Effect of whole body resistance training on arterial compliance in young men

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The effect of resistance training on arterial stiffening is controversial. We tested the hypothesis that resistance training would not alter central arterial compliance. Young healthy men (age, 23 ± 3.9 (mean ± s.e.m.) years; n = 28,) were whole-body resistance trained five times a week for 12 weeks, using a rotating 3-day split-body routine. Resting brachial blood pressure (BP), carotid pulse pressure, carotid cross-sectional compliance (CSC), carotid intima–media thickness (IMT) and left ventricular dimensions were evaluated before beginning exercise (PRE), after 6 weeks of exercise (MID) and at the end of 12 weeks of exercise (POST). CSC was measured using the pressure-sonography method. Results indicate reductions in brachial (61.1 ± 1.4 versus 57.6 ± 1.2 mmHg; P < 0.01) and carotid pulse pressure (52.2 ± 1.9 versus 46.8 ± 2.0 mmHg; P < 0.01) PRE to POST. In contrast, carotid CSC, β-stiffness index, IMT and cardiac dimensions were unchanged. In young men, central arterial compliance is unaltered with 12 weeks of resistance training and the mechanisms responsible for cardiac hypertrophy and reduced arterial compliance are either not inherent to all resistance-training programmes or may require a prolonged stimulus.

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Exercise training is (1) used in the treatment and prevention of cardiac events, (2) recommended for preventing muscle atrophy and enhancing quality of life and (3) recommended for the maintainance of independence in the elderly (American College of Sports Medicine Position Stand, 1998; Health Canada, 2003). Arterial compliance, is enhanced following aerobic exercise training (Vaitkevicius et al. 1993; Tanaka et al. 2000). However, the effects of resistance training on the arterial vasculature have been inadequately addressed. Cross-sectional comparisons between sedentary and resistance-trained individuals have shown reduced whole-body arterial compliance (Bertovic et al. 1999), and augmented age-associated central arterial stiffening in resistance-trained athletes (Miyachi et al. 2003). One longitudinal study yielded results supporting reduced arterial compliance with this training method (Miyachi et al. 2004).

The acute cardiovascular responses to resistance exercise demonstrate a rapid increase of blood pressure and heart rate during specific movements, with a return to a relatively low blood pressure between repetitions (MacDougall et al. 1985). In addition, the duration of a resistance-training set is limited in comparison to aerobic exercise and post-exercise hypotension is observed following each set of resistance exercise (MacDonald, 2002).

The aim of the present study was to assess arterial vascular adaptations to 12 weeks of whole body resistance training in young healthy males. We hypothesize that arterial compliance would be unchanged as a result of resistance training as the acute BP response to resistance training is relatively short in duration and small in magnitude.

Methods

Twenty-eight young healthy male subjects with an average age of 23 ± 3.9 years (mean ± s.e.m.) participated in this study. All participants were physically active, but had not
participated in a structured resistance-training protocol for at least 6 months. All subjects were normotensive (<140/90 mmHg), not obese (Table 1) and free from chronic diseases as assessed by medical history, physical examination and blood lipid levels. The McMaster Research Ethics Board approved the experimental protocol and all participants provided written informed consent before participating in the study.

### Resistance training

Participants completed 12 weeks (up to 60 sessions) of whole-body resistance training to induce muscle hypertrophy. Using a 3-day split routine (pushing, pulling and leg exercises), participants completed resistance-training sessions five times per week on a rotating schedule. The pull session consisted of parallel arm pull-down, seated wide grip row, seated narrow grip row, reverse fly and biceps curl. The push session included shoulder press, bench press, vertical bench press, triceps extension and chest flys. Finally, the leg session consisted of extension, curl, incline press and seated calf raises. Participants exercised based on one of the predetermined regimens, either pull, push or legs, on the first day of the week and rotated to the subsequent exercise regimen the following day. This continued throughout the 60 sessions of the overall training protocol. Training was conducted in the fitness centre on the McMaster University Campus. Sessions were held on Monday to Friday. Participants were instructed not to participate in other resistance-type exercises. Also, participants who were aerobically active (two sessions maximum per week) were asked to maintain their activity level, while those not participating in aerobic activity were instructed to abstain from such activities.

Prior to training, participants were familiarized with the resistance-training equipment. Motion-guided machines were employed due to ease of use and adjustment and to minimize potential participant injury. Each training session lasted approximately 1 h. One repetition maximum (1RM) lifts were performed on each apparatus four times: prior to the initiation of training, following the 4th, 8th and the 12th week of training. The 1RM testing performed at each time point was coordinated to coincide with the appropriate day of the 3-day cycle.

### Training sessions

A trainer accompanied participants or was available at all training sessions. Participants were required to complete all exercises at a defined intensity and frequency (Table 2). If a participant was successful in completing more than the required repetitions yet was unable to increase their 1RM, the resistance was increased. Re-assessment of each participant’s 1RM on each apparatus was used to increase the intensity of subsequent exercise sessions. Recovery between exercises and sets was 2 min with the participants performing a warm-up set of 10–12 repetitions at less than 50% of their 1RM prior to all exercises.

### Protocol

Measurements of fat- and bone-free mass were obtained prior to and following the training protocol using dual photon X-ray absorbiometry (DXA: Model

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### Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th>MID</th>
<th>POST</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>82.6 ± 2.4</td>
<td>84.1 ± 2.4</td>
<td>84.8 ± 2.4*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>FBFM (kg)</td>
<td>64.1 ± 1.6</td>
<td>ND</td>
<td>66.4 ± 1.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BMI (kg m(^{-2}))</td>
<td>25.8 ± 0.78</td>
<td>26.3 ± 0.76</td>
<td>26.5 ± 0.78</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>126.3 ± 2.1</td>
<td>122.8 ± 1.7</td>
<td>123.0 ± 1.8</td>
<td>0.12</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>62.9 ± 1.2</td>
<td>61.8 ± 1.3</td>
<td>65.4 ± 1.2†</td>
<td>0.022</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>84.0 ± 1.3</td>
<td>79.4 ± 3.0</td>
<td>79.0 ± 4.0</td>
<td>0.11</td>
</tr>
<tr>
<td>PP (mmHg)</td>
<td>63.3 ± 1.9</td>
<td>59.0 ± 2.4</td>
<td>57.6 ± 2.8*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Resting HR (beats min(^{-1}))</td>
<td>64.5 ± 1.78</td>
<td>65.3 ± 2.0</td>
<td>61.8 ± 1.7</td>
<td>0.08</td>
</tr>
<tr>
<td>Carotid IMT (mm)</td>
<td>4.04 ± 0.08</td>
<td>4.17 ± 0.11</td>
<td>4.15 ± 0.10</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Data are mean ± s.e.m. FBFM, fat and bone free mass; BMI, body mass index; BP, blood pressure, MAP, mean arterial pressure; PP, brachial pulse pressure; HR, heart rate; IMT, intima–media thickness. * \( P < 0.05 \) versus PRE. † \( P > 0.05 \) versus MID.

### Table 2. Progression of the resistance-training programme

<table>
<thead>
<tr>
<th>Week</th>
<th>Number of sets</th>
<th>Number of repetitions</th>
<th>Participant instructions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–2</td>
<td>2</td>
<td>10–12</td>
<td>Focus on form</td>
</tr>
<tr>
<td>3–5</td>
<td>3</td>
<td>10–12</td>
<td>80% of 1RM</td>
</tr>
<tr>
<td>6–7</td>
<td>3</td>
<td>8–10</td>
<td>3rd set to failure 80% of 1RM</td>
</tr>
<tr>
<td>8–10</td>
<td>3</td>
<td>6–8</td>
<td>3rd set to failure, intensity &gt; 80% of 1RM</td>
</tr>
<tr>
<td>11–12</td>
<td>3</td>
<td>5–6</td>
<td>3rd set to failure, intensity &gt; 80% of 1RM</td>
</tr>
</tbody>
</table>

1RM, one repetition maximum.
QDR-1000/W, Hologic Inc., Waltham, MA, USA). Following a familiarization session, arterial compliance, intima–media thickness (IMT), heart dimensions and resting brachial BP were measured prior to the initiation of training, once during the seventh, and the twelfth week of training with the same investigator conducting all the tonometry measurements. The procedures involving BP, cardiac and arterial measurements were conducted using an identical protocol under the same control conditions at all time points. Measurements were obtained a minimum of 24 h following the last training session and at the same time of the day (within ∼2 h). Testing sessions were conducted at the same time of the day for each individual participant. Participants were asked to abstain from nicotine and caffeine for at least 12 h prior to testing and evaluations were made ∼4 h after the consumption of 237 ml of a commercial meal replacement (BOOST, Mead Johnson Nutritional, Ottawa, ON, Canada).

Upon arrival, participants were instrumented with an oscillometric automated BP measurement device (model CBM-7000, Colin Medical Instruments, San Antonio, TX, USA) and instructed to lie motionless for 10 min. Measurements of brachial systolic (SBP), diastolic (DBP), pulse pressure (PP), and calculated mean arterial pressure (MAP = 1/3(PP) + DBP) were obtained in triplicate at intervals of 2 min. As described by Carter et al. (2003), the first measurement of arterial BP from the automated sphygmomanometer is consistently high. Thus, the average of the last two measures was used to determine resting brachial BP.

Central arterial compliance was assessed at the common carotid artery according to the method described by Miyachi et al. (2003). The main difference involved calibration of carotid blood pressure to arterial blood pressure measured continuously using arterial tonometry at the wrist rather than by an arm cuff. Ten measurements of arterial BP and two measurements of arterial diameter change were used to determine cross-sectional compliance. Beat-by-beat changes in BP were obtained in the radial and carotid arteries using two planation tonometry instruments. Continuous automated radial artery BP was measured (CBM-7000, Colin Medical Instruments, San Antonio, TX, USA), while at the carotid artery a manual pen-like device containing a high-fidelity transducer (model SPT-301, Millar Instruments Inc., Houston, TX, USA) was used to obtain continuous BP waveforms. Carotid artery BP was obtained in the right carotid artery while ultrasound images were obtained simultaneously in the left carotid artery.

The pen-like arterial tonometer (Millar) is sensitive to manual hold-down pressure; thus requiring adjustment of the obtained values based on several assumptions (Kelly et al. 1989). Briefly, it was assumed that DBP and MAP are similar in all conduit arteries when an individual is in the supine position (Nichols et al. 1998) while SBP is amplified through the arterial tree. The mean and minimum BP obtained from the carotid waveform were equated to the MAP and DBP of the radial artery. The maximum BP waveform recorded in the carotid artery was then used as an extrapolation point from the calibrated MAP and DBP (Kelly et al. 1989).

Arterial and cardiac images were obtained using B-Mode Ultrasound (System FiV e, GE Medical Systems, Horten, the Netherlands). For arterial images, a 10-MHz linear array probe was positioned longitudinal to the left carotid artery and an image ∼2 cm proximal to the bifurcation that divides the vessel into the external and internal portions of the artery was obtained. Two video clips of one complete heart cycle were obtained and digitally stored at a frame rate of 12 frames/s for off-line analysis of diameter change. Participants were also instrumented with an electrocardiograph (model Cardiomatic MSC 7123, Medical Systems Corp, Miami, FL, USA) for simultaneous recording of R–R intervals. All physiological measurements of HR and BP were input to a data acquisition board (Powerlab model ML795, ADInstruments, Colorado Springs, CO, USA) for analog to digital conversion and stored on a personal computer (IBM Netvista X86 compatible processor, White Plains, NY, USA) using available software (Chart 4.2, ADInstruments) with analog signals sampled at 200 Hz. Digital images obtained with B-mode ultrasound were converted to digital imaging and communication in medicine (DICOM)-compressed JPEG stacked image files for further analysis using automated software (AMS II, Chalmers University of Technology, Göteborg, Sweden).

During semi-automated artery analysis the arterial diameter from leading edge to leading edge was assessed for all frames of the digital video clip. A minimum of 100 measurements of vessel diameter was used to obtain a mean arterial diameter for each frame. The maximum and minimum diameters obtained during the heart cycle were then used to determine diameter change. Subsequently, the two measurements of diameter change were used to calculate cross-sectional compliance (CSC) (Miyachi et al. 2003):

\[
\text{CSC} = \frac{\Delta \text{Area}}{\text{PP}} = \frac{\pi r^2_{\text{max}} - \pi r^2_{\text{min}}}{\text{PP}} = \frac{\pi (d^2_{\text{max}}/2) - \pi (d^2_{\text{min}}/2)}{\text{PP}}
\]
Table 3. One repetition maximum performance

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Pre training</th>
<th>Post training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder press</td>
<td>632.1 ± 33.4</td>
<td>827.7 ± 39.3*</td>
</tr>
<tr>
<td>Bench press</td>
<td>705.8 ± 32.4</td>
<td>1080.3 ± 38.3*</td>
</tr>
<tr>
<td>Lat pull down</td>
<td>662.5 ± 20.6</td>
<td>856.2 ± 25.6*</td>
</tr>
<tr>
<td>Biceps curl</td>
<td>467.9 ± 27.5</td>
<td>728.4 ± 30.5*</td>
</tr>
<tr>
<td>Seated rear deltoid fly</td>
<td>620.3 ± 27.5</td>
<td>894.5 ± 31.5*</td>
</tr>
<tr>
<td>Double leg incline press</td>
<td>2157.7 ± 40.3</td>
<td>3841.6 ± 232.8*</td>
</tr>
</tbody>
</table>

Values are in Newtons (N). Data are mean ± s.e.m. *P < 0.05 versus PRE (n = 28). Lat, Latissimus Dorsi.

where, CSC is cross-sectional compliance, PP is pulse pressure, r is the radius of the artery, d is diameter of the artery, max is the maximal value and min is the minimal value. While \( \beta \)-stiffness index, \( \beta \) was calculated as (O'Rourke et al. 2002):

\[
\beta = \frac{\ln(PP)}{(d_{\text{max}} - d_{\text{min}})/d_{\text{min}}}
\]

IMT was determined through automated determination of the proximal aspect of the intima and the proximal interface of the adventitia based on gradient and intensity differences from pixel to pixel. The average IMT from two images of the common carotid artery were compared at each time point. Left ventricular dimensions and functional characteristic measurements were made in a subset of 19 participants after selection of the most appropriate images available for analysis. Images obtained from nine of the participants were either improperly aligned or of insufficient quality to warrant analysis at one of the three time points. Two B-mode full heart cycle high-resolution (60 Hz) video clips obtained in the parasternal long axis were acquired while subjects were positioned in the left lateral decubitus position. One additional M-mode image was taken according to the American Society of Echocardiography guidelines when the orientation of the heart was appropriate. Digital video clips and appropriate M-mode images were subsequently transferred for off-line analysis using commercially available software (Echopac M-mode, GE Medical Systems, Horten, the Netherlands).

Analysis of heart images involved the use of anatomical M-mode. This feature allows B-mode digital video clips, acquired in the parasternal long axis, to be reconstructed as M-mode images. This enables the inclusion of patients with heart orientations that are not ideal for typical M-mode acquisition. Once anatomical M-mode images were constructed, subsequent analysis was conducted (Sahn et al. 1978). Intra-observer and inter-observer coefficients of variability (CV) ranged from 4.5 to 11% and 6 to 12%, respectively, for various individual cardiac parameters (e.g. intraventricular septum during systole (IVSs)).

Results

Participants had increases in lean tissue and whole body mass following resistance training (Table 1). Whole body lean tissue gains were substantial at 3.5% and similar to previous findings (Tarnopolsky et al. 2001). 1RM performance for all exercises increased (\( P < 0.001 \)); we present data for six exercises that are representative of the range of changes noted following training (Table 3).

Resting arterial BP and heart rate

Brachial systolic BP and MAP did not change throughout training; that is, before beginning exercise (PRE), after 6 weeks of exercise (MID) and at the end of 12 weeks of exercise (POST) (Table 1). However, diastolic BP increased (\( P = 0.02 \)) from MID to POST. PP decreased (\( P = 0.003 \)) from PRE to POST as a result of an increased diastolic BP. This reduced PP was also apparent when assessed at the level of the carotid artery PRE to POST (\( P = 0.03 \)) (Fig. 1A). Resting heart rate was not significantly reduced with training (\( P = 0.08 \)).

Central arterial structure

Carotid artery diameter measured as a mean (PRE, 6.75 ± 0.08; MID, 6.65 ± 0.08; POST, 6.78 ± 0.09 mm), at systole (PRE, 7.11 ± 0.08; MID, 7.02 ± 0.09; POST; 7.08 ± 0.08 mm), or at end diastole (PRE, 6.39 ± 0.08, MID, 6.31 ± 0.09; POST, 6.44 ± 0.09 mm) did not change significantly with training. The change in carotid diameter throughout the cardiac cycle also showed no change with training (PRE, 0.72 ± 0.03; MID, 0.71 ± 0.03; POST, 0.67 ± 0.04 mm). Cross-sectional compliance (CSC) measured at the carotid artery did not change significantly throughout the training (Fig. 1B). Also carotid \( \beta \)-stiffness index (Fig. 1C) showed no significant alterations at the carotid artery. Finally, carotid IMT was not significantly altered.

Cardiac dimensions

Intraventricular septum (IVS), left ventricular internal diameter (LVID), left ventricular posterior wall (PWT), and left ventricular ejection fraction taken at end diastole

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were not different at any time point of training (Table 4). However, these measurements taken during systole showed differences or trends when comparing MID to POST measures. Specifically, PWT during systole was elevated \((P = 0.03)\) at MID compared to POST. There were also trends for reduced left ventricular dimensions (LVD) \((P = 0.06)\), increased IVS \((P = 0.09)\), reduced LVEF \((P = 0.08)\) and percentage fractional shortening (FS) \((P = 0.07)\) at MID.

**Discussion**

The current study is in contrast to a number of reports that have found resistance-exercise training to result in reductions of carotid (Miyachi *et al.* 2004) and whole-body arterial compliance (Bertovic *et al.* 1999). Our most important findings were a reduction in brachial and carotid pulse pressure, no change in central arterial stiffness and no changes in carotid intima–media thickness, left ventricular wall thickness or mass. These findings suggest that short-duration resistance training does not result in artery stiffening as shown in cross-sectional studies (Bertovic *et al.* 1999; Miyachi *et al.* 2003) or a longitudinal study (Miyachi *et al.* 2004).

The effectiveness of the resistance-training protocol in inducing compensatory hypertrophy was evident by the gains of FFM. The gains in strength as evidenced by the increases in 1RM support the effectiveness of the resistance training. Participant adherence to the protocol was ∼98%.

The primary findings related to resting BP were an increase in DBP, a reduction of resting brachial and carotid PP with high-intensity resistance training. The reduction of resting PP is an observation that has not been reported with resistance-exercise training. The importance of this finding stems from the negative impact of augmented PP on cardiac work and vascular function (Dart & Kingwell, 2001). In contrast Bertovic *et al.* (1999) observed an augmented PP both in the carotid and brachial arteries of their resistance-trained group compared to sedentary controls. However, Miyachi *et al.* (2003) did not observe an elevated PP in a subsequent cross-sectional study. Some possible mechanisms include alterations of sympathetic flow to the renal or muscle systems, or alterations of circulating vasoactive agents including angiotensin II and vasopressin. Muscle sympathetic nerve activity differs between resistance-trained athletes and sedentary controls; however, longitudinal resistance training has failed to alter muscle sympathetic nerve activity (Carter *et al.* 2003).

**Arterial compliance and resistance training**

Central arterial compliance is an adaptable attribute of the vascular system; however, the current resistance-training protocol did not affect this variable when measured in the carotid artery as was suggested by Bertovic *et al.* (1999) and Miyachi *et al.* (2003). This finding supports the hypothesis, which predicted no change in the elasticity of the carotid artery.

**Carotid IMT with resistance-exercise training**

The absence of a significant increase in carotid artery IMT with resistance training is expected given the absence of carotid artery stiffening. The age-associated decrease of

![Figure 1. Carotid artery pulse pressure (A) cross-sectional compliance (B) and beta-stiffness index (C) at PRE, MID and POST resistance exercise training](image)

Values are means ± s.e.m. *Different from PRE, \(P < 0.05\).
Carotid arterial compliance noted by Miyachi et al. (2003) was correlated with an augmented IMT, compared with sedentary controls. Therefore, more extensive training may result in increased IMT, which may contribute to reduced carotid or central compliance.

**Cardiac parameters with resistance training**

The relationship between central arterial stiffness and concentric left ventricular hypertrophy in disease populations is established (Roman et al. 2000; Deague et al. 2001). However, concentric left ventricular hypertrophy following resistance-exercise training is an equivocal finding (Haykowsky et al. 2002). Therefore, the suggestion that resistance training causes left ventricular hypertrophy secondary to central arterial stiffening (Miyachi et al. 2003) may not be the case with resistance training.

There was no significant PRE to POST changes of any heart variable; however, notable trends were apparent at MID. These trends may be a result of the resistance-training programme and the alterations of the programme that were incorporated throughout the 12 weeks of training. Adaptations of PWT, IVS and left ventricular internal diameter (LVID) may be dependent on the method of resistance training (Haykowsky et al. 2002). Observations of concentric left ventricular hypertrophy may be specific to body building (lower intensity, higher repetitions), whereas an absence of concentric hypertrophy may be specific to power lifting (i.e. higher intensity, lower repetitions). We followed a progressive design, which could be described as a hybrid programme between that of the typical body builder and that of a power lifter. Therefore, the acute effects of resistance training on left ventricular wall stress may have changed from the beginning to the end of training, contributing to the trends and returns to baseline by the termination of training. Another possible explanation for the aforementioned trends may be the measurement techniques. The relatively large variability (6–12% intra-observer CV) of the cardiac measurements may have contributed to a trend that was not physiological.

Differences between the training protocol implemented by Miyachi et al. (2004) and our study exist with regards to the intensity and periodization. These differences may account for the opposing findings observed. Miyachi et al. (2004) incorporated 4 months of training at a frequency of three times per week. In addition, the various exercises were identical at all training sessions. The training programme in the present study involved five sessions of resistance training per week. Each training session consisted of different exercises (3-day repeating cycle) thus allowing greater recovery for specific muscle groups. The effectiveness of this technique is evident in our strength gains, which are substantially greater than those of Miyachi et al. (2004). The repetitions were reduced and sets to fatigue were incorporated late in the protocol to enhance hypertrophy. These differences indicate that our training protocol was more intense and involved the same number of resistance-training sessions in a shorter period.

It is not known whether the reduced arterial compliance noted in resistance-trained athletes is a maladaptive process that is similar to that occurring in vascular disease. Before conclusions can be drawn as to whether resistance exercise-induced changes in vascular compliance are due to changes that are similar to disease processes (Miyachi et al. 2003), long-term training studies in humans (6–12 months) evaluating cardiovascular risk with resistance training are needed. Furthermore, measuring compliance at rest may not be a valid representation of compliance because during exercise it may be different from rest and different in resistance-trained individuals compared to sedentary controls.

In the present study we did not include a non-exercising control group. To improve the stability of the PRE measures we had a number of familiarization sessions to reduce the variability caused by apprehension that our subjects may have had, which could have affected

![Table 4. Heart morphology](#)

<table>
<thead>
<tr>
<th>Heart parameter</th>
<th>PRE</th>
<th>MID</th>
<th>POST</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVSd (cm)</td>
<td>1.12 ± 0.05</td>
<td>1.20 ± 0.04</td>
<td>1.11 ± 0.04</td>
<td>0.09</td>
</tr>
<tr>
<td>IVSs (cm)</td>
<td>0.872 ± 0.03</td>
<td>0.847 ± 0.04</td>
<td>0.865 ± 0.03</td>
<td>0.79</td>
</tr>
<tr>
<td>LVIDd (cm)</td>
<td>3.73 ± 0.10</td>
<td>3.58 ± 0.09</td>
<td>3.82 ± 0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>LVIDs (cm)</td>
<td>5.22 ± 0.10</td>
<td>5.22 ± 0.08</td>
<td>5.39 ± 0.10</td>
<td>0.43</td>
</tr>
<tr>
<td>PWTd (cm)</td>
<td>1.68 ± 0.04</td>
<td>1.79 ± 0.04</td>
<td>1.67 ± 0.05†</td>
<td>0.03</td>
</tr>
<tr>
<td>PWTs (cm)</td>
<td>1.03 ± 0.04</td>
<td>1.07 ± 0.04</td>
<td>1.02 ± 0.03</td>
<td>0.43</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.63 ± 0.02</td>
<td>0.67 ± 0.02</td>
<td>0.63 ± 0.02</td>
<td>0.08</td>
</tr>
<tr>
<td>FS (%)</td>
<td>28.6 ± 1.3</td>
<td>31.3 ± 1.6</td>
<td>28.8 ± 1.4</td>
<td>0.07</td>
</tr>
<tr>
<td>LVmass (g)</td>
<td>216.6 ± 12.2</td>
<td>216.2 ± 7.7</td>
<td>224.0 ± 8.6</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Data are mean ± s.e.m. s, during systole; d, during diastole; IVS, intraventricular septum diameter; LVID, left ventricular internal diameter; PWT, posterior wall thickness; pctFS, percentage fractional shortening; LVmass, left ventricular mass. †P < 0.05 versus MID.
sympathetic activity. The same investigator also made all measurements, while blinded to condition, of vascular and cardiac variables PRE, MID and POST. Finally, we included a large number of subjects, which resulted in normally distributed data.

**Conclusion**

Resistance-exercise training does not alter central arterial compliance and has no effect on cardiac dimensions. Further, the thickness of the intima–media layer of the artery, which has been correlated with reduced arterial compliance (Miyachi et al. 2003), was not altered with training. Thus, 12 weeks of progressive high-intensity resistance training in healthy young men does not adversely affect carotid artery compliance or left ventricular dimensions.

**References**


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