

A comparative study of left ventricular structure and function in elite athletes

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Adaptations to left ventricular (LV) structure and function appear to be dependent on the type, intensity and duration of exercise training. We therefore studied two clearly defined groups of elite athletes, by M-mode and Doppler echocardiography, with a group of inactive individuals as controls. All groups were age matched. Group 1 comprised ten elite endurance athletes with maximal oxygen consumption (VO₂ max) of 74.7 ± 1.43 (mean ± SEM). Group 2 consisted of ten elite weightlifters with VO₂ max 45.3 ± 2.00. Group 3 comprised ten inactive individuals with VO₂ max 44.5 ± 2.13. Left ventricular end diastolic dimension was significantly higher in group 1 (5.72 ± 0.07) than in groups 2 or 3 (5.29 ± 0.09 and 5.19 ± 0.09 respectively, *p* < 0.001). Left ventricular mass index was significantly higher in groups 1 and 2 (156.4 ± 5.97 and 138.6 ± 7.27 respectively) than in group 3 (104.1 ± 3.16 *p* < 0.001). Percentage fractional shortening was used as an index of systolic function and no significant difference was found between groups. Doppler E:A ratio was taken as an index of diastolic function and was found to be significantly elevated in group 1 at rest (3.37 ± 0.24) compared with 2.38 ± 0.16 and 1.99 ± 0.10 in groups 2 and 3 respectively (*p* < 0.003). On exercise, the E:A ratio in group 1 was significantly higher than in group 3 (1.95 ± 0.14 and 1.23 ± 0.05 respectively *p* < 0.001), and tended to be higher than group 2 (1.68 ± 0.15 *p* = ns). These data show that both modes of intense training produce left ventricular hypertrophy. Diastolic function is not impaired in the athletes and may be augmented in the endurance athletes.

Keywords: Left ventricular hypertrophy, elite athletes, Doppler echocardiography, diastolic function

Introduction

All forms of athletic training are associated with left ventricular hypertrophy (LVH). However, the exact effects on cardiac structure and function depend upon the type of training¹⁻³. Endurance training exerts a volume overload on the left ventricle and produces left ventricular cavity enlargement with proportional increases in myocardial thickness⁴⁻⁶. Conversely, resistance training exerts a predominant pressure overload, which results in increased septum

and free wall thickness without affecting cavity dimensions⁴⁻⁶.

LVH also occurs in pathological states such as hypertension and aortic valve stenosis. Pathological LVH is associated with abnormal left ventricular diastolic function⁷. However, the effects of 'physiological' LVH on diastolic function are controversial⁸⁻¹⁰.

The development of 2-D echocardiography and the recent introduction of Doppler techniques has facilitated the investigation of the effect of LVH on diastolic function. The ratio of the peak velocity of blood flowing into the left ventricle in early diastole to the peak velocity during atrial contraction – the E:A ratio – is now widely accepted as a measure of left ventricular diastolic function¹¹. We have employed this Doppler method, along with standard 2-D echocardiographic techniques, to compare two clearly defined categories of elite athletes with age matched controls.

Subjects

Thirty male subjects were studied in three groups. Group 1 consisted of ten highly trained endurance runners of national and international standard. Their mean age was 24.5 years (range 18–31 years). All subjects had been competing and training at a high level for at least 4 years. They were running an average of 45 miles per week at the time of the study.

Group 2 comprised ten highly trained weightlifters, again of national and international standard. Their mean age was 24.3 years (range 20–30 years). Their average Scottish national ranking in their

Table 1. Subject characteristics

	<i>Endurance runners</i>	<i>Weight lifters</i>	<i>Normals</i>
Age (years)	24.5 (1.45)	24.3 (1.23)	24.2 (1.23)
Weight (kg)	65.2 (1.76)	78.7 (3.62)	76.6 (2.74)
Height (cm)	176.3 (2.16)	171.7 (3.05)	179.3 (2.36)
Percentage body fat	9.14 (0.41)	16.6 (1.23)	19.1 (1.29)
Body surface area (M ²)	1.82 (0.04)	1.91 (0.06)	1.95 (0.04)

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particular weight category was number two. All subjects performed intense resistance training programmes ranging from 12 to 20 h per week, and each individual had been competing and training for a minimum of 3 years prior to the study.

At the time of the study, all athletes were training hard in a bid to gain selection for the 1990 Commonwealth games. Subjects taking any pharmacological agents were excluded; in particular we asked any athlete who had used anabolic steroids not to volunteer.

Group 3 comprised ten normal healthy volunteers, mean age 24.2 years (range 20–31 years), who were not trained or taking part in any competitive sport or activity at the time of the study. Table 1 shows the subject characteristics.

Methods

2-D and M-mode echocardiography were performed at rest to determine the pattern of structural changes associated with intense training. These were obtained with a Hewlett Packard (South Queensferry, UK) Ultrasound Imaging System incorporating a 2.5 MHz phased array transducer. The transducer was positioned in the intercostal space to the left side of the sternum which provided the clearest cardiac images.

Septal, posterior free wall and chamber dimension, at both systole and diastole were measured, using the leading edge method (from the most anterior edge of the endocardial and epicardial lines)¹². LV mass was measured using the Penn convention. End diastole was taken at the onset of the QRS complex, and end systole at the point of minimal left ventricular dimension. Variability in measurements taken from the M-mode data was estimated at ± 0.05 mm for septal and posterior free wall measurements, and ± 2 mm for chamber dimensions.

Doppler echocardiography was performed in the supine position at rest and during sub-maximal exercise. The transducer was positioned at the cardiac apex and orientated to obtain a four chamber view of the heart that provided good visualization of the left ventricular cavity and maximal excursion of the mitral valve leaflets. Care was taken to attain the smallest possible angle between the presumed direction of diastolic blood flow and the orientation of the ultrasound beam. The sample volume was placed in the inflow area of the left ventricle just below the mitral valve annulus. This position was then adjusted until maximal diastolic flow velocities were recorded and the Doppler waveform most clearly defined. Doppler analysis was carried out using video playback and integrated software. Variability in analysing Doppler peaks was estimated to be ± 5 cm/s. All measurements quoted are values averaged over at least four cardiac cycles.

The subjects were asked to cycle in the supine position on a specially adapted bicycle ergometer. Workload was initiated at 75 W and held at this level for 1 min, when the workload was increased to 100 W for a further 2 min. Echocardiographic measurements were made during the third minute of sub-maximal exercise. Heart rate was continuously monitored and

systolic blood pressure determined by palpation at the end of each minute of exercise.

All subjects also performed a maximal treadmill exercise test to determine maximal oxygen uptake using an automated gas analysis system (Beckman Metabolic Measurement Cart, Sensor Medics Co., Anaheim, California, USA). Prior to each test the oxygen and carbon dioxide sensors were calibrated using a standard gas mixture of 16% O₂, 4% CO₂, with a balance of N₂. The runners were tested using a protocol specifically designed for elite athletes¹³, while the normals and weightlifters were tested using a different protocol¹³. Plateauing of VO₂, max at exhaustion occurred in all the runners and in almost all the weightlifters and normals. All test lasted between 10 and 14 min and resulted in a respiratory quotient in excess of 1.10 at peak exercise. Maximum heart rate was determined from continuously monitored ECG.

Student unpaired t-tests were used for comparison of intergroup parameters, paired t-tests were used for intragroup comparisons. Overall differences were considered significant if $p < 0.05$. All data are expressed as mean \pm s.e.m. unless otherwise noted.

Results

Cardiorespiratory data at rest and during maximal treadmill exercise are shown in Table 2. Resting heart rate was significantly lower in the group of endurance runners. Resting blood pressures and maximal heart rates were similar between groups. Maximal oxygen uptake was significantly higher in the endurance runners compared with the other two groups ($p < 0.001$).

Resting left ventricular parameters are shown in Table 3. Left ventricular mass index was significantly greater in both groups of athletes compared with control ($p < 0.001$). The endurance runners had significantly larger LVEDD (left ventricular internal dimension at end diastole) than normals ($p < 0.03$). While there was no significant difference in EDD between the weightlifters and normals, LVEDD was

Table 2. Cardiorespiratory data

	Endurance runners	Weight lifters	Normals
Resting heart rate (bpm)	53.1 (3.41)	68.5 (2.36)*	69.3 (2.81)†
Resting systolic BP (mmHg)	120.6 (1.63)	117.7 (1.98)	122.6 (1.86)
Submaximal heart rate (bpm)	81.4 (3.54)	100.1 (2.47)*	100.5 (3.37)*
Maximum heart rate (bpm)	191.6 (3.03)	194.0 (2.36)	194.3 (2.18)
Maximal oxygen uptake (mls/kg/min)	74.7 (1.43)	45.3 (2.0)*	44.5 (2.13)*

* $p < 0.001$ c.f. endurance runners

† $p < 0.002$ c.f. endurance runners
BP, blood pressure

Table 3. Left ventricular parameters

	Endurance runners	Weight lifters	Normals
LV Mass (g)	283.4 (10.4)	260.6 (8.77)	202.1 (5.75)
LVMi (g/m ²)	156.4 (5.97)*	138.6 (7.27)*	104.1 (3.16)
SWT (cm)	1.18 (0.03)	1.15 (0.04)	1.00 (0.03)
PWT (cm)	1.05 (0.03)	1.06 (0.03)	0.88 (0.02)
LVEDD (cm)	5.72 (0.07)†	5.29 (0.09)	5.19 (0.09)
Proportional wall thickness	0.39 (0.01)‡	0.42 (0.01)†	0.36 (0.01)
Fractional shortening (%)	34.5 (2.6)	35.7 (1.9)	35.7 (1.44)

LV, left ventricular; LVMi, left ventricular mass corrected for body surface area; LVEDD, left ventricular internal dimension at end diastole; PWT, left ventricular posterior wall thickness; SWT, ventricular septal thickness

* $p < 0.01$ c.f. normals

† $p < 0.03$ c.f. normals

‡ $p < 0.05$ c.f. normals

higher in the runners than weightlifters, but not significantly so ($p < 0.079$).

Total wall thickness divided by end diastolic dimension was used as an index of relative myocardial thickening. The weightlifters had increased relative thickening compared with controls ($p < 0.003$). Endurance runners also had thickening in excess of increased left ventricular end diastolic dimension compared with control ($p < 0.05$). Fractional shortening, a measure of left ventricular systolic function, was not significantly different in any of the three groups.

Doppler measurements made at rest and during submaximal exercise, are shown in Figure 1. At rest, the ratio of early to late diastolic filling (E:A ratio), an index of left ventricular diastolic function, was higher in the endurance runners ($p < 0.001$). On exercise, the E:A ratio was significantly higher in the endurance runners ($p < 0.001$) and to a lesser extent in the weightlifters ($p < 0.05$) than the controls. When corrected for the differences in resting and sub-maximal heart rate, the endurance runners continued to have significantly higher values ($p < 0.03$ at rest and $p < 0.008$ on exercise).

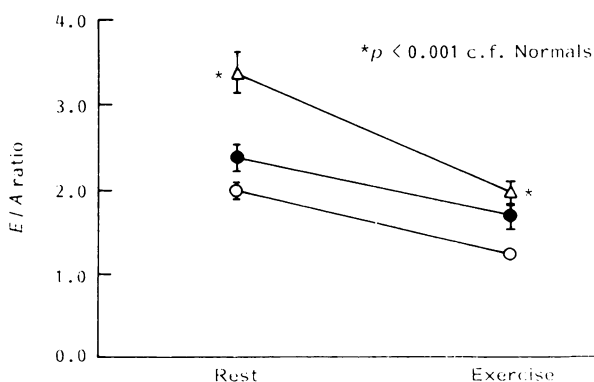


Figure 1. Doppler measurements at rest and during submaximal exercise. Key: △—△: endurance runners; ●—●: weightlifters; ○—○: normals

Discussion

Our finding of increased left ventricular dimensions in the runners agrees with previous studies⁴⁻⁶. Most found that septal and free wall thickness increased proportionately with left ventricular end diastolic dimension, but we found a relatively greater increase in myocardial wall thickness than in left ventricular end diastolic dimension. This is in agreement with the studies of Gilbert *et al.*³ and Granger *et al.*¹⁴ suggesting that disproportionate myocardial thickening can occur in endurance runners as well as resistance trained athletes. The elite calibre of our subjects may have had some bearing on these findings.

The weightlifters showed only marginal, non-significant, increases in left ventricular end diastolic dimension. The predominant pressure overload exerted on the left ventricle resulted in increased LV mass predominantly due to increased wall thickness.

Previous studies have shown that LVH associated with pathological states such as hypertension or aortic stenosis is associated with impaired diastolic function. We found no evidence of impaired diastolic function in either group of athletes despite similar degrees of LVH. In fact, diastolic function was greatly enhanced in the endurance athletes both at rest and on exercise. This would support the hypothesis that two different types of ventricular hypertrophy can occur. 'Physiological' LVH occurs as an adaptation to intense training, while 'pathological' LVH results from disease states. Impaired diastolic function in the pathological condition would not appear to be related to increased left ventricular mass *per se*. It may be due to concomitant left ventricular structural changes such as fibrosis or due to biochemical changes¹⁵⁻¹⁷. In trained athletes, enhancement in diastolic function may be a compensatory mechanism for the decreased diastolic period on exercise. Many mechanisms have been suggested to facilitate this compensation. While augmented venous return from working muscles may increase atrio-ventricular pressure gradient Sabbah and Stein¹⁸ demonstrated that ventricular filling is not a passive process. Penpargkul *et al.*¹⁹ proposed that improved diastolic function could be due to increased calcium transport in the sarcoplasmic reticulum of trained subjects hearts and could probably be associated with physical training.

The present study is limited by the disparity between the resting and submaximal heart rates of the endurance runners compared with the other two groups. At slower heart rates, early diastolic filling may be facilitated by an increase in the diastolic period. Thus a lower heart rate may potentiate a higher E:A ratio^{20,21}. However, enhanced diastolic function in endurance runners was still significant after correction for heart rate. Also, supine bicycle ergometry may not reflect exercise in the upright position, due to increased preload and increased peak filling rate^{22,23}.

In conclusion, these data demonstrate that elite endurance runners and weightlifters develop different forms of left ventricular hypertrophy. The runners had increased chamber dimensions and wall thickness, while the weightlifters exhibited increased

wall thickness only. Neither type of LVH impaired diastolic function, in fact the endurance runners had evidence of augmented left ventricular relaxation.

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