Acute effects of resistance exercise on arterial compliance

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DeVan, Allison E., Maria M. Anton, Jill N. Cook, Daria B. Neidre, Miriam Y. Cortez-Cooper, and Hirofumi Tanaka. Acute effects of resistance exercise on arterial compliance. J Appl Physiol 98: 2287–2291, 2005. First published February 17, 2005; doi:10.1152/japplphysiol.00002.2005.—Decreased central arterial compliance is an emerging risk factor for cardiovascular disease. Resistance training is associated with reductions in the elastic properties of central arteries. Currently, it is not known whether this reduction is from one bout of resistance exercise or from an adaptation to multiple bouts of resistance training. Sixteen healthy sedentary or recreationally active adults (11 men and 5 women, age 27 ± 1 yr) were studied under parallel experimental conditions on 2 separate days. The order of experiments was randomized between resistance exercise (9 resistance exercises at 75% of 1 repetition maximum) and sham control (seated rest in the exercise room). Baseline hemodynamic values were not different between the two experimental conditions. Carotid arterial compliance (via simultaneous B-mode ultrasound and applanation tonometry) decreased and β-stiffness index increased (P < 0.01) immediately and 30 min after resistance exercise. Immediately after resistance exercise, carotid systolic blood pressure increased (P < 0.01), although no changes were observed in brachial systolic blood pressure at any time points. These measures returned to baseline values within 60 min after the completion of resistance exercise. No significant changes in these variables were observed during the sham control condition. These results indicate that one bout of resistance exercise acutely decreases central arterial compliance, but this effect is sustained for <60 min after the completion of resistance exercise.

arterial stiffness; carotid artery; weight training

CARDIOVASCULAR DISEASE is the leading cause of death in the United States and most industrialized countries, and the stiffening of central or cardiothoracic arteries is an emerging risk factor for cardiovascular disease (5, 12). Decreases in the elastic properties of the arteries reduce the buffering capacity of the arteries, leading to increased pulse pressure, aortic impedance, and left ventricular wall tension (11, 19, 25, 27, 29), all of which augment the workload of the heart. In addition, the vascular structure of the carotid sinus determines the deformation and strain on arterial baroreceptor endings during changes in arterial blood pressure. Because of this, reduced arterial compliance is associated with impaired arterial baroreflex regulation of heart rate (16).

Our laboratory has previously demonstrated that aerobic exercise training increases the elastic properties of central arteries (26, 28). In contrast, recent studies indicate that central arterial compliance in young and middle-aged resistance-trained men is decreased compared with sedentary age-matched controls (3, 14). Similarly, central arterial compliance decreases after 4 mo of resistance training in young and middle-aged adults (15). Interestingly, in this study, a reduction in arterial compliance was achieved in the initial 2 mo of resistance training, and no further changes were observed between month 2 and month 4 of the exercise intervention (15). The ability to modify arterial compliance over such a short period of time is thought to be due to the modulation of the contractile states of the vascular smooth muscle cells in the arterial wall (4). If such mechanisms are involved in modulating the chronic effects of resistance exercise on arterial compliance, it is plausible to hypothesize that an acute bout of resistance exercise would alter arterial compliance as well. In fact, plasma norepinephrine levels are elevated after an acute bout of resistance exercise, giving rise to the possibility that sympathetic vasoconstrictor tone may also be elevated after exercise (17). However, it is currently unknown whether arterial compliance is reduced after one bout of resistance exercise and, if so, how long the acute effects of a resistance exercise bout persist.

Accordingly, the primary aim of this investigation was to determine the acute effects of one bout of resistance exercise on central arterial compliance. We hypothesized that acute resistance exercise would decrease central arterial compliance and that the postulated decrease in arterial compliance would gradually return to baseline levels after the completion of resistance exercise.

METHODS

Subjects. Sixteen apparently healthy sedentary or recreationally active adults (11 men and 5 women) aged 23–35 yr were studied (Table 1). All subjects were nonsmokers, nonobese, and free of overt cardiovascular or other chronic diseases as assessed by medical history. None of the subjects were taking cardiovascular-acting medications. Activity status was documented by the Godin physical activity questionnaire (7), and subjects had not performed resistance exercise in the past 6 mo. The Human Research Committee reviewed and approved all procedures, and written, informed consents were obtained from all subjects.

Procedures. At least 1 wk before the main experimental sessions, body composition was measured by dual-energy X-ray absorptiometry (Lunar DPX, General Electric Medical Systems, Fairfield, CT), one repetition maximums were obtained for nine resistance exercises (1), and upright seated brachial blood pressure measurements were taken after 5–10 min of seated rest.

The order of the two testing sessions, resistance exercise and sham control (quiet seated rest in the exercise room), were randomized. Before each testing session, subjects were at least 4-h fasted, and female subjects were in the early-follicular phase of their menstrual...
cycle. In an attempt to control for dietary intake and the associated differences in hormonal milieu, diet before the second testing session was reproduced the 3 days before the last testing session. Both sessions were initiated around the same time of day to minimize possible diurnal changes in the dependent variables.

Brachial blood pressure was measured two to three times using the oscillometric pressure sensor method after subjects had rested in the supine position at least 10 min in a quiet, dimly lit, temperature-controlled room. Oscillometric blood pressure measurements have been found to be valid (13), and reproducibility of resting blood pressure measurements using this procedure in our laboratory is excellent. The coefficient of variation for two trials performed 1 wk apart is 1% for systolic blood pressure. The correlation coefficient between the trial one and trial two values is $r = 0.98$. Arterial stiffness and arterial compliance were measured as previously described (15). Briefly, while imaging subject’s left common carotid artery by B-mode ultrasound (HD1 5000CV, ATL Instruments, Bothel, WA), blood pressure was obtained by applanation tonometry (Millar TCB-500, Millar Instruments, Houston, TX) secured by the neck collar in the right common carotid artery. Because 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 as previously described (2). These calibrations were performed at each measurement time point. After these preexercise measures, subjects either sat quietly in the exercise room for 30–45 min or performed a weight training session consisting of one warm-up set of 8–12 repetitions at 50% of one repetition maximum and one set to exhaustion at 75% of one repetition maximum for nine exercises in the following order: seated row, seated chest press, leg press, seated overhead press, seated biceps curl, leg extension, seated leg curl, standing triceps extension, and seated calf raise. After this, the preexercise measurements described above were repeated immediately after (5–10 min post-sham control or postexercise) and 30, 60, 90, 120, and 150 min post-sham control or postexercise.

Ultrasound images were transferred to digital viewing software (Access Point 2000, Freeland, Westfield, IN) where pulsatile changes in carotid artery diameters were analyzed 1–2 cm past the carotid bulb as described previously (15). Carotid artery blood pressure waveforms were analyzed at each time point by waveform browsing software (WinDaq Waveform Browser Software, 2000, Dataq Instruments, Akron, OH). A combination of ultrasound imaging at the common carotid artery with simultaneous applanation of tonometrically obtained arterial pressures from the contralateral carotid artery permitted noninvasive determinations of arterial compliance. In addition to arterial compliance, we also calculated β-stiffness index, which provides an index of arterial compliance adjusted for distending pressure (9, 30). Arterial compliance and β-stiffness index were calculated using the following equations: $[(D_1 - D_0)/D_0]/[2(P_1 - P_0)] \times \pi \cdot D_0^2$ and $(\ln P_1/P_0)/[(D_1 - D_0)/D_0]$, where $D_1$ and $D_0$ are the maximum and minimum carotid diameters measured from intima to intima, and $P_1$ and $P_0$ are the highest and lowest blood pressure at the carotid artery after adjusting for hold-down force. To eliminate interinvestigator variability, one investigator analyzed all arterial images and blood pressure waveforms. At each time point, an average of 15 heart cycles were analyzed.

**RESULTS**

Baseline hemodynamic values were not different between the two conditions. Carotid arterial compliance and β-stiffness index (Fig. 1) after resistance exercise were both different from baseline measures immediately and 30 min post-sham control or postexercise ($P < 0.005$). These values returned to baseline by 60 min after resistance exercise. Mean arterial blood pressure was not significantly different from baseline values at any time point (Table 2). Heart rate increased (from 8 to 16 beats/min), and diastolic blood pressure decreased immediately and 30 min postexercise. Brachial systolic blood pressure did not change significantly (Fig. 2), whereas carotid artery systolic blood pressure increased ($P < 0.001$) immediately after resistance exercise. No significant changes were observed in any variables in the sham control condition except for a decrease in heart rate immediately, 30, 60, and 90 min post-sham control or postexercise.

**DISCUSSION**

The primary findings of this study are as follows. First, central arterial compliance was decreased immediately and 30 min after resistance exercise. These measures returned to

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SE</th>
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<tbody>
<tr>
<td>Men/women</td>
<td>11/5</td>
</tr>
<tr>
<td>Age, yr</td>
<td>27±1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172±3</td>
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<tr>
<td>Body mass, kg</td>
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<td>Body fat, %</td>
<td>27±1</td>
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<td>Physical activity score, units</td>
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<tr>
<td>Brachial systolic BP, mmHg</td>
<td>118±2</td>
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<tr>
<td>Brachial diastolic BP, mmHg</td>
<td>73±2</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>67±3</td>
</tr>
</tbody>
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BP, blood pressure.
baseline levels within 60 min after resistance exercise. These results suggest that the increased central arterial stiffness and reduced central arterial compliance previously observed in resistance-trained adults may be due to the chronic effects of resistance exercise training. Second, one bout of resistance exercise was associated with elevations in carotid systolic blood pressure, although there were no significant changes in brachial systolic blood pressure. These results suggest that measurements of central blood pressure may reveal the vascular effects of resistance exercise that may not be unmasked by routine brachial blood pressure assessments.

In the present study, measures of arterial compliance decreased after resistance exercise, but these measures were no longer different from baseline values 60–150 min after resistance exercise. Thus the acute effects of resistance exercise on the vasculature appear to be transient in nature, lasting no longer than 1 h. In previous intervention studies, which found decreased central arterial compliance after chronic weight training, arterial stiffness was assessed 24–48 h after the last bout of resistance exercise (15). Taken together, these results suggest that the lower arterial compliance or higher arterial stiffness previously observed in those who habitually engage in weight training seem to be a result of the chronic effects of daily resistance exercise.

It is not clear why arterial stiffness is elevated acutely after resistance exercise. It is possible that the increase in central arterial stiffness and decrease in central arterial compliance with one bout of resistance exercise may be due to an epiphenomenon of corresponding blood pressure changes. However, mean arterial blood pressure, which is used to display the pressure-compliance relation, did not change significantly, and there was an ~5% reduction in carotid distention following the resistance exercise bout. Additionally, the decrease in the elastic properties of the carotid artery was also observed even when β-stiffness index, which provides an index of arterial compliance adjusted for distending pressure (9, 30), was used to represent these changes. Other possibilities include increased sympathetic adrenergic vasoconstrictor tone (6, 22, 23) and/or impaired endothelial function (31), both of which were not measured in the present study.

In the present study, brachial blood pressure responses after one bout of resistance exercise are similar to other studies of sedentary and recreationally active adults (18, 24). Two sets of 8–12 repetitions for 12 resistance exercises had no significant effect on brachial systolic or mean blood pressure during the
24 h after resistance exercise when compared with control values (24). Our present study is consistent with these previous observations. A novel finding of the present study is that, although peripheral systolic blood pressure at the brachial artery remained constant, central systolic blood pressure was significantly elevated above baseline levels immediately after resistance exercise. O’Rourke (19) argued that the effects of disease states or drugs on the vasculature may be underappreciated when one relies solely on brachial blood pressure measurements. For example, it has been reported that nitroglycerin causes substantial reductions in aortic systolic pressure (>10 mmHg via direct intra-aortic blood pressure measurements) without causing any significant alterations in brachial systolic blood pressure (20). Similarly, in response to lower body negative pressure, carotid blood pressure displayed twice as much change as brachial blood pressure (21). Our present study extends this concept to the stimuli of resistance training and suggests that measurements of central blood pressure may unmask the vascular effects of resistance exercise that an assessment of brachial blood pressure would not.

In summary, one bout of moderate-intensity full-body resistance exercise increases central arterial stiffness and decreases central arterial compliance in young, healthy adults, but these changes persist <60 min. Therefore, the acute effects of resistance exercise on arterial stiffness are transient in nature, and the reduced elastic properties of central arteries previously observed in resistance-trained adults appear to be due to the chronic effects of resistance exercise training. Further research into the mechanisms, time course, and severity of the resistance training-induced decrease in the elastic properties of central arteries is crucial because many exercise professionals are prescribing resistance training to increase musculoskeletal strength (1, 8, 10). In particular, such exercise prescriptions might be contraindicated for those at risk or who already have cardiovascular disease. With the increasingly accumulating knowledge regarding the effects of resistance exercise on the vasculature, exercise prescriptions can be better tailored to individuals whose aims are to prevent and treat cardiovascular disease.

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REFERENCES

