Original Research

The Impact of Moderate Aerobic Physical Training on Left Ventricular Mass, Exercise Capacity and Blood Pressure Response During Treadmill Testing in Borderline and Mildly Hypertensive Males

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Introduction: We sought to investigate the effect of moderate physical exercise on left ventricular mass, exercise tolerance and blood pressure response during treadmill testing, in middle-aged pre-hypertensive and mildly hypertensive men without any evidence of coronary heart disease.

Methods: Forty of 52 male borderline and mildly hypertensive subjects (mean age 53 ± 7 years old) with a normal treadmill exercise test and echocardiographic evaluation were randomly assigned to an exercise rehabilitation programme. Patients in the exercise group participated in an aerobic exercise program for 16 weeks, three times per week, at 60 to 80 percent of the maximum heart rate achieved during the preceding exercise test.

Results: At baseline no statistically significant differences between the two groups were observed in METS, in left ventricular mass index, or in blood pressure measurements at rest and during treadmill testing. Sixteen weeks later the exercise group showed higher values of METS compared to the control group ($p<0.001$), while changes in METS from baseline to 16 weeks' follow up differed significantly between the two groups ($p<0.001$ for group-time interaction). Additionally, 16 weeks after randomisation, systolic/diastolic blood pressure and heart rate were significantly lower in the exercise group compared to the control group at all stages of stress testing. Finally, the left ventricular mass index decreased significantly in the exercise group (118.80 ± 3.89 to 96.10 ± 8.95 kg/m) during the 16 weeks of intervention.

Conclusion: This study revealed the beneficial effect of regular exercise training on left ventricular mass index, exercise capacity and systolic/diastolic blood pressure levels in borderline hypertensive patients.

Physical exercise is associated with haemodynamic changes and modulations in the loading conditions of the heart.[1] Furthermore, many epidemiological studies have suggested that a sedentary lifestyle increases the risk for hypertension, whereas increased occupational or leisure-time physical activity is associated with lower levels of blood pressure,[2-4] as well as a reduction in left ventricular mass.[5-9]

Although precise exercise prescriptions for controlling blood pressure and reducing cardiovascular risk vary, recent findings strongly suggest that low-to-moderate intensity exercise (35% to 79% of age-predicted maximum heart rate or 30% to 74% of maximal oxygen uptake) may be more effective in lowering blood pressure than higher intensity exercise.[10] Also, several studies, including a meta-analysis of

29 studies, concluded that exercising more than three times per week had no additional impact on blood pressure reduction.[11-13]

Many studies have illustrated the beneficial role of exercise training in the management of arterial hypertension and improvement of exercise capacity.[14-16] Even in mildly hypertensive subjects, 12 weeks of exercise training has shown a beneficial effect on blood pressure levels and regression of left ventricular hypertrophy.[17] However, data in the literature regarding the effect of training programmes on left ventricular mass in normo- and pre-hypertensive subjects are sparse. The purpose of this study was to evaluate the role of moderate intensity physical training on exercise capacity, blood pressure response during treadmill test, and left ventricular mass index in middle-aged male subjects, with high normal or mildly elevated blood pressure levels at rest and without any clinical evidence of coronary heart disease.

Methods

For the purposes of the study we initially enrolled 100 men who had a negative treadmill test for ischaemic heart disease and high normal or mildly elevated blood pressure levels at rest (systolic blood pressure <160 mmHg). All patients underwent echocardiographic evaluation, where the left ventricular mass was calculated as described below. From the initial population we finally recruited 52 men who had no clinical evidence of coronary heart disease, who agreed to participate in this protocol, who had not been following a regular exercise programme during the last six months, and who were not taking any antihypertensive medication. Inclusion criteria were age between 40 and 60 years, a treadmill test negative for ischaemia, and normal or high normal resting blood pressure measurements, as defined according the latest guidelines.[18] Patients with a history of stroke, heart failure, diabetes mellitus, atherosclerotic disease, alcohol or drug abuse, smoking habits, psychiatric disease, or physical inability to comply with the exercise protocol, were excluded from the study.

Arterial blood pressure was measured at the end of the physical examination with the subject in a sitting position. Blood pressure was recorded three times, with the right arm relaxed and well supported by a table, at an angle of 45° to the trunk. The systolic blood pressure level was determined by the first perception of sound (tapping quality). The diastolic blood pressure I (or phase IV) level was determined when the sounds ceased to be of tapping quality and became fully muffled. Changes in loudness were not considered. Patients whose mean blood pressure levels, calculated from the three measurements, were lower than 140/90 mm Hg (phase V) and were not on antihypertensive medication were classified as non-hypertensive subjects.

Patients who satisfied the inclusion criteria were randomly assigned to the exercise or non-exercise (control) group (26 men in each). The duration of the exercise training was 16 weeks. Six patients from the exercise group were dropped from the study because they did not complete the 16-week exercise programme. From the control group two patients were excluded because they started antihypertensive medications during the study, one patient because he appeared to suffer from depression, and finally three more patients because they did not appear at their appointment for re-evaluation at the end of the four months period. Finally, 40 patients completed the study protocol, 20 in the exercise group and 20 in the control group.

Exercise testing

All patients underwent an exercise treadmill test (ETT) at entry according to the standard Bruce protocol.[19] Patients were encouraged to reach symptom-limited maximal exercise. They were included in the study if they exhibited no evidence of ischaemia during the ETT and achieved at least 90% of their age-predicted maximal heart rate, defined as 220 minus age.[19] In order to allow a more accurately estimated workload, the patients were not allowed to lean on the handrails.

Blood pressure (measured by indirect arm-cuff sphygmomanometry in the right arm), heart rate and estimated workload in METs (1 MET equals 3.5 ml of oxygen uptake per kg of body weight per minute) were recorded at the end of each of exercise stage, at peak exercise, and each minute after the cessation of exercise for a total of three minutes. Exercise capacity (in METS) was estimated on the basis of the speed and grade of the treadmill.[20] The recovery period consisted of a three-minute walk on the treadmill at 2 km per hour and 2.5% grade. Heart rate recovery value was defined as the reduction from peak exercise heart rate to the rate one minute after the cessation of exercise. Exercise pulse pressure was calculated as the difference between systolic and diastolic blood pressure at the end of each exercise stage. The treadmill exercise was evaluated by two physicians who were unaware of this study protocol.

Echocardiography

The echocardiographic evaluation was performed with the patient lying in the left decubitus position, by two physicians who were unaware of the study protocol. Left ventricular diastolic dimensions (LVDD), systolic dimensions (LVSD), interventricular septal thickness (IVS), and posterior wall thickness (PW) were measured in all participants from the left parasternal long axis, according to the guidelines of the American Society of Echocardiography. Left ventricular mass (LVM) was calculated with the use of the anatomically validated formula

$$LVM = 1.04\ (IVS+LVDD+PW)^3-(LVDD)^3-13.6\ g$$

Left ventricular mass was divided by body-surface area (BSA) to obtain the left-ventricular-mass index (LVMI), according the Devereaux formula[21] below:

$$LVMI = LVM / BSA$$

while body-surface area was calculated using the following formula:

$$BSA = height^{0.725} \times weight^{0.425} \times 0.007184$$

Subjects with an LVMI above 116 (kg/m^2) were classified as having moderate left ventricular hypertrophy.[22]

Exercise programme

Patients in the exercise group participated in an aerobic exercise program of 16 weeks' duration. Exercise training consisted of stationary cycling (SportsArt C570r) three times per week. Exercise sessions started with a five-minute warm-up period (stretching and slow cycling) before the prescribed aerobic exercise and ended with an appropriate cooling-off period. Patients exercised for a mean (± SD) of 44 ± 9 minutes, at 60 to 80 percent of the maximum heart rate achieved during an exercise tolerance test.

Heart rate was measured continuously with a portable heart rate monitor (Polar RS200). The monitor was set to maintain a target heart rate and to signal (by a beep) when heart rate fell outside the chosen exercise target range. Blood pressure was monitored periodically during each workout to ensure that it was within safe limits (systolic blood pressure <220 mmHg, diastolic blood pressure <110 mmHg). The workload was adjusted to maintain the target heart rate and blood pressure within the prescribed limits throughout the exercise session. During the exercise training, heart rate was maintained between 111-142 bpm (average 126 bpm). The participants exercised at an average of 75% of their maximum heart rate. The blood pressure was measured three times every 20 minutes during exercise and the average of those measurements was calculated each time.

Bioethics

The study's protocol was approved by the Ethical Committee and Internal Review Board of our Institute (2003). All participants were informed about the aims and procedures of the study and gave their written consent.

Statistical analysis

We calculated that at least 20 patients in each group were needed to achieve a statistical power of at least 80% for the detection of a two-sided difference in LVMI change of 45 ± 7.3% between study groups from baseline to the end of the study, at a significance level $p<0.05$. Every patient enrolled in the study was randomised to control or exercise group by turns, and the intention to treat (ITT) principle was followed during the analysis. Data are presented as mean ± standard deviation. The normality of all continuous variables was evaluated using the Kolmogorov-Smirnov test. The difference between baseline and 16-week values of various echocardiographic and stress test parameters in each group was evaluated using the sign test, when the variable describing the difference in the parameters was not normally distributed, or the paired t-test, when it was normally distributed. For the comparisons of continuous variables between the two groups at each time point we used the Student t-test for normally distributed variables and the Mann-Whitney test for skewed variables. Correlations between continuous variables were evaluated using the Spearman correlation coefficient. Finally, repeated measures ANOVA was used to account for potential baseline differences between study groups. All statistical analyses were performed using SPSS 14 (SPSS Inc., Chicago, IL, USA).

Results

The baseline age, weight and body mass index of the participants are presented in Table 1. At baseline, no significant difference was observed in the mean

Table 1. Baseline characteristics of the study population.

	Control group	Exercise group	p*
Age (years)	55.3 ± 6.4	51.7 ± 8.2	0.135
Weight (kg)	80.4 ± 6.1	75.4 ± 7.9	0.032
Body mass index (kg/m^2)	26.00 ± 1.9	25.02 ± 2.4	0.165
METS	11.4 ± 1.6	11.0 ± 1.9	0.528

*Student's t-test.

values of age, and BMI. Regarding LVM values, no baseline differences were observed between the two groups; however, after 16 weeks of follow up, LVM was higher in the control group compared to the exercise group (Table 2). Changes of all echocardiographic parameters from baseline to 16 weeks' follow up differed significantly between the control and exercise groups (Table 2). Furthermore, 16 weeks after the randomisation, all parameters had decreased significantly in the exercise group, while in the control group LV diameters and LVM had increased significantly (Table 2).

Tables 3, 4 and 5 show the values of systolic/diastolic blood pressure and heart rate at different stages of stress testing in both groups at each time point, and the changes from baseline to 16-week follow up in each group. Sixteen weeks after randomisation, systolic/diastolic blood pressure and heart rate had decreased significantly in the exercise group at all stages of stress test, whereas the control group showed no such change. In addition, resting pulse pressure value (PP), defined as the difference between systolic and diastolic blood pressures, decreased significantly from baseline to 16 weeks after randomisation only in the exercise group, while changes in heart rate recovery value did not differ significantly between the control

Table 2. Results from the echocardiographic evaluation.

	Control group	Exercise group	p
End diastolic posterior LV wall thickness (cm)			
Baseline	1.090 ± 0.05	1.110 ± 0.04	0.35
After 16 weeks	1.093 ± 0.05	1.007 ± 0.06	<0.001
Change	0.003 ± 0.02	-0.103 ± 0.06†	<0.001
End diastolic ventricular septum thickness (cm)			
Baseline	1.115 ± 0.05	1.110 ± 0.05	0.82
After 16 weeks	1.093 ± 0.06	1.009 ± 0.05	<0.001
Change	-0.023 ± 0.06	-0.100 ± 0.04†	<0.001
Left ventricular end-systolic diameter (cm)			
Baseline	2.795 ± 0.25	2.880 ± 0.29	0.31
After 16 weeks	2.780 ± 0.24	2.595 ± 0.28	0.03
Change	-0.015 ± 0.22	-0.285 ± 0.16*	<0.001
Left ventricular end-diastolic diameter (cm)			
Baseline	4.785 ± 0.14	4.725 ± 0.27	0.90
After 16 weeks	4.865 ± 0.12	4.531 ± 0.23	<0.001
Change	0.080 ± 0.11*	-0.194 ± 0.23*	<0.001
Left ventricular mass (g)			
Baseline	227.733 ± 13.34	225.100 ± 16.95	0.58
After 16 weeks	231.131 ± 13.11	181.877 ± 18.65	<0.001
Change	3.398 ± 6.76*	-43.224 ± 18.63*	<0.001
Left ventricular mass index (kg/m$^{2.7}$)			
Baseline	115.948 ± 5.98	118.80 ± 3.89	0.08
After 16 weeks	117.527 ± 6.78	96.10 ± 8.95	<0.001
Change	1.579 ± 3.60	-22.69 ± 9.67†	<0.001

p-values for differences between groups at each time point were calculated using the Mann-Whitney test, while other p-values were calculated using Students' t-test.
*p<0.05 from paired t-test for differences between two time points within each group. †p<0.05 from sign test for differences between two time points within each group.
The p-value for differences between the changes in the two groups comes from the test for group-time interaction of repeated measures ANOVA.

Table 3. Systolic blood pressure (mmHg) during stress testing.

	Control group	Exercise group	p
Supine position before start of stress test			
Baseline	137.25 ± 17.13	131.50 ± 13.48	0.24
After 16 weeks	134.25 ± 14.89	119.45 ± 6.87	<0.001
Change	-3.00 ± 5.93*	-12.05 ± 8.17*	<0.001
Upright position before start of stress test			
Baseline	133.50 ± 14.69	133.00 ± 10.80	0.90
After 16 weeks	133.50 ± 14.96	121.25 ± 7.23	0.003
Change	0.00 ± 2.29	-11.75 ± 8.32†	<0.001
End of 1st stage			
Baseline	162.00 ± 17.50	156.76 ± 13.98	0.30
After 16 weeks	163.6 ± 16.72	134.50 ± 8.47	<0.001
Change	1.6 ± 2.54†	-22.25 ± 11.52†	<0.001
End of 2nd stage			
Baseline	181.05 ± 12.95	176.85 ± 9.19	0.24
After 16 weeks	181.15 ± 11.79	147.70 ± 8.57	<0.001
Change	0.1 ± 2.42	-29.15 ± 10.63†	<0.001
End of 3rd stage			
Baseline	190.00 ± 17.95	186.00 ± 18.04	0.49
After 16 weeks	190.74 ± 16.18	164.2 ± 7.57	<0.001
Change	0.74 ± 6.86	-21.85 ± 17.66*	<0.001
End of 4th stage			
Baseline	191.66 ± 21.4	190.18 ± 10.22	0.84
After 16 weeks	191.66 ± 16.32	171.10 ± 6.56	0.03
Change	0.00 ± 5.47	-18.00 ± 7.86*	<0.001
Maximum systolic blood pressure			
Baseline	192.50 ± 17.80	192.60 ± 12.05	0.98
After 16 weeks	193.60 ± 16.23	173.10 ± 6.57	<0.001
Change	1.10 ± 5.02	-19.0 ± 13.00*	<0.001

*p<0.05 from paired t-test for differences between two time points within each group. †p<0.05 from sign test for differences between two time points within each group.
The p-value for differences between the changes in the two groups comes from the test for group-time interaction of repeated measures ANOVA.

and exercise groups (p= 0.107 for group-time interaction). Finally, changes in LVM were highly correlated with changes in both systolic and diastolic blood pressure (rho>0.45, p<0.001).

No significant difference between the two groups was observed at baseline in the values of METS (11.4 ± 1.6 vs. 11 ± 1.9, p=0.528), while 16 weeks after randomisation the exercise group had higher values of METS compared to the control group (13.8 ± 1.6 vs. 10.8 ± 1.9, p<0.001). Changes of METS from baseline to 16 weeks' follow up differed significantly between the two groups (p< 0.001 for group-time interaction).

Discussion

This study revealed that regular aerobic exercise in a programme of 16 weeks promotes a reduction in both resting and exercise blood pressure levels, and leads to an improvement in exercise tolerance in middle-aged men who have no evidence of coronary heart disease and high normal or mildly elevated rest blood pressure levels. Furthermore, LVMI was significantly reduced in the intervention group but not in the control group.

A physically activity lifestyle has been recognised to have beneficial effects on overall health, and especially on cardiovascular morbidity and mortality. Furthermore, most of the beneficial effects of physical activity on cardiovascular health have been linked to the modification of several "traditional" coronary risk factors, such as blood pressure levels and body mass index.[22] Although the duration, intensity and volume of physical activity are still under consideration, it is becoming more apparent that most of the health benefits at a minimal risk are derived from low to moderate intensity physical activities. Many studies have illustrated the beneficial role of exercise training in the management of arterial hypertension and improvement of exercise capacity.[14-16] Even in mildly hy-

Table 4. Diastolic blood pressure (mm Hg) during stress testing.

	Control group	Exercise group	p
Supine position before start of stress test			
Baseline	83.75 ± 6.25	83.00 ± 4.97	0.64
After 16 weeks	82.60 ± 5.22	76.55 ± 4.88	0.001
Change	-1.15 ± 3.26	-6.45 ± 4.92*	0.001
Upright position before start of stress test			
Baseline	83.75 ± 6.46	84.50 ± 5.35	0.69
After 16 weeks	82.75 ± 5.25	77.35 ± 4.49	0.003
Change	-1.00 ± 3.07	-7.15 ± 4.30*	<0.001
End of 1st stage			
Baseline	85.50 ± 6.67	85.50 ± 6.46	0.98
After 16 weeks	84.00 ± 5.98	78.25 ± 4.94	0.003
Change	-1.50 ± 2.85*	-7.25 ± 6.78*	0.004
End of the 2nd stage			
Baseline	84.50 ± 6.26	86.50 ± 7.27	0.44
After 16 weeks	83.50 ± 5.40	79.00 ± 5.53	0.01
Change	-1.00 ± 5.03	-7.50 ± 7.69*	0.002
End of the 3rd stage			
Baseline	83.68 ± 8.13	88.25 ± 8.31	0.11
After 16 weeks	84.74 ± 6.76	80.6 ± 6.31	0.038
Change	1.05 ± 2.67	-7.65 ± 9.91*	<0.001
End of the 4th stage			
Baseline	81.66 ± 6.83	90.90 ± 9.43	0.03
After 16 weeks	81.67 ± 6.83	82.25 ± 7.52	0.79
Change	0.00 ± 0.00	-8.64 ± 8.68*	0.03
Maximum diastolic blood pressure			
Baseline	84.50 ± 7.93	88.75 ± 8.25	0.12
After 16 weeks	85.25 ± 6.97	82.25 ± 7.52	0.10
Change	0.75 ± 2.45	-6.50 ± 8.59*	<0.001
Rest pulse pressure at standing position			
Baseline	49.75 ± 13.81	48.5 ± 10.64	0.75
After 16 weeks	50.75 ± 13.5	43.90 ± 6.47	0.05
Change	-1.00 ± 4.16	-4.60 ± 8.84*	0.01

p-values for differences between groups at each time point were calculated using the Mann-Whitney test.
*$p<0.05$ from sign test for differences between two time points within each group.
The p-value for differences between the changes in the two groups comes from the test for group-time interaction of repeated measures ANOVA.

pertensive subjects, 12 weeks of exercise training has shown a beneficial effect on blood pressure levels.[17]

Exercise is beneficial because it causes heart muscle cells to enlarge, leading to a type of hypertrophy. However, this is quite different from hypertrophy due to chronic hypertension, which involves an excessive increase in fibroblasts, extracellular matrix, fibrosis, death of cardiomyocytes and ultimately heart failure.[23] Although the increase in heart mass is similar in both physiological and hypertensive remodelling, the former is homogeneous and leads to a proportionate increase in the myocyte and non-myocyte components of the myocardium. By contrast, hypertensive remodelling is inhomogeneous and results in a disproportionate contribution of cardiac fibroblasts that produce excessive interstitial fibrillar collagen, leading to a condition that is similar in some respects to the formation of scar tissue. These conditions can also lead to a loss of cardiomyocytes by apoptosis or necrosis and to their replacement by fibroblasts and extracellular collagen.[24] Additionally, hypertensive patients with left ventricular hypertrophy seem to have reduced exercise tolerance.[25,26]

Cardiovascular exercise training is the most effective mode of exercise in the prevention and treatment of hypertension, and the current recommendations suggest exercise lasting 20-60 minutes, 3-5 days per week, at 40-70% of maximum oxygen uptake. Recent statements from the Surgeon General,[27] the National Institutes of Health Consensus Development Panel on Physical Activity and Cardiovascular Health,[28] and the Centre for Disease Control and Prevention and the American College of Sports Medicine[20] recommend at least 30 minutes of moderate-intensity physi-

Table 5. Heart rate (beats/ min) during stress test.

	Control group	Exercise group	p
Supine position before start of stress test			
Baseline	78.20 ± 12.14	75.65 ± 11.12	0.49
After 16 weeks	79.45 ± 11.46	69.20 ± 7.78	0.002
Change	1.25 ± 3.35	-6.45 ± 8.13*	0.001
Upright position before start of stress test			
Baseline	83.00 ± 14.33	81.45 ± 12.53	0.71
After 16 weeks	82.25 ± 12.35	73.20 ± 5.97	0.005
Change	-0.75 ± 4.73	-8.25 ± 9.63*	0.003
End of 1st stage			
Baseline	118.25 ± 15.80	113.85 ± 18.55	0.42
After 16 weeks	120.50 ± 14.42	100.75 ± 7.90	<0.001
Change	2.25 ± 2.31*	-13.10 ± 15.88*	<0.001
End of 2nd stage			
Baseline	135.50 ± 15.59	135.45 ± 17.03	0.99
After 16 weeks	137.45 ± 14.04	119.25 ± 8.39	<0.001
Change	1.95 ± 2.82*	-16.20 ± 13.57*	<0.001
End of 3rd stage			
Baseline	155.05 ± 16.05	158.10 ± 17.43	0.57
After 16 weeks	157.32 ± 11.25	138.70 ± 12.86	<0.001
Change	2.26 ± 9.28	-19.40 ± 14.87*	<0.001
End of 4th stage			
Baseline	157.83 ± 10.13	168.00 ± 12.64	0.11
After 16 weeks	164.50 ± 4.13	163.05 ± 9.64	0.72
Change	6.67 ± 7.60	-5.55 ± 5.20*	0.001
Maximum heart rate			
Baseline	160.35 ± 11.69	166.15 ± 13.50	0.15
After 16 weeks	161.10 ± 9.52	163.05 ± 9.65	0.52
Change	0.75 ± 7.79	-3.10 ± 8.69	0.01

*p<0.05 from paired t-test for differences between two time points within each group.
The p-value for differences between the changes in the two groups comes from the test for group-time interaction of repeated measures ANOVA.

cal activity on most, preferably all, days of the week to prevent coronary heart disease and other chronic diseases. It is unclear whether durations longer than 30 minutes per session produce significantly greater reductions in blood pressure, while there is evidence to suggest that high intensity exercise (>75% VO-2max) may not be as effective as low intensity exercise (<70% VO2max) in reducing elevated blood pressures.[29,30] The combination of pharmaceutical treatment with exercise training seems to be independently effective for the reduction of left ventricular hypertrophy, leading to enhancement of myocardial capillarisation in adults.[31,32] In this study, in patients with high normal and mildly elevated blood pressure levels at rest, exercise training led to a significant reduction in LVMI, even in those with hypertrophy.

Recently, much attention has been given to the relation between pulse pressure and cardiovascular mortality and morbidity.[33,34] In the present study, we found that the change in pulse pressure at rest from baseline to 16 weeks after randomisation was significantly higher in the exercise group compared to the controls. Specifically, the subjects in the exercise group showed a significant reduction in resting pulse pressure levels after the 16 weeks of intervention, reflecting a beneficial effect of exercise on the arterial elastic properties, whereas the control subjects showed no such change. Increased pulse pressure levels have been recognised to reflect increased stiffness of the large arteries and have been reported to have a predictive value for an ischaemic response during exercise.[35,36]

Limitations

The use of treadmill time to quantify exercise tolerance, as used in this study, is not as precise as the measurement of peak oxygen consumption with cardiopulmonary exercise testing. Another limitation is the lack of ambulatory blood pressure measurement in all subjects in order to recognise those with mild hypertension and to follow the response of blood pressure levels after the 16 weeks' intervention in both groups. Another observation is that, in order to obtain a correct interpretation of the result of the physical conditioning, it is necessary to have specificity between the type of treadmill test and the type of training. Therefore, it would have been better to carry out the training assessment with a stationary cycle ergometer, as used in the exercise programme, and not with a treadmill exercise test. Another limitation of this study is that, although we used brachial blood pressure recorded in the conventional way, we know that its precision can be reduced as the treadmill speed increases, because it becomes increasingly difficult to measure. Since blood pressure assessment during the treadmill test was one of the main points of the study, it would have been better to evaluate the patient with a stationary cycle ergometer.

Conclusions

Our study revealed the beneficial effect of regular physical training on the cardiovascular system in middle-aged men with high normal or mildly elevated blood pressure. Specifically, regular aerobic exercise in a programme of 16 weeks promotes reduction in blood pressure levels, both at rest and during treadmill exercise testing, as well as an improvement in exercise tolerance. Furthermore, exercise training leads to a significant reduction in LVMI, even in those with hypertrophy.

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