Aftereffects of Exercise on Regional and Systemic Hemodynamics in Hypertension

Jean Cléroux, N'guessan Kouamé, André Nadeau, Denis Coulombe, and Yves Lacourcière

Several studies have indicated that a single bout of physical exercise induced a significant antihypertensive effect during the hours after the activity. However, little information is presently available on the underlying hemodynamic changes. We examined 13 essential hypertensive patients and nine normotensive subjects in a randomized, crossover study design during 3 hours after a 30-minute period of upright leg cycling at 50% of peak aerobic capacity and during 3 hours after a 30-minute control period of rest. Blood pressure, heart rate, cardiac output, total peripheral resistance, and regional vascular resistance in the forearm as well as venous plasma catecholamine concentrations were measured repeatedly. After exercise, systolic (−11±2 mm Hg) and diastolic (−4±1 mm Hg) blood pressures, total peripheral resistance (−27±5%), forearm vascular resistance (−25±6%), and plasma norepinephrine levels (−21±7%) were significantly (p<0.05) decreased, and cardiac output was increased (+31±8%) compared with control in hypertensive subjects. In contrast, in normotensive subjects blood pressure, forearm vascular resistance, and plasma norepinephrine were unchanged, and systemic hemodynamics changed to a lesser extent than in hypertensive subjects after exercise. It is concluded that a decrease in regional vascular resistance in skeletal muscles and possibly in the skin in hypertensive patients may contribute importantly to the antihypertensive effect of prior exercise. A decreased sympathetic nervous activity, as seen from lower plasma norepinephrine levels, may be involved in this effect. (Hypertension 1992;19:183-191)
in blood pressure and systemic hemodynamics, and possibly in specific regional hemodynamics, would be consistent with changes in sympathetic activity.

**Methods**

**Subjects**

Thirteen patients with uncomplicated established mild-to-moderate essential hypertension (11 men, two women) and nine normotensive subjects (seven men, two women) gave informed consent to participate in this study, which was approved by our institutional ethics committee on human research. Secondary hypertension was ruled out by clinical and laboratory evaluation. Patients with labile hypertension or women of childbearing potential were excluded from the study. None of the subjects were receiving antihypertensive therapy or any other medication during this study. Hypertensive patients previously on antihypertensive therapy were gradually weaned off therapy over 2 weeks and remained without treatment during an additional 6-week period before entering the study.

All subjects had their blood pressure measured weekly during the 3 weeks preceding entry into the study. Office blood pressure was taken as the mean of three measurements in the sitting position at 5-minute intervals not differing by more than 5 mm Hg. Hypertensive patients qualified for the study if their diastolic blood pressure was between 90 and 114 mm Hg in the last two visits of this period. Normotensive subjects with diastolic pressure below 85 mm Hg qualified for the study. As part of the screening procedure, all subjects underwent a clinical treadmill test (Bruce protocol) with 12-lead electrocardiogram recording. None of the subjects displayed an abnormal response to this test, defined as a downsloping or a depression of the ST segment by 1 mm or more within 0.085 seconds of the R wave when exercising at up to 90% of the maximal age-predicted heart rate.

**Protocol**

The present study used a randomized crossover design to compare the aftereffects of exercise on hemodynamics with those of a nonexercise control situation. This study design was chosen to establish control values over the same time period as that required for the postexercise evaluation while avoiding an unduly extended stay in the laboratory. The two evaluations were 1 week apart. On the exercise day, leg exercise was performed for 30 minutes on a cycle ergometer at 50% of the measured peak oxygen uptake. On the nonexercise control day, the subject rested in the sitting position for the same duration. During exercise, oxygen uptake was monitored such that the work load could be adjusted to elicit 50% of individual peak oxygen uptake determined 2 weeks before the first hemodynamic study.

Each evaluation required 3.5 hours. The first hour was used for exercise or rest and for setting up measurement methods, and hemodynamics were examined during the remaining 2.5 hours. On arrival at the laboratory, a small catheter was placed in an antecubital vein of the subjects, who then either exercised or rested for 30 minutes. After a further 15-minute period of recovery while seated in a chair, the subjects moved to a bed where the measurement methods were readied. Hemodynamic measurements began 30 minutes after the end of the exercise or control rest period. Regional hemodynamics in the forearm and hand were studied during 1.5 hours, and systemic hemodynamics were examined during the last hour. Regional hemodynamic measurements were made during three 15-minute baseline periods with 15-minute intervals between. Systemic hemodynamic measurements were made during three 10-minute baseline periods with 10-minute intervals between. During the intervals, leg raising and lower body negative pressure maneuvers were applied. These did not affect resting values, and their circulatory effects have been reported elsewhere in a preliminary report. On study days, all subjects were instructed to have a light breakfast 1 hour before coming to the laboratory and to abstain from caffeine and smoking.

**Measurements**

**Oxygen uptake.** Oxygen uptake was measured from expired gases drawn at 250 ml·min⁻¹ from a 7-l mixing chamber with infrared absorption and zirconium cell analyzers for carbon dioxide and oxygen, respectively (Energy Expenditure Unit 2900, Sensormedics, Anaheim, Calif.). In this system, the analyzers and mass flowmeter are interfaced with a computer (model PS/2, IBM, Boca Raton, Fla.) to give 20-second averages of values sampled at 100 Hz. Peak oxygen uptake was measured during a progressively increasing work load test on a cycle ergometer (Ergomedic 829E, Monark, Varberg, Sweden) with increments of 50 W every 2 minutes. Peak value was considered to be attained when an increase in work load did not further elicit an increase in oxygen uptake or in heart rate, and respiratory exchange ratio was greater than 1.1.⁸

**Blood pressure and heart rate.** Blood pressure was measured with a standard mercury sphygmomanometer taking the first and the fifth Korotkoff sounds as the systolic and diastolic values, respectively, with a cuff of appropriate size. Mean blood pressure was calculated as diastolic plus one-third pulse pressure. Heart rate was measured with a tachograph triggered by the R wave of the electrocardiogram recorded in lead III (model 7P4, Grass Instrument Co., Quincy, Mass.), and both traces were recorded on polygraph paper.

**Regional hemodynamics.** Forearm blood flow was measured by venous occlusion plethysmography (model EC-4, D.E. Hokanson Inc., Bellevue, Wash.) using mercury-in-Silastic strain gauge⁹ applied around the arm contralateral to that used for blood pressure measurements and blood sampling. The strain gauge was placed 4–5 cm below the antecubital crease. Measurements were made at constant
room temperature (23°–24°C) while circulation to the hand was included and then excluded by inflating a wrist cuff 40 mm Hg above systolic blood pressure. Hand blood flow was obtained by subtracting forearm flow (wrist cuff inflated) from flow to the hand and forearm (wrist cuff deflated). Five-minute cycles with 2 minutes of circulation arrest at the wrist and 3 minutes of unrestricted flow to the hand were used. Measurements were derived from the average of three consecutive flow curves. Blood flow variability (standard deviation) calculated on sequential averages was ±5%, in agreement with previous reports.10,11 Forearm and hand vascular resistances were calculated by dividing mean arterial pressure by the respective blood flow. The values reported at each time point are the means of three series of measurements at 5-minute intervals.

**Plasma catecholamines.** Samples for catecholamine assay were taken during the last minute of each baseline period during regional hemodynamic measurements in the forearm and hand. Blood was drawn through an indwelling catheter (Cathlon IV, Critikon Inc., Markham, Canada) inserted in an antecubital vein at the beginning of each evaluation that was maintained patent with a heparin-lock solution. After discarding the first 2 cc, samples were collected in ice-chilled tubes containing EDTA and glutathione, processed at 4°C and stored at −70°C until analysis. Plasma catecholamines were assayed in duplicate with a specific and sensitive radioenzymatic assay using thin-layer chromatography for separation of methylated derivatives.12 In our hands, this method allows detection of levels of norepinephrine and epinephrine as low as 60 pM.

**Systemic hemodynamics.** Stroke volume was determined from M mode echocardiographic measurements (Mark III Ultrasonograph, ATL Company, Seattle, Wash.) of end-diastolic and end-systolic left ventricular internal diameters over three consecutive cardiac cycles during which heart rate was stable. Systolic and diastolic volumes were derived from the dimensions with the formula of Teichholz and associates.13 The transverse axis was located in two-dimensional mode. In our laboratory, the intra-subject variability (standard deviation) of stroke volume measurements 1 week apart in a mixed control group consisting of hypertensive and normotensive subjects was ±5.0% with a correlation coefficient of 0.85 (p<0.001).

Cardiac output was obtained from the product of stroke volume and heart rate recorded at the moment of echocardiographic measurement. End-systolic wall stress was calculated using the method of Wilson and coworkers14 and divided by end-systolic volume, thus providing an index of myocardial contractility that is independent of ventricular preload or afterload.15 Total peripheral resistance was calculated by dividing mean arterial pressure by cardiac output and multiplying by 80. The blood pressure value used for this calculation was the mean of the reading preceding and the reading following echocardiographic measurements. Changes in plasma volume that could have occurred as a result of the exercise were estimated from hematocrit and hemoglobin concentration in samples taken at midpoint during the control evaluation and after exercise.16

**Statistical Analysis**

Results, expressed as mean±SEM, were compared with analysis of variance for repeated measurements. When a significant (p≤0.05) F ratio was observed, Duncan's test was used to locate significant differences.17

**Results**

**Subjects' Characteristics**

Hypertensive and normotensive subjects were characterized by similar age, height, weight, and maximal oxygen uptake (44±2 and 41±2 years, 175±2 and 173±3 cm, 75±3 and 73±6 kg, 35±1 and 36±3 ml·kg⁻¹·min⁻¹, respectively). Office blood pressure (mean of three weekly visits) was significantly higher in hypertensive (151±3/99±1 mm Hg) than in normotensive (118±2/79±4 mm Hg) subjects. The hemodynamic and plasma catecholamine results obtained during the nonexercise control evaluation in hypertensive and normotensive subjects are presented and compared in Table 1. Regional hemodynamic and plasma catecholamines were measured in all 22 subjects. Systemic hemodynamics were performed in nine hypertensive and six normotensive subjects. It can be seen that supine systolic and diastolic blood pressure levels were greater in the

### Table 1. Regional and Systemic Hemodynamics and Plasma Catecholamine Levels in Hypertensive and Normotensive Subjects During the Control Evaluation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hypertensives</th>
<th>Normotensives</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>58±1</td>
<td>57±4</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>138±2*</td>
<td>105±3</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>98±2*</td>
<td>75±3</td>
</tr>
<tr>
<td>FBF (ml·100 cc⁻¹·min⁻¹)</td>
<td>1.8±0.1*</td>
<td>2.4±0.2</td>
</tr>
<tr>
<td>FVR (units)</td>
<td>62.7±2.6*</td>
<td>37.2±2.7</td>
</tr>
<tr>
<td>HBF (ml·100 cc⁻¹·min⁻¹)</td>
<td>0.9±0.1</td>
<td>0.8±0.2</td>
</tr>
<tr>
<td>HVR (units)</td>
<td>252±48*</td>
<td>143±22</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>68±6*</td>
<td>89±6</td>
</tr>
<tr>
<td>Cardiac output (l·min⁻¹)</td>
<td>3.9±0.3*</td>
<td>5.0±0.4</td>
</tr>
<tr>
<td>TPR (dynes·sec·cm⁻²)</td>
<td>2325±63*</td>
<td>1406±118</td>
</tr>
<tr>
<td>Contractility index (ESWS:ESVI) (10⁸ dynes·m²·ml⁻¹·cm⁻²)</td>
<td>2.71±0.21*</td>
<td>1.92±0.13</td>
</tr>
<tr>
<td>NE (pM)</td>
<td>1445±128*</td>
<td>746±109</td>
</tr>
<tr>
<td>EPI (pM)</td>
<td>76±11</td>
<td>101±16</td>
</tr>
</tbody>
</table>

Results are mean±SEM. *p≤0.05 compared with respective value in normotensive subjects. HR, heart rate; SBP and DBP, systolic and diastolic blood pressure, respectively; FBF and FVR, forearm blood flow and vascular resistance, respectively; HBF and HVR, hand blood flow and vascular resistance, respectively; TPR, total peripheral resistance; ESWS-ESVI, end systolic wall stress: end systolic volume index ratio; NE and EPI, norepinephrine and epinephrine, respectively. To convert pM to ng/ml, divide NE by 5.9 and EPI by 5.5.
hypertensive subjects, whereas heart rate was similar in both groups. With regard to regional hemodynamics, forearm blood flow was lower and forearm and hand vascular resistances were higher in hypertensive than in normotensive subjects, whereas hand blood flow was similar in both groups. As for systemic hemodynamics, stroke volume and cardiac output were lower and calculated total peripheral resistance and myocardial contractility were higher in hypertensive than in normotensive subjects. Plasma norepinephrine concentrations were higher in hypertensive than in normotensive subjects, whereas plasma epinephrine levels were similar in both groups.

**Exercise Results**

During exercise, systolic blood pressure increased to higher values in hypertensive (170±5 mm Hg) than in normotensive (138±4 mm Hg) subjects; diastolic pressure decreased slightly from resting levels in both groups but remained greater in the hypertensive subjects (90±2 versus 68±4 mm Hg). However, the magnitude of the increases in systolic blood pressure above resting values (measured before exercise) was identical in hypertensive (+20±3 mm Hg) and in normotensive (+20±4 mm Hg) subjects. Exercise also induced similar increases in heart rate (128±5 and 133±4 beats • min⁻¹) and in percent peak oxygen uptake in hypertensive (53±1%) and in normotensive (52±1%) subjects. The work loads maintained were not significantly different (NS) in hypertensive and normotensive individuals (84±6 and 91±11 W). Thus, the exercise represented the same absolute and relative intensity for hypertensive and normotensive subjects.

**Aftereffects of Exercise**

In hypertensive subjects, systolic, mean, and diastolic blood pressures were significantly lower after exercise compared with the nonexercise control evaluation (Figure 1). This antihypertensive effect was well sustained between 30 and 90 minutes after exercise (i.e., during the period of regional hemodynamic measurements) and averaged −11±2 mm Hg for systolic and −4±1 mm Hg for diastolic pressure. The antihypertensive effect was equally sustained between 2 and 3 hours after exercise (i.e., during systemic hemodynamic measurements) with the systolic pressure 9±2 mm Hg and the diastolic pressure 4±2 mm Hg below control values. These values did not differ significantly from the 30-90-minute postexercise period. As estimated from changes in hematocrit and hemoglobin concentration, plasma volume was not different between control and postexercise evaluations in hypertensive (−0.9±1.7%; NS) or normotensive (−0.4±1.0%; NS) subjects.

In hypertensive subjects, a well-sustained increase in forearm blood flow (+0.6±0.2 ml • 100 cc⁻¹ • min⁻¹, on average; p≤0.05) and decrease in forearm vascular
resistance (Figure 3) was seen during the 30–90-minute measurement period after exercise compared with the control evaluation. In contrast, no significant change in forearm blood flow (+0.2±0.3 ml·100 cc·min⁻¹; NS) or vascular resistance (Figure 3) was found in normotensive subjects during the same period after leg exercise compared with the control period. With regard to hemodynamics in the hand, significantly higher blood flows (hypertensive subjects, 2.6±0.4 versus 0.9±0.2 ml·100 cc·min⁻¹; normotensive subjects, 2.6±0.6 versus 0.8±0.1 ml·100 cc·min⁻¹; both p≤0.05) and lower vascular resistances (Figure 3) were observed in both groups 30 minutes after the end of exercise, and in normotensive subjects 60 minutes (hand blood flow, 1.7±0.3 versus 0.6±0.2 ml·100 cc·min⁻¹; p≤0.05) after the end of the exercise compared with the control evaluation.

Figure 4 shows the effects of prior exercise on plasma catecholamine levels in both groups of subjects. Plasma norepinephrine concentrations were significantly lower from 30 to 90 minutes after exercise than during the same period after the control period of rest in hypertensive subjects, whereas they were unchanged in normotensive subjects. Plasma epinephrine levels were also unchanged in normotensive subjects but were greater after exercise than after rest in hypertensive subjects (significantly at 60 minutes postexercise).

The results concerning systemic hemodynamics appear in Figure 5. Mean values of the results collected during the three 10-minute measurement periods at 10-minute intervals on a given day are presented because no significant differences were found between these periods. These values are therefore representative of the hemodynamic profile between 2 and 3 hours after rest and after exercise. In hypertensive subjects, stroke volume was significantly greater (+22±8%; p≤0.05) after exercise than control, whereas it was unchanged in normotensive subjects (+3±3%; NS). Cardiac output increased in both groups but significantly more in hypertensive than in normotensive subjects (+31±8% and +15±3%, respectively; p≤0.05). The ratio of end-systolic wall stress and end-systolic volume was unaffected by prior exercise in either group, indicating no impairment of myocardial contractility after exercise. Total peripheral resistance was significantly lower after exercise than during control in both groups. However, the decrease was almost twice as pronounced in hypertensive as in normotensive subjects (−27±5% and −15±3%, respectively; p≤0.05). Left ventricular internal diameter in diastole was not altered after exercise in hypertensive subjects (+0.9±0.8 mm, from
a control value of 50.1±1.8 mm; NS) but increased significantly in normotensive subjects (+1.1±0.4 mm from a control value of 50.8±0.8 mm; p≤0.05).

Discussion

The results of this study show that 30 minutes of mild leg exercise induced a significant and sustained depressor aftereffect on supine systolic, mean, and diastolic blood pressures for up to 3 hours in hypertensive subjects. These results agree with previous findings, although a decreased diastolic blood pressure has not always been found. The most important and novel findings, in view of the inconsistencies among earlier studies, are that this effect was associated with an important and significant fall in total peripheral resistance and also with sustained reductions in forearm vascular resistance and plasma norepinephrine in hypertensive subjects. These observations arise from the comparison of results obtained after a period of exercise to those of a nonexercise control period of the same duration, with the two evaluations performed in random order 1 week apart. Therefore, all responses reflect changes from values measured at the same time of day without prior exercise, and they do not depend on the cardiovascular reaction associated with anticipation of physical exercise or on a stressful reaction to an unfamiliar laboratory environment.

In addition, a group of normotensive individuals was evaluated according to the same protocol, and no significant change in blood pressure was found after exercise. The comparison of postexercise responses in both groups therefore allows identification of circulatory changes with which the decrease in blood pressure was associated.

Proposed mechanisms for the postexercise decrease in blood pressure include decreased blood volume, accumulation of vasodilatory metabolites, and thermoregulatory vasodilation. However, the results of the present study suggest that blood volume was not significantly altered by exercise in either group of subjects. Furthermore, exercise was performed at the same intensity in hypertensive and normotensive subjects. Thus, it is unlikely that metabolic mechanisms, thermoregulatory mechanisms, or both, were importantly involved in the decreased blood pressure that was found exclusively in hypertensive subjects. In addition, because most of the blood flow to the hand is distributed to skin, our results showing that hand vascular resistance was decreased transiently in both groups after exercise is consistent with the notion that hypertensive and normotensive subjects thermoregulated similarly af-
ter exercise. In hypertensive subjects, the fall in systolic blood pressure tended to be more marked at 30 minutes after exercise (i.e., when a significant fall in hand vascular resistance was detected), suggesting that thermoregulatory vasodilation could have contributed to the decrease in blood pressure. However, the magnitude of the fall in blood pressure was not significantly different from that observed at other times (−13±3 mm Hg at 30 minutes versus −10±3 and −11±2 mm Hg at 60 and 90 minutes, respectively; NS) when hand vascular resistance was no longer different from control.

The important fall in total peripheral resistance was the primary hemodynamic mechanism responsible for the decreased blood pressure after exercise in hypertensive subjects. Total peripheral resistance decreased significantly but to a lesser extent in normotensive individuals after exercise. In spite of the lack of effect of vasodilatory metabolites on blood pressure, their involvement in the reduced total peripheral resistance after exercise can therefore not entirely be ruled out. Cardiac output increased to a greater extent after exercise in hypertensive than normotensive subjects. This was related to differences in stroke volume, which increased only in hypertensive subjects, whereas a similar tachycardia was found in both groups. Factors affecting stroke volume include contractility, preload, and afterload. A decreased left ventricular function has earlier been suggested to be involved in the antihypertensive effect of mild exercise. However, in the present study, the contractility index, which has been shown to be independent of loading conditions, was unchanged in both groups after exercise. Because left ventricular internal diameter in diastole was unchanged after exercise in hypertensive subjects, this suggests that preload was unaffected, and the increased stroke volume is therefore likely to be related to the reduced afterload. Moreover, left ventricular internal diameter in diastole increased after exercise in normotensive subjects, and this did not affect stroke volume significantly.

Our results further show that parallel decreases in forearm vascular resistance and in blood pressure occurred after leg exercise in hypertensive subjects, whereas both forearm hemodynamics and blood pressure were unchanged in normotensive subjects. This indicates that hemodynamic changes in the main vascular tissues of the forearm (i.e., skeletal muscle and skin) were most probably involved in the antihypertensive effect of exercise. Although skeletal muscles constitute more than 60% of the forearm volume and skin represents less than 10%, the relative proportion of blood flow distributed to each tissue can vary considerably. Because forearm skin circulation is under different regulatory control than skin circulation in the hand, changes in the latter cannot be used to make conclusions regarding the regulation of the forearm skin or, by inference, forearm skeletal muscle circulation. Furthermore, if thermoregulatory vasodilation as seen from hemodynamics in the hand was reflected in forearm hemodynamics, then forearm vascular resistance should be lower after 30 minutes of exercise than after 60 and 90 minutes. Although there was a tendency for such an effect, this difference was not significant (−17±4 vs. −15±4 and −13±4 units at 60 and 90 minutes, respectively; NS).

Additional observations nevertheless suggest that the reduced forearm vascular resistance after exercise may have been related to changes occurring predominantly at the level of skeletal muscle vascular beds. These observations are related to plasma noradrenaline concentrations. Indeed, it has been shown that antecubital venous plasma norepinephrine is derived mainly from local release in the forearm and that skeletal muscle vascular beds are a major source of circulating norepinephrine, whereas skin contributes to a lesser extent. In the present study, the observation that plasma norepinephrine concentrations were decreased together with forearm vascular resistance from 30 to 90 minutes after exercise in hypertensive subjects and that both norepinephrine and forearm hemodynamics were unchanged in normotensive individuals is consistent with the hypothesis that sympathetic nervous activity to skeletal muscle vascular beds is reduced after exercise in hypertensive subjects. Although the increased flow in the forearm could theoretically have contributed to the lower norepinephrine levels through increased clearance, it has previously been demonstrated that a fivefold increase in forearm blood flow is not associated with any significant change in plasma norepinephrine levels. Thus, an altered clearance is not likely to have contributed importantly to the reduced norepinephrine levels.

Another observation linking the changes in plasma norepinephrine with skeletal muscle vascular resistance comes from the report of a decreased sympathetic nervous activity to skeletal muscles of the leg measured with microneurography after exercise. Indeed, in resting subjects, a close relation has been reported between sympathetic nervous activity in skeletal muscles of the leg and forearm, and there is a good correlation between microneurographic sympathetic nervous activity and venous plasma norepinephrine concentrations. To the extent that these relations are also valid after exercise when the leg has been active and the forearm has not, this suggests that a decrease in sympathetic nervous activity may contribute to the fall in blood pressure via an effect on skeletal muscle vascular resistance.

In contrast to lower norepinephrine levels, mildly elevated epinephrine concentrations were found after exercise in hypertensive subjects. There are a number of situations where a dissociation between sympathetic nervous and adrenal medullary responses can be found. As earlier suggested, greater circulating epinephrine levels could have contributed to the lower total peripheral resistance after exercise. However, since the changes in plasma epinephrine levels were not sustained through the recovery period.
and were small even when statistically significant (i.e., at 60 minutes postexercise), it is not likely that β2-receptor–mediated vasodilation plays an important role in the reduction in forearm vascular resistance after exercise in the hypertensive patients.

Relation With Previous Studies

Our findings on systemic and regional hemodynamics are at variance with earlier results showing decreased heart rate, stroke volume, and cardiac output and increased total peripheral resistance and increased forearm vascular resistance after exercise in hypertensive patients. In one of these studies, the fact that the subjects were evaluated during seated rest, rather than supine as in the present study, and were older than our subjects may have influenced outcome. It is also possible that the preexercise versus postexercise design used by Hagberg et al and Bennett et al might have elicited a defense reaction that could have induced a higher preexercise cardiac output and forearm blood flow and thus have masked the postexercise increase. Several points support this assumption. First, in these studies, baseline measurements were performed over a short period (15–20 minutes) immediately before exercise. Second, in the Hagberg et al study resting cardiac output tended to be greater in the subjects who were about to exercise than in their control group (4.5 versus 3.9 l/min). Third, in the Bennett et al study, preexercise forearm blood flow was much higher (approximately 6.5 ml·100 cc⁻¹·min⁻¹) than during the control evaluation of the present study, whereas postexercise levels were within the same range (approximately 3 ml·100 cc⁻¹·min⁻¹). Last, with regard to the study of Bennett et al, higher preexercise forearm blood flows were also reported in normotensive subjects compared with control levels in the present study, thus additionally supporting the concept of the anticipatory response.

The randomized crossover design used in the present study allowed us to avoid this effect. In contrast, our results agree with the observation that skeletal muscle sympathetic neural activity was reduced after exercise in hypertensive patients. It is interesting to note that this method required 30–45 minutes to set up and that the baseline period can be presumed to have lasted at least 1 hour in that study, thus reducing the possibility that the acute anticipatory response was present.

In conclusion, the results of the present study indicate that the antihypertensive aftereffect of exercise in hypertensive individuals is related to a greater decrease in total peripheral resistance than that found in normotensive subjects. Because significant reductions in forearm vascular resistance and in plasma norepinephrine were found exclusively in hypertensive subjects after exercise, it is suggested that a reduced sympathetic activity to skeletal muscles and also possibly to the skin contributes importantly to the antihypertensive action of prior exercise. Our results further indicate that the antihypertensive effect of exercise was not associated with thermoregulatory vasodilation or with cardiac function impairment.

Acknowledgments

We thank Marie Bouchard for skillful technical echocardiographic assistance, Jacques Renaud for plasma catecholamine determinations, and Diane Leroux for statistical analysis of the results.

References


---

**KEY WORDS** • cardiac output • catecholamines • vascular resistance • exercise • hemodynamics • essential hypertension
Aftereffects of exercise on regional and systemic hemodynamics in hypertension.
J Cléroux, N Kouamé, A Nadeau, D Coulombe and Y Lacourcière

Hypertension. 1992;19:183-191
doi: 10.1161/01.HYP.19.2.183
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://hyper.ahajournals.org/content/19/2/183

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not
the Editorial Office. Once the online version of the published article for which permission is being requested
is located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org/subscriptions/