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Unfavorable Effects of Resistance Training on Central Arterial Compliance

A Randomized Intervention Study

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Background—Reductions in the compliance of central arteries exert a number of adverse effects on cardiovascular function and disease risk. Endurance training is efficacious in increasing arterial compliance in healthy adults. We determined the effects of resistance training on carotid arterial compliance using the intervention study design.

Methods and Results—Twenty-eight healthy men 20 to 38 years old were randomly assigned to the intervention group (n=14) and the control group (n=14). Control subjects were instructed not to alter their normal activity levels throughout the study period. Intervention subjects underwent 3 supervised resistance training sessions per week for 4 months and detraining for a subsequent 4 months. The resistance training increased maximal strength in all muscle groups tested ($P<0.001$). There were no significant differences in baseline arterial compliance and β -stiffness index between the intervention and control groups. In the intervention group, carotid arterial compliance decreased 19% ($P<0.05$), and β -stiffness index increased 21% ($P<0.01$) after resistance training. These values returned completely to the baseline levels during the detraining period. Arterial compliance did not change in the control group. In both groups, there were no significant changes in brachial and carotid blood pressure, carotid intima-media thickness, lumen diameter, and femoral arterial compliance. Changes in carotid artery compliance were significantly and negatively related to corresponding changes in left ventricular mass index ($r=-0.56$, $P<0.001$) and left ventricular hypertrophy index ($r=-0.68$, $P<0.001$).

Conclusions—In marked contrast to the beneficial effect of regular aerobic exercise, several months of resistance training “reduces” central arterial compliance in healthy men. (*Circulation*. 2004;110:2858-2863.)

Key Words: arteries ■ echocardiography ■ elasticity ■ exercise ■ ultrasonics

Arterial compliance in the central (cardiothoracic) circulation reflects the ability of an artery to expand and recoil during cardiac contraction and relaxation, thereby damping down the fluctuation in arterial pressure and blood flow.¹ Reductions in arterial compliance or increases in arterial stiffness impair this buffering function and contribute to elevations in systolic blood pressure, left ventricular (LV) hypertrophy, coronary ischemic disease, and reductions in arterial baroreflex sensitivity.²⁻⁴ Indeed, higher arterial stiffness is associated with a greater rate of mortality in patients with end-stage renal failure and essential hypertension.⁵ Accordingly, the prevention and treatment of arterial stiffness are of paramount importance.

We and others have demonstrated that regular aerobic exercise is efficacious in preventing and reversing arterial stiffening in healthy adults.⁶⁻⁸ In recent years, resistance exercise, another common exercise modality, has gained

widespread acceptance in exercise prescription and cardio-pulmonary rehabilitation programs and has become an integral component in the comprehensive health program endorsed by the major health organizations.^{9,10} These recommendations are based primarily on the documented impact of resistance training on the attenuation of osteoporosis and sarcopenia and related risks, including falling and functional disability.^{9,11} However, there is very little information on the potential influence of resistance training on nonmusculoskeletal components, in particular, cardiovascular function. In marked contrast to the favorable effects of regular aerobic exercise that we observed on arterial compliance, we recently found in a cross-sectional study that strength-trained middle-aged men exhibited rather “decreased” levels of arterial compliance.¹² Given the well-known limitation of cross-sectional study design and the conflicting results between aerobic and strength training, we

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deemed that our previous cross-sectional study should be confirmed prospectively with the interventional approach.

With this information as background, the primary aim of the present study was to determine the effects of strength training intervention on central arterial compliance. On the basis of our previous cross-sectional study,¹² we hypothesized that a period of strength training would decrease arterial compliance in previously sedentary men. In addition, after the strength training period, we used the detraining phase to further confirm that the changes we observed after strength training were indeed because of strength training per se. Our working hypothesis was that changes in arterial compliance would return to the baseline levels when the stimuli of daily weight lifting were removed.

Methods

Subjects

Twenty-eight healthy men were studied for the present study. None of the subjects had participated in any resistance or endurance training on a regular basis. All subjects were normotensive (blood pressure <140/90 mm Hg), nonobese (body mass index <30 kg/m²), and free of overt chronic diseases as assessed by medical history, physical examination, and complete blood chemistry and hematological evaluation. Candidates who had smoked in the previous 4 years, who were taking medications or anabolic steroids, or who had significant intima-media thickening, plaque formation, and/or other characteristics of atherosclerosis (eg, ankle-brachial index <0.9) were excluded. All subjects gave their written informed consent to participate, and all procedures were approved by the Institutional Review Board. Subjects were subsequently randomized into either the exercise intervention group or the nonexercising control group.

Measurements

The intervention group was studied 5 times: before training (baseline), at 2 months (midpoint of resistance training), at 4 months (at the completion of resistance training), at 6 months (midpoint of detraining), and at 8 months (at the completion of detraining). The control group was studied 3 times: at baseline, at 4 months, and at 8 months. To avoid potential diurnal variations, subjects were tested at a same time of day throughout the study period. Before each testing, subjects abstained from caffeine and fasted for at least 4 hours (a 12-hour overnight fast for determination of metabolic risk factors). Subjects in the intervention group were studied 20 to 24 hours 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) physiological state.

Incremental Exercise

To demonstrate that the subjects had not been sedentary, we measured maximal oxygen consumption during an incremental cycle ergometer exercise.¹³ Oxygen consumption (coefficient of variation [CV] 4±1), heart rate, and ratings of perceived exertion were measured throughout the protocol.

Strength Testing

Maximal muscular strength in the intervention group was tested before and after resistance training using the following exercises: half squat, bench press, leg extension, leg curls, lateral row, and abdominal bend. After 10 warm-up repetitions, 1-repetition maximums (1RM) were obtained according to the established guidelines. Because of the potential risks involved in 1RM testing, this test was not performed in the control group.

Metabolic Risk Factors for Coronary Heart Disease

To screen for the presence of coronary heart disease, fasting plasma concentrations of cholesterol and glucose were determined by use of enzymatic techniques.⁶

Arterial Blood Pressure at Rest

Chronic levels of arterial blood pressure at rest were measured with a semiautomated device (Form PWV/ABI, Colin Medical Technology) over the brachial and dorsalis pedis arteries. Recordings were made in triplicate with subjects in the supine position.

Carotid Artery Intima-Media Thickness

Carotid artery intima-media thickness (IMT) was measured from the images derived from an ultrasound machine equipped with a high-resolution linear-array broad-band transducer (5 to 10 MHz; axial resolution of 0.06 mm) as previously described.¹² Ultrasound images were analyzed by use of computerized image analysis software. At least 10 measurements of IMT were taken at each segment, and the mean values were used for analysis. Plaque was considered to be present if a localized irregular thickening was at least 1.5 mm thick. This technique has excellent day-to-day reproducibility (CV, 3±1%) for the carotid IMT.

Artery Stiffness and Compliance

The combination of ultrasound imaging of a common carotid artery with simultaneous applanation of tonometrically obtained arterial pressure from the contralateral carotid artery permits noninvasive determination of arterial compliance.^{6,14} Carotid artery diameter was measured from the images derived from an ultrasound machine equipped with a high-resolution linear-array transducer. A longitudinal image of the cephalic portion of the common carotid artery was acquired 1 to 2 cm distal to the carotid bulb. To assess the effects of peripheral artery compliance, the same procedure was repeated on the common femoral artery. The computer images were analyzed with the use of image analysis software. All image analyses were performed by the same investigator, who was blinded to the group assignments.

The pressure waveform and amplitude were obtained from the common carotid artery with a pencil-type probe incorporating a high-fidelity strain-gauge transducer (SPT-301, Millar Instruments).^{6,15} Because the baseline levels of carotid blood pressure are subjected to hold-down force, the pressure signal obtained by the tonometry was calibrated by equating the carotid mean arterial and diastolic blood pressure to the brachial artery value.^{6,12} The pressure waveforms were also used to obtain carotid augmentation index, which has been proposed as an indicator of the magnitude of wave reflections.^{8,5} In addition to arterial compliance,¹⁶ we also calculated β -stiffness index, which provides an index of arterial compliance adjusted for distending pressure.¹⁷ Arterial compliance and β -stiffness index were calculated by use of the equations $[(D1-D0)/D0]/[2(P1-P0)] \times \pi \times (D0)^2$ and $(\log P1/P0)/[(D1-D0)/D0]$, where D1 and D0 are the maximal and minimum diameters and P1 and P0 are the highest and lowest blood pressures. The day-to-day CVs were 2±1%, 7±3%, and 5±2% for carotid artery diameter, pulse pressure, and arterial compliance, respectively.⁶ The CV for femoral arterial compliance was 7±4%.

LV Dimensions, Mass, and Function

Echocardiography was used to measure LV dimension, wall thickness, and functions according to established guidelines.¹⁸ The LV mass was then calculated.¹⁹ The ratio of average LV wall thickness to LV internal end-diastolic diameter was used as an index of LV hypertrophy (CV, 7±3%).¹²

Body Composition

Body composition was determined by use of the bioelectric impedance method (CV, 4±2%).

Resistance Training Intervention

In the first 4 months of the study period, subjects in the intervention group underwent 3 supervised resistance-training sessions per week. During each training session, subjects completed 3 sets of 8 to 12 exercises at 80% of 1RM in the following order: leg extension, seated chest press, leg curls, lateral row, squat, and sit-ups. Subjects performed 12 repetitions in sets 1 and 2 and as many repetitions as possible to concentric failure in set 3. Resistance was increased for

TABLE 1. Selected Subject Characteristics

Variables	Control	Intervention
No.	14	14
Age, y	22±1	22±1
Height, cm	172±1	173±2
Body weight, kg	68.1±2.1	66.5±2.4
Body mass index, kg/m ²	22.9±0.7	22.2±0.7
Body fat, %	19±1	18±2
Lean body mass, kg	55.2±1.6	54.5±1.5
Heart rate, bpm	58±3	56±2
Total cholesterol, mmol/L	4.26±0.39	4.42±0.13
HDL cholesterol, mmol/L	1.46±0.07	1.62±0.10
Plasma glucose, mmol/L	4.7±0.1	5.0±0.2
Plasma insulin, μ U/mL	5.9±0.6	5.4±0.5
$\dot{V}O_2$ max, mL · min ⁻¹ · kg ⁻¹	49±3	49±2

Data are mean±SEM. $\dot{V}O_2$ max indicates maximal oxygen consumption.

the following exercise sessions when subjects were able to complete at least 10 repetitions in the final set. Recovery time between exercises was controlled at 2-minute intervals. Each training session lasted \approx 45 minutes. Trained assistants verbally encouraged the subjects and ensured proper form and technique. Subjects were instructed to refrain from any other regular exercise workouts during the entire study period. Subjects in the control group were instructed not to alter their normal activity levels throughout the study period.

Statistical Analysis

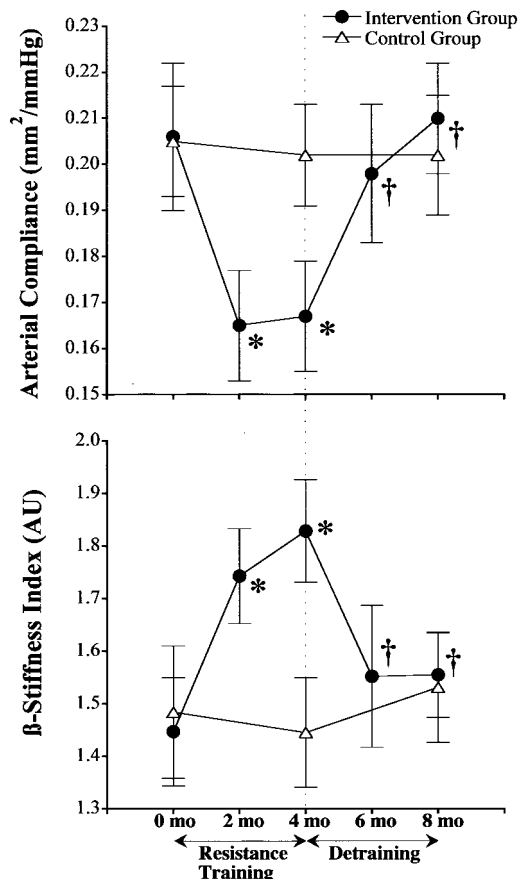
Changes were assessed by 2-way ANOVA (group \times period) with repeated measures. In the case of significant F values, a post hoc test (Newman-Keuls method) was used to identify significant differences among mean values. Pearson's correlation and regression analyses were performed to determine the relation between variables of interest.

Results

Before the intervention period, there were no significant differences in any of the variables between the groups (Table 1).

Twelve subjects in the intervention group completed 100% of all training sessions as scheduled (ie, 50 total training sessions in 4 months). For the remaining 2 subjects, the training period was extended by 1 week to ensure that each subject underwent the required 50 training sessions. The resistance training increased 1RM strength in all muscle groups tested ($P<0.001$). Percent increases for each of the exercises were 30% in squat, 20% in bench press, 47% in leg extension, 26% in leg curl, 25% in lat row, and 32% in abdominal bend.

There were no significant differences in baseline arterial compliance and β -stiffness index between the intervention and control groups (Figure). Carotid arterial compliance decreased after 2 months of resistance training ($P<0.01$). No further decreases in arterial compliance occurred between 2 and 4 months of resistance training. After the detraining period, the reduced arterial compliance values returned to the baseline level. Alterations in arterial compliance were primarily a result of changes in arterial distension, because carotid pulse pressure remained unchanged. In general, qualitatively similar results (although inverse in direction) were obtained by use of β -stiffness index ($P<0.01$). There were no



Changes in carotid arterial compliance (top) and β -stiffness index (bottom) in the intervention group (black circles) and control group (white triangles). Values are mean±SEM. * $P<0.05$ vs baseline; † $P<0.05$ vs resistance training period (2- and 4-month values).

changes in arterial compliance or β -stiffness index in the control group throughout the 8-month period. Femoral arterial compliance, an index of the compliance of peripheral muscular artery, did not change. In both groups, there were no significant changes in brachial and carotid blood pressures, carotid augmentation index, carotid IMT, and carotid lumen diameter (Table 2).

There were no significant changes in resting heart rate and stroke volume throughout the study period (Table 2). Resistance training increased LV wall thickness, LV mass index, and LV hypertrophy index ($P<0.001$). The values returned to the baseline levels during the detraining period, because there was no longer a significant difference from the baseline values. In the intervention and control groups, changes in carotid artery compliance during resistance training and detraining periods were significantly and negatively related to the corresponding changes in LV hypertrophy index ($r=0.68$, $P<0.001$) and LV mass index ($r=0.56$, $P<0.001$). There was no significant association between changes in carotid IMT and LV hypertrophy index ($r=0.17$, $P>0.05$).

Discussion

The salient findings of the present study were as follows. First, a few months of resistance training significantly

TABLE 2. Hemodynamic and Cardiovascular Indices

Variable/Group	Baseline	After Training	After Detraining	Interaction
Heart rate, bpm				
Control	57±2	56±2	57±2	F=0.218
Intervention	55±2	54±2	53±2	P=0.805
Brachial systolic BP, mm Hg				
Control	118±3	120±2	120±2	F=0.324
Intervention	116±3	116±3	116±3	P=0.728
Brachial diastolic BP, mm Hg				
Control	69±2	72±2	73±1	F=2.487
Intervention	69±1	66±1	70±2	P=0.093
Brachial mean BP, mm Hg				
Control	87±2.3	89±1.5	90±1.5	F=0.988
Intervention	85±1.6	84±1.9	87±2.2	P=0.379
Carotid systolic BP, mm Hg				
Control	100±2	103±2	103±1	F=1.477
Intervention	103±3	104±2	102±3	P=0.238
Carotid intima-media thickness, mm				
Control	0.49±0.01	0.52±0.02	0.50±0.02	F=0.400
Intervention	0.47±0.01	0.52±0.02	0.51±0.01	P=0.677
Carotid lumen diameter, mm				
Control	5.91±0.11	5.94±0.14	6.02±0.12	F=0.496
Intervention	5.87±0.12	5.98±0.11	6.00±0.10	P=0.612
IMT/lumen diameter, mm/mm				
Control	0.084±0.002	0.087±0.004	0.085±0.002	F=0.380
Intervention	0.084±0.003	0.088±0.004	0.084±0.004	P=0.686
Carotid augmentation index, %				
Control	-19±3	-18±3	-16±3	F=0.979
Intervention	-18±3	-13±3	-16±2	P=0.382
Femoral artery compliance, mm ² /mm Hg				
Control	0.09±0.01	0.09±0.01	0.08±0.01	F=0.180
Intervention	0.10±0.01	0.10±0.02	0.09±0.01	P=0.836
Stroke volume index, mL/kg				
Control	1.19±0.03	1.26±0.05	1.26±0.09	F=1.150
Intervention	1.21±0.06	1.20±0.06	1.21±0.57	P=0.326
LV mass index, g/kg				
Control	3.1±0.2	3.1±0.2	3.1±0.2	F=15.912
Intervention	2.8±0.1	3.4±0.1*	2.9±0.1†	P<0.0001
LV hypertrophy index, mm/mm				
Control	19±1	19±1	19±1	F=22.432
Intervention	18±1	21±1*	18±1†	P<0.0001

BP indicates blood pressure; LV, left ventricular.

**P*<0.05 vs Baseline; †*P*<0.05 vs After Training.

reduces central arterial compliance in healthy men. Second, the reduced arterial compliance returned to the baseline levels a few months after the cessation of resistance training, confirming that the change in central arterial compliance was indeed an effect of resistance training per se. Third, effects of resistance training on the compliance of the peripheral muscular artery (ie, femoral artery) were not apparent, indicating that the effect of resistance training involves only central elastic arteries

whose cushioning function dampens fluctuations in pressure and flow. Fourth, changes in central arterial compliance induced by resistance training and detraining were significantly associated with corresponding structural changes in LV. Thus, in marked contrast to the beneficial effect of regular aerobic exercise that we observed on arterial compliance, the present findings are not consistent with the idea that resistance training exerts beneficial influences on arterial wall buffering functions.

Previous cross-sectional studies found that individuals who performed resistance training on a regular basis demonstrated lower levels of arterial compliance than their sedentary peers.^{12,20} Because the cross-sectional nature of these observations precluded us from attributing the observed group difference to the effects of resistance training per se, we performed the present intervention study. Consistent with the previous cross-sectional findings,^{12,20} in the present study, several months of resistance training induced $\approx 20\%$ reductions in carotid arterial compliance. Moreover, to isolate the effects of resistance training on arterial compliance as much as possible, we also implemented the detraining program at the conclusion of resistance training. We reasoned that if the changes in arterial compliance were mediated by resistance training, such changes should return to the baseline level when the stimuli of daily resistance exercise were removed. Indeed, during the detraining period, arterial compliance, which was reduced with resistance training, was reversed to the baseline values. Taken together, these results would further support the view that resistance training reduces central arterial compliance.

It is generally thought that arterial compliance is a relatively static measure and that it would take years to change the elastic properties of arteries. In marked contrast to this prevailing thought, arterial compliance has a large reserve and can be altered over a much shorter period, even acutely.^{21,22} In the present study, we observed an $\approx 20\%$ reduction in central arterial compliance in the initial 2 months of resistance training, and no further changes were observed between 2 and 4 months of the exercise intervention. The magnitude of the reduction in arterial compliance achieved in the present intervention study is similar to $\approx 20\%$ difference in arterial compliance between sedentary and resistance-trained young men that we observed in our previous cross-sectional study.¹² These results are consistent with previous pharmacological studies²³ that, in contrast to the prevailing thought, arterial compliance can be altered over a relatively short time period.

It is not clear what physiological mechanisms explain the arterial stiffening with resistance training. During each bout of resistance exercise, arterial blood pressure is known to increase to as high as $\approx 320/250$ mm Hg.²⁴ These acute intermittent elevations in arterial blood pressure during resistance exercise may have altered the arterial structure and/or the arterial load-bearing properties of collagen and elastin,²⁵ thereby causing arterial stiffening. Although there were no changes in carotid artery IMT or IMT/lumen ratio, it would not exclude the possibility of some qualitative changes within the arterial wall (eg, fracture of elastic lamellae). Intense resistance training is also known to be a strong stimulus to increase sympathetic nervous system activity,^{26,27} which may have acted to reduce arterial compliance by providing chronic restraint on the arterial wall via greater sympathetic adrenergic vasoconstrictor tone.²⁸ However, because the influence of sympathetic vasoconstrictor tone would be expected to be greater in peripheral muscular arteries, the preferential changes observed in central versus peripheral artery in the present study argue against this possibility. Other potential mechanisms may include impaired endothelial function²⁹ and

increased formation of collagen cross-linking and advanced glycation end products in arterial wall.³⁰ Because we observed effects of resistance training only on the central elastic artery (carotid artery) but not on the peripheral muscular artery (ie, femoral artery), it is also possible to hypothesize that some mechanical/physical factors may have interacted to reduce arterial compliance. Future studies will be needed to determine the physiological mechanisms underlying the influence of resistance training on central arterial compliance.

The traditional view on the mechanism underlying LV concentric hypertrophy is that the intermittent pressor responses during weight-lifting sessions increase cardiac afterload and LV wall tension, resulting in LV hypertrophy.³¹ However, because the training bouts last for only brief periods per day, it is possible that other more chronic factors may be responsible for LV hypertrophy. Previous studies conducted on hypertensive individuals have indicated that arterial stiffness may be causally linked with LV hypertrophy index via its influence on afterload.³² Consistent with these observations, changes in LV mass and LV hypertrophy index with resistance training were significantly associated with changes in arterial compliance in the present study. These results raise the possibility that central arterial stiffening induced by resistance training may contribute, at least in part, to the concentric LV hypertrophy.

It may be feared that our present findings may discourage the practice of resistance training. We should emphasize, however, the important difference between the training protocol used in the present study and those recommended by the major health organizations.^{9,10} The intensity, volume, and frequency of the resistance training used in the present study were much greater than those recommended for the comprehensive health programs.^{9,10} In light of the role of resistance training on the maintenance of functional ability and the prevention of osteoporosis, the "properly prescribed" resistance training should still be highly encouraged, particularly for older adults. Our present study raises a caution when heavy and strenuous weight training is to be prescribed especially to high-risk populations.

In summary, in marked contrast to the beneficial effect of aerobic training, several months of resistance training reduces the central arterial compliance in healthy men. The reduction in arterial compliance returned to baseline levels during the subsequent detraining period, confirming that the change in central arterial compliance was because of the effect of resistance training per se. In addition, structural changes in the LV induced by the resistance training and detraining were associated with corresponding changes in central arterial compliance. The underlying physiological mechanisms and clinical implications of these findings warrant further investigation.

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