Effect of Low-Intensity Aerobic Exercise Training on Arterial Compliance in Postmenopausal Women

Jun SUGAWARA, Hironobu INOUE*, Koichiro HAYASHI, Takashi YOKOI, and Ichiro KONO*

Regular aerobic exercise training attenuates age-related reduction in central arterial compliance, an independent risk factor of cardiovascular diseases. We tested the hypothesis that even low-intensity exercise training could increase central arterial compliance in postmenopausal women. Using B-mode ultrasound, we studied the central arterial compliance of 15 postmenopausal females (age: 52–66 years) before and after a 12-week aerobic exercise intervention. Subjects performed aerobic exercise training of the same energy expenditure (cycle exercise, total 900 kcal/week, 3–5 sessions/week) at two different exercise intensities: 7 trained at low intensity (40% heart rate reserve: L-TR) and 8 trained at moderate intensity (70% heart rate reserve: M-TR). Arterial compliance increased after exercise training in the L-TR group (0.70 ± 0.32 vs. 1.06 ± 0.55 mm²/mmHg × 10⁻¹, p < 0.05) and in the M-TR group (0.82 ± 0.37 vs. 1.14 ± 0.39 mm²/mmHg × 10⁻¹, p < 0.05). There was no significant difference in increases of arterial compliance in either group (L-TR: 0.35 ± 0.38 vs. M-TR: 0.32 ± 0.33 mm²/mmHg × 10⁻¹). These results suggest that the improvement of central arterial compliance by aerobic exercise training might not be influenced by the intensity of exercise training if the energy expenditure of the training is the same. Accordingly, even low-intensity exercise training may have the effect of improving central arterial compliance. (Hypertens Res 2004; 27: 897–901)

Key Words: arterial stiffness, aging, ultrasound

Introduction

The large artery in the cardiothoracic region has rich elasticity, and performs two functions, i.e., as a low-resistance conduit and as a cushion (or buffer) of flow pulsations at its input (1). The latter function, assessed as arterial compliance, has physiological and pathophysiological significance (2, 3). Arterial compliance decreases with ageing in sedentary people (2, 4–6). The reduction in central arterial compliance results in progressive age-related increase in systolic blood pressure, which raises left ventricular afterload, and decrease in diastolic blood pressure, which alters coronary perfusion and elevates pulse pressure (1). The elevations in systolic and pulse pressure are associated with the development of cardiovascular disease (2, 3). Berry et al. (7) indicated that stiffer large arteries likely connect to the greater prevalence of systolic hypertension in elderly women and may partly explain the acceleration in postmenopausal cerebrovascular and cardiac complications.

Currently, it is recognized that regular physical activity can be a primary preventive measure for hypertension (8), cardiovascular disease and stroke (9, 10). Several exercise intervention studies have indicated that regular aerobic exercise training improves arterial compliance in healthy young (11) and elderly (5, 6, 12) people, and patients with congestive heart failure (13). The exercise-induced increase in central arterial compliance could contribute to the prevention of the diseases mentioned above. However, in most of those studies, subjects performed at approximately 50–70% of...
maximal oxygen consumption or heart rate (HR) reserve. Therefore, it is unclear whether relative low-intensity exercise training could increase central arterial compliance. We thus tested the hypothesis that even low-intensity exercise training could increase central arterial compliance in postmenopausal women. In order to address this issue effectively, we compared changes in carotid arterial compliance with the 12-week aerobic exercise training in postmenopausal women who trained 900 kcal per week at different intensities (40% and 70% heart rate reserve).

Methods

Subjects

Fifteen postmenopausal sedentary females, including 4 who had hyperlipidemia (total cholesterol plasma concentrations > 240 mg/dl), were studied before and after 12 weeks of aerobic exercise training. All subjects were normotensives and did not smoke, take medications, or have significant intima-media thickening, plaque formation, and/or other characteristics of atherosclerosis. None of the subjects had taken hormone replacement therapy. All potential risks and procedures of the present study were explained, and subjects gave their written informed consent. This study was reviewed and approved by the Human Research Committee (National Institute of Advanced Industrial Science and Technology).

Exercise Intervention

The subjects underwent exercise training under supervised conditions. They performed aerobic exercise training (3–5 sessions/week, 12 weeks) of the same energy expenditure (see below) at two different exercise intensities: a low-intensity training (40% HR reserve: L-TR) or moderate-intensity training (70% HR reserve: M-TR). Each subject selected the training intensity by themselves before the start of the training period. Seven females, including all subjects with hyperlipidemia, selected the low-intensity training and the others selected the moderate-intensity training. The target HR was 128 ± 5 bpm in the M-TR group and 103 ± 5 bpm in the L-TR group. Using an electric-braked ergometer, subjects trained at the intensity corresponding to each target HR. During the training, the work load of the ergometer was automatically modulated so as to maintain the HR of the subject at the target level. Subjects performed the training corresponding to 180–300 kcal of the energy expenditure. The weekly total energy expenditure of training was set at 900 kcal.

Measurements

All the studies were done after a fast of at least 4 h. The subjects abstained from alcohol, caffeine, and intense exercise for at least 24 h before the experiments. Fasting blood samples were obtained. Serum and EDTA plasma samples were stored at -4°C and analyzed at the end of the study. Fasting plasma concentrations of total cholesterol and low-density lipoprotein (LDL) cholesterol were evaluated by the enzymatic method. Fasting plasma concentrations of high-density lipoprotein (HDL) cholesterol were evaluated by the synthetic polymer method. After the blood sampling and the body mass measurement, arterial compliance and blood pressure were measured under quiet supine resting conditions after at least 15 min of resting. Properties of the common carotid artery were measured by an ultrasound system with a high-resolution (10 MHz) linear transducer (SonoSite180PLUS; SonoSite Inc., Bothell, USA). The transducer was placed at 1 to 2 cm proximal to the carotid bulb, with an approximately 90° angle to the vessel so that the near and far wall interfaces were clearly discernible (6, 12), and 10–20 pulsations of the common carotid artery (B-mode longitudinal image) were recorded on digital videotape. All scans and image analyses were performed by the same investigator who was blinded to the group assignments of the subjects. Ultrasound images were analyzed by image-analysis software (Scion Image; Scion Corporation, Frederick, USA). Carotid arterial lumen diameter was determined as the distance between the vessel far-wall boundary, corresponding to the interface between the lumen and intima, and a near-wall boundary, corresponding to the interface of the adventitia and media at the minimum diastolic relaxation and at the maximal systolic expansion of the vessel. Intima-media thickness (IMT) was defined as the distance from the leading edge of the lumen-intima interface to the leading edge of the media-adventitia interface at the minimum diastolic relaxation of the vessel (14). Arterial lumen diameters at the minimum diastolic relaxation and at the maximal systolic expansion of the vessel and IMT were measured at 3 points per frame and averaged, respectively. Moreover, 3–5 continuous measurements of each parameter were averaged and statistically analyzed. To adjust for individual differences in lumen diameter, the IMT/diastolic lumen diameter ratio was also calculated. The arterial compliance and distensibility coefficient were obtained according to following equations (6, 15):

\[
\text{Arterial compliance} = \frac{(\text{CSA}_t - \text{CSA}_a)}{\Delta P}
\]

\[
\text{Distensibility coefficient} = \frac{[\text{CSA}_t - \text{CSA}_a]}{\text{CSA}_a/\Delta P},
\]

where CSA\(_a\) and CSA\(_t\) are cross-sectional areas at the maximal systolic expansion and at the minimum diastolic relaxation of the carotid artery, respectively, and \(\Delta P\) is the carotid arterial pulse pressure.

Carotid arterial pulse pressure was recorded with the appplanation tonometry probe (TU-100; COLIN, Komaki, Japan) at the right common carotid artery. Blood pressure was measured at the upper right arm with a semiautomated oscillometric device (form PWV/ABI; COLIN). Recordings were made in duplicate with subjects in the supine position.
Table 1. Physiological Characteristics

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<thead>
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<th>Low-intensity exercise training group</th>
<th>Moderate-intensity exercise training group</th>
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<tbody>
<tr>
<td></td>
<td>Before training</td>
<td>After training</td>
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<tr>
<td>Age (years)</td>
<td>58 ± 4</td>
<td>59 ± 6</td>
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<tr>
<td>Height (cm)</td>
<td>154.8 ± 3.3</td>
<td>155.1 ± 2.9</td>
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<tr>
<td>Body mass (kg)</td>
<td>63 ± 9</td>
<td>62 ± 8</td>
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<tr>
<td>Heart rate (bpm)</td>
<td>123 ± 9</td>
<td>123 ± 7</td>
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<tr>
<td>Systolic BP (mmHg)</td>
<td>79 ± 7</td>
<td>80 ± 6</td>
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<tr>
<td>Diastolic BP (mmHg)</td>
<td>45 ± 4</td>
<td>43 ± 4</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>236 ± 18</td>
<td>218 ± 22</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>61 ± 14</td>
<td>57 ± 14</td>
</tr>
<tr>
<td>HDL cholesterol (nU/ml)</td>
<td>142 ± 15</td>
<td>127 ± 23*</td>
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Data are mean ± SD. All data are reported as the mean ± SD. Statistical significance was set at p < 0.05 for all comparisons.

Table 2. Carotid Artery Characteristics

<table>
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<tr>
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<th>Low-intensity exercise training group</th>
<th>Moderate-intensity exercise training group</th>
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<tbody>
<tr>
<td></td>
<td>Before training</td>
<td>After training</td>
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<tr>
<td>IMT (mm)</td>
<td>0.60 ± 0.07</td>
<td>0.61 ± 0.05</td>
</tr>
<tr>
<td>IMT/lumen diameter (ratio)</td>
<td>0.10 ± 0.01</td>
<td>0.10 ± 0.01</td>
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<tr>
<td>Diastolic lumen diameter (mm)</td>
<td>6.2 ± 0.5</td>
<td>6.3 ± 0.6</td>
</tr>
<tr>
<td>Carotid artery systolic pressure (mmHg)</td>
<td>114 ± 9</td>
<td>114 ± 6</td>
</tr>
<tr>
<td>Carotid artery pulse pressure (mmHg)</td>
<td>35 ± 4</td>
<td>34 ± 3</td>
</tr>
<tr>
<td>Arterial compliance (mm²/mmHg</td>
<td>0.70 ± 0.32</td>
<td>1.06 ± 0.55*</td>
</tr>
<tr>
<td>Distensibility coefficient (10⁻³/kPa)</td>
<td>2.3 ± 0.9</td>
<td>3.4 ± 1.8*</td>
</tr>
</tbody>
</table>

Data are mean ± SD. IMT, intima-media thickness. * p < 0.05 vs. before training.

Statistical Analyses

Repeated-measures analysis of variance (ANOVA) was used to evaluate the effects of exercise intensity. In the case of a significant F value, a post hoc test using the Fischer’s PLSD method identified significant differences among mean values. Analysis of covariance (ANCOVA) was performed to remove influences of initial levels of arterial compliance from the training effects. All data are reported as the mean ± SD. Statistical significance was set at p < 0.05 for all comparisons.

Results

In response to aerobic exercise training, body mass significantly decreased in the M-TR group (p < 0.05, Table 1). Body mass in the L-TR group also decreased, but not significantly (p = 0.07). Resting HR, blood pressure and pulse pressure did not change significantly in either group (Table 1). After the exercise training period, the fasting plasma concentration of total cholesterol significantly decreased in the M-TR group (p < 0.05, Table 1), while that in the L-TR group tended to decrease (p = 0.09). The fasting plasma concentration of LDL cholesterol, but not HDL cholesterol, significantly decreased in both training groups (all p < 0.05).

Table 2 shows the properties of the carotid artery. There were no significant differences in any parameter between the two groups. Carotid arterial diastolic lumen diameter, IMT, IMT/lumen diameter, and systolic and pulse pressures did not change in either group. Carotid arterial compliance increased after the exercise training in the L-TR group and M-TR group (both p < 0.05). The distensibility coefficient of the carotid artery was also significantly increased after the training in the L-TR group and M-TR group (both p < 0.05). The initial level of arterial compliance and distensibility in the L-TR group were not significant but lower than those in the M-TR group. Therefore, these initial levels were taken into account when the training effects were examined. However, the increase in arterial compliance of the L-TR group (0.35 ± 0.38 mm²/mmHg 10⁻³) did not differ from that of the M-TR group (0.32 ± 0.33 mm²/mmHg 10⁻³). The increase in the distensibility coefficient in the L-TR group (1.1 ± 1.4 ± 10⁻³/kPa) did not differ from that in the M-TR group (1.1 ± 1.0 ± 10⁻³/kPa).
Discussion

The primary findings of the present study were as follows. First, arterial compliance estimated by two different indices improved after the low- and moderate-intensity training programs. Second, increases in these parameters were not different between the two groups. These results suggest that the improvement of central arterial compliance by aerobic exercise training might not be influenced by the intensity of exercise training if the exercise intensity ranges from low to moderate and the energy expenditure of the training is the same. In turn, even low-intensity exercise training may have the effect of increasing arterial compliance.

We first demonstrated that even low-intensity exercise training could improve central arterial compliance. In the present study, the duration of exercise training was markedly longer in the low-intensity training group than in the moderate-intensity training group, which might have contributed to the improvement of central arterial compliance in the low-intensity exercise training. These results may imply that the total energy expenditure is more important than the exercise intensity for the improvement of central arterial compliance. The recommendation from the Centers for Disease Control and Prevention (CDC) and the American College of Sports Medicine (ACSM) is that individuals should engage in 30 min or more of physical activity at a relative intensity of 40% to 60% of maximal oxygen consumption (or absolute intensity of 4 to 6 METs) on most (preferably all) days of the week (9, 16). The results of the present study provide the first evidence of an improvement of arterial compliance by an exercise regimen at the lowest limit of the recommendation by the CDC and ACSM. In Japan, it is recommended that exercise treatment consist of isotonic exercise at a relative intensity of 40% to 60% of maximal oxygen consumption for 30–40 min, 3–5 times a week (17). While it is not practical to measure the maximum oxygen consumption in all patients, a target of 110 bpm is used for patients aged 60 years or older, as estimated from the relation between HR and physical activity at 50% maximum oxygen consumption at various ages. The target HR in the L-TR group (103 ± 5 bpm) was lower than that. If the training intensity is relatively low, it may be easy to introduce it into an ordinary lifestyle and easy for an individual to keep up with training safely. Therefore, our findings have important clinical implications and the potential of affecting exercise prescriptions.

Arterial compliance is determined by structural and functional factors. The former include the intrinsic elastic property of the arterial wall, i.e., the composition of elastin and collagen and the calcium content of elastin, and the latter include the alteration of vasoconstrictor tone. In animal studies, regular physical exercise could attenuate the age-related increase in the arterial stiffness of old rat aorta, but not the age-related compositional changes (i.e., increased elastin content and decreased collagen content) in the arterial wall (18). In the present study, we could not observe apparent changes in IMT and IMT normalized by lumen diameter after exercise training in either exercise training group. These results were consistent with a previous study (14). Chronic exercise training increases the production of nitric oxide (19–22), an endothelium-derived vasodilator, and reduces the plasma concentration of endothelin-1 (23), a vasoconstrictor, in humans. With this information as background, we can speculate about substances which improve arterial compliance via a reduction in the vasoconstrictor tone. However, the present study design did not aim to determine the mechanism underlying the improvement of arterial compliance by exercise training.

There were several limitations to this study. First, we did not examine an “untrained” control group, which is the crucial problem among interventional studies generally. However, it has been well-established that moderate-intensity regular aerobic exercise improves the central arterial compliance (5, 6, 11–13). Therefore, the fact that the increase in central arterial compliance in the L-TR group was not significantly different from that in the M-TR group was important. The initial level of central arterial compliance in the L-TR group was not significant, but was lower than that in the M-TR group. This factor may affect the training-induced improvement of central arterial compliance in the L-TR group. However, there was no significant difference in the increases in arterial compliance between the two exercise training groups after the initial arterial compliance level was taken into account (ANCOVA). Accordingly, even low-intensity exercise training (corresponding to 40% HR reserve) may have the effect of improving central arterial compliance. The beneficial effect of low-intensity exercise training on arterial compliance has important clinical implications and the potential of improving exercise prescriptions. Secondly, all subjects with hyperlipidemia belonged to the L-TR group. We could not rule out the possibility that a bias on subject assignment affected the changes in arterial compliance. However, Saba et al. (24) demonstrated the absence of a significant relationship between arterial stiffness and hyperlipidemia. Miyagi et al. (25) indicated that there was no significant relation between hypercholesterolemia and pulse pressure, the latter of which has been associated with a lower arterial compliance. Tanaka et al. (6) observed an increase in carotid arterial compliance after 3 months of regular exercise training in previously sedentary middle-aged and older men, and this increase was not associated with an alteration of plasma cholesterol level. Finally, as we did not assess the change in aerobic capacity (i.e., maximal oxygen consumption) with the training program, it is unclear whether there was a difference in the effect of training on aerobic capacity between the two training groups.

In summary, 12-week aerobic exercise training at low intensity increased central arterial compliance in postmenopausal females to the same extent as moderate intensity training did. These results suggest that the improvement of central...
arterial compliance by aerobic exercise training might not be influenced by the intensity of exercise training if the energy expenditure of the training is the same.

References