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**Original Scientific Paper**

# The effects of strength training on central arterial compliance in middle-aged and older adults

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***Background*** Contrary to aerobic exercise, strength training (ST) is associated with decreased central arterial compliance in young men. It is unknown whether ST, with or without concurrent endurance training, would have a similar effect in older adults with reduced baseline arterial compliance.
***Objective*** The primary aim of this study was to determine the effect of a ST program on central arterial compliance in middle-aged and older adults.
***Design*** Randomized, controlled intervention study in which 37 healthy, sedentary men and women (52 ± 2 years) performed 13 weeks of ST ($n$=13), ST + aerobic exercise ($n$=12) or stretching exercises as a control group ($n$=12).
***Methods*** Participants were rigorously screened for cardiovascular disease and underwent pre-post testing for carotid arterial compliance (via simultaneous ultrasound and applanation tonometry), carotid-femoral pulse wave velocity, plasma endothelin-1 and angiotensin II concentrations and carotid artery vasoreactivity (cold pressor test).
***Results*** ST performed alone, or in conjunction with aerobic exercise, improved maximal muscle strength and increased total lean body mass (both $P < 0.01$). No significant changes were observed in carotid artery compliance or carotid-femoral pulse wave velocity following ST or ST + aerobic exercise. Carotid artery compliance increased significantly (23%) following stretching which may be attributed to a reduction in carotid pulse pressure. No significant changes were observed in plasma vasoconstrictor hormones or carotid artery vasoreactivity following the interventions.
***Conclusion*** Thirteen weeks of moderate ST two or three times per week does not reduce central arterial compliance in middle-aged and older adults. *Eur J Cardiovasc Prev Rehabil* 15:149–155 © 2008 The European Society of Cardiology

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## Introduction

Arteries lose their compliance (become stiffer) with advancing age, independent of atherosclerosis, hypertension and other disease states [1]. This age-related reduction in central arterial compliance is associated with an increased risk for coronary heart disease [2], hypertension [3], left ventricular hypertrophy and diastolic dysfunction [4,5], and decreased baroreceptor sensitivity [6]. Fortunately, the reduction in central arterial compliance seen in middle-aged and older adults can be partially reversed by daily physical activity involving moderate aerobic exercise [7,8].

Conversely, present evidence suggests that strength training (ST) is associated with reduced central arterial compliance in young men [9–11]. Whether or not ST would further reduce the already low arterial compliance of middle-aged and older adults has not been investigated. This is a clinically relevant question given the negative consequences of reduced central arterial compliance. If chronic ST does in fact exacerbate the age-related increase in arterial stiffness, then resistance exercise prescription to combat sarcopenia, osteopenia and glucose intolerance may need to be revisited and modified.

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Accordingly, the primary aim of this study was to determine the effect of a short-term ST program on selected measures of central arterial compliance in middle-aged and older adults. The resistance exercise program used in this study was consistent with the recommendations established by the American Heart Association [12]. We reasoned that this would increase the generalizability of the study to a larger population (presumably following these guidelines), and would shed some light on the vascular effects of such recommendations.

Additionally, strength exercises are typically prescribed in conjunction with aerobic exercise to target the musculoskeletal as well as cardiovascular needs of the older adult. Therefore, an additional aim of this study was to determine the effects of combined aerobic and strengthening exercises on arterial compliance in middle-aged and older adults. Given the opposing effects of aerobic and ST exercise on central arterial compliance, we hypothesized that the performance of both modes of exercise would not reduce carotid artery compliance.

## Methods

### Participants

Over 320 people from the city of Austin and surrounding areas identified themselves by phone or email as meeting the study inclusion criteria which were (i) 40–80 years of age, (ii) not receiving hormone replacement therapy currently or in the past 12 months, (iii) nonsmoker, (iv) sedentary ($\leq$ 20 min of exercise, $\leq$ 2 times per week), (v) no history of heart disease (vi) not taking cardiovascular-acting or cholesterol-lowering medications, (vii) nonhypertensive (< 140/90 mmHg), nondiabetic and nondyslipidemic, (viii) BMI < 30 kg/m$^2$, and (ix) no musculoskeletal problems that would limit their ability to exercise. After a telephone interview, 63 qualified for further testing and were screened for cardiovascular disease risk factors and overt cardiovascular and chronic disease by a medical history questionnaire, determination of fasting plasma lipid/lipoprotein profile and glucose, seated brachial blood pressure measurements performed in triplicate, and resting and exercise 12-lead electrocardiogram. Twenty-one participants were excluded from the study on the basis of this further screening process [13,14].

A total of 42 participants (11 men, 31 women) were randomized into either the stretching control group ($n = 13$), the ST group ($n = 14$), or the combined aerobic and ST group ($n = 15$). An intervention group that performed aerobic training alone was not included in this study given the added expense, investigator time, burden to participant recruitment and the previously documented benefits of aerobic training on arterial compliance [7,8]. This study was approved by the local Human Research Committee, and every participant signed informed consent to participate.

### Measurements

To avoid potential diurnal variations, participants were tested at the same time of day throughout the study. Participants were overnight fasted for all testing sessions except for exercise testing. To minimize the effect of dietary intake, participants kept a 3-day diet record before testing and repeated the same diets at follow-up testing. Arterial compliance, arterial stiffness and carotid artery reactivity were conducted with the participant in the supine position in a quiet, dimly lit, temperature-controlled (22°C) room. All post testing occurred 24–48 h after the last intervention session to avoid the acute effects of exercise [15]. Premenopausal women were tested during the early follicular phase of their menstrual cycle.

### Maximal oxygen consumption

To verify the participant's sedentary status and document any changes in maximal aerobic capacity as a result of the study interventions, $VO_2$ max was determined during a modified Balke incremental treadmill exercise protocol. $VO_2$ max was determined via open-circuit spirometry (Max-1, Physio-dyne Instrument Corp., Quogue, New York, USA).

#### *Body composition*

Total fat mass, fat-free mass, and percent body fat were determined using dual-energy radiograph absorptiometry (Lunar DPX, GE Medical Systems, Fairfield, Connecticut, USA).

#### *Maximum strength*

Maximal voluntary dynamic strength for upper and lower extremity exercises was determined by one-repetition maximum (1-RM) strength testing using selectorize equipment (Cybex International, Owatonna, Minnesota, USA) according to established guidelines [16]. Before the maximum strength test, each participant was familiarized fully with the equipment over 3–5 ST sessions.

#### *Plasma lipid, lipoprotein and glucose concentrations*

Fasting plasma concentrations of total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides and glucose were determined by enzymatic methods with a semiautomated chemistry analyzer (Vitros DT60 II, Johnson & Johnson, Rochester, New York, USA).

#### *Basal plasma concentrations of vasoactive hormones*

Plasma concentrations of endothelin-1 were measured by an enzyme-linked immunoassay kit (R&D systems, Minneapolis, Minnesota, USA). Angiotensin II concentrations were determined by double antibody $I^{125}$ radioimmunoassay after a solid phase phenylsilylsilica column extraction (ALPCO Diagnostics, Windham, New Hampshire, USA). To eliminate interassay variance, all presamples and postsamples were analyzed within the same assay kit in duplicate.

### *Arterial blood pressures at rest*

Seated brachial blood pressure was measured in triplicate on two separate occasions to verify the participant as nonhypertensive. An automated oscillometric device (Press-Mate 7800, Colin Medical, San Antonio, Texas, USA) was use to measure blood pressure to avoid investigator bias. Supine brachial blood pressure measurements were performed in triplicate approximately 5 min apart between 07 00 and 09 00 h. and throughout the vascular testing procedures.

### *Measures of arterial stiffness*

Pulse wave velocity (PWV) was measured with an automatic medical device (VP-2000, Colin Medical, San Antonio, Texas, USA) [17]. Carotid-femoral PWV was measured on the left side of the body in triplicate. The day-to-day coefficient of variation is 7% [17].

### *Arterial compliance*

Arterial compliance of the carotid artery was determined by the technique of ultrasound imaging with simultaneous applanation tonometry [8,11]. Briefly, the left common carotid artery diameter was measured from the images derived from an ultrasound machine (HDI-5000CV, Philips Medical, Bothel, Washington, USA) equipped with a high-resolution, multifrequency probe and were transferred to a computer with digital viewing software (Access Point 2000, Freeland, Westfield, Indiana, USA) for further analyses.

Common carotid blood pressure and pressure waveforms were obtained from the contralateral artery with a pencil-type probe incorporating a high-fidelity pulse transducer (SPT-301, Millar Instruments, Houston, Texas, USA) and corrected for hold-down pressure [18]. Arterial compliance was calculated as $\{[(D1–D0)/D0]/[2(P1–P0)]\}\pi D0^2$, where D1 and D0 are the maximal and minimum diameters, and P1 and P0 are the highest and lowest blood pressure values. The day-to-day coefficient of variation is 7–9%.

### *Carotid artery reactivity to cold pressor test*

The cold pressor test (CPT) has been used to assess coronary blood flow in response to sympathetic activation [19]. More recently, the vasodilatory response or vasoreactivity to the CPT has been shown to reflect endothelial function of the large epicardial [20,21] and carotid arteries [22]. Briefly, the CPT was performed by submersion of the right foot to the ankle in iced slush for 120 s with measurements of common carotid artery diameter and mean blood velocity made at baseline and during the last 30 s of the test [22]. Before testing, the participant was instructed to avoid breath holding, muscle contractions and Valsalva's maneuver during the test. To account for changes in shear stress stimuli [23], shear stress was calculated as (mean blood velocity × blood viscosity × 4)/(carotid artery end-diastolic diameter) [24]. Mean blood velocity was measured by Doppler ultrasound with an insonation angle $< 60°$ and the sample volume gate spanning the width of the blood vessel. Carotid artery dilation in response to the CPT was expressed as relative diameter change (Δdiameter/end-diastolic diameter × 100) and diameter change normalized for shear stress during the CPT.

### *Strength training intervention*

ST was performed 3 days/week for 13 weeks on commercially available selectorize machines (Cybex International, Owatonna, Minnesota, USA) at approximately 70% of 1 RM. The exercise program consisted of 10 different exercises performed in the following order: seated chest press, horizontal leg press, shoulder press, abdominal crunches, seated hamstring curls, seated row, seated calf raises, low back extension, tricep curls, and bicep dumbbell curls. The exercises were performed for a single set of 8–12 repetitions following a warm-up set of 10 repetitions at approximately 50% of 1 RM and light stretching of the target muscle group. Participants rested 1 min between the warm-up and target weight set and 2–3 min between exercises. When the participant could perform more than 12 repetitions with the target weight, resistance was increased and the number of repetitions reduced accordingly. The ST session was supervised by an experienced personal trainer and lasted 30–45 min.

### *Strength training + aerobic exercise (COMBO)*

Participants performed the ST exercises as described above, 2 days/week on days separate from the aerobic training sessions. Each aerobic training session consisted of either walking or cycling at 60–75% of heart rate reserve for 30–45 min, 2 days/week. Once familiarized with the aerobic component of the exercise program, participants performed one of the aerobic sessions on their own, using a heart rate monitor (Polar Electro, Oy, Kempele, Finland) to document their exercise performance.

### *Stretching intervention*

The stretching group served as the time and attention control group by participating in a mild stretching program designed by a physical therapist to take the major muscle groups through full range 3 days/week. Lower extremity stretches were for the quadriceps, hamstring, adductor and gastrocnemius muscles. Upper extremity stretches were for pectoralis major and minor, triceps and latissimus dorsi muscles. Neck and trunk stretching exercises were performed to increase flexion, extension and rotation. Most of the stretches were actively held for 20 s at end range (point of minimal discomfort) for three repetitions. Two of the stretching sessions were supervised and one session was performed at home. Stretching sessions lasted from 30 to 45 min.

### Statistical analysis

One-way analysis of variance was used to determine baseline group differences. As no group baseline differences existed, a mixed model, repeated measures analysis of variance (group × time) was used to compare the means of the ST, COMBO and stretching groups following their respective interventions for the dependent variables. When a significant interaction effect was found, pair-wise comparisons were made to determine within group differences for the variables of interest. All statistical analyses were performed with SPSS 11.5 software (SPSS Inc., Chicago, Illinois, USA) with the α level set *a priori* at 0.05.

## Results

One person dropped from the stretching group due to a work conflict, and one person had to withdraw from the ST group because of a spouse's illness. Three participants dropped from COMBO group due to a work conflict or health status change unrelated to study participation. Selected participant characteristics are presented in Table 1. At baseline, there were no group differences with regard to age, height, weight, body composition or aerobic capacity (ranged from 28 ± 2 to 29 ± 2 ml/kg/min). The ST group completed 87% of the ST sessions whereas the COMBO group completed 92% of the ST sessions and 96% of the aerobic exercise sessions. The stretching group completed 100% of their required sessions.

### Cardiovascular risk factors

Cardiovascular risk factors were measured to assess the health status of the participants and to determine if these variables might confound the interpretation of the effects of the interventions on arterial compliance. The groups were not significantly different from one another with regard to metabolic risk factors before and following exercise intervention (Table 2).

### Muscle strength, lean body mass and maximal oxygen consumption

Maximal strength increased significantly in the major flexors and extensors of the lower and upper extremities of the ST (25–35%; $P < 0.001$) and COMBO (18–33%; $P < 0.05$) groups and are reported in Table 3. Lean body mass increased ∼1.5 kg in both groups ($P < 0.01$; Table 1). Maximal aerobic capacity did not increase significantly in either the COMBO or ST group ($P = 0.34$; Table 1).

### Blood pressure

Baseline values for brachial and carotid systolic, mean and diastolic blood pressure were not different between the three groups (Table 4). Carotid pulse pressure declined 6 ± 10 mmHg in the stretching group, and the decrease was greater than that of the ST and COMBO groups (both $P < 0.01$; Table 4).

### Arterial compliance and structure

Carotid artery compliance decreased slightly ($P = 0.12$) following ST and was unchanged with COMBO training (Fig. 1). Carotid artery compliance increased 23% following stretching ($P < 0.05$; Fig. 1). Carotid-femoral PWV did not change in response to ST (1109 ± 37 vs. 1048 ± 31 cm/s), COMBO (1100 ± 26 vs. 1045 ± 32 cm/s) or stretching (1133 ± 55 vs. 1132 ± 54 cm/s).

### Vasoconstrictor hormones and vasoreactivity to the cold pressor test

Basal concentrations of the plasma vasoconstrictor hormones, endothelin-1 and angiotensin II were not

**Table 1 Selected participant characteristics**

| Variable | Stretching | | Strength training | | Combination training | |
|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post |
| Sex (M/F) | 4/8 | – | 3/10 | – | 3/9 | – |
| Age (years) | 54 ± 2 | – | 52 ± 2 | – | 51 ± 1 | – |
| Height (cm) | 168 ± 3 | – | 169 ± 4 | – | 167 ± 4 | – |
| Body weight (kg) | 72.6 ± 3.5 | 72.5 ± 3.8 | 76.4 ± 3.0 | 77.3 ± 2.8 | 76.3 ± 4.7 | 76.6 ± 4.8 |
| Body mass index (kg/m$^2$) | 25.7 ± 1.1 | 25.7 ± 1.2 | 26.8 ± 1.1 | 27.1 ± 1.1 | 27.0 ± 1.1 | 27.1 ± 1.0 |
| Body fat (%) | 37 ± 3 | 37 ± 3 | 39 ± 3 | 38 ± 3 | 41 ± 3 | 40 ± 3 |
| Lean body mass (kg) | 43.6 ± 3.1 | 43.7 ± 3.1 | 45.0 ± 3.4 | 46.7 ± 3.6* | 42.8 ± 4.0 | 44.4 ± 3.9* |
| $VO_2$ max (ml/kg/min) | 27.7 ± 1.8 | 26.2 ± 1.4 | 29.4 ± 1.7 | 28.7 ± 2.2 | 28.7 ± 1.3 | 29.1 ± 1.4 |

Data are mean ± SEM. *$P < 0.01$ vs. pre.

**Table 2 Metabolic risk factors for cardiovascular disease**

| Variable | Stretching ($n$ = 12) | | Strength training ($n$ = 13) | | Combination training ($n$ = 12) | |
|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post |
| Total cholesterol (mmol/l) | 5.1 ± 0.2 | 5.2 ± 0.2 | 4.8 ± 0.2 | 4.8 ± 0.1 | 5.1 ± 0.1 | 4.8 ± 0.1 |
| LDL cholesterol (mmol/l) | 3.1 ± 0.2 | 3.2 ± 0.2 | 2.9 ± 0.2 | 2.8 ± 0.1 | 2.9 ± 0.2 | 2.8 ± 0.2 |
| HDL cholesterol (mmol/l) | 1.5 ± 0.1 | 1.4 ± 0.2 | 1.5 ± 0.1 | 1.6 ± 0.1 | 1.6 ± 0.1 | 1.6 ± 0.1 |
| Triglycerides (mmol/l) | 1.4 ± 0.2 | 1.4 ± 0.2 | 1.0 ± 0.2 | 0.9 ± 0.1 | 1.2 ± 0.1 | 1.1 ± 0.2 |
| Fasting glucose (mmol/l) | 5.6 ± 0.1 | 5.7 ± 0.1 | 5.5 ± 0.1 | 5.7 ± 0.2 | 5.3 ± 0.1 | 5.5 ± 0.2 |

Data are mean ± SEM. HDL, high-density lipoprotein; LDL, low-density lipoprotein.

**Table 3 Strength changes following 13 weeks of training**

| Variable | Strength training | | | Combination training | | |
|---|---|---|---|---|---|---|
| | Pre | Post | % Change | Pre | Post | % Change |
| Chest press (lbs) | 60 ± 6 | 79 ± 8† | 32 | 65 ± 11 | 79 ± 11* | 21 |
| Shoulder press (lbs) | 50 ± 6 | 67 ± 7† | 34 | 52 ± 8 | 69 ± 9* | 33 |
| Hamstring curl (lbs) | 83 ± 7 | 112 ± 12† | 35 | 85 ± 13 | 112 ± 15* | 32 |
| Leg press (lbs) | 121 ± 15 | 151 ± 15† | 25 | 119 ± 15 | 140 ± 16* | 18 |

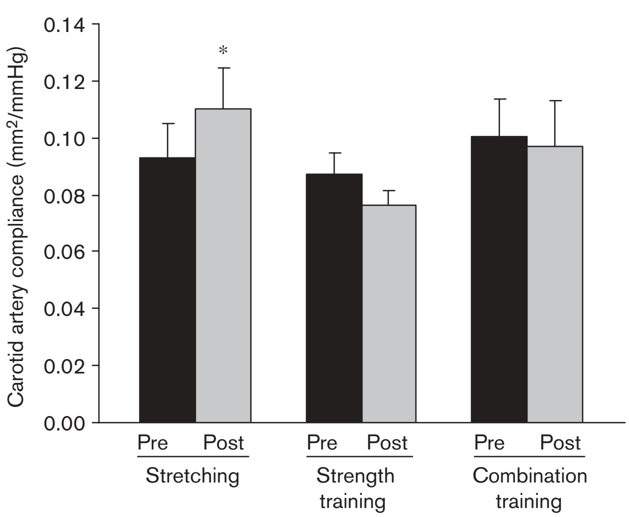
Data are mean ± SEM. *$P < 0.05$ vs. pre. †$P < 0.001$.

**Table 4 Hemodynamic and cardiovascular indices for stretching, strengthening and combination training groups**

| Variable | Stretching | | Strength training | | Combination training | |
|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post |
| Heart rate at rest (bpm) | 65 ± 3 | 65 ± 3 | 60 ± 1 | 61 ± 1 | 62 ± 2 | 59 ± 2 |
| Brachial systolic BP (mmHg) | 122 ± 4 | 119 ± 3 | 113 ± 3 | 113 ± 3 | 118 ± 3 | 117 ± 4 |
| Brachial Mean BP (mmHg) | 87 ± 3 | 85 ± 3 | 84 ± 2 | 83 ± 3 | 86 ± 2 | 86 ± 2 |
| Brachial diastolic BP (mmHg) | 66 ± 3 | 67 ± 2 | 66 ± 2 | 64 ± 2 | 68 ± 2 | 68 ± 3 |
| Brachial pulse pressure (mmHg) | 55 ± 3 | 52 ± 2 | 48 ± 2 | 49 ± 3 | 50 ± 3 | 50 ± 3 |
| Carotid systolic BP (mmHg) | 113 ± 4 | 106 ± 3 | 107 ± 3 | 106 ± 4 | 106 ± 3 | 108 ± 3 |
| Carotid pulse pressure (mmHg) | 46 ± 4 | 40 ± 3* | 43 ± 3 | 46 ± 4 | 38 ± 3 | 40 ± 3 |

BP, blood pressure. Data are mean ± SEM. *$P < 0.05$ vs. pre.

**Fig. 1**




Carotid arterial compliance before and after stretching, strength training and combination training. Data are mean ± SEM. *$P < 0.05$ vs. pre.

different before or after the intervention period for all groups (Table 5). Absolute and relative diameter changes of the carotid artery in response to the CPT were similar for all groups at baseline and tended to increase as a result of the interventions ($P = 0.09$). Normalization of the percent change in arterial diameter by the shear stress did not change these findings.

## Discussion

The major finding of this study was that no significant decrease in central arterial compliance occurred with ST in previously sedentary middle-aged and older adults. Given the clear benefits of ST on the musculoskeletal and metabolic systems of older adults, the present findings are heartening.

### Strength training and arterial compliance

Earlier studies involving young men have shown a marked decrease in central arterial compliance associated with ST [9–11]. In this study involving middle-aged and older adults, ST did not result in arterial stiffening as measured by carotid artery compliance and aortic PWV. Several possible explanations exist for this finding. The intensity and/or training volume of the strengthening program may have been insufficient to cause arterial adaptation. This, however, is not likely as moderate ST seems to induce arterial stiffening to a similar extent as more strenuous ST [25]. In the context of our study population, it is important to emphasize that the moderate training program used in the study more accurately reflects what middle-aged and older adults are likely to perform if following the present recommendations to increase muscle strength while minimizing risk of injury. Indeed, our participants significantly increased lean muscle mass, increased 1 RM strength by 25–35%, and remained injury-free throughout the training period. It is, however, possible that a longer intervention period might be needed for 'old' arteries to adapt to an ST stimulus.

In support of this, middle-aged men with a $\geq 2$ year history of vigorous ST had central arterial compliance values that were 30% lower than their sedentary peers [10]. We found a small, non-significant, decrease in central arterial stiffness in the course of 13 weeks which, theoretically, could progress with continued training. The baseline carotid artery compliance of our participants,

Table 5 Plasma concentrations of vasoconstrictor hormones and vasoreactivity to the cold pressor test

| Variable | Stretching | | Strength training | | Combination training | |
|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post |
| Hormones | | | | | | |
| Endothelin-1 (pg/ml) | 0.91 ± 0.12 | 1.18 ± 0.17 | 0.92 ± 0.15 | 0.77 ± 0.18 | 0.91 ± 0.12 | 1.20 ± 0.29 |
| Angiotensin II (pg/ml) | 4.6 ± 0.6 | 6.0 ± 1.9 | 3.7 ± 0.7 | 4.5 ± 1.0 | 4.5 ± 0.7 | 4.0 ± 0.3 |
| Cold pressor test (CPT) | | | | | | |
| Basal carotid shear stress (dyne/cm$^2$) | 5.3 ± 0.4 | 5.9 ± 0.5 | 5.4 ± 0.7 | 6.2 ± 0.8 | 6.0 ± 0.4 | 5.9 ± 0.4 |
| CPT shear stress (dyne/cm$^2$) | 4.2 ± 0.4 | 4.7 ± 0.4 | 4.8 ± 0.7 | 5.5 ± 0.6 | 4.6 ± 0.4 | 4.7 ± 0.4 |
| ΔCarotid diameter (mm) | 0.18 ± 0.06 | 0.24 ± 0.07 | 0.19 ± 0.03 | 0.26 ± 0.05 | 0.20 ± 0.05 | 0.26 ± 0.03 |
| ΔCarotid diameter (%) | 2.8 ± 1.0 | 4.0 ± 1.0 | 3.1 ± 0.6 | 4.4 ± 0.8 | 3.3 ± 0.9 | 4.4 ± 0.5 |
| ΔCarotid diameter %/shear stress (AU) | 0.75 ± 0.29 | 1.06 ± 0.34 | 0.82 ± 0.27 | 1.18 ± 0.47 | 0.74 ± 0.24 | 1.13 ± 0.20 |

AU, arbitrary units. Data are mean ± SEM.

however, was 18% lower than that of the weightlifters in that study before intervention. Thus it is possible that further reductions in compliance following ST would not be likely to occur because of a floor effect.

Finally, the proposed mechanisms by which short-term ST reduces arterial compliance were not evident in our study. Miyachi *et al.* [11] reported ~19% decrease in carotid artery compliance following 2 months of ST which was reversed by 2 months of detraining. The authors proposed an increase in vasoconstrictor tone or reduction in endothelium function as possible mechanisms. We did not find an increase in plasma angiotensin II or endothelin-1, both potent vasoconstrictors, nor did we find evidence of increased carotid artery vasoreactivity suggestive of endothelium dysfunction following ST. Therefore, ST does not seem to decrease central arterial compliance in healthy older adults with reduced baseline arterial compliance.

### Combination training and arterial compliance

Although moderate aerobic exercise has consistently been shown to increase central arterial compliance [7,8], we found that when performed in conjunction with ST, no improvement in central artery compliance occurred. By making the aerobic and ST stimuli equal in terms of frequency and duration in the present study, we sought to more specifically address the opposing effects of these two modes of exercise on central arterial compliance. Additionally, we wanted to include the minimum frequency recommended for ST. As ST did not substantially reduce central artery compliance as hypothesized, we cannot conclude that the lack of improvement in arterial compliance was due to the opposing effect of aerobic exercise. The excellent participant adherence to the training protocol, however, does offer some insight for exercise prescription as it relates to arterial compliance. These previously sedentary participants increased their moderate physical activity from virtually none to 45 min, four times per week and yet this amount of exercise was insufficient to increase central arterial compliance. On the basis of the earlier studies [7,8], had the mode of exercise been solely aerobic, we would have likely seen an increase in central arterial compliance. The exact proportion of ST to endurance training to elicit an improvement in central arterial compliance is unknown, as this is the first study to specifically address such a question. It, however, appears that aerobic exercise volume should be greater than that of ST to increase arterial compliance. We have recently demonstrated that rowing, an exercise mode which includes both aerobic and resistance training components, is associated with higher carotid artery compliance [26], suggesting that performance of both types of training can result in favorable changes in carotid arterial compliance as long as the volume of aerobic exercise is high enough.

### Stretching and arterial compliance

An unexpected finding of the study was that a stretching program significantly increased carotid arterial compliance. The stretching group was included as an attention control group to account for the 'attention' that the resistance training participants would receive from their frequent visits to the laboratory and interactions with the investigative team. Although this particular finding is rather surprising, stretching may be capable of modifying arterial compliance. Arteries are under longitudinal strain by virtue of their tethering to surrounding tissues [27] and show an age-related loss of longitudinal compliance. Sustained axial stretching of the artery has been shown to increase elastin and collagen content in the arterial wall and induce matrix reorganization [27]. Alternatively, the whole-body stretching exercises may have acted like progressive muscle relaxation techniques to reduce blood pressure and stress [28]. In this regard, stretching resulted in a significant reduction in carotid pulse pressure. Therefore, the improvement in carotid artery compliance could be attributed to a reduction in distending pressure.

## Conclusion

Given the clear benefits of ST on the musculoskeletal system of older adults, the present findings that moderate intensity ST does not accelerate the age-related reduction in central arterial compliance is heartening. Present exercise guidelines for older adults recommend the performance of both aerobic and ST exercise, thus it is expected that ST would not be performed exclusively. The positive effect of stretching on blood pressure and arterial compliance indicates that this type of exercise should not be overlooked in the complete exercise prescription for middle-aged and older adults.

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