Progressive Resistance Training Without Volume Increases Does Not Alter Arterial Stiffness and Aortic Wave Reflection

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Endurance exercise is efficacious in reducing arterial stiffness. However, the effect of resistance training (RT) on arterial stiffening is controversial. High-intensity, high-volume RT has been shown to increase arterial stiffness in young adults. We tested the hypothesis that an RT protocol consisting of progressively higher intensity without concurrent increases in training volume would not elicit increases in either central or peripheral arterial stiffness or alter aortic pressure wave reflection in young men and women. The RT group (*n* = 24; 21 ± 1 years) performed two sets of 8–12 repetitions to volitional fatigue on seven exercise machines on 3 days/week for 12 weeks, whereas the control group (*n* = 18; 22 ± 1 years) did not perform RT. Central and peripheral arterial pulse wave velocity (PWV), aortic pressure wave reflection (augmentation index; AIx), brachial flow–mediated dilation (FMD), and plasma levels of nitrate/nitrite (NOx) and norepinephrine (NE) were measured before and after RT. RT increased the one-repetition maximum for the chest press and the leg extension ($P < 0.001$). RT also increased lean body mass ($P < 0.01$) and reduced body fat (%; $P < 0.01$). However, RT did not affect carotid-radial, carotid-femoral, and femoral-distal PWV (8.4 ± 0.2 vs. 8.0 ± 0.2 m/sec; 6.5 ± 0.1 vs. 6.3 ± 0.2 m/sec; 9.5 ± 0.3 vs. 9.5 ± 0.3 m/sec, respectively) or AIx (2.5% ± 2.3% vs. 4.8% ± 1.8 %, respectively). Additionally, no changes were observed in brachial FMD, NOx, NE, or blood pressures. These results suggest that an RT protocol consisting of progressively higher intensity without concurrent increases in training volume does not increase central or peripheral arterial stiffness or alter aortic pressure wave characteristics in young subjects. Exp Biol Med 232:1228–1235, 2007

Key words: arterial stiffness; resistance training; vascular function; nitric oxide; norepinepherine

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Introduction

Cardiovascular disease remains the major cause of mortality in the United States and in other industrialized countries. Increased arterial stiffness is an important determinant of cardiovascular risk (1) and is an independent predictor of cardiovascular events and mortality (2). Aerobic exercise training has been shown in cross-sectional studies to be associated with reduced arterial stiffness and central aortic pressure wave reflections in both young competitive endurance athletes (3) and in older healthy individuals (4). Interventional studies have also demonstrated that aerobic exercise training is efficacious in reducing arterial stiffness and central pressure wave reflection in young (5) and older healthy individuals (4) as well as in patients with coronary artery disease (6). Resistance training (RT) is another exercise modality recommended by the American Heart Association (AHA) (7) and by the American College of Sports Medicine (ACSM) (8) to help prevent osteoporosis, sarcopenia, obesity, and the clustering of cardiovascular risk factors associated with metabolic syndrome (7). However, less is known about the independent effects of RT on arterial function. Cross-sectional studies suggest that chronic, high-intensity, high-volume RT reduces arterial compliance (i.e., increased stiffness) in both young and middle-aged men (9, 10). Interventional studies have yielded conflicting results regarding the effects of RT on arterial function. Miyachi *et al*. (11) reported that 4 months of high-intensity RT decreased central arterial compliance in young healthy men. The same authors later reported that moderate-intensity RT resulted in similar decreases in central arterial compliance (12). Cortez-Cooper *et al*. (13) reported that 11 weeks of high-intensity RT resulted in increases in arterial stiffness and wave reflection in young healthy women. In contrast, Rakobowchuk *et al*. (14) found that central arterial compliance was unaltered after 3 months of RT in young men. However, all of the aforementioned studies used high-intensity and concurrent high-volume RT protocols that are not commonly recommended for the majority of the population.

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Therefore, the primary purpose of the present study was to determine the effects of RT, performed in accordance with AHA and ACSM recommendations, on arterial function in young men and women. Although acute blood pressure (b/p) responses to RT range from 255/190 mm Hg (upper body measurement) to as high as 320/250 mm Hg (lower body measurement) (15), this response is transient; b/p returns to baseline within approximately 1 min and is often followed by a decline in pressure to below prior resting values (16). It is unclear if this transient stimulus elicits chronic vascular adaptations. Indeed, acute resistance exercise has been shown to decrease peripheral arterial stiffness while having no effect on central arterial stiffness (17). We hypothesized that an RT protocol consisting of progressively higher intensity without concurrent increases in training volume would not alter arterial function. To test this hypothesis, we measured central and peripheral pulse wave velocity (PWV), central aortic pressure wave reflection, brachial flow–mediated dilation (FMD), and plasma concentrations of norepinephrine (NE) and nitrate/nitrite (NOx) before and after 12 weeks of RT.

Materials and Methods

A total of 48 healthy men and women were recruited to participate in the study, which investigated the effects of 12 weeks of progressive RT on arterial function. All of the subjects were either sedentary or recreationally active and had not participated in a structured exercise program for at least 6 months before entering the study. Forty-eight subjects were nonrandomly assigned to either the RT group ($n = 30$) that performed two sets of 8–12 repetitions to volitional fatigue on seven exercise machines on 3 days/week for 12 weeks or to an age-matched, nonexercise control group (CON; $n = 18$). None of the subjects were hypertensive (b/p > 140/90), were cigarette smokers, were obese (body mass index > 30 kg/m^2), or were receiving medications. Data were collected and analyzed at the beginning and at the end of exercise training (48–72 hrs after the last RT session). All measurements were performed by the same investigator in a quiet, temperature-controlled room (21°–23°C) when the subjects were in a fasting state of 10–12 hrs. Subjects were asked to abstain from caffeine and alcohol for at least 24 hrs prior to vascular measurements. To avoid potential diurnal variations, all measurements were conducted at the same time of day. All measurements for female subjects were completed in the same phase of their menstrual cycle before and after the intervention and no subjects were taking birth control medications. The study was approved by the University of Florida Health Science Center Institutional Review Board, and all subjects signed written informed consent prior to participating in the study.

Pulse Wave Analysis. Following a 15-min rest period in a supine position, heart rate (HR) and brachial b/p measurements were performed in triplicate in the left arm using an automated, non-invasive b/p cuff (HEM-773, Omron Inc., Bannockburn, IL). An average of the three HR and b/p measurements were used for resting values of each. The assessment of arterial wave reflection characteristics was performed noninvasively, using the SphygmoCor system (AtCor Medical, Sydney, Australia). High-fidelity radial artery pressure waveforms were recorded by applanation tonometry of the radial pulse using a pencil-type micromanometer (Millar Instruments, Houston, Texas). The aortic pressure waveform is derived noninvasively from the radial pulse using applanation tonometry and the application of a generalized transfer function, which corrects for pressure wave amplification in the upper limb (18). The generalized transfer function has been validated using both intra-arterially (19, 20) and noninvasively (21) obtained radial pressure waves. The test-retest reproducibility of this procedure was previously established by others (22). In our laboratory, reproducibility was established previously by triplicate measurement on nonconsecutive days in young, healthy men, with a mean coefficient of variation of 6.5% (23).

The central aortic pressure wave is composed of a forward traveling wave, which is generated by left ventricular ejection, and a reflected wave that is returning to the ascending aorta from the periphery (24). The aortic augmentation index (AIx) is defined as reflected wave amplitude divided by pulse pressure and is expressed as a percentage (25). The forward and reflected waves travel in opposite directions along the artery at the same velocity. The round trip travel time (Δt_p) of the forward traveling wave from the ascending aorta to the major reflection site and back is measured from the foot of the forward traveling pressure wave to the foot of the reflected wave. The Δt_p is inversely related to arterial PWV and arterial stiffness and is directly related to the distance to the reflecting site (24). AIx is an index of wave reflection, which is a manifestation of systemic arterial stiffness. Assessment of central arterial pressure waves is described in detail by Nichols and Singh (24).

PWV. With the subject supine, tonometry transit distances from the suprasternal notch to the radial (SSN-R), femoral (SSN-F), and carotid (SSN-C) sites and from the femoral to the dorsalis pedis (F-DP) recording sites were measured as straight lines between these points on the body surface with a tape measure. To determine PWV, pressure waveforms were recorded at the following three sites sequentially: carotid-radial (C-R), carotid-femoral (C-F), and femoral-dorsalis pedis (F-DP) waveforms. Pressure waveforms were gated with simultaneous electrocardiographs (EKG) and were used to calculate the PWV between the two sites. Foot-to-foot PWV to each peripheral site (DP, R, and F) was calculated by determining the delay between the appearance of the pressure waveform foot in the carotid and peripheral sites (26). The distance between recording sites was adjusted for parallel transmission in the aorta and the carotid by subtracting SSN-C from SSN-R and SSN-F,

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respectively. These corrected distances were divided by the respective foot-to-foot transmission delays (C-R, C-F) to give PWV. Central PWV (in the mostly elastic aorta) was evaluated using the C-F data, and peripheral PWV (in the more muscular conduits) was evaluated using the F-DP and C-R data. The PWV between the various measuring sites was used as an indirect measure of regional arterial stiffness. The reliability of the PWV between the different regions was established by sequential measurement in young, healthy men on 3 separate days. The mean coefficients of variation for C-R, C-F, and F-DP were 4.5%, 2.1%, and 5.3%, respectively (23).

FMD Testing. Brachial artery FMD was assessed noninvasively in the right arm using a high-resolution ultrasound machine (ATL HDI 3000; Advanced Technologies Laboratories, Bothell, WA) equipped with a 10.5MHz transducer, as described originally by Celermajer *et al*. (27). Briefly, resting baseline end diastolic brachial diameters and blood velocity were obtained with the transducer placed 3–5 cm above the antecubital crease. Following baseline measurements, reactive hyperemia was produced by inflating a b/p cuff placed on the upper forearm 1–2 cm below the elbow for 5 mins at 200 mm Hg, followed by a rapid deflation. Blood velocity envelopes were obtained during the first 10 secs following cuff release to help establish the magnitude of the hyperaemic response. The transducer was held in the same position for the duration of cuff inflation to ensure that the same section of the brachial artery was measured before and after cuff inflation. The brachial artery was imaged and recorded for 3 mins following cuff deflation. Ultrasound images were recorded on a super-VHS videocassette for later, off-line, manual analysis using specialized image analysis software (Image Pro; Data Translation Inc., Marlboro, MA) Brachial artery diameters were determined during end diastole by measuring the distance between the near and far walls of the intima. Brachial FMD was expressed as absolute (Δmm) and as a percent increase from baseline (FMD%). In our laboratory, the day-to-day coefficient of variation was 10.4% for peak brachial FMD%.[1]

Because the amount of dilation has been shown to depend on the resultant hyperemic flow stimulus, all measurements of FMD were normalized to the mean shear rate (4 × mean blood velocity/mean diameter). This allowed for proper interpretation of potential baseline group (RT vs. CON) or intervention (pre- vs. post-) differences.

Blood Collection and Analysis. Blood samples were collected from an indwelling venous catheter in either the left or right forearm. Samples were drawn following a 15–20-min equilibration period. Plasma blood samples were used to determine venous levels of NE and NOx. Blood was collected in tubes containing diethylenetriamine pentaacetic, immediately underwent centrifugation at 2000 *g* for 15 mins at 4°C, and then were stored immediately at –80°C until analysis at the end of the study.

Plasma NE was measured using a commercially available competitive enzyme immunoassay (EIA; Labor Diagnostika Nord GmbH & Co. KG, Nordhorn, Germany). The plasma NE concentration was used as an indirect humoral index of autonomic nervous system sympathetic activity. Because NO is rapidly converted to nitrate and nitrite (NOx) in plasma, NOx was used to estimate NO production. Plasma NOx was measured using a commercially available kit (Nitrate/Nitrate Colormetric Assay Kit; Cayman Chemical Inc., Ann Arbor, MI), which converts all nitrate to nitrite using nitrate reductase. Spectrophotometric analysis of total nitrite was performed using the Griess reagent. Subjects were asked to follow the National Institute of Health, low-nitrate diet guidelines for 36 hrs prior to each blood draw (28). All samples were run in triplicate.

Muscular Strength. Muscle strength was assessed by determining one repetition maximum (1-RM), using variable resistance MedX training equipment (MedX Corp., Ocala, FL). Two exercises were used for 1-RM strength testing: the chest press (upper body) and the leg extension (lower body). Prior to testing, subjects warmed up each muscle group by doing 10 repetitions using a light weight. After a 2–3 min rest, each subject began the process of reaching their 1-RM. The initial weight used for 1-RM strength testing was 50% of the subject's body weight. Each attempt was followed by 2–3 mins of rest. The determination of each subject's 1-RM was achieved within five attempts. Strength testing was conducted at the start of the study and after 12 weeks of training in both the RT and CON groups.

Exercise Training. Exercise training was performed at the Living Well Center, located at the University of Florida, Gainesville, FL. The RT group exercised on 3 nonconsecutive days/week for a 12-week period. Each session consisted of a 5-min warm-up on a treadmill followed by approximately 30–40 mins of RT. All subjects underwent a familiarization session on each piece of equipment prior to starting the 12-week training period. Training consisted of seven exercises using variable-resistance MedX training equipment: leg extension, leg curl, leg press, lat pulldown, chest press, overhead press, and bicep curl. Subjects completed two sets of 8–12 repetitions to volitional fatigue on each machine. The weight was increased by approximately 5% after the subject could successfully complete 12 or more repetitions with proper form and control. Recovery time between sets was controlled at 90-sec intervals. All RT sessions were supervised by trained exercise physiologists.

Body Composition Measurements. Body composition was estimated from skinfold thickness measurements. Skinfold measurements were obtained from seven sites (chest/pectoral, midaxillary, triceps, subscapular, abdominal, suprailiac, and thigh). Skinfold thickness was measured with a Lange skinfold caliper (Cambridge Scientific Inc., Cambridge, MD). Body density was predicted from age-adjusted equations for males (29) and

[1] Casey DP. 2006. Unpublished data.

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females (30). Percent fat was calculated from the Siri equation (31). Total body mass and percent fat values were used to calculate fat-free mass.

Statistical Analysis. Analysis of variance (ANOVA) was used to analyze baseline group differences between the RT and CON groups. Changes in the continuous dependent variables were analyzed by ANOVA, with repeated measures before and after 12 weeks of RT or of the control period. When a significant group-by-time interaction was observed, within-group comparisons between time points and between-group comparisons at each time point were performed using Tukey's post hoc analysis. All statistical analyses were performed using SPSS 14.0 for Windows (SPSS Inc., Chicago, IL). All data are reported as mean ± standard error of the mean (SEM). An alpha level of $P < 0.05$ was required for statistical significance.

Results

Twenty-four (11 men, 13 women) of the 30 subjects who underwent initial testing completed the exercise intervention. Two of the participants did not complete the study because of injuries unrelated to the exercise intervention. Four other participants were dropped because of noncompliance with the training regimen. Therefore, all data presented are the means for 24 RT participants and 18 (8 men, 10 women) CON subjects. Baseline characteristics did not differ between the six participants that failed to complete the study and those that were included in the analyses. The participants in the intervention group completed approximately 97% of their scheduled training sessions. Before the intervention period, no significant differences were observed in baseline characteristics between the two groups (Table 1). Changes in vascular function following RT did not differ between men and women. Therefore, the data for the men and women were pooled together for analyses.

Regional Arterial Stiffness Measures and Aortic Wave Reflection. There were no differences in regional PWVs, AIx, Δt_p, b/p, or HR between the two groups at study entry. The 12-week RT intervention did not elicit changes in C-R, C-F, or F-D PWV measurements in the RT group (8.4 ± 0.22 vs. 8.0 ± 0.19 m/sec, $P > 0.06$; 6.5 ± 0.14 vs. 6.3 ± 0.19 m/sec, $P > 0.47$; 9.5 ± 0.29 vs. 9.5 ± 0.29 m/sec, $P > 0.83$, respectively) or in the CON group (8.4 ± 0.16 vs. 8.3 ± 0.25 m/sec, $P > 0.61$; 6.9 ± 0.15 vs. 7.0 ± 0.16 m/sec, $P > 0.51$; 9.0 ± 0.33 vs. 8.9 ± 0.30 m/sec, $P > 0.46$, respectively; Fig. 1). The AIx and the Δt_p did not change in the RT group (2.5% ± 2.3% vs. 4.8% ± 1.8 %, $P > 0.25$; 158.5 ± 5.6 vs. 159.4 ± 3.7 ms, $P > 0.88$, respectively) or in the CON group (1.0% ± 1.9% vs. 1.9% ± 2.3%, $P > 0.43$; 158.4 ± 4.5 ms vs. 162.8 ± 5.4 ms, $P > 0.18$, respectively) following the 12-week intervention (Fig. 2). Brachial and aortic b/ps and HR did not change throughout the study in either group (Table 2).

FMD. Relative and absolute brachial FMD did not change following 12 weeks of RT (6.1% ± 0.5% vs. 6.0% ± 0.3%, $P > 0.61$; 0.27 ± 0.02 mm vs. 0.26 ± 0.02 mm, $P > 0.84$, respectively; Fig. 3). When normalized for shear stimulus, FMD remained unchanged after RT (0.19 ± 0.02 s^{-1} vs. 0.18 ± 0.01 s^{-1}, $P > 0.72$). Relative, absolute, and normalized brachial FMD did not change in the CON group (6.1% ± 0.4% vs. 5.7% ± 0.4%, $P > 0.74$; 0.27 ± 0.02 mm vs. 0.26 ± 0.02 mm, $P > 0.42$; 0.22 ± 0.02 s^{-1} vs. 0.20 ± 0.01 s^{-1}, $P > 0.40$, respectively).

Plasma NOx and NE Concentrations. Plasma levels of NOx did not change over the 12 weeks in either the RT group (19.9 ± 1.0 μ*M* vs. 19.4 ± 1.2 μ*M*, $P > 0.16$) or CON group (18.9 ± 2.3 μ*M* vs. 18.2 ± 1.7 μ*M*, $P > 0.82$). Plasma levels of NE did not change in either the RT (0.52 ± 0.10 ng/ml vs. 0.52 ± 0.09 ng/ml, $P > 0.90$) or the CON group (0.44 ± 0.06 ng/ml vs. 0.45 ± 0.06 ng/ml, $P > 0.84$).

Body Composition and Muscle Strength. Body mass did not change following 12 weeks of RT ($P > 0.45$). However, there was a reduction in subcutaneous adipose tissue (% fat; $P < 0.01$) and an increase in lean body mass ($P < 0.01$) following 12 weeks of RT. Criterion measures of upper and lower body 1-RM strength were increased following 12 weeks of RT ($P < 0.001$). Chest-press strength increased 37%, and leg-extension strength increased 42% in the RT group. There were no changes in body composition or strength in the CON group (Table 1).

Discussion

The first principal finding of this study is that an RT protocol consisting of progressively higher intensity without concurrent increases in training volume does not increase central or peripheral arterial stiffness, as assessed by PWV in young healthy men and women. Moreover, RT did not alter aortic pressure wave reflection in this group. These findings are in contrast to previous reports that RT increases arterial stiffness and wave reflection in young, healthy women (13) and reduces central arterial compliance in healthy men (11, 12). Our results are in agreement with Rakobowchuk *et al.,* (14) who found no changes in central arterial compliance following RT. The second principal finding is that there were no training-associated changes in peripheral vascular function, basal circulating levels of NOx and NE, and peripheral and aortic b/ps.

Differences between the RT protocol used in the current and previous studies may explain the disparate findings. For example, Cortez-Cooper *et al.* (13) used an RT protocol consisting of high-intensity super-sets and an extremely high volume (up to six sets per exercise), both of which are not commonly recommended for the majority of the population and are usually performed only by competitive athletes (7, 8). Interestingly, although Cortez-Cooper *et al.* found a significant increase in augmentation index of the carotid artery, both the RT and control groups demonstrated a significant increase in the C-F PWV, with the greatest increase in PWV occurring in the untrained control group

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Table 1. Subject Characteristics and Changes in Body Composition and in Muscular Strength with Resistance Intervention Compared with Control[a]

	Resistance ($n = 24$)		Control ($n = 18$)	
	Baseline	12 weeks	Baseline	12 weeks
No. of males	11	—	8	—
No. of females	13	—	10	—
Age (years)	21 ± 0.5	—	22 ± 0.7	—
Height (cm)	173.6 ± 2.4	—	174.5 ± 2.3	—
Weight (kg)	70.8 ± 3.3	71.3 ± 3.4	72.7 ± 3.7	72.8 ± 3.8
BMI (kg/m^2)	23.3 ± 0.7	23.5 ± 0.7	23.8 ± 1.0	23.8 ± 1.0
% body fat	23.7 ± 2.2	21.6 ± 2.0*	20.9 ± 1.7	20.8 ± 1.6
Lean body mass (kg)	53.1 ± 3.1	54.8 ± 3.0*	55.9 ± 2.8	56.2 ± 2.9
Chest press 1-RM (AU)	249.5 ± 24.8	335.1 ± 32.0**	277.9 ± 34.7	280.3 ± 35.3
Leg extension 1-RM (AU)	300.7 ± 21.2	423.5 ± 28.2**	297.1 ± 25.0	298.3 ± 24.5

[a] Data are mean ± SEM. 1-RM, one repetition maximal strength; AU, arbitrary unit.
* $P < 0.05$; ** $P < 0.001$.

(13). Miyachi *et al.* (11) also utilized a high-volume RT protocol, which consisted of 18 sets per training session, and found decreased carotid compliance. In a follow-up study, the same investigators found that a moderate-intensity RT protocol also resulted in decreased carotid compliance when the training volume was high (18 sets) (12). In contrast, Rakobowchuk *et al.* (14) employed similar vascular measurement techniques, which demonstrated that central arterial compliance was unaltered after 3 months of RT in young men when they used a progressive training protocol which increased intensity but not the volume (15 sets) of exercise over 12 weeks. It must be noted that the training volume used in the present study (14 sets) elicited strength gains comparable to the aforementioned studies as well as significant improvements in body composition.

The differences between our findings and that of others (11–13), might be due to the techniques used to measure arterial function. In the present study, we used PWV to assess central and peripheral arterial stiffness, whereas others have used ultrasonography to assess common carotid artery compliance. Although PWV is generally considered the gold standard for the direct measurement of arterial stiffness (32), it is dependent on manual surface distance measurements. Surface measurements may differ slightly from the true length of the arterial pathway because of anatomical particularities (33). However, this limitation is nullified by carefully standardizing PWV measurements both before and after the RT intervention. Indeed, Paini *et al.* (33) recently showed a strong correlation ($r^2 = 0.41$; $P < 0.001$) between C-F PWV and carotid stiffness measurements *via* ultrasonagraphy in normotensive individuals. Although both of the available cross-sectional ultrasonography studies report that carotid compliance is significantly reduced in strength-trained men compared with age-matched sedentary controls (9, 34), one of the studies reported no change in PWV, whereas the other reported increased PWV in strength-trained men. The explanation for this inconsistency in PWV results is not completely understood. It is important to note that measurement techniques are not the sole explanation for disparate findings, as Rakobowchuk *et al.* (14) found no change in carotid compliance following 3 months of RT using ultrasonography techniques. In aggregate, the available evidence is contradictory, and differences in measurement

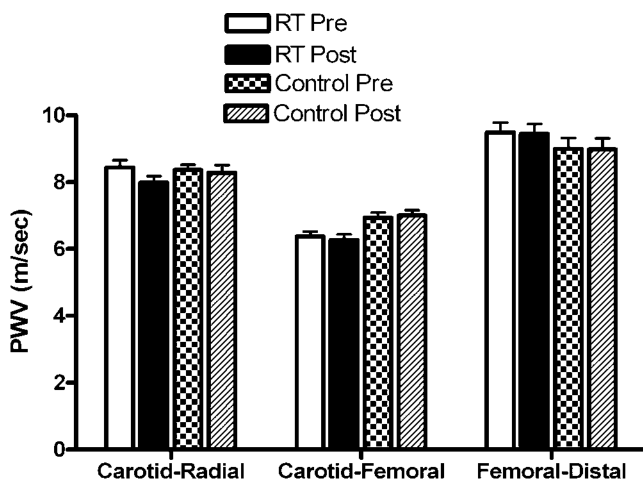

Figure 1. PWVs before and after 12 weeks of RT intervention or CON. Data represent mean ± SEM.

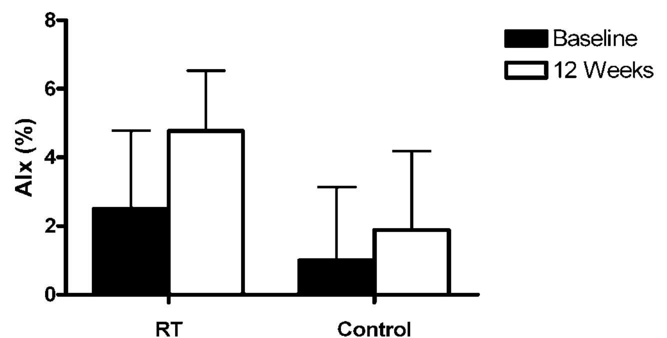

Figure 2. The AIx before and after 12 weeks of RT intervention or CON. Data represent mean ± SEM.

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Table 2. Hemodynamic Variables for RT and Control Interventions[a]

	Resistance ($n = 24$)		Control ($n = 18$)	
	Baseline	12 weeks	Baseline	12 weeks
Brachial SBP (mmHg)	117.6 ± 1.7	116.9 ± 2.0	114.2 ± 2.2	115.4 ± 2.3
Brachial DBP (mmHg)	73.9 ± 1.3	71.3 ± 0.8	70.2 ± 1.6	69.7 ± 1.7
Brachial PP (mmHg)	43.9 ± 1.6	45.5 ± 1.8	44.2 ± 1.7	45.9 ± 2.0
Aortic SBP (mmHg)	101.7 ± 1.2	101.0 ± 1.5	97.3 ± 1.9	98.7 ± 2.0
Aortic DBP (mmHg)	74.2 ± 1.2	72.2 ± 0.9	70.9 ± 1.7	70.6 ± 1.7
Aortic PP (mmHg)	27.5 ± 1.1	28.9 ± 1.2	26.4 ± 1.1	28.1 ± 1.5
MABP (mmHg)	86.6 ± 1.2	85.3 ± 1.1	83.0 ± 1.7	83.4 ± 1.7
HR (bpm)	65.6 ± 2.2	62.5 ± 2.1	61.0 ± 2.4	61.8 ± 2.1

[a] Data are mean ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MABP, mean arterial blood pressure; HR, heart rate.

technique do not completely explain the discrepancies between our findings and that of others.

To date, studies that reported increases in arterial stiffness following RT have not determined the possible physiological mechanisms responsible for this phenomenon. Stiffness of a vessel is controlled by distending pressure; structural elements within the vessel wall, mainly elastin and collagen; and functional components of the smooth muscle cells. Therefore, changes in any one of these may contribute to an increase in arterial stiffness. Unfortunately, the present study did not permit the determination of possible arterial wall changes following training. Although possible, it is unlikely that structural changes in the arterial wall would occur over short durations (e.g., 2–4 months). Indeed, Miyachi *et al.* (11) reported that, although they observed reduced central artery compliance following high-intensity RT, there were no changes in carotid artery intimamedia thickness or carotid lumen diameter.

Increased levels of sympathetic nervous system activity can enhance the basal vasoconstrictor tone of vascular smooth muscle and ultimately can play a role in the compliance of peripheral arteries. Although plasma NE levels are acutely elevated after a bout of resistance exercise (35, 36), basal NE levels following prolonged periods of RT have not been previously determined in young men and women. In the current study, we found that 12 weeks of RT did not alter basal levels of NE. This finding is in agreement with Carter *et al.*, (37) who demonstrated that 8 weeks of RT did not alter sympathetic tone as assessed by muscle sympathetic nerve activity (MSNA). Unfortunately, sympathetic nervous system activity was not assessed directly by MSNA or indirectly *via* humoral NE in any of the studies that have reported increases in arterial stiffness following RT. We reasoned that greater sympathetic nerve activity would be expected to have a greater influence on peripheral muscular arteries compared with central elastic arteries. However, only increases in central arterial stiffness have been observed, which argues against increased sympathetic tone as the possible mechanism behind changes in arterial stiffness following RT.

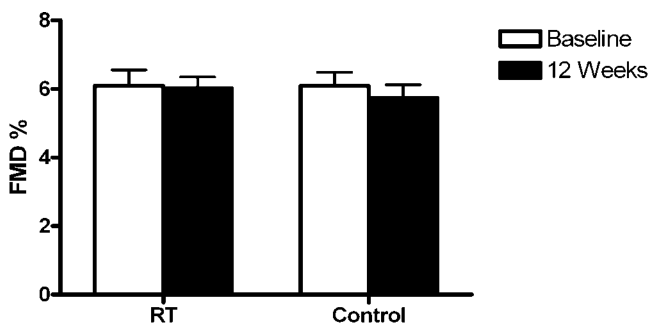

Figure 3. Peak brachial artery FMD (%) before and after 12 weeks of RT intervention or CON. Data represent mean ± SEM.

In large elastic and muscular arteries, stiffness can be influenced by endothelial function (38). Peripheral vascular endothelial function, as assessed by brachial FMD, has been shown to have a significant inverse relationship with central artery stiffness (39). Unfortunately, previous studies that reported increased arterial stiffness following RT did not assess endothelial function (11–13). Otsuki *et al.* (34) recently demonstrated, using a cross-sectional design, that strength-trained men with increased levels of arterial stiffness have elevated levels of endothelin-1 compared with sedentary men, which may suggest an impairment in endothelial function in strength-trained men. In the present study, we did not observe any changes in brachial FMD or in plasma levels of NOx following 12 weeks of RT. Rakobowchuk *et al.* (40) reported no change in brachial FMD following 12 weeks of RT in young healthy men, but postocclusion blood flow was improved, suggesting downstream vessel adaptation. Olson *et al.* (41) observed improvements in brachial FMD in overweight young and middle-aged women following 1 year of RT.

The findings of the present study may not be easily generalized to older adults or to vascular disease patients. It should be noted, however, that we recently demonstrated that moderate-intensity, whole-body RT in previously sedentary, normotensive postmenopausal women does not alter central aortic pressure wave reflection (42). Further, Maeda *et al.* (43) demonstrated that short-term, lower-body

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RT does not induce arterial stiffening in older men. Future studies with larger randomized samples, utilizing high-intensity/high-volume RT versus RT of high intensity without concurrent increases in training volume, should be performed to elucidate the impact of training volume on arterial stiffness.

In summary, the present study demonstrates that 12 weeks of RT consisting of progressively higher intensity without concurrent increases in training volume does not increase central or peripheral arterial stiffness or alter aortic pressure wave characteristics in young men and women. These findings are in contrast to previous cross-sectional reports and to high-volume interventional studies, which have found that RT results in increases in arterial stiffness and/or decreases in central artery compliance. Additionally, RT does not change brachial vascular function or alter basal levels of NOx and NE. These results support the RT recommendations of the governing bodies (AHA and ACSM) to help prevent osteoporosis, sarcopenia, obesity, and the clustering of cardiovascular risk factors associated with metabolic syndrome. Properly prescribed RT should be highly encouraged in an exercise prescription for young and older adults.

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