Effect of training on left ventricular structure and function

An echocardiographic study

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SUMMARY To study the cardiac effects of running, 15 healthy non-athletic male volunteers underwent a moderate training schedule for six weeks; thereafter five continued to run at a similar intensity for a further six weeks and 10 stopped. Left ventricular wall thickness and dimension, relaxation, and diastolic function were studied by digitised echocardiography at entry and at two, four, and six weeks, and again at 12 weeks. Significant increases in running ability and maximum oxygen consumption were observed. Maximum oxygen consumption was unchanged in those who continued to run but a reduction was noted in those who stopped. No significant changes in left ventricular dimensions were observed, but the thickness of the posterior wall and the septum increased during six weeks' running. Left ventricular mass (cube volume formula) increased significantly during the first six weeks. Comparison of those who continued to train and those who stopped showed that the former had no change in mass, whereas in the latter mass returned towards pre-exercise values. Systolic function (fractional shortening) was unchanged throughout the study. In addition, no abnormalities of relaxation or diastolic function were detected.

Exercise induced left ventricular hypertrophy may develop rapidly during training and the increased myocardial mass resulting from six or 12 weeks' running is not associated with impaired relaxation and diastolic function as found in pathological forms of hypertrophy.

Running has recently become an increasingly popular leisure activity for the healthy population and for those requiring cardiac rehabilitation. The cardiac effects of exercise training have been studied in high class or champion athletes, weight lifters, dancers, and swimmers and have shown significant increases in left ventricular cavity size and volume.¹⁻⁶ A previous study found that isotonic exercise tends predominantly to increase cavity size, whereas isometric exercise increases wall thickness.⁷ The effect of training on left ventricular function is less clear, with apparently normal, increased, or decreased resting ejection fraction (or fractional shortening).¹⁻⁸ In primary and secondary left ventricular hypertrophy an increase in mass is associated with abnormal diastolic properties such as prolonged and incoordinate relaxation and reduced rate of filling and wall thinning.⁸⁻⁹ The purpose of this study was to observe the rate at which changes in left ventricular structure may take place

during training and detraining of volunteers and to find out whether the increase in mass is associated with impairment of diastolic myocardial properties.

Subjects and methods

Twenty three healthy normotensive and non-smoking volunteers were recruited from male members of the university staff and students who had not recently performed active sport. A physical examination was performed and height, weight, and skin fold thickness (at four sites) were measured.

TRAINING PROGRAMME

The training programme consisted of running five times a week, initially for six weeks. Subjects started at 15 minutes a day for two weeks, rising to 20 and then 30 minutes a day for the latter two periods of two weeks. After six weeks the subjects were asked to continue running for a further six weeks (at 30 minutes a day, five times a week) or to stop completely.

Volunteers were supervised and motivated by one of us (RGS) and the number and duration of each training session were recorded. Assessments (exercise and running tests and echocardiogram) were performed at entry (test 1) and at two (test 2), four (test 3), six (test 4), and 12 weeks (test 5). Resting heart rate was measured after five to 10 minutes' supine rest (after echocardiogram).

EXERCISE ASSESSMENT

Treadmill test

Volunteers were initially tested twice on a motorised treadmill (Powerjog) to achieve skill in running on this apparatus. A third test was used for further analysis and volunteers were thereafter tested once at fortnightly intervals for six weeks and again at 12 weeks. Horizontal running for three minutes (at four, six, and eight miles/h) were interspersed with three minute rests. Pre-exercise and maximum heart rate was measured on an electrocardiograph and oxygen uptake (maximum oxygen consumption calculated) was measured using an Oxycon gas analyser and expressed as ml/kg/min.

Running test

The purpose of this test was to cover as much ground as possible in 12 minutes on a 400 m cinder running track. A supervised test was performed at test 1 to 4 and the result expressed in metres.¹⁰ A running test was not performed at 12 weeks because the track was persistently covered with snow.

ECHOCARDIOGRAPHY

Echocardiograms were performed by one observer (LMS) using a standardised technique in the partial left lateral position with an Electronics for Medicine Echo IV with a 2.25 MHz transducer at a paper speed of 10 cm/s. A left ventricular echocardiogram at the level of the mitral valve tips showing clear continuous echoes from septum and posterior wall endocardium and epicardium was used for further analysis. Echocardiograms were manually digitised using a Summergraphics digitiser and a Prime 750 computer system.¹¹ Measurements were made to the nearest 0.1 cm and 5 ms, which are within the resolution of the echocardiographic apparatus and digitiser respectively. From the records the following measurements were made with no knowledge of the training state of the individual:

- (1) End diastolic (DD) and end systolic cavity dimension (DS) (cm). Fractional shortening was calculated from $(DD - DS/DS) \times 100$.
- (2) Posterior wall (PW) and septal (VS) thickness (cm) at the R wave of the electrocardiogram.
- (3) Peak rate of change of left ventricular dimension

and posterior wall thickness during early diastole (cm/s).

(4) Change in cavity dimension during isovolumic relaxation, expressed as a percentage of the total dimension change during the cardiac cycle.

(5) Time interval (in ms) from minimum left ventricular dimension to the onset of mitral valve opening (measured at cusp separation).

Left ventricular mass was calculated and corrected for body surface area (g/m^2). The simplest equation (cube volume formula) was used as all had normal function and cavity shape¹²: $1.055[(DD + PW + VS)^3 - DD^3]$

CONTROL SUBJECTS

Five healthy male volunteers had a similar fortnightly echocardiographic study to determine the reproducibility of the method.

RELIABILITY STUDY

Twenty randomly selected echocardiograms from subjects during training were remeasured to determine the reliability of the method.

STATISTICAL METHOD

Values are quoted mean $\pm$ 1 standard deviation (SD). Analysis of variance was used to determine the differences between repeat estimations of one measurement. Retest reliability coefficients ($1 - [\text{error variance}/\text{total variance}]$) were calculated, which assumes an ideal value of unity.¹³

Results

Only 15 of 23 volunteers completed at least 27 of 30 possible training sessions and had high quality echocardiograms available for each assessment. These 15 men, who were aged 18 to 49 (mean 26) years, form the basis of this study. After six weeks' running five elected to continue running and 10 to stop.

The reproducibility and reliability of measurements were adequate, with retest reliability coefficients of between 0.91 and 0.97 for remeasuring 20 echocardiograms. Similar measurements of cavity size, wall thickness, and function were obtained from five serial echocardiograms (entry, two, four, six, and 12 weeks) in the five non-training healthy controls. Comparison of the greatest with the smallest of those measurements showed an 11% change for diastolic and 9% for systolic cavity dimensions and the remaining measurements showed a change of less than 5%.

No significant change was observed in the body weight or resting heart rate during six weeks' training, but running ability improved by 11% ($p < 0.01$) (Table 1). At entry, 2775 $\pm$ 122 m was covered in 12 minutes, and this rose to 2900 $\pm$ 134, 2972 $\pm$ 145, and 3075 $\pm$ 114 m (test 4). Similarly, maximum oxygen

Table 1 *Heart rate, weight, and training details*

Subjects	Heart rate (beats/min)	Body weight (kg)	Distance run in 12 mins (m)	Maximum oxygen consumption (ml/kg/min)
Test 1				
All	66±8	75.9±6	2775±122	48.6±4
Continued	68±5	74.3±6	2710±416	47.8±6
Stopped	65±4	77.1±7	2801±154	50±4
Test 2				
All	64±4	75.1±6	2900±134	48.6±5
Continued	63±4	73.1±5	2780±390	50.2±5
Stopped	65±4	76.5±6	2955±158	51±6
Test 3				
All	61±5	75.1±9	2972±145	51.9±6
Continued	59±4	73.4±4	2872±301	51.3±5
Stopped	62±4	76.3±5	3037±114	51.2±4
Test 4				
All	60±6	74.7±11	3075±114*	55.6±7*
Continued	59±7	73.2±8	2984±475*	56.8±6*
Stopped	61±7	76±11	3159±112*	55.2±6*
Test 5				
All	—	—	—	—
Continued	59±6	73.5±7	NA	56.1±6†
Stopped	67±4	77.5±5	NA	48.3±5

NA, not available.

*Significant change during first six weeks' running ($p<0.01$).†Significant difference between those who continued or stopped running ($p<0.05$).Table 2 *Left ventricular dimensions and mass in volunteers during six weeks' training*

Subjects	Diastolic dimension (cm)	Systolic dimension (cm)	Posterior wall (cm)	Septum (cm)	Left ventricular mass (g/m ²)	Fractional shortening (%)
Test 1						
All	5.2±0.4	3.3±0.5	0.9±0.1	0.9±0.1	105±15	36±4
Continued	5.3±0.3	3.4±0.4	0.9±0.1	0.9±0.1	110±14	36±4
Stopped	5.2±0.4	3.2±0.5	0.9±0.2	0.9±0.1	101±12	37±5
Test 2						
All	5.2±0.4	3.2±0.5	0.95±0.1	0.9±0.1	113±16	38±5
Continued	5.3±0.3	3.3±0.3	0.9±0.1	0.9±0.1	111±18	38±5
Stopped	5.2±0.4	3.3±0.4	0.95±0.2	0.9±0.1	115±19	37±5
Test 3						
All	5.3±0.5	3.3±0.3	1.05±0.2	0.95±0.1	135±23	37±4
Continued	5.4±0.5	3.3±0.3	1.0±0.1	1.0±0.1	132±21	37±4
Stopped	5.3±0.4	3.3±0.4	1.1±0.1	0.9±0.2	138±24	38±4
Test 4						
All	5.5±0.4	3.4±0.4	1.0±0.1	1.0±0.1	134±22	38±4
Continued	5.6±0.4	3.3±0.5	1.1±0.1	1.0±0.1	137±18	40±5
Stopped	5.3±0.5	3.4±0.4	1.1±0.1	1.0±0.1	132±17	37±6
Significance*	NS	NS	$p<0.01$	$p<0.05$	$p<0.01$	NS
Test 5						
Continued	5.6±0.3	3.4±0.2	1.1±0.1	1.0±0.1	139±16	39±3
Stopped	5.2±0.3	3.3±0.3	0.9±0.1	0.9±0.1	119±17	37±4

*Analysis of variance.

NS, not significant.

No significant differences between the groups who stopped or continued to train.

consumption increased by 14% from 48.6±4 ml/kg/min at test 1 to 55.6±7 ml/kg/min at test 4 ($p<0.01$). Maximum oxygen consumption was unchanged in those who continued to run (56.1 ml/kg/min±6), but was significantly reduced in those who stopped (48.3±5 ml/kg/min, $p<0.05$).

No significant changes were observed in diastolic or

systolic cavity dimensions during six weeks' training: 5.2±0.4 and 3.3±0.4 cm respectively at test 1 and 5.5±0.4 and 3.4±0.4 cm at test 4 (Table 2). By test 5 those still training had dimensions of 5.6±0.3 and 3.4±0.2 cm respectively and in those who stopped they were 5.2±0.3 and 3.3±0.3 cm (Fig.).

The thickness of the posterior wall and septum

Fig. Serial M mode echocardiograms of a subject during six weeks' training. Test 1 (entry), test 2 (two weeks), test 3 (four weeks) and test 4 (six weeks). Note the increasing thickness of the posterior wall without significant cavity enlargement.

increased significantly from 0.9 ± 0.1 and 0.9 ± 0.1 cm respectively at test 1 to 1.1 ± 0.1 ($p < 0.01$) and 1.0 ± 0.1 cm ($p < 0.05$) at test 4. Left ventricular mass increased significantly ($p < 0.01$) from test 1 (105 ± 15 g/m²) to test 4 (134 ± 22 g/m²). On cessation of running, wall and septal thickness returned towards normal (0.95 ± 0.2 and 0.8 ± 0.2 cm) and therefore mass was reduced to 119 ± 17 g/m². In the

five who continued, the degree of hypertrophy was unchanged (mass 139 ± 16 g/m², difference not significant).

At test 1 all had normal function, and despite a 28% increase in mass no changes in fractional shortening or diastolic function were found during training or detraining (Table 3). There appeared to be no significant differences between the two groups of runners during the first six weeks' training in weight, training details, and left ventricular hypertrophy and function.

Discussion

We have shown that a significant increase in left ventricular wall thickness and mass may develop in untrained volunteers after a period of moderate intensity repetitive exercise. The exercise programme we used was predominantly endurance training, and this appeared to induce wall thickening at quite a rapid rate. In most subjects wall thickness was normal after two weeks of running but in the next four weeks changes of up to 30% in thickness were observed with no significant changes in cavity size. By four to six weeks most had a mass similar to that achieved by professional dancers and elite athletes.¹⁻⁶ No further increase in hypertrophy was observed in the five who continued to run, but a small reduction in wall and septal thickness and mass was shown on cessation of exercise. Other human studies using similar techniques and animal experiments have also shown relatively rapid increases of a similar amount in wall thickness and mass.¹⁴⁻¹⁶ Although all of our subjects would have been classified as moderate to good in running ability¹⁰ and were well motivated, they were essentially non-athletic (in contrast to most studies using high class sportsmen), and from the evidence presented here it would appear that any such individual can obtain the cardiac effects of physical training.

The reliability and reproducibility of the non-invasive cardiac investigations are important in longitudinal studies where relatively small differences in measurements are expected. Although M mode echocardiography lacks spatial resolution, the excellent edge definition and temporal resolution are ideal for the study of wall thickness, relaxation, and diastolic function.^{8,9} To reduce the effect of different body positions and transducer angulation a standardised examination position was used by a single observer.¹⁷ Repeat measurements from the same echocardiogram at a different time and serial fortnightly echocardiograms in normal subjects gave reliability and reproducibility that would appear to be adequate to detect the expected changes.

While there is little doubt that exercise increases

Table 3 *Left ventricular diastolic function during training and detraining*

Subjects	Peak rate wall thinning (cm/s)	Peak rate dimension increase (cm/s)	Minimum dimension to mitral opening (ms)	Dimension change during isovolumic relaxation (%)
Test 1				
All	10±2	15.5±3	4±3	4±4
Continued	10±2	15±3	3±3	3±4
Stopped	10.5±2	15.5±4	4±3	5±3
Test 2				
All	10±3	15±2	2±4	3±2
Continued	10.5±3	16±4	3±3	4±3
Stopped	10±3	15±4	2±2	1±3
Test 3				
All	10.5±3	16±3	7±6	6±5
Continued	11±3	15.5±3	6±5	4±5
Stopped	10.5±3	16±5	8±5	7±4
Test 4				
All	11.5±3	17±2	3±3	4±2
Continued	11±3	16.5±4	2±3	4±3
Stopped	12±3	17.5±4	3±2	3±3
Test 5				
All	—	—	—	—
Continued	12±3	17±2	3±2	3±4
Stopped	11±3	16±3	4±4	3±3

No significant changes.

left ventricular mass, its effect on function is less well defined. Increase in cardiac output during exercise in an untrained individual (maximum two to three times resting level) is due to changes in heart rate; the ejection time is shortened but its rate rises due to the positive inotropic action of increased sympathetic activity and reduced peripheral vascular resistance.¹⁸ A period of training allows exercise to be performed at a lower level of cardiac work (with reduced oxygen requirements) as heart rate changes are augmented by the raised end diastolic volume resulting in increased stroke volume.¹⁴⁻¹⁹ Previous studies have shown increased, normal, or reduced ventricular shortening after exercise training¹⁻⁶; it would seem unlikely, however, that a substantial change in the percentage of the resting ventricular shortening will occur because normal cardiac muscle functions over a relatively small range of lengths and therefore at any particular level of inotropic stimulation the ratio of stroke volume to end diastolic volume (ejection fraction) remains constant. This is true during normal human growth and in many mammalian species, regardless of the relative ratio of body to heart weight.¹⁹⁻²⁰ In pathological causes of left ventricular hypertrophy (aortic stenosis, hypertension, and hypertrophic cardiomyopathy), ventricular shortening is usually normal but diastolic function is often impaired. Relaxation is prolonged and incoordinate, and there is a reduction in the peak rate of wall thinning and filling.⁸⁻⁹ We have shown that an equivalent degree of exercise induced hypertrophy (at least in the short term) is not associated with detectable abnormalities

of diastolic function.

The combination of exercise related changes in wall thickness or cavity size, or both, seems to depend on the nature of the exercise performed. It is suggested that isometric exercise (weight lifting and field events) will induce hypertrophy and endurance training will result in dilatation.⁷ This hypothesis does not appear to be widely applicable, however, as most sports are a combination of isotonic and isometric exercise and dilatation or concentric hypertrophy alone would appear to be non-physiological.²¹ In addition, weight lifters and shot putters often have enormous body mass, and correction of left ventricular mass or cavity size to a normalised end diastolic dimension is probably inappropriate. It may be that these extremes form the spectrum of physiological hypertrophy, but most athletes develop a combination of hypertrophy and dilatation. Other possible explanations are that particular forms of training at the critical periods in development before maturity may be responsible²² or that individuals by their physical (and cardiac) endowment select a sporting activity that suits them best.

The physical properties of the ventricle suggest a direct relation between heart size and workload.¹⁴ During normal growth an increase in cardiac size of up to 20 fold occurs by the addition of new myofibrils in series and parallel. This progressive myocardial cell enlargement occurs in response to the volume load of increasing red cell mass and circulating blood volume to maintain a constant relation between wall stress and systolic pressure.²⁰ After a period of training the left

ventricle responds in a similar fashion with dilatation and hypertrophy. Whether the increased mass of myocardium functions normally depends on various factors which probably differentiate physiological from pressure overload hypertrophy. In the former, mitochondria increase their number, coronary capillary neoformation is induced, and myosin ATPase activity remains normal in contrast to pressure overload hypertrophy.^{14 19-21} We have shown that increased mass itself does not affect function and the differences compared with the pathological forms may be due to the associated ischaemia and fibrosis.²¹

In conclusion, we have shown that in non-athletic individuals the cardiac effects of training may be shown during only six weeks of moderate exercise. Continued running had little additional cardiac effect whereas stopping resulted in reversal of hypertrophy. In addition, significant increases in left ventricular wall thickness and mass were not associated with impairment of systolic or diastolic function.

References

- DeMaria AN, Neumann A, Lee G, Fowler W, Mason DT. Alterations in ventricular mass and performance induced by exercise training in man, evaluated by echocardiography. *Circulation* 1978; 57: 237-44.
- Ehsani AA, Hagberg JM, Hickson RC. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *Am J Cardiol* 1978; 42: 52-6.
- Cohen JL, Gupta PK, Lichstein E, Chadda KD. The heart of a dancer: noninvasive cardiac evaluation of professional ballet dancers. *Am J Cardiol* 1980; 45: 959-65.
- Ikäheimo MJ, Palatsi IJ, Takkunen JT. Noninvasive evaluation of the athletic heart: sprinters versus endurance runners. *Am J Cardiol* 1979; 44: 24-30.
- Gilbert CA, Nutter DO, Felner JM, Perkins JV, Heymsfield SB, Schlant RC. Echocardiographic study of cardiac dimensions and function in the endurance trained athlete. *Am J Cardiol* 1977; 40: 528-33.
- Wieling W, Borghols EAM, Hollander AP, Danner SA, Dunning AJ. Echocardiographic dimensions and maximal oxygen uptake in oarsmen during training. *Br Heart J* 1981; 46: 190-5.
- Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med* 1975; 82: 521-4.
- Gibson DG, Traill TA, Hall RJC, Brown DJ. Echocardiographic features of secondary left ventricular hypertrophy. *Br Heart J* 1979; 41: 54-9.
- Traill TA, Gibson DG, Brown DJ. Study of left ventricular wall thickness and dimension changes using echocardiography. *Br Heart J* 1978; 40: 162-9.
- Cooper T. *New aerobics*. New York: Bantam Books, 1970.
- Gibson DG, Brown D. Measurement of instantaneous left ventricular dimension and filling rate in man, using echocardiography. *Br Heart J* 1973; 35: 1141-9.
- Reichek N. Echocardiographic assessment of left ventricular hypertrophy. *Eur Heart J* 1982; 3 (suppl A): 49-57.
- Guildford JP. *Fundamental statistics in psychology and education*. Tokyo: McGraw-Hill, 1973.
- Wikman-Coffelt J, Parmley WW, Mason DT. The cardiac hypertrophy process. *Circ Res* 1979; 45: 697-707.
- Hickson RC, Holloszy JO. Time course of the regression of cardiac hypertrophy following cessation of endurance exercise training. *Med Sci Sports* 1977; 9: 73.
- Fagard R, Aubert A, Lysens R, Staessen J, Vanhees L, Amery A. Noninvasive assessment of seasonal variations in the cardiac structure and function in cyclists. *Circulation* 1983; 67: 896-901.
- Wong M, Shah PM, Taylor RD. Reproducibility of left ventricular internal dimensions with M mode echocardiography: effects of heart size, body position and transducer angulation. *Am J Cardiol* 1981; 47: 1068-74.
- Sonnenblick EH. The structural basis and importance of restoring forces and elastic recoil for the filling of the heart. *Eur Heart J* 1980; 1 (suppl A): 107-10.
- Ford LE. Heart size. *Circ Res* 1976; 39: 297-303.
- Grossman W. Cardiac hypertrophy: useful adaptation or pathological process? *Am J Med* 1980; 69: 576-84.
- Rost R. The athlete's heart. *Eur Heart J* 1982; 3 (suppl A): 193-8.
- Stein RA, Michielli D, Diamond J, Horwitz B, Krasnow N. The cardiac response to exercise training: echocardiographic analysis at rest and during exercise. *Am J Cardiol* 1980; 46: 219-25.

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