Changes in Vascular Hemodynamics in Older Women Following 16 Weeks of Combined Aerobic and Resistance Training

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The purpose of this study was to determine whether combined (aerobic and anaerobic) training decreases blood pressure (BP) and improves vascular properties. Seventy-nine postmenopausal women were randomly assigned to 3 groups that trained at different frequencies. Maximum oxygen uptake, body composition, BP, and arterial elasticity were evaluated prior to training and after 16 weeks of training. There was a significant time effect (decrease) for resting systolic BP (SBP) and rate pressure product. Exercise SBP, diastolic BP (DBP), heart rate, and RPP also decreased. Changes in total vascular impedance were related to SBP and changes in systemic vascular resistance were related to changes in DBP independent of body composition changes. Our findings suggest that combined training reduces SBP and improves vascular properties and that combined training 1 d/wk decreases BP similar to more frequent combined training. Training-induced changes in arterial resistance and impedance may be involved in inducing changes in BP. *J Clin Hypertens (Greenwich)*. 2013;15:241–246 ©2012 Wiley Periodicals, Inc.

Hypertension (HTN) is one of the leading risk factors for the development of cardiovascular disease (CVD). High blood pressure (BP) causes acceleration of atherosclerosis, arterial smooth muscle hyperplasia and hypertrophy, and increased collagen synthesis, all of which lead to structural and functional alterations to the arterial wall.[1] High BP is associated with small artery and organ damage.[2] The treatment and prevention of specific organ damage is not identical and reversibility varies.[3]

Evidence suggests that performing regular physical activity decreases BP and the risk of CVD.[4–7] A strong inverse relationship between fitness level and mortality was shown in the Aerobic Center Longitudinal Study (ACLS). Individuals who exercised at >4 standard metabolic equivalence showed a significant reduction in all-cause mortality.[8]

Previous research suggests that both aerobic and resistance training decrease BP.[4,6,7,9–15] However, few studies have evaluated the effects of combined aerobic and resistance training on BP. Altered vascular integrity as measured by artery elasticity and vascular resistance/impedance possibly contributes to elevated BP.[1] Figueroa and colleagues[16] suggests that combined training most likely improves the functional adaptations within arterial walls. Endothelial-dependent vasodilation may induce a reduction in vasomotor tone within the peripheral arteries, which results in decreased BP.[16] Similarly, Vona and colleagues[17] found that with combined aerobic and resistance training endothelial dysfunction decreased. These changes may be sufficient enough to improve overall vascular integrity.

Exercise frequency might be particularly important when considering the effects of a combined aerobic and resistance training program. Older women may require more time to recover after exercise training so an increased volume of training could induce an overtraining response. This type of exercise response can counteract the beneficial exercise-induced adaptations.[18] Determining an optimal training frequency may decrease the possibility of overtraining.

The objective of this study was to determine what affect 3 different frequencies of combined aerobic and resistance training have on both resting and submaximal exercise BP, artery elasticity, vascular resistance, and vascular impedance.

METHODS AND PROCEDURES

Study Participants

All participants were healthy African American and European American women 60 years and older enrolled in a larger study designed to look at metabolic factors in women older than 60, across 3 different training frequencies. Patients were all sedentary and did not participate in any regular exercise training. Exclusion criteria included clinical evidence of heart disease, abnormal electrocardiography (either at rest or during screening exercise testing), smoking, diabetes mellitus, or medications that affected energy expenditure, insulin levels, thyroid status, or heart rate. All participants were randomized, using Block Randomization stratified by race, to one of 3 exercise groups: group 1, 1 d/wk aerobic and one different d/wk strength training; group 2, 2 d/wk aerobic and 2 d/wk strength training; and group 3, 3 d/wk aerobic and 3 d/wk strength training. All patients adhered to more than 95% of their sessions, and there was no

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significant difference in adherence between groups. Methods and procedures were approved by the appropriate institutional review board, and all participants signed appropriate informed consent forms.

Study Design and Methods

All participants maintained a stable weight through diet control and maintained dietary records prior to evaluations. Patients were evaluated for muscle performance, maximal and submaximal VO_2, heart rate, and BP (modified Balke treadmill protocol), resting BP and resting artery elasticity, vascular resistance, and vascular impedance, prior to and after 16 weeks of training.

Exercise Testing

Maximum oxygen uptake (VO_2max) was evaluated with a physician-supervised modified Balke treadmill test protocol. A metabolic cart, calibrated prior to testing (Vmax Spectra29; SonsorMedics, Inc, Yorba Linda, CA), was used to evaluate ventilatory expired gases. Monitoring consisted of 12-lead electrocardiogram and BP was measured every 2 minutes (Omron Blood Pressure Monitor, model HEM-780; Omron Healthcare, Inc, Bannockburn, IL). Patients began walking at 2 mph at a 0% grade. The grade increased 3.5% every 2 minutes and the speed was increased to 3 mph at 12 minutes. All patients exercised until voluntary fatigue. VO_2max was defined as the highest 20-second average value during the last stage of the exercise test, a maximal respiratory exchange ratio (RER) $\geq$1.1, and a maximal HR that was $\leq$10 beats per minute of the age-predicted maximum HR (220–age). Two of the 3 criteria for VO_2max were required. Seven days following the maximal VO_2max test, patients performed a submaximal walk test and their BP response was measured at 0% and 5% grade. They walked at 2 mph with a 0% grade for 4 minutes and 4 minutes at a 5% grade. BP was measured by auscultation between 3:30 and 4:00 time points for both stages. Due to equipment malfunction data for exercise HR, SBP, and DBP data (Table III) analysis was for 62 (HR; group 1 [n=21], group 2 [n=22], group 3 [n=19]), 57 (SBP and RPP; group 1 [n=20], group 2 [n=18], group 3 [n=19]), and 56 (DBP; group 1 (n=20), group 2 (n=18), group 3 (n=19) patients.

Body Composition

Dual-energy x-ray absorptiometry (Lunar DPX-L densitometer; LUNAR Radiation, Madison, WI) in the Department of Nutrition Sciences at UAB was used to determine total fat and lean mass. Adult Software, version 1.33, was used to analyze the scans.

Resting BP

Resting supine BP was taken on 3 consecutive days (and the measurement from the second day was reported) with automatic auscultation between 7 AM and 8 AM in a fasted state prior to exercise (Omron Blood Pressure Monitor, model HEM-780; Omron Healthcare, Inc). This was done to ensure that a stable measurement was used. Resting BP was also measured after training.

Arterial Elasticity

Arterial elasticity was measured using noninvasive radial artery pulse wave analysis. Pulse wave analysis was performed in duplicate, and average values were reported. The radial artery waveform was obtained with a sensor positioned over the artery and calibrated using an oscillometric method on the opposite arm. Thirty seconds of analog waveforms were digitized at 200 samples/sec, and a beat marking algorithm determined the beginning of systole, peak systole, onset of diastole, and end diastole for all beats in the 30-second measurement period. An average beat determination was constructed, and a parameter-estimating algorithm (Hypertension Diagnostics, Eagan, MN) was applied to define a third-order equation that replicated the diastolic decay and waveform. The estimates of arterial elasticity are based on the asymptotic behavior of a Windkessel mode (1, 2). Mathematically (CR-2000 operator's manual), the pulse waveform P(t), the pressure (mm Hg) at time t elapsed since the beginning of diastole, is modeled as a decaying exponential function plus a sinusoidal function dampened by a decaying exponential:

$$P(t) = \{a1 * \exp(-a2t)\} + \{a3 * \exp(-a4t) * \cos(a5t + a6)\}.$$

The modified Windkessel model then uses the parameters a1 − 6 to estimate:

$$LAE * SVR = 2a4[(a2 + a4)2 + a52]/[a2(2a4 + a2)(a42 + a52)]$$

SAE*SVR=1/(2a4+a2). SVR is the systemic vascular resistance=mean arterial BP/cardiac output. Cardiac output (L/min) is estimated as HR*(−6.6+(0.25*(ET-35)−(0.62*HR))+(40.4*BSA)−(0.51*Age))/1000, where ET is ejection time in milliseconds, HR is heart rate in beats per minute, and BSA is body surface area in millimeters squared (estimated as 0.007184*WT 0.425*HT 0.725). ET in milliseconds is directly observable from the pulse waveform. Information from the pulse waveform only provides estimates of LAE*SVR and SAE*SVR. LAE and SAE are estimated by dividing each of LAE*SVR and SAE*SVR by SVR. Due to equipment malfunction, data were collected for only 70 patients (group 1 [n=20], group 2 [n=27], group 3 [n=21]).

Exercise Training

Training sessions lasted 50 minutes in a facility dedicated to research and under the supervision of exercise physiologists. Each session began with a 3 to 4 minute warmup on a bike ergometer or treadmill and 3 to 4 minutes of stretching.

Aerobic Training. During the first week, patients performed 20 minutes of continuous exercise at 67% maximum heart rate. Each week, intensity and duration were increased so that at 8 weeks, patients were working at 80% maximum heart rate for 40 minutes. Exercise modalities included bike ergometer and treadmill exercise.

Resistance Training. Strength exercises included leg press, squats, leg extension, leg curl, elbow flexion, lateral pull-down, bench press, military press, lower back extension, and bent-leg sit-ups. Each exercise consisted of 2 sets of 10 repetitions with a 2-minute rest between sets. The intensity was gradually increased to 80% of the maximum weight the patient could lift at one time (1RM). Patient 1RM was determined every fifth week to ensure that intensity was increased appropriately.

Statistical Approach

One-way analysis of variance (ANOVA) was used to analyze all descriptive data. One-way ANOVA with repeated measures was used to analyze all main variable outcomes. Pearson product correlations were used to evaluate relationships between changes in variables of interest. Two multiple linear regression models for estimating changes in SBP were developed (first model age, ΔSVR, ΔFM, ΔFFM were independent variables and the second model age, ΔTVI, ΔFM, ΔFFM). DBP was also modeled using the same 2 sets of independent variables. Due to missing data, analyses for arterial elasticity and exercise BP were performed using a reduced cohort (sample sizes for each variable are included in the Tables).

RESULTS

Descriptive statistics are shown in Table I. At baseline, age and height were not significantly different between groups. Body weight and percentage of body fat were significantly different; however, there was little change in the values after training. Changes in large arterial elasticity (LAE), small arterial elasticity (SAE), systemic vascular resistance (SVR), and total vascular impedance (TVI) from pre-training to post-training are shown in Table II. There was no significant time effect for any of these variables. A significant group affect as well as time by group interaction was observed for small arterial elasticity.

Resting and exercise BP and heart rate for the 3 groups at baseline and 16 weeks are shown in Table III. At rest, a significant time effect was observed for systolic BP (SBP), diastolic BP (DBP), heart rate (HR), and rate pressure product (RPP), showing that after 16 weeks of training, older women had significantly reduced resting SBP, DBP, HR, and RPP. There was no significant group effect or time by group effect for any variable at rest.

During exercise there was a significant time effect for all variables, showing a significant reduction in SBP, DBP, HR, and RPP during exercise. There was no significant group effect or time by group interaction for any exercise variable.

Correlations between changes in systolic BP (ΔSBP) and diastolic BP (ΔDBP) with age, changes in fat mass (ΔFM), fat free mass (ΔFFM), systemic vascular resistance (ΔSVR), total vascular impedance (ΔTVI), large artery elasticity (ΔLAE), and small artery elasticity (ΔSAE) are shown in Table IV. There was a positive relationship between ΔDBP and ΔFFM, ΔSVR, and ΔTVI. A negative relationship was seen between ΔDBP and age, ΔFM, ΔLAE. A positive relationship was seen between ΔSBP and ΔTVI.

Four linear regression models for estimating resting ΔSBP and ΔDBP are shown in Table V. The first model includes age, ΔSVR, ΔFM, ΔFFM, for prediction of ΔSBP. After adjusting for ΔFM and

TABLE I. Baseline Characteristics of Participants

	Group 1	Group 2	Group 3	*P* Value
Age	65.6±0.7	63.7±0.5	64.8±0.7	.11
Height	166.5±1.1	165.2±1.0	164.4±0.8	.33
Δ Body weight				
Pre-training	78.2±2.7	75.0±1.7	68.4±2.0	.01
Post-training	77.7±2.4	73.8±1.7	68.2±2.0	
Δ Percent body fat				
Pre-training	44.7±1.2	43.0±0.9	39.5±1.4	.01
Post-training	43.6±1.3	41.5±0.8	38.8±1.4	

Values are reported as mean±standard error. Group 1 (n=27); group 2 (n=30); group 3 (n=22).

TABLE II. Changes in Arterial Properties in Response to Exercise Training

	Group 1	Group 2	Group 3	*P* Value
ΔLAE				
Pre-training	13.3±0.9	13.8±1.2	11.9±0.7	T=.33
Post-training	15.3±1.2	12.8±0.7	13.1±1.0	G=.26
	(n=22)	(n=27)	(n=21)	T*G=.21
ΔSAE				
Pre-training	4.2±0.4	4.3±0.4	3.0±0.3	T=.87
Post-training	4.2±0.3	3.7±0.3	3.7±0.4	G=.16
	(n=22)	(n=27)	(n=21)	T*G=.01
ΔSVR				
Pre-training	1538.6±43.9	1528.9±52.3	1673.3±63.6	T=.77
Post-training	1517.2±57.8	1625.7±65.4	1625.4±57.7	G=.28
	(n=22)	(n=27)	(n=21)	T*G=.12
ΔTVI				
Pre-training	165.4±9.4	161.8±7.1	179.3±10.4	T=.55
Post-training	156.6±10.8	170.0±7.6	167.4±10.2	G=.49
	(n=22)	(n=27)	(n=21)	T*G=.42

Abbreviations: LAE, large artery elasticity; SAE, small artery elasticity; SVR, systemic vascular resistance; TVI, total vascular impedance from pre-training to post-training. Values are reported as mean ± standard error. T = time; G = group; T*G = time by group.

TABLE III. Changes in Blood Pressure in Response to Exercise Training

	Group 1	Group 2	Group 3	*P* Value
Resting				
ΔSBP				
Pre-training	124.7±2.8	124.7±3.0	124.1±3.1	T=.01
Post-training	116.4±2.2	123.8±2.6	120.5±3.3	G=.55
	(n=27)	(n=30)	(n=22)	T*G=.17
ΔDBP				
Pre-training	68.2±2.1	69.7±2.1	66.5±2.1	T=.02
Post-training	63.9±1.4	67.3±2.0	66.0±1.7	G=.53
	(n=27)	(n=30)	(n=22)	T*G=.35
ΔHR				
Pre-training	63.9±1.3	63.3±1.3	65.1±1.5	T=.02
Post-training	59.9±1.3	62.3±1.2	64.2±1.7	G=.28
	(n=27)	(n=30)	(n=22)	T*G=.22
ΔRPP				
Pre-training	7977.3±256.3	7888.1±243.4	8093.9±293.1	T<.01
Post-training	6978.2±199.9	7704.1±204.3	7780.4±385.5	G=.34
	(n=27)	(n=30)	(n=22)	T*G=.08
Exercise				
ΔSBP				
Pre-training	168.8±5.7	164.9±5.3	164.2±5.3	T<.01
Post-training	153.5±3.4	151.5±5.3	141.7±4.3	G=.38
	(n=20)	(n=18)	(n=19)	T*G=.43
ΔDBP				
Pre-training	73.5±2.9	78.2±2.5	76.6±2.0	T=.01
Post-training	71.8±2.2	73.6±2.9	69.7±2.2	G=.47
	(n=20)	(n=17)	(n=19)	T*G=.45
ΔHR				
Pre-training	113.6±3.0	115.1±2.1	114.4±3.0	T<.01
Post-training	104.8±3.0	112.1±2.6	106.0±2.1	G=.39
	(n=21)	(n=22)	(n=19)	T*G=.16
ΔRPP				
Pre-training	19354.4±1007.5	19181.0±875.5	18967.1±1029.0	T<.01
Post-training	16116.0±577.1	17254.8±994.4	15058.8±642.8	G=.54
	(n=20)	(n=18)	(n=19)	T*G=.30

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure; RPP, rate pressure product. Values are reported as the mean±standard error. Group 1 trained 1 d/wk; group 2 trained 2 d/wk group 2 trained 2 d/wk; group 3 trained 3 d/wk. T = time; G = group; T*G = time by group.

TABLE IV. Correlations Between Changes in Body Composition, Arterial Properties, and Blood Pressure

	ΔSBP (*P*)	ΔDBP (*P*)
Age	−0.118 (.193)	−0.221 (.051)
ΔFFM	0.058 (.335)	0.211 (.059)
ΔFM	0.164 (.113)	−0.133 (.165)
ΔSVR	0.189 (.381)	0.269 (.023)
ΔTVI	0.240 (.037)	0.348 (.004)
ΔLAE	−0.122 (.186)	−0.246 (.034)
ΔSAE	0.175 (.292)	0.075 (.292)

Abbreviations: ΔSBP, systolic blood pressure with age; ΔDBP, diastolic blood pressure with age; ΔFM, changes in DEXA total fat mass; ΔFFM, changes in DEXA fat free mass; ΔSVR, systemic vascular resistance; ΔTVI, total vascular impedance; ΔLAE, large artery elasticity; ΔSAE, small artery elasticity.

ΔFFM, age was the only significant independent correlate. The second model was identical to the first except for the substitution of ΔDBP for ΔSBP. After adjusting for ΔFM and ΔFFM, both age and ΔSVR were significant independent correlates. The third model includes age, ΔTVI, ΔFM, and ΔFFM for prediction of ΔSBP. After adjustment for ΔFM and ΔFFM, both ΔTVI and ΔFM were significant independent correlates. The fourth model was identical to the third except for the substitution of ΔDBP for ΔSBP. After adjusting for ΔFM and ΔFFM, age and ΔTVI were significant correlates.

DISCUSSION

After 16 weeks of combined aerobic and resistance training, similar decreases in BP and RPP (an indicator of myocardial oxygen demand) were observed in all 3 training groups both during exercise and at rest. This is particularly important in older adults who may have compromised cardiovascular systems and increased risk of stroke and myocardial infarction while performing routine daily activities. Similar changes in

TABLE V. Multiple Regression for Estimating Changes in Blood Pressure

	Intercept	Slope	Adjusted β	*P* Value
Regression for ΔSBP				
Adjusted R^2=0.361	9.47			.77
Age		−1.26	−0.29	.01
ΔSVR		0.01	0.09	.41
ΔFM		0.34	0.05	.70
ΔFFM		0.20	0.02	.89
Regression for ΔDBP				
Adjusted R^2=0.124	47.61			.03
Age		−0.69	−0.28	.04
ΔSVR		0.01	0.28	.03
ΔFM		−0.21	−0.05	.71
ΔFFM		1.42	0.21	.13
Regression for ΔSBP				
Adjusted R^2=0.083	46.62			.21
Age		−0.70	−0.16	.23
ΔTVI		0.08	0.32	.02
ΔFM		2.01	0.28	.05
ΔFFM		1.48	0.13	.37
Regression for ΔDBP				
Adjusted R^2=0.172	50.47			.02
Age		−0.75	−0.30	.02
ΔTVI		0.06	0.37	.01
ΔFM		0.05	0.01	.93
ΔFFM		1.29	0.19	.16

Abbreviations: ΔSBP, systolic blood pressure from age; ΔDBP, diastolic blood pressure from age; ΔSVR, changes in systemic vascular resistance; ΔTVI, total vascular impedance; ΔFM, DEXA total fat mass; ΔFFM, DEXA fat free mass.

SVR, BP, and RPP between the 3 groups indicate that exercise training 1 d/wk aerobic and 1 d/wk resistance induces BP and RPP changes similar to training for up to 3 d/wk, provided the one resistance and the one aerobic exercise session take place on nonconsecutive days.

Interestingly, changes in TVI were independently related to changes in resting SBP and changes in resting SVR were significantly related to changes in DBP after adjusting for FFM and FM. Although causality cannot be directly inferred, it does suggest that BP changes following exercise training may at least be partly induced by changes in vascular resistance and vascular impedance.

Endurance training alone has been known to improve cardiovascular health.[4–7,13,19,20] Kikkinos and Gregg show that aerobic exercise of moderate to vigorous intensities decreased BP in patients with mild to moderate HTN.[4,6] A study conducted by Tanasescu and colleagues[21] showed a 42% CVD risk reduction in men who ran for an hour 1 or more times per week. Tanaka and colleagues found that middle-aged individuals who performed low- to high-intensity endurance training had a lower SBP and DBP than their sedentary peers, and Higashi and colleagues found that moderate-intensity aerobic training performed 5 times per week lowered SBP and DBP.[7,9] Physiological adaptations, such as an increase in parasympathetic tone and increased endothelial function lead to increased vasodilation.[17,22,23] These physiological changes induced by performing regular exercise reduces BP.[24] Therefore, we hypothesize that in the present study it is likely that endurance training–induced changes in parasympathetic tone may have contributed to the reductions in resting and exercising SBP and DBP we observed.

In the study the group that performed aerobic exercise 1 day per week had similar changes in resting BP (with the exception of SAE) as the groups that performed aerobic and resistance exercise 2 or 3 days per week. The observed changes could be in response to the strength training performed on a nonconsecutive day from the aerobic training. Previous research suggests that resistance training is related to decreases in DBP.[10–13] Fagard and colleagues[13] found that resistance training performed 2 times per week reduced resting DBP and had no affect on SBP. Collier and colleagues[11] reported that women exhibit greater decreases in DBP than SBP following resistance training. Casey and colleagues[12] found that performing resistance training on 2 nonconsecutive days per week decreased DBP. In addition, Tanasescu and colleagues showed a 23% cardiovascular risk reduction in individuals who performed 30 minutes of resistance exercise $\geq$1 times per week. Taken together, these studies support the view that resistance training can also have a positive effect on BP, particularly DBP. The interpretation of our data is that aerobic and resistance training on nonconsecutive days at a frequency of as little as 1 day a week is sufficient to achieve improvements in BP in older healthy women. It is difficult to interpret the time by group interaction. It is probable that the apparent increase in SAE in group 3 may have been primarily the result of regression toward the mean, a consequence of the abnormally low initial SAE in group 3. There was no significant group effect or time by group interaction for large artery elasticity, systemic vascular resistance, or total vascular impedance.

Similar to previous research, we observed decreases in HR, BP, and RPP during submaximal exercise.[15] A 9.0% decrease was seen during exercise and 3.0% decrease at rest for SBP. A 5.0% decrease was seen during exercise and 3.0% decrease at rest for DBP. A 14.7% decrease was seen during exercise and a 5.0% decrease was seen at rest for RPP. Reduction in sympathetic stimulation probably contributed to the reduction of BP observed in the submaximal exercise tasks observed after 16 weeks of training. Stimulation of the sympathetic nervous system and thus adrenal activity (primarily norepinephrine) is reduced during an absolute submaximal work level (ie, walk at 2 mph) in trained individuals compared with untrained.[18] Vasodilation in response to decreases in sympathetic stimulation causes a reduction in SVR and thus BP.[18]

Decreases in HR and BP contribute to decreases in RPP.[18] A lower RPP during absolute submaximal exercise is indicative of less myocardial stress, making a cardiac event less likely to occur during daily activities.[18]

LIMITATIONS

The patients in this study were older women who trained for 16 weeks (as this was the group of interest for our study) so the results of this study are not applicable to other age and/or sex groups. In addition, we had relatively small numbers of patients in each group, although our study is still larger than most in the literature. We also had no control group. Patients were randomly assigned throughout the 5-year duration of the study, so it is unlikely that a seasonal bias occurred in the evaluations. Also, it is unlikely that an aging-related increase in HR and BP would have occurred during only a 16-week timespan.

CONCLUSIONS

The present findings indicate that compared with more frequent combined aerobic and resistance training, in this population of women 60 years and older, combined aerobic and resistance training 1 day a week reduces both resting and exercise SBP and DBP. Our data also suggest that following exercise training alterations in vascular properties may at least partly explain some of the decrease in resting BP.

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