diastole were measured on the same plane. The early (E) and late (A) diastolic peak filling velocities (in m/s) were measured using pulsed Doppler, and the E/A ratio was calculated. The same professional cardiologist, who was blind to the subject's training group, took three measurements, and the average was calculated. Resting cuff blood pressure and heart rate were measured after echocardiographic examination.

Relative wall thickness was obtained by dividing the sum of end diastolic LV posterior wall thickness and IVS thickness by LV diameter. LV mass (in g) was calculated using the following formula:²²

$$LV_{\text{mass}} = 0.8(1.04 \times ((IVS + LVP + LVD)^3 - (LVD)^3)) + 0.6$$

where LVP is left ventricular posterior wall thickness and LVD is internal left ventricular diameter (all in cm at end diastole).

The LV mass index was obtained by dividing calculated LV mass by body surface area. Stroke volume and ejection fraction were computed after calculation of LV end diastolic and end systolic volumes according to the standard equation.²³ End systolic LV wall stress was calculated as described by Grossman *et al.*²⁴

Statistical analysis

Values presented are mean (SD). To estimate the significance of the differences between means, Student's *t* test (for dependent or independent samples where appropriate) was used. The α level was set at 0.05.

RESULTS

During the one year study period, no athlete from either group lost his desire and ability to train because of injuries or other causes. Subjects in the intervention group managed to increase their training volume by on average 58% (range 20–125) without any great accumulation of subjective feelings of fatigue. A typical training session consisted of 80 minutes (about 16 km) before and 120 minutes (about 24 km) during the intervention. The lactate threshold in all runners in the intervention group increased (range 0.1–1.8 km/h; from 14.4 (0.6) to 15.1 (0.6), $p < 0.05$). Table 2 presents echocardiographic and clinical data before and after the intervention as well as those of the control group.

Whereas none of the echocardiographic values or the lactate threshold changed significantly in the control group ($p > 0.05$), athletes in the intervention group responded to the training programme by substantial concentric myocardial hypertrophy. Their relative wall thickness increased significantly by 12% (10 out of 12 athletes in the intervention group showed an increase of 0.02 or more), mainly because of similarly pronounced thickening of both IVS and posterior LV wall (table 2).

These changes resulted in a significant increase in LV mass (by 16.4%) and LV mass index (as body weight did not change). Interestingly, LV internal diameter did not evidence further dilation, and calculated LV ejection fraction and stroke volume remained unchanged ($p > 0.05$). Double product decreased by about 8% ($p < 0.05$), mainly because of lowered heart rate ($p < 0.001$). Doppler indices and calculated end systolic LV wall stress did not change ($p > 0.05$). Although the increases in end diastolic right ventricular free wall thickness and diameter did not reach significance ($p > 0.05$), the sum of these variables (right ventricular cross section) was greater after the intervention ($p < 0.05$).

DISCUSSION

The major finding of our study was the increase in myocardial wall thickness without concomitant dilation of the LV cavity after the increase in training volume in

Table 2 Clinical and echocardiographic characteristics of all the subjects before and after the one year training period

Variable	Intervention group (n = 12)		Control group (n = 11)	
	Before	After	Before	After
Body weight (kg)	69.9 (6.0)	69.5 (6.3)	67.4 (6.6)	68.6 (6.4)
Systolic blood pressure (mm Hg)	133.7 (15.2)	132.5 (17.2)	128.8 (10.2)	131.6 (11.1)
Diastolic blood pressure (mm Hg)	76.8 (11.3)	71.3 (11.9)	69.7 (10.9)	71.0 (7.2)
Heart rate (beats/min)	55.1 (7.3)	50.9 (6.9)‡	58.5 (12.4)	53.2 (9.0)
Double product	7367 (1426)	6744 (1460)*	7529 (1723)	6933 (1296)
Training volume (h/week)	8.0 (3.0)	12.5 (3.9)‡	8.3 (3.2)	8.4 (2.5)
IVS at diastole (mm)	10.4 (1.8)	11.5 (1.7)†	10.2 (1.4)	10.3 (1.1)
IVS at systole (mm)	14.9 (1.8)	15.3 (2.3)	13.5 (1.7)	14.1 (1.3)
LV diameter at diastole (mm)	56.1 (2.9)	56.7 (3.5)	55.4 (3.4)	55.3 (3.1)
LV diameter at systole (mm)	36.5 (3.6)	35.9 (3.5)	35.1 (3.8)	35.5 (4.6)
LV posterior wall at diastole (mm)	10.4 (1.6)	11.5 (1.6)‡	10.5 (1.3)	10.5 (1.1)
LV posterior wall at systole (mm)	17.7 (2.6)	17.9 (1.8)	17.5 (2.4)	17.5 (1.4)
IVS/LV posterior wall at diastole	1.00 (0.06)	1.00 (0.04)	0.97 (0.06)	0.98 (0.07)
LV mass (g)	240.4 (53.8)	279.5 (60.6)‡	233.3 (39.7)	233.5 (38.3)
LV mass index (g/m ²)	126.7 (28.2)	147.6 (32.3)‡	125.7 (23.6)	124.9 (21.3)
Relative wall thickness	0.360 (0.045)	0.400 (0.048)*	0.374 (0.051)	0.376 (0.049)
LV ejection fraction (%)	63.8 (6.7)	65.7 (5.3)	65.0 (6.0)	65.1 (6.8)
Stroke volume (ml)	97.5 (14.0)	104.5 (14.3)	98.5 (11.5)	96.1 (10.8)
ESWS (kdyn/cm ²)	64.7 (20.6)	60.6 (15.6)	59.1 (16.7)	61.3 (15.0)
E (m/s)	0.74 (0.09)	0.76 (0.13)	0.78 (0.08)	0.82 (0.07)
A (m/s)	0.45 (0.07)	0.45 (0.07)	0.47 (0.07)	0.49 (0.05)
E/A	1.68 (0.37)	1.70 (0.29)	1.67 (0.27)	1.67 (0.18)
RV diameter (mm)	20.7 (4.5)	22.8 (4.2)	18.8 (4.0)	22.0 (4.6)
RV free wall thickness (mm)	5.9 (1.1)	6.3 (1.1)	5.9 (0.7)	5.8 (0.5)
RV cross section (mm)	26.6 (4.8)	29.1 (4.7)*	24.7 (4.0)	27.8 (4.6)

Data are mean (SD).

*p<0.05, †p<0.01, ‡p<0.001 compared with before intervention period.

IVS, Interventricular septum; LV, left ventricular; ESWS, end systolic wall stress; E, early peak filling velocity; A, late peak filling velocity; RV, right ventricular.

experienced distance runners. Thus an increase in relative wall thickness was detected. At the same time, resting heart rate and double product decreased, but no changes in E/A ratio (the most commonly used index of LV diastolic function), arterial blood pressure, or end systolic LV wall stress (afterload) were reported.

Interestingly, although it is generally accepted that weight (isometric) or interval—that is, intense—training rather than isotonic endurance workouts, if performed regularly and to a sufficient extent, tends to induce concentric myocardial hypertrophy, whereas the latter voluminous workouts are considered to be adequate stimulus to induce eccentric hypertrophy of the heart,¹ the echocardiographic data after one year of extensive running training applied by our subjects in both the control and (particularly) intervention groups suggest that already trained adult distance runners of subelite level are not able to enlarge their LV chamber size further by using a traditional training regimen. Instead, increased training volume was sufficient to induce further concentric remodelling rather than chamber dilation in our experienced (trained for an average of 10 years) runners. Although problems regarding the ability to detect changes in LV mass using echocardiography in relatively small groups have been reported,²⁵ in our study a prolonged and substantial increase in training volume produced an appreciable concentric myocardial hypertrophy. How and whether the observed changes in cardiac geometry were related to exercise capacity are beyond the scope of this study.

Consistently, Italian investigators have reported that male distance runners extensively involved in professional training develop myocardial hypertrophy mainly through wall thickening.¹⁸ At the end of the intervention period, relative wall thicknesses of our runners had reached values very similar to those presented by Fagard in his meta-analysis.² Together with unchanged echocardiographic data in our control group, this indicates that such voluminous training triggers concentric myocardial hypertrophy in trained, white, male endurance runners.

In contrast, during a three year period of training and competitions, an increase in LV diameter together with thinning of LV walls was reported in top class endurance runners⁴ and professional road cyclists.²⁶ This eccentric LV hypertrophy may be attributable to the very large training volume of these subjects, exceptionally high performance level, and possible use of blood volume expanding medicines and methods. In addition, the LV end diastolic diameter was larger than 70 mm in 33 (11.34%) Japanese 100 km runners.²⁷ All this implies that LV chamber dilation is influenced by a complex interaction of genetic, training—for example, monthly running distance up to 900 km in Japanese athletes—and environmental—for example, ambient temperature—factors, as well as possible drug abuse. Although the exact mechanisms underlying differences in myocardial hypertrophy among athletes remain to be established, evidence exists²⁸ (and our unpublished data indicate) that they are significantly related to the type, magnitude, and duration of exercise related haemodynamic myocardial pressure overload.

The finding of no significant change in LV ejection fraction corresponds to the findings from other studies that even dramatic changes in training loads do not influence this index of integrated LV systolic function at rest.^{13 17 29} It has been reported that endurance athletes differ significantly from untrained healthy subjects with respect to right ventricular morphology similarly to LV morphology.^{30–32} No change in the ratios of LV to the corresponding right ventricular variables (end diastolic diameter or wall thickness) in response to increased training volume in our study agrees with the results of Scharhag *et al.*,³¹ who revealed no differences in proportions of left to right ventricular dimensions between endurance athletes and untrained healthy men. The increase in right ventricular cross section by the end of the intervention supports, and the finding of no change in the ratio of IVS to posterior LV wall thickness confirms, the notion that the heart hypertrophies symmetrically in response to endurance athletic conditioning.

What is already known on this topic

- Moderately increased myocardial mass due to thickening of walls and dilation of cavities is usually observed in endurance athletes such as distance runners
- It is known that the process is partly dependent on the kind, intensity, and amount of endurance training, but how separate training parameters influence the extent and type of adaptation remains controversial

What this study adds

- Augmentation of the training volume of male distance runners without concomitantly increased intensity results in the thickening of myocardial walls but has no effect on the cavities—that is, it induces further concentric hypertrophy of the already dilated left ventricle in experienced subelite endurance athletes
- The increase in myocardial mass was observed to be due to hypertrophy of both ventricles, and was not associated with deteriorating cardiac function

In conclusion, we have shown that, in experienced subelite distance runners, the prolonged augmentation of training volume results in symmetrical thickening of myocardial walls but causes no further dilation in cavities.

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Competing interests: none declared

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