

The effects of Aerobic Exercise Training on resting Blood Pressure in Hypertensive Patients

M. A. Mughal (Department of Pharmacology Ziauddin Medical University, Clifton, Karachi.)

I. A. Alvi (Department of Therapeutics, Ziauddin Medical University, Clifton, Karachi.)

I. A. Akhund (Department of Medicine, Ziauddin Medical University, Clifton, Karachi.)

A. K. Ansari (Department of Physiology, Ziauddin Medical University, Clifton, Karachi.)

Abstract

Objective: To see the effects of aerobic exercise, on changes in blood pressure, in patients with essential hypertension. A 12-weeks aerobic exercise intervention trial was conducted, to examine the influence of brisk walking on resting systolic and diastolic blood pressure, pulse pressure, mean arterial blood pressure, body weight and body mass index in patients with essential hypertension.

Subjects and methods: Twenty-seven men with stage 1 or 2 essential hypertension (not on antihypertensive medication) participated in the study. The aerobic exercise training protocol consisted of 30 minutes of brisk walking 3 to 5 times per week, at 50% of VO₂max on an ergometer cycle. The data were analyzed by comparing exercise responses at baseline and 12-weeks.

Results: Statically significant decrease in resting systolic [mean+SEM, 143.2+1.4 to 137.5+1.2 mmHg, mean reduction -5.7 mmHg, p<0.01] and diastolic [mean+SEm, 91.8+1.0 to 90.4+0.9 mmHg, the mean reduction -1.4 mmHg] blood pressure wre found (p<0.05). Reduced pulse pressure from baseline value of -3.7mmHg, (p<0.01) and mean arterial pressure of -3.4 mmHg (p<0.01) was noted. No discernible effects on mean body mass index was observed although mean body weights decreased -1.1kg, (p<0.05). Brisk walking yielded significant increase in VO₂max (p<0.05).

Conclution: Aerobic exercise caused small reduction in resting systolic and diastolic blood pressures in men with stage 1 or 2 essential hypertension. A lifestyle change such as exercising. may play a role in reducing the risk of hypertension (JPMA 51:222;2001).

Introduction

Hypertension (systolic and diastolic blood pressure >140/90 mmHg) is a major modifiable risk factor for cardiovascular disease. Elevated blood pressure levels have been shown to be a risk factor for stroke, congestive heart failure, myocardial infarction, peripheral vascular disease, and end-stage renal disease¹. The recent Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure, recommended that optimal blood pressure levels should be less than 120/80 mmHg, for resting systolic and diastolic blood pressure respectively. Non-pharmacological intervention in the form of regular aerobic exercise has been recommended for the prevention of high blood pressure as well as the lowering of blood pressure among those individuals, with elevated level¹. Physical inactivity is associated with increased risk of development of both hypertension and coronary artery disease (CAD)²⁻⁴. No studies yet have assessed the effect of aerobic exercise for lowering the blood pressure in patients with essential hypertension in our indigenous population. Thus, the purpose of this study was to measure the influence of brisk walking on stage I or II essential hypertension.

Patients and Methods

Patients with primary hypertension, stages I or II, who had not been previously treated with antihypertensive drugs, were recruited from Nawabshah and studied during 1999-2000. The design

and intention of the study was thoroughly explained and informed consent was obtained from all study participants. The subjects underwent a standardized baseline assessment. All subjects were free of overt disease, as assessed by medical history, physical examination and fasting blood chemistries. Subjects were excluded from the study if they presented a history or evidence of hepatic, renal, or haematological disease; stroke, diabetes (fasting blood glucose >7.0 mmol/L)⁵, or body mass index of (BMI) >35 .

All the study participants had physician-diagnosed stage I or 2 hypertension (blood pressure $>140/90$ mmHg)¹; no subjects were taking antihypertensive medication and all subjects were nonsmokers and had not participated in a regular aerobic exercise programme for a minimum of 12 months. The duration of aerobic exercise was set at 50% of VO₂max using mean \pm SEM age was 39.8 ± 0.7 years, ranging from 34-48 years, initial blood pressure $143.2 \pm 1.4/91.8 \pm 1.0$ mm Hg, body weight 68.6 ± 0.6 Kg and body mass index was 23.1 ± 0.3 . To initiate the 12-week aerobic exercise training programme, the subjects underwent a supervised orientation, after which they exercised on their own. The aerobic exercise training protocol consisted of 30 minutes of brisk walking 3 to 5 times weekly. Exercise intensity was determined by Karvonen's formula⁶. Intensity of training was expressed as a percentage of maximum oxygen consumption, which was estimated using a cycle ergometry test. The subjects were asked to record their prescribed exercise duration on a daily basis.

The study patients were forbidden to take any medication and were also requested to maintain routine lifestyle habits with regard to diet intake throughout the study. Before the intervention period, blood pressure (BP) was obtained on 2 separate visits with an interval of 2 weeks, and the average values were used as the baseline value. In an attempt to examine the time course of BP reduction with exercise, BP was measured at least every 4 weeks thereafter; however, the data were analyzed and presented at baseline and week-12. Post-exercise training BP was defined as the mean of 2 measurements immediately after and within a week after the last bout of exercise. The different measurements were made to eliminate any contribution of the immediate (acute) effects of exercise. All blood pressure measurements were conducted during the morning hours.

Arterial blood pressure was measured using a standard mercury sphygmomanometer with appropriate cuff size, after subjects had rested in the sitting position for at least 5 minutes. Two readings were taken at 2 minutes interval, and the latter was used in statistical analysis. Systolic and diastolic blood pressures were read to the nearest 2 mmHg at Korotkoffs sounds phase I and V. Pulse pressure was calculated as systolic blood pressure minus diastolic blood pressure. Mean arterial pressure (MAP) was calculated, by the formula $\text{MAP} = \text{diastolic pressure} + \frac{1}{3} \text{ pulse pressure}$. Body weight was measured on a balance scale, while participants were without shoes and heavy outer garments. Height was measured in the standing position following weight measurement by a stadiometer. Body mass index (BMI) was calculated as $\text{BMI} = \frac{\text{weight (kg)}}{\text{height (m)}^2}$ at the end of a 12-week, minus values at the baseline; a negative and positive value implies a lowering or increasing of that value. Differences between means of parameters were tested for significance using the paired student's t test. A value of $P < 0.05$ was considered significant.

Results

Forty male patients initially took part in an exercise training programme. Of the 40 patients, 13 (32.5%) were withdrawn early in the study. The reasons of withdrawal were 8 subjects were lost to follow-up, refusal in 2, and intercurrent illness in 3 and did not complete the study and remaining 27 patients, who remained compliant with the exercise programme were included in the final evaluation. However, the rate of compliance to the study protocol was 67.5%. Baseline measurements and changes from baseline to the end of training for study parameters are presented in Table.

Table. Characteristics of study patients before and after the period of physical exercise training [n=27, age 39.8± 0.7 years, sex (M/F) 27/0].

	Before	After	Difference
Body Weight (kg)	68.6 ± 0.6	67.5 ± 0.5	-1.1
Body mass index (kg/m ²)	23.1 ± 0.3	23.0 ± 0.4	-0.1
Systolic blood pressure (mmHg)	143.2 ± 1.4	137.5 ± 1.2	-5.7
Diastolic blood pressure (mmHg)	91.8 ± 1.0	90.4 ± 0.9	-1.4
Pulse pressure (mmHg)	50.9 ± 0.8	47.2 ± 1.0	-3.7
Mean arterial pressure (mmHg)	109.4 ± 1.1	106.0 ± 0.9	-3.4
VO ₂ max (mL/kg/min)	33.2 ± 0.3	38.3 ± 0.2	+5.1

The changes in anthropometric variables did not differ significantly with brisk walking at the end of aerobic exercise intervention. No adverse clinical events or exercise-related complications were experienced.

insignificant p value. shows significant change at p value <0.01. and shows p value <0.05 at the end of 12-week physical training.

Discussion

Multiple studies have shown that regular aerobic exercise can lower the arterial blood pressure in both hypertensive and healthy subjects^{2,7-9}. Several non- pharmacological interventions, including exercise, are recommended, in primary prevention of hypertension and other cardiovascular diseases, in which the pathogenetic role of endothelial dysfunction has been suggested¹⁰. Multiple exercise intervention studies have shown consistently that aerobic exercise reduces systolic blood pressure¹¹ and diastolic blood pressure in patients with essential hypertension^{12,13}.

The mechanisms responsible for these hypotensive effects remain clearly unknown¹⁴ and warrant further studies. Although, exercise acutely raises blood pressure, there is growing evidence that repeated

physical exertion reduces blood pressure in both normotensive and hypertensive subjects¹⁵. Exercise might exert its hypotensive effects directly through hemodynamic mechanisms. Since mean arterial pressure (MAP) is equal to the cardiac output (CO) and total peripheral resistance (TPR), a lowering of resting blood pressure must result from a decrease in CO, TPR, or both, as we found significant reduction ($p < 0.01$) in MAP at the end of study.

There is now increasing evidence that high pulse pressure, which is an indicator of large artery stiffness, is an independent risk factor for cardiovascular mortality, especially from coronary artery disease. The study of Benetos et al¹⁶, in a large French population, showed that in male subjects aged 40 to 60 years, increased pulse pressure was a strong predictor of cardiovascular mortality, especially coronary mortality. In this recent report, cardiovascular and coronary death rates were similar in the group of normotensive men with a pulse pressure > 50 mmHg and in the group of hypertensive men with a pulse pressure < 45 mmHg. No association between cardiovascular mortality and pulse pressure was observed in either normotensive or hypertensive women. In the present study, we found significant decrease in pulse pressure ($p < 0.01$) in males and as no women were studied; the results could not be generalized. However, pulse pressure could aid in evaluating cardiovascular risk.

It is known that many of the cardiovascular complications associated with sedentary aging, such as hypertension, coronary artery disease and thrombosis are pathogenetically linked to endothelial dysfunction^{17,18}. In men, endothelium-dependent vasodilation has been shown to decline progressively with advancing age^{18,19}, starting as early as 20 years. The reduction of blood pressure in our study, although not to the acceptable range, indirectly indicates vasorelaxation, as regular exercise can restore the loss of endothelium-dependent vasodilation in previously sedentary middle aged and older men²⁰. We did not assess these parameters directly, however, our findings indirectly reflect these underlying changes.

DeSouza et al²⁰, reported that 3 months of regular aerobic exercise (primarily walking) resulted in a 30% increase in endothelium-dependent vasodilation in previously sedentary middle aged and older men. Moreover, this improvement occurred without concomitant significant changes in body mass and adiposity suggesting a primary effect of aerobic exercise on endothelial cell function. Higashi et al¹⁰, reported that long-term physical exercise improves endothelium-dependent vasorelaxation through an increase in the release of nitric oxide in normotensive as well as hypertensive subjects. These apparently beneficial changes in vasodilator capacity and vascular structure may be produced with intensity and frequency of exercise²¹.

Our findings demonstrate, that the fall in blood pressure is not associated with reduction in body weight. Our subjects showed little change in body weight, whereas the change in body mass index did not reach statistical significance, suggesting that the observed changes were not due to dietary factors and also these subjects were not treated for hypertension. Thus, it seems reasonable to attribute the observed changes with the type of physical activity (brisk walking) for 12-weeks. Most clinical studies on exercise therapy for hypertension failed to show the effects of weight loss^{22,23}. However, other studies have also shown that exercise lowers blood pressure of overweight hypertensives without producing either weight loss or a reduction in body fat^{24,25}. The results of our study compare with other publications^{26,27}. These findings show that, at least in the short-term, exercise-training programme (brisk walking) had no effect on mean body mass index.

VO₂max (a measure of aerobic fitness) significantly increased in the present study ($P < 0.05$) and in others ($P < 0.01$)²⁸. Ishikawa et al⁶, have also reported increased VO₂max, especially in hypertensive subjects. This would suggest that the increase in VO₂max and improvement in hypertension is the response of physical exercise training. The result shows that 30-minute of brisk walking at 50% of VO₂max, 3-5 times weekly is sufficient for most of the beneficial hemodynamic effects to occur.

We do not know that whether blood pressure would subsequently have improved, if the period of daily

exercise had continued for more than 12-weeks. However, our findings suggest that the optimum amount of regular exercise may be somewhere between 3 and 5 times per week and that there should be some rest days.

In conclusion, these findings provide a preliminary indication that brisk walking, a low cost and acceptable form of physical activity in patients with essential hypertension could be incorporated into strategies for improving cardiorespiratory fitness and has effects that should reduce the possibility of subsequent development of cardiovascular disease.

Acknowledgments

We would like to thank all the subjects who participated in the study.

References

1. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation and treatment of High Blood Pressure Arch. Intern. Med.. 1997; 157: 2413-46.
2. Paffenbarger RS, Hildebrand RI, Wing AL, et al. Physical activity, all cause mortality, and longevity of college alumni. N. Engl. J Med., 1986;314: 605-13.
3. The American college of sports medicine: Resource manual for guidelines for exercise testing and prescription. Baltimore, Williams and Wilkins edition. 1993
4. Petrie JC, O'Brien ET, Little WA, et al. British hypertension society recommendations on blood pressure measurements. Br. Med. j., 1986; 293: 611-5.
5. American Diabetes Association. Clinical Practice Recommendations 1998. Diabetes Care. 1999; 22: S 20-S23.
6. Ishikawa K, Ohta T, Zhang J, et al. Influence of age and gender on exercise training-induced blood pressure reduction in systemic hypertension. Am. J. Cardiol., 1999; 84: 192-196.
7. Mersev DJ. Health benefit of aerobic exercise. Postgrad. Med., 1991; 90: 1103-7
8. Rogers MW, Probst MM, Comber JJ, et al. Differential effects of exercise training intensity on blood pressure amid cardiovascular responses to stress in border line hypertensive humans. J. Hypertens., 1996; 14: 1369-75.
9. Bond V, Hatfield B, Davis GC, et al, Aerobic exercise attenuates blood pressure reactivity in normotensive young adults. Eihn. Dis., 1999;9: 104.10.
10. Higashi Y, Sasaki S, Kurisu S, et al. Regular aerobic exercise augments endothelium-dependent vascular relaxation in normotensive as well as hypertensive subjects: Role of endothelium-derived nitric oxide. Circulation., 1999; 100: 1194-202.
11. Bertovic DA, Waddell IK, Gatzka CD, et al. Muscular strength training is associated with low arterial compliance and high pulse pressure. Hypertension., 1999; 33: 1385-91.
12. Arida RM, Naffah-Mazzacoratti-M-da-G, Soares J, et al. Monoamine responses to acute and chronic aerobic exercise in nonnotensive and hypertensive subjects. Rev Paul Med 1998; 116: 1618-24.
13. Kokkinos PF, Papademetriou V. Exercise and hypertension. Coron Artery Dis., 2000; 11. 99-102.
14. Orabach P, Lowenthal DT. Evaluation and treatment of hypertension in active individuals, Med. Sci. Sports Exerc., 1998; 10; S 354-66.
15. Bjornorp P. Hypertension and exercise, Hypertension., 1982; 4 JSuppl. III]: 6-9.
16. Benetos A, Rudnichi A, Safar M, et al. Pulse pressure and cardiovascular mortality in nonnotensive and hypertensive subjects. Hypertension..1998; 32:560-4.
17. Vita JA, Treasure CB, Nabel EG, et al Coronary vasomotor response to acetylcholine relates to risk factors for coronary artery disease. Circulation., 1990; 81: 491-497.

18. Taddei S, Virdis A, Mattei P, et al. Hypertension causes premature aging of endothelial function in humans. *Hypertension*, 1997; 29: 736-743.
19. Taddei S, Virdis A, Mattem P, et al. Aging and endothelial function in normotensive subjects and patients with essential hypertension. *Hypertension*, 1995; 91: 1981-87.
20. DeSouza CA, Shapiro LF, Clevenger CM, et al. Regular aerobic exercise prevents and restores age-related declines in endothelium-dependent vasodilation in healthy men. *Circulation*, 2000; 102: 1351-57.
21. Tanaka H, Reiling MJ, Seals DR. Regular walking increases peak limbic vasodilator capacity of older individuals. *J. Hypertens.* 1998; 16: 423-8.
22. Seals DR, Hagberg JM. The effect of exercise training on human hypertension A review: *Med. Sci. Sports Exerc.*, 1984; 16: 207-215.
23. Lipton CM. Exercise training and hypertension. *Exerc Sport Sci. Rev.*, 1984; 2, 245-306.
24. Krotkiewski M, Mandroukas K, Stostrom L, et al. effects of long-term physical training on body fat, metabolism and blood pressure in obesity. *Metabolism*, 1979, 28: 650-658.
25. deChamplain J, Cousineau D, Lapointe L, et al. Sympathetic abnormalities in human hypertension. *Clin. Exp. Hypertens*, 1981, 3: 417-438.
26. Moreira WD, Fuchs FD, Appel Li. The effects of two aerobic training intensities on ambulatory blood pressure in hypertensive patients. *J. Clin Epidemiol*, 1999; 52: 637-42.
27. Young DR, Appel Li, Jee S, et al. The effects of aerobic exercise and Tai Chi on blood pressure in older people: Results of a randomized trial. *J. Am. Geriatr Soc.*, 1999; 47: 277-84.
28. Dengel DR, Hagberg JM, Pratley RE, et al. Improvements in blood pressure, glucose metabolism, and lipoprotein lipids after aerobic exercise plus weight loss in obese, hypertensive middle-aged men. *Metabolism*, 1998; 47: 1075, 82.