

Jacopo M. Legramante, Alberto Galante, Michele Massaro, Antonio Attanasio, Gianfranco Raimondi, Fabio Pigozzi and Ferdinando Iellamo

Am J Physiol Regulatory Integrative Comp Physiol 282:1037-1043, 2002. First published Jan 24, 2002; doi:10.1152/ajpregu.00603.2001

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Hemodynamic and autonomic correlates of postexercise hypotension in patients with mild hypertension

JACOPO M. LEGRAMANTE,¹ ALBERTO GALANTE,¹ MICHELE MASSARO,¹
ANTONIO ATTANASIO,¹ GIANFRANCO RAIMONDI,¹
FABIO PIGOZZI,² AND FERDINANDO IELLAMO¹

¹*Dipartimento di Medicina Interna, Centro di Riabilitazione Cardiologica "San Raffaele" and Università di Roma "Tor Vergata," 00173 Rome; and* ²*Istituto Universitario di Scienze Motorie, 00194 Rome, Italy*

Received 3 October 2001; accepted in final form 21 November 2001

Legramante, Jacopo M., Alberto Galante, Michele Massaro, Antonio Attanasio, Gianfranco Raimondi, Fabio Pigozzi, and Ferdinando Iellamo. Hemodynamic and autonomic correlates of postexercise hypotension in patients with mild hypertension. *Am J Physiol Regulatory Integrative Comp Physiol* 282: R1037–R1043, 2002. First published January 24, 2002; 10.1152/ajpregu.00603.2001.—We investigated the interplay of neural and hemodynamic mechanisms in postexercise hypotension (PEH) in hypertension. In 15 middle-aged patients with mild essential hypertension, we evaluated blood pressure (BP), cardiac output (CO), total peripheral resistance (TPR), forearm (FVR) and calf vascular resistance (CVR), and autonomic function [by spectral analysis of R-R interval and BP variabilities and spontaneous baroreflex sensitivity (BRS)] before and after maximal exercise. Systolic and diastolic BP, TPR, and CVR were significantly reduced from baseline 60–90 min after exercise. CO, FVR, and HR were unchanged. The low-frequency (LF) component of BP variability increased significantly after exercise, whereas the LF component of R-R interval variability was unchanged. The overall change in BRS was not significant after exercise vs. baseline, although a significant, albeit small, BRS increase occurred in response to hypotensive stimuli. These findings indicate that in hypertensive patients, PEH is mediated mainly by a peripheral vasodilation, which may involve metabolic factors linked to postexercise hyperemia in the active limbs. The vasodilator effect appears to override a concomitant, reflex sympathetic activation selectively directed to the vasculature, possibly aimed to counter excessive BP decreases. The cardiac component of arterial baroreflex is reset during PEH, although the baroreflex mechanisms controlling heart period appear to retain the potential for greater opposition to hypotensive stimuli.

autonomic nervous system; baroreflex; heart rate variability; hemodynamics

AFTER A SINGLE BOUT OF DYNAMIC exercise, arterial blood pressure (BP) is usually reduced in hypertensive patients. This reduction in BP below preexercise control value has been defined as postexercise hypotension (PEH) (21). However, there is no general consensus on the mechanisms of PEH. Although PEH has been re-

ported to be mainly sustained by a persistent decrease in systemic vascular resistance, it has been debated whether changes in cardiac output (CO) also contribute to PEH (6, 9, 13). In addition, forearm vascular resistances (FVR) have been variously reported as increased (1) or decreased (6) after exercise, whereas leg vascular resistances have been evaluated only in one study (31) and found to be decreased after exercise. Alterations in neural cardiovascular regulation are also controversial. Some studies (6, 8, 13) reported a tachycardia during hypotension following exercise, whereas other studies (1, 9, 34) observed no significant change in heart rate (HR). The lack of the anticipated tachycardia suggested an alteration in arterial baroreflex modulation of HR. However, it has not been established whether, and if so how, the arterial baroreflex is altered during PEH in hypertensive patients (21). Surprisingly, only one study (34) has directly addressed the issue of baroreflex control of HR during PEH in hypertensive patients, showing an enhanced baroreflex sensitivity (BRS) in response to hypotensive stimuli. However, to our knowledge, the baroreflex control of HR in response to hypotensive stimuli, which would be more relevant to the mechanics of PEH, has never been addressed.

Accordingly, in the current study we investigated in hypertensive patients the neural mechanisms of cardiovascular regulation and systemic and regional hemodynamics associated with reduced BP after a single bout of maximal exercise. Through this study, we aimed to obtain further insights on the interplay of those mechanisms involved in PEH of hypertensive patients.

METHODS

Subjects

We studied 15 patients (12 male and 3 female patients; mean age 48.6 ± 5.3 yr) with uncomplicated, mild essential hypertension. Mild hypertension was defined on the basis of

Address for reprint requests and other correspondence: F. Iellamo, Dipartimento Medicina Interna, Università di Roma Tor Vergata, Via O. Raimondo, 8, 00173 Roma, Italy (E-mail: iellamo@med.uniroma2.it).

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medical history and sitting systolic BP (SBP) between 140 and 160 mmHg and/or diastolic BP (DBP) between 90 and 105 mmHg, as measured by conventional sphygmomanometry. All patients had BP measured on three or more occasions during the 3 wk before the study, in the laboratory in which the investigation was subsequently performed. BP was measured three times at 5-min intervals, after patients sat quietly for 15 min. All patients had no other diseases based on history, physical examination, electrocardiogram, routine laboratory analyses, and echocardiographic examination. Hypertensive patients had either never been treated or discontinued therapy for at least 2 wk before the study. All patients were nonsmokers, and no patient was engaged in regular physical activity. The work fully conforms with the "Guiding Principles for Research Involving Animals and Human Beings." Informed consent was obtained from each patient, and the study was approved by the Ethics Committee of the San Raffaele Cardiac Rehabilitation Center.

Measurements

BP and HR. BP was measured in the sitting position with a conventional sphygmomanometer. Patients were also connected to an analog multichannel signal conditioner and amplifier/filter (Marazza) to measure arterial BP continuously and noninvasively by Finapres while the electrocardiographic signal was simultaneously recorded. Respiratory excursions were also recorded by means of a piezoelectric thoracic belt. The three analog signals were sampled at 300 Hz/channel and stored for subsequent analyses. These signals were used to assess autonomic function (see below).

Systemic hemodynamics. With patients in the left lateral position, the parasternal long-axis view was used to measure end diastolic and end systolic left ventricular diameters as well as the aortic ring diameter. The instantaneous flow velocity in the ascending aorta was measured using continuous-wave Doppler directed through the suprasternal window with the patient supine. HR was derived from electrocardiographic trace monitoring. Stroke volume (SV) was calculated as the product of mean time-velocity integrals and the cross-sectional area of the aortic orifice, as described previously (13). Two assumptions were made in the measurements of SV: 1) the cross-sectional area was constant throughout the study and 2) the maximal flow signal was obtained at the aortic orifice area. These assumptions did not affect within-subject comparison of CO, because the aortic ring diameter is constant and independent of SV (13, 20). Doppler signals were recorded on a VHS videotape simultaneously with the electrocardiogram. CO was calculated as the product of SV and HR recorded at the moment of echocardiographic measurement. At least three consecutive cardiac cycles, when HR was stable, were averaged to derive a mean value for the time-velocity integral. Total peripheral resistance (TPR) was calculated by dividing mean BP (MBP) by CO. The BP value used for this calculation was the mean of the readings taken in the supine position immediately before and after the echocardiographic measurements. Because of unreliable echocardiographic windows, 1 of the 15 patients was discarded from central hemodynamic analyses.

Regional hemodynamics. We simultaneously measured forearm (FBF) and calf blood flow (CBF) by a computer-assisted venous occlusion plethysmographic device with the patients in the supine position, as previously reported (16). FVR and calf vascular resistance (CVR) were calculated as MBP divided by recorded flows. Because of artifacts in the plethysmographic traces, 2 of the 15 patients were eliminated from the CVR analysis.

Experimental Protocol

After the patients sat quietly for 15 min, BP was measured in the seated position twice (5 min apart), and the measurements were averaged. After instrumentation and a 20- to 25-min adaptation period to the supine position, patients underwent baseline echocardiographic examination, after which they were shifted into a stable supine position. Continuous arterial BP and HR recordings were performed for 10 min for autonomic assessment. In the last 2 min of this recording, sequential inflations and deflations of the cuffs were timed to provide three estimates of FBF and CBF per minute, which were averaged to obtain blood flows at baseline.

After baseline measurements, patients underwent a symptom-limited incremental (25 W/2 min) exercise test on a cycle ergometer. Sixty minutes after the end of the exercise, during which patients sat quietly in the laboratory, postexercise BP was measured by sphygmomanometer twice (5 min apart), and the measurements were averaged, as described above. Thereafter, patients underwent postexercise echocardiographic examination, after which continuous BP, HR, and peripheral blood flow measurements were repeated in the supine position, according to the same protocol used before exercise. The strain gauges on the forearm and calf were placed at exactly the same sites used before exercise, according to marks made on the skin with a dermatographic pencil. After the postexercise measurements obtained in all patients 60–90 min following exercise cessation, BP was measured again twice (at 5-min intervals) in the sitting posture by sphygmomanometry.

Spontaneous baroreflex analysis. Details of this analysis have been previously described (16, 18, 19). Briefly, the beat-by-beat time series of SBP and R-R interval were scanned by a computer to identify sequences of three or more consecutive beats in which SBP and R-R interval changed in the same direction [either increasing ("up sequences") or decreasing ("down sequences")]. A linear regression was applied to each sequence, and the mean individual slope of the SBP/R-R interval relationship, obtained by averaging all slopes computed within the test period, was calculated and taken as a measure of the integrated BRS (2). In addition, "nonbaroreflex" sequences, characterized by consecutive beats in which the SBP and R-R interval of the following beat changed not in the same direction but in the opposite direction (i.e., hypertensive/tachycardic and hypotensive/bradycardic sequences) were also calculated. These sequences have been considered as an expression of neural mechanisms regulating the cardiovascular function with positive feedback characteristics (23). The ratio between the number of baroreflex and nonbaroreflex sequences (the B-to-NB ratio), an estimate of the dynamic balance between negative and positive feedback mechanisms of short-term neural cardiovascular regulation (24), was also calculated.

Power spectral analysis. The methodology for spectral analysis has been described previously (17, 19). Briefly, a derivative-threshold algorithm provided the continuous series of R-R interval from the electrocardiographic signal. The harmonic components of R-R interval variability were evaluated by the autoregressive method. Spectral analysis of finger SBP and DBP and respiratory activity was performed on the signals sampled once for every cardiac cycle, employing a procedure similar to that described for the R-R interval. Stationary segments of data were selected by visual examination and analyzed (35). In short-term R-R interval and BP variabilities two main oscillatory components can be identified: one component is in the high frequency (HF) range

(0.15–0.4 Hz) synchronous with respiration, and the other component is usually centered on 0.1 Hz (low frequency; LF), but can vary from 0.04 to 0.15 Hz (25, 28, 30, 35). The very low-frequency component (<0.03 Hz) was not addressed in this study and is considered to be a direct current component (35). The power density of each spectral component was calculated in both absolute values and normalized units (28). The LF component of R-R interval and BP variability is considered a marker of efferent sympathetic cardiac and vascular modulation, respectively, whereas the HF component of R-R interval variability would reflect respiratory-driven vagal modulation to the sinoatrial node (17, 25, 28–30, 35).

Statistics

Each variable was checked for normal distribution by the Kolmogorov-Smirnov test. For variables that passed the test, we then used the paired *t*-test to compare pre- and postexercise values. The Wilcoxon signed-rank test was used for nonnormally distributed variables. Values are expressed as means \pm SE. Differences were considered statistically significant at $P < 0.05$.

RESULTS

Exercise Test

The maximum workload attained during the exercise test was 142 ± 2.9 W. HR increased from 72 ± 2.1 to 146 ± 2.9 beats/min ($87 \pm 2.0\%$ of the maximal age-predicted HR). All patients ended the exercise test because of muscular fatigue, and no complications were observed during or after the exercise.

BP

Arterial BP was significantly lower than control values 60 min after exercise. SBP decreased from 149 ± 4 to 137.9 ± 3.7 mmHg ($P < 0.01$) and DBP from 103 ± 1.9 to 97.7 ± 2.2 mmHg ($P < 0.05$) in the group as a whole. However, in 2 of 15 patients, BP did not decrease after exercise. In the patients with PEH ($n = 13$), SBP decreased from 150.4 ± 4.4 to 135.6 ± 3.9 mmHg ($P < 0.001$), and DBP from 103.5 ± 2.1 to 96.6 ± 2.3 mmHg ($P < 0.01$). Because this study aimed to investigate the mechanisms underlying the postexercise decrease in BP, only those patients exhibiting PEH were included in the subsequent analyses. Data from the two patients who did not have a postexercise decrease in BP are shown in Table 1.

Systemic and Regional Hemodynamics

PEH was accompanied by a significant decrease in TPR whereas CO and SV showed no significant changes after exercise. In addition, HR was not significantly different before and after exercise (Table 2). FVR did not change significantly, whereas CVR showed a marked and significant reduction from baseline values (Fig. 1). At the end of the recording sessions following exercise, BP was still lower than at baseline [136.9 ± 2.8 mmHg for SBP ($P < 0.001$) and 100.6 ± 2.2 mmHg for DBP (not significant)].

Baroreflex Control of HR and Power Spectral Analysis

The integrated BRS, as estimated by pooling together up and down sequences, showed no significant trend toward an increase during supine recovery after exercise compared with preexercise values (Table 3). However, when up and down sequences were analyzed separately, we found that BRS was significantly, albeit slightly, increased in response to hypotensive stimuli (i.e., decreasing BP ramps), whereas it was unchanged in response to hypertensive stimuli (i.e., increasing BP ramps), although a tendency toward increase was detected. The occurrence of baroreflex down sequences was also greater with respect to the preexercise period (Fig. 2). The B-to-NB ratio increased significantly during recovery after exercise (from 5 ± 2.1 to 9.5 ± 1.9 , $P < 0.05$), mainly due to a significant decrease in the occurrence of nonbaroreflex sequences (from 19.1 ± 3 to 14 ± 1.7 , $P < 0.05$). R-R interval spectral characteristics showed no significant changes after exercise (Table 3). LF power was significantly increased and normalized HF power was significantly reduced after exercise for both SBP and DBP. Changes in BP spectral powers are summarized in Table 4.

DISCUSSION

The novel findings of this study are as follows. First, postexercise vasodilation does not occur to the same extent in all vascular beds. Second, the vasodilator effect appears to override a concomitant reflex sympathetic activation directed to the vasculature. Finally, the cardiac component of the arterial baroreflex is reset during the hypotension after exercise, although the

Table 1. Hemodynamic data under baseline conditions and after exercise from patients without PEH

	BP, mmHg	HR, beats/min	CO, l/min	TPR, AU	CVR, AU	FVR, AU
<i>Patient 1</i>						
Baseline	130/100	93	8.8	7.1	14	6.8
Postexercise	150/110	100	8.6	9.6	12.6	1.3
<i>Patient 2</i>						
Baseline	150/100	60	8.1	9.5	24	13.7
Postexercise	155/110	57	7.6	10.3	24.4	17.7

PEH, postexercise hypotension; BP, systolic and diastolic blood pressure; HR, heart rate; CO, cardiac output; TPR, total peripheral resistance; CVR, calf vascular resistance; FVR, forearm vascular resistance; AU, arbitrary units.

Table 2. Systemic hemodynamics under baseline conditions and during PEH

	Baseline	PEH
TPR, AU	19.6 ± 1.5	18.4 ± 1.3*
CO, l/min	6.4 ± 0.4	6.2 ± 0.4
SV, ml	98.6 ± 7.7	91.1 ± 6.1
HR, beats/min	68.1 ± 2.6	69.5 ± 2.6

Values are means ± SE. SV, stroke volume. * $P < 0.05$ vs. baseline.

baroreflex mechanisms controlling heart period appear to retain the potential for greater opposition to hypotensive stimuli.

Central and Peripheral Hemodynamics

In keeping with most of the studies (6, 13) performed so far, the present investigation indicates that the main hemodynamic mechanism sustaining the reduction in BP after exercise in hypertensive patients is a decrease in TPR, and the lack of the decrease in TPR in the two patients who did not show a postexercise decrease in BP would confirm this view. However, investigating the vasomotor responses in different regional vascular beds allowed us to show, for the first time, that vasodilation may not occur in all vascular beds during PEH.

We observed a marked and significant reduction in CVR, i.e., the active limb during exercise, whereas vascular resistances in the nonactive limb, i.e., the forearm, were not significantly affected by the previous exercise. The observation of differential control of FVR and CVR is not new (7, 36); however, within the framework of PEH this finding might shed some light on the physiological mechanisms involved in the postexercise vasodilation (and hypotension) in hypertensive patients.

It has been suggested that in hypertensive patients, PEH is mainly due to a decrease in sympathetic outflow to the peripheral vessels (8). This suggestion was mainly based on the observation of a decrease in muscle sympathetic nerve activity (MSNA) following acute exercise. The different behavior of the active and inac-

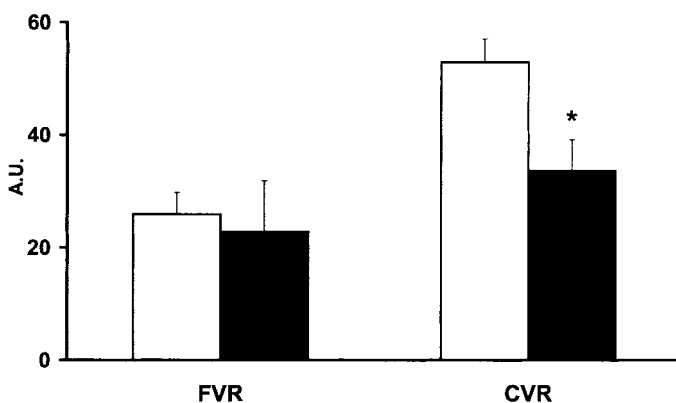


Fig. 1. Forearm (FVR) ($n = 13$) and calf vascular resistances (CVR) ($n = 11$) during baseline conditions (open bars) and 60 min after exercise (filled bars). AU, arbitrary units. * $P < 0.05$ vs. baseline.

Table 3. Spontaneous BRS and power spectrum analysis of R-R interval variability under baseline conditions and during PEH

	Baseline	PEH
BRS, ms/mmHg	6.7 ± 0.9	7.4 ± 1.1
Baroreflex sequences, no.	62.7 ± 11.2	73.1 ± 10.1
<i>R-R interval variability</i>		
Variance, ms ²	1,134.3 ± 202.2	1,242.5 ± 266.3
LF		
ms ²	341.1 ± 61.2	387.6 ± 103.7
NU	69.3 ± 3.6	67.5 ± 3.8
HF		
ms ²	145.7 ± 37.3	180.7 ± 53.5
NU	24.7 ± 3.1	24.8 ± 3.2

Values are means ± SE. BRS, baroreflex sensitivity; NU, normalized units. There were no significant differences for any variable between baseline and PEH.

tive vascular beds, along with the increase in indirect indexes of efferent sympathetic vascular modulation (see below) observed in this study, prompted us to suggest that mechanisms other than sympathoinhibi-

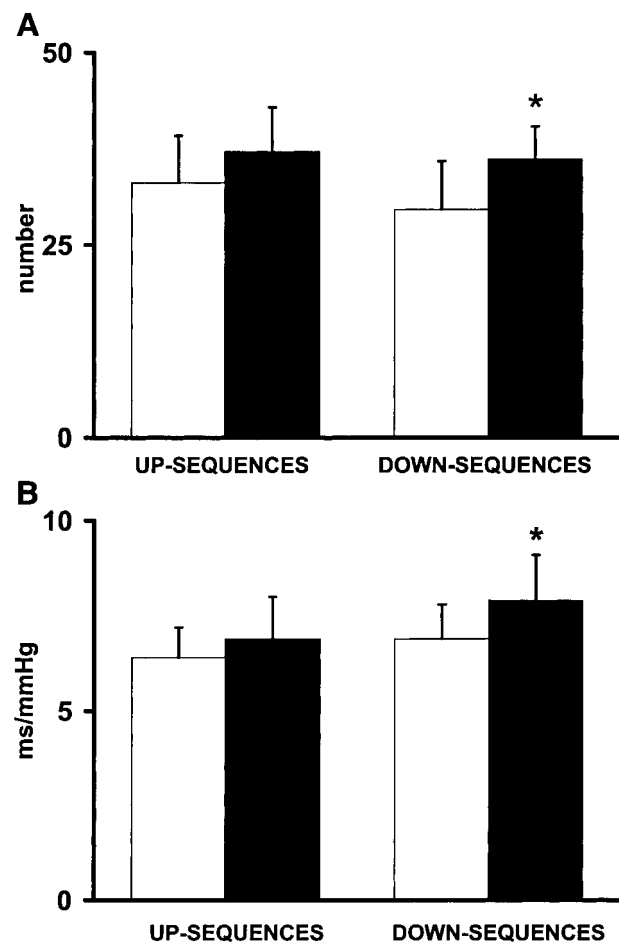


Fig. 2. The number of up and down baroreflex sequences (A) and baroreflex sensitivity values calculated separately for up and down baroreflex sequences (B) in baseline conditions (open bars) and 60 min after exercise (filled bars). * $P < 0.05$ vs. baseline.

Table 4. Power spectrum analysis of SBP and DBP variabilities under baseline conditions and during PEH

	Baseline	PEH
<i>SBP variability</i>		
Variance, ms ²	40.7 ± 7.8	46.8 ± 8.8
LF, ms ²	11.5 ± 2.4	18.6 ± 3.6*
HF, ms ²	3.2 ± 1	2.4 ± 0.7
<i>DBP variability</i>		
Variance, ms ²	12 ± 2.8	11.2 ± 1.7
LF, ms ²	4.4 ± 1	7.4 ± 1.4*
HF, ms ²	0.5 ± 0.1	0.4 ± 0.06

Values are means ± SE. SBP, systolic blood pressure; DBP, diastolic blood pressure. **P* < 0.05 vs. baseline.

tion might contribute to the reduction in TPR and thus to PEH in hypertensive patients (10).

The significant decrease in vascular resistances in the limb vascular bed involved in the exercise, which account for a large fraction of TPR (but not in the inactive limb), suggests that substances involved in postexercise hyperemia might play a crucial role in sustaining vasodilation and in turn PEH. Locally released vasodilator metabolites play a primary role in increasing muscle blood flow during exercise, and it has been suggested (15) that the marked increase in blood flow in the lower limbs after high-intensity exercise is controlled by complex interactions of various vasoactive metabolites. In addition, mechanical factors associated with increased arterial blood flow and arterial pressure in the vascular tree (i.e., the shear stress) are thought to be involved in the release of endothelium-derived vasodilating substances (27). Hence, we speculate that in hypertensive patients PEH is sustained by a decrease in vascular resistances related mainly to factors involved in postexercise hyperemia. Of note, Hara and Floras (13) also reported a decrease in CVR (not linked to a concomitant decrease in peroneal SNA) in a group of hypertensive patients similar to those in our study; however, they did not measure FVR. On the other hand, in previous studies, FVR has been variously reported as increased (2) or decreased (6) during PEH in hypertensive patients. The reasons for these discrepancies are not readily apparent. In any case, the contribution afforded by the forearm to TPR would be less than that of the legs, because of the smaller extent of the vasculature of the upper limbs.

In our hypertensive patients, CO did not show significant changes after exercise. Indeed, an earlier study by Hagberg et al. (9) was the only one to report a decrease in CO associated with increased TPR during PEH. In all other studies, CO has been found to be increased (6, 13) or unchanged (present study) in relation to augmented or unchanged HR. The elderly age of the hypertensive patients and the seated posture in which they were evaluated in the study by Hagberg et al. (9) might explain these discrepancies, since both factors could influence CO, TPR, and HR (3, 22).

Autonomic Function

As in other studies (1, 9, 34), the reduction in BP was not accompanied by the anticipated reflex increase in HR. This finding led to the hypothesis (1, 34) that in hypertensive patients there was a resetting of the arterial baroreflex control of HR after exercise. However, this possibility had not been directly tested in hypertensive patients. The only study (34) we are aware of that addressed the role of the arterial baroreflex in PEH in hypertensive patients investigated solely the reflex chronotropic response to phenylephrine-induced increases in BP. The present study is the first to examine the arterial baroreflex modulation of HR in response to both hypertensive and hypotensive stimuli during PEH in hypertensive patients. We observed a leftward shift in the baroreceptor-cardiac stimulus-response relationship to the lower BP level of postexercise recovery, with an unchanged overall BRS from the preexercise value, which is what defines baroreflex resetting (32). However, our results also indicate that the arterial baroreflex would not be simply reset along the prevailing (decreased) BP values of the postexercise period [i.e., a pressure-dependent, acute baroreceptor resetting (5)] but would also remain more sensitive to hypotension possibly to prevent an excessive postexercise decrease in BP, as would be suggested by the slightly augmented BRS in response to hypotensive stimuli (i.e., the decreasing BP ramps) and the concomitant increase in the number of baroreflex down sequences. However, because under normal conditions the baroreceptors are continuously engaged on a recurring basis by spontaneous, bidirectional fluctuations in BP above and below the mean pressure, the slightly increased BRS in response to hypotensive stimuli was not sufficient to induce the anticipated tachycardic effect. It should be mentioned that the spontaneous baroreflex method reflects mainly vagally mediated baroreflex responses (17, 19); it cannot address the contribution of the sympathetic component of the arterial baroreflex (32). However, the absence of a reflex increase in the LF component of HR variability during PEH, by reflecting no changes in the efferent sympathetic cardiac modulation may indicate, although indirectly, that during PEH the cardiac component of the arterial baroreflex was entirely reset toward the prevailing lowered BP. The unchanged HF component of HR variability also supports the fact that vagal control mechanisms were not altered to a great extent 60–90 min after cessation of a single bout of maximal exercise. As a result, HR was unchanged.

Interestingly, a resetting of baroreflex control of peripheral sympathetic outflow should have not occurred in our hypertensive patients, inasmuch as the LF component of BP variability, a marker of efferent sympathetic vascular modulation (17, 25, 28–30), increased significantly during the hypotension and vasodilation following exercise. This is a novel finding, strongly suggesting that in hypertensive patients there could be a differential reflex control of cardiac and peripheral sympathetic outflow in response to a sustained physi-

ological decrease in BP, as occurs after exercise. In this context, it might be important to recall that in hypertensive patients baroreceptor control of the sinoatrial node is impaired, but baroreflex control of the peripheral circulation is largely preserved (26, 33). The vasodilator effect had to override the concurrent reflex sympathoexcitation to the vasculature, for BP to be maintained persistently lower in recovery than during preexercise. The possibility that a decreased vascular responsiveness to α -adrenergic receptor activation might also contribute to the overriding vasodilator effect cannot be excluded (12, 14).

Our finding of a peripheral sympathoexcitation during PEH varies, in part, with other studies showing a sympathoinhibition after exercise. Floras et al. (8) reported a decrease in MSNA during PEH in young subjects (~ 25 yr) with intermittently elevated BP. However, Hara and Floras (13) subsequently reported a lack of sympathoinhibition (as inferred from MSNA and plasma norepinephrine concentration) in patients of slightly older age with mild essential hypertension subjected to the same exercise protocol. The patients enrolled in the present investigation were older than in the above studies. It is conceivable that the neural mechanisms associated with PEH may be heterogeneous among hypertensive patients and also differ as the patients age (and the hypertensive state) progresses. Another possible explanation is a differential sympathetic modulation of the different vascular beds. We observed a decrease in CVR but not in FVR, a finding that could be compatible with the decrease in peroneal MSNA observed by Floras et al. (8). However, it is not possible to ascertain whether in other vascular beds (e.g., renal and splanchnic regions) SNA was reduced or enhanced. The LF component of BP variability assessed in this study, on the contrary, would reflect, although indirectly, the overall rather than regional efferent sympathetic vascular modulation. Interestingly, our results extend to hypertensive patients those results obtained in normotensive subjects by Piepoli et al. (31), who also used spectral analysis of BP variability to assess changes in overall autonomic modulation during PEH.

When taken together, the results of this study, obtained in a fully unobtrusive experimental setting, and the findings of previous investigations (11, 13) support the view of a limited contribution of sympathoinhibition to PEH (10) and might help to delineate a new physiological concept for PEH in hypertension: the vasodilation with the attendant hypotension induced by a previous bout of exercise elicits a peripheral sympathoexcitation, possibly aimed at countering excessive decreases in BP. The observation of a significant increase in BRS in response to hypotensive stimuli along with the increase in the B-to-NB ratio supports the concept of an attempt by the autonomic nervous system to prevent excessive postexercise decreases in BP. Obviously, the possibility that differences in exercise modalities (short-term maximal vs. prolonged submaximal exercise) used in the various studies might differently affect the hemodynamic and autonomic

changes attending PEH in hypertensive patients should also be considered.

Potential Limitations

Our study did not define the vasodilator stimulus (or stimuli) responsible for PEH. Many substances have been proposed and subsequently abandoned as possible mediators of PEH (4, 11, 13). However, to date the nature of the vasodilating substances involved in PEH of hypertensive patients has yet to be determined (10).

A potential limitation of this study includes the indirect method used to assess changes in autonomic function. The issue of the validity of our approach for assessing autonomic function has been addressed by experiments in humans (30), in whom direct recordings of MSNA were performed during various states of autonomic regulation, as produced by graded infusions of vasodilator and vasoconstrictor drugs. The presence of similar oscillations at LF and HF in nerve activity and R-R interval and systolic arterial pressure variabilities at various levels of induced pressure changes provides support for the use of LF_{R-R} and HF_{R-R}, to infer the changing state of, respectively, sympathetic and vagal modulation of the sinoatrial node, and of LF_{SAP}, as an index of efferent sympathetic vascular modulation (30).

In conclusion, we investigated the interplay of the hemodynamic and neural mechanisms involved in PEH in middle-age hypertensive patients. Our results indicate that PEH is mediated mainly by a peripheral vasodilation, which may involve metabolic factors linked to postexercise hyperemia. The vasodilator effect appears to override a concomitant, reflex sympathetic activation directed to the vasculature, with the possible aim of countering excessive decreases in BP. Finally, the cardiac component of the arterial baroreflex is reset during hypotension following exercise, although the baroreflex mechanisms controlling heart period appear to retain the potential for greater opposition to hypotensive stimuli.

This study was supported in part by Agenzia Spaziale Italiana Grant ASI-99 and by Ministero dell'Università e della Ricerca Scientifica e Tecnologica (quota 40%-2000).

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