Comparison of effects of exercise and diuretic on left ventricular geometry, mass, and insulin resistance in older hypertensive adults

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Am J Physiol Regul Integr Comp Physiol 287: R360 –R368, 2004. First published April 29, 2004; 10.1152/ajpregu.00409.2003.—To compare the effects of exercise training and hydrochlorothiazide on left ventricular (LV) geometry and mass, blood pressure (BP), and hyperinsulinemia in older hypertensive adults, we studied 28 patients randomized either to a group (age 66.4 ± 1.3 yr; n = 16) that exercised or to a group (age 65.3 ± 1.2 yr; n = 12) that received hydrochlorothiazide for 6 mo. Endurance exercise training induced a 15% increase in peak aerobic power. The reduction in systolic BP was twofold greater with thiazide than with exercise (26.6 ± 12.2 vs. 11.5 ± 10.9 mmHg). Exercise and thiazide reduced LV wall thickness (LV mass index: 14% in each group), and the LV wall thickness-to-radius ratio (h/r) similarly (exercise: before 0.48 ± 0.2, after 0.42 ± 0.01; thiazide: before 0.47 ± 0.04, after 0.40 ± 0.04; P = 0.017). The reductions in systolic BP and h/r were correlated in the exercise group (r = 0.70, P = 0.005) but not in the thiazide group. Exercise training reduced glucose-stimulated hyperinsulinemia (before: 13.65 ± 2.6 vs. 9.84 ± 1.5 mU·ml−1·min−1; P = 0.04) and insulin resistance. Thiazide did not affect plasma insulin levels. The results suggest that although exercise is less effective in reducing systolic BP than thiazide, it can induce regression of LV hypertrophy similar in magnitude to thiazide. Unlike hydrochlorothiazide, exercise training can improve insulin resistance and aerobic capacity in older hypertensive people.

SYSTOLIC HYPERTENSION is a common disorder in older men and women. It is a major but modifiable risk factor for cardiovascular morbidity and mortality in old age (3, 14). Left ventricular (LV) hypertrophy (LVH), commonly observed in hypertension, is a powerful predictor of cardiovascular morbidity and mortality independent of the level of blood pressure (BP) in hypertensive adults (2, 14, 29). Reduction of systolic BP can reduce the risk of stroke and heart failure in hypertensive older individuals (2, 4). Furthermore, regression of LVH is likely to reduce cardiovascular risks in hypertension (40).

Endurance exercise training has been recommended for management of hypertension because it is effective in reducing BP (16, 19, 46, 47). Recent studies showed that exercise training may also reduce LV concentric remodeling and LVH (18, 27, 51). As endurance exercise training improves hyperinsulinemia and insulin resistance (20, 23, 39), it is plausible that this adaptation may play a role in regression of LV remodeling and LVH because insulin is a stimulus for the development of cardiac hypertrophy (30, 36, 37). It is not clear, however, whether exercise can induce regression of LV remodeling in older adults or whether it is as effective as antihypertensive medications in reducing LV mass in older hypertensive patients.

To address these issues, we studied older adults with mild hypertension randomized into exercise and thiazide groups to characterize 1) adaptive changes in LV mass, geometry, and function and 2) metabolic and hormonal adaptations in response to exercise training and to determine whether these adaptive responses were associated with alterations in LV mass, geometry, and function. Our hypotheses were that endurance exercise training in older adults with mild hypertension can reduce BP, relative LV wall thickness expressed as a reduction in the LV wall thickness-to-radius ratio (h/r), LVH, and hyperinsulinemia and that the effect of endurance exercise training on regression of LVH and LV remodeling is similar in magnitude to that induced by thiazide diuretic. In this study, we focused on LV relative wall thickness in addition to LV mass because in hypertension LV remodeling appears to be as good or even better than LVH as a predictor of LV dysfunction and cardiac risk factor (30, 49).

METHODS

Subject Recruitment

We screened 639 subjects. Among these, we identified and recruited 51 eligible subjects with grade I and II hypertension (4) who fulfilled the following criteria: 1) age ≥55 yr; 2) no current treatment with antihypertensive medications; 3) absence of symptomatic coronary artery and peripheral arterial diseases, history of myocardial infarction or coronary artery bypass surgery, aortic aneurysm, significant valvular heart disease, congestive heart failure, or noncardiac chronic conditions that might interfere with exercise testing or training; 4) absence of geographic or job constraints that might prevent the volunteer from regular participation in a supervised exercise training program; 5) no current tobacco abuse; and 6) sedentary lifestyle (defined as regular exercise less than once per week). The other 588 respondents to our solicitation were either normotensive or did not fulfill the inclusion criteria (most were treated with antihypertensive medications).

Among the volunteers that were recruited, none was taking any cardiac medications and none had segmental LV wall motion abnormalities at rest or unequivocal ECG changes suggestive of myocardial ischemia during maximal exercise test. All volunteers signed an informed written consent form. The study protocol was approved by...
the Human Studies and the Radioactive Drug Research Committees of Washington University School of Medicine. The volunteers were then randomized to an exercise group (n = 32) and a medication group (n = 19). In the exercise group, 16 volunteers (66.4 ± 1.3 yr, 13 men and 3 women) completed the training program. The other 16 did not finish the exercise program. Of these, nine dropped out for personal (nonmedical) reasons, two developed orthopedic problems precluding continuation of training, one developed LV dysfunction and heart failure, one had a new onset of exercise-induced angina with ECG evidence of myocardial ischemia, and three did not show a reduction of BP and were therefore referred to their personal physicians for treatment of hypertension. In the thiazide group, 12 volunteers (65.3 ± 1.2 yr, 10 men and 2 women) completed the study. Among the other seven volunteers who dropped out, four withdrew from the study for personal reasons and the other three did not show any decrease in BP even with the higher doses of hydrochlorothiazide and were referred to their physicians for further treatment. The volunteers in both groups who dropped out did not return for final evaluation. Among the subjects who completed the study, eight in the exercise group and seven in the thiazide group had an impaired or diabetic glucose tolerance test on initial evaluation.

Exercise Tests and Determination of Maximal O2 Consumption

Exercise tests were performed on a treadmill for determination of maximal O2 consumption (V\textsubscript{O2max}) as described previously (24). Briefly, during warm-up the subjects walked flat on a treadmill at a pace adjusted to elicit ~70% of the maximal age-predicted heart rate for 3–4 min. The subjects then began to exercise at the same speed with incremental increases in grade of 2% every 2 min until exhaustion or development of symptoms or signs including ECG changes that were considered to make it unsafe to continue. O2 consumption (V\textsubscript{O2}) was measured with the use of a metabolic measurement cart (MAX 1, FITCO, Farmingdale, NY) for determination of V\textsubscript{O2max}. V\textsubscript{O2max} was established when two of the following criteria were met: 1) attainment of plateau of VO\textsubscript{2} with increasing exercise intensity, 2) respiratory exchange ratio (RER) of >1.10, and 3) the highest VO\textsubscript{2} recorded during exhaustion lower than the expected value based on the speed and elevation of the treadmill. Because many subjects did not meet the criteria for V\textsubscript{O2max} the data are presented as peak VO\textsubscript{2} instead of V\textsubscript{O2max}.

Assessment of LV Size, Geometry, and Function

We used two-dimensional (2D) and 2D-guided M-mode echocardiography for evaluation of LV size, geometry, and function as previously described (51). LV end-diastolic and end-systolic diameters (EDD and ESD, respectively) and posterior wall and septal thicknesses (PWT and Sep, respectively) were measured with the use of the standard formulas (10, 45, 51). End-diastolic and end-systolic wall thicknesses (PWT and Sep, respectively) were measured with the use of 2D-guided M-mode echocardiography for evaluation of LV size, geometry, and function as described previously (51). Echocardiographic data of LV size, geometry, and function were collected using a V\textsubscript{O2} and were referred to their physicians for further treatment. The volunteers in both groups who dropped out did not return for final evaluation. Among the subjects who completed the study, eight in the exercise group and seven in the thiazide group had an impaired or diabetic glucose tolerance test on initial evaluation.

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Arterial Stiffness

We evaluated arterial stiffness with the use of pulse pressure (PP) normalized for stroke volume (SV). PP has been shown to be affected by arterial stiffness (33, 41), SV, and heart rate. Because there were no significant changes in heart rate in response to the interventions in this study, we used the ratio PP/SV as an index of arterial stiffness. Previous studies demonstrated that PP normalized for SV is a valid and reliable surrogate for arterial stiffness (9, 13). We used arterial elastance (E\textsubscript{a}) as an index for the effective arterial load (48). E\textsubscript{a} was calculated as the ratio of end-systolic pressure to SV (48).

Body Composition and Diet

We evaluated the effects of the interventions on body composition with the use of hydrostatic weighing, as described previously (26). Waist circumference was measured at the midaxillary line as the smallest measurement between the iliac crest and inferior border of the rib cage (25). Abdominal circumference was measured 2 cm above the umbilicus. Anthropometric measurements were made by the same technician before and after the interventions. No specific dietary intervention was implemented. All subjects were advised to continue their daily sodium and caloric intake. Those with high levels of plasma total or low-density lipoprotein-cholesterol, however, were advised to lower their daily fat intake and were also referred to their primary care physicians. Plasma cholesterol and triglycerides were measured (35) with the use of automated enzymatic commercial kits (Miles/Technician, Tarrytown, NY). Plasma high-density lipoprotein-cholesterol was measured by the dextran sulfate-magnesium precipitation procedure (54).

Blood Pressure

Casual BP measurement. Each volunteer had at least two BP measurements, 1 wk apart, in the morning by standard mercury sphygmomanometry after 10 min of rest. Three BP readings were recorded in both arms during each visit. All BP recordings were averaged.

Ambulatory BP monitoring. Each volunteer had a 24-h ambulatory BP recording with the use of a portable BP recording device (Space Lab Ambulatory Blood Pressure Monitor, model 90207ABP) before and after training or drug therapy. The effect of the interventions on ambulatory BP profile was evaluated with the following variables: 1) average whole day BP, 2) average BP during daytime, 3) average BP during nighttime, and 4) BP load. BP load was defined as the proportion (%) of elevated BP recordings above an arbitrary cut-off threshold over the total number of readings (55). We considered the cut-off threshold to be 140/90 mmHg for daytime hours and 120/80 mmHg during sleep hours. BP evaluations at the completion of the study (at rest and 24-h monitoring) were made 24 h after the last bout of exercise or the last dose of hydrochlorothiazide.

Oral Glucose Tolerance Test and Plasma Insulin Assay

A 75-g oral glucose tolerance test (OGTT) was administered to determine glucose tolerance and insulin response to glucose stimulation. The participants were instructed to consume a weight-maintain-
ing diet containing a minimum of 150 g of carbohydrate for 3 days preceding the OGTT. A diet history was obtained to establish compliance. The test was performed in the morning ~14 h after the previous meal. Blood samples were obtained in the fasting state and 30, 60, 90, 120, and 180 min after ingestion of glucose. Blood glucose was measured by the standard glucose oxidase method (Beckman glucose analyzer). Insulin was assayed by the double-antibody radioimmunoassay specific for insulin with the method described by Morgan and Lazarow (34). The National Diabetes Data Group criteria were used to classify subjects as having impaired or diabetic glucose tolerance (50). OGTT was administered ~16 h after the last bout of exercise in the trained state. In the exercise group, only the subjects \( n = 8 \) who were found to have an impaired or diabetic glucose tolerance test during initial evaluation underwent a final OGTT. In the thiazide group, we performed a glucose tolerance test on all volunteers. The rationale for testing the entire group was that impaired glucose tolerance is considered to be one of the side effects of thiazide diuretics. The areas under the glucose and insulin curves were calculated with the trapezoidal rule. The product of glucose and insulin area was used as an estimate of peripheral insulin resistance (32).

### Plasma Volume

Blood and plasma volume were determined with the use of the standard radionuclide technique \( ^{51} \text{Cr}-\text{labeled red blood cells and } ^{125} \text{I}-\text{labeled human serum albumin; Ref. 15a} \) before and after training only in the exercise group.

### Plasma Catecholamines, Renin Activity, and Angiotensin

For plasma catecholamine assay, venous blood samples were obtained from an intravenous catheter placed in the forearm after the subjects had rested for 30 min in the recumbent position and 10 min in the standing position. Heart rate and BP were measured simultaneously. Plasma norepinephrine and epinephrine concentrations were determined with the use of the single isotope derivative (radioenzymatic) method as described previously (31). Plasma renin activity and angiotensin concentration were measured with the use of the standard radioimmunoassay procedure and commercially available kits (Biotech). Plasma samples were taken under basal conditions in the supine position after 30 min of rest and in the upright position after the subjects had been standing for 10 min concurrent with measurements of BP and heart rate before and after training. Plasma norepinephrine, epinephrine, renin, and angiotensin levels were not measured in the thiazide group.

### Interventions

**Exercise.** The training program was for 6 mo and consisted of two phases: a flexibility program (1 mo) and an endurance exercise training program (5 mo).

**FLEXIBILITY EXERCISE PROGRAM.** The purpose of the flexibility program was to overcome the significant reduction in flexibility (i.e., range of motion) of the major joints that is common in this age group (8). The participants were expected to attend three sessions of the stretching program per week, for ~40 min per session, for 1 mo. The flexibility exercises involved all the major joints and muscle groups to provide stretch to connective tissue in all areas likely to have reduced flexibility. The program was individualized to emphasize exercises designed to help correct specific problems (i.e., stiffness and restricted range of motion). A shortened individualized stretching flexibility program was continued during the endurance exercise training program, as part of both the warm-up and cool-down exercises.

**ENDURANCE EXERCISE TRAINING.** After completion of the flexibility program the volunteers participated in a 5-mo-long program of endurance exercise training. A 10-min warm-up with stretching, light calisthenics, and walking was followed by 30–50 min of more strenuous exercise consisting of brisk walking and/or jogging and/or cycle ergometer and treadmill exercise. Initially, the exercise intensity was adjusted to require between 60% and 70% of heart rate reserve that usually consisted of brisk walking only. As their exercise capacity improved, the participants progressed to alternate walking and jogging exercise, a greater resistance during cycle ergometer exercise, or higher speed and elevation during treadmill exercise. The desired intensity of the exercise, monitored by heart rate, was regulated by appropriate setting of the resistance on the cycle ergometer or by adjusting walking-running speed or elevation of the treadmill. After 2 mo, the intensity of exercise was increased to 70–85% of heart rate reserve. The volunteers were required to attend at least three exercise sessions per week. The duration of the exercise sessions, not including the warm-up, was progressively increased from 30 to 50 min during the first 2 mo and then maintained at 50 min/session for the remaining 3 mo. The participants were also encouraged to walk for 40–50 min on the days on which they did not attend an exercise session. The exercise prescription, which was evaluated weekly, was carefully geared to the subjects’ exercise capacity and their reaction to the exercise in terms of fatigue, musculoskeletal symptoms, and BP response.

**Thiazide diuretic.** Each participant in the medication group received 25–50 mg of hydrochlorothiazide daily with a supplemental 20 meq of K+ daily for 6 mo. The rationale for using thiazide was based on the recommendation by the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure for older people with mild-to-moderate hypertension (3). Compliance with medication was verified by the “pill count.” This group was seen in our clinical facilities on a regular basis (see Safety Monitoring).

### Safety Monitoring

Subjects in both groups were evaluated on a weekly basis in our clinical facilities to evaluate their BP responses to the interventions and their clinical status. In the thiazide group, blood samples were obtained on a regular basis to monitor serum K+ level and adjust the dose of oral K+ supplement if indicated. The participants who were considered to have a suboptimal response to treatment (i.e., a decrease in SBP ≤5 mmHg) underwent an additional 24-h BP monitoring. If 24-h monitoring confirmed the resting BP data, the dose of hydrochlorothiazide was increased to 50 mg daily in the thiazide group and the duration of exercise sessions and frequency of exercise were increased in the exercise group. If there was no satisfactory response within 2 wk, the subjects were withdrawn from the study and referred to their physicians for further treatment.

### Statistics

A two-way analysis of variance was used to assess the effects of interventions in the two groups. Furthermore, we used a group \( t \)-test to compare the changes (after ~ before) in the variables in response to exercise and drug therapy between the two groups. Student’s paired \( t \)-test was also used where appropriate. Least squares linear regression analysis was used to evaluate the relationship between the physiological variables. We used nonparametric tests (Wilcoxon or Mann-Whitney rank sum test) for all data that either were not normally distributed or were of unequal variances. Data are shown as means ± SE.

### RESULTS

#### Training

The subjects trained 2.6 ± 0.16 days/wk (range 1.6–4.1 days/wk) at an intensity of 83.0 ± 1.2% of their maximal heart rate. The weekly energy expenditure during exercise sessions averaged 938 ± 124 kcal.
Thiazide Diuretic

Twelve subjects received 25 mg of hydrochlorothiazide daily, but in two subjects the dosage had to be increased to 50 mg daily to attain an adequate reduction (i.e., >5 mmHg) in SBP.

Casual Blood Pressure

Baseline SBP and DBP levels were similar in the two groups (Table 1). There were significant decreases in resting SBP and DBP in both groups in response to the interventions. However, the magnitude of decrease in casual SBP was significantly greater ($P < 0.05$) in the thiazide group. Hydrochlorothiazide resulted in a decrease in casual SBP by 26.6 ± 12.2 mmHg, whereas the decrease in SBP in the exercise group was almost 50% smaller (11.5 ± 10.9 mmHg). In contrast, the decreases in DBP did not differ significantly between the groups. Rate-pressure product (product of casual systolic pressure and heart rate) was lower in the thiazide group. Both interventions resulted in a significant decrease in the rate-pressure product.

Twenty-Four-Hour Ambulatory Blood Pressure

Two groups had similar 24-h SBP and DBP at baseline (i.e., before the interventions; Table 1). Although exercise training and hydrochlorothiazide reduced both SBP and DBP, the latter was three to four times more effective compared with the former in reducing 24-h, daytime, and nighttime average SBP. We found a strong significant correlation between the reductions in casual SBP and 24-h SBP in the exercise ($r = 0.62$, $P = 0.014$) and thiazide ($r = 0.70$, $P = 0.010$) groups. The reduction in average DBP was only slightly greater in the thiazide group than in the exercise group, and the differences were not significant statistically. The baseline systolic load was significantly greater in the exercise group than in the thiazide group. Both interventions reduced SBP load. The difference in the magnitude of the reduction in the systolic load between the two groups did not reach statistical significance.

Maximal Heart Rate and Blood Pressure

Exercise training had no significant effect on heart rate (exercise group: 155.8 ± 6.2 vs. 154.9 ± 4.4 beats/min, $P = 0.81$; thiazide group: 154.9 ± 3.7 vs. 155.2 ± 3.9 beats/min, $P = 0.92$) or SBP and DBP during maximal exercise. DBP was lower during maximal exercise in the diuretic group, but the difference was not significant (Table 1). The difference in the changes ($\Delta$) in SBP during maximal exercise (before vs. after) in response to the interventions was of borderline significance (ΔSBP: exercise 4.4 ± 6.8 mmHg, thiazide −13.8 ± 5.4 mmHg; $P = 0.06$).

Peak Aerobic Power

The initial peak VO$_2$ values were similar in the two groups. In the exercise group, peak VO$_2$ increased 15%, from 1.876 ± 0.14 to 2.16 ± 0.14 l/min ($P < 0.001$). When normalized for body weight, peak VO$_2$ increased from 21.5 ± 1.9 to 24.2 ± 1.3 ml·kg$^{-1}$·min$^{-1}$ ($P < 0.001$) in response to training. Peak VO$_2$ did not change in the thiazide group (21.2 ± 3 vs. 22.1 ± 2 ml·kg$^{-1}$·min$^{-1}$; $P = 0.57$). Seven subjects from each group were able to attain true VO$_2$max.

LV Size, Geometry, and Function

In the exercise group, six subjects had concentric LVH, five had eccentric LVH, two had concentric remodeling, and one had a normal LV geometry before training (Table 2). In the diuretic group, four subjects had concentric LVH, four had eccentric LVH, two had concentric remodeling, and two had a normal LV geometry during the initial evaluation. The baseline values for LV wall thickness (PWT and Sep), EDD and ESD, h/r, LV mass index, LV EDV and ESV, LV end-systolic wall stress, and EF were similar in the exercise and thiazide groups. LV PWT and Sep, LV mass normalized for body surface area or FFM, and h/r decreased significantly in both groups in response to the interventions. There were no significant differences in the magnitude of the reduction in LV wall thickness, LV geometry (h/r), or LV mass normalized for body surface area (exercise 14%, thiazide 14%) or height (exercise 15%, thiazide 16%) between the exercise and thiazide groups.

There was a significant and strong correlation ($r = 0.701$, $P = 0.005$) between reduction in casual SBP and the LV remodeling (h/r) in response to exercise training (Fig. 1A). Similarly, there was a significant relationship between reductions in SBP and LV PWT ($r = 0.60$, $P = 0.024$) in the exercise group (Fig. 1B). In contrast, in the group treated with

<table>
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<th>Variable</th>
<th>Exercise</th>
<th>Thiazide</th>
<th>Initial</th>
<th>Final</th>
<th>Initial</th>
<th>Final</th>
<th>$P$ (group)</th>
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<td>151.0 ± 2.3</td>
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Values are means ± SE. SBP, systolic blood pressure; DBP, diastolic blood pressure; max, maximal exercise; HR, heart rate. *$P < 0.05$, exercise vs. thiazide (after intervention).
hydrochlorothiazide, we found no correlation between reductions of SBP and LV remodeling (r = 0.158, P = 0.62) or LV wall thickness (r = 0.15, P = 0.62).

LV EDV and ESV did not change significantly in response to either intervention. Neither exercise training nor thiazide had any significant effect on EF, Ees, SV, cardiac output, or end-systolic wall stress (Table 2). However, the changes (before vs. after) in Ees in response to interventions were different, with the thiazide group showing a greater decrease compared with the exercise group (ΔEes; exercise group −0.20 ± 0.3 vs. thiazide group −1.42 ± 0.49 mmHg/ml; P = 0.04). Total peripheral resistance decreased in both groups. However, the differences were not statistically significant. There was a strong inverse correlation between the change (before vs. after) in EF and LV end-systolic wall stress both in the exercise group (r = −0.79, P < 0.001) and in the thiazide group (r = −0.90, P < 0.001), showing that those subjects who decreased their LV wall stress in response to the interventions had a greater increase in EF (r = −0.82, P < 0.001). We found an inverse relationship between changes in end-systolic wall stress and those in the relative LV wall thickness (h/r) in the entire study group (r = −0.64; P < 0.001, n = 26).

**Arterial Stiffness**

The changes in PP normalized for SV (Table 2) in response to the interventions were not significant. Effective arterial load (i.e., Ea) also did not change significantly in response to either exercise or thiazide.

**Body Composition and Anthropometric Data**

There were no significant changes in body weight or FFM in response to either exercise training or thiazide (Table 3). There were no significant changes in body mass index (BMI) or abdominal, waist, and hip circumferences in the two groups. Ten subjects in the exercise group and seven in the medication group had a baseline BMI >25%. We found that those subjects in the exercise group with a baseline BMI >25% exhibited a lower abdominal circumference (before 109 ± 12 cm, after 105 ± 9 cm; P = 0.024, n = 10) and waist circumference (before 106 ± 13 cm, after 103 ± 10 cm; P = 0.045, n = 10) in response to training. Changes in BMI did not correlate with changes in BP.

---

**Table 2. Effects of exercise and antihypertensive drug on LV size, geometry, and function**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Exercise</th>
<th>Thiazide</th>
<th>P (group)</th>
<th>P (status/pre/post)</th>
<th>P (group × status)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>Final</td>
<td>Initial</td>
<td>Final</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVPWT, mm</td>
<td>11.4 ± 0.3</td>
<td>10.0 ± 0.4</td>
<td>11.2 ± 0.7</td>
<td>9.6 ± 0.8</td>
<td>0.58</td>
</tr>
<tr>
<td>LVSeptum, mmHg</td>
<td>11.2 ± 0.50</td>
<td>10.1 ± 0.60</td>
<td>11.2 ± 0.5</td>
<td>9.8 ± 0.7</td>
<td>0.76</td>
</tr>
<tr>
<td>h/r</td>
<td>0.48 ± 0.02</td>
<td>0.42 ± 0.01</td>
<td>0.47 ± 0.04</td>
<td>0.40 ± 0.04</td>
<td>0.70</td>
</tr>
<tr>
<td>LVM/FFM, g/kg × 10⁻³</td>
<td>4.20 ± 0.17</td>
<td>3.46 ± 0.31</td>
<td>4.32 ± 0.34</td>
<td>3.48 ± 0.32</td>
<td>0.813</td>
</tr>
<tr>
<td>LVMI, g/m²¹⁰</td>
<td>120.4 ± 5.9</td>
<td>103.6 ± 9.8</td>
<td>116.0 ± 8.0</td>
<td>99.3 ± 8.5</td>
<td>0.60</td>
</tr>
<tr>
<td>*LVMI, g/m²</td>
<td>55.0 ± 3.0</td>
<td>47.0 ± 4.5</td>
<td>55.0 ± 4.6</td>
<td>46.0 ± 4.4</td>
<td>0.88</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>48.7 ± 1.8</td>
<td>48.6 ± 2.1</td>
<td>48.6 ± 2.1</td>
<td>49.1 ± 1.8</td>
<td>0.93</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>30.7 ± 1.6</td>
<td>29.6 ± 1.3</td>
<td>29.4 ± 1.8</td>
<td>31.1 ± 2.0</td>
<td>0.95</td>
</tr>
<tr>
<td>LVse, g/cm²</td>
<td>44.0 ± 2.4</td>
<td>41.4 ± 2.4</td>
<td>46.3 ± 5.3</td>
<td>45.8 ± 5.0</td>
<td>0.38</td>
</tr>
<tr>
<td>LVEDV, ml</td>
<td>108.8 ± 7.7</td>
<td>109.1 ± 8.8</td>
<td>110.0 ± 8.3</td>
<td>108.7 ± 10.0</td>
<td>0.88</td>
</tr>
<tr>
<td>LVESV, ml</td>
<td>36.2 ± 3.5</td>
<td>32.3 ± 3.1</td>
<td>33.9 ± 4.9</td>
<td>37.8 ± 5.7</td>
<td>0.71</td>
</tr>
<tr>
<td>SV, ml</td>
<td>72.6 ± 4.7</td>
<td>75.6 ± 6.3</td>
<td>74.8 ± 5.9</td>
<td>72.2 ± 4.6</td>
<td>0.96</td>
</tr>
<tr>
<td>Q, l/min</td>
<td>4.93 ± 0.38</td>
<td>5.02 ± 0.45</td>
<td>4.81 ± 0.37</td>
<td>4.84 ± 0.25</td>
<td>0.69</td>
</tr>
<tr>
<td>TPR, dyn/cm⁻⁵</td>
<td>1801 ± 156</td>
<td>1634 ± 165</td>
<td>1888 ± 138</td>
<td>1479 ± 92</td>
<td>0.82</td>
</tr>
<tr>
<td>EF, %</td>
<td>66.5 ± 1.8</td>
<td>68.5 ± 1.6</td>
<td>69.8 ± 2.3</td>
<td>66.4 ± 2.7</td>
<td>0.91</td>
</tr>
<tr>
<td>Ees, mmHg/ml</td>
<td>3.90 ± 0.48</td>
<td>3.7 ± 0.31</td>
<td>4.59 ± 0.54</td>
<td>3.16 ± 0.28</td>
<td>0.97</td>
</tr>
<tr>
<td>Ea, mmHg/ml</td>
<td>1.80 ± 0.17</td>
<td>1.66 ± 0.17</td>
<td>1.83 ± 0.11</td>
<td>1.51 ± 0.11</td>
<td>0.62</td>
</tr>
<tr>
<td>PP/SV, mmHg/ml</td>
<td>0.95 ± 0.10</td>
<td>0.90 ± 0.1</td>
<td>0.94 ± 0.06</td>
<td>0.72 ± 0.05</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Values are means ± SE; LV, left ventricular; h/r, LV wall thickness-to-radius ratio; LVEDD, LV end-diastolic diameter; LVESD, LV end-diastolic diameter; LVES, LV end-systolic diameter; LVM/FFM, LV mass/fat-free mass; LVMI, LV mass index; LVPWT, LV posterior wall thickness; LVSeptum, LV septal thickness; LVse, LV end-systolic wall stress; PP, pulse pressure; SV, stroke volume; EF, ejection fraction; EDV, end-diastolic volume; ESV, end-systolic volume; Ea, arterial elastance; Q, cardiac output; TPR, total peripheral resistance.

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Fig. 1. Effects of exercise training on regression of left ventricular (LV) remodeling. There is a significant correlation between the magnitude of the reduction in systolic blood pressure (SBP) and the LV wall thickness-to-radius ratio (h/r) (n = 14).
Metabolic Adaptations

The plasma lipid profile did not change in either group (Table 3). In those volunteers from the exercise group with abnormal glucose tolerance, fasting plasma glucose (before 129.9 ± 9.0, after 128.0 ± 11.5 mg/dl; \( P = 0.88 \)) and insulin (before 19.5 ± 3.0, after 14.1 ± 1.4 \( \mu \text{U/ml} \); \( P = 0.18 \)) did not change significantly in response to training. The glucose area under the curve did not change (before 35,851.9 ± 2,888.8 vs. after 34,893.8 ± 2,416.0 mg·dl\(^{-1}\)·min; \( P = 0.61 \)), but the insulin area was significantly smaller (before 13,645.1 ± 2,564.4 vs. after 9,840.0 ± 1,457.9 \( \mu \text{U·ml}^{-1} \cdot \text{min}^{-1} \); \( P = 0.04 \); Fig. 2A) in response to training. Furthermore, training induced a significant reduction (34%) in the product of glucose and insulin areas (before 8,003.0 ± 661.1, after 7,859.3 ± 730.6 \( \mu \text{U·ml}^{-1} \cdot \text{min}^{-1} \); \( P = 0.82 \)), or the product of glucose and insulin areas (before 189.5 ± 22.05 \( \times 10^6 \) U, after 189.12 ± 30.84 \( \times 10^6 \) U; \( P = 0.43 \)).

Plasma Hormones

There were no changes in plasma renin activity or angiotensin level either in the recumbent or upright position in response to exercise training (Table 4). Exercise training also did not result in any significant changes in norepinephrine concentration (supine or upright). However, we found that plasma epinephrine was higher in the upright position after training.

Plasma Volume

Exercise training had no significant effect on plasma volume (before training 4,270 ± 212 vs. after training 4,180 ± 200 ml; \( P = 0.30 \)).

DISCUSSION

The findings of this study suggest that endurance exercise training of mild-to-moderate intensity can induce a partial regression of LHV with reductions in LV mass index and relative wall thickness in older adults with mild hypertension. Our data also suggest that the extent of this reversal is likely to

Table 3. Metabolic and body composition changes in response to exercise and thiazide diuretic

<table>
<thead>
<tr>
<th>Variable</th>
<th>Initial</th>
<th>Final</th>
<th>Exercise</th>
<th>Thiazide</th>
<th>( P ) (group)</th>
<th>( P ) (status/pre/post)</th>
<th>( P ) (group ( \times ) status)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol, mg/dl</td>
<td>196.4 ± 16.5</td>
<td>206.3 ± 20.1</td>
<td>212.2 ± 11.2</td>
<td>211.8 ± 9.4</td>
<td>0.44</td>
<td>0.10</td>
<td>0.12</td>
</tr>
<tr>
<td>LDL-cholesterol, mg/dl</td>
<td>116.8 ± 12.3</td>
<td>126.6 ± 14.1</td>
<td>127.5 ± 8.6</td>
<td>125.9 ± 7.7</td>
<td>0.65</td>
<td>0.69</td>
<td>0.58</td>
</tr>
<tr>
<td>HDL-cholesterol, mg/dl</td>
<td>52.3 ± 5.7</td>
<td>51.9 ± 4.2</td>
<td>45.4 ± 4.4</td>
<td>51.9 ± 5.3</td>
<td>0.53</td>
<td>0.59</td>
<td>0.45</td>
</tr>
<tr>
<td>Triglyceride, mg/dl</td>
<td>148.0 ± 27.3</td>
<td>138.6 ± 26.6</td>
<td>204.5 ± 50.1</td>
<td>170.0 ± 26.3</td>
<td>0.26</td>
<td>0.57</td>
<td>0.76</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>90.5 ± 4.0</td>
<td>89.0 ± 3.8</td>
<td>90.1 ± 7.4</td>
<td>90.5 ± 7.4</td>
<td>0.93</td>
<td>0.92</td>
<td>0.86</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>57.8 ± 3.5</td>
<td>57.9 ± 3.5</td>
<td>56.8 ± 4.9</td>
<td>52.9 ± 3.7</td>
<td>0.45</td>
<td>0.64</td>
<td>0.62</td>
</tr>
<tr>
<td>BMI, %</td>
<td>30.5 ± 4.3</td>
<td>29.8 ± 4.0</td>
<td>28.2 ± 4.5</td>
<td>28.5 ± 5.0</td>
<td>0.19</td>
<td>0.89</td>
<td>0.71</td>
</tr>
<tr>
<td>Abdominal circumference, cm</td>
<td>107.0 ± 12.0</td>
<td>104.0 ± 10.0</td>
<td>103.0 ± 14</td>
<td>103 ± 11</td>
<td>0.54</td>
<td>0.74</td>
<td>0.65</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>104.0 ± 15</td>
<td>101.0 ± 12</td>
<td>97.0 ± 15</td>
<td>97.9 ± 15.1</td>
<td>0.31</td>
<td>0.83</td>
<td>0.70</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td>111.7 ± 3.0</td>
<td>109.5 ± 2.4</td>
<td>108.3 ± 3.5</td>
<td>108.4 ± 4.0</td>
<td>0.49</td>
<td>0.75</td>
<td>0.73</td>
</tr>
</tbody>
</table>

Values are means ± SE. BMI, body mass index; FFM, fat-free mass; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Fig. 2. Adaptive changes in hyperinsulinemia (A) and insulin resistance (B). Exercise training results in significantly smaller insulin area (A) and product of insulin (ins) and glucose (gluc) areas (B) in response to oral glucose challenge. mcu, Microunit.
be similar to that induced by thiazide diuretic. Consistent with previous reports, however, the reduction in SBP with thiazide was greater than with exercise training (3). The regression of LVH in our volunteers is evidenced by reductions in h/r and LV PWT and Sep without an increase in LV EDD or EDV and LV mass normalized for body surface area or FFM. The absence of a significant change in LV EDD or EDV suggests that the training stimulus was not sufficiently vigorous to induce superimposed volume-overload hypertrophy. The strong inverse correlation between EF and end-systolic wall stress provides evidence for the reliance of LV systolic function on the systolic load.

The basis for the partial reversal of increased LV relative wall thickness and LVH in our patients is not clear. It is likely, however, that multiple factors might have played a role because we used two different types of interventions. The observation of an association between the reductions in SBP and h/r in the exercise group but not in the thiazide group supports this notion. Furthermore, the mechanisms underlying development of LVH and remodeling per se are complex and include both mechanical and humoral factors. Therefore, the reversal of this adaptation is likely to be mediated by more than one mechanism. Cardiac myocyte hypertrophy is induced by direct mechanical stretch (43, 44). In contrast, proliferation of myocardial fibroblasts with augmented collagen synthesis and eventual myocardial fibrosis are mediated by release of growth factors in the myocardium (22, 53). This may account for the lack of strong relationship between SBP and the magnitude of LVH reported in some studies. Previous studies showed only a modest relationship between LV mass and BP in hypertension (1, 17). A recent study showed a better correlation between arterial stiffness and LV relative wall thickness in an elderly population that included both normotensive and hypertensive older adults (38).

The adaptations to exercise training that could have contributed to the partial reversal of LVH include removal of an excess mechanical stimulus, as evidenced by a significant correlation between the extent of reductions in SBP and h/r, and amelioration of insulin resistance. Although the correlation between SBP and h/r does not necessarily signify a "cause-and-effect" relationship, it nevertheless suggests a connection between reductions in SBP and LV relative wall thickness in the exercise group. Several investigators have demonstrated a significant relationship between insulin resistance and LV remodeling or LVH in hypertension (36, 37, 49, 52). Therefore, insulin may play a role in the development of LVH. In view of these observations, it is plausible that the decreases in insulin resistance and glucose-stimulated hyperinsulinemia in response to training could have contributed to reduction of LVH and LV relative wall thickness even though we did not find a relationship between improvement in insulin resistance and altered LV geometry. Notable metabolic adaptations to exercise training in older adults are reductions in insulin resistance and hyperinsulinemia with improvement in metabolic syndrome (20, 23, 39). A lower abdominal circumference after exercise training is compatible with a decrease in abdominal fat and may account in part for improvement in insulin resistance as reflected in a positive correlation between these variables. It is possible that the lower plasma insulin in the trained state is also, at least in part, due to the residual effect of acute exercise, because our patients had their final glucose tolerance test soon after the last bout of exercise. Therefore, it seems likely that the improvement in insulin resistance in response to training in our participants is a consequence of metabolic adaptations (i.e., a decrease in abdominal adiposity) as well as the effect of acute exercise. The absence of changes in plasma norepinephrine, renin, and angiotensin in response to training suggests that at least plasma levels of these hormones may not play a role in the adaptive changes in LV structure and geometry. The mechanisms responsible for reduction of LV mass index and relative wall thickness by thiazide diuretics are unknown. Our findings suggest that, in contrast to exercise, lowering of SBP per se may not have a direct impact on regression of LVH even though we cannot rule out the role of BP entirely.

There is conflicting information with regard to the effects of exercise training on reversal of LVH in hypertensive adults. Our findings are consistent with recent studies that reported reduction of LVH by exercise training in hypertension (18, 27) and confirm the findings of a preliminary study that showed that reduction in LV remodeling was related to a lower SBP in older adults (51). However, others have shown that endurance exercise training does not induce a significant decrease in LV mass in hypertensive subjects (5). The reasons for these discrepancies are unknown. It is possible that with higher-intensity exercise training the superimposition of physiological LVH resulting in an increase in LV EDD and EDV could have blunted the decrease in LV mass by lowering BP. Because of this confounding effect, the LV relative wall thickness, i.e., h/r, may be a more desirable marker in exercise training studies.

The limitations of this study are that the implications of our findings are applicable only to patients with mild hypertension with modest LVH. Although the absence of a statistically significant difference in reduction of LV relative wall thickness between the two groups may be attributed to a small sample size, the observed differences between the two groups were too small to be physiologically significant. Furthermore, a similar reduction in LVMi by exercise training and thiazide supports the notion that the effects of these interventions on regression of LVH were comparable. We could not use the intention-to-treat analysis in this study because the participants who dropped out did not return for their final tests. The absence of a relationship between improvements in insulin resistance and reversal of LVH and altered LV geometry may be due in part to the fact that we did not measure plasma insulin levels in all the participants in the exercise group. A high dropout rate in this study suggests that the efficacy of exercise in treatment of hypertension in older adults may be limited. It is possible that

Table 4. Hormonal adaptations to exercise training

<table>
<thead>
<tr>
<th>Variable</th>
<th>Initial</th>
<th>Final</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reninsup, ng · ml⁻¹ · h⁻¹</td>
<td>0.78 ± 0.10</td>
<td>0.85 ± 0.12</td>
<td>0.75</td>
</tr>
<tr>
<td>Reninsup, ng · ml⁻¹ · h⁻¹</td>
<td>0.92 ± 0.21</td>
<td>1.4 ± 0.53</td>
<td>0.19</td>
</tr>
<tr>
<td>Angiotensinsup, U/l</td>
<td>33.6 ± 5.1</td>
<td>36.1 ± 7.1</td>
<td>0.44</td>
</tr>
<tr>
<td>Angiotensinsup, U/l</td>
<td>35.4 ± 5.9</td>
<td>39.6 ± 7.6</td>
<td>0.28</td>
</tr>
<tr>
<td>Epinephrine, pg/100 ml</td>
<td>12.3 ± 1.0</td>
<td>15.0 ± 4.7</td>
<td>0.04</td>
</tr>
<tr>
<td>Epinephrine, pg/100 ml</td>
<td>14.8 ± 2.4</td>
<td>20.5 ± 2.5</td>
<td>0.053</td>
</tr>
<tr>
<td>NEsup, pg/100 ml</td>
<td>152.8 ± 36.8</td>
<td>183.3 ± 33.0</td>
<td>0.31</td>
</tr>
<tr>
<td>NEsup, pg/100 ml</td>
<td>251.9 ± 67.0</td>
<td>318.2 ± 58.2</td>
<td>0.30</td>
</tr>
</tbody>
</table>

Values are means ± SE. E, epinephrine; NE, norepinephrine; sup, supine; up, standing.
angiotensin-converting enzyme inhibitors and angiotensin receptor blockers or even thiazides could be more effective in reducing LV relative wall thickness and LVH than exercise training in individuals with severe or moderately severe hypertension with marked LVH. Nevertheless, we believe that exercise training can be a useful strategy in those who can tolerate it and are willing to perform regular exercise.

In conclusion, the findings of this study suggest that a program of mild-to-moderate intensity exercise training can result in partial regression of increased LV relative wall thickness and LVH that is similar in magnitude to that induced by a thiazide diuretic. Although hydrochlorothiazide is considerably more effective in reducing SBP than exercise, metabolic adaptations that occur only with exercise training can provide significant additional clinical benefits that are not attainable with a thiazide diuretic. Therefore, endurance exercise training appears to be a suitable treatment strategy in some older adults with mild hypertension because, despite a smaller decrease in BP, it can induce a comparable regression of LV mass with improvements in insulin resistance and aerobic capacity and because aggressive reduction of BP by antihypertensive medications may not necessarily confer a greater protection against mortality in hypertensive individuals.

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REFERENCES


