Original Article

Acute Exercise Increases Systemic Arterial Compliance after 6-Month Exercise Training in Older Women

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High physical activity or aerobic exercise training increases central arterial distensibility in older humans. However, the effect of a single bout of exercise on central arterial distensibility in older humans is unknown. Furthermore, the effect of exercise training on central arterial distensibility during exercise is unclear. We investigated whether systemic arterial compliance (SAC) changes after acute exercise in older humans, and, if so, whether this change in SAC is enhanced by aerobic exercise training. Seven untrained older women (61–69 years old) participated in a 6-month exercise intervention study. We measured SAC after acute exercise (cycling exercise at 80% of their individual ventilatory threshold for 30 min) before and after 6 months of aerobic exercise training. After exercise training, the individual ventilatory threshold was significantly increased. In addition, both the SAC at rest and that 30 min after acute exercise were significantly increased after the exercise training program. Before exercise training, there was no significant increase in SAC after acute exercise, whereas, after exercise training, the SAC was significantly increased 30 min after acute exercise. The present study suggests that, after aerobic exercise training, SAC increases after acute exercise in older humans, and that the SAC at rest and after acute exercise is enhanced by aerobic exercise training, thereby causing an effective adaptation in increase in cardiac output during exercise. (*Hypertens Res* 2008; 31: 377–381)

***Key Words*: systemic arterial compliance, aging, acute exercise, exercise training**

Introduction

Large arteries, and central arteries in particular, act as a conduit delivering blood to the tissues and organs, and have a buffering action to level off fluctuations in blood pressure created by cardiac pulsation and intermittent blood flow. A decreased arterial distensibility results in a decline in this buffering capacity, causing increases in systolic blood pressure and left ventricular afterload. The arterial distensibility decreases with advancing age (*1*, *2*). Several studies have reported that central arterial distensibility is higher in physically active humans compared with sedentary humans (*2–9*). Furthermore, aerobic exercise training induces an elevation of central arterial distensibility (*10–13*).

Many studies have reported that high physical activity or aerobic exercise training induced an increase in central arterial distensibility (*2–13*). However, there have been few studies on the effects of a single bout of exercise on central arterial distensibility. Kingwell *et al*. (*14*) reported that sys-

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Table 1. Age, Height, Body Weight, and BMI before and after Exercise Training in Older Women

	Before training	After training
Age, years	64±4	—
Height, cm	152±5	—
Body weight, kg	51±6	51±6
BMI, kg/m^2	22±3	22±3

BMI, body mass index. Values are means±SD.

temic arterial compliance (SAC) increased 30 min after acute exercise in their young human subjects. This finding suggests that a single bout of exercise increases SAC, and this elevation may effectively regulate the increase in cardiac output during exercise. However, the effect of acute exercise on SAC in older humans is unknown. Furthermore, the effect of exercise training on SAC during exercise is unclear.

We hypothesized that SAC increases after acute exercise, even in older humans who have decreased arterial distensibility, because the elevated SAC may effectively regulate the increase in cardiac output during exercise, and that the acute exercise–induced increase in SAC is reinforced by aerobic exercise training, thereby causing an effective adaptation in increase in cardiac output during exercise. To test our hypothesis, we measured SAC after acute exercise before and after 6 months of aerobic exercise training in older women. Our findings may be helpful to establish guidelines for the use of aerobic exercise training to help prevent cardiovascular diseases in older humans.

Methods

Subjects

Seven untrained older women (61–69 years old) participated in an exercise intervention study. None of the participants had a history of smoking, and none were currently taking any medications. All subjects were free from the signs and symptoms of any overt chronic diseases.

The study was approved by the Ethical Committees of the Institute of Health and Sport Sciences of the University of Tsukuba. The study conformed with the principles outlined in the Helsinki Declaration, and all subjects provided their written informed consent before inclusion in the study.

Experimental Protocol

All seven subjects completed an exercise intervention study. Ventilatory threshold (VT), blood pressure, and SAC were measured before and after 6 months of aerobic exercise training. Blood pressure and SAC were measured before and 30 min after acute exercise. Before they were tested, subjects were asked to refrain from alcohol and caffeine consumption and intense physical activity (exercise) for 24 h to avoid possible acute effects on exercise capacity, blood pressure, and arterial distensibility. All measurements were performed at a constant room temperature (25°C).

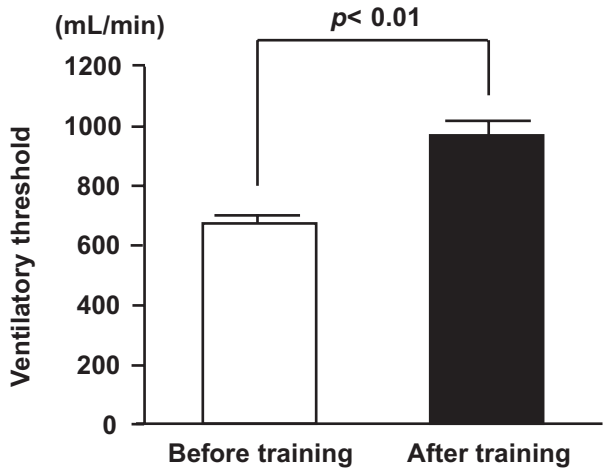

Fig. 1. *Individual ventilatory threshold during a cycle exercise test before and after 6 months of exercise training in older women (n = 7). Values are the means±SD.*

Exercise Test, Acute Exercise, and Exercise Training

The subjects performed a symptom-limited ramp-fashion cycling exercise (after 2 min at 20 W, with 10-W increases every 1 min) until they felt exhausted or reached 85% of the age-predicted maximal heart rate, before and after the exercise training program. Their individual VT was calculated by using regression analysis of the slopes of CO_2 production, O_2 uptake, and minute ventilation plots (*15–18*). Before and after the 6-month exercise training program, the subjects performed a steady state cycling exercise at the initial 80% of their individual VT for 30 min (acute exercise). The subjects submitted to a 6-month exercise training program on a cycle ergometer for 30 min/day, 5 days/week. The work rate of the cycle ergometer was set at 80% of the individual VT level, and was reset once a week using the heart rate corresponding to the initial 80% VT level.

Measurement of SAC

SAC can be calculated from the central arterial pressure waveform and stroke volume (SV) using the area method as follows (*19*):

$$SAC = SV/([A_s + A_d]/A_d)/(P_s - P_d),$$

where A_s is the area under the arterial waveform during systole, A_d is that during diastole, P_s is the end-systolic pressure, and P_d is the end-diastolic pressure. A carotid arterial waveform has been used as the central arterial waveform to esti-

Table 2. Systolic and Diastolic Blood Pressures, Heart Rate, and Stroke Volume at Rest and 30-min after Exercise

	Before training		After training	
	Before Ex	After Ex	Before Ex	After Ex
SBP, mmHg	137±20	128±13	114±19*	104±15
DBP, mmHg	79±6	81±3	64±9*	62±8
HR, beats/min	76±12	80±13	74±12	80±11
SV, mL	44±10	33±7	54±12	48±10

SBP, systolic blood pressure; DBP, diastolic blood pressures; HR, heart rate; SV, stroke volume; Ex, exercise. *$p<0.05$ *vs.* before training. Values are means±SD.

mate SAC non-invasively (*10*). In the present study, a finger arterial waveform was transformed to a brachial arterial waveform using analysis software (Beatscope; TNO-BMI, Amsterdam, the Netherlands) (*20*, *21*); the transformed arterial waveform was used to calculate SAC instead of the carotid arterial waveform (*22*, *23*). We estimated SV using the Modelflow method from the transformed arterial waveform using Beatscope (*24*, *25*).

Statistics

Values are expressed as the means±SD. To evaluate the differences in body weight, body mass index (BMI), and VT before and after exercise training, Student's *t*-test for paired data was used. To evaluate the differences in systolic blood pressure, diastolic blood pressure, heart rate, SV, and SAC before and after exercise training and acute exercise, statistical analysis was carried out by analysis of variance with repeated measures. In the case of significant *F* values, a Bonferroni post hoc test was used to identify significant differences among mean values. Values of $p<0.05$ were accepted as statistically significant.

Results

There were no significant differences in body weight or BMI between before and after the 6-month exercise training period (Table 1). After exercise training, individual VT during the exercise test was significantly increased (Fig. 1). Systolic and diastolic blood pressure at rest significantly decreased after exercise training, whereas there were no significant differences in heart rate or SV between before and after exercise training (Table 2). In addition, there was no significant interaction (exercise training × acute exercise) in the values of systolic and diastolic blood pressures, heart rate, and SV.

Figure 2 shows the SAC before and after exercise training. There were significant main effects of exercise training and acute exercise in the SAC. The SAC at rest (before acute exercise) and that 30 min after acute exercise (after acute exercise) were significantly increased after the exercise training program compared to before the program (Fig. 2). We also found interaction between exercise training and acute exercise in the SAC. Before exercise training, there was no significant change in the SAC after acute exercise, whereas, after exercise training, the SAC increased significantly 30 min after acute exercise (Fig. 2).

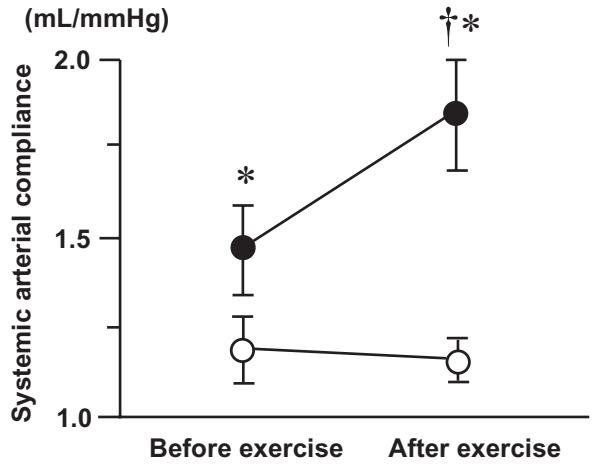

Fig. 2. *Systemic arterial compliance before and 30 min after acute exercise in older women ($n=7$). Values are the means±SD. Open circle, before 6 months of exercise training; closed circle, after 6 months of exercise training. *$p<0.05$ vs. before exercise training. †$p<0.01$ vs. before exercise.*

Discussion

In designing the present study, we hypothesized that SAC increases after acute exercise, even in older humans, and that the acute exercise–induced increase in SAC is reinforced by aerobic exercise training. Our results demonstrated that exercise training increased the SAC at rest in older women. We also revealed that there was no significant difference in SAC between before and 30 min after acute exercise in older women with a sedentary lifestyle, whereas, after 6 months of moderate exercise training, the SAC was increased significantly 30 min after acute exercise. Many studies have reported that central arterial distensibility is increased by habitual physical activity in young and older humans (*2–13*). On the other hand, it has been reported that systemic arterial distensibility was increased by a single bout of exercise in young humans (*14*). However, the effect of a single bout of exercise on systemic arterial distensibility in older humans is unknown. The present study showed that a single bout of exercise induced an increase in systemic arterial distensibility in older women after undergoing exercise training, but not before. These findings suggest that exercise training causes an improvement in SAC in older women, thereby contributing to an effective circulatory adaptation during exercise.

A decreased central arterial distensibility results in an

increase in left ventricular afterload (*22*, *26*). It has been reported that acute exercise induces an increase in arterial distensibility in young humans (*14*, *27*). The present study demonstrated for the first time that, after exercise training, the SAC increased significantly 30 min after exercise in older women. Therefore, it is considered that the acute exercise induced the increase in arterial distensibility in young and older humans. These regulations may help to increase cardiac output and inhibit excessive blood pressure rise during acute exercise.

In the present study, acute exercise did not cause a change in SAC before exercise training in older humans. However, after exercise training, SAC increased 30 min after acute exercise. There have been few studies on the effects of a single bout of exercise on arterial distensibility. Kingwell *et al*. (*14*) reported that SAC increased 30 min after acute exercise at an intensity of 60% $V_{O_2\,max}$ for 30 min in young humans with a sedentary lifestyle. Furthermore, we reported that light cycling exercise (30 W for 5 min) caused a decrease in arterial pulse wave velocity (PWV), which is an index of arterial distensibility, in the lower extremity in young men (*27*). These findings suggest that acute exercise induces an increase in arterial distensibility in young humans. In the present study, acute exercise did not cause a change in SAC before exercise training in older humans. This finding may have been due to a decrease in arterial distensibility by aging (*13*), rather than a low exercise intensity. This is because, in the present study, we used the same exercise intensity for the acute exercise both before and after exercise training. The present study suggests that acute exercise, even in older humans, may cause an increase in arterial distensibility after moderate exercise training.

The mechanism underlying the acute exercise–induced increase in SAC after exercise training remains to be elucidated. Arterial distensibility is defined by changes of function and structure in arteries (*28*, *29*). The critical factors of arterial distensibility are mainly changes and amounts of elastic and collagen fibers and tonus on vascular smooth muscle cells (*28*, *29*). We recently reported that the concentration of plasma nitric oxide (NO), which is a potent vasodilator substance produced by vascular endothelial cells, was significantly increased by aerobic exercise training in older women (*30*), and that the concentration of plasma endothelin-1 (ET-1), which is a potent vasoconstrictor peptide produced by vascular endothelial cells, was significantly decreased by aerobic exercise training in older women (*31*). Furthermore, we demonstrated that the production of endothelial NO synthase (eNOS) in the aorta decreases with aging, and that the decreased production is upregulated by exercise training in aged rats (*32*). Thus, the exercise training–induced improvement of vascular endothelial function is one of the causal factors for the acute exercise–induced increase in SAC after exercise training in older humans. Alternatively, the following mechanism is also possible. Exercise training causes changes in the autonomic nervous system and circulating hormones, which exert an influence on arterial distensibility. Therefore, it is possible that these factors affect the acute exercise–induced increase in SAC after exercise training in older humans.

The present study had several important limitations. First, the subjects were untrained and healthy older women, and the number of subjects was small ($n=7$). Therefore, further studies will be needed to generalize our findings. Second, this study did not employ blood sampling. Thus, we could not measure metabolic risk factors for cardiovascular disease before and after the exercise training program.

In conclusion, we demonstrated that exercise training increased the SAC at rest in older women. The present study also revealed that the SAC did not change by acute exercise in older women with a sedentary lifestyle, whereas, after moderate exercise training, the SAC significantly increased 30 min after acute exercise. These findings suggest that exercise training causes an improvement in SAC in older women, thereby contributing to an effective circulatory adaptation during acute exercise, *i.e.*, an increase in cardiac output during exercise and an inhibition of excessive rise in blood pressure during exercise.

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