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# Effects of High Intensity Resistance Training on Arterial Stiffness and Wave Reflection in Women

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**Background:** Cross-sectional studies reported that chronic resistance training is associated with arterial stiffening in men. These findings are in marked contrast to those found with aerobic exercise and may have important clinical relevance with regard to cardiovascular disease risk. However, the effect of resistance training on arterial stiffness has not been confirmed by interventional studies nor has this relation been investigated in women.

**Methods:** To determine whether a strength training program increases regional and central arterial stiffness in women, 23 healthy young women (29 ± 1 years; mean ± SD) participated in a high-intensity strength and power training program for 11 weeks. Ten other women (27 ± 2 years) served as time controls.

**Results:** In the intervention group, one repetition maximal strength increased 12% to 17% ($P < .0001$), and leg fat-free mass (via DEXA) increased significantly. Brachial blood pressure (BP) and fasting plasma lipid and lipoprotein concentrations did not change across the 11 weeks. Carotid augmentation index, a measure of arterial wave reflection and arterial stiffness, increased from −8% ± 13% to 1% ± 18% ($P < .05$), and carotid–femoral pulse wave velocity increased (791 ± 88 *v* 833 ± 96 cm/sec; $P < .05$). There were no changes in femoral–ankle pulse wave velocity, a segmental measure of peripheral arterial stiffness.

**Conclusions:** We concluded that a high-intensity resistance training program increases arterial stiffness and wave reflection in young healthy women. Our present interventional results are consistent with the previous cross-sectional studies in men in which high-intensity strength training is associated with arterial stiffening. Am J Hypertens 2005; 18:930–934 © 2005 American Journal of Hypertension, Ltd.

**Key Words:** Arterial compliance, strength training, exercise, C-reactive protein.

Cardiovascular disease is the number one cause of mortality in women, and coronary heart disease is the predominant cardiovascular event comprising more than half of the events in women.[1] According to the Framingham Heart Study, 63% of women who died suddenly of coronary heart disease, had no previous symptoms of this disease. Therefore, there is mounting interest in finding new risk factors to better predict the presence of occult coronary heart disease. Emerging evidence indicates that elevated arterial stiffness may play a role in the pathogenesis of coronary heart disease and serve as an early marker for the detection of asymptomatic atherosclerotic lesions.[2]

It is widely accepted that regular participation in physical activity confers protective effects against coronary heart disease.[3] However, the type of the physical activity that has been examined in these epidemiologic studies is generally confined to aerobic exercises, and it is not clear whether strength training exerts similar cardioprotective effects. The benefits of strength training, including increased bone and muscle mass and increased strength of connective tissue, are being increasingly recognized.[4] Strength training could become an even more essential component of overall exercise and fitness programs if it was shown to have positive effects on the cardiovascular system as well. However, in marked contrast to regular aerobic exercise, we[5] and other investigators[6] have previously reported that strength training is associated with

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greater, rather than smaller, arterial stiffness in young and middle-aged men. Because these two previous studies rely on the cross-sectional comparison between weight-trained men and sedentary peers, it is not clear whether the increased arterial stiffness observed in men is a direct result of regular strength training. Furthermore, there are no data addressing this issue in women. Because of the well-known sex-related differences in cardiovascular adaptations to regular exercise,[7] the results obtained in men cannot necessarily be extrapolated to women.

Accordingly, the purpose of the present study was to determine whether a strength training program increases arterial stiffness in women. In the present study, we used a high-intensity, progressive weight training protocol to maximize the stimuli of weight training on arterial stiffness.

# Methods

## Subjects

Twenty-three young healthy women (29 ± 1 years, 165 ± 1 cm) were studied for the present study. All the healthy subjects were either sedentary or recreationally active, but none had been performing weight training exercises. An additional 10 healthy age-matched, non-strength training women (27 ± 2 years, 166 ± 2 cm) served as time controls. All subjects were normotensive (<140/90 mm Hg), nonobese (body mass index [BMI] <30 kg/m$^2$), and free of overt chronic diseases as assessed by medical history. Candidates who smoked in the past 2 years, were taking medications or anabolic steroids, or had significant intima–media thickening, plaque formation, or other characteristics of atherosclerosis (eg, ankle-brachial index [ABI] <0.9) were excluded. All subjects gave their written informed consent to participate, and all procedures were approved by the Institutional Review Board.

## Measurements

To avoid potential diurnal variations, subjects were tested at a same time of day throughout the study period. All the women were studied in the same phase of their menstrual cycle before and after the intervention. Before each testing session, subjects abstained from caffeine and fasted for at least 4 h. Subjects in the intervention group were studied 20 to 24 h after their last exercise training session to avoid the acute effects of exercise, but they were still considered to be in their normal (ie, habitually exercising) physiologic state.

Measures of arterial stiffness (pulse wave velocity [PWV]), wave reflection (carotid augmentation index [AI]), and blood pressure (BP) were obtained in duplicate using a recently validated Colin VP-2000 (Colin Medical Instruments, San Antonio, TX).[8] The automatic device simultaneously measured electrocardiogram (ECG), phonocardiogram (PCG), bilateral brachial and ankle BPs, and carotid and femoral arterial pulse waves. Arterial BPs on

**Table 1.** Changes in selected variables (eg, blood pressure and metabolic risk factors) with the respective intervention

| | Nonexercising Controls | | | Strength Training | | |
|---|---|---|---|---|---|---|
| | Before | After | Change | Before | After | Change |
| Brachial systolic BP (mm Hg) | 109 ± 7 | 111 ± 7 | 2.3 ± 6.2 | 115 ± 8 | 115 ± 8 | 0.6 ± 6.7 |
| Brachial diastolic BP (mm Hg) | 64 ± 8 | 67 ± 5 | 2.1 ± 6.9 | 67 ± 6 | 65 ± 7 | −1.3 ± 5.1 |
| Brachial mean arterial BP (mm Hg) | 80 ± 8 | 82 ± 5 | 1.5 ± 2.6 | 83 ± 6 | 83 ± 7 | −0.7 ± 4.8 |
| Pulse pressure (mm Hg) | 45 ± 6 | 45 ± 6 | 0.3 ± 5.4 | 48 ± 5 | 50 ± 4 | 2.0 ± 4.6 |
| Ankle systolic BP (mm Hg) | 115 ± 10 | 122 ± 10 | 6.1 ± 9.6 | 121 ± 13 | 120 ± 12 | −1.1 ± 9.9 |
| Ankle-brachial index (unit) | 1.07 ± 0.06 | 1.09 ± 0.05 | 0.02 ± 0.07 | 1.05 ± 0.09 | 1.04 ± 0.06 | −0.01 ± 0.08 |
| Heart rate (beats/min) | 55 ± 5 | 56 ± 8 | 0.7 ± 5.1 | 62 ± 8 | 58 ± 8* | 4.0 ± 6.6 |
| Blood viscosity (mPa · s) | 3.58 ± 0.20 | 3.68 ± 0.25 | 0.09 ± 0.26 | 3.10 ± 0.40 | 3.38 ± 0.31 | 0.28 ± 0.50 |
| Total cholesterol (mmol/L) | 4.4 ± 0.7 | 4.3 ± 0.6 | −0.07 ± 0.37 | 4.5 ± 0.7 | 4.6 ± 0.7 | 0.12 ± 0.38 |
| HDL-cholesterol (mmol/L) | 1.4 ± 0.4 | 1.5 ± 0.4 | 0.07 ± 0.30 | 1.5 ± 0.3 | 1.5 ± 0.2 | 0.02 ± 0.26 |
| LDL-cholesterol (mmol/L) | 2.5 ± 0.3 | 2.5 ± 0.3 | −0.02 ± 0.19 | 2.6 ± 0.6 | 2.7 ± 0.6 | 0.11 ± 0.23 |
| Triglycerides (mmol/L) | 0.72 ± 0.34 | 0.69 ± 0.18 | −0.13 ± 0.18 | 0.86 ± 0.49 | 0.86 ± 0.49 | 0.00 ± 0.30 |
| Plasma glucose (mmol/L) | 4.9 ± 0.6 | 4.8 ± 0.3 | −0.1 ± 0.5 | 4.8 ± 0.4 | 4.9 ± 0.3 | 0.2 ± 0.4 |
| C-reactive protein (mg/L) | 0.6 ± 0.7 | 0.5 ± 0.7 | −0.1 ± 0.3 | 0.5 ± 0.5 | 0.5 ± 0.7 | −0.1 ± 0.6 |

BP = blood pressure.
Data are mean ± SD.
* $P < .05$ from before training.

**Table 2.** Changes in body composition and muscular strength with the strength training intervention

| | Before Training | After Training | Change |
|---|---|---|---|
| Body mass (kg) | 71 ± 15 | 71 ± 14 | −0.3 ± 2.4 |
| Body fat (%) | 30.5 ± 9.9 | 28.7 ± 9.2* | −1.9 ± 2.6 |
| Lean body mass (kg) | 44.8 ± 4.5 | 46.0 ± 4.3* | 1.1 ± 1.4 |
| Limb muscle mass (kg) | 19.7 ± 2.3 | 20.4 ± 2.0* | 0.7 ± 0.7 |
| Bench press 1RM (kg) | 49.1 ± 11.8 | 55.4 ± 10.1* | 6.3 ± 3.7 |
| Squat 1RM (kg) | 74.1 ± 16.5 | 89.0 ± 14.3* | 15.0 ± 10.1 |

1RM = one repetition maximal strength.
Data are mean ± SD.
* $P < .05$ from before training.

the four limbs as well as ankle arterial pulse waves were measured with the modified oscillometric pressure sensor method. The average of two right brachial BP measurements was recorded. Carotid and femoral arterial pulse waves were obtained using arterial applanation tonometry incorporating an array of 15 micropiezoresistive transducers. Carotid–femoral PWV, a measure of the speed at which the pressure wave travels, was determined by measuring the distance between the carotid and femoral pulses and dividing by the time delay between the "foot" (that is, the start of the sharp systolic upstroke) of the carotid and femoral pressure waves. The distance between carotid and femoral arteries (carotid–femoral distance) was measured as a straight surface distance between the carotid and femoral tonometer placement sites using a random zero method. A minimum of two measurements were taken and if values varied from each other by more than 1.0 cm, additional measurements were taken. The coefficient of variation for carotid–femoral PWV was 5%. Heart–femoral and femoral–ankle distances were automatically calculated by the Colin VP-2000 based on the subject's height. Heart–femoral PWV was obtained by using the transit time between the second sound of the PCG and the dichrotic notch plus the transit time between the foot of the carotid and the foot of the femoral pulse waveforms. Ankle arterial pulse waves were obtained with the modified oscillometric pressure sensor method. During the measurement of PWV, ankle BP was inflated at a low pressure to obtain posterior tibial artery BP waveforms, from which the machine calculated the transit time between the femoral artery site (tonometer) and ankle site. The coefficients of variation for heart–femoral and femoral–ankle PWV were both 3%. The carotid pressure waveforms were used to obtain carotid AI, which has been proposed as an indicator of the magnitude of wave reflections and arterial stiffness.[8] The coefficient of variation for this measure was 12%.

A blood sample was drawn from an antecubital vein for the determinations of blood viscosity (Brookfield viscometer, Middlebavo, MA), and plasma lipids, lipoproteins (Vitros DT60 analyzer, Orthoclinical Diagnostics, Raritan, NJ), and C-reactive proteins (Alpco Diagnostics EIA, Windham, NH). Blood viscosity was measured at 37°C using a Brookfield cone and plate viscometer. Body composition was measured using a dual-energy X-ray absorptiometry (DEXA; Lunar DPX, GE Medical Systems, Fairfield, CT). Maximal muscular strength in the intervention group was tested before and after resistance training with the bench press and squat. After the warm-up, one-repetition maximal (1RM) strength was obtained according to the established guidelines. Due to the potential risks involved in 1RM strength testing and body composition measures (X-ray exposure during the DEXA scan), these tests were not performed in the control group.

## Strength Training

Subjects in the intervention group underwent four supervised strength training sessions/week for 11 weeks using a light-day/heavy-day periodized approach. Specific resistance exercises included bench press, overhead press, weight-assisted parallel bar dip, dumbbell crossover pull,

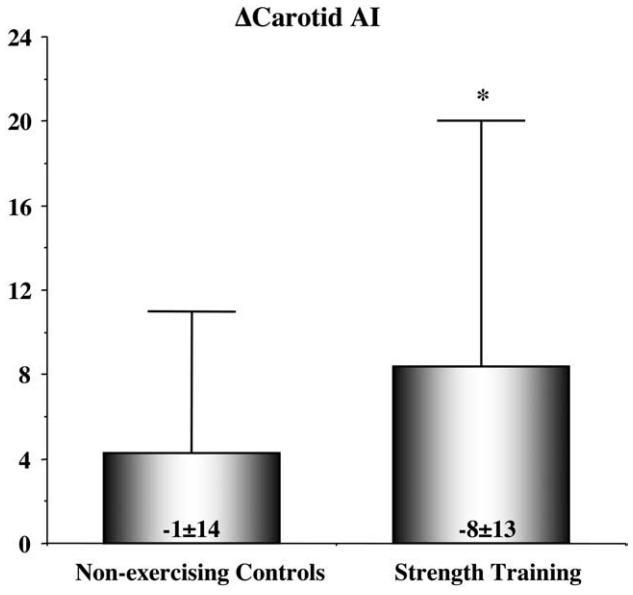


**FIG. 1.** Changes in carotid augmentation index (AI) in the control and intervention groups. Values are means ± SD. Baseline carotid augmentation values are shown in the bar. Carotid augmentation index increased from −8% ± 13% to 1% ± 18% ($P < .05$) in the intervention group. In the control group, the augmentation index was −1% ± 14% before and 3% ± 14% after with no significant change ( *$P < .05$).

**Table 3.** Pulse wave velocity (PWV) before and after the respective interventions

| | Nonexercising Controls | | | Strength Training | | |
|---|---|---|---|---|---|---|
| | Before | After | Change | Before | After | Change |
| Carotid–femoral PWV (cm/sec) | 724 ± 83 | 780 ± 66* | 56 ± 51 | 791 ± 88 | 833 ± 96* | 42 ± 80 |
| Carotid–femoral distance (cm) | 59.3 ± 2.6 | 59.3 ± 2.8 | 0.0 ± 1.3 | 61.6 ± 3.4 | 60.0 ± 2.4 | −1.6 ± 2.6 |
| Heart–femoral PWV (cm/sec) | 639 ± 57 | 649 ± 45 | 10 ± 44 | 652 ± 46 | 665 ± 58 | 12 ± 57 |
| Femoral–ankle PWV (cm/sec) | 835 ± 77 | 848 ± 64 | 13 ± 63 | 871 ± 88 | 862 ± 106 | −9 ± 55 |

Data are mean ± SD.
* $P < .05$ from baseline values.

dumbbell rowing motion, latissimus dorsi pulldown, dumbbell curl, squat/leg press, high pull, deadlift, medicine ball drills, and abdominal exercises. In the first 4 weeks, subjects performed three sets of 10-repetition exercises to concentric failure. In the subsequent 4 weeks, three sets of 5-repetition exercises were performed. In the last 3 weeks of the intervention, timed supersets—an upper body exercise paired with a lower body exercise—were incorporated using six sets of 5 repetitions during which the weights increased for four sets then decreased for two sets. During the entire study, resistance was increased for the next exercise sessions when subjects were able to complete correct repetitions on the final set on the heavy days. Subjects in the control group were instructed not to alter their normal activity levels throughout the study period.

### Statistics

Changes in the dependent variables were assessed by ANOVA with repeated-measures. All data were reported as mean ± SD. Statistical significance was set a priori at $P < .05$ for all comparisons.

## Results

Of the 37 subjects who underwent the initial testing, 14 dropped out during the course of the study, and the data presented for the training group are the means of 23 subjects. The subjects in the intervention group completed ~80% of the scheduled training sessions. As shown in Table 1, brachial and ankle BP and plasma concentrations of lipids, lipoproteins, and glucose did not change significantly in either group. There were no significant changes in blood viscosity or plasma C-reactive protein concentration.

Body mass did not change with the strength training intervention (Table 2). There were small but significant increases in lean body mass and limb muscle mass, and a corresponding reduction ($P < .05$) in percent body fat. Maximal muscle strength obtained during bench press and squat increased 13% and 20% (both $P < .05$), respectively. There was no change in body weight in the control group.

As depicted in Fig. 1, carotid AI increased ($P < .05$) in the intervention group. The magnitude of increase was significantly greater in the intervention group. No such significant change in AI was observed in the control group. Carotid–femoral PWV increased significantly in both groups (Table 3). Peripheral measures of arterial stiffness (femoral–ankle /PWV) did not change significantly in either group.

## Discussion

The major finding of the present investigation is that a short-term high-intensity progressive weight training program increases carotid AI and aortic PWV in young healthy women. To the best of our knowledge, this is the first study to determine the effects of resistance training intervention on arterial stiffness in women. The present intervention study is consistent with the previous cross-sectional studies in young men in which high-intensity weight training is associated with arterial stiffening.

Aortic PWV and AI are probably two of the most extensively used measures of arterial stiffness. Although these methodologies are considered to be indirect measures of arterial stiffness, a recent consensus identified PWV to be the best available technique for assessing arterial stiffness.[9] In the present study, weight training increased aortic PWV as well as AI, whereas femoral–ankle PWV did not change. This indicates that the effect of weight training appears to be manifested preferentially in the central, more elastic arteries and that peripheral, more muscular arteries may be affected to a lesser extent by the intervention.

The present findings should not discourage the general public from participating in regular resistance training. As evidenced by the increases in 1RM strength, the workout performed by subjects was rather intense and deviates from the weight training programs that are typically prescribed to the public.[4] In the present study, to maximize the stimuli of resistance training and the resultant adaptation in the cardiovascular system, we intentionally used a high-intensity resistance training program. In addition, subjects participating in the present study were young healthy women with smaller baseline levels of arterial stiffness. A more clinically important and unanswered question is whether a similar effect of resistance training

may be observed in older subjects with greater baseline arterial stiffness values. Further studies are warranted on this issue.

In addition to the primary dependent variable of arterial stiffness, we determined the effects of resistance training on such emerging risk factors as C-reactive protein[10] and blood viscosity,[11] which could potentially impact arterial stiffness. We are not aware of any previous studies that have determined the changes in these risk factors with resistance training. Three months of resistance training had no effect on these variables or on the more traditional risk factors, including fasting plasma glucose, cholesterol, and brachial BP. These results suggest that changes in arterial stiffness and wave reflection may not be mediated by these risk factors and that the effect of resistance training on arterial stiffness may be independent of the changes in these risk factors.

We can only speculate on the mechanisms involved in the change in arterial stiffness and wave reflection with strength training. The elastic properties of the arterial wall are determined by both structural components (eg, relative composition of elastin and collagen) and functional components (eg, vasoconstrictor tone exerted by the vascular smooth muscle cells). Given that structural changes in the arterial wall would take considerably longer than 11 weeks, it is likely that changes in the arterial elastic properties observed in the present study were mediated by mechanisms that modulate smooth muscle cell tone. In this context, potential mechanisms include greater sympathetic nerve activity, decreased nitric oxide bioavailability, greater local endothelin-1, and increased concentrations of vasoconstrictor hormones (eg, angiotensin II).[12,13] Another possibility is that reductions in heart rate at rest observed in the intervention group may have acted to increase AI as there is an inverse relation between heart rate and AI.[14] However, heart rate decreased an average of 4 beats/min, which is less than a 7% reduction; therefore, it is unlikely that this would completely explain the increase in AI with strength training. Future studies are warranted to investigate the mechanisms underlying the arterial stiffening effects of chronic resistance training.

An unexpected finding of the present study is an increase in carotid–femoral PWV in the control group. We do not believe that this is due to measurement error or artifact as we have established good reliability of our arterial stiffness measurements using this particular automatic device[8] and the coefficients of variation were small in the present study. As lifestyle activities of the control group were not changed, we can only attribute the causes of the changes to spontaneous factors (eg, seasonal changes).

In summary, the present study demonstrated that weight training induces increases in arterial stiffness and wave reflection in young healthy women. This finding is in contrast to previous aerobic exercise training studies demonstrating beneficial effects on arterial stiffness. Considering the recent guidelines that resistance training be incorporated into preventive and rehabilitative programs of overall physical activity, the present findings may have an important implication for providing proper exercise prescription.

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