Effect of heavy exercise on spectral baroreflex sensitivity, heart rate, and blood pressure variability in well-trained humans

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Cottin F, Médiuge C, Papelier Y. Effect of heavy exercise on spectral baroreflex sensitivity, heart rate, and blood pressure variability in well-trained humans. Am J Physiol Heart Circ Physiol 295: H1150–H1155, 2008. First published July 11, 2008; doi:10.1152/ajpheart.00003.2008.—The aim of the study was to assess the instantaneous spectral components of heart rate variability (HRV) and systolic blood pressure variability (SBPV) and determine the low-frequency (LF) and high-frequency baroreflex sensitivity (HF-BRS) during a graded maximal exercise test. The first hypothesis was that the hyperpnea elicited by heavy exercise could entail a significant increase in HF-SBPV by mechanical effect once the first and second ventilatory thresholds (VTs) were exceeded. It was secondly hypothesized that vagal tone progressively withdrawing with increasing load, HF-BRS could decrease during the exercise test. Fifteen well-trained subjects participated in this study. Electrocardiogram (ECG), blood pressure, and gas exchanges were recorded during a cycloergometer test. Ventilatory equivalents were computed from gas exchange parameters to assess VTs. Spectral analysis was applied on cardiovascular series to compute RR and systolic blood pressure power spectral densities, cross-spectral coherence, gain, and α index of BRS. Three exercise intensity stages were compared: below (A1), between (A2), and above (A3) VTs. From A1 to A3, both HF-SBPV (A1: 45 ± 6, A2: 65 ± 10, and A3: 120 ± 23 mmHg, P < 0.001) and HF-HRV increased (A1: 20 ± 5, A2: 22 ± 8, and A3: 40 ± 11 ms², P < 0.02), maintaining HF-BRS (gain, A1: 0.68 ± 0.12, A2: 0.63 ± 0.08, and A3: 0.57 ± 0.09; α index, A1: 0.58 ± 0.08, A2: 0.48 ± 0.06, and A3: 0.50 ± 0.09 ms/mmHg, not significant). However, LF-BRS decreased (gain, A1: 0.39 ± 0.06, A2: 0.17 ± 0.02, and A3: 0.11 ± 0.01, P < 0.001; α index, A1: 0.46 ± 0.07, A2: 0.20 ± 0.02, and A3: 0.14 ± 0.01 ms/mmHg, P < 0.001). As expected, once VTs were exceeded, hyperpnea induced a marked increase in both HF-HRV and HF-SBPV. However, this concomitant increase allowed the maintenance of HF-BRS, presumably by a mechanoelectric feedback mechanism.

heavy exercise; cardio-respiratory interactions; baroreflex sensitivity; ventilatory thresholds; smoothed power spectral density

AT REST AND DURING exercise, short-term variabilities of heart rate (HR) and blood pressure (BP) result from complex interactions between central and peripheral regulators of different origins: mechanical, nervous, and hormonal. Among these determinants, the mechanical effect of breathing on BP has been well described (33). In healthy humans, during the inspiration phase of the breathing cycle generate the breathing variability of BP. Furthermore, these changes in BP secondarily induce a breathing variability of HR via baroreflex effect, which contributes to the respiratory sinus arrhythmia (RSA).

To quantify these short-term modulations of BP and HR, spectral analysis has been used (9, 10, 19, 23) providing two main frequency components: a low frequency (LF) ranging from 0.04 to 0.15 Hz (34) and a high frequency (HF) centered at the breathing frequency (34). At rest, the quantification of spectral components gave indexes of the autonomic control of HR and BP. On the one hand, it has been shown that the HF spectral component of HR variability (HF-HRV) is an index of the vagal tone (5, 6, 34), whereas both sympathetic and vagal activities contributed to the LF (LF-HRV) spectral component of HRV (5, 34). On the other hand, the LF (LF-SBPV) spectral component of systolic BP variability (SBPV) only reflected the sympathetic activity to the α-adrenergic receptors of vascular (14) whereas HF-SBPV probably reflected the mechanical effect of breathing on systolic blood pressure (SBP) (10).

Moreover, previous studies (1, 10, 19) have shown that, during graded moderate exercise (i.e., intensity below the first ventilatory threshold, V1), vagal tone progressively decreased, and SBPV increased, suggesting that SBPV could be counterbalanced at rest via HRV (baroreflex). During heavy exercise (i.e., intensity above V1), despite the complete vagal withdrawal, even though LF-HRV remained negligible (8, 9), HF-HRV after having reached a nadir around V1 increased via mechanoelectric feedback of hyperpnea to the sinus node (3, 4, 7, 8, 16, 17, 29).

In relation to SBPV, to our knowledge there is no study about SBPV during heavy exercise, probably due to the fact that noninvasive measurement of BP is rather difficult to perform during high exercise load. However, during heavy-intensity cycling, the substantial increase in breathing muscle favors a high venous return at inspiration. In addition, being very low at these exercise loads, HR amplitude modulations cannot buffer the SBPV. Thus the breathing variability of BP should markedly increase when the exercise intensity exceeds...
VT1 and should further increase when the intensity exceeds the second ventilatory threshold (VT2). Therefore, whereas HF-SBPV might be quite similar during two consecutive stages at low intensity, it should be different at high workloads, particularly just once VT1 and a fortiori VT2 are exceeded. It should be therefore relevant to compare spectral components during three distinct intensities: just below VT1, between VT1 and VT2, and above VT2.

Previous studies have explored the baroreflex sensitivity (BRS) at exercise with administration of different levels of pressure to the subject’s carotid baroreceptors with a neck collar (24, 25, 27, 31). They all showed no alteration of the BRS when exercise intensity increased but reported a resetting-offsetting of the stimulus-response curve that was linearly related to the exercise intensity. As an alternative to this method, it has been suggested that spectral analysis could provide a technique to noninvasively assess BRS (23, 26). In addition, BRS sensitivity can be assessed specifically in LF and HF (respiratory) ranges. A recent study (25) has shown that vagal pharmacological blockade decreased the baroreflex gain, whereas sympathetic blockade had no effect. Thus, with vagal tone decreasing and being no longer effective with increasing exercise load, breathing (HF) cardiac BRS should decrease during heavy exercise. Compared with the above-mentioned studies, the main advantage of assessing BRS from spectral analysis was that nonphysiological stimuli (neck chamber or other physiological maneuvers such as the phenylephrine method; see Ref. 26) can be avoided. Therefore, spontaneous BRS can be assessed during exercise without any potentially disturbing maneuvers from a physiological point of view.

The aim of the present work was to assess the instantaneous spectral components of HRV and SBPV, allowing LF and HF-BRS determination during a graded maximal exercise test. The first hypothesis of this study was that the hyperpnea elicited by heavy exercise should entail a significant increase in HF-BRS determination during a graded maximal exercise test.

METHODS

Fifteen competitive cyclists or triathletes (4 females and 11 males) participated in the study. All subjects were free of cardiac or pulmonary disease. The anthropometric and physiological characteristics of the subjects are summarized in Table 1. Before participating in the study, subjects were familiarized with the experimental procedure and informed of the risks associated with the protocol. All subjects gave their written voluntary informed consent in accordance with the guidelines of the University of Evry. In addition, the study was approved by the ethical committee of the University of Evry, France.

Anthropometric measurements. Height and weight were measured before each test. Four skinfold measurements were taken (triceps, biceps, suprailiac, subscapular) with percent body fat computed using the Durnin and Womersley’s (11) formula.

Table 1. Subjects characteristics

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
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<tr>
<td>Number</td>
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<td>4</td>
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<tr>
<td>Age, yr</td>
<td>27 ± 4</td>
<td>22 ± 12</td>
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<tr>
<td>Height, cm</td>
<td>178 ± 4</td>
<td>163 ± 2</td>
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<tr>
<td>Weight, kg</td>
<td>72 ± 8</td>
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<tr>
<td>Body fat, %</td>
<td>11 ± 6</td>
<td>22 ± 3</td>
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<tr>
<td>Absolute VO2max, ml/min</td>
<td>4,425 ± 439</td>
<td>3,374 ± 425</td>
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<tr>
<td>Relative VO2max, ml·min⁻¹·kg⁻¹</td>
<td>62 ± 10</td>
<td>51 ± 5</td>
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<tr>
<td>Maximal power, watts</td>
<td>359 ± 47</td>
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<tr>
<td>VT1, watts</td>
<td>252 ± 49</td>
<td>155 ± 38</td>
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<tr>
<td>VT2, watts</td>
<td>317 ± 48</td>
<td>194 ± 31</td>
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<tr>
<td>VT1, %maximal power</td>
<td>70 ± 9</td>
<td>66 ± 10</td>
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<tr>
<td>VT2, %maximal power</td>
<td>88 ± 6</td>
<td>83 ± 5</td>
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</table>

Data are presented as averages ± SD. VT1 is the first ventilatory threshold, while VT2 is the second ventilatory threshold.

Seat and handlebar heights were set for each subject and kept constant for all tests. During exercise, pedaling rate was kept between 70 and 100 revolutions/min.

Data collection procedures. Electrocardiogram (ECG) signals were digitized and recorded with a PowerLab device (ADInstruments) with a sampling frequency of 1,000 Hz. Beat-to-beat RR intervals were extracted from ECG using Chart5 software (Chart5, v5.5; ADInstruments). Artifacts, cumulative RR periods, and extrasystoles were manually processed by computation of interpolated or extrapolated values.

The Finometer (TNO; BMI) was used to measure BP from a cuff placed on a middle finger. Each subject was instructed to keep his/her finger relaxed on a special handlebar that supported the elbows and forearms during exercise. Systolic and diastolic BP yielded by the Finometer device have been previously validated during bicycle exercise and other laboratory tests (12, 13, 28). The Finometer was connected to the same analog/digital converter (PowerLab). As for ECG, BP signal was digitized and sampled at 1,000 Hz. The SBP series were assessed using a detection technique provided with the Chart5 program. Beat-to-beat SBP values were then measured with a 1 mmHg accuracy. Every SBP series was examined and corrected by interpolation when necessary. This procedure was thoroughly performed within the areas of analysis to eliminate all artifacts.

Gas exchange (VO2, VCO2, and VE) were measured with a Quark device (Quark PFT; Cosmed, Rome, Italy). Before each test, the O2 analysis system was calibrated using ambient air (20.9% O2 and 0.04% CO2) and calibration gas (12.01% O2 and 5% CO2). The calibration of the turbine flowmeter of the analyzer was performed with a 3-liter syringe (Quinton Instruments).

Ventilatory threshold assessment. Ventilatory thresholds were assessed from gas exchange components. The ventilatory equivalent method was used to determine VT1 and VT2 (32, 36). Therefore, VE/VO2 and VE/VCO2 were plotted vs. time during the incremental exercise test (see Fig. 2). VT1 corresponded to the first deflection point of the increase in the VE/VO2 curve, whereas the VE/VCO2 slope remained constant (32). In addition, VT2 related to the deflection point of the increase in the VE/VCO2 curve concomitant to a second sudden increase in VE/VO2 with a further increase in exercise intensity (32). Based on the above criteria, two experienced researchers have independently assessed the ventilatory thresholds. When there was a disagreement, a third experienced investigator was involved in the process. When he agreed with one investigator, the corresponding threshold was kept. When all of the investigators found different thresholds, the threshold of the considered subject was not taken into account.

Analysis areas. To explore the time course of the spectral components of SBPV, HRV, and BRS close to VTs, the following three areas of interest were defined: A1, below VT1, during the two successive
power stages immediately before VT1; A2, during all stages between VT1 and VT2; and A3, during all stages above VT2.

**Signal processing.** Before any signal processing, all SBP and RR series were resampled at 8 Hz using a cubic spline function (Scicos-Scilab; INRIA).

Signal processing was performed with a special fast-Fourier transform (Scicos-Scilab, INRIA, France) that provides a smoothed power spectral density (SPSD) of the SBPV and HRV. SPSD was used to compute LF and HF power of SBPV and HRV within the areas of interest (A1, A2, and A3). These spectral components were used to achieve the statistical analysis of the effect of exercise. SPSD has been described in previous studies (22, 23). Briefly, according to the kind of cardiovascular variability analyzed (LF or HF), SPSD was applied to Hanning windows of different sizes.

For LF ranging from 0.04 to 0.15 Hz (periods from 6.66 to 20 s) (34), a 64-s window (512 points) corresponding to a minimum of three maximal periods was chosen for LF analysis.

For HF, the breathing frequency of the 15 subjects ranging from 0.39 ± 0.07 to 1.04 ± 0.11 Hz (periods from 1 to 2.5 s), a 16-s window (128 points) corresponding to a minimum of 6.5 breathing periods was therefore chosen.

The spectral power was computed in LF and HF ranges (spectral components) by integrating the power spectral density. The LF band was set between 0.04 and 0.15 Hz as recommended by the Task Force (34), whereas the HF band was delimited for each subject in the range between the minimal and maximal values of its own breathing frequency as follows.

**BRS.** Because far as a minimal coherence between HRV and SBPV spectra was required to determine BRS, a cross-spectral analysis was applied on these spectra (22, 23, 26). Ranging from 0 to 1, the coherence function provided an estimate of the linearity between two spectra. It was considered that HRV and SBPV spectra were coherent when the coherence index was higher than 0.5. The coherence was required to validate spectral BRS computation. Next, BRS was computed by the following two parameters: the spectral gain, which was the modulus of the transfer function between SBP-SPSD and RR-SPSD; and the alpha index, which was the square root of the RR-SPSD/SPSBD ratio.

**Statistical analysis.** All results are given as means ± SE of the spectral power over the consecutive windows (for LF and HF) of each A1, A2, and A3 area. BRS (gain and alpha index) were computed only in A1, A2, and A3 area. BRS (gain and alpha index) were computed only when the spectral coherence of a window was higher than 0.5.

Parameters are mean values of LF and HF-HRV, LF and HF-SBPV, spectral coherence, BRS gain, BRS alpha index, and the percentage of spectra with coherence higher than 0.5.

The one-way ANOVA for repeated measures (Tukey test; SigmaStat 2.03) was used to compare the above-computed components between the three analysis areas. When the variables were not normally distributed, the Friedman repeated-measures ANOVA on ranks was then used. A P value <0.05 has been chosen as the significance threshold.

### RESULTS

**Ventilatory threshold detection.** The two ventilatory thresholds were detected for all the subjects.

**Duration of analysis areas.** Durations were as follows: A1 duration = 120 ± 0 s; A2 duration = 170 ± 15 s, and A3 duration = 159 ± 21 s. There was no significant difference between the durations of analysis areas.

**HR and SBP data.** From A1 to A3, HR increased (A1: 151 ± 19, A2: 165 ± 18, and A3: 178 ± 16 beats/min for all pairwise P < 0.001) and SBP increased (A1: 211 ± 28, A2: 215 ± 27, and A3: 219 ± 30 mmHg for all pairwise P < 0.001). An example of the ECG and BP waveforms at the different exercise intensity stages was shown in Fig. 1.

**HRV.** LF power decreased between the three areas [overall P < 0.008 and for all pairwise, A1: 208 ± 88 vs. A2: 30 ± 9 ms², P < 0.01; A1: 208 ± 88 vs. A3: 12 ± 2 ms², P < 0.01; A2: 30 ± 9 vs. A3: 12 ± 2 ms², not significant (NS), Fig. 3], whereas HF power increased between areas (overall P < 0.02 and for all pairwise, A1: 20 ± 5 vs. A2: 23 ± 11 ms², NS, A1: 20 ± 5 vs. A3: 39 ± 11 ms², P < 0.01; A2: 23 ± 11 vs. A3: 39 ± 11 ms², P < 0.05, Fig. 3).

**SBPV.** LF power of SBPV did not change between areas (A1: 1,234 ± 341, A2: 1,087 ± 194, and A3: 695 ± 184 mmHg, NS, Fig. 3), whereas HF power increased between areas (overall P < 0.001 and for all pairwise, A1: 45 ± 6 vs. A2: 65 ± 10 mmHg, NS; A1: 45 ± 6 vs. A3 120 ± 23 mmHg, P < 0.001; A2: 65 ± 10 vs. A3 120 ± 23 mmHg, P < 0.001, Fig. 3).

**Spectral coherence between SBPV and HRV.** The spectral coherence was not different between areas in LF (Table 2) and HF (Table 2). However, if the percentage of coherence within areas was not different between areas in LF (Table 2), it decreased in HF (P < 0.03, Table 2). In addition, the LF-BRS could not be computed in 1 subject out of 15 during A1 and
The first part of the discussion refers to the hypotheses regarding LF, BRS decreased with the spectral gain (overall $P < 0.001$ and for all pairwise, A1: 0.39 ± 0.06 vs. A2: 0.17 ± 0.02, $P < 0.001$; A1: 0.39 ± 0.06 vs. A3: 0.11 ± 0.01, $P < 0.001$; A2: 0.17 ± 0.02 vs. A3: 0.11 ± 0.01, NS, Fig. 3) and with the alpha index (overall $P < 0.001$ and for all pairwise, A1: 0.46 ± 0.07 vs. A2: 0.20 ± 0.02, $P < 0.001$; A1: 0.46 ± 0.07 vs. A3: 0.14 ± 0.01, $P < 0.001$; A2: 0.20 ± 0.02 vs. A3: 0.14 ± 0.01, NS, Fig. 3). Conversely, in HF, both BRS spectral gain and alpha index did not change between areas (spectral gain, A1: 0.68 ± 0.12, A2: 0.63 ± 0.08, and A3: 0.57 ± 0.09, NS and alpha index, A1: 0.58 ± 0.08, A2: 0.48 ± 0.06, and A3: 0.50 ± 0.09 ms/mmHg, NS, Fig. 3).

**DISCUSSION**

To our knowledge, SBPV has never been investigated during heavy to maximal exercise load. The new findings of this study are linked to the relationship between ventilation, HR, and BP variability during a maximal graded exercise test. Two hypotheses have been suggested: first, the hyperpnea elicited by heavy exercise could entail a significant increase in SBPV once VTs are exceeded. It was secondly hypothesized that, with vagal tone decreasing and being no longer effective with increasing exercise load, HF-BRS should progressively decrease during the exercise test. This study showed an increase in both HF-SBPV and HF-HRV when VT2 was exceeded, leading to preservation of HF-BRS and confirming the first hypothesis but rejecting the second. The first part of the discussion refers to the hypotheses regarding HF modulations while the second part refers to the other new findings of the study in relation to the LF modulation of HRV and SBPV. Last, a third part discusses the methodological aspects of the BRS assessment.

With HF-SBPV suddenly increasing only when VT2 was exceeded, the first hypothesis was therefore partly verified: the hyperpnea elicited by heavy exercise induced a significant increase in SBPV concomitant with the subjects reaching VT2 (Fig. 2 and 3). Thus hyperpnea has probably increased the respiratory modulations of BP via the venous return mechanism described at the beginning of the introduction. However, the hyperpnea elicited by exceeding VT1 was not strong enough to induce a significant difference between HF-SBPV in A1 compared with A2. Concomitantly, HF-HRV increased also when VT2 was exceeded (Figs. 2 and 3). This study therefore confirmed the results obtained in previous studies (4, 7, 8). Furthermore, Casadei et al. (5, 6) have shown that the residual HF-HRV observed during exercise was induced by nonneural mechanisms, including the mechanical effect of breathing, which rhythmically stretches the sinus node (16, 17, 18, 29). This phenomenon is called the mechanoelectric feedback (16). In addition, the contribution of these nonneural mechanisms in HRV genesis increased with increasing exercise intensity (5, 29). Because HF-HRV is vagally mediated at rest or during moderate exercise and cardiac vagal tone is no longer effective during heavy exercise, HF-HRV increased necessarily by mechanoelectric feedback (16, 17, 29). Consequently, when VT2 was exceeded, concomitant increases in both HF-SBPV and HF-HRV were observed. This putative double effect could preserve HF-BRS during heavy exercise (Fig. 3). The second hypothesis was therefore not verified. This result differs from the results of Macor et al. (19) who found a decrease in HF-BRS during moderate exercise (20 and 40% of maximal oxygen uptake). Conversely, some studies (24, 27, 31) reported a resetting of the operating point of the baroreflex curve during heavy exercise, demonstrating the preservation of the overall BRS. It is worthwhile to point out that the response of respiratory HRV induced by respiratory SBP modulations is a local baroreponse presumably determined by mechanoelectric feedback. The highly coherent
ent interaction between HF-SBPV and HF-HRV (coherence index > 0.5) allowed the BRS computation. However, because no reflex was involved in this phenomenon, this baroresponse cannot be an actual baroreflex. Indeed, the implicit model of spontaneous baroreflex (either in frequency domain, i.e., alpha index, or time domain, i.e., baroreflex slope) considers that arterial pressure changes directly and fully drives the sinus node (i.e., RR). This simplification might hold in control conditions, where respiration is slow and shallow, but does not stand during exercise conditions where respiration rate and volumes are high and RR variance extremely low. In these instances, high coherence does not protect from mistakenly considering SBP and RR variabilities as causally linked, through a reflex, instead of being simultaneously driven by a central signal as respiratory drive, but more complex (e.g., trivariate: RR, SBP, respiration) models are better suited. This point is clearly evidenced by a study of Mancia et al. (21) showing that a high RR-SBP coherence was maintained even after complete baroreceptor denervation in conscious cats. At any rate, contrary to the above study, the present study found that, despite the persistence of HF-BRS when VTs were exceeded, the percentage of HF spectral coherence phases decreased from A1 to A3. Thus a decay in baroresponse efficiency appeared at high exercise intensity.

Regarding LF-SBPV, the results of this study reported no change when VTs were respectively exceeded. So far, no study has investigated SBPV during heavy exercise. On the other hand, previous studies showed an increase in LF-SBPV with increasing load during moderate exercise (10, 19). However, it has been suggested that, during heavy exercise, the sympathetic nervous system became quickly saturated (20). Another report suggested the sympathetic nervous system response could be replaced by hormonal adjustment mechanisms (15). In addition, it has been shown that \( \alpha \)-adrenergic blockade with prazosin decreased LF-SBPV in conscious rats (14). Thus it could be possible that the high level of circulating catecholamines observed during heavy exercise (37) entailed a saturation of the \( \alpha \)-adrenergic receptors in muscular vessels, which could inhibit a further increase in LF power of SBPV. Regarding HRV, previous studies found a decrease in LF with increasing intensity during moderate (10, 19, 34) and heavy (8, 9) exercise. The results of this study confirm those of previous studies, namely LF-HRV decreasing when VTs were exceeded. On the one hand, with the vagal tone participating in the LF-HRV genesis, the observed decrease in LF could be attributed to vagal withdrawal during moderate exercise. On the other hand, with LF-HRV being also linked to sympathetic activity, another plausible explanation is the saturation of the cardiac \( \beta \)-receptors by catecholamines. This phenomenon could reduce the sympathetic variability entailing the decrease in LF power of HRV (15). Regarding LF-BRS, because there was no significant change in LF-SBPV from A1 to A3 while LF-HRV decreased, a drop of LF-BRS occurred despite no difference in the percentage of coherence between LF-SBPV and LF-HRV. This result confirmed those obtained during moderate exercise (19).

Methodological aspects of the BRS assessment. As a matter of interest, baroreflex gain is usually estimated by administration of vasovagal drugs (26) or pressure stimuli to the subject’s carotid baroreceptors with a neck collar (24, 25, 27, 31). With these methods, substantial SBP and HR variabilities can be observed. Porta et al. (30) suggested to remain cautious about these procedures, since they are based on nonphysiological stimuli that may alter the actual baroreflex gain. Several techniques have been proposed to evaluate the baroreflex gain based on the spontaneous variability of SBP and HR. Hence, one can distinguish two kinds of methods: sequential (2) and spectral (23, 26). In the present study, no significant difference was found between the results obtained with BRS gain and alpha index computation. Pagani et al. (26) compared phenylephrine with spectral results in both LF and HF ranges to estimate changes in autonomic regulation induced by training in mildly hypertensive subjects. They found a correlation between the two methods with a gain of similar magnitude for the two frequency ranges, inferring that the two methods provided complementary information. In this study, the choice of the spectral method allowed investigation of the spontaneous interactions between HRV and SBPV during a maximal graded exercise test with no other physiological or pharmacological maneuvers, which might have corrupted the effect of heavy exercise. Furthermore, the spectral method allowed distinguishing LF- from HF-BRS, with each one corresponding to different mechanisms. Therefore, the spectral method seemed to be more appropriate than the sequential method to quantify BRS during heavy exercise.

Conclusion

To our knowledge, this is the first study that investigated BP variability during heavy to maximal exercise and examined its relationship with different levels of exercise intensity correspond-
ing to VTs. The exercise-induced hyperpnea was associated with a marked increase in both respiratory HRV and SBPV only when VT2 was exceeded, whereas this effect was not strong enough at exercise intensities between VT1 and VT2. Thus BP oscillation buffering, via HRV, was partially preserved by the baroresponse due to mechanoelectrical feedback. Conversely, a decrease in BRS was observed in the LF range. The second hypothesis was therefore verified only in the LF range but not in the HF one. Last, on the one hand, the results of this study confirm those of previous studies (5, 6) that have shown that nonneural mechanisms are mainly involved in the RSA genesis during heavy exercise. On the other hand, this study gives every indication that the breathing variability of the SBP during heavy exercise is also mainly due to the mechanical effect of hyperpnea.

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GRANTS

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REFERENCES


