Health Education and Public Health
2019; 2(3): 191 – 194 . doi: 10.31488 /heph.122

Research article

Intermittent, Moderate-Intensity Aerobic Exercise For Eight Weeks And High-Intensity Aerobic Exercise For Two Weeks Both Reduce Arterial Stiffness: Evaluation By Measurement Of Stiffness Parameter Using Ultrasonic Echo Tracking

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Received: April 15, 2019; Accepted: May 20, 2019; Published: May 23, 2019

Abstract

Background and purpose: Aerobic exercise has been reported to be associated with reduced arterial stiffness. We studied the effects of short-term, intermittent, moderate-intensity and high-intensity exercise training on arterial stiffness. Methods：A group of 25 young healthy volunteers (Group A) and a group of 15 young healthy volunteers (Group B) were recruited for 8 weeks and 6 weeks intermittent, moderate-intensity exercise training. Another group of 26 young healthy volunteers (Group C) were recruited for 2 weeks high-intensity exercise training. Using ultrasonic diagnostic equipment we measured changes in arterial stiffness of the carotid artery before and after each exercise training. Results: In Group A, after the 8 weeks exercise training, arterial stiffness decreased significantly. In Group B, after the 6 weeks training, all the parameters did not change significantly. In Group C, after two weeks exercise training, arterial stiffness decreased significantly. Conclusions: Eight weeks of moderate-intensity exercise training was required to decrease carotid arterial stiffness significantly. On the other hand, only 2 weeks of high-intensity exercise training reduced carotid arterial stiffness significantly.

Keywords: Arterial stiffness, tread mill exercise, bicycle ergometer exercise, echo tracking

Introduction

It has been shown that increased arterial stiffness is a significant risk factor for future cardiovascular disease independent of well-known cardiovascular risk factors. Arterial stiffness increases with age inevitably, and is not improved by medications such as vasodilator and diuretics. Many studies have been conducted on the association between exercise training and changes in arterial stiffness in different subject groups, for example, young healthy, hypertensive, hyperlipidemic, diabetic, and cardiovascular disease groups [1-5]. However, the intensity, duration, and frequency of aerobic exercise required to change arterial stiffness have not been established. In addition, most reports base their conclusions on changes in pulse wave velocity, which is an indirect index of arterial stiffness. We studied the effects of moderate-intensity and high-intensity exercise training on arterial stiffness based on measurements of the stiffness parameter (β) which is a direct index of arterial stiffness calculated from arterial diameter change due to arterial pressure change.

Methods

Evaluation of arterial stiffness

To evaluate arterial stiffness, we used stiffness parameter β. β has the advantage that it does not depend on pressure. β is defined as

$$\beta = \ln (Ps/Pd)/[(Ds-Dd)/Dd],$$

where ln means a natural logarithm. Ps and Pd are systolic and diastolic pressure (mmHg) and Ds and Dd are the maximum and minimum diameters (mm) of the carotid artery in a cardiac cycle, respectively [6, 7]. β is obtained noninvasively with an ultrasonic system (α10, Hitach-Aloka, Tokyo, Japan) which measures arterial diameter-change waveform by echo tracking [8] (Figure 1).

Subjects

Three groups of subjects were recruited: Group A, 25 young healthy volunteers (18 men, mean age 20.8 ± 1.0 years) (Table 1) ; Group B, 15 young healthy volunteers (5 men, mean age 20.7 ± 0.5 years); Group C, 26 young healthy volunteers (20

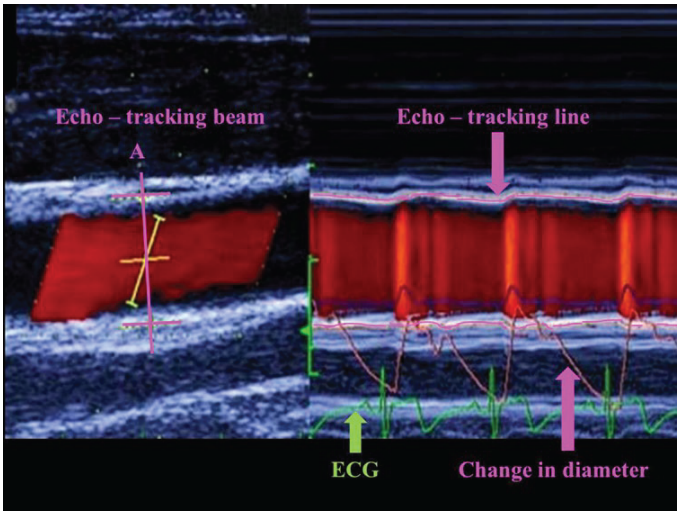

Figure 1. Measurement of diameter-change waveform. Left: long-axis view of the common carotid artery and ultrasound beam steering. By setting the tracking positions displayed as small bars on the echo-tracking beam (line A) to arterial walls, echo tracking automatically starts. Right: the diameter-change waveform, which is calculated by subtracting the distance to the near wall from that to the far wall, is displayed on the M-mode view (from (5)).

Table 1. Changes in hemodynamic and carotid arterial parameters before and after 8 weeks moderate-intensity exercise.

Exercise period		8 weeks	
	before	after	P value
HR [bpm]	67 ± 12	65 ± 11	n.s.
Ps [mmHg]	113 ± 8	111 ± 9	n.s.
Pd[mmHg]	61 ± 6	61 ± 8	n.s.
Puls pressure [mmHg]	51 ± 9	50 ± 11	n.s.
maxU [m/s]	0.84 ± 0.16	0.89 ± 0.15	P<0.05
Ds [mm]	7.38 ± 0.49	7.20 ± 0.60	n.s
Dd [mm]	6.78 ± 0.40	6.56 ± 0.47	n.s
(Ds – Dd) /Dd	0.09 ± 0.02	0.10 ± 0.02	P <0.01
SV [ml]	9.73 ± 2.75	9.76 ± 2.68	n.s.
Output [ml/min]	624 ± 109	616 ± 131	n.s.

HR: heart rate; Ps: systolic pressure; Pd: diastolic pressure; max: maximum velocity; Ds: systolic diameter; Dd: diastolic diameter; SV: carotid srterial stroke volume; output: carotid arterial flow volume

men, mean age 20.7 ± 0.5 years) (Table 2). All subjects provided informed consent and the ethics committee of Himeji Dokkyo University approved the study protocol.

Measurement protocol

Preliminary measurements before the commencement of the exercise

The preliminary test for Group A and Group B consisted of taking of health histories, degree of physical activity, measurements of β at rest, and measurements of peak aerobic capacity (peak VO2). For the measurements of β, the subjects lay down in the supine position and rested for 10 min before the first recording was made. The location to be measured was the common carotid artery at about 2 cm proximal to the carotid

Table 2. Changes in hemodynamic and carotid arterial parameters before and after 2 weeks high-intensity exercise.

Exercise period		8 weeks	
	before	after	P value
HR [bpm]	71 ± 11	65 ± 10	P <0.01
Ps [mmHg]	111 ± 8	101 ± 9	n.s.
Pd[mmHg]	60 ± 7	59 ± 8	n.s.
Puls pressure [mmHg]	51 ± 10	51± 9	n.s.
maxU [m/s]	0.86 ± 0.16	0.85 ± 0.16	n.s.
Ds [mm]	7.32 ± 0.53	7.15 ± 0.45	P<0.05
Dd [mm	6.71 ± 0.45	6.50± 0.37	P<0.01
(Ds – Dd) /Dd	0.09 ± 0.03	0.10 ± 0.02	P<0.05
SV [ml]	8.66 ± 2.05	8.92 ± 1.41	n.s.
Output [ml/min]	601 ± 125	570 ± 82	n.s.

HR: heart rate; Ps: systolic pressure; Pd: diastolic pressure; maxU: maximum velocity; Ds: systolic diameter; Dd: diastolic diameter; SV: carotid srterial stroke volume; output: carotid arterial flow volume

bulb. We used scanning in the long axis view, and obtained a B-mode image of a longitudinal section of the artery. The echo-tracking system tracked the vessel wall movements to produce displacement waveforms of the anterior and posterior artery walls. This gave the maximum and minimum diameters (Figure 1). After the measurements of β, peakVO2 was assessed during a graded exercise test on an electrically braked cycle ergometer. The ergometer test started with a warm-up of 2-min exercise at 20 W workrate, and then the workrate was increased by 20 W/min until limitation. The limitation criteria for the establishment of peak VO2 included a plateau in the oxygen consumption with increasing workrate, attainment of pulsation to 158 bpm and attainment of fatigue to the maximum, or impossibility of continuing exercise.

The protocol of the preliminary measurements of β at rest in Group C is the same as Group A and Group B. For the measurements of peak VO2, treadmill test was performed according to Brace protocol. The limitation criteria for the establishment of peak VO2 included a plateau in the oxygen consumption with increasing workrate, attainment of pulsation to 180 bpm and attainment of fatigue to the maximum, or impossibility of continuing exercise. In addition, we fixed electrocardiographic limits at an ST elevation of 0.1 mV and an ST depression of 0.2 mV, respectively.

Exercise test

The exercise sessions for Group A and Group B were 30 min in duration on the bicycle ergometer. Subjects were asked to make an effort to maintain the heart rate during the session at around 60% of that at the peak VO2. The sessions were conducted two or three times per week over a period of 8 weeks for Group A and 6 weeks for Group B.

For Group C, the subjects were asked to perform 30 min treadmill exercise and to make an effort to maintain the heart rate during the session at around 70% of that at the peak VO2. The sessions were conducted five times per week over a period of two weeks.

Statistical analysis

The obtained data are presented as mean ± SD. A paired t-test was used to evaluate the changes before and after exercise training. Furthermore, a two-way analysis of variance (ANOVA) was used for comparing the changes before and after 6 weeks and 8 weeks exercise training. A value of $p < 0.05$ was considered to be statistically significant.

Results

After the moderate 8 weeks exercise training (Group A), systolic pressure (Ps), diastolic pressure (Pd), pulse pressure, heart rate (HR), systolic arterial diameter (Ds) and diastolic arterial diameter (Dd) did not change significantly. However, the pulsatile change in diameter ((Ds-Dd) / Dd) increased significantly, and ß decreased significantly (Figure 2). The maximum blood flow velocity in the carotid artery was also significantly increased (Table 1). After the moderate 6 weeks exercise training (Group B), on the other hand, all the parameters did not change significantly. After the high-intensity 2 weeks exercise training (Group C), Ps, Pd, and pulse pressure did not change significantly. However, HR decreased significantly, and Ds and Dd decreased and (Ds – Dd)/Dd increased significantly (Table 2). Accordingly, β decreased significantly (Figure. 3).

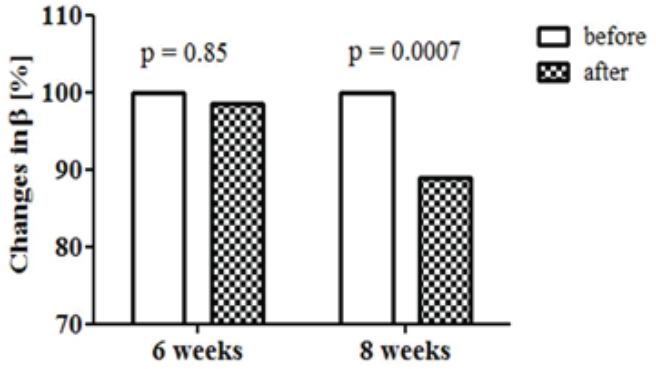

Figure. 2. Changes in β before and after 6 weeks and 8 weeks of moderate-intensity exercise training. Data are displayed as percent change, normalized to the value before the training.

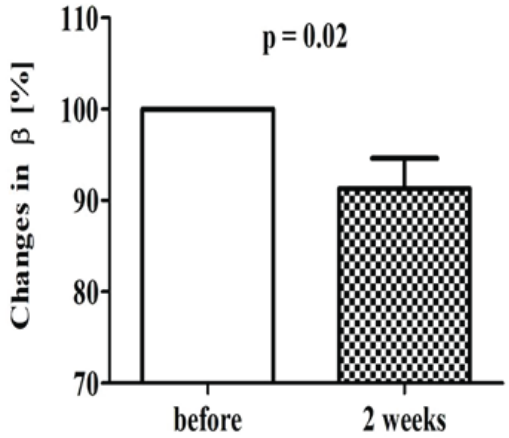

Figure 3. Changes in β before and after 2 weeks of high-intensity exercise training. Data are displayed as percent change, normalized to the value before the training.

Discussion

Two types of exercise (8 weeks moderate-intensity ergometer exercise and 2 weeks high-intensity treadmill exercise) both decreased arterial stiffness β. The mechanisms underlying the effects of aerobic exercise on arterial stiffness are largely unknown. Nevertheless, it has been suggested that the ability of exercise to change arterial stiffness may reflect vascular structural remodeling associated with increased nitric oxide bioactivity via shear-stress stimulation of endothelial nitric oxide (NO) synthase [1, 9, 10]. Indeed, the maximum velocity in the carotid artery was increased significantly after 8 weeks exercise, which caused an increase in shear stress on the luminal surface of the endothelium. However, the vasodilator effects of NO does not seem to be the cause of the decrease in arterial stiffness, since Ds and Dd did not change significantly after 8 weeks exercise. On the contrary, it has been reported that administration of nitroglycerin increases arterial diameter and arterial stiffness as well [11, 12]. In our 2 weeks exercise, though Ds and Dd decreased significantly, β also decreased significantly. Superficially, these results seem paradoxical. However, we have to consider which layer of the arterial wall withstands the internal blood pressure. When Ds and Dd are decreased, the tonus of the smooth muscle in the arterial wall is considered to be increased. When the tonus of the smooth muscle is increased, the internal pressure is withstood by circumferential tension of the smooth muscle layer in the arterial wall. The smooth muscle is more distensible compared with collagen fiber in the outer layer. Therefore, arteries withstanding luminal pressure mainly by smooth muscles have lower stiffness. However, the role of the smooth muscle is a matter of continuing debate.

Limitations

This study only enrolled young healthy subjects. Furthermore, we could not show any direct evidence as to the cellular and molecular mechanisms responsible for changes in arterial stiffness caused by exercise training, since we had no methods of physically accessing human arteries in situ and experimentally manipulating potential signaling pathways.

Conclusions

In healthy young subjects, 8 weeks of intermittent, moderate-intensity exercise training (HR 60% of that at the peak VO2, two or three times per week) with cycle ergometer improved (reduced) β, which is an index of arterial stiffness. However, 6 weeks of the same intensity exercise did not change β. On the other hand, 2 weeks of high-intensity exercise (HR 70% of that at the peak VO2, five times per week) with treadmill reduced β significantly.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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To cite this article: Tanaka M, Sugawara M, Niki K, et al. Intermittent, Moderate-Intensity Aerobic Exercise For Eight Weeks And High-Intensity Aerobic Exercise For Two Weeks Both Reduce Arterial Stiffness: Evaluation By Measurement Of Stiffness Parameter Using Ultrasonic Echo Tracking. Health Educ Public Health. 2019: 2:3.