Effect of Regular Exercise on 24-Hour Arterial Pressure in Older Hypertensive Humans

Douglas R. Seals and Mary Jo Reiling

The experimental goals were to determine if regular low-intensity aerobic exercise reduces 24-hour arterial blood pressure in middle-aged and older (aged 50 years or older) humans with mild diastolic (90–105 mm Hg) essential hypertension and, if so, whether this is accurately reflected by changes in casual recordings made at rest. Fourteen subjects walked 3–4 days/wk for 6 months, with 10 exercising an additional 6 months; 12 other subjects served as nonexercising controls. In the exercising subjects, maximal oxygen consumption increased 7–14% (p<0.05) with little or no change in body weight or fat. Conventional casual readings of systolic, mean, and diastolic arterial pressure at rest were lower (5–10 mm Hg, p<0.05) in all body positions after 6 months of exercise and changed little thereafter. Casual recordings made during additional circulatory measurements showed 6-month decreases of only half this magnitude and were specific to a particular blood pressure phase and body position; however, all changes were significant after 12 months of exercise. The reductions in arterial pressure at rest were associated with decreases in heart rate (p<0.05) and cardiac output (p<0.05). Ambulatory-determined 24-hour arterial pressure was unchanged after 6 months of exercise, but mean levels were slightly lower (4 mm Hg, p<0.05) after 12 months due to reductions in daytime (7 mm Hg, p<0.05) and nighttime (4 mm Hg, NS) systolic pressure; diastolic pressure was unchanged throughout the year of training. In the controls, conventionally recorded casual blood pressure levels were lower after 6 months (p<0.05), but no other changes were observed in any other variable over the 12 months of study. We conclude 1) regular low-intensity aerobic exercise at best produces only small reductions in 24-hour levels of arterial pressure in middle-aged and older humans with mild (diastolic) essential hypertension and 2) training-associated changes in casually determined blood pressure at rest are dependent on the measurement conditions and, most importantly, do not necessarily reflect the magnitude or even the direction of changes in arterial pressure throughout an entire day. (Hypertension 1991;18:583–592)

Arterial blood pressure rises with advancing age in industrialized societies. This contributes to the increased risk of cardiovascular disorders such as stroke and ischemic heart disease in older people. Lowering chronic levels of arterial pressure via drug therapy reduces cardiovascular risk in older patients with essential hypertension, but the associated side effects are generally greater than in younger patients. Thus, nonpharmacological interventions have been recommended as an initial approach for older patients with mild-to-moderate blood pressure elevations.

Regular physical exercise lowers arterial pressure at rest by 7–9% (8–10 mm Hg) on average in young and middle-aged hypertensive humans, but its influence in older people is less well documented. Recently Hagberg et al reported that 3 months of low-intensity aerobic exercise training reduced arterial pressure at rest by 8–12 mm Hg in middle-aged and older men and women with mild essential hypertension; an additional 6 months of training produced further (4–8 mm Hg) reductions. These decreases in arterial pressure were as great or greater than those observed in subjects who performed higher intensity exercise. Interestingly, training had a much smaller effect on blood pressure determined at rest during...
experimental sessions in which additional circulatory measurements (i.e., cardiac output) were made.

In both young and older patients with essential hypertension, levels of arterial blood pressure recorded acutely in the clinic or laboratory under resting conditions (i.e., casual readings) often overestimate levels measured throughout the entire day under freely behaving (ambulatory) conditions; the latter correlate better with end organ damage and therefore with patient prognosis. In longitudinal studies, casually determined pressures can decrease over time without corresponding changes in ambulatory-measured levels. Thus, it is possible that intervention-induced changes in casually recorded arterial pressure at rest may not accurately reflect the magnitude or even the direction of changes in levels throughout the entire day. This could be the case in studies examining the influence of physical training on blood pressure. For example, two recent investigations reported little or no decrease in daytime or nighttime ambulatory-determined arterial blood pressure after young and middle-aged normotensive or borderline hypertensive subjects; in one of these studies, casually measured levels decreased to a much greater extent than the ambulatory pressures.

The primary aims of the present investigation were to determine if regularly performed low-intensity aerobic exercise reduces arterial blood pressure throughout the entire day in middle-aged and older men and women with mild essential (diastolic) hypertension and, if so, whether this is accurately reflected by changes in casually determined blood pressure at rest measured at least under two different experimental conditions. Our secondary aims were to determine, in this population, whether extending the length of exercise training would produce further reductions in arterial pressure and to gain insight into the systemic hemodynamic mechanisms underlying training-associated decreases in arterial pressure.

Methods

Subjects

A total of 34 subjects (24 men and 10 women) aged 50–74 years were enrolled in this investigation after providing their written, informed consent. All procedures and protocols used in this study had been approved by the Human Subjects Committee at the University of Arizona. Eight of the subjects dropped out due to illness or lack of compliance with the requirements of the study. Twenty-six subjects completed at least two rounds of testing separated by 6 months of either regular aerobic exercise (n = 14; nine men and five women) or unchanged physical activity levels (nonexercising controls) (n = 12; 10 men and two women). Eighteen of these subjects (10 exercise and eight controls) participated in an additional 6 months of study and completed a third round of testing. At enrollment every subject had an average level of diastolic arterial blood pressure at rest between 90 and 105 mm Hg based on repeated casual recordings (see session 1). The subjects were free of overt disease based on a medical history, physical examination, and electrocardiogram at rest and during maximal treadmill exercise. They were within 20% of ideal body weight based on their body mass index and had no orthopedic complications that would prohibit their participation in testing or physical training. Only one (training group) subject smoked; her responses were not different from those of the other exercising subjects. Before enrollment, four subjects in the exercise group (diuretics only) and seven controls (diuretics, n = 3; β-blockers, n = 4) were taking antihypertensive medications. Each of these subjects stopped their medications at least 2 weeks before the first session in a particular round of testing and remained off drugs throughout the testing period. Most of the subjects resumed drug therapy immediately after the final session of each round of testing to ensure proper blood pressure control; however, three subjects (one exercise, two controls) did not continue therapy after their initial round of testing because their arterial pressure remained at a satisfactorily low level. No subject had performed any regular exercise for at least 2 years before enrollment. Subjects were instructed not to alter their diet during the period of study. To document this, a comprehensive food frequency questionnaire was administered and analyzed during each round of testing.

Experimental Procedures

Except where indicated, all subjects underwent each of the following procedures during each round of testing. For subjects in the physical training group, these procedures were performed on days other than those on which they exercised.

Maximal $O_2$ Consumption

Maximal $O_2$ consumption was measured by open circuit spirometry during graded treadmill walking as described in detail previously and was used to document training-induced adaptations resulting in an increase in aerobic exercise capacity. Subjects were required to exercise to the point of subjective exhaustion. All subjects attained 1) a plateau in $O_2$ consumption despite further increases in work load, 2) a respiratory exchange ratio ($CO_2$ production/$O_2$ consumption–hyperventilatory index of exercise effort) of more than 1.15, and 3) their age-predicted maximal heart rate. Concerning the latter two variables, the levels achieved over the three rounds of testing were similar within and between the two groups ($1.20±0.02–1.22±0.03$ and $168±4–171±5$ beats/min and $1.19±0.02–1.21±0.02$ and $170±4–173±4$ beats/min for the exercising subjects and controls, respectively).

Estimation of Body Fat

Body fat was estimated from skinfold thickness at six sites using a Harpenden caliper. Body density and
percent body fat were estimated using the equations of Jackson and Pollock\textsuperscript{23} and Siri,\textsuperscript{24} respectively.

**Determination of Arterial Blood Pressure: Casual Recordings at Rest**

**Session 1. Arterial pressure only.** In the initial round of testing, these sessions were used to determine whether the subject’s level of diastolic pressure met the criterion for enrollment and, if so, to establish the baseline level of arterial pressure from which values obtained in subsequent testing periods could be compared. In this initial round (i.e., before enrollment), subjects were studied once or twice per week for 3–6 weeks to ensure that a stable level of arterial pressure was obtained. Consistent recordings over a period of 3 weeks were required before a subject was allowed to continue with testing. The readings from these 3 weeks were averaged and used as the baseline arterial pressure. During subsequent rounds of testing, similarly stable, multiple recordings were obtained over a 2–3 week period and averaged. Measurements were made by the auscultatory method over the brachial artery using a Hawksley random-zero sphygmomanometer (Hawksley and Sons, West Sussex, England). During the first session of the initial testing period, arterial pressure was determined in both arms, and the arm with the highest reading was used for all subsequent recordings. The first and fifth phases of the Korotkoff sounds were used to determine the systolic and diastolic arterial pressures, respectively. All measurements conformed strictly to American Heart Association guidelines.\textsuperscript{25}

To document that changes in arterial pressure observed over time were not specific for a particular body position, measurements were made with the subject in the supine, sitting, and standing positions. Briefly, in each session subjects rested in the supine position on a padded table in a quiet laboratory (−24°C) for 15 minutes, after which time three or more recordings were made over a 10–15-minute period. The back of the table was then raised, and subjects sat upright for 5 minutes followed by at least three readings over 10–15 minutes. Subjects then stood for 5 minutes, and three or more measurements were obtained in this position.

**Session 2. Arterial pressure during systemic hemodynamic measurements.** The aims of this session were 1) to gain insight into the systemic hemodynamic mechanisms responsible for training-induced reductions in arterial pressure at rest and 2) to obtain an additional set of arterial pressure recordings made at rest but under somewhat different experimental conditions than those obtained in the sessions described above; this would indicate whether any training-induced reductions observed at rest were measurement condition-specific. Subjects rested in the supine position for 20 minutes after which cardiac output (Colliers CO\textsubscript{2} rebreathing technique\textsuperscript{26}), heart rate, and arterial pressure were measured three to five times over a 30–45-minute period. The arterial pressure measurements were made before the rebreathing maneuver when the subject was resting quietly. The table then was adjusted, and the subjects sat upright for ~10 minutes, at which point these measurements were repeated. At least three consistent (i.e., ±1 l/min) values of cardiac output were obtained in each position for each subject and averaged. Stable measurements could not be obtained in two of the controls; thus, their hemodynamic data were deleted from subsequent analysis. Cardiac output was divided by the corresponding heart rate to calculate stroke volume and into the corresponding mean arterial pressure to calculate systemic vascular resistance. Because the CO\textsubscript{2} rebreathing technique appears to underestimate absolute (i.e., l/min) levels of cardiac output at rest in hypertensive older subjects,\textsuperscript{13} the data on cardiac output, stroke volume, and systemic vascular resistance are presented as percent changes from levels obtained during the initial round of testing.

**Ambulatory Measurements**

Levels of arterial blood pressure throughout an entire day were obtained using a noninvasive ambulatory monitor (model 5200, Spacelabs Inc., Redlands, Wash.). The monitor operates by the auscultatory method when Korotkoff sounds are audible (phase five for diastolic) and by oscillometry when not; extraneous noise (body movement) is differentiated from pressure signals by an internal microprocessor. Ambulatory readings from this system correlate well with ambulatory measurements made during direct (intra-arterial) recordings.\textsuperscript{27,28} On a weekday in which no other measurements (or training) were performed (typically in the morning) subjects were oriented to the monitor and fitted with a standard, properly sized blood pressure cuff; in their second (or third) rounds of testing, subjects reported to the laboratory at the same time and day of the week as during this initial trial. The ambulatory system was calibrated against conventional sphygmomanometer-determined readings and programmed to inflate automatically every 15 minutes between 6:00 AM and midnight and every 60 minutes between midnight and 6:00 AM; thus, 78 recordings were possible over a 24-hour period. To reduce noise, each subject was instructed to stop and relax the arm throughout the inflation/deflation cycle and to record the time, location, body position, and state of activity at each reading. Recordings were stored on magnetic tape and subsequently analyzed and printed using a desktop computer (IBM PC). Each reading was edited both by computer and manually, and outliers (systolic pressure less than 70 or more than 260 mm Hg, diastolic less than 40 or more than 150, and heart rate less than 40 or more than 150 beats/min) were deleted. Averages were then calculated for each hour and also for the entire daytime (6:00 AM to 6:00 PM), evening/nighttime (6:00 PM to 6:00 AM), and all day (24-hour) time periods. Over the three rounds of testing, meaningful readings were obtained on 85–90% of the total possible recordings. To ensure that
differences in daily activity levels did not confound the interpretation of serial measurements, on the second and third rounds of testing subjects performed the same activities as during their initial 24-hour time periods; the activity logs were checked to ensure that this procedure was followed. It was determined that one training group subject performed a substantially greater amount of physical activity during the day of his second round of testing. All of his ambulatory data were deleted from subsequent analysis. In addition, in one control group subject the magnetic tape was accidentally erased while he was working, and a repeat trial was not possible.

Physical Activity Levels

After completing the initial round of testing, the 14 subjects in the exercise group performed low-intensity aerobic training for approximately 6 months. Subjects were instructed to walk at least 3 days/wk for at least 30 min/day at an intensity associated with an exercise heart rate of 40–50% of their individually determined heart rate reserve (calculated as heart rate at rest + 0.40–0.50 [maximal heart rate−heart rate at rest]). Subjects actually exercised for an average of 27.6±1.8 (mean±SEM) weeks, 3.5±0.1 days/wk, for 41±4 min/day, at 47±2% of heart rate reserve. On completion of the second round of testing, 10 of these subjects agreed to continue training for an additional 6 months; in exchange, these subjects were allowed to gradually increase their exercise intensity and duration if they so chose. This subgroup walked an average of 3.5±0.1 days/wk for 43±5 min/day at an intensity of 46±2% of heart rate reserve during their initial 6 months of training and 3.6±0.2 days/wk for 50±2 min/day at an intensity of 57±3% of their heart rate reserve during the final period (30.3±2.4 weeks) of training. The 12 nonexercising controls maintained their normal physical activity patterns throughout the 6-month period between the first and second rounds of testing as determined by analysis of their individual activity logs; subsequently, eight of these subjects continued to maintain their normal activity levels for an additional 6 months. Due to the length of the study period, to ensure the highest possible compliance with their respective assignments subjects were allowed to choose between training and maintained physical activity. This strategy was effective in eliminating the “crossover” effect associated with training studies described previously by Holloszy et al29 (i.e., failure of some randomly assigned training group subjects to exercise regularly and the covert initiation of training by some control subjects).

Statistics

Changes in a variable from the initial round of testing over time within a group were evaluated by analysis of variance. Differences between two specific time points were determined by Scheffe post hoc analysis. Differences of \(p < 0.05\) were considered significant. All data are presented as mean±SEM.

Results

Subject Characteristics

Body weight and estimated body fat were unchanged after 6 months of regular aerobic exercise, but maximal \(\dot{V}O_2\) consumption was increased 7% (\(p < 0.05\)) (Table 1). In the subset of subjects who completed 1 year of training, body weight decreased slightly (~1 kg; \(p < 0.05\), but estimated body fat was unchanged during the final 6 months. In this subgroup, maximal \(\dot{V}O_2\) consumption was increased by 9% and 14% after 6 and 12 months of training, respectively (both \(p < 0.05\)). There were no changes in any of these variables over corresponding periods of time in the nonexercising control subjects. There also were no changes across time in any component of diet in either group.

Arterial Blood Pressure

Casual Blood Pressure at Rest

Session 1. Arterial pressure only. Six months of regular exercise resulted in decreases in systolic (range, 7–10 mm Hg), diastolic (5–7 mm Hg), and mean (7–8 mm Hg) arterial pressure at rest in all three body positions studied (\(p < 0.05\)) (Table 2, Figures 1 and 2). In the subset of subjects who trained for 1 year, the decreases in arterial pressure during the initial 6 months of training were slightly greater than those for the whole group, but there were no further reductions over the final 6 months of exercise. Arterial blood pressure decreased to a similar (6 month group) or slightly lesser (12 month subgroup) extent in the nonexercising controls over the same periods of time (\(p < 0.05\)).

Session 2. Systemic hemodynamics. During the initial round of testing, there were no consistent
Figure 1. Bar graphs show initial levels (top of each bar, mean±SEM) and average changes in arterial blood pressure in response to 6 (n=14) and 12 (n=10) months of regular aerobic exercise recorded under three different experimental conditions. Top panel: Casual recordings at rest during no other measurements (average of three body positions). Middle panel: Casual recordings during measurements of cardiac output (average of supine and sitting positions). Bottom panel: 24-hour ambulatory recordings. Note that the data from the three conditions differed with respect to whether a significant change occurred, the phase of arterial pressure that was altered, and the magnitudes and timecourses of the reductions. *p<0.05 vs. initial level. †p<0.05 only for supine position.

Figure 2. Bar graphs show initial levels and average changes in arterial blood pressure over 6 (n=12) and 12 (n=8) months of unchanged physical activity in the controls recorded under three different experimental conditions (see Figure 1 for details). Note that significant reductions were observed over time during sessions in which only blood pressure was measured at rest (top panel). In contrast, levels of arterial pressure recorded at rest during other circulatory measurements (middle panel) and ambulatory recordings during 24 hours of normal activities (bottom panel) remained constant over time. *p<0.05 vs. initial levels.

Differences in the absolute levels of arterial pressure determined in these hemodynamic measurement sessions compared with those obtained in the sessions in which only blood pressure was measured (Table 3, Figures 1 and 2). After 6 months of aerobic training, there was a trend for small (3-4 mm Hg) reductions in systolic, diastolic, and mean arterial pressure at rest in both the supine and upright seated body positions; however, the changes were statistically significant only for supine diastolic and mean arterial pressure. In the subjects completing 1 year of exercise, trends similar to those described for the entire group were observed during the initial 6 months of training. In this subgroup, an additional 6 months of regular exercise resulted in further small (2-4 mm Hg) decreases in arterial pressure such that the final levels of systolic, diastolic, and mean pressure were all significantly (p<0.05) lower than the initial values in both body positions. In contrast to the sessions in which only blood pressure was measured, in these hemodynamic sessions there were no changes in arterial pressure over time in the nonexercising controls.

Ambulatory Recordings

Depending on the time of day used for comparison, the absolute levels of ambulatory-determined pressure were slightly to markedly lower than the casually determined levels (Table 4, Figures 1 and 2). Average casual levels at rest were most closely associated with daytime ambulatory pressures and least associated with nighttime ambulatory levels; the latter were 9-14 mm Hg lower than the corresponding levels during the day. Six months of training did not significantly alter daytime, nighttime, or 24-hour levels of arterial blood pressure; however, there was a trend for a reduction in daytime and 24-hour systolic and mean pressure. In the 1-year training subgroup, an additional 6 months of regular exercise resulted in a further, albeit slight (1-2 mm Hg) trend for a lowering of daytime and 24-hour systolic and mean pressure such that their final (i.e., 12-month) levels were significantly lower than their initial values (6-7 mm Hg for systolic, 4-5 mm Hg for mean; all p<0.05). There was also a trend (3-4 mm Hg, NS) for reductions in nighttime systolic and mean pressure such that their final (i.e., 12-month) levels were significantly lower than their initial values.

Systemic Hemodynamics

Session 2

The reductions (or trends for reductions) in arterial blood pressure at rest after 6 months of training
were associated with small but statistically significant decreases in heart rate and cardiac output and unchanged levels of stroke volume and systemic vascular resistance in both body positions (Figure 3, only supine data shown). In the 1-year training subgroup, the trend for slight, further reductions in arterial pressure over the final 6 months of exercise was associated with trends for further decreases in heart rate and cardiac output. The hemodynamic variables were quite constant over time in the non-exercising controls.

**Ambulatory Recordings**

There was a trend (NS) for a small (4 beats/min) reduction in 24-hour heart rate after 6 months of training in the entire exercise group; this was associated with a small (5 beats/min) but significant (p<0.05) decrease in daytime heart rate and no change in nighttime heart rate (Table 4). The same trend was observed over the initial 6 months in the 1-year training subgroup; however, after the final 6 months of training, 24-hour heart rate had returned toward the initial levels in these subjects. There were no changes in ambulatory-determined levels of heart rate over corresponding periods of time in the non-exercising controls.

**Discussion**

These findings provide the experimental basis for two primary new conclusions regarding the influence of physical training on blood pressure. First, regularly performed low-intensity aerobic exercise, at best, produces only small decreases in 24-hour levels of arterial pressure in middle-aged and older patients with mild diastolic essential hypertension. Second, exercise training-induced reductions in casually determined arterial pressure were associated with small but statistically significant decreases in heart rate and cardiac output and unchanged levels of stroke volume and systemic vascular resistance in both body positions (Figure 3, only supine data shown). In the 1-year training subgroup, the trend for slight, further reductions in arterial pressure over the final 6 months of exercise was associated with trends for further decreases in heart rate and cardiac output. The hemodynamic variables were quite constant over time in the non-exercising controls.

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Table 4. Average Levels of Arterial Blood Pressure and Heart Rate Determined From All-Day Ambulatory Recordings

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>24-Hour</th>
<th>Day</th>
<th>Night</th>
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<tr>
<td></td>
<td>Initial</td>
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<td>12 mo</td>
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<td>Arterial pressure (mm Hg)</td>
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<tr>
<td>Systolic</td>
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<tr>
<td>Exercise (13)</td>
<td>137±3</td>
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<td>142±3</td>
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<td>139±3</td>
</tr>
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<td>Exercise (9)</td>
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<td>132±4</td>
<td>131±3*</td>
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<td>82±2</td>
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<td>93±1</td>
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<tr>
<td>Mean</td>
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<td></td>
<td></td>
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<tr>
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<td>Exercise (9)</td>
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<tr>
<td>Heart rate (beats/min)</td>
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<tr>
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<td>Control (8)</td>
<td>76±3</td>
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All data are mean±SEM. *p<0.05 vs. initial value.

determined blood pressure at rest are dependent, in part, on the measurement conditions and, most importantly, do not always reflect the magnitude or even the direction of changes in arterial pressure throughout an entire day.

Influence of Physical Training on 24-Hour Blood Pressure

In the present study, 6-12 months of regular low-intensity aerobic exercise produced small increases in maximal oxygen consumption. These small improvements in aerobic capacity are consistent with previous observations and indicate that our exercise stimulus was sufficient to produce physiologically significant adaptations in circulatory or skeletal muscle function, or both. In spite of this training effect, 24-hour levels of arterial blood pressure were unchanged after 6 months of regular exercise. In a subgroup that performed an additional 6 months of slightly more intense exercise, 24-hour levels of mean arterial pressure were slightly (4 mm Hg on average) but significantly lower at the end of 1 year of training compared with their initial levels. This reduction was due primarily to a decrease in daytime systolic pressure, although there was also a trend for a reduction in nighttime systolic pressure. In contrast, 24-hour levels of diastolic pressure were unchanged throughout the entire year of training in these subjects. The decreases in daytime and 24-hour systolic and mean arterial pressure after 1 year of training were likely the result of the exercise stimulus per se, because 24-hour levels of arterial pressure remained remarkably constant over the same period of time in the nonexercising controls.

Little information is available concerning the effects of physical training on blood pressure through-
out an entire day. In young and middle-aged normotensive men, Fortmann et al. observed small (2–3 mm Hg) decreases in daytime and nighttime systolic and diastolic pressure after a 1-year program of exercise plus diet-induced weight loss. In a similar population, Van Hoof et al. reported that 4 months of aerobic training lowered daytime diastolic pressure by 5 mm Hg but did not alter nighttime levels or 24-hour systolic pressure. Finally, Gilders et al. recently observed no change in 24-hour levels of arterial blood pressure after 16 weeks of regular aerobic exercise in young and middle-aged normotensive or borderline hypertensive men and women. In each of these studies, training produced significant increases (13–14%) in aerobic capacity. Taken together, the results of these investigations and those from the present study indicate that low-to-moderate intensity aerobic exercise training has, if any, only a small effect on 24-hour levels of arterial blood pressure in normotensive or mildly hypertensive healthy humans. It is possible that high-intensity training would evoke larger reductions in 24-hour arterial pressure, but this postulate is not supported by experimental findings on casual measurements, which indicate that low-level exercise exerts as great or greater hypotensive effect than more strenuous training.11-13

Training-Induced Changes in Casual Versus Ambulatory Measured Blood Pressure

In the present investigation, 6 months of regular aerobic exercise was associated with significant reductions in conventionally measured systolic, mean, and diastolic arterial blood pressure at rest, and the changes were consistent among the three body positions studied. The data from the 1-year training subgroup indicated that essentially no further change occurred in this measure of arterial pressure with an additional 6 months of exercise. The magnitudes of the training-evoked reductions in these casual readings were similar to the average decreases reported previously in young and middle-aged hypertensive humans, especially when expressed as percent change from initial levels.9,11,12 Compared with our findings, Hagberg et al. recently reported larger absolute (i.e., mm Hg) reductions in arterial pressure at rest after 9 months of low-intensity aerobic training in older subjects with mild diastolic hypertension. However, the percent changes from initial levels to the end of training were not markedly different in the two studies, in part because of the higher initial levels of systolic pressure in their subjects. Thus, based on these casual measurements, the data from our exercising subjects per se and the findings of Hagberg et al. suggest that regular aerobic exercise has a blood pressure–lowering effect in older hypertensive humans of similar magnitude to that observed in younger hypertensive subjects.

Acute measurements of arterial pressure made under slightly different experimental conditions, however, provide a somewhat different picture. In the present study, during the initial round of testing the absolute levels of blood pressure determined at rest during a protocol in which cardiac output was also measured (i.e., session 2) were similar to the other casually determined pressures. In spite of this, reductions in the session 2 blood pressures over 6 months of training were 1) observed only for diastolic and mean levels, not systolic; 2) significant only in the supine position; and 3) 50% or less of the magnitude observed in the other casual sessions. A similar discrepancy in results between the two casual blood pressure measurement conditions were observed by Hagberg et al., but their intersession differences were even greater than those observed here. Collectively, our results and those of Hagberg et al. demonstrate that the magnitude of training-induced reductions in casually determined arterial pressure at rest is protocol-specific.

Do these casual measurements at rest accurately reflect training-induced changes in arterial pressure throughout an entire day under freely behaving conditions? In contrast to the reductions observed at rest under laboratory conditions, ambulatory-measured pressures were not altered after 6 months of training in the present study. In a subset of the subjects, 24-hour levels of mean arterial pressure were lower after 1 year of training. However, unlike the casually recorded levels, these reductions in 24-hour pressure were mediated solely by reductions in systolic pressure. This discrepancy was not likely due to differences in initial absolute levels (i.e., law of initial baseline) because the ambulatory systolic pressures were lower than, and the diastolic pressures (especially daytime) were similar to, the corresponding casually determined levels.

The results of the three previous studies on physical training and ambulatory blood pressure fail to provide any consistent insight into this issue. The data of Fortmann et al. are similar to our findings in that the magnitudes of the training-induced reductions in casually recorded pressures at rest were much greater than the changes in ambulatory levels. In contrast, Van Hoof et al. reported 5 mm Hg reductions in both casually determined and ambulatory-determined diastolic blood pressure and no changes in systolic levels after training, suggesting a good correlation between the two measures on average. Gilders et al. observed no training-associated changes in either casual or ambulatory pressures in their study. Considered together, the present findings and those from previous reports demonstrate that casual readings at rest and 24-hour ambulatory recordings can provide quite different information regarding the influence of physical training on chronic levels of arterial blood pressure, including 1) whether a change occurred, 2) the particular phase of the blood pressure cycle that was altered, and 3) the magnitude of change.

The same could be said with respect to measurements over time in nonexercising control subjects. In our investigation, the conventionally determined pres-
sures at rest (Table 2) fell as much over 6 months in the control subjects as in the training group. Most importantly, 24-hour ambulatory-recorded levels were unchanged over the same period of time in the control subjects (Table 4). Similar discrepancies between the two measurements have been reported previously for control subjects in longitudinal studies involving both physical training and other interventions. Furthermore, because the pressures in session 2 of the present investigation did not change over time in the control subjects, our data indicate that casual determinations made under different experimental conditions also can markedly influence conclusions drawn on this portion of the study population.

As discussed in earlier reviews on this topic, several laboratories have reported reductions in casually recorded blood pressure levels in nonexercising controls of similar magnitude to that observed in their training subjects. Insufficiently long screening periods before enrollment probably contributed to some of these observations (i.e., blood pressure decreased over time as subjects became more familiar with the measurement conditions). This clearly was not the case in the present investigation because we documented that blood pressure was stable in every subject before they were allowed to continue in the study. Thus, some common, unknown factor specifically influenced this measure of arterial pressure in both of our groups. From an interpretive standpoint, these data suggest that the decreases in conventionally recorded arterial pressure observed in our exercising subjects may not have been caused by physical training but instead by a general effect associated with study participation.

Systemic Hemodynamics

The decreases in casually determined blood pressure at rest after 6 months of training were associated with small but significant reductions in heart rate and cardiac output and no changes in stroke volume or systemic vascular resistance (Figure 3). In the subgroup that completed 1 year of training, the further slight reduction in arterial pressure over the latter 6 months of exercise was associated with similar trends in heart rate and cardiac output. These observations confirm the recent findings of Hagberg et al on older men and women with mild diastolic hypertension and indicate that in this population, decreases in arterial pressure at rest after low-intensity aerobic training are mediated solely by bradycardia-evoked reductions in cardiac output.

The magnitude of the decrease in heart rate at rest was similar to the reduction we reported previously in normotensive middle-aged and older men who underwent more strenuous aerobic training. In the present study, this training-induced bradycardia at rest was associated with a trend for a decrease in 24-hour heart rate (Table 4). This trend was, in turn, solely the result of a significantly lower daytime heart rate. The fact that 24-hour levels of heart rate were unchanged in the control subjects indicates that this bradycardia was an adaptation to the chronic exercise stimulus.

Clinical Significance

Cardiovascular disease increases dramatically with advancing age and contributes greatly to morbidity, disability, and mortality in the elderly. Hypertension appears to be the key factor underlying this increased risk because of its high prevalence in older people. There is now convincing experimental support that lowering levels of arterial pressure at rest reduces cardiovascular risk in elderly hypertensive patients. However, because of the side-effects and economic considerations associated with drug therapy, nonpharmacological interventions may be most efficacious in older people with mild essential hypertension. The present findings (session 2 data) and those of Hagberg et al demonstrate that regularly performed low-intensity aerobic exercise lowers arterial blood pressure at rest in middle-aged and older men and women with mild diastolic hypertension and that the degree of reduction can be as much or more than that observed in younger hypertensive subjects. Such changes should result in a lowering of cardiovascular risk in these older subjects based on data from clinical trials and epidemiological investigations using casual recordings of blood pressure. Therefore, it is not clear how discrepancies in training-induced blood pressure changes such as those observed in the present study affect the clinical importance of this intervention. Our data and earlier reports do indicate, however, that to obtain accurate information on chronic levels of arterial pressure over time, 24-hour ambulatory recordings should be performed whenever possible along with traditional casual readings.

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