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Greater Age-Related Reductions in Central Arterial Compliance in Resistance-Trained Men

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Abstract—Reductions in the compliance of central arteries exert a number of adverse effects on systemic cardiovascular function and disease risk. Using the cross-sectional study design, we determined the relation between chronic resistance training and carotid arterial compliance. A total of 62 healthy normotensive men, 20 to 39 years of age (young) and 40 to 60 years of age (middle-aged), who were either sedentary or resistance-trained, were studied. In both activity groups, carotid arterial compliance (simultaneous ultrasound and applanation tonometry) was lower ($P<0.05$) in the middle-aged compared with the young men. There was no significant difference between young sedentary and resistance-trained men. In the middle-aged group, carotid arterial compliance in the resistance-trained men was $\approx 30\%$ lower ($P<0.01$) than their sedentary peers. Femoral artery compliance and arm pulse wave velocity (measures of peripheral artery stiffness) were not different among any groups. Left ventricular hypertrophy index (echocardiography) was greater ($P<0.05$) in resistance-trained compared with sedentary men and was associated with carotid arterial compliance ($r=-0.35$; $P<0.01$). We concluded that (1) resistance training is associated with the smaller central arterial compliance in healthy middle-aged men; (2) age-related reductions in arterial compliance was greater in resistance-trained men than in sedentary men; and (3) the lower arterial compliance in the resistance-trained men is associated with left ventricular hypertrophy. In marked contrast to the beneficial effect of regular aerobic exercise, the present findings are not consistent with the idea that resistance training exerts beneficial influences on arterial wall buffering functions.

Key Words: aging arterial elasticity exercise ultrasonography

The compliance of central arteries buffers pulsatile ventricular output through mechanical distention during systole and the elastic recoil during diastole. This compliance function effectively converts the pulsatile flow at the level of the aorta to continuous flow in the capillaries.1 Reductions in this buffering action, as seen in sedentary aging, exert a number of adverse effects on systemic cardiovascular function and disease risk, including elevations in systolic blood pressure and pulse pressure, increased aortic impedance and left ventricular (LV) wall tension, and a reduction in arterial baroreflex gain.2–4 As such, decreased central arterial compliance has been identified as an independent risk factor for future cardiovascular disease.5

Regular physical activity is regarded as an important component of prevention and treatment of age-related increases in cardiovascular disease.6 Recent findings from our laboratory indicate that regular aerobic exercise attenuates age-associated reductions in central arterial compliance and partially restores compliance in previously sedentary middle-aged and older adults.7 One remaining and often asked question is whether regular resistance training is also associated with the favorable effect on arterial compliance. Before 1990, this training modality was emphasized only as a means to develop muscular strength, power, and muscle mass.8,9 In recent years, however, statements on physical activity by various health organizations10–15 have recommended resistance training as part of a preventive and rehabilitative program of physical activity. These recommendations are based primarily on the documented impact of resistance training on the attenuation of osteoporosis and sarcopenia13,16 as well as on the emerging evidence indicating associations between resistance training and metabolic risk factors. Given this, it is reasonable to hypothesize that regular resistance exercise would be associated with increased arterial compliance. However, a recent cross-sectional study found that young men who performed resistance training on a regular basis demonstrated lower levels of systemic arterial compliance than did their sedentary peers17 and suggests that we may not observe the similar trend for resistance training. Moreover, older individuals are at greater risks for develop-
ment of cardiovascular disease as well as for having functional disability associated with sarcopenia (age-related loss of muscle mass and strength), and resistance training is being strongly recommended as a preventive intervention for functional capacity with advancing age. As such, it is important to understand the interaction between age and resistance training for this key cardiovascular risk factor/function.

Accordingly, the primary aim of the present cross-sectional study was to determine the relation between resistance training and central arterial compliance. Based primarily on the only available data on this topic, we hypothesized that regular strength exercise would be associated with smaller central arterial compliance in any given age. Because age exerts an independent effect in reducing arterial compliance in humans, we further hypothesize that an interaction whereby age and resistance training combined would result in a greater suppression of central arterial compliance.

Methods

Subjects
A total of 62 healthy men 20 to 39 years of age (young) and 40 to 60 years of age (middle-aged) participated in the study (Table 1). The sedentary subjects were recruited through various forms of advertisements and had not participated in a regular exercise program for at least the previous 2 years. The resistance-trained men were recruited from various fitness clubs and had been performing vigorous resistance training for >2 years. The average 1-repetition maximum for the bench press for young and middle-aged resistance-trained men was 115±3 kg, respectively, and was not significantly different (P=0.07). Average years of training were 5.0±1.2 and 21.3±2.8 years in young and middle-aged resistance-trained men. To better isolate the effect of resistance exercise training, those who had been concurrently performing regular aerobic exercise (ie, cross-training) were excluded. All subjects were normotensive (<140/90 mm Hg), nonobese, and free of overt chronic diseases as assessed by medical history, physical examination, and complete blood chemistry and hematological evaluation. Men >40 years of age were further evaluated by ECG at rest and, along with blood pressure, during incremental treadmill exercise performed to exhaustion. Candidates who smoked in the past 4 years or were taking medications, anabolic steroids, or other performance-enhancing drugs or had significant intima-media thickening, plaque formation, and/or other characteristics of atherosclerosis were excluded. All subjects gave written informed consent to participate. All procedures were reviewed and approved by the Human Research Committee.

Measurements

Before they were tested, subjects abstained from caffeine and fasted for at least 4 hours (a 12-hour overnight fast was used for determination of metabolic risk factors). Subjects were studied 20 to 24 hours after their last exercise training session to avoid the immediate (acute) effects of exercise, but they were still considered to be in their normal (ie, habitually exercising) physiological state.

Arterial Compliance

Subjects were studied under quiet resting conditions while they were in the supine position. 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. Common 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 cecal portion of the common carotid artery was acquired 1 to 2 cm proximal to the carotid bulb, with the transducer placed at a 90 degree angle. To assess the age-related and physical activity–related differences in peripheral artery compliance, the same procedure was repeated on the common femoral artery. The computer images were digitized with a video frame grabber (DT-3152, Data Translation) and 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 or conditions of the subjects. Time points that corresponded with maximal systolic expansion of the carotid artery and basal (minimum) diastolic relaxation were selected.

### Table 1. Selected Subject Characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Young (n=17)</th>
<th>Resistance-Trained (n=16)</th>
<th>Middle-Aged (n=15)</th>
<th>Resistance-Trained (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>27±2</td>
<td>29±1</td>
<td>51±1*</td>
<td>51±2*</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176±3</td>
<td>176±2</td>
<td>176±1</td>
<td>174±2</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77.0±4.2</td>
<td>83.5±3.4</td>
<td>84.0±2.8</td>
<td>82.1±2.1</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>24±2</td>
<td>17±2†</td>
<td>26±2†</td>
<td>19±2†</td>
</tr>
<tr>
<td>Lean body mass, kg</td>
<td>60.0±3.4</td>
<td>67.7±1.6†</td>
<td>61.6±1.5</td>
<td>66.5±2.2†</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.91±0.03</td>
<td>0.90±0.02</td>
<td>0.97±0.01</td>
<td>0.91±0.02†</td>
</tr>
<tr>
<td>Brachial systolic BP, mm Hg</td>
<td>115±3</td>
<td>116±3</td>
<td>118±3</td>
<td>121±3</td>
</tr>
<tr>
<td>Brachial diastolic BP, mm Hg</td>
<td>65±2</td>
<td>65±2</td>
<td>74±2</td>
<td>72±2</td>
</tr>
<tr>
<td>Brachial mean BP, mm Hg</td>
<td>83±2</td>
<td>84±2</td>
<td>89±2</td>
<td>90±3</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>56±2</td>
<td>59±2</td>
<td>59±2</td>
<td>56±2</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>4.16±0.26</td>
<td>4.08±0.20</td>
<td>4.75±0.22*</td>
<td>4.74±0.18*</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.41±0.09</td>
<td>1.58±0.30</td>
<td>1.35±0.22</td>
<td>1.34±0.12</td>
</tr>
<tr>
<td>Plasma glucose, mmol/L</td>
<td>4.7±0.1</td>
<td>5.0±0.2</td>
<td>5.5±0.1*</td>
<td>5.6±0.2</td>
</tr>
<tr>
<td>Plasma insulin, µU/mL</td>
<td>6.2±0.9</td>
<td>5.1±0.8</td>
<td>7.0±1.2</td>
<td>6.2±0.8</td>
</tr>
<tr>
<td>VO2max, mL/min per kg</td>
<td>42±2</td>
<td>46±2</td>
<td>35±2*</td>
<td>40±2*</td>
</tr>
</tbody>
</table>

Data are mean±SE. BP indicates blood pressure; and VO2max, maximal oxygen consumption.

*P<0.05 vs young; †P<0.05 vs sedentary of same age group.
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, and TCB-500, Millar Instruments). Because the baseline levels of carotid blood pressure are subjected to hold-down force, the pressure signal obtained by tonometry was calibrated by equating the carotid mean arterial and diastolic blood pressure to the brachial artery value as previously described. The pressure waveforms were also used to obtain carotid augmentation index, which has been proposed as an indicator of the magnitude of wave reflections and arterial stiffness. To calculate femoral artery compliance, we used the blood pressure values obtained at the posterior tibial artery. In our laboratory, the day-to-day coefficients of variation were 2±1%, 7±3%, and 5±2% for carotid artery diameter, pulse pressure, and arterial compliance, respectively.

**Arm Pulse Wave Velocity**

Pulse wave velocity (PWV) was measured from the “foot” of pressure waves recorded at brachial and radial artery as previously described. PWV is calculated from the measurement of the distance divided by pulse transit time. Arm PWV was used as a measure of peripheral arterial stiffness.

**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 transducer, as previously described. Ultrasound images were digitized with a video frame grabber and were analyzed by use of computerized image analysis software. All image analyses were performed by the same investigator, who was blinded to the group assignment of subjects. 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. In our laboratory, this technique has excellent day-to-day reproducibility (coefficient of variation 3±1%) for the carotid IMT.

**LV Dimensions, Mass, and Function**

Echocardiography was used to measure LV dimension, wall thickness, and functions, according to established guidelines. The LV mass was calculated by use of the equation by Devereux et al. The ratio of average LV wall thickness (mean of interventricular septal wall thickness and posterior wall thickness) to LV internal end-diastolic diameter was used as an index of LV hypertrophy.

**Body Composition**

Body composition was determined by using dual-energy x-ray absorptiometry (DPX-IQ, Lunar Radiation) with subjects in the supine position. Waist circumference was measured at the narrowest part of the torso and was used as a measure of total abdominal fat.

**Treadmill Exercise**

To demonstrate that the subjects had not been performing aerobic exercise on regular basis, we measured maximal oxygen consumption during a modified Balke incremental treadmill exercise, as described previously. Oxygen consumption, heart rate, and ratings of perceived exertion (Borg scale) were measured throughout the protocol.

**Metabolic Risk Factors for Coronary Heart Disease**

To determine the relations between central arterial compliance and metabolic risk factors for coronary heart disease, fasting plasma concentrations of cholesterol and glucose were determined by the clinical laboratory affiliated with the General Clinical Research Center, as previously described.

**Brachial Blood Pressure at Rest**

Chronic levels of arterial blood pressure at rest were measured with a semiautomated device (Dinamap XL, Johnson & Johnson) over the brachial artery. Recordings were made in triplicate, with subjects in the upright sitting position, and conformed strictly to American Heart Association guidelines.

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**Carotid arterial compliance of sedentary and resistance-trained men.**

*P<0.05 vs young of same activity group; †P<0.01 vs sedentary of same age group.

**Statistical Analyses**

Statistical analyses were performed with the Statistica software. Data were analyzed by 2-way ANOVA (age×physical activity status) and ANCOVA. In the case of a significant F value, a post hoc test with the Newman-Keuls method identified significant differences among mean values. When young and middle-aged men were compared within the same activity group (eg, bench press), 1-way ANOVA was used to analyze the data. Relations of interest were initially identified by univariate correlational and regressional analysis. Independent relations among the dependent variables were determined by means of partial correlation analysis. All data are reported as mean±SEM. Statistical significance was set a priori at P<0.05 for all comparisons.

**Results**

There were no significant differences in height, body mass, or arterial blood pressure among all 4 groups (Table 1). Body fat and waist/hip ratio were smaller (P<0.01) and lean body mass was higher (P<0.01) in resistance-trained men compared with their age-matched sedentary peers. Although all metabolic risk factors were well within clinically normal levels in all groups, plasma total cholesterol and glucose concentrations were higher (P<0.05) in middle-aged compared with young groups.

In both activity groups, carotid arterial compliance was lower (P=0.002) in middle-aged men compared with the young men (Figure). In the young group, carotid arterial compliance was not different (P=0.09) between the sedentary and the resistance-trained men. In the middle-aged men, carotid arterial compliance in the resistance-trained men was ≈30% lower (P=0.01) than their sedentary peers. As such, when the age-related decrease in arterial compliance was calculated, the magnitude of reduction was greater in resistance-trained compared with the sedentary control subjects (31% versus 21%). Age-related and resistance training–related differences remained significant after adjusting carotid arterial compliance for carotid systolic blood pressure (P<0.05). In general, qualitatively similar results (although inverse in direction) were obtained by use of the carotid augmentation index (Table 2). In contrast to the apparent group difference observed in central arteries, arterial distensibilities of peripheral arteries, femoral artery compliance,
and arm PWV were not different among the groups \((P>0.05)\).

There were no group differences in carotid pulse pressure (Table 2). Carotid artery IMT was greater \((P<0.05)\) in middle-aged men compared with the young men, but no statistically significant differences were observed between sedentary and resistance-trained men. Stroke volume, fractional shortening, and ejection fraction were not different among all groups. LV mass and LV hypertrophy index were larger \((P<0.05)\) in resistance-trained men compared with the sedentary peers.

In the overall study population, carotid arterial compliance was related to carotid systolic blood pressure \((r=-0.49)\), carotid pulse pressure \((-0.42)\), carotid IMT \((-0.27)\), and LV hypertrophy index \((-0.35)\) (all \(P<0.01)\). There was a negative and significant association between carotid arterial compliance and years of strength training among resistance-trained adults \((r=-0.66; P<0.01)\). Because of the colinearity between age and years of training, we performed partial correlational analyses. The relation between arterial compliance and years of training remained significant even after age was partialed out \((r=-0.33)\). Moreover, this relation was stronger \((r=-0.71, P<0.05)\) among the young resistance-trained men. LV hypertrophy index was related to carotid artery IMT \((r=0.39, P<0.01)\) and carotid systolic blood pressure \((r=0.28, P<0.05)\). The measures of arterial elasticity were not associated with brachial blood pressure, height, metabolic risk factors, or adiposity.

**Discussion**

The salient findings of this study were as follows. First, central arterial compliance in the resistance-trained middle-aged men were \(\approx30\%\) lower than in their sedentary healthy control subjects. Second, because arterial compliance was not significantly different between sedentary and resistance-trained young men, the age-related decrease in arterial compliance was greater in the resistance-trained compared with the sedentary men. Third, in the pooled population, the LV hypertrophy index was significantly related to carotid artery compliance and carotid IMT, consistent with the concept of the vascular-ventricular coupling on cardiovascular function/structure. These results suggest that in marked contrast to aerobic exercise training, resistance training is associated with a greater age-related reduction in central arterial compliance.

We have previously reported that regular aerobic exercise attenuates age-associated reductions in central arterial compliance and partially restores arterial compliance in previously sedentary middle-aged and older adults. We considered the recent emphasis placed on resistance training for health and disease prevention, we extended our research effort to the modality of resistance training. As an initial approach to answer this question, we used a cross-sectional study design in the present investigation. Because of the well-recognized limitations associated with this design, we attempted to isolate the influence of resistance training as much as possible. To do so, sedentary and resistance-trained men were carefully matched for age, height, brachial blood pressure, and metabolic risk factors. Additionally, in an attempt to isolate the effect of chronic resistance training per se, we excluded those who had been concurrently performing endurance training or those taking anabolic steroids or other performance enhancing drugs. Our present results indicate that chronic resistance training is associated with lower central arterial compliance in healthy middle-aged men. Nevertheless, these results of the present cross-sectional study need to be confirmed prospectively with the exercise intervention study in the future.

This study extends our understanding of the relation between resistance training and arterial compliance in at least two additional ways. First, by establishing that central arterial compliance was not different in resistance-trained and seden-
mentary young men, our findings indicate that the lower levels of arterial compliance observed in middle-aged trained subjects appear to be caused by a greater reduction in arterial compliance with age. Second, in contrast to the central arteries studied, there was no obvious group differences in the stiffness of the peripheral arteries as assessed by femoral arterial compliance and arm PWV, indicating that the effect of resistance training only pertains to central arteries whose cushioning function dampens fluctuations in pressure and flow. The present study is the first to suggest that the age-related reduction in central arterial compliance is greater in resistance-trained men than in sedentary men. The clinical significance associated with the decline in arterial compliance in this population remains to be determined.

What physiological mechanisms might explain the reduced central arterial compliance in resistance-trained men? A likely explanation may be that the acute intermittent elevations in arterial blood pressure in the cardiothoracic region during resistance exercise may result in chronic increases in the smooth muscle content of the arterial wall and the load-bearing properties of collagen and elastin. During each bout of resistance exercise, arterial blood pressure is known to increase to as high as \( \approx 320/250 \) mm Hg. Previous observations in experimental animals indicate that elevations in local distending pressure stimulate smooth muscle hypertrophy and synthesis of extracellular materials in the arterial walls. Indeed, in the current study, central arterial compliance was modestly but significantly associated with carotid IMT. It is a matter of debate as to whether the arterial stiffening and wall hypertrophy associated with resistance training is a beneficial adaptation to strengthen arterial wall against the risk of rupture, a maladaptation that subsequently increases the risk of cardiovascular disease, or both. There are many examples in physiological responses/adaptations in which this appears to be the case. For instance, a fight-or-flight response, associated with increases in sympathetic nervous system activity and epinephrine release, is a beneficial acute response for facilitating blood clot and for minimizing blood loss from the wounds that one may incur during the fight. But if sustained, it may lead to increases in arterial blood pressure and the development of atherosclerosis.

Another possible physiological mechanism is that the higher sympathetic nervous system activity in resistance-trained men may have acted to reduce arterial compliance by providing chronic restraint on the arterial wall through greater sympathetic adrenergic vasoconstrictor tone. Other potential mechanisms may include increased formation of advanced glycation end-products and collagen cross-linking in the arterial wall. One may argue that all of the mechanisms described above should be present in both young and middle-aged populations and that reductions in arterial compliance should have been observed in both populations. We speculate that the degree of adaptation may be greater in the middle-aged men because they have simply been lifting weights for a longer duration. Indeed, among the resistance-trained men, arterial compliance was significantly and negatively associated with years of resistance training. Alternatively, the nature of the adaptive process (e.g., collagen and other extracellular matrix protein deposition in the arterial walls) might be greater because their arteries had already undergone some changes by virtue of their older age. Future studies will be needed to determine the physiological mechanisms underlying the influences of habitual resistance exercise on central arterial compliance.

Consistent with previous studies, we observed a greater LV mass and LV hypertrophy index in resistance-trained men versus age-matched sedentary peers. The mechanism underlying LV concentric hypertrophy in the resistance-trained men was thought to be the intermittent marked pressor responses during vigorous resistance-training exercise sessions acting to increase cardiac afterload and LV wall tension. However, because such conditions last for only brief periods per day, it is plausible to speculate that other factors may be associated with LV concentric hypertrophy. Previous studies conducted on hypertensive individuals have indicated that arterial stiffness may be causally linked with LV hypertrophy index. Our findings extend these concepts gained in clinical medicine to the functional status of resistance training in healthy humans. Specifically, in the current study, we found that middle-aged resistance-trained men demonstrated a lower arterial compliance and a higher LV hypertrophy index. Moreover, central arterial compliance was significantly and inversely related to the LV hypertrophy index. Taken together, these results raise the possibility that chronic stiffening of the central arteries may contribute, at least in part, to the concentric LV hypertrophy observed in healthy resistance-trained men. Our present findings are consistent with the concept of “vascular-ventricular coupling” in that morphological and functional changes in the left ventricle and peripheral vasculature are closely coupled.

Perspectives
In marked contrast to our previous findings involving regular aerobic exercise, the current results indicate that chronic resistance training is associated with lower rather than higher central arterial compliance in middle-aged men. Additionally, age-related reductions in arterial compliance appear to be greater in resistance-trained than sedentary men. The lower arterial compliance observed in resistance-trained men is further related to LV concentric hypertrophy, presumably through vascular-ventricular coupling. The underlying physiological mechanisms and clinical implications of these findings warrants further investigation.

Acknowledgments
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References