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Effects of Acute Resistance Exercise on Arterial Stiffness in Young Men

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ABSTRACT

Background and Objectives: Increased central arterial stiffness is an emerging risk factor for cardiovascular disease. Acute aerobic exercise reduces arterial stiffness, while acute resistance exercise may increase arterial stiffness, but this is not a universal finding. We tested whether an acute resistance exercise program was associated with an increase in arterial stiffness in healthy young men. **Subjects and Methods:** Thirteen healthy subjects were studied under parallel experimental conditions on 2 separate days. The order of experiments was randomized between resistance exercise (8 resistance exercises at 60% of 1 repeated maximal) and sham control (seated rest in the exercise room). Carotid-femoral pulse wave velocity (PWV) and aortic augmentation index as indices of aortic stiffness were measured using applanation tonometry. Measurements were made at baseline before treatments, 20 minutes, and 40 minutes after treatments (resistance exercise and sham control). **Results:** There was no difference in resting heart rate or in arterial stiffness between the two experimental conditions at baseline. At 20 minutes after resistance exercise, heart rate, carotid-femoral PWV and augmentation index@75(%) were significantly increased in the resistance exercise group compared with the sham control ($p < 0.05$). Brachial blood pressure, central blood pressure and pulse pressure were not significantly increased after resistance exercise. **Conclusion:** An acute resistance exercise program can increase arterial stiffness in young healthy men. Further studies are needed to clarify the effects of long-term resistance training on arterial stiffness. (**Korean Circ J 2010;40:16-22**)

KEY WORDS: Arterial stiffness; Resistance training.

Introduction

Increased arterial stiffness has been associated with increased risk of cardiovascular disease and cardiovascular mortality.^{1,2)} It is well known that regular aerobic exercise improves cardiorespiratory fitness, decreases arterial stiffness,³⁾ reduces cardiovascular risk factors, and reduces risks of cardiovascular mortality.⁴⁾ In contrast, resistance exercise increases skeletal muscle strength and plays a key role in preventing aging-related muscle loss.⁵⁾

Also, because of evidence demonstrating positive effects of resistance exercise on cardiovascular diseases, the American Heart Association has recommended the use of resistance exercise in preventing and treating cardiovascular diseases.⁶⁾

While it has recently been reported that resistance exercise has a detrimental effect on vascular function, this effect has been of increasing interest to researchers. According to some studies that have been conducted in Japan, arterial stiffness is significantly decreased after acute resistance exercise.⁷⁾ However, other studies have reported contradictory results, that arterial stiffness is not significantly increased after acute resistance exercise.⁸⁾ Because studies to date are not consistent, further studies are needed to evaluate the effects of resistance exercise on arterial stiffness. Recently, the number of young male adults in Korea who participate in resistance exercise has been increasing day by day. Nevertheless, only a small number of studies have been conducted to examine the effects of resistance exercise on arterial stiffness. Therefore, the purpose of this study was to examine the effects of an acute, moderately intense, resistance exercise pro-

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gram on arterial stiffness in healthy young men.

Subjects and Methods

Study subjects and design

The current study used as subjects 13 healthy non-smoking men (aged 20-29 years) (Table 1). These subjects had no cardiovascular or metabolic diseases and had a habit of performing resistance exercise approximately three times a week. All measurements were performed after 8 or more hours of fasting. Twenty four hours before the measurement, subjects abstained from alcohol and caffeine. To rule out possible confounding effects of other transient exercises, other exercises were prohibited during the 24-hour period prior to the test. The room temperature of the laboratory was maintained at 21-23 °C. The measurement of arterial stiffness was performed by a well-trained researcher during the same period. The study involved a cross-over design in which the same subject was treated twice. All subjects randomly participated in a resistance exercise program or a sham control. For the resistance exercise program, eight types of systematic resistance exercises were performed. Sham controls rested on a comfortable chair while performing no exercises. All variables were measured at baseline before treatments, and again at 20 and 40 minutes after treatments.

Study methods

Resistance exercise

The resistance exercise program was performed by implementing eight types of systematic resistance exercise. Each exercise involved using a weight that was 60% of the 1 repetition maximum (RM). Subjects did 15 repetitions per set and completed two sets. This program has been recommended by the American College of Sports Medicine.⁹⁾ The eight types of resistance exercise included the following: Bench press, Squat, Lat pull down, Biceps curl, Leg extension, Leg curl, Upright row, Triceps extension.

Brachial and central blood pressure measurements

Brachial blood pressure was obtained by averaging

Table 1. Characteristics of subjects (n=13)

Variable	Mean ± SD
Age (years)	20.8 ± 2.2
Body Mass Index (kg/m ²)	23.4 ± 1.9
Brachial SBP (mmHg)	117.5 ± 8.8
Brachial DBP (mmHg)	70.7 ± 11.3
Brachial PP (mmHg)	47.5 ± 18.2
Heart rate (bpm)	58.8 ± 10.8

SBP: systolic blood pressure, DBP: diastolic blood pressure, PP: pulse pressure

the blood pressure readings from a mercury sphygmomanometer with the subject in the supine position following a resting period that was a minimum of 5-10 minutes. The interval between measurements was set at three minutes. For the measurement of central arterial pressure, the pulse wave was obtained from the radial artery using high fidelity applanation tonometry (Millar Instruments, Houston, TX, USA) and SphygmoCor (AtCor Medical, Sydney, Australia). Then, an auto measurement was done using a generalized radial-to-aortic transfer function formula.

Arterial stiffness measurements

Carotid-femoral pulse wave velocity (PWV) and Aortic augmentation index were measured using SphygmoCor. The measurement of these two parameters was done according to guidelines specified by the Clinical Application of Arterial stiffness, Task Force III.¹⁰⁾ For measurement of Carotid-femoral PWV, the distance between the carotid artery and the femoral artery was measured using a measuring tape. Using the distance between the carotid artery and aortic notch, the distance between the carotid artery and femoral artery (D) was calculated. According to a "foot-to-foot" method, with the use of a high-fidelity applanation tonometry, the time difference (Δt) between the points where the form of the pulse wave recorded on the carotid artery and femoral artery began to rise at systolic phase was measured. Then, the distance between the two arteries (D) was divided by the time difference (Δt).

$$PWV = D / (\Delta t) \text{ (m/s)}$$

The aortic augmentation index was obtained by analyzing the pulse wave that was recorded from the radial artery using applanation tonometry. Its magnitude was expressed as a percentile value. Because the aortic augmentation index is affected by changes in heart rate, aortic the augmentation index at a heart rate of 75 beats/min (Aix@75) was concurrently calculated.¹¹⁾ To enhance the accuracy of measurements, only values whose quality index exceeded 80% were used.

Statistical analysis

All measurements are expressed as mean ± SD or mean and 95% confidence interval (CI). Comparisons between and within groups were done using two way repeated analysis of variance (ANOVA) (group × time). Statistical significance was set at $p < 0.05$. All statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) 12.0 (SPSS Inc, Chicago, IL, USA).

Results

The characteristics of subjects are presented in Table 1. At baseline, prior to the treatment, all the measurement variables were similar between the two experimen-

Table 2. Changes in hemodynamic parameters and pulse wave analysis at baseline and 20 and 40 minutes after resistance exercise

Variables	Resistance exercise			Sham control (seated rest)		
	Baseline	After 20 minutes	After 40 minutes	Baseline	After 20 minutes	After 40 minutes
Heart rate (bpm)*	59.2 ± 9.3	80.4 ± 10.6	73.5 ± 9.6	58.5 ± 10.4	54.9 ± 13.0	55.2 ± 13.9
Brachial SBP (mmHg)	116.6 ± 10.3	119.8 ± 9.6	114.2 ± 10.2	118.0 ± 11.1	116.0 ± 8.6	114.8 ± 10.9
Brachial DBP (mmHg)	66.9 ± 6.3	71.2 ± 7.9	68.0 ± 7.8	70.8 ± 10.2	71.8 ± 7.6	70.8 ± 7.5
Brachial PP (mmHg)	49.7 ± 9.4	48.7 ± 5.8	46.1 ± 5.8	47.3 ± 7.3	45.2 ± 5.7	44.0 ± 7.2
Aortic SBP (mmHg)	97.9 ± 6.9	101.7 ± 8.1	96.6 ± 8.7	101.2 ± 10.5	98.8 ± 7.3	98.2 ± 10.2
Aortic DBP (mmHg)	67.4 ± 6.4	73.2 ± 7.8	69.4 ± 7.9	71.5 ± 10.2	71.2 ± 8.0	71.5 ± 7.8
Aortic PP (mmHg)	30.6 ± 5.9	28.5 ± 3.6	27.2 ± 3.3	29.7 ± 3.6	27.7 ± 3.1	26.7 ± 5.3
AIx@75 (%)*	-13.8 (-21.0–-6.7)	-0.3 (-7.7–7.0)	-6.5 (-13.9–0.9)	-7.6 (-14.8–-0.4)	-11.3 (-18.7–-3.9)	-15.1 (-22.5–-7.6)
AIx(%)	-6.3 (-13.4–0.8)	-2.6 (-10.3–5.1)	-5.4 (-12.6–1.9)	0.2 (-6.9–7.3)	-1.7 (-9.4–6.0)	-5.6 (-12.8–1.6)

* $p < 0.05$ for interact effects. SBP: systolic blood pressure, DBP: diastolic blood pressure, PP: pulse pressure, AIx: aortic augmentation index, AIx@75: aortic augmentation index at a heart rate of 75 beats per minute



Fig. 1. Changes in pulse wave velocity at baseline and 20 and 40 minutes after treatment.

tal conditions. Changes in hemodynamic variables and arterial stiffness after an acute resistance exercise program are shown in Table 2. Heart rate was 59.2 ± 9.3 bpm at baseline before resistance exercise and this was significantly increased to 80.4 ± 10.6 bpm at 20 minutes and 73.5 ± 9.6 bpm at 40 minutes after the acute resistance exercise program ($p < 0.05$). Systolic pressure and diastolic pressure of the brachial artery were 116.6 ± 10.3 mmHg and 66.9 ± 6.3 mmHg at baseline before resistance exercise. These values increased to 119.8 ± 9.6 mmHg and 71.2 ± 7.9 mmHg at 20 minutes after exercise (this difference did not reach statistical significance). Also, central systolic and diastolic blood pressure values were 97.9 ± 6.9 mmHg and 67.4 ± 6.4 mmHg at baseline, which increased to 101.7 ± 8.1 mmHg and 73.2 ± 7.8 mmHg at 20 minutes after exercise (this did not reach statistical significance). However, the carotid-femoral PWV was significantly elevated at 20 minutes following exercise ($p < 0.05$) (Fig. 1). Aortic AIx@75 was also significantly elevated at 20 minutes following exercise (Table 2).

Discussion

We examined the effects of an acute, moderate re-

sistance exercise program on arterial stiffness in healthy young men between the ages of 20 and 29 years. Heart rate, carotid-femoral PWV and Aortic AIx@75 were all significantly increased at 20 minutes after a session of exercise. Our findings are consistent with those of a previous study for which the investigators reported that an acute resistance exercise program increased the arterial stiffness in young subjects.¹²⁾

It is well known that physical activity protects against cardiovascular diseases. To date, however, most previous studies have focused on aerobic exercise. According to cross-sectional studies¹³⁻¹⁵⁾³⁵⁾ and interventional studies,¹³⁾¹⁵⁾ aerobic exercise reduces cardiovascular risk factors and is associated with decreased cardiovascular mortality and decreased arterial stiffness.

Arterial stiffness can be decreased by changes in vascular structural and functional factors. According to Seals et al.³⁾ a long-term aerobic exercise program can reduce or cause recovery from structural degeneration of the vascular wall that is related to aging, and thereby can reduce arterial stiffness. Also, a short-term aerobic exercise program can improve the functional capacity of blood vessels.¹³⁾¹⁶⁾ It is also known that arterial stiffness is decreased by increased expression of genes associated with vasodilation-inducing factors¹⁷⁾ and decreased oxidative stress.¹⁸⁾ Furthermore, the reduction in arterial stiffness following regular aerobic exercise was independent of the decrease in traditional cardiovascular risk factors. These results confirm that aerobic exercise reduces arterial stiffness.¹³⁾¹⁶⁾

In contrast, resistance exercise programs have focused on improvements in musculoskeletal functions rather than cardiovascular functions. Resistance exercise has a beneficial effect on maintenance of functional capacity and on traditional cardiovascular risk factors.⁵⁾ Based on this, the American Heart Association has recommended that resistance exercise be included in exercise programs for the prevention and treatment of cardio-

vascular disease.⁶⁾ Nevertheless, there is insufficient evidence to demonstrate a protective effect of resistance exercise on cardiovascular diseases compared with aerobic exercise. Furthermore, few studies have examined the correlation between resistance exercise and arterial stiffness, and results in this field have been contradictory.

Some studies reported that resistance exercise increases arterial stiffness.⁷⁾¹²⁾¹⁹⁻²¹⁾ Also, arterial compliance was decreased in middle-aged men who had been doing resistance exercise as compared with sedentary men in the same age range.²²⁾

Although the mechanism underlying the increase in arterial stiffness after resistance exercise is unknown, there are some possible explanations based on previous studies. These changes in arterial stiffness after resistance exercise and aerobic exercise are assumed to originate from changes in blood pressure during exercise. Because aerobic exercise mainly uses the large muscles in a rhythmical manner, changes in blood pressure are moderate during aerobic exercise. In contrast, resistance exercise increased blood pressure up to 310/250 mmHg during exercise. Long-term exposure to resistance exercise leads to increased content of smooth muscle cells on the vascular wall and changes in the load-bearing characteristics of elastin and collagen. This can play a pathophysiologic role in which resistance exercise increases arterial stiffness.²³⁾

DeVan et al.¹²⁾ reported that the increase in arterial stiffness immediately after resistance exercise was associated with increased central systolic pressure. In their study, young male subjects took part in an acute resistance exercise bout at 75% intensity of 1 RM. As a result, systolic blood pressure of the brachial artery showed no significant changes immediately after the resistance exercise. But central systolic blood pressure and arterial stiffness were significantly elevated. These results indicate that an assessment of the blood pressure of a central artery rather than the blood pressure of the brachial artery is more helpful for evaluating the effects of resistance exercise on blood vessels. In our study, although the result was not statistically significant, central blood pressure tended to increase following resistance exercise. PWV was significantly increased.

Another possible reason that arterial stiffness is increased after resistance exercise may be due to the effects of the Valsalva maneuver during resistance exercise. The Valsalva maneuver is commonly utilized during a high-intensity or a maximum-intensity resistance exercise maneuver. According to Heffernan et al.²⁴⁾ the PWV of the central artery is significantly increased in cases in which the Valsalva maneuver is deliberately generated. Increased arterial stiffness due to the Valsalva maneuver may be associated with increased intrathoracic and intraabdominal pressure, and these pressures are directly transferred to the aorta and arteries.²⁵⁾

The abrupt increase in pressure raises the pressure on intracellular space and vascular wall. Thus, it causes fatigue of the elastin fibers and thereby transfers the load from the circumferential to the relaxing collagen fibers. Owing to this, vascular elasticity is reduced and arterial stiffness is elevated.²⁶⁾

Third, the increase in arterial stiffness due to the effects of the autonomic nervous system should also be considered. It has been proposed that aerobic exercise improves heart rate variability, an indicator of autonomic nervous system function. In contrast, it has been reported that resistance exercise elevates catecholamine levels and activates the sympathetic nervous system.²⁷⁾ According to Eller,²⁸⁾ there are significant inverse correlations between increased catecholamine levels, increased arterial stiffness, and cardiac regulation of the vagus nerve. In other words, resistance exercise can activate the sympathetic nervous system. It is presumed that an imbalance in the autonomic nervous system via increased sympathetic activation and decreased parasympathetic activation during or right after resistance exercise can increase arterial stiffness. Activation of the sympathetic nervous system during resistance exercise raises the tension developed by vascular smooth muscle cells, and can thereby increase arterial stiffness.

Finally, differences in the sensitivity of the carotid baroreceptors may also play a role in the increase in arterial stiffness following resistance exercise. Heffernan et al.¹⁹⁾ compared baroreceptor sensitivity and arterial stiffness following acute aerobic exercise and acute resistance exercise. There were no changes in arterial stiffness following aerobic exercise, but the stiffness of the central artery increased following resistance exercise. Following both resistance exercise and aerobic exercise, the sensitivity of the baroreceptors was decreased. However, it decreased to a greater degree following resistance exercise.

As mentioned earlier, a very small number of studies have examined the effects of resistance exercise on arterial stiffness. Because the studies that have been done have reported contradictory results, no definite conclusions could be drawn regarding whether resistance exercise has an effect on arterial stiffness. It has been demonstrated, however, that resistance exercise has various beneficial effects on patients with cardiovascular diseases as well as on individuals in the general population. Accordingly, it might be essential to identify the specific causes of the difference in arterial stiffness following resistance exercise. Attempts should also be made to identify resistance exercise programs that have no detrimental effects on the vasculature and yet can provide the advantages of resistance exercise. The current study was conducted in male subjects. Therefore our results may not apply as much to female subjects.

Only a very small number of studies have been con-

ducted to examine changes in the arterial stiffness following resistance exercise in female subjects. Similar to male subjects, results for females have been contradictory. The difference between male and female subjects has not been clarified. In the case of aerobic exercise Moreau et al.¹⁶⁾ studied the effect of walking at an intensity of 70% of the maximal heart rate, five times a week for 40 minutes a day in middle-aged and elderly women. Tanaka et al.¹³⁾ utilized the same exercise program but implemented it in middle-aged and elderly men. Together, their findings show that aerobic exercise causes greater recovery of arterial elasticity in women as compared with men (40% vs. 25%). Collier²⁹⁾ implemented aerobic exercise and resistance exercise during a 4-week period in hypertensive middle-aged men and women. They found that aerobic exercise and resistance exercise decreased blood pressure in both men and women. In particular, it was shown that the decrease in diastolic pressure was greater in women than in men. However, they found no changes in arterial stiffness after resistance exercise. These results indicate that resistance exercise does not elevate arterial stiffness in women and it also has an anti-hypertensive effect in women. Casey et al.³⁰⁾ studied the effect of progressive resistance exercise in healthy postmenopausal women. The exercise was performed approximately twice a week for about 30 minutes each session for three months. There were no changes in compliance of the central artery or in aortic augmentation index. Another recent study was conducted by Yoshizawa et al.³⁶⁾ He used middle-aged women from Japan. Resistance exercise was performed twice a week at 60% intensity of 1 RM for a 12-week period. They found that carotid-femoral PWV was slightly decreased. To summarize, arterial stiffness was not decreased or changed following resistance exercise in women. It is possible that there is a difference between males and females in the change in arterial stiffness after resistance exercise. According to a study conducted by Cortez-Cooper et al.²⁰⁾ in young women, in which high-intensity resistance exercise was performed for an 11-week period, carotid artery augmentation index and central stiffness increased.

It has been reported that women have lower blood pressure and a lower incidence of hypertension than men. Premenopausal women have a lower risk of developing cardiovascular diseases than men of the same age. Similarly, the degree of arterial stiffness has been reported to be lower in women than men. With regard to changes in arterial stiffness following exercise, however, no definite results have been reported on possible mechanisms for gender differences. Further efforts are needed to clarify these possible gender differences.

Because all of the study subjects who showed no changes or a decrease in arterial stiffness following resistance exercise were middle-aged or elderly women, the

impact of age should also be considered.

A review of previous studies on the effects of resistance exercise on arterial stiffness in young adults was conducted. DeVan et al.¹²⁾ showed that compliance of the carotid artery was significantly decreased and beta stiffness was significantly increased following resistance exercise. Additionally, Heffernan et al.¹⁹⁾ showed that carotid-femoral PWV was significantly increased after an acute bout of resistance exercise. In training studies arterial stiffness was shown to increase¹⁹⁾²⁰⁾ and the compliance of the carotid artery was shown to decrease⁷⁾¹²⁾²¹⁾ after resistance training in young adults; but this is not a universal finding.⁸⁾¹⁸⁾²⁴⁾

A review of previous studies that were conducted in elderly people was also done. In most cases, these studies showed no changes or a slight decrease in arterial stiffness following resistance exercise. Poelkens et al.³¹⁾ implemented a 10-week resistance training study in which exercise intensity was gradually raised from 50% to 80% of 1 RM. They found no changes in compliance of the carotid artery and arterial stiffness. Yoshizawa et al.³⁶⁾ implemented a 3-month resistance exercise training study and showed that carotid-femoral PWV was slightly decreased. Maeda et al.³²⁾ showed that there were no changes in arterial stiffness following 12 weeks of lower body resistance exercise.

The difference seen between elderly people and young adults may be due to a poor adaptation of the blood vessels to stimuli, as described by Ferrier et al.³³⁾ It has been shown that sensitivity to stimuli is relatively higher in younger subjects compared to older subjects. According to epidemiological studies that were previously reviewed, arterial stiffness is elevated in elderly men who have done long-term resistance exercise as compared with sedentary men in the same age range. These results indicate that a long-term resistance exercise program can elevate arterial stiffness.

The intensity of resistance exercise may also help explain the contradictory results between elderly people and young adults. According to studies that have been conducted in young adults, the intensity of resistance exercise was greater in order to maximize the effects of resistance exercise. In studies that have been conducted in elderly people, because of safety concerns, resistance exercise of moderate or progressively raised intensity has mainly been performed. No definite mechanisms have been discovered regarding how resistance exercise increases arterial stiffness. According to Otsuki et al.³⁴⁾ the increase in arterial stiffness may be related to increased levels of endothelin-1 (ET-1), one of the vasoconstriction-inducing factors in young male subjects who have long performed resistance exercise. In other words, a high-intensity resistance exercise raises the concentration of ET-1 and this leads to vasoconstriction. Thus, it causes decreased functioning of vascular endothelial cells

and thereby increases arterial stiffness. Based on findings that there were no changes in arterial stiffness in young adults after 12 weeks of progressive resistance exercise, it can be inferred that the intensity of resistance exercise is a major factor affecting changes in arterial stiffness.³⁰⁾ In the current study, arterial stiffness was increased following a moderate degree of an acute resistance exercise bout. According to Rakobowchuk et al.⁸⁾ there were no changes in arterial stiffness following 12-weeks of high-intensity resistance training. The difference in the intensity and period of resistance exercise may explain the difference in changes following the resistance exercise. Further discussion is mandatory to clarify this. In association with the current study, it is possible that previous exercise habits can have an effect on the degree of vascular stiffness. All the study subjects who were enrolled in the current study had been performing resistance exercise at an average frequency of three times a week. It is therefore possible that the subject's previous exercise habits affected the changes in arterial stiffness following resistance exercise. According to a study conducted by DeVan et al.¹²⁾ arterial stiffness was increased following an acute resistance exercise program despite the fact that the subjects did not exercise during the previous six months. Further studies are warranted to examine the effects of current exercise habits on the arterial stiffness response following an acute resistance exercise program.

There are several limitations of the current study. The current study was conducted in a small number of healthy young male subjects. Therefore, our results cannot be generalized to people with clinical diseases or other subjects with a different age range. An assessment of the transient changes in arterial stiffness after an acute resistance exercise session was made. The changes in arterial stiffness cannot predict changes following long-term resistance exercise training. Despite these limitations, however, the current study might be of great value as it was designed to examine the effects of resistance exercise on the degree of vascular stiffness in Korean people.

To summarize, consistent conclusions cannot be drawn yet regarding the effects of resistance exercise on arterial stiffness. We showed that an acute resistance exercise bout increases arterial stiffness in young male subjects aged between 20 and 29 years. Therefore, resistance exercise should be performed carefully by people who are at increased risk of developing cardiovascular diseases. Further studies are also warranted to examine the effects of an acute and a long-term resistance exercise program on arterial stiffness.

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