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Blood Pressure Reductions With Exercise and Sodium Restriction in Postmenopausal Women With Elevated Systolic Pressure: Role of Arterial Stiffness

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OBJECTIVES This study determined the relative efficacy of aerobic exercise (daily walking) and moderate dietary sodium restriction (sodium intake <100 mmol/day) for reducing systolic blood pressure (SBP) and pulse pressure (PP) in postmenopausal women with elevated initial levels, and the potential role of reductions in large artery stiffness in these changes.

BACKGROUND Lifestyle behaviors are recommended for lowering blood pressure (BP) in adults with elevated baseline levels, but there is little information as to the relative efficacy of different interventions or the mechanisms underlying their potential beneficial effects.

METHODS After baseline measurements and random assignment, 35 nonmedicated healthy postmenopausal women with SBP between 130 and 159 mm Hg completed three months of either aerobic (walking) exercise (n = 18; 62 ± 9 years, mean ± SD) or moderate dietary sodium restriction (SR) (n = 17; 65 ± 10 years, mean ± SD).

RESULTS Body mass and composition, plasma volume, and fasting concentrations of metabolic coronary risk factors did not differ between the groups at baseline or change with intervention. Systolic BP and PP at rest decreased with both exercise and SR ($p < 0.05$); however, the reductions were three- to fourfold greater with SR ($p < 0.05$). Sodium restriction, but not exercise, also reduced 24-h SBP and PP ($p < 0.05$). Aortic pulse wave velocity (PWV) and carotid augmentation index were reduced only with SR ($p < 0.05$). Changes in SBP and PP at rest and over 24 h correlated with the corresponding changes in aortic PWV ($r = 0.53$ to 0.61, $p < 0.01$).

CONCLUSIONS Moderate SR lowers SBP and PP in postmenopausal women with elevated baseline levels more than does daily walking. The greater blood pressure reductions with SR may be mediated in part by a decrease in the stiffness of the large elastic arteries. (J Am Coll Cardiol 2001;38:506–13) © 2001 by the American College of Cardiology

The stiffness of the large elastic arteries in cardiothoracic (central) circulation increases with age in adult humans, although the muscular peripheral arteries are not obviously affected (1–4). The increase in central arterial stiffness results in progressive age-associated elevations in systolic blood pressure (SBP) and pulse pressure (PP) (5–7). Importantly, the increases in SBP and PP after age 50 are much greater in women than in men (5,7).

The elevations in SBP and PP with age are clinically significant. Based on relative risk, SBP and PP represent the single largest contributor to overall cardiovascular disease (CVD) risk in middle-aged and older adults (7–11). Even modest elevations in SBP above 120 mm Hg are associated with increased CVD risk (7,9,10).

Lowering SBP reduces CVD mortality in middle-aged and older patients with elevated baseline levels (9,12). Current treatment guidelines for this population emphasize lifestyle modifications such as regular exercise, weight loss and dietary changes as the initial therapeutic approach (13,14). Of these, regular aerobic exercise might be unique because in hypertensive adults it may both lower SBP and produce other benefits such as improvements in fitness and changes in other health behaviors (15,16). However, there is currently little information from randomized trials in middle-aged and older adults, particularly postmenopausal women, upon which to base treatment recommendations on the use of aerobic exercise. Moreover, to our knowledge there are no data as to the relative efficacy of regular exercise compared with other currently recommended behavioral (lifestyle) interventions for lowering SBP in this clinically important population. In this context, dietary sodium restriction (SR) lowers SBP in adults with elevated baseline

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Abbreviations and Acronyms

AI = augmentation index
BMI = body mass index
BP = blood pressure
CVD = cardiovascular disease
DBP = diastolic blood pressure
EX = aerobic exercise
MAP = mean arterial blood pressure
PP = pulse pressure
PWV = pulse wave velocity
SBP = systolic blood pressure
SR = dietary sodium restriction

levels, and the magnitude of the reduction appears to be greater in older patients (17,18).

If aerobic exercise and SR lower SBP in middle-aged and older adults, reductions in central arterial stiffness could play an important mechanistic role. Cross-sectional group comparisons indicate that both regular exercise and reduced sodium intake are associated with lower stiffness in the large elastic arteries of the central circulation (3,4,19). However, whether habitual exercise and/or SR reduce central arterial stiffness in middle-aged and older adults with chronic elevations in SBP is unknown.

Accordingly, the primary experimental aims of the present investigation were to determine: 1) the relative efficacy of regular aerobic exercise and moderate SR in lowering SBP and PP in postmenopausal women with elevated baseline SBP, and 2) whether one of these interventions is more efficacious in reducing central arterial stiffness, and whether such reductions are related to the corresponding decreases in SBP and PP.

METHODS

Subjects. Subject inclusion criteria were postmenopausal status (amenorrheic for at least two years and follicle stimulating hormone plasma concentrations >40 IU/l); ≥50 years of age; SBP in the high normal or Stage 1 hypertensive range (130 to 159 mm Hg) with diastolic BP (DBP) ≤99 mm Hg (i.e., Stage 1 hypertensive range or below) during sitting rest; no antihypertensive medications taken either two months prior to screening (n = 1) or ever (n = 35); absence of other chronic disease (based on blood chemistries, medical history, physical examination, and resting and maximal exercise electrocardiogram); not on a low-sodium diet nor having performed regular exercise during the preceding two years; not smoking; and a body mass index (BMI) <35. A total of 35 women completed a three-month intervention involving either walking exercise (EX; n = 18) or moderate SR (n = 17). Four women (3 EX, 1 SR) dropped out of the interventions owing to personal reasons. Six of the women who completed each intervention had baseline SBP in the high normal (130 to 139 mm Hg) range, whereas the others had initial values in the Stage 1 systolic hypertensive range (140 to 159 mm Hg). Nine of the 17 women in the SR group and 10 of the 18 women in the EX group were on hormone replacement therapy. Of the subjects completing the interventions, 34 were white and 1 was Asian. The Asian subject completed the SR condition and demonstrated changes in BP similar to the mean changes for her group. The nature, purpose, and risks of the study were explained to each subject before written informed consent was obtained.

Experimental design. A parallel group design with an active treatment control group (i.e., SR) was employed rather than a placebo control group; the latter could be considered unethical in that it would deny patients either the experimental or usual care treatment likely to lower CVD risk. Moreover, stable baseline resting blood pressure (BP) values were documented in each subject prior to randomized assignment (see below), thus minimizing the need for a placebo control group for the key outcome variable. Subjects satisfying the inclusion criteria underwent baseline measurements and subsequently were randomly assigned to either the EX or SR group. The duration of each condition was ~13 weeks (3 months). During these intervention periods subjects were asked to maintain their preenrollment body weight; they reported to the laboratory every two weeks for intervention-specific counseling, compliance monitoring, and measurements of body weight and casual BP at rest. At the end of their assigned condition, subjects again underwent the measurements performed at baseline.

Measurements. Treadmill exercise testing was performed using a modified Balke walking protocol as described in detail previously (20). Body composition was determined by dual energy X-ray absorptiometry. Total caloric intake and its composition, including sodium intake, were estimated from food records analyzed by experienced research dietitians. Urinary sodium excretion was determined from 24-h urine collections. Plasma volume was measured using the modified Evans blue dye procedure as described previously (21). Fasting plasma concentrations of glucose, insulin, lipids and lipoproteins, and fibrinogen were measured by the Core Laboratory of the University of Colorado Adult General Clinical Research Center using conventional procedures (3).

Casual BP at rest was recorded in the upright seated position between 7 and 11 AM after an overnight fast in strict accordance with American Heart Association guidelines as described in detail previously by our laboratory (22,23). Recordings were obtained in triplicate in three separate sessions at least one week apart in order to establish the stability of baseline levels; this was repeated at end-intervention. The mean values from the three sessions were used to establish pre- and end-intervention levels. To avoid any possibility of investigator bias, recordings were obtained over the brachial artery with a semi-automated device (Dinamap XL, Critikon, Florida). Casual recordings of BP at rest also were obtained manually using auscultation over the brachial artery with a random zero sphygmomanometer

(Hawksley and Sons, West Sussex, United Kingdom). These values were used to independently confirm the results obtained with the semi-automated device. In addition, BP recordings were obtained over a 24-h period of normal daily activity using a noninvasive ambulatory monitor (Model 90207, Spacelabs, Redlands, Washington) as described in detail previously (22,23). The following values were obtained: 24-h, daytime, and nighttime SBP and DBP; SBP load (% of recordings >140 mm Hg) and DBP load (% of recordings >90 mm Hg); SBP and DBP variability (SD of 24-h, daytime, and nighttime values); and day-to-night differences (nocturnal decline) in SBP and DBP.

Details and reliability of the noninvasive measurements of arterial stiffness (carotid augmentation index [AI] aortic and arm pulse wave velocities [PWV]) have been described in detail previously (3). Because of logistical and technical reasons, measurements of AI and PWV were obtained before and at the end of the interventions on only 30 (14 EX, 11 SR) and 24 (14 EX, 16 SR) of the subjects, respectively. For all measurements, analyses were performed by the same investigator who was blinded to the group assignment of the subject.

Aerobic exercise. Following orientation, subjects exercised on their own using a general program that has been described previously (22). During the initial few weeks, subjects were asked to walk for ~30 min/day at an intensity of 40% to 50% of their individually determined maximal heart rate for three to four days/week. As their exercise tolerance increased, subjects were asked to increase their walking to 40 to 45 min/day at an intensity equivalent to 65% to 80% of maximal heart rate for as many days a week as possible. Compliance to the exercise program was documented using heart rate monitors combined with activity logs that were returned to the laboratory for analysis every two weeks during the intervention period. Subjects assigned to this condition were asked not to alter their diet from their baseline levels.

Dietary sodium restriction. Moderate SR was employed using procedures described previously for peer-reviewed clinical trials (24,25). In accordance with current Joint National Committee (on Prevention, Detection, Evaluation and Treatment of High Blood Pressure) VI guidelines (14), subjects were asked to reduce their sodium intake to <100 mmol/day (<2.4 g of sodium or <6 g of sodium chloride) without altering their total caloric intake or otherwise changing their diet composition. After an initial orientation, subjects in this condition visited the laboratory every two weeks to receive dietary education and counseling by a research dietitian. This was exactly the same frequency at which EX group subjects underwent monitoring. Compliance to the intervention was documented by determining the reductions in dietary sodium intake as well as 24-h urinary sodium excretion rates. Subjects assigned to this intervention were asked to maintain their habitual activity at their individual baseline (sedentary) levels.

Data analysis and statistics. Differences at baseline and changes over the three-month EX and SR intervention periods in the dependent variables were assessed by two-way repeated measures analysis of variance (ANOVA). When indicated by a significant main effect or interaction, specific mean comparisons were performed to identify significant differences within and between each intervention. Relations between variables of interest were determined by univariate correlation and regression analysis. Analyses were performed only on subjects who completed the interventions. Data are presented as mean ± SD. Statistical significance was set at $p < 0.05$.

RESULTS

Subject characteristics. The mean ages of the women in the EX and SR groups were 62 ± 9 and 65 ± 10 years, respectively (NS). No differences were seen in any subject characteristic in the two groups at baseline, nor did any of these variables change in response to either intervention (Table 1). Total caloric intake, fat, carbohydrate, and protein macronutrient diet composition, and calcium, magnesium, and potassium micronutrient diet composition did not differ between the groups at baseline, and they did not change with either intervention (data not shown).

Intervention compliance. Subjects in the EX group walked for 5.8 ± 1.1 days/week, 40 ± 4 min/day, at an intensity equivalent to 70 ± 2% of their maximal heart rate over the three-month intervention period. Resting heart rate and submaximal exercise heart rate and perceived exertion were reduced, and maximal treadmill walking time was increased, in response to EX (all $p < 0.05$), but all were unchanged with SR (Table 2). Maximal oxygen consumption was not significantly changed with either intervention (Table 2). Urinary sodium excretion and dietary sodium intake were reduced over the three-month period in the SR group (both $p < 0.05$) but were unchanged in the EX group (Table 2).

Blood pressure. There were no group differences in baseline casual BP at rest (Fig. 1). The manual and semi-automated measures provided similar information for these baseline values as well as for the changes in response to the interventions (Fig. 1). Significant main effects and interactions were observed for each of the casual BP values: SBP, DBP, PP, and mean arterial blood pressure (MAP) were lower after compared with before EX and SR (all $p < 0.05$; Fig. 1); however, the decreases were consistently greater in response to SR compared with EX, especially for SBP (~16 vs. 5 mm Hg) and PP (~10 vs. 2 mm Hg) (both $p < 0.05$). In EX the numbers of subjects with ΔSBP >−2, −2 to 2, and >+2 mm Hg were 10 (55%), 3 (17%) and 5 (28%), respectively, whereas with SR it was 15 (88%), 2 (12%) and 0; thus, there was a higher percentage of "responders" to SR than to EX. The mean decreases in BP with SR were consistently greater (all $p < 0.05$) for the subgroup of women with Stage 1 systolic hypertension at baseline

Table 1. Selected Subject Characteristics

	Intervention			
	Exercise		Sodium Restriction	
	Preintervention	Postintervention	Preintervention	Postintervention
Body mass (kg)	72.4 ± 12.3	72.0 ± 12.8	73.0 ± 10.5	72.8 ± 10.3
Body fat (%)	41.8 ± 5.1	41.2 ± 6.3	42.1 ± 8.3	42.3 ± 8.5
Fat-free mass (kg)	41.6 ± 4.5	41.7 ± 4.4	41.7 ± 4.2	41.4 ± 4.1
BMI (kg/m^2)	28.0 ± 4.3	27.9 ± 4.4	28.1 ± 4.9	28.0 ± 4.8
Waist circumference (cm)	89.9 ± 11.5	89.7 ± 11.3	92.2 ± 12.8	92.4 ± 12.9
Waist:hip ratio	0.83 ± 0.06	0.82 ± 0.06	0.85 ± 0.08	0.85 ± 0.08
Fibrinogen (g/l)	3.2 ± 0.9	3.2 ± 0.9	3.4 ± 0.9	3.5 ± 0.9
Total cholesterol (mmol/l)	5.1 ± 0.8	5.0 ± 0.7	5.2 ± 0.9	5.2 ± 0.9
HDL (mmol/l)	1.6 ± 0.5	1.6 ± 0.5	1.5 ± 0.4	1.4 ± 0.4
HDL_3 (mmol/l)	1.2 ± 0.3	1.3 ± 0.3	1.2 ± 0.2	1.2 ± 0.3
HDL_2 (mmol/l)	0.3 ± 0.3	0.4 ± 0.5	0.3 ± 0.2	0.2 ± 0.2
LDL (mmol/l)	2.7 ± 0.6	2.7 ± 0.6	3.0 ± 0.8	3.0 ± 0.8
Triglycerides (mmol/l)	1.5 ± 0.6	1.6 ± 0.7	1.7 ± 0.7	1.8 ± 0.9
Insulin (pmol/l)	46.2 ± 18.3	48.6 ± 23.7	39.6 ± 16.7	38.4 ± 15.3
Glucose (mmol/l)	5.2 ± 0.5	5.2 ± 0.4	5.1 ± 0.5	5.1 ± 0.5
Plasma volume (ml)	2911 ± 465	3001 ± 351	3038 ± 116	3120 ± 312

Values are mean ± SD.
BMI = body mass index; HDL = high density lipoprotein; LDL = low density lipoprotein.

compared with the subgroup of women with initial SBP values in the high normal range; for EX the differences were not significant owing to the smaller reductions in BP associated with that intervention. In women taking hormone replacement compared with those not taking hormone replacement therapy, no baseline differences were seen in BP, nor were any differences detected in their responses to the respective interventions.

There were no group differences in baseline 24-h, daytime or nighttime ambulatory BP (Fig. 2). The 24-h SBP, DBP, MAP and PP levels were lower after compared with before SR due primarily to reductions in daytime levels (all $p < 0.05$; Fig. 2); nighttime levels were not significantly changed. In contrast, 24-h BP was not changed by EX. As such, all of the reductions in ambulatory BP with SR were greater than in response to EX (interaction $p < 0.05$). As was the case with casual BP at rest, the decreases in 24-h and daytime BP generally were greater in the women with Stage 1 systolic hypertension at baseline compared with those with values initially in the high normal range. Moreover, there was no difference in any ambulatory-determined BP value at baseline in women taking hormone replacement compared with women not taking hormone replacement therapy, nor were the responses to either intervention different in those two subgroups.

There were no baseline group differences in ambulatory-determined BP loads, variabilities or nocturnal declines. The SR decreased 24-h SBP load by ~35% (53 ± 26% vs. 35 ± 24%, $p < 0.01$) owing to a reduction in daytime SBP load (66 ± 27% vs. 45 ± 32%, $p < 0.01$) and a tendency for a decrease in nighttime SBP load (23 ± 27% vs. 10 ± 19%, $p = 0.06$). The SR tended to reduce 24-h (21 ± 27% vs. 12 ± 19%, $p = 0.06$) and daytime (26 ± 22% vs. 16 ± 16%, $p = 0.06$) DBP loads. Also, SR reduced 24-h SBP variability (15 ± 3 mm Hg vs. 12 ± 4 mm Hg, $p < 0.05$) due to a reduction in daytime SBP variability (13 ± 2 mm Hg vs.

Table 2. Responses to the Dietary Sodium Restriction and Exercise Interventions

	Intervention			
	Exercise		Sodium Restriction	
	Preintervention	Postintervention	Preintervention	Postintervention
Urinary sodium excretion (mmol/day)	119 ± 48	132 ± 49	124 ± 46	86 ± 32*
Sodium intake (mg/day)	2851 ± 1149	2923 ± 1118	2685 ± 559	1421 ± 512*
Resting heart rate (beats/min)	71 ± 7	68 ± 8*	66 ± 10	66 ± 9
Submaximal exercise heart rate (beats/min) (workload ~70% of baseline $\dot{V}O_{2max}$)	153 ± 10	143 ± 13*	144 ± 9	143 ± 14
Submaximal rating of perceived exertion	16.2 ± 1.5	14.4 ± 1.5*	16.6 ± 1.5	16.6 ± 2.0
Maximal treadmill time (min)	9.7 ± 1.6	10.4 ± 2.4*	10.4 ± 2.3	10.2 ± 2.3
$\dot{V}O_{2max}$ (ml/kg/min)	21.3 ± 4.1	22.4 ± 4.7	20.0 ± 4.5	20.0 ± 4.3

*Change from baseline $p < 0.05$. Values are mean ± SD.

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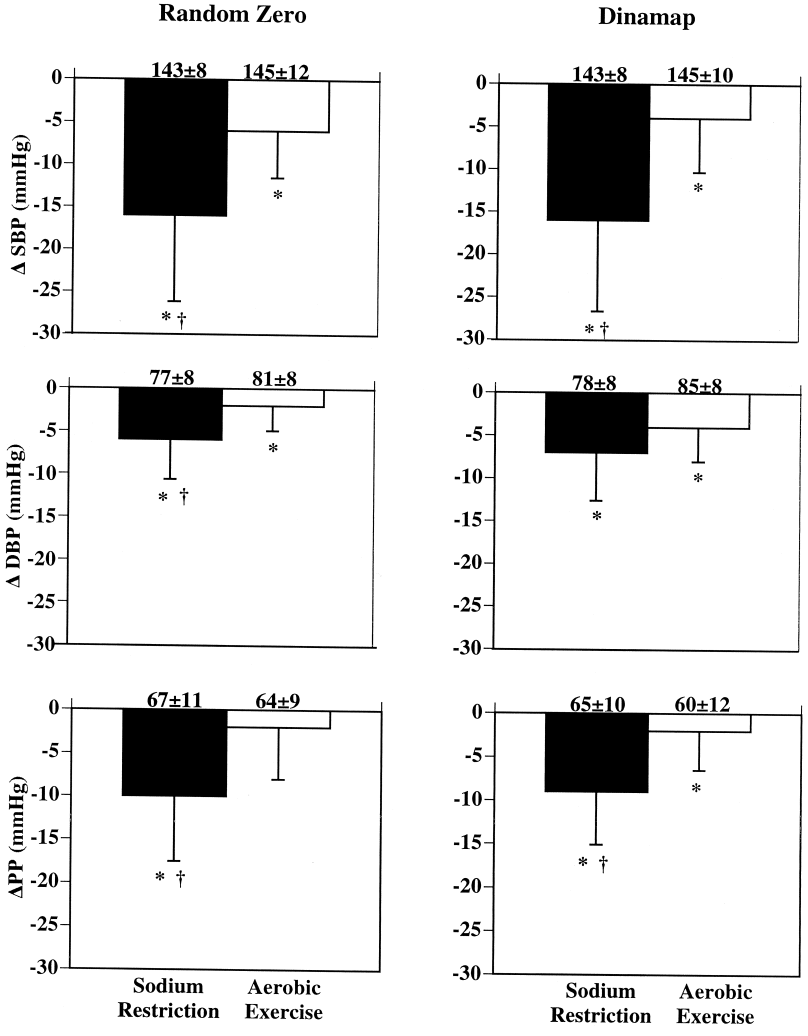

Figure 1. Mean ± SD changes in arterial blood pressure at rest measured either by a semiautomated device **(Dinamap)** or manually **(Random Zero)** in response to aerobic exercise and dietary sodium restriction. Baseline values are shown above each bar. *Change from baseline $p < 0.05$; †$p < 0.05$ vs. aerobic exercise. DBP = diastolic blood pressure; PP = pulse pressure; SBP = systolic blood pressure.

10 ± 2 mm Hg, $p < 0.01$); nighttime SBP variability and DBP variabilities were unchanged. In contrast, EX had no influence on either BP load or variability. Neither intervention altered the nocturnal decline in BP.

There were no significant correlations between compliance to either intervention and the corresponding reductions in BP.

Arterial stiffness. No baseline group differences were seen in any measure of arterial stiffness (Fig. 3). Carotid AI and aortic PWV (both $p < 0.05$) were reduced by SR but had no effect on arm PWV. In contrast, EX did not alter any measure of arterial stiffness.

Relations between reductions in BP and arterial stiffness. Among the pooled individual subjects, Δaortic PWV was significantly (all $p < 0.01$ or better) related to Δcasual SBP ($r = 0.53$), Δcasual PP ($r = 0.58$), Δ 24-h SBP ($r = 0.61$), Δ24-h PP ($r = 0.57$), Δ24-h DBP ($r = 0.59$), and Δ24-h MAP ($r = 0.61$). The Δcarotid AI was significantly (all $p <$ 0.01 or higher) related to both Δcasual SBP ($r = 0.49$) and Δcasual PP ($r = 0.46$).

DISCUSSION

The key findings from the present investigation were as follows. First, in otherwise healthy postmenopausal women with elevated SBP, SR lowered BP at rest more than did EX. Second, SR, but not EX, was effective in reducing 24-h levels of BP, as well as SBP load and variability. Third, SR reduced the stiffness of the large elastic arteries in the central circulation, whereas EX did not obviously do so in this population. Finally, the reductions in central arterial stiffness correlated with the corresponding reductions in SBP and PP, suggesting a potential mechanistic role in the hypotensive effects of SR. Overall, our results support the view that SR is more efficacious for lowering resting and 24-h BP and large artery stiffness than is EX in this highly prevalent, clinically important population.

Exercise intervention. The reductions in resting BP observed with EX were significant, although slightly less than what we had observed in a preliminary investigation in a similar population of women (22). This previous study,

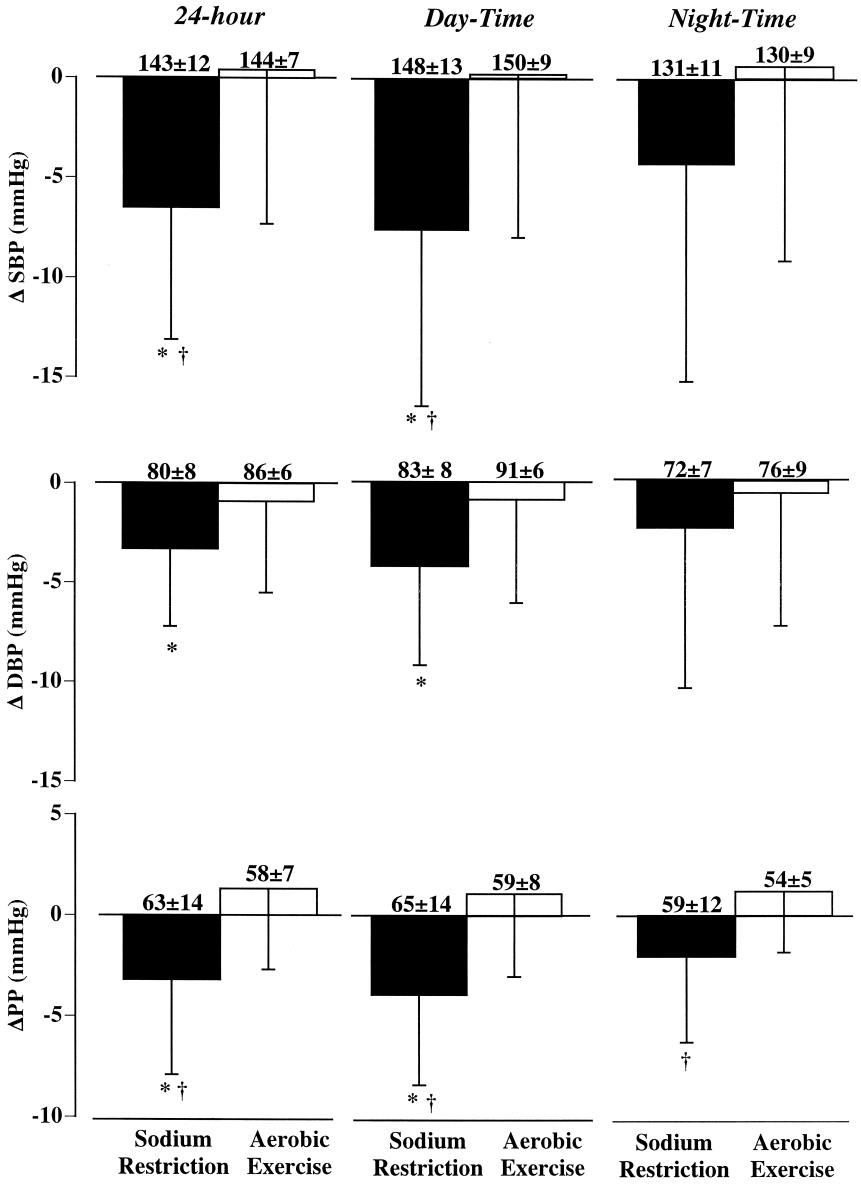

Figure 2. Mean ± SD changes in ambulatory-determined 24-h, daytime, and nighttime systolic blood pressure (SBP), diastolic blood pressure (DBP), and arterial pulse pressure (PP) in response to aerobic exercise and dietary sodium restriction. Baseline values are shown above each bar. *Change from baseline $p < 0.05$; †$p < 0.05$ vs. aerobic exercise.

however, was not a randomized trial. Moreover, the reductions in casual resting BP observed in the present study are well within the range previously reported in hypertensive adult humans in response to regular aerobic exercise (16). The facts that our population included women with high normal baseline SBP and normal DBP, and that nonhypertensive adults often demonstrate smaller responses to EX than their hypertensive counterparts (16), also may have contributed to our results. Moreover, it is possible that the higher exercise frequency in the present study (~6 days/week) compared with our previous investigation (~3 days/week) could have played a role (the influence of essentially daily exercise on BP has not been studied to our knowledge). Consistent with the findings of our earlier study (22), EX did not lower 24-h BP in the present investigation.

Dietary sodium restriction. The Trial of Nonpharmacologic Interventions in the Elderly (TONE) has shown that SR is an effective surrogate for BP control in some older patients on antihypertensive drug therapy (25). The results of the present study extend these previous findings in several ways. First, we have shown that SR is an effective lifestyle intervention for lowering resting SBP, DBP, and PP in postmenopausal women with mild to moderate elevations in baseline resting SBP who are not currently on antihypertensive medications. Second, the response is consistent, with ~90% of the subjects demonstrating >2 mm Hg reduction in SBP. Third, the magnitude of the reductions in SBP and PP with SR can be impressive in this population. For example, the mean 16 mm Hg decrease in resting SBP in the present study was at least twofold greater than that

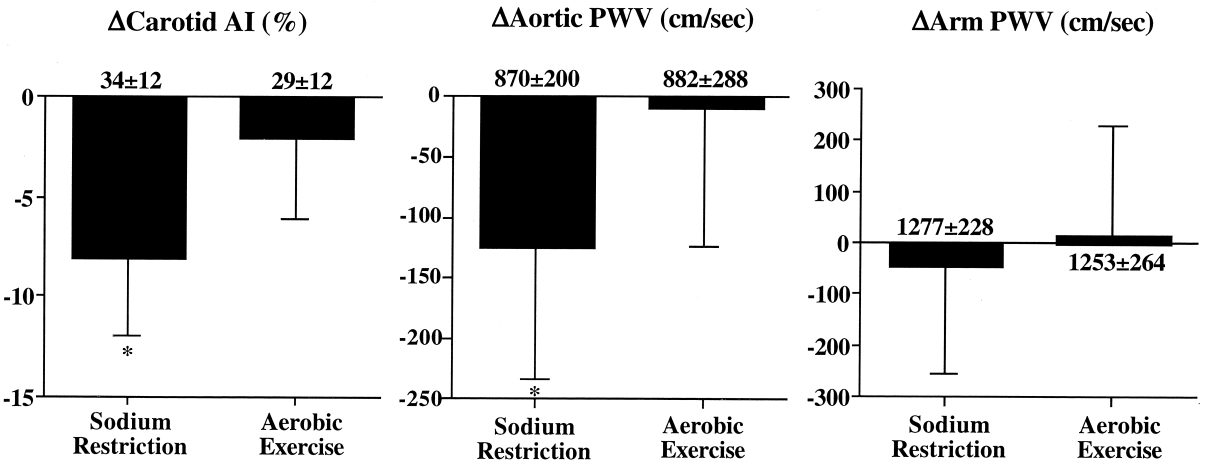

Figure 3. Mean ± SD changes in carotid augmentation index (AI), aortic pulse wave velocity (PWV), and arm PWV in response to aerobic exercise and dietary sodium restriction. Baseline values are shown above each bar. *Change from baseline $p < 0.05$.

observed previously (17,18,26,27). This occurred despite the fact that our study sample included a number of subjects with initial SBP values in the normotensive range, and this may have been the result, at least in part, of: a) excellent compliance to the intervention, b) the female gender of the sample (27), and c) the moderate level of sodium intake of the subjects at baseline, reductions from which are associated with greater decreases in SBP than are high baseline levels (27).

Fourth, in contrast to EX, SR lowered 24-h SBP, DBP, and PP. Similarly SR, unlike EX, reduced the mean daily SBP load and variability, which are independent predictors of CVD (28,29). Finally, these consistent and marked BP-lowering effects of SR were not obviously dependent on corresponding changes in body weight or composition, diet, or other CVD risk factors. We also found that hormone replacement therapy status, an increasingly important issue in this population, does not modulate these SR-induced reductions in resting BP.

Role of arterial stiffness. We observed significant reductions with SR in measures that reflect largely (aortic PWV) or in part (carotid AI) the stiffness of the large elastic arteries in the central circulation, whereas EX had no effect. Neither intervention affected peripheral arterial stiffness, which is not increased with age (1,3). Importantly, the changes in resting and 24-h SBP and PP consistently correlated with the corresponding changes in one or both measures of central arterial stiffness. These relations were stronger and more robust with aortic PWV, a more precise measure of the stiffness of the large elastic arteries in the central circulation per se than carotid AI. Associations between absolute levels of BP and measures of central arterial stiffness have been noted previously in cross-sectional comparisons of subjects differing in dietary sodium intake (19). To our knowledge, however, the present investigation is the first to provide experimental evidence for an association between these events in response to an intervention. As such, our findings provide initial support for the hypothesis that reductions in central arterial stiffness may play a mechanistic role particularly in the SBP- and PP-lowering effects of SR. It should be noted, however, that the decreases in mean BP with SR may have contributed to the reduction in aortic PWV independent of any SR-evoked changes in the intrinsic mechanical properties of the aorta.

Clinical implications. Our results are noteworthy in at least two ways. First, elevated SBP is prevalent in this population, particularly in the high normal and Stage 1 systolic hypertensive ranges (7). Given the number of postmenopausal women in the future based on current demographic projections, an increasing portion of the overall BP-related CVD risk in middle-aged and older adults will be contributed by this group. Second, lifestyle (behavioral) interventions are the recommended first-line therapeutic approach for middle-aged and older adults with mild to moderate elevations in SBP (13,14). As such, determining the relative efficacy of two of the most widely recommended lifestyle interventions, EX and SR, for lowering SBP in this population provides new insight for health professionals interested in BP control. Moreover, the fact that SR lowers 24-h SBP and PP mean levels, and SBP load and variability, all of which are significant independent predictors of CVD risk (28–31), further supports its clinical use in this patient population. Finally, the results of the present study provide new evidence supporting a mechanistic role for reductions in central arterial stiffness in the hypotensive effects of SR.

There are, however, at least two limitations of our study related to clinical implications. First, the population studied was almost entirely Caucasian. African-American and other minority women may respond differently to these interventions. Second, the three-month duration of our interventions precludes extrapolation of the results to long-term BP control, which obviously is the ultimate goal of therapy.

Conclusions. In postmenopausal women with elevated baseline SBP, SR has a greater hypotensive effect on resting and 24-h BP than EX. The greater reductions in SBP and

PP associated with SR may be mediated in part by a reduction in the stiffness of the large elastic arteries in the central circulation.

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