

EFFECTS OF REGULAR EXERCISE ON BLOOD PRESSURE AND LEFT VENTRICULAR HYPERTROPHY IN AFRICAN-AMERICAN MEN WITH SEVERE HYPERTENSION

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Abstract *Background.* The prevalence of hypertension and its cardiovascular complications is higher in African Americans than in whites. Interventions to control blood pressure in this population are particularly important. Regular exercise lowers blood pressure in patients with mild-to-moderate hypertension, but its effects in patients with severe hypertension have not been studied. We examined the effects of moderately intense exercise on blood pressure and left ventricular hypertrophy in African-American men with severe hypertension.

Methods. We randomly assigned 46 men 35 to 76 years of age to exercise plus antihypertensive medication (23 men) or antihypertensive medication alone (23 men). A total of 18 men in the exercise group completed 16 weeks of exercise, and 14 completed 32 weeks of exercise, which was performed three times per week at 60 to 80 percent of the maximal heart rate.

Results. After 16 weeks, mean (\pm SD) diastolic blood pressure had decreased from 88 ± 7 to 83 ± 8 mm Hg in the patients who exercised, whereas it had increased slightly, from 88 ± 6 to 90 ± 7 mm Hg, in those who did not exercise ($P=0.002$). Diastolic blood pressure remained significantly lower after 32 weeks of exercise, even with substantial reductions in the dose of antihypertensive medication. In addition, the thickness of the interventricular septum ($P=0.03$), the left ventricular mass ($P=0.02$), and the left-ventricular mass index ($P=0.04$) had decreased significantly after 16 weeks in the patients who exercised, whereas there was no significant change in the nonexercisers.

Conclusions. Regular exercise reduced blood pressure and left ventricular hypertrophy in African-American men with severe hypertension. (N Engl J Med 1995;333:1462-7.)

CHRONIC essential hypertension is a major risk factor for cardiovascular disease.¹⁻³ Morbidity and mortality from cardiovascular causes increase curvilinearly as blood pressure rises, with no evidence of a threshold of risk.⁴

Antihypertensive therapy has reduced the incidence of congestive heart failure, renal failure, and stroke⁴⁻⁶ but has failed to produce the expected reductions in coronary morbidity and mortality.⁴⁻⁷ Various explanations have been proposed, including adverse effects of some antihypertensive agents on carbohydrate and lipid metabolism^{8,9} and poor compliance with medication regimens. Even when drug therapy is effective, side effects and cost considerations lead to poor compliance and poorly controlled hypertension. Thus, nonpharmacologic therapy as an adjunct treatment or alternative to drug therapy is desirable.

Aerobic exercise of moderate intensity reduces blood pressure in patients with mild-to-moderate essential hypertension¹⁰⁻¹⁶ and is now recommended to lower blood pressure in such patients.^{3,17,18} Exercise of lower intensity reduces the risk of injury and cardiac complications^{19,20} and makes exercise feasible for most patients. These factors, along with the low cost, absence of side effects, and additional cardiovascular benefits,²¹ make the use of exercise to lower blood pressure appealing.

Patients with systolic blood pressure of at least 180 mm Hg and diastolic blood pressure of at least 110 mm Hg (those with severe hypertension)¹⁷ have a risk of coronary heart disease that is about five times the risk in those with systolic blood pressure of 120 mm Hg

or less and diastolic blood pressure of 80 mm Hg or less.² Most patients with severe hypertension have left ventricular hypertrophy²² and require a combination of antihypertensive medications, which increases the chance of side effects and poor compliance.

Information about the effects of exercise on blood pressure in such patients, and more specifically in African Americans, is lacking. Since the prevalence of hypertension and its cardiovascular sequelae is higher in African Americans than in whites,²³ interventions to control blood pressure optimally in this population are particularly important.

We undertook this study to assess the effects of regular aerobic exercise on blood pressure and left ventricular hypertrophy in African-American men with severe hypertension.

METHODS

We recruited 46 African-American men with essential hypertension, 35 to 76 years of age, for this study. All the patients had sedentary lifestyles, as defined by the absence of a regular exercise program during the preceding six months. Inclusion criteria were an age over 21 years and a history of essential hypertension, with untreated diastolic blood pressure of at least 110 mm Hg or systolic blood pressure of at least 180 mm Hg. Patients were excluded from the study if they had a history of congestive heart failure; stroke; evidence of coronary heart disease, based on stress tests or stress thallium scintigraphy; insulin-dependent diabetes mellitus; or alcoholism, drug abuse, or psychiatric disease.

Each patient signed a consent form, approved by the local institutional review board, and was randomly assigned to drug therapy plus exercise (23 patients) or drug therapy alone (23 patients).

Antihypertensive Drug Therapy

Antihypertensive medication was prescribed for all the patients to reduce diastolic blood pressure to 95 mm Hg or less, or 10 mm Hg below pretreatment levels. Blood pressure was measured every two weeks and medication was adjusted, until the target blood pressure was achieved. Treatment was initiated with 2.5 mg of indapamide (Lozol) per day. If this was not effective in controlling blood pressure, 120 mg of sustained-release verapamil (Verelan) per day was added. Verapamil was increased as necessary to a maximal dose of 480 mg per day, depending on the patient's needs and ability to tolerate the drug. If the target blood pressure was not achieved with

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this combination, enalapril (Vasotec) was added. The dose of enalapril ranged from 2.5 to 40 mg per day.

Patients were evaluated on three occasions to ensure that their blood pressure was persistently controlled. Target blood-pressure control was achieved in all patients.

Patients were advised and frequently reminded not to alter their dietary habits during the study. Patients in the no-exercise group were also advised to maintain the same level of physical activity. Patients were seen at least monthly in our clinic.

Peak Oxygen Uptake and Blood Pressure

Peak oxygen uptake was assessed in the exercise group at base line and after 16 weeks of training. Each patient breathed through a mouthpiece with a two-way valve while running on a treadmill at a speed that elicited 75 percent of the age-predicted maximal heart rate, at a grade of 0 percent. The grade was increased from 0 to 4 percent at the beginning of the fourth minute, and by 2 percent every minute after the end of the fifth minute. Oxygen uptake was recorded every 30 seconds (Medical Graphics Cardiopulmonary Exercise System 2001). Peak oxygen uptake was considered to have been reached when the patient became too tired to continue.

Exercise blood pressure, heart rate, and the product of rate times pressure were also measured at maximal workloads attained at base line and after 16 weeks of training. The maximal workload attained at base line was used as the submaximal workload in evaluations of the same variables after training.

Resting and Exercise Blood Pressure

Resting blood pressure was measured with the patient sitting comfortably for 5 to 10 minutes and the cuff arm supported at the heart level.²⁴ Measurements were made by a Hawksley random-zero sphygmomanometer. All readings were taken between 8 and 10 a.m. To ensure that readings were made under similar conditions, blood pressure in the exercise group was recorded during the first four exercise sessions but before the patients began to exercise. Four readings were obtained per session, for a total of 16 readings. The mean of these 16 readings is reported as the base-line resting blood pressure. The procedure was repeated during the last four exercise sessions to measure the post-training resting blood pressure. Blood pressure for the control group was recorded during the first and last four visits attended.

Echocardiography

M-mode and two-dimensional echocardiograms were recorded with patients in the left lateral decubitus position, by a technician unaware of the study assignments. M-mode tracings that were guided two-dimensionally were recorded on light-sensitive paper at 50 mm per second (Hewlett-Packard Sonos 1000, with a 2.5-MHz transducer), from the short parasternal axis at the chordal level between the free edges of the mitral leaflets at the tips of the papillary muscles. To decrease variability, five cardiac cycles were read and the values averaged.²⁵ Only tracings with optimal visualization of left ventricular interfaces were used. In our echocardiographic laboratory, the range of variability of observations by a single reader is 0 mm to 1.5 mm for the dimensions of the left ventricular cavity, and 0 mm to 0.5 mm for wall thickness. Tracings were read at the end of the study independently by two cardiologists, who were unaware of the study groups and the date of echocardiographic assessment. When there was a disagreement between the two readers, the final value was settled by consensus.

Left ventricular diastolic dimensions (LVDD), systolic dimensions (LVSD), interventricular septal thickness (IVS), and posterior-wall thickness (PW) were measured in all patients according to the guidelines of the American Society of Echocardiography.²⁶ Left ventricular mass was calculated with the use of the anatomically validated formula

$$\text{left ventricular mass} = 1.04 [(IVS + LVDD + PW)^3 - (LVDD)^3] - 13.6 \text{ g.}$$

Left ventricular mass was divided by body-surface area to obtain the left-ventricular-mass index. Left ventricular fractional shortening was calculated with the use of the standard formula

$$\text{fractional shortening (\%)} = [(LVDD - LVSD) / LVDD] \times 100.$$

Exercise Program

Patients in the exercise group participated in an aerobic-exercise program that consisted of two phases, each 16 weeks long. Exercise

Table 1. Base-Line Characteristics of the Exercise and No-Exercise Groups.

CHARACTERISTIC	EXERCISE (N = 23)	NO EXERCISE (N = 23)
	mean \pm SD	
Age (yr)	57 \pm 10	58 \pm 11
Weight (kg)	97 \pm 16	95 \pm 16
Height (m)	1.77 \pm 0.05	1.76 \pm 0.08
Body-surface area (m ²)	2.2 \pm 0.20	2.15 \pm 0.21
Body-mass index*	31 \pm 5.5	31 \pm 4.3
Heart rate (bpm)	73 \pm 9	74 \pm 9
Systolic blood pressure (mm Hg)	138 \pm 10	139 \pm 13
Diastolic blood pressure (mm Hg)	88 \pm 7	88 \pm 6
Posterior-wall thickness (mm)	13.0 \pm 1.4	11.9 \pm 1.8
Intraventricular septal thickness (mm)	14.5 \pm 2.0	13.7 \pm 1.8
Left ventricular mass (g)	347 \pm 84	318 \pm 68
Left-ventricular-mass index†	162.2 \pm 39.7	147.1 \pm 28.7
Left ventricular systolic dimension (mm)	31.2 \pm 4.9	34.7 \pm 5.3
Left ventricular diastolic dimension (mm)	50 \pm 6	51 \pm 7
Ejection fraction (%)	71 \pm 10	64 \pm 11
Fractional shortening (%)	37.4 \pm 6.7	31.8 \pm 5.3

*The body-mass index is the weight in kilograms divided by the square of the height in meters.

†The left-ventricular-mass index is the left ventricular mass divided by the body-surface area.

training for both phases consisted of stationary cycling on Windracer exercise bicycles (Randal Sports Medicine Equipment, Kirkland, Wash.) three times per week. Exercise sessions started with a five-minute warm-up period (stretching and slow cycling) before the prescribed aerobic exercise and ended with an appropriate cooling-off period. Patients exercised for a mean (\pm SD) of 44 \pm 9 minutes, at approximately 74 percent of the predicted maximal heart rate.

Phase 1

Patients initially cycled for 5 to 10 minutes at 60 percent of their maximal heart rates. The duration of exercise increased progressively and on an individual basis as patients tolerated longer exercise sessions. The duration and intensity of the actual exercise phase were maintained for 20 to 60 minutes at 60 to 80 percent of the predicted maximal heart rate. Heart rate was measured continuously with a portable heart-rate monitor (Pacer). Blood pressure was monitored periodically during each workout to ensure that it was within safe limits (systolic blood pressure, <220 mm Hg; diastolic blood pressure, <110 mm Hg). The workload was adjusted to maintain the target heart rate and blood pressure within the prescribed limits throughout the exercise session.

Phase 2

This phase consisted of continued exercise and follow-up for an additional 16 weeks. If the diastolic blood pressure fell below 90 mm Hg and at least 5 mm Hg below the base-line value in any patient, the dose of antihypertensive medication was reduced. The reduction generally started with enalapril, in a stepwise fashion and in the reverse of the order in which the medications had been initiated, and occurred at weeks 20, 24, and 28 of the training program. During the period of medication reduction, blood pressure was measured weekly and during routine monthly follow-up visits to the clinic.

Statistical Analysis

The t-test for independent groups was used in the following analyses: base-line comparisons of the randomized groups, comparisons of changes from base line to weeks 16 and 32 between the two groups, and base-line comparisons of patients for whom 16-week echocardiographic

Table 2. Clinical Characteristics of the Patients in the Exercise and No-Exercise Groups at Base Line and after 16 Weeks.*

CHARACTERISTIC	EXERCISE GROUP			NO-EXERCISE GROUP			P VALUE
	NO.	BASE LINE	16 WK	NO.	BASE LINE	16 WK	
Weight (kg)	23	97±16	97±17	23	95±16	97±20	0.56
Body-surface area (m ²)	18†	2.2±0.2	2.2±0.2	22‡	2.1±0.2	2.2±0.2	0.80
Body-mass index§	18†	30±3	30±4	22‡	31±4	31±5	0.44

*Plus-minus values are means ±SD. P values are for group comparisons of changes between values at base line and those at 16 weeks.

†Complete data at both base line and 16 weeks were available for only 18 patients.

‡Complete data at both base line and 16 weeks were available for only 22 patients.

§Body-mass index is the weight in kilograms divided by the square of the height in meters.

graphic data were available with patients for whom they were not. Multiple-regression analysis was used to examine treatment-related changes in echocardiographic measurements after adjustment for base-line differences. The exercise metabolic data, which were collected only on the exercise group, were recorded at base line and at 16 weeks; the one-sample t-test was used to compare values at the two time points. All tests of significance were two-sided, and differences were considered statistically significant when $P \leq 0.05$. SAS version 6.08 was used for all analyses.

RESULTS

The clinical characteristics of the patients are shown in Table 1. Comparisons of the exercise and no-exercise groups revealed no base-line differences in any variables examined except the thickness of the left ventricular posterior wall (13.0 ± 1.4 vs. 11.9 ± 1.8 mm; $P = 0.02$), the systolic dimension (31.2 ± 4.9 vs. 34.7 ± 5.3 mm; $P = 0.04$), the ejection fraction (71 ± 10 percent vs. 64 ± 11 percent; $P = 0.03$), and fractional shortening (37.4 ± 6.7 percent vs. 31.8 ± 5.3 percent; $P < 0.003$). There was a trend toward a larger left-ventricular-mass index in patients in the exercise group (162.2 ± 39.7 vs. 147.1 ± 28.7 ; $P = 0.14$).

Eighteen of the 23 patients assigned to the exercise group completed phase 1. One patient dropped out early in the study because of an interim diagnosis of deep venous thrombosis, one was withdrawn for not complying with the medication regimen, and three were withdrawn for not complying with the exercise program. In

the no-exercise group, three patients were withdrawn for not taking their medication.

After phase 1, 14 of the 18 patients in the exercise group continued with phase 2. Three patients could not attend clinic exercise sessions because of work schedules, and one moved out of town. Two patients from the no-exercise group did not want to continue in the study. Medication was successfully reduced in 10 of the 14 patients in

the exercise group. The average reductions in the dose of medication for the group were 24 percent, 38 percent, and 37 percent for indapamide, verapamil, and enalapril, respectively. The numbers of exercise-group patients whose medication was reduced or discontinued was four and two, respectively, for indapamide, eight and three for verapamil, and two and one for enalapril. None of the patients in the no-exercise group met the criteria for a reduction of medication. The percentage of patients whose medication was reduced was significantly higher (by Fisher's exact test) in the exercise group than in the no-exercise group ($P < 0.001$).

Changes from base line to week 16 in body weight, body-surface area, and body-mass index did not differ between the two randomized groups (Table 2). Table 3 shows exercise data for the 14 patients in the exercise group who completed the metabolic exercise test at 16 weeks. Peak oxygen uptake increased significantly. The maximal heart rate was unchanged, but the heart rate at the submaximal workload was significantly lower at 16 weeks than at base line. Exercise systolic and diastolic blood pressure and the rate-pressure product at maximal and submaximal workloads were also significantly lower at 16 weeks (Table 3).

The decrease in diastolic blood pressure at 16 weeks in the patients randomly assigned to the exercise group was significantly different from the slight increase in those assigned to the no-exercise group. Differences in the reductions in systolic blood pressure and resting heart rate between the exercise group and the no-exercise group approached significance. Among the patients who completed 32 weeks of study, the change in diastolic blood pressure from base line was significantly greater in the exercise group (Table 4).

The echocardiographic data are shown in Table 5. For 32 of the 46 patients (15 in the exercise group and 17 in the no-exercise group), complete data were available at 16 weeks. There were no base-line differences between patients for whom echocardiographic data were available at 16 weeks and those for whom they were not, except for trends toward lower values for base-line posterior-wall thickness ($P = 0.10$) and higher values for body-mass index ($P = 0.13$) in the patients with echocardiographic data available. Eleven exercising and 10 nonexercising patients had left ventricular hypertrophy, defined as a left-ventricular-mass index greater than 134 g per square meter of body-surface area. At 16 weeks, the exercise group had significantly

Table 3. Exercise Data for the Exercise Group at Base Line and after 16 Weeks of Exercise Training.

VARIABLE	BASE LINE	16 WEEKS	P VALUE*
	mean ±SD		
Maximal heart rate (bpm)	153±15	153±11	0.90
Submaximal heart rate (bpm)	153±15	146±14	0.009
Peak oxygen uptake (ml/kg/min)	21±4	23±3	<0.001
Maximal systolic blood pressure (mm Hg)	218±23	199±34	0.002
Maximal diastolic blood pressure (mm Hg)	108±10	98±13	0.002
Submaximal systolic blood pressure (mm Hg)	218±23	187±30	<0.001
Submaximal diastolic blood pressure (mm Hg)	107±10	94±9	<0.001
Maximal rate-pressure product	33,272±4686	30,407±5178	0.01
Submaximal rate-pressure product	32,926±4710	27,179±4809	<0.001

*Data on 14 patients are shown. P values are for comparisons of values at base line and those at 16 weeks.

Table 4. Resting Blood Pressure and Heart Rate at Base Line and after 16 and 32 Weeks.*

VARIABLE	16-WEEK STUDY					32-WEEK STUDY				
	EXERCISE GROUP (N = 23)		NO-EXERCISE GROUP (N = 23)		P VALUE	EXERCISE GROUP (N = 14)†		NO-EXERCISE GROUP (N = 18)‡		P VALUE
	Base line	16 Wk	Base line	16 Wk		Base line	32 Wk	Base line	32 Wk	
Systolic blood pressure (mm Hg)	138±10	131±15	139±13	138±10	0.13	135±8	129±10	139±13	138±15	0.15
Diastolic blood pressure (mm Hg)	88±7	83±8	88±6	90±7	0.002	89±7	85±6	89±6	90±7	0.04
Heart rate (bpm)	73±9	71±9	73±9	75±9	0.11	—	—	—	—	—

*Plus-minus values are means ±SD. P values are for the comparison of changes from base line between groups. Data on heart rates were not obtained at 32 weeks.

†Complete data at both base line and 32 weeks were available for only 14 patients.

‡Complete data at both base line and 32 weeks were available for only 18 patients.

larger reductions from base line in interventricular septal thickness, left ventricular mass, and left-ventricular-mass index than the no-exercise group. Changes in left ventricular systolic and diastolic dimensions, ejection fraction, and fractional shortening were not significantly different.

DISCUSSION

Reduction in Blood Pressure with Exercise

We observed a significant reduction in diastolic blood pressure and regression of left ventricular hypertrophy in African-American men with severe hypertension after 16 weeks of moderately intense aerobic exercise. In addition, exercising for more than 16 weeks resulted in a further fall in blood pressure, permitting a substantial reduction in antihypertensive medication.

Systolic blood pressure also decreased, by 7 mm Hg and 6 mm Hg after 16 and 32 weeks of exercise, respectively. Although these changes are clinically important,² neither reached statistical significance.

Severe hypertension is difficult to manage and often requires multiple antihypertensive medications for effective control. Such complex treatments burden patients financially and increase the possibility of adverse effects and poor compliance. Our results show that severe hypertension can be managed more effectively with a combination of drug therapy and regular, moderately intense exercise. Most important, medications necessary to control blood pressure without exercise can be curtailed substantially as patients continue exercising.

Blood pressure at the maximal exercise level, blood pressure at a submaximal workload, and the rate-pressure product — an indicator of myocardial oxygen consumption — were also lower after 16 weeks of regular exercise. The lower rate-pressure product observed at maximal workload was entirely due to lower systolic blood pressure, since values for maximal heart rate before and after exercising were similar. The rate-pressure product at submaximal workload was 18 percent lower than at base line.

The general consensus is that regular aerobic exercise reduces blood pressure significantly in patients with mild-to-moderate essential hypertension.¹⁰⁻¹⁵ Our results show that the effectiveness of moderately intense exercise extends to patients with severe hypertension. Moderately intense exercise carries a lower risk of musculoskeletal injuries and acute cardiovascular events than does intense exercise.^{19,20} Patients are also more likely to initiate and continue a lower-intensity exercise program than a higher-intensity one.

The underlying mechanism or mechanisms responsible for the exercise-induced reduction in blood pressure remain unclear. Recent evidence shows that insulin resistance and hyperinsulinemia may contribute to the pathogenesis of hypertension.^{27,28} Insulin levels at a 16-week follow-up visit were 33 percent lower than those at base line in the exercise group (22.2 ± 18 vs. 14.8 ± 9 μ U per milliliter [133 ± 108 vs. 89 ± 54 pmol per liter]) and 9 percent lower than base-line levels in the no-exercise group (26.7 ± 23 vs. 24.2 ± 21 μ U per milliliter [160 ± 138 vs. 145 ± 126 pmol per liter]). However, differences in the changes in insulin levels between the two groups were not statistically significant.

Regression of Left Ventricular Hypertrophy

Left ventricular hypertrophy is an independent risk factor for cardiovascular disease.^{22,29} Morbid events from cardiovascular causes, including sudden cardiac

Table 5. Echocardiographic Data on 15 Patients in the Exercise Group and 17 Patients in the No-Exercise Group.*

VARIABLE	EXERCISE GROUP (N = 15)		NO-EXERCISE GROUP (N = 17)		P VALUE	
	BASE LINE	16 WK	BASE LINE	16 WK	UNADJUSTED	ADJUSTED
Posterior-wall thickness (mm)	13.3±1.5	12.3±1.3	11.9±2.0	11.9±1.9	0.04	0.20
Interventricular septal thickness (mm)	14.9±2.3	14.0±1.7	13.7±2.0	13.7±2.1	0.008	0.03
Left ventricular systolic dimension (mm)	31±5	30±5	35±7	34±6	0.49	0.92
Left ventricular diastolic dimension (mm)	49±5	48±4	51±6	51±6	0.62	0.29
Left ventricular mass (g)	346±95	304±72	323±69	326±83	0.01	0.02
Left-ventricular-mass index†	163±45	143±34	150±27	149±32	0.02	0.04
Ejection fraction (%)	72±11	74±11	64±11	69±12	0.43	0.81
Fractional shortening (%)	37±8	38±9	32±5	36±8	0.18	0.53

*Plus-minus values are means ±SD. P values are for the comparison of changes from base line between groups.

†The left-ventricular-mass index is the left ventricular mass divided by the body-surface area.

death, are three times as frequent in patients with left ventricular hypertrophy.^{22,29}

Regression of left ventricular hypertrophy after a reduction in blood pressure has been reported with most antihypertensive medications.³⁰⁻³² However, the effects of prolonged exercise on ventricular structure and function are not clear. The rigorous exercise endured by athletes can provoke cardiac enlargement. The "athlete's heart," as it is commonly called, is exercise-specific. Purely aerobic exercises induce enlargement of the left ventricular cavity, with no changes in left-ventricular-wall thickness.³³ In contrast, combined isotonic and isometric exercise (e.g., weight training and rowing) may lead to substantial hypertrophy of the left ventricular wall.³⁴ Nevertheless, exercise-induced left ventricular hypertrophy is considered a normal physiologic adaptation to the particularly rigorous training of athletes. It is not associated with diastolic dysfunction, arrhythmias, or an adverse prognosis, as is hypertension-induced left ventricular hypertrophy,^{35,36} and it regresses quickly with detraining.³⁷

In contrast to athletes, our patients exercised at a relatively low intensity and duration. After 16 weeks of such training, we observed a significant reduction in the thickness of the interventricular septum and 12 percent reductions in left ventricular mass and left-ventricular-mass index. The reduction in left ventricular mass was similar to the reduction of 8 to 15 percent in overall left ventricular mass that has been reported in drug studies.³¹ There were no changes in any echocardiographic measurements in the no-exercise group. Our findings and those of studies of highly trained athletes^{33,34} suggest that the intensity and duration of exercise may have an important effect on left ventricular structure.

The mechanisms involved in the regression of left ventricular hypertrophy in the patients in our exercise group are not readily apparent. It is likely that the reduction in blood pressure has played a part. In general, most antihypertensive regimens that lower blood pressure induce various degrees of reduction in left ventricular mass without any drug-specific effects.^{31,32} Other factors, however, such as levels of angiotensin II and catecholamines, may be involved.³⁸ Prolonged physical activity decreases plasma catecholamines in some hypertensive patients.¹⁰⁻¹²

In conclusion, we found that African-American men with severe hypertension and left ventricular hypertrophy benefit from a combined regimen of regular, moderately intense aerobic exercise and antihypertensive drug therapy. The antihypertensive effects of exercise substantially reduced the amount of medication required to control blood pressure.

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