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Cardiovascular effects of training for a marathon run in unfit middle aged men

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Abstract

The effects of a 30 week exercise programme on serum lipid values, blood pressure, and cardiac function were assessed in a group of sedentary men aged 35-50 training for their first marathon. Mean serum cholesterol concentration (n=33) fell by 12% from 6·54 (SE 0·18) to 5·76 (0·15) mmol/l (mean fall 0·78 mmol/l; 95% confidence interval 0·52 to 1·04 mmol/l), serum triglyceride concentration (n=33) by 22% from 1·56 (0·17) to 1·21 (0·09) mmol/l (mean fall 0·34 mmol/l; 95% confidence interval 0·12 to 0·56 mmol/l), and mean blood pressure (n=27) by 10% from 102 (2) to 92 (2) mm Hg (mean fall 10 mm Hg; 95% confidence interval 7 to 13 mm Hg). These changes were not explained by changes in body composition. Peak exercise left ventricular end diastolic volume (n=16) increased with training; as a result of this and an increased exercise left ventricular ejection fraction peak exercise cardiac output increased from 19·9 (1·2) to 23·1 (3·0) l/min (mean rise 3·2 l/min; 95% confidence interval 1·5 to 5·0 l/min). Maximum oxygen consumption increased from 33·9 (1·6) to 39·0 (1·3) ml/kg/min (mean rise 5·0 ml/kg/min; 95% confidence interval 1·8 to 8·2 ml/kg/min).

This study showed favourable effects on coronary risk factors and cardiac function and supports the place of regular exercise in coronary prevention programmes.

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Introduction

The sight of runners on the streets of Britain has become commonplace. This burgeoning of interest has been most evident in non-athletes and is reflected in the number of popular marathons that have taken place in recent years. It is interesting to speculate on the motives behind this activity, though it is a popular belief that exercise is good for the individual and especially for the heart.

Though much is known about the effect of training on cardiorespiratory function in athletes, there is little information concerning the benefits (or otherwise) of prolonged endurance training in previously unfit runners, many of whom are middle aged. Indeed, concern has been expressed about the possible hazards of prolonged exercise in this age group, given the reports of sudden death in marathon runners.

That regular dynamic exercise enhances a feeling of wellbeing seems undisputed; whether it prolongs life is less certain, though it has been shown to ameliorate the effects of coronary heart disease in patients with angina pectoris.[1 2] Retrospective and prospective studies of physical activity have suggested a beneficial effect on cardiovascular mortality, though the evidence is not conclusive.[3 4] Cross sectional studies of endurance athletes and sedentary controls show significant differences in serum lipid values and cardiovascular function.[5-7] Greater credence for a potential beneficial effect of exercise on coronary heart disease in sedentary people might accrue from identifying favourable effects on coronary risk factors.

The purpose of this study was to assess the effects of a structured exercise programme on serum lipid values, blood pressure, and smoking habits in a group of previously sedentary and unfit middle aged men. In addition, we examined in detail the effects of exercise on cardiac function at rest and during exercise.

Subjects and methods

We interviewed 210 respondents to an advertisement in the local newspaper handling the organisation of the Glasgow marathon. We considered only those who had not participated in any form of regular exercise in the previous two years. Fifty six subjects were eligible, but after a medical examination five were excluded, four because of undiagnosed hypertension (blood pressure>170/110 mm Hg) and one because of undiagnosed aortic stenosis; the remaining 51 subjects entered the study. Their mean age was 40 (range 35-50) and mean serum cholesterol concentration 6·40 (SD 1·06) mmol/l, high density lipoprotein cholesterol concentration 1·23 (0·26) mmol/l, and triglyceride concentration 1·60 (1·29) mmol/l. Sixteen were cigarette smokers with a mean consumption of 28 cigarettes a day (range 10-60) and 14 were ex-smokers. Three, 33, 13, and two were from social classes I to IV respectively. Thirty had a family history of coronary heart disease.

Training schedule—Each subject undertook a 30 week structured training programme with assessments before and after 15 and 30 weeks (T1, T2, and T3, respectively). The programme was based on minutes run; training diaries kept by the subjects allowed assessment of the total training time and the milage covered.[8] Only 39 subjects completed the training schedule satisfactorily. The remainder withdrew because of musculoskeletal injury (five subjects), intercurrent illness (two), and occupational commitments (five). Initially the subjects ran on three days a week (around 60 minutes a week), increasing to six days a week by the 20th week (around 360 minutes a week). The mean running time over the 30 weeks was 5659 minutes (range 3027 to 8164) and the mean distance covered 663 miles (1067 km) (range 362 to 1047) miles; 583 to 1685 km).

Variables assessed—The study population recruited was larger than anticipated and we were unable to measure every variable in every subject at each assessment. Occupational commitments and musculoskeletal injury were the commonest reasons for incomplete data in the 39 subjects. The following variables were recorded: body weight and percentage of body fat[9]; fasting total serum cholesterol, high density lipoprotein cholesterol, and triglyceride concentrations; resting heart rate and blood pressure after 20 minutes' recumbency; standard 12 lead electrocardiogram; and cardiac function at rest and at maximum upright dynamic exercise as measured by first pass radionuclide ventriculography. Paired data on serum lipid values were recorded at T1 and T3 in 33 of the 39 subjects. At all three assessments serum lipid values, body weight, and percentage of body fat were available in 20 subjects and blood pressure in 27.

Laboratory methods—Serum total cholesterol and triglyceride concentrations were measured on a COBAS-BIO centrifugal analyser (Roche Diagnostics) employing fully enzymatic methods—that is, with cholesterol oxidase and aminoantipyrine for cholesterol and glycerol kinase and pyruvate kinase for triglyceride—MERCKOTEST reagents being used in each case. High density lipoprotein cholesterol was separated by using Biomerieux precipitating reagents and the cholesterol concentration estimated as above.

Radionuclide ventriculography—Radionuclide ventriculograms were obtained by using a multicrystal gammacamera (Baird Atomic System 77) at rest and peak exercise. The methods and reproducibility of this technique have been reported.[10] Left ventricular ejection fraction was calculated from the standard formula; stroke volume, end systolic volume, and cardiac

output were calculated from this and the end diastolic volume and heart rate. Exercise was carried out on an upright bicycle ergometer starting at 75 W and increasing by 25 W every three minutes. Heart rate, blood pressure, and oxygen uptake were recorded during the last minute of each exercise stage; electrocardiogram leads V1, V5, and aVF were monitored continuously. The time of assessment was kept constant for each subject to minimise possible differences due to diurnal variation. Twenty six subjects were randomly selected for cardiac assessment, of whom 21 completed the training schedule. Technically adequate data were recorded at rest and during exercise at each visit in 16.

Statistical analysis was by Student's paired *t* test. A p value of <0·05 was considered significant in comparisons between T1 and T3 only. In multiple comparisons (T1 *v* T2, T1 *v* T3, and T2 *v* T3) p<0·02 was considered significant. The data are presented as means and standard error (SE) followed by the 95% confidence intervals. Pearson's correlation was used to compare associations between variables.

Results

SERUM LIPID VALUES, BODY WEIGHT, AND PERCENTAGE OF BODY FAT

The groups with data at T1 and T3 and at T1, T2, and T3 (n=33 and n=20, respectively) were comparable before and in their response to the training programme. In the 20 subjects with data at all three assessments total body weight, percentage of body fat, and total serum cholesterol
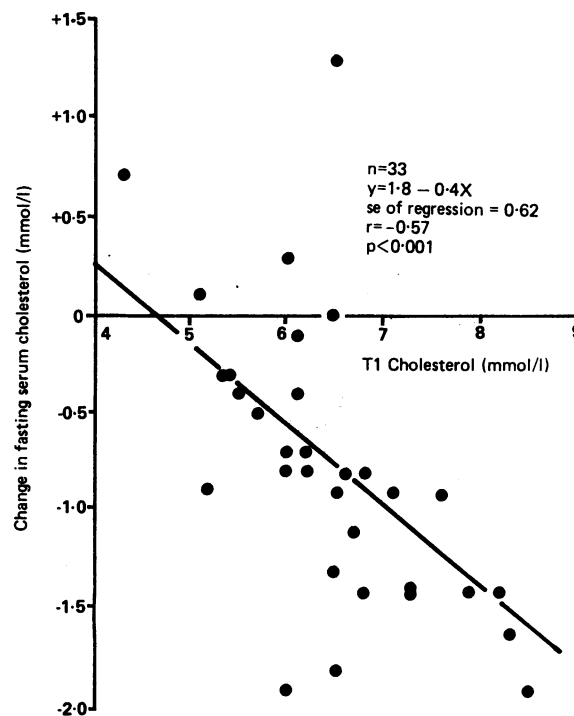
FIG 1—Change in fasting serum cholesterol concentrations after 30 weeks of training. (T1=Pretraining value.)

TABLE I—*Serum cholesterol and plasma triglyceride concentrations, body weight, and percentage body fat before and after 15 and 30 weeks of training (T1, T2, and T3, respectively). Except where stated otherwise values are means with SE in parentheses (n=20)*

| | T1 | T2 | T3 | Mean difference (95% confidence interval) | | |
				T1 *v* T2	T2 *v* T3	T1 *v* T3
Total cholesterol (mmol/l)	6·10 (0·02)	5·75 (0·02)	5·55 (0·02)	−0·36 (0·02 to 0·70) 0·02<p<0·05	−0·20 (−0·11 to 0·51) NS	−0·56 (0·20 to 0·90) p<0·005
High density lipoprotein cholesterol (mmol/l)	1·18 (0·06)	1·19 (0·06)	1·23 (0·08)	0·04 (−0·07 to 0·07) NS	0·04 (−0·11 to 0·19) NS	0·05 (−0·11 to 0·21) NS
Plasma triglyceride (mmol/l)	1·50 (0·19)	1·35 (0·28)	1·24 (0·12)	−0·16 (−0·45 to 0·14) NS	−0·11 (−0·54 to 0·33) NS	−0·26 (−0·03 to 0·50) NS
Body weight (kg)	78·1 (2·5)	75·3 (2·5)	74·1 (2·5)	2·8 (1·5 to 4·1) p<0·001	1·2 (0·2 to 2·3) 0·02<p<0·05	4·0 (2·5 to 5·5) p<0·001
Body fat (%)	22·7 (0·9)	19·7 (0·9)	17·0 (0·7)	2·9 (1·8 to 3·6) p<0·001	2·8 (1·7 to 3·8) p<0·001	5·7 (4·0 to 6·9) p<0·001

TABLE II—*Pre-exercise (upright) heart rate and blood pressure and peak exercise heart rate, blood pressure, and maximum oxygen uptake. Except where stated otherwise values are means with SE in parentheses (n=16)*

| | T1 | T2 | T3 | Mean difference (95% confidence interval) | | |
				T1 v T2	T2 v T3	T1 v T3
Resting:						
Heart rate (beats/min)	74 (4)	61 (3)	62 (3)	−13 (6 to 20) p<0·003	11 (−2 to 4) NS	−13 (6 to 19) p<0·003
Systolic blood pressure (mm Hg)	133 (4)	122 (4)	114 (4)	−11 (5 to 7) p<0·005	−11 (5 to 17) p<0·005	−22 (16 to 27) p<0·001
Diastolic blood pressure (mm Hg)	87 (4)	81 (3)	73 (3)	−6 (1 to 11) 0·02<p<0·05	−8 (1 to 15) 0·02<p<0·05	−14 (5 to 23) p<0·005
Mean blood pressure (mm Hg)	102 (4)	95 (3)	86 (2)	−7 (3 to 12) p<0·005	−9 (3 to 15) p<0·01	−17 (10 to 23) p<0·001
Exercise:						
Heart rate (beats/min)	178 (3)	172 (3)	174 (2)	−5 (−12 to 1) NS	2 (−3 to 7) NS	−4 (−9 to 2) NS
Systolic blood preesure (mm Hg)	213 (7)	222 (6)	209 (4)	9 (−3 to 21) NS	−13 (1 to 25) 0·02<p<0·05	−4 (−17 to 7) NS
Maximum oxygen consumption (ml/kg/min)	33·9 (1·6)	37·8 (1·5)	39·0 (1·3)	3·8 (1·2 to 6·5) p<0·02	1·2 (−1·8 to 4·2) NS	5 (1·8 to 8·2) p<0·01

concentration fell significantly (table I). The concentration of high density lipoprotein cholesterol did not rise significantly. A modest but significant relation was noted between the change in body weight and serum triglyceride value (r=0·54; p<0·02). There was no relation between the change in body weight or percentage of body fat and the change in total serum cholesterol concentration.

In the 33 subjects the serum cholesterol concentration fell by 12% from 6·54 (SE 0·18) to 5·76 (0·15) mmol/l (mean fall 0·78 mmol/l; 95% confidence interval 0·52 to 1·04; p<0·001) (fig 1). There was no relation between the change in maximum oxygen uptake or milage covered and the change in serum cholesterol value. The relation between the duration of exercise (minutes) and the fall in serum cholesterol concentration approached but did not achieve statistical significance (r=0·4; p=0·07). The serum triglyceride value fell in this group from 1·56 (0·17) to 1·21 (0·09) mmol/l (mean fall 0·34 mmol/l; 95% confidence interval 0·12 to 0·56; p<0·005).

Thirteen of the original smokers completed the study and only three persisted in smoking; there was no obvious effect of stopping smoking on any variable.

HEART RATE AND BLOOD PRESSURE

Resting supine heart rate fell significantly from T1 and T2 with no further change at T3 (69 (SE 3), 58 (2), and 58 (2) beats/min, respectively; T2 and T3 p<0·005 v T1). Resting supine blood pressure did not change significantly from T1 to T2 but fell significantly from T2 to T3. Pre-exercise (upright) blood pressure fell significantly throughout the study (fig 2). The change in maximum oxygen consumption, training variables, total body weight, and percentage of body fat were not related to the change in resting supine blood pressure. There was a modest relation between the height of the initial blood pressure and the fall noted at 30 weeks (systolic: r=−0·67, p<0·001; diastolic: r=−0·53, p<0·005). Peak exercise heart rate and systolic blood pressure did not change (178 (3), 172 (3), and 174 (2) beats/min and 213 (7), 222 (6), and 209 (4) mm Hg at T1, T2, and T3, respectively).

ELECTROCARDIOGRAMS

There was no change in the voltages recorded in the electrocardiograms to suggest increased left ventricular mass. In one subject concave upward ST segment elevation developed in the precordial leads after 15 weeks of training. Minor inferior T wave changes often isolated to lead III were common. No ST segment depression was observed during exercise.

RADIONUCLIDE VENTRICULOGRAPHY

In the 16 subjects with technically adequate rest and exercise first pass radionuclide ventriculograms pre-exercise (upright) heart rate and blood pressure fell progressively (table II); this response was similar to that of the whole group (fig 2). Their peak exercise heart rate and systolic blood pressure did not change (178 (SE 3), 172 (3), and 174 (3) beats/min and 213 (7), 222 (6), and 209 (4) mm Hg, respectively).

CARDIAC FUNCTION

Pre-exercise values—There was no change in any resting parameter of cardiac function except pre-exercise cardiac output, which was lower at T2 than T1 owing to the bradycardia induced by training (table III). Resting cardiac output was lower at T3 than T1 but not at the p<0·02 level of significance (p=0·04).

Peak exercise values—Over the 30 week period the peak exercise left ventricular ejection fraction increased by 3 (SE 1) units (1 to 5 units; p<0·02) and end diastolic volume and stroke volume by 22 (9) ml (6 to 37 ml; p<0·01) and 20 (5) ml (9 to 31 ml; p<0·002), respectively (table III). Peak exercise cardiac output increased by 3·2 (0·8) l/min (1·5 to 5·0 l/min; p<0·002); peak cardiac output was also higher at T3 than T2 but not at the p<0·02 level of significance (p=0·04).

Discussion

We have investigated the changes in cardiac function and coronary risk factors that occurred in a group of unfit middle aged men during an exercise programme. The intensity of training was such as to ensure a high degree of cardiovascular fitness.
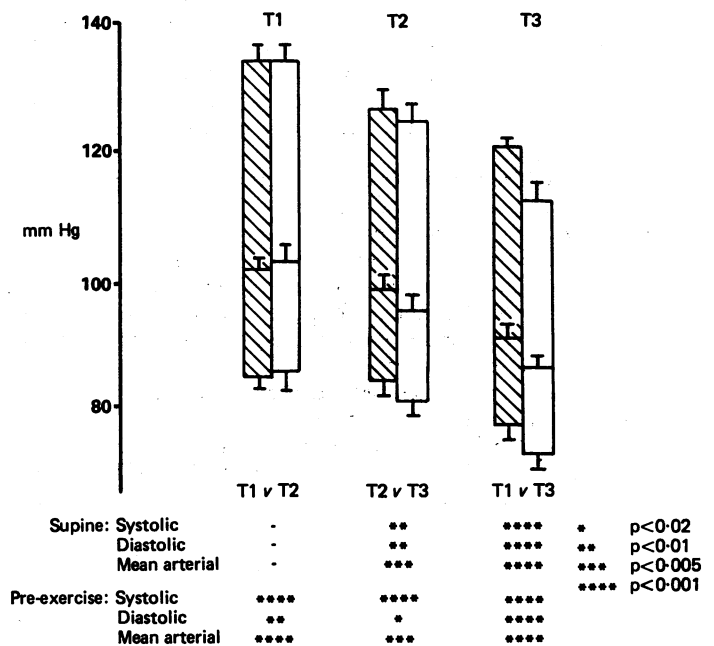
FIG 2—Systolic, diastolic, and mean arterial blood pressures before and after 15 and 30 weeks of training (T1, T2, and T3, respectively). ▨=Supine blood pressure. □=Pre-exercise (upright) blood pressure. Bars are SE. (n=27.)

TABLE III—*Cardiac function at rest and at peak exercise assessed by upright first pass radionuclide ventriculography. Except where stated otherwise values are means with SE in parentheses (n=16)*

		T1	T2	T3	Mean difference (95% confidence interval)		
					T1 v T2	T2 v T3	T1 v T3
Left ventricular ejection fraction	Rest	62 (2)	62 (2)	61 (2)	5 (2 to 8) $p<0.005$	5 (0.3 to 10) $0.02<p<0.05$	9 (5 to 13) $p<0.001$
	Exercise	67 (1)	67 (1)	70 (2)			
End diastolic volume (ml)	Rest	166 (10)	166 (8)	176 (7)	3 (−10 to 17) NS	14 (−2 to 31) NS	15 (−0.3 to 32) NS
	Exercise	170 (12)	180 (8)	192 (9)			
Stroke volume (ml)	Rest	101 (5)	101 (5)	106 (4)	12 (2 to 22) $p<0.005$	20 (3 to 37) $p<0.05$	27 (16 to 38) $p<0.001$
	Exercise	113 (8)	121 (7)	133 (6)			
End systolic volume (ml)	Rest	65 (6)	65 (5)	70 (5)	−8 (−16 to −1) $0.02<p<0.05$	−5 (−15 to 5) NS	−11 (−22 to −1) $0.02<p<0.05$
	Exercise	57 (5)	60 (3)	59 (4)			
Cardiac output (l/min)	Rest	7.3 (0.3)	6.1 (0.3)	6.5 (0.3)	13 (10 to 15) $p<0.001$	15 (12 to 17) $p<0.001$	17 (15 to 19) $p<0.001$
	Exercise	19.9 (1.2)	20.7 (1.1)	23.1 (3)			
Systolic blood pressure: end systolic volume at peak exercise		4.3 (0.5)	3.9 (0.2)	4.0 (0.4)	0.4 (−0.5 to 1.2)	−0.01 (−0.6 to 0.6)	0.3 (−0.4 to 1.1)

A significant fall in total serum cholesterol concentration was observed; as high density lipoprotein cholesterol did not change, the decrease affected the putative atherogenic low density and very low density lipoprotein subfractions. The effects of exercise on serum cholesterol have been extensively studied with variable and inconclusive results.[11][12] The greatest effects have been noted when significant weight loss has occurred, but this association is weak and poorly predictive.[13] We found no significant relation between the change in serum cholesterol concentration and the change in either body weight or percentage of body fat. This suggests that exercise by itself influenced serum cholesterol, and it is interesting to note a possible relation with the total time spent training. Concomitant changes in diet, however, cannot be excluded. Interestingly, the energy expenditure calculated from the subjects' running diaries exceeded that available from the loss of body fat by around 4·2 MJ (1000 kcal) a week,[9] implying an increase in energy intake, despite which the serum cholesterol concentration fell.

Resting supine blood pressure fell, but mainly in the second half of the study. Nevertheless, in the absence of a control group caution should be observed before interpreting these changes as being due solely to the effects of exercise. The fall in blood pressure may have been due to familiarisation with the techniques and personnel concerned. Great efforts were taken to achieve this as soon as possible by fortnightly training sessions; despite this the resting supine blood pressure did not fall until the second 15 weeks.

Weight loss is associated with a reduction in circulating catecholamine values, which may partly explain its hypotensive effect.[14] The fall in blood pressure exceeded that predicted for the observed weight loss:[15] The loss of weight was greatest between T1 and T2, whereas the fall in blood pressure occurred later; no relation was detected between the change in weight and the fall in blood pressure. Thus these data suggest an independent effect of exercise on blood pressure.

In the 16 subjects in whom cardiac output was determined pre-exercise cardiac output fell significantly from T1 to T2 but was similar at T2 and T3. Pre-exercise upright arterial blood pressure fell progressively and was lower at T3 than T2. As resting cardiac output was similar, this implies a continuing reduction in peripheral vascular resistance. The fall in blood pressure was in keeping with reports of a moderate reduction in blood pressure in normotensive and mildly hypertensive subjects.[16-18]

The increase in peak exercise cardiac output at T3 was due to an increased stroke volume, which resulted from an increased end diastolic volume and left ventricular ejection fraction. That the peak exercise left ventricular ejection fraction was highest at T3 does not necessarily imply an increase in contractility. More probably it

reflects changes in ventricular loading conditions. The similar systolic blood pressure to end systolic volume ratio at peak exercise (said to be a measure of contractility relatively independent of loading conditions[19]) supports this.

In conclusion our findings show that a structured exercise programme leading to the completion of a marathon had beneficial effects not only on cardiac function but also on the various components that make up the coronary risk factor profile. The possible long term advantages of these short term improvements are interesting and support the encouragement of regular exercise as part of the general coronary preventive message.

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