Postexercise Blood Pressure Reduction in Elderly Hypertensive Patients

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OBJECTIVES
We sought to study: 1) the impact of hemodynamic and left ventricular function on short-term postexercise blood pressure reduction in elderly hypertensive patients; and 2) the 22-h postexercise effects on ambulatory blood pressure in elderly hypertensive patients.

BACKGROUND
Although early exercise provokes postexercise blood pressure reduction, the mechanisms underlying this response are not completely understood. Besides, it is unclear whether the reduction in blood pressure after exercise lasts long enough to have clinical relevance in elderly hypertensive patients.

METHODS
We studied 24 elderly hypertensive patients (age 68.9 ± 1.5 years) and 18 age-matched normotensive control subjects (age 68.1 ± 1.2 years). Cardiac output (carbon dioxide rebreathing) and blood pressure (auscultatory) were measured at rest and after a 45-min period of low-intensity bicycle exercise (50% maximal oxygen uptake) and at 15, 30, 60 and 90 min after exercise. Left ventricular function (by Doppler echocardiography) was also evaluated. Ambulatory blood pressure monitoring was evaluated after 45 min of exercise or 45 min of rest, in a randomized order.

RESULTS
In the hypertensive patients, exercise provoked a significant reduction in blood pressure, cardiac output, stroke volume and left ventricular end-diastolic volume. It also provoked a significant reduction in systolic, mean and diastolic blood pressure during a 22-h period, at daytime and nighttime.

CONCLUSIONS
The short-term reduction in blood pressure after exercise in elderly hypertensive patients is associated with a decrease in stroke volume and left ventricular end-diastolic volume. The 22-h postexercise reduction in blood pressure demonstrates the clinical relevance of low-intensity exercise in elderly hypertensive patients. (J Am Coll Cardiol 2002;39:676–82) © 2002 by the American College of Cardiology

There is accumulated evidence that a single period of exercise significantly reduces blood pressure during the postexercise period in spontaneously hypertensive animals (1) and in patients with hypertension (2–10). Nevertheless, the mechanisms underlying this response are still controversial. The reduction in blood pressure after early exercise has been attributed to reductions in cardiac output (CO) (8) and systemic vascular resistance (4,11) or both an early decrease in systemic vascular resistance and a later reduction in CO (2). On the basis of these findings, we have studied the hemodynamic responses during the postexercise period, as well as the left ventricular diastolic and systolic function after an exercise period in elderly hypertensive patients.

Despite the fact that a single period of exercise provokes a reduction in blood pressure during the postexercise period, it is not clear whether such a reduction lasts long enough to have clinical relevance, particularly in elderly hypertensive patients, in whom high blood pressure is of primary concern.

The aims of the present study were: 1) to test the hypothesis that 45 min of low-intensity dynamic exercise would cause postexercise hypotension in nonobese, elderly hypertensive patients, as a consequence of a decrease in CO, associated with changes in left ventricular function; 2) to test the hypothesis that the postexercise hypotension, associated with a decrease in CO, would significantly reduce cardiac work during the postexercise period; and 3) to test the hypothesis that low-intensity exercise would reduce blood pressure for a 22-h period in nonobese, elderly hypertensive patients.

METHODS
A total of 55 elderly, nonobese (body mass index <30 kg/m²) patients enrolled in the Exercise Cardiology Ambulatory of the Heart Institute (InCor), University of São Paulo Medical School, were selected according to the following criteria: age >60 years, clinical blood pressure levels not exceeding 160/110 mm Hg and no evidence of metabolic disorders, renal vascular hypertension, cerebral ischemic disease or obstructive coronary artery disease at the time of the study.

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None of the participants were taking any medications, nor were they involved in a regular exercise training program at the time of the study. Thirteen subjects were excluded from the study: three did not want to participate after having been submitted to the maximal progressive exercise test; five were involved in a regular exercise training program for at least six months; two were taking medications; and three presented with an exaggerated blood pressure response during the maximal exercise test. The Ethical Committee for Human Research Protocols of the University of São Paulo Medical School approved the study. After providing written, informed consent, 24 nonobese, elderly hypertensive patients (15 men and 9 women) and 18 age-matched, nonobese, elderly normotensive control subjects (9 men and 9 women) participated in the study. Hypertensive patients and normotensive control subjects were paired by age (68.9 ± 1.5 years vs. 68.1 ± 1.2 years; p = 0.6429), weight (75.2 ± 4.1 kg vs. 72.4 ± 2.9 kg; p = 0.5425), height (1.61 ± 0.03 m vs. 1.63 ± 0.02 m; p = 0.5891) and body mass index (28.1 ± 1 kg/m² vs. 27.4 ± 0.9 kg/m²; p = 0.2336).

Blood pressure levels were determined by taking three blood pressure measurements after a 5-min rest in the supine position, on two weekdays. The subjects were classified as hypertensive if the average of the systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels were similar or above 140 and 90 mm Hg, respectively (12), or normotensive if the average of the SBP and DBP levels were lower than 140 and/or 90 mm Hg, respectively (12). The SBP and DBP levels were significantly higher in hypertensive patients than in normotensive control subjects (157 ± 4 mm Hg vs. 126 ± 2 mm Hg and 93 ± 2 mm Hg vs. 79 ± 2 mm Hg; p < 0.0000 and p = 0.0000, respectively). Peak oxygen uptake (V\textsubscript{O\textsubscript{2}}peak) was not different between hypertensive patients and normotensive control subjects (17.9 ± 1.2 ml/kg per min vs. 16.1 ± 0.6 ml/kg per min, respectively; p = 0.1518). Maximal exercise capacity was determined by means of a maximal progressive exercise test on an electromagnetically braked cycle ergometer (Medifit 400L\textsuperscript{ }, Medical Fitness Equipment, Maarn, The Netherlands), with work load increments of 15 W every 3 min at 60 rpm until exhaustion. Oxygen uptake (V\textsubscript{O\textsubscript{2}}) and carbon dioxide production (V\textsubscript{CO\textsubscript{2}}) were determined by means of gas exchange, on a breath-by-breath basis, in a computerized system (CAD/Net 2001, Medical Graphics Corp., St. Paul, Minnesota). Peak V\textsubscript{O\textsubscript{2}} was defined as the maximal V\textsubscript{O\textsubscript{2}} attained at the end of the exercise period, in which the subject could no longer maintain the cycle ergometer velocity of 60 rpm. The peak heart rate achieved during the progressive exercise test was 143 ± 4 beats/min by hypertensive patients and 140 ± 4 beats/min by normotensive control subjects. The peak respiratory exchange ratio achieved during the progressive exercise test was 1.16 ± 0.02 by hypertensive patients and 1.18 ± 0.03 by normotensive control subjects.

Blood pressure was monitored noninvasively by means of a standard mercury sphygmomanometer. The heart rate was monitored continuously by electrocardiography. Cardiac output was measured noninvasively by means of the equilibration of the carbon dioxide rebreathing technique, as described previously (13). To reach the carbon dioxide equilibrium during the rebreathing maneuver and to estimate the oxygenated mixed venous partial arterial pressure of carbon dioxide, a carbon dioxide concentration between 10% and 14% was employed, and the equilibrium had to start in the first 10 s of rebreathing (14). To determine CO, a three-way valve on a computerized system with a breath-by-breath analysis of V\textsubscript{O\textsubscript{2}} and V\textsubscript{CO\textsubscript{2}} was connected to the subjects. Stroke volume (SV) was calculated by dividing CO (l/min) by heart rate (beats/min). Total peripheral resistance (TPR) was calculated by dividing the mean blood pressure (MBP) (mm Hg) by CO (l/min). The arterial-venous oxygen difference (\(a\text{-}V\text{O}_2\)) was calculated by dividing V\textsubscript{O\textsubscript{2}} (ml/min) by CO (ml/min). Cardiac work was calculated by the product of SBP (mm Hg), SV (ml/heart beat) and heart rate (beats/min).

Doppler echocardiography was performed with ultrasound equipment (Sequioa 512 model, Acuson Corp., Mountainview, California), before and at 30 min after exercise. The left ventricular mass, interventricular septum, posterior wall thickness, left ventricular end-diastolic diameter, end-diastolic volume, end-systolic volume, fractional systolic shortening and ejection fraction were evaluated.

Ambulatory blood pressure monitoring was performed using Spacelabs 90270 (Spacelabs Medical, Cedex, France), that was previously programmed to perform measurements at 10-min intervals for 22 h on two different days. The subjects were asked to document their activities, such as time in bed, time after retiring from work, time of awakening and time on the bus or in the car. They were instructed to perform the same activities on both experimental days, including exercise and rest, and at the same time on these days, as much as possible. In addition, on both days they were instructed not to exercise, not to take a shower, not to sleep during the recording interval of daytime and to relax and straighten out the arm during their waking hours. Three periods were taken into consideration for data analysis. First, the average time was obtained during the 22-h period of the two experimental days. This included the period from the time the subject left the laboratory until the
Protocol 1. The purpose of this protocol was to determine whether an early period of exercise provokes a blood pressure reduction during the postexercise period in elderly hypertensive patients and elderly normotensive control subjects and, furthermore, to study the hemodynamic mechanisms underlying this response in blood pressure. One week after the measurements of maximal exercise capacity, 23 hypertensive patients and 18 normotensive control subjects performed bicycle exercise for 45 min at 52 ± 1% and 51 ± 1% VO₂peak, respectively. Initially, the subjects were electrocardiographically monitored while sitting in a cycling position. A cuff for blood pressure evaluation was placed on the left arm. Then, the computerized ergospirometer with a three-way valve was connected to the subjects. After a 5-min period of adaptation, the measurements were started. Blood pressure was measured 30 s before each carbon dioxide rebreathing maneuver. The heart rate was recorded for 10 s immediately before the rebreathing maneuver. The measurements of blood pressure, heart rate and CO were performed at rest and at 15, 30, 60 and 90 min during the postexercise period. The work rate performed during the 45 min of exercise was 18.6 ± 5.7 W for hypertensive patients and 11.1 ± 3.1 W for normotensive control subjects. The average heart rate was 97 ± 3 beats/min and 93 ± 3 beats/min for hypertensive patients and normotensive control subjects, respectively, which corresponded to 68% and 66.5% of the maximal heart rate achieved in the progressive exercise test. Because one hypertensive patient could not tolerate high carbon dioxide concentrations during the CO measurements, she was excluded from this part of the study.

Protocol 2. The purpose of this study was to determine whether the hemodynamic changes during the postexercise period in elderly hypertensive patients were due to alterations in left ventricular diastolic and systolic function. Before cycling-positioned and baseline maneuvers, Doppler echocardiography was performed in nine hypertensive patients in the supine position. The same procedure was performed at 30 min after exercise. Cardiac output by carbon dioxide rebreathing was also evaluated in this study. Protocol 3. The purpose of this study was to determine whether an early period of exercise provokes a 22-h reduction in blood pressure in elderly hypertensive patients and in elderly normotensive control subjects. Ambulatory blood pressure monitoring was performed after exercise or rest, in randomized order, in 16 hypertensive patients and 18 normotensive control subjects. The time between sessions was five to seven days, and blood pressure monitoring always began at the same time of day (7:30 AM to 8:00 AM). Initially, the ambulatory blood pressure monitor was placed on a nondominant arm, with the subject in the supine position. After a 5-min rest period, the monitor was started, and the subject performed one of the two following experimental protocols: 1) a 20-min period in the supine position, a 7-min period in a cycle position, a 5-min period upright at rest, 3 min of cycling at 50% of a predetermined work load to elicit 50% VO₂peak for warm-up, a 45-min exercise period at 50% VO₂peak, a 5-min period for cool-down and a 55-min period of recovery in the supine position; or 2) a 20-min exercise period in the supine position, a 7-min period in an upright position in a comfortable chair, a 58-min rest period in an upright position and a 55-min period in the supine position. After these protocols, the subjects left the laboratory and returned the next day for the removal of the monitor.

Statistical analysis. Data are expressed as the mean value ± SEM. Two-way analysis of variance with repeated measures was used to compare the short-term postexercise effects on blood pressure and related hemodynamic responses, as well as the 22-h postexercise effects on SBP, DBP and MBP between the exercise day and control day in hypertensive patients and normotensive control subjects. The Scheffé post-hoc comparisons were performed as needed. The paired Student t test was used to compare pre-exercise and postexercise left ventricular function and demographic data. A p value <0.05 was considered as statistically significant.

RESULTS

Protocol 1. Systolic blood pressure, DBP and MBP levels at baseline and at 15, 30, 60 and 90 min during the postexercise period were significantly higher in hypertensive patients than in normotensive control subjects (group effect: p = 0.0000, p = 0.0005 and p = 0.0001, respectively; interaction: p = 0.0000, p = 0.0284 and p = 0.0000, respectively) (Fig. 1). Systolic blood pressure, DBP and MBP levels at 15, 30, 60 and 90 min in the postexercise period were significantly lower than those at baseline (time effect: p = 0.0000, p = 0.0003 and p = 0.0000, respectively). This reduction in SBP, DBP and MBP was due to hypertension, because SBP, DBP and MBP were lower at 15, 30, 60 and 90 min versus baseline only in the hypertensive group. Cardiac output levels throughout the experimental protocol were significantly higher in hypertensive patients than in normotensive control subjects (group effect: p = 0.0295; interaction: p = 0.1289) (Table 1). Cardiac output levels at 15 and 30 min during the postexercise period were significantly decreased, as compared with baseline (time effect: p = 0.0010). Stroke volume levels at baseline and at 15, 30, 60 and 90 min during the postexercise period were significantly higher in hypertensive patients than in normotensive control subjects (group effect: p = 0.0680; interaction: p = 0.0427) (Table 1). Stroke volume levels at 15 and 30 min during the postexercise period were
**Table 1.** Hemodynamic Responses at Baseline and During Postexercise (90 Min) in Elderly Normotensive Control Subjects and Elderly Hypertensive Patients

<table>
<thead>
<tr>
<th></th>
<th>Normotensive Control Subjects (n = 18)</th>
<th>Hypertensive Patients (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>15 min After Exercise</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>2.7 ± 0.2</td>
<td>2.6 ± 0.2</td>
</tr>
<tr>
<td>SV (ml/heartbeat)</td>
<td>38.5 ± 3.1</td>
<td>35.8 ± 2.7</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>72.4 ± 3.1</td>
<td>74.4 ± 2.7</td>
</tr>
<tr>
<td>TPR (U)</td>
<td>38.7 ± 2.2</td>
<td>40.2 ± 2.5</td>
</tr>
<tr>
<td>a-Vo2 (ml/100 ml)</td>
<td>7.3 ± 0.4</td>
<td>7.8 ± 0.4</td>
</tr>
<tr>
<td>CW (U·10⁻³)</td>
<td>352 ± 32</td>
<td>343 ± 28</td>
</tr>
</tbody>
</table>

*p < 0.05 for intergroup comparisons; †p < 0.05 for pooled data of both groups vs. baseline; ‡p < 0.05 for intragroup comparisons vs. baseline. Data are presented as the mean value ± SEM.

a-Vo2 = arterial-venous oxygen difference; CO = cardiac output; CW = cardiac work; HR = heart rate; SV = stroke volume; TPR = total peripheral resistance.
90 min in the postexercise period were significantly lower than those at baseline (time effect: $p < 0.0000$). This reduction in cardiac work was due to hypertension, because cardiac work at 15, 30, 60 and 90 min was lower than at baseline only in the hypertensive group.

**Protocol 2.** Exercise did not change the early diastolic filling rate to late diastolic filling rate ratio during the postexercise period (Table 2). However, exercise significantly decreased the left ventricular end-diastolic volume during the postexercise period, as compared with baseline. Exercise decreased the left ventricular end-systolic volume, but caused no change in the fractional systolic shortening and ejection fraction.

**Protocol 3.** The 22-h SBP and DBP levels are shown in Figure 2. The average of 22-h SBP, DBP and MBP levels were significantly higher in hypertensive patients than in normotensive control subjects (group effect: $p < 0.0000$, $p = 0.0000$ and $p = 0.0000$, respectively). In hypertensive patients, but not in normotensive control subjects, the 22-h SBP, DBP and MBP levels after exercise were significantly lower than the levels after the rest (control) condition (day effect: $p = 0.1408$, $p = 0.5061$ and $p = 0.0655$, respectively; interaction: $p = 0.0018$, $p = 0.0172$ and $p = 0.0125$, respectively). In hypertensive patients, but not in normotensive control subjects, the average daytime SBP, DBP and MBP levels after exercise were significantly lower than after the rest condition (day effect: $p = 0.1881$, $p = 0.3717$ and $p = 0.0677$, respectively; interaction: $p = 0.0043$, $p = 0.0258$ and $p = 0.0346$, respectively). Similarly, in hypertensive patients, but not in normotensive control subjects, the average nighttime SBP, DBP and MBP levels after exercise were significantly lower than after the rest condition (day effect: $p = 0.1363$, $p = 0.5069$ and $p = 0.0717$, respectively; interaction: $p = 0.0018$, $p = 0.0054$ and $p = 0.0016$, respectively).

**DISCUSSION**

The major new findings of the present study are: 1) the early postexercise reduction in blood pressure and CO after an early period of dynamic exercise in nonobese, elderly hypertensive patients is associated with a decrease in left ventricular end-diastolic volume; 2) the reduction in both blood pressure and CO after exercise provokes a significant decrease in cardiac work; and 3) the postexercise hypotension in nonobese, elderly hypertensive patients lasts for 22 h.

The hemodynamic data obtained in this study show that

### Table 2. Doppler Echocardiographic Characteristics at Baseline and After Exercise (30 min) in Elderly Hypertensive Patients

<table>
<thead>
<tr>
<th>Hypertensive Patients (n = 9)</th>
<th>Baseline</th>
<th>After Exercise</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular mass (g/m²)</td>
<td>161.7 ± 23.2</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Interventricular septum (mm)</td>
<td>11.1 ± 0.5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Posterior wall thickness (mm)</td>
<td>11.2 ± 0.5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>End-diastolic dimension (mm)</td>
<td>48.3 ± 1.6</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>End-diastolic volume (ml)</td>
<td>114.3 ± 10</td>
<td>99.6 ± 8.4</td>
<td>0.029</td>
</tr>
<tr>
<td>End-systolic volume (ml)</td>
<td>34.2 ± 7.1</td>
<td>23.2 ± 4.2</td>
<td>0.018</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.89 ± 0.08</td>
<td>0.85 ± 0.03</td>
<td>0.652</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>74.4 ± 5.1</td>
<td>76.5 ± 2.6</td>
<td>0.617</td>
</tr>
<tr>
<td>Fractional shortening (%)</td>
<td>44.9 ± 4.1</td>
<td>43.4 ± 2.2</td>
<td>0.852</td>
</tr>
</tbody>
</table>

Data are presented as the mean value ± SEM. E/A = early diastolic filling rate to late diastolic filling rate ratio.

![Figure 2](image-url) Levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP) during 22 h, at daytime and nighttime, on a control day and an exercise day in elderly normotensive control subjects (NC) and elderly hypertensive patients (HT). Note that blood pressure during 22 h, at daytime and nighttime, on the exercise day was significantly ($p < 0.05$) decreased in HT, but not in NC. *$p < 0.05$ for intragroup comparisons.
the postexercise hypotension in hypertensive patients is associated with a decrease in CO. In addition, during the first 30 min, this reduction in CO is primarily due to a decrease in SV, as a consequence of a lower left ventricular end-diastolic volume. From 60 to 90 min, the postexercise hypotension seems to be mediated by both the sustained reduction in SV and the progressive reduction in heart rate.

In normotensive individuals, in whom CO or SV was unchanged, no postexercise hypotension was found.

A previous study (8) of elderly hypertensive patients has attributed the reduction in blood pressure to a lower CO. The present investigation extends this finding to the left ventricular end-diastolic volume mediation. A reduction in venous return, or a decrease in blood volume, seems to explain the decrease in left ventricular end-diastolic volume after exercise. Both the increased muscle blood flow to eliminate the accumulation of metabolites after exercise (3,9) and the vasodilatory responses, as a consequence of the increased nitric oxide released during exercise (15), decrease the venous return during the postexercise period. Heat dissipation after exercise (16), which increases blood flow to the skin, can also reduce venous return. However, it is unlikely that these vasodilatory mechanisms, per se, explain the reduction in left ventricular end-diastolic volume. These mechanisms, whether maximally activated, would induce a decrease in TPR after exercise, which was not found in the present study. The reduction in blood volume during exercise, mediated by a lower plasma volume or intravascular to extravascular fluid shift during exercise, can decrease the venous return. Despite the fact that exercise at 50% VO₂peak, performed for 45 min, does not change the hematocrit rate in middle-aged individuals (data not published), we cannot guarantee that a decrease in blood volume has not taken place in elderly hypertensive patients. Nevertheless, a definite explanation for the reduction in left ventricular end-diastolic volume after exercise in elderly hypertensive patients is open for further investigation.

In the present study, there is no evidence of a decrease in left ventricular systolic function, because no change was found in the fractional systolic shortening and ejection fraction during the postexercise period as compared with baseline.

Some investigators (4,11) have attributed the reduction in blood pressure after exercise to a decrease in peripheral vascular resistance. Hara and Floras (11) demonstrated that postexercise hypotension was caused by peripheral vasodilation in young normotensive and hypertensive individuals. In addition, they reported that the reduction in SV was offset by persistent tachycardia, which resulted in higher CO after exercise. In the present study, we found no reduction in peripheral vascular resistance. There is, at least, a potential explanation for this controversy. The mechanisms underlying the postexercise vasodilatory response in elderly hypertensive patients are different from those in young hypertensive patients. In older patients, in whom vascular changes are expected (17), the blood pressure reduction depends primarily on decreasing CO rather than decreasing peripheral vascular resistance.

The transient tachycardic response in hypertensive patients, concurrently with hypotension after exercise, may be attributed to increased postexercise baroreflex sensitivity. In elderly hypertensive patients, in whom aging (18) and hypertension (10) reduce the baroreflex control, exercise that causes a decrease in blood pressure may improve baroreflex sensitivity. In fact, other investigators (19) have observed that baroreflex sensitivity is enhanced after an early period of exercise.

The novelty of the present study is the observation that low-intensity exercise provokes a significant decrease in cardiac work during the postexercise period in elderly hypertensive patients. These results have clinical implications, because cardiac work (flow-pressure product) is well correlated with myocardial VO₂ (20). In the present study, the decrease in cardiac work is primarily due to a reduction in SBP and SV. In normotensive individuals, in whom SBP and SV were unchanged after exercise, no reduction in cardiac work was found.

Low-intensity dynamic exercise decreases ambulatory blood pressure in elderly hypertensive patients. Furthermore, this reduction in blood pressure took place during both daytime and nighttime. One could argue that the quantity and/or quality of sleep may have been better in the night after exercise, simply because these patients were more tired as a result of the 45 min of exercise. This does not seem to be the case, because the reports on nighttime activity, with regard to time in bed and quality of sleep, were similar for both the control and exercise days. Quinn (21) reported that 50% to 75% of maximal oxygen consumption significantly decreased SBP and DBP for 24 h in middle-aged hypertensive patients. In a recent study, Taylor-Tolbert et al. (22) demonstrated that a period of moderate-intensity exercise provokes a decrease in blood pressure for 24 h in obese, elderly hypertensive patients. Our findings extend this finding to nonobese, elderly hypertensive patients.

At least one piece of information links our findings to the clinical relevance of exercise in elderly hypertensive patients. Elderly hypertensive patients can maintain lower blood pressure levels for 22 h, possible preventing cardiovascular events.

Conclusions. Forty-five minutes of low-intensity exercise decreases left ventricular end-diastolic volume and, as a consequence, SV and CO in nonobese, elderly hypertensive patients. These hemodynamic changes explain, in great part, the postexercise hypotension in nonobese, elderly hypertensive patients. Low-intensity dynamic exercise should be strongly recommended to elderly hypertensive patients, because, in addition to reducing cardiac work during the postexercise period, it provokes a decrease in blood pressure that lasts for 22 h.
REFERENCES