Original Article

Effect of Acute and Long-Term Aerobic Exercise on Arterial Stiffness in the Elderly

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Arterial stiffness is an important factor for cardiovascular performance and a predictor of cardiovascular risk. We evaluated the effects of both acute and long-term aerobic exercise on arterial stiffness in community-dwelling healthy elderly subjects. In addition, we evaluated the relationship between the effects of long-term exercise and those of acute exercise. The study subjects were participants in the Shimanami Health Promoting Program study (J-SHIPP), which was designed to investigate factors relating to cardiovascular disease, dementia, and death (67±6 years). They performed mild-to-moderate aerobic exercise lasting for 30 min twice a week for 6 months. Arterial stiffness was assessed before and after the first 30-min acute exercise (n=99) and long-term 6-month aerobic training (n=40). The radial arterial augmentation index (AI) obtained from the radial pulse waveform by the tonometry method was used as a parameter of arterial stiffness. Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were significantly decreased after 30-min of aerobic exercise, however no significant change in AI was observed. On the other hand, there were significant decreases in AI (from 87 to 84%, p<0.01), SBP (from 136 to 129 mmHg, p<0.01), and DBP (from 75 to 70 mmHg, p<0.01) after the 6-month exercise period. Long-term exercise–induced changes in AI were significantly and inversely correlated with the pre-exercise AI (r=−0.40, p<0.01). In addition, AI changes after the 6-month exercise period were significantly related to those observed after first 30-min exercise (r=0.48, p<0.01). These findings indicate that apparently healthy and sedentary elderly subjects with higher AI may benefit from mild-to-moderate aerobic exercise to improve arterial stiffness. (*Hypertens Res* 2007; 30: 895–902)

***Key Words*: arterial stiffness, aerobic exercise, augmentation index, radial pressure waveform**

Introduction

The central arterial system serves as a conduit delivering blood to the organs and tissues, and acts as a "buffer" by distending during cardiac ejection to provide an optimal and continuous peripheral blood flow. Since the buffering function of large arteries is determined by the arterial wall mechanical properties, deteriorations in the arterial structure lead to reduced diastolic blood pressure and impairment of coronary perfusion (*1*). Epidemiological studies provide evidentiary support of a significant association between central arterial stiffness and coronary artery diseases, including myocardial infarction (*2–4*). Alterations in arterial structure and function also occur with aging in healthy subjects. The increase in central arterial stiffness with age has been thought to be associated with age-related increases in the morbidity of cardiovascular disease (*5–7*). In addition, in subjects with metabolic-atherogenic disorders, including hypertension, diabetes mellitus, and hypercholesterolemia, the stiffness of the central arteries is known to be more pronounced (*8–10*).

Since aerobic exercise increases vascular wall shear-stress

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via an increase in blood flow, aerobic exercise could acutely reduce arterial tone (*11*, *12*). Reductions in peripheral resistance rather than cardiac output are postulated to be one of the underlying mechanisms for post-exercise hypotension (*13*). However, there have been no studies evaluating the acute effects of aerobic exercise on arterial properties. On the other hand, repetitive aerobic exercise for a long period of time induces anti-atherogenic effects through numerous mechanisms, including improvement of lipid metabolism, insulin sensitivity, and blood pressure reduction (*14–16*). Furthermore, habitual aerobic exercise has been shown to reduce menopause-related structural changes in the carotid arteries (*17*, *18*). These findings suggest that acute and long-term aerobic exercise would have different effects on the vasculature. In addition, it is conceivable that the effects of acute exercise on arterial properties would be altered after a long period of aerobic training.

In the present study, we evaluated the effects of both short-term (30 min) and long-term (6 months) aerobic exercise on arterial stiffness in community-dwelling healthy and sedentary elderly subjects. In addition, we investigated whether the changes in arterial stiffness induced by a single aerobic exercise session could predict the effects of long-term exercise training.

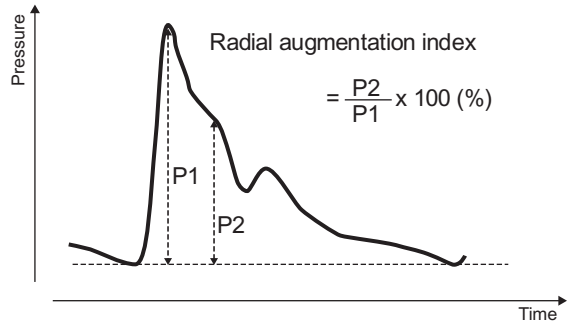

Fig. 1. *Schema of the radial arterial pressure contour. P_1, the height of the early systolic shoulder/peak pressure; P_2, the height of the late systolic shoulder/peak pressure. AI is defined as $(P_2/P_1) \times 100$.*

Methods

Study Subjects

The study subjects were participants in the Shimanami Health Promoting Program study (J-SHIPP), which is a longitudinal study evaluating factors relating to cardiovascular disease, dementia, and death (*19*, *20*). In brief, subjects aged 50 years or older and free from any history of medications for cardiovascular diseases were recruited from among participants of an exercise seminar held by the Matsuyama City Office (Matsuyama is the largest city in Ehime Prefecture, Japan). Among the participants, 99 subjects (mean age: 67±6 years; range: 53 to 86 years) who provide written informed consent and completed all measurements were enrolled in this study. All participants in the exercise seminar were from the general population of Matsuyama. None of them had performed regular physical exercise before the study. The study was approved by the ethical committee of Ehime University School of Medicine.

Exercise Intervention

Subjects were instructed to perform mild-to-moderate (heart rate [HR] of 110 to 120 bpm) aerobic exercise lasting for 30 min twice a week for 6 months under the instruction of an exercise therapist (*21*). This exercise training was carried out with 50 attendees at once, and mainly consisted of aerobic dancing and hopping. Each 30-min session began and ended with a 5-min warm up/cool down. Since participants were sedentary elderly, the intensity of the exercise was increased in a stepwise fashion throughout the training period. To maintain the intensity during the exercise session, supervision was provided by several therapists (approximately 1 therapist per 10 participants). None of the subjects had physical problems that interfered with their performance of the exercises.

Study Procedure

To evaluate the acute effects of aerobic exercise, brachial blood pressure (BP) and radial pressure waveform were measured before and after the exercise. At the pre-exercise measurement, brachial BP was measured twice on the right upper arm using an automatic cuff-oscillometric device (HEM-907; OMRON HEALTHCARE Co., Ltd., Kyoto, Japan) after an at least 5-min rest in a sitting position (*22*). If the difference in systolic BP (SBP) between the two measurements was larger than 10 mmHg, another BP measurement was performed. The final measured value was used for the analysis. Immediately after measuring brachial BP, the left radial pressure waveform was recorded twice for 40 s by the tonometry method using a non-invasive radial pulse wave monitor (HEM-9010AI; OMRON HEALTHCARE Co., Ltd.) (*22*). Post-exercise measurement was carried out with the same methods after the subjects had recovered to their baseline HR. The effects of long-term exercise were assessed by the pre-exercise measurements performed before and after 6 months of continuous training. We evaluated 40 subjects due to a limited opportunity to measure the long-term exercise effect. The average attendance rate was 76.3%. All measurements were carried out in air-conditioned rooms, and no one smoked during the procedure. Subjects were permitted to drink water freely.

Table 1. Background Characteristics of Subjects and the Effect of the 30-min Acute Aerobic Exercise

n (male/female)	99 (12/87)		
Age (years old)	67±6		
Body height (cm)	153±7		
Body weight (kg)	52±7		
Body mass index (kg/m^2)	22±3		
	Effect of 30 min acute exercise		
	Pre	Post	*p*
Systolic BP (mmHg)	135±19	131±20	0.0029
Diastolic BP (mmHg)	76±11	74±10	0.0068
Heart rate (bpm)	73±8	73±10	0.51
AI (%)	86±11	85±10	0.69
AI@75 (%)	85±9	84±8	0.45

Values are mean±SD. BP, blood pressure; AI, augmentation index; AI@75, AI corrected at heart rate 75 bpm.

Measurement of Augmentation Index

The augmentation index (AI) of the radial pressure waveform was calculated as an index of the wave reflection (*23–25*). AI was defined as the ratio of the height of the late systolic shoulder/peak (P_2) to that of the early systolic shoulder/peak (P_1) in each pulse (Fig. 1). The P_1 and P_2 were determined by multidimensional derivatives of the original pressure wave. Larger values of AI indicate increased wave reflection from the periphery or earlier return of the reflected wave as a result of increased arterial stiffness (*26*). To eliminate the influence of HR, an adjusted AI based on an HR of 75 bpm (AI@75) was also used (*27*). The mean value of AI calculated from the two radial waveform measurements was applied to the statistical analyses.

The pulse wave monitor device consists of a sensor unit for radial pulse measurement and a laptop computer. The sensor unit has a pressure sensor with an array of multiple 40 microtransducer elements. One of these 40 sensor elements was selected automatically to obtain the optimal radial pressure waveforms. The signals were digitized at 500 Hz. The radial pulse waveform obtained with this device has been shown to be identical to the waveform obtained by intra-arterial recordings. The intra- and inter-observer variability of the method were 3.6% and 2.4%, respectively (*25*).

Statistical Analysis

Comparisons between before and after exercise were evaluated with a paired *t*-test. Linear regression analysis was used to assess the correlation between variables and Pearson's correlation coefficient was calculated. Multiple regression analysis was performed for the effect of long-term aerobic exercise on AI. All results were expressed as the mean±SD unless otherwise specified. All statistical analysis was performed using SPSS software, version 12.0 (SPSS Inc., Chicago, USA). A probability value less than 0.05 was considered to indicate statistical significance.

Results

Baseline characteristics of the study subjects are summarized in Table 1. Thirty-eight (38.4%) subjects were hypertensive with a SBP of more than 140 mmHg and/or diastolic BP (DBP) of more than 90 mmHg. In the twenty-two subjects, the pre-exercise BP was measured three times due to the large differences between the initial two observations. The averaged SBP values for each of the three measurements in these 22 subjects were as follows: 1st measurement, 148±20 mmHg; 2nd measurement, 138±22 mmHg; 3rd measurement, 139±20 mmHg. In 2 of these subjects, an additional (4th) measurement was performed. Similar trends were observed in the post-exercise BP measurements, which were performed in 17 subjects: the average SBP values were 148±19, 134±22, and 133±23 mmHg for the three measurements. Basal AI was within the standard range as determined by our previously developed nomogram (*22*).

Effects of Acute Aerobic Exercise

The effects of acute aerobic exercise on BP and AI are shown in Table 1. Both SBP (ΔaSBP) and DBP (ΔaDBP) were significantly decreased after 30-min of aerobic exercise. Subjects with higher initial BP showed a larger exercise-induced BP reduction ($r=-0.291$, $p=0.004$ for basal SBP and ΔaSBP; $r=-0.394$, $p<0.001$ for basal DBP and ΔaDBP). However, no significant changes were observed in the AI or in the AI@75. Acute exercise–induced changes in the AI (ΔaAI) and AI@75 (ΔaAI@75) were significantly and inversely correlated with pre-exercise AI ($r=-0.47$, $p<0.001$) and pre-exercise AI@75 ($r=-0.54$, $p<0.001$), respectively (Fig. 2A, B), indicating that the subjects with higher basal AI showed more profound reduction in AI after the 30-min aerobic exercise. Furthermore, changes in SBP after the 30-min exercise were significantly correlated with ΔaAI ($r=0.24$, $p=0.015$) as well as ΔaAI@75 ($r=0.22$, $p=0.027$). Since there were

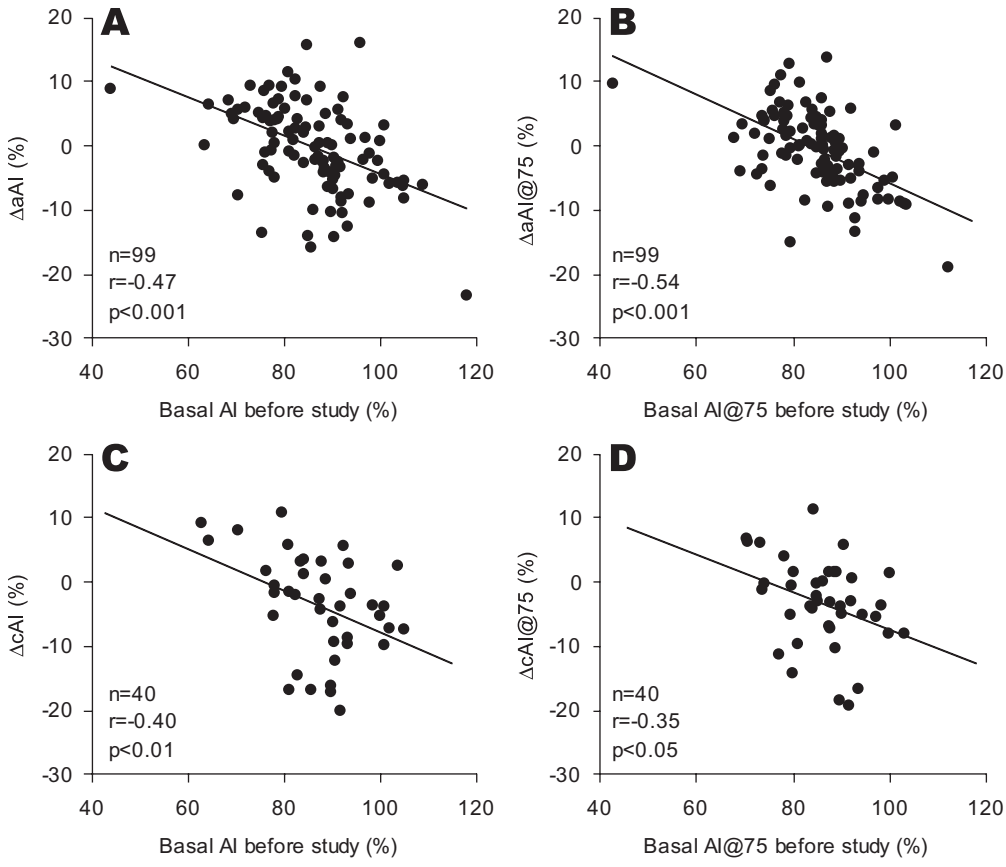

Fig. 2. *Exercise-induced changes in AI in correlation with basal AI. A: The relationship between basal AI and the change in AI after 30-min acute aerobic exercise (ΔaAI). B: The relationship between basal AI@75 and the change in AI@75 after 30-min acute aerobic exercise (ΔaAI@75). C: The relationship between basal AI and the change in AI after 6-month long-term aerobic exercise (ΔcAI). D: The relationship between basal AI@75 and the change in AI@75 after 6-month long-term aerobic exercise (ΔcAI@75). AI@75, AI corrected for a heart rate of 75 bpm.*

two cases located in the edge of the distribution area (Fig. 2A, B), further analyses were carried out without these two cases. However, the observed correlations between basal AI and *Δ*aAI, as well as *Δ*aAI@75, were still significant ($p<0.001$ for both). SBP and DBP also were significantly reduced after the 30-min exercise (SBP: 135±20 to 131±20 mmHg, $p=0.008$; DBP: 76±11 to 74±10 mmHg, $p=0.011$). Again, no significant changes were observed in AI (86±10 to 86±9%, $p=0.836$) or AI@75 (85±8 to 85±7%, $p=0.519$).

Effects of Long-Term Aerobic Exercise

The effects of long-term exercise training were evaluated in 40 subjects. Their baseline characteristics and the effects of long-term aerobic exercise on BP and AI are shown in Table 2. Significant reductions of BP, AI and AI@75 were observed after the 6-month aerobic exercise. Changes in the AI (*Δ*cAI) and AI@75 (*Δ*cAI@75) after the 6-month training were also significantly and inversely correlated with pre-exercise basal AI and AI@75 ($r=-0.40$, $p<0.01$, and $r=-0.35$, $p<0.05$), respectively (Fig. 2C, D). However, the *Δ*cAI was not associated with the changes in SBP observed after long-term exercise (*Δ*cSBP) ($r=0.103$, $p=0.528$).

Table 2 also shows the effects of acute 30-min exercise performed after 6-month training. As in the first acute exercise, both SBP and DBP were significantly decreased after the 30-min aerobic exercise, while no significant changes were observed in the AI or AI@75. HR was increased after the 30-min exercise, presumably as a compensatory response to the reduced SBP. The reduction in SBP was larger at the post-exercise measurement than at the pre-exercise measurement.

Relationship between the Effects of Acute and Long-Term Aerobic Exercise

In 40 subjects, *Δ*cAI was significantly and positively related to *Δ*aAI (Fig. 3). The subjects who showed a greater reduction of AI at the first acute aerobic exercise also showed a greater reduction in AI after the 6-month long-term aerobic exercise training. In contrast, no correlation was observed between the

Table 2. Background Characteristics of Subjects Who Completed Long-Term Study and the Effect of 6-Months Long-Term Aerobic Exercise

n (male/female)	40 (3/37)		
Age (years old)	67±6		
Body height (cm)	151±7		
Body weight (kg)	49±6		
Body mass index (kg/m^2)	22±3		
	Effect of 6 months long-term exercise		
	Pre	Post	*p*
Systolic BP (mmHg)	136±19	129±17	0.0056
Diastolic BP (mmHg)	75±11	70±10	0.0008
Heart rate (bpm)	73±9	73±8	0.56
AI (%)	87±10	84±10	0.0068
AI@75 (%)	86±8	83±9	0.0048
	Effect of last 30 min acute exercise		
	Pre	Post	*p*
Systolic BP (mmHg)	129±17	121±15	0.37×10^{-5}
Diastolic BP (mmHg)	70±10	69±10	0.10
Heart rate (bpm)	73±8	77±11	0.0028
AI (%)	84±10	84±10	0.91
AI@75 (%)	83±9	85±9	0.11

Values are mean±SD. BP, blood pressure; AI, augmentation index; AI@75, AI corrected at heart rate 75 bpm.

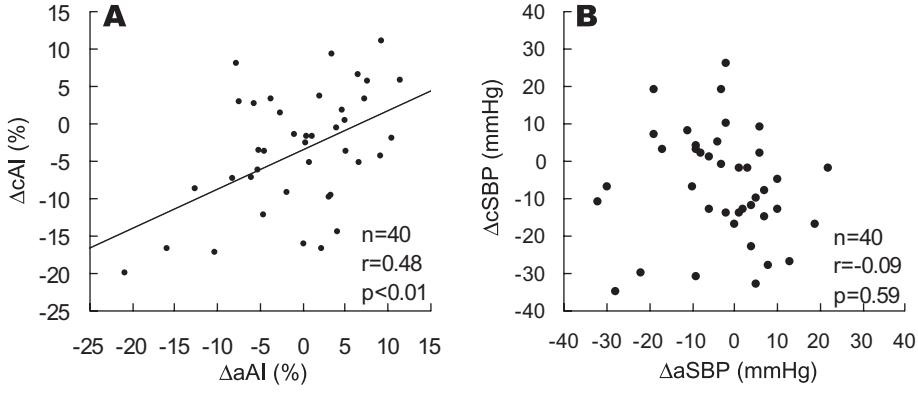

Fig. 3. *Correlations between acute and long-term changes in AI and SBP. A: The relationship between the changes in AI after the first 30-min of aerobic exercise (ΔaAI) and after 6-month exercise training (ΔcAI). B: The relationship between the changes in SBP after the first 30-min aerobic exercise (ΔaSBP) and after 6-month training (ΔcSBP).*

changes in SBP after a single acute exercise (*Δ*aSBP) and *Δ*cSBP. Four subjects exhibiting a marked *Δ*aSBP (more than −20 mmHg) showed higher basal BP compared with the rest of the subjects (SBP: 165±12 *vs*. 133±16 mmHg, $p<0.001$; DBP: 88±14 *vs*. 73±10 mmHg, $p=0.010$). Seven subjects exhibiting prominent *Δ*cSBP also showed higher basal SBP compared with the rest of the subjects (150±17 *vs*. 133±18 mmHg, $p=0.021$).

To further evaluate which parameters can predict the effects of long-term aerobic exercise on arterial stiffness, multiple regression analysis for *Δ*cAI was performed with the following parameters: age, sex, basal AI, basal SBP, *Δ*aAI, and *Δ*aSBP (Table 3). Both basal AI and *Δ*aAI were significantly and independently associated with *Δ*cAI.

Discussion

In the present study, we investigated the acute and long-term effects of aerobic exercise on radial AI in community-dwelling elderly subjects. Acute aerobic exercise significantly

Table 3. Multiple Regression Analysis for the Effect of 6-Months Aerobic Exercise on Radial AI

Parameters	β	p
Basal AI (%)	−0.39	0.015
AI change by first 30-min exercise (%)	0.37	0.02
Age (years old)	−0.09	0.50
Sex (male)	0.21	0.16
Basal systolic BP (mmHg)	0.07	0.31
Systolic BP change by first 30-min exercise (mmHg)	0.16	0.33

BP, blood pressure; AI, augmentation index.

reduced BP, while no significant reduction of AI was observed after single 30-min aerobic exercise. On the other hand, a significant reduction of both BP and AI was observed after 6-month long-term exercise training. The exercise-related reduction in AI was especially pronounced in subjects with higher basal AI. Furthermore, the changes in AI after long-term exercise were significantly related to those observed after the first 30-min exercise performed at the beginning of the study.

The magnitude of BP reduction after exercise has been shown to be subject to the "law of initial values," *i.e.*, the higher the initial BP, the larger the exercise-induced BP reduction (*28*–*30*). In the present study, we observed similar findings in AI changes after exercise. The subjects with higher basal AI showed larger reduction in AI after both acute and long-term exercise. It has been shown that the acute effect of exercise on BP is a low-threshold phenomenon that occurs after energy expenditures requiring only 40% maximal capacity (*28*). This is consistent with the present findings that mild-to-moderate aerobic exercise significantly reduced BP.

The BP reduction after long-time exercise observed in the present study was slightly higher than the values expected based on recent meta-analyses (*31*, *32*). These reports estimated that aerobic exercise induced a BP reduction of 3 to 4 mmHg in SBP, and 2 to 3 mmHg in DBP. In the present study, several subjects with systolic hypertension showed marked ΔcSBP resulting in a prolonged BP reduction. Since we carried out careful repeated readings of BP at both the pre- and post-exercise measurements, and the correlation between the two measurements was highly significant (r=0.688), the discrepancies were more likely to be due to differences in the clinical backgrounds of the patients, rather than technical errors during the BP readings.

Long-term exercise training produces a dominant vagal control of the heart and a resting bradycardia in young adults. In older adults, a similar phenomenon has also been reported in a meta-analysis (*33*). However, no significant changes were observed in HR in the present study after the 6-month exercise period, whereas both BP and AI were markedly reduced. To obtain a exercise-related changes in HR, a longer period of time or a higher intensity of exercise may require in elderly subjects, thought rapid training adaptation in younger subjects has also been reported (*34*). Finally, the lack of HR reduction in the present study may also have been partly due to compensatory responses to the reduced BP.

Several studies have reported the favorable effects of long-term aerobic exercise and physical fitness on arterial stiffness. In healthy young subjects, 4-week exercise training significantly increased systemic arterial compliance (*35*). Tanaka and colleagues reported a lack of post-menopause increase in central arterial stiffness in physically active women (*17*, *18*). Furthermore, Edwards *et al*. (*36*) prospectively demonstrated an improvement of systemic arterial stiffness with endurance exercise training in patients with coronary artery disease. However, the long-term effects of aerobic exercise in elderly subjects have not been consistent.

A previous study reported a significant positive correlation between systemic arterial compliance and the duration of treadmill exercise in elderly subjects (*37*). Vaitkevicius *et al*. (*38*) have also shown that endurance-trained elderly men had lower arterial stiffness than their sedentary peers, suggesting that regular aerobic exercise can mitigate the age-associated augmentation of arterial stiffness. On the other hand, Ferrier *et al*. (*39*) failed to demonstrate a favorable effect of 8-week home-based aerobic exercise training on arterial stiffness in elderly patients with isolated systolic hypertension. They concluded that the large arterial stiffness underlying isolated systolic hypertension was resistant to relatively short-term aerobic training. These findings may indicate that the effect of aerobic exercise on arterial stiffness in elderly subjects depends on the duration of the exercise training. One intriguing finding in the present study is that the acute effect of aerobic exercise was significantly and independently associated with the outcome of 6-month long-term exercise on AI.

AI is partially dependent on aortic and large-artery PWV. A higher PWV results in earlier arrival of the reflected pressure wave, and increases AI at both the aortic and peripheral arteries (*40*). Since it has been demonstrated that aerobic exercise significantly reduces PWV, it is conceivable that long-term exercise–induced reduction of AI is, at least in part, due to the deceleration of PWV (*41*). The relaxation of vascular smooth muscle induced by the endothelial production of NO in response to the shear stress caused by increased blood flow (*11*, *12*) is likely to account for the acute elevation in arterial compliance and deceleration of PWV by exercise (*41*), although a different mechanism has also been shown to play a role (*42*). Furthermore, it is also well known that BP itself can influence AI as well as PWV. Accordingly, the reduction of AI observed in this study might also have been induced by BP reduction in addition to the improvement of arterial compliance. However, Cameron and Dart (*35*) revealed that the increase in systemic arterial compliance by 4-week exercise training is greater than that expected by BP-dependent improvement, indicating that improvement of intrinsic arterial compliance is the main mechanism of the exercise-related decrease in AI. The present finding that long-term aerobic

exercise decreased AI independently of BP changes may further support the hypothesis that aerobic exercise improves arterial compliance more than expected by BP reduction.

In contrast, AI did not change after acute exercise at either pre- or post-intervention measurement, whereas BP showed a marked reduction. It has been previously demonstrated that acute aerobic exercise decreases femoral to posterior tibial PWV even after low-intensity and short-duration exercise (*42*). Since both aortic and peripheral arterial properties influence AI, it is not clear why AI was not reduced by exercise in the present study. The intensity of our exercise training may not have been sufficient to evoke the post-exercise reduction in AI. Further investigations with simultaneous measurement of PWV and AI under quantification of the intensity of exercise will be needed.

Subjects with higher basal AI showed more profound reduction in AI after the 30-min aerobic exercise. A larger exercise-induced BP reduction was also observed in the subjects with higher initial BP, and this change was significantly correlated with that of AI. The exercise-induced peripheral vasodilation may occur more intensively in the subjects with higher basal peripheral vascular resistance.

There were several limitations in this study. First, we did not evaluate the effects of long-term training in a case-control manner. Undefined factors, *e.g.*, seasonal variations, may have affected our results. Second, we did not give careful consideration to smoking habituation, because the majority of the participants were female. Frequency of smokers in elderly females is quite low in Japan.

In the present study, AI measured at the radial artery was used as an index of arterial stiffness. The radial AI can be easily obtained non-invasively from the radial pulse waveform (*43*). Furthermore, radial AI has been shown to have a close linear correlation with the aortic AI (*43*). In the present study, we also provided evidence that the radial AI could be a highly sensitive index for the improvement of vascular stiffness even prior to BP reduction. These findings indicate that radial AI could be a useful index for arterial stiffness in fields related to public health care as well as clinical assessment.

In summary, acute 30-min aerobic exercise significantly decreased BP without any change in radial AI in elderly subjects. In contrast, repetitive aerobic exercise training for 6 months significantly reduced both BP and AI. These results indicate that elderly subjects with higher AI may benefit from mild-to-moderate aerobic exercise training to improve arterial stiffness.

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