Heart Rate Variability during Exercise Performed below and above Ventilatory Threshold

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ABSTRACT
COTTIN, F., C. MÉDIGUE, P.-M. LEPRÈTRE, Y. PAPELIER, J.-P. KORALSZTEIN, and V. BILLAT. Heart Rate Variability during Exercise Performed below and above Ventilatory Threshold. Med. Sci. Sports Exerc., Vol. 36, No. 4, pp. 594–600, 2004. Purpose: To examine whether differences in heart rate variability (HRV) can distinguish sub- from supra-ventilatory-threshold exercise and whether the exercise duration at supra-threshold intensity alters cardiorespiratory synchronization. Methods: Beat-to-beat RR interval, \( \overline{V\dot{O}_2}, \overline{V\dot{CO}_2}, \overline{V\dot{E}} \), and blood lactate concentration of 11 healthy well-trained young subjects were collected during two exercise tests: 1) a moderate-intensity test: 15 min performed below the power at ventilatory threshold (pVT); and 2) a heavy-intensity test: above pVT until exhaustion. Fast Fourier transform, smoothed pseudo Wigner-Ville distribution, and complex demodulation were applied to RR time series. Results: 1) Moderate exercise shows a prevalence of low-frequency (LF) spectral energy compared with the high-frequency (HF) one (LF \( = 80 \pm 10\% \) vs HF \( = 20 \pm 10\%, p < 0.001 \)), whereas the reverse is observed during heavy exercise (LF \( = 11 \pm 8\% \) vs HF \( = 89 \pm 8\%, p < 0.001 \)). 2) During heavy exercise, the HF amplitude and the tidal volume (Vt) remained constant, whereas the breathing frequency (BF) increased (BF \( = 0.70 \pm 0.18 \) vs 0.93 \( \pm 0.31 \), \( p < 0.01 \)) and mean RR decreased (342 \( \pm 15 \) vs 317 \( \pm 16 \), \( p < 0.01 \)). Despite the RR series and the breathing signal remaining synchronized, HR/BF ratio decreased and stabilized at 3 RR for one breathing cycle, whatever the initial ratio. Conclusion: 1) HRV allows us to differentiate sub- from supra-ventilatory-threshold exercise and 2) exercise duration at supra-threshold intensity does not alter the cardiorespiratory synchronization. Key Words: CARDIORESPIRATORY INTERACTIONS, BREATHING FREQUENCY, SPECTRAL ANALYSIS, TIME FREQUENCY ANALYSIS

Heart rate variability (HRV) analysis has been broadly used in sports science to assess the autonomic control of heart rate (HR) at rest (15,24,25) and during moderate exercise (10,20), whereas it has been less studied during heavy exercise (i.e., with exercise intensity above ventilatory threshold).

In healthy subjects, the spectral analysis of short-term HRV recordings (5–20 min) provides two main frequency peaks: a high-frequency peak (HF) corresponding to the breathing frequency (BF), i.e., usually between 0.2 Hz at rest and to 1 Hz during severe exercise. This phenomenon is called “respiratory sinus arrhythmia” (RSA). The HF peak resulting mainly from baroreflex buffering changes induced by the mechanical effect of breathing (30) is mediated by cardiac parasympathetic nervous system (PNS) activity (12,23). Another peak can be found within the low-frequency band (LF) (close to 0.1 Hz). It is now admitted that this LF variability is induced by both sympathetic nervous system (SNS) and PNS cardiovascular control (12,23,29).

Cardiorespiratory (CR) interactions have been also investigated by spectral analysis. The breathing pattern has a strong effect on HF-HRV in resting subjects with the spectral energy in the HF band decreasing when breathing frequency (BF) increases and increasing when tidal volume (Vt) increases (16,18). The effect of exercise intensity on breathing pattern is also well known. At low levels of exercise, increasing the work intensity entails an increase in both BF and Vt. At high levels of exercise, increasing the work intensity entails an increase in BF only with Vt remaining constant (8,13). In addition, during incremental exercise, an increase in sympathetic tone and the withdrawal of parasympathetic activity were associated with a decrease in both LF and HF-HRV components when HR, BF, and Vt increased (10,20).

However, autonomic cardiovascular control is not the only mechanism that induces HRV. For instance, previous studies of heart transplant recipients showed an expected
disappearance of HRV at rest, because of the complete denervation of the graft (1,4). However, during exercise, HRV appeared unexpectedly and was related to the effect of breathing (4). In healthy subjects, during heavy exercise, i.e., a work rate higher than the power at ventilatory threshold, cardiac vagal control is no longer effective (27). Although vagal withdrawal is conflicting with an increase in HF-HRV, the large hyperventilation induced by heavy exercise could be the result of a mechanical effect on the sinus node, inducing an increase in HF-HRV (like in heart transplant recipients). During the inspiration phase, the expansion of the rib cage induces a decrease in intrathoracic pressure that in turn increases the venous return and consequently increases the filling of the right pump (27). This increase induces a stretch of the sinus node, provoking an increase in the sinus node activity and an increase in HR. During the expiration phase, the emptying of the lungs provokes the opposite effect with a decrease in HR. In agreement with this hypothesis, others showed that the sinus node could in fact work like a stretch receptor (17).

To our knowledge, short-term HRV during sub- versus supra-ventilatory threshold has not been investigated and gives the opportunity to compare two different conditions of cardiac control. In agreement with the conclusion of Casadei et al. (6,7), we hypothesize that HRV could be mainly generated by neural mechanisms at rest and also during moderate exercise, whereas the residual HRV remaining during heavy exercise could be due to the mechanical effect of breathing on the sinus node. Therefore, the aim of the present study was twofold: 1) to compare HRV spectral components during steady state exercise, at two different exercise intensities corresponding to sub- (moderate exercise) and supra-ventilatory threshold (heavy exercise) and 2) to study the effect of exercise duration on HRV and dynamic cardiorespiratory interactions by analyzing the instantaneous spectral parameters (amplitude, frequency, and phase) and comparing spectral energies at the beginning and at the end of the heavy exercise test, as changes in BF during heavy exercise could entail a modification of the HRV components (8,13).

METHODS

Subjects

Eleven healthy well-trained pubertal subjects (nine males and two females; 14.6 ± 1.1 yr) participated in this study. These subjects were regional elite triathletes. All subjects were free of cardiac and pulmonary disease. The anthropometric and physiological characteristics of the subjects are summarized in Table 1. Before participation, each subject was 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 Hôpital St. Louis (Paris).

Experimental Design

Subjects performed two sets of exercise at 4-d intervals in the upright position on an electronically braked bicycle ergometer (ERGOLINE 900, Hellige, Market). Seat and handlebar heights were set for each subject and kept constant during all tests, as was the pedaling frequency, which was selected by each subject at a rate between 70 and 110 rev.min⁻¹.

The first exercise protocol was an incremental test to determine the power at ventilatory threshold (PVT) and the power at VO₂max (PVVO₂max). According to the performance level of the subjects, the initial power was set between 30–80 W and was increased by 15 W every minute. VO₂max was defined as the highest oxygen uptake obtained in two successive 15-s interval periods during the incremental test with a respiratory exchange ratio (RER = VO₂/CO₂) greater than 1.1, a blood lactate greater than 8 mmol.L⁻¹ and a HR peak at least equal to 90% of the age-predicted maximum. A plateau of VO₂ was assumed if the VO₂ of the last stage was not greater than the previous one by 1.75 mL.min⁻¹.kg⁻¹. PVVO₂max was defined as the lowest power associated with VO₂max (5). The power at the ventilatory threshold (PVT) was detected using the V-slope method, which computes a regression analysis of the slopes of the CO₂ uptake (VCO₂) versus O₂ uptake (VO₂) plot (3). The beginning of the excess CO₂ output determines PVT.

During the second exercise protocol, subjects performed a 15-min warm-up on a bicycle ergometer at a work rate corresponding to 30% below PVT (P₃₀) immediately followed by an all-out constant-load exercise at 10% above PVT (P₁₀⁻). Data Collection Procedures

**Time series.** R-R intervals were recorded beat-to-beat using an S 810 HR monitor, (Polar Electro Oy, Kempele, Finland) with a sampling frequency of 1000 Hz from EKG signal providing an accuracy of 1 ms for each RR period.

**Gas measurements.** VO₂, VCO₂, and VE were performed throughout each test using a telemetric system (K4b², COSMED, Rome, Italy) (19). Expired gases were measured breath by breath and averaged every 5 s. The response time of the oxygen and carbon dioxide analyzers were less than 120 ms to reach 90% of the flow sample. The time delay of the gas analyzer (time necessary for the gas to transit through the sampling line before being analyzed) was about 500 ms. This time delay was automatically assessed and taken into account in the metabolic calculations. The algorithms used in the K4b² for the determination of ventilatory threshold have been implemented according to Beaver et al. method (3). 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 flow-meter of the analyzer

| TABLE 1. Subjects characteristics. |
|-------------------------------|----------------|----------------|--------|----------------|----------------|----------------|----------------|
| Subjects                       | Mean           | SD             | N      | Age (yr)       | 14.6           | 1.1            | 11               |
|                               | Height (cm)    | Weight (kg)    | Body Fat (%) | VO₂max (mL.min⁻¹.kg⁻¹) | 61.0           | 13.4           | 85.6             |
|                               |                |                |        | PVT (W)        | 263.4          | 7.3            | 60.1             |

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was performed with a 3-L syringe (Quinton Instruments, Seattle, WA).

**Measurements of blood lactate concentration.** Capillary blood samples were taken from the ear lobe using a Lactate Pro LT (Arkay Inc., Kyoto, Japan) (26). For the incremental test, samples were taken at rest, in the last 30 s of each stage while sustaining the work rate, and at 2 and 4 min after the end of the incremental test. For the P_{30} work rate, samples were taken at rest, 30 s before, and 5 min after the end of the P_{30} exercise test. For the P_{+10} work rate, samples were taken at rest, in the last 30-s of every 3 min, at exhaustion, and 2 and 4 min after the end of the P_{+10} exercise test.

**Anthropometry.** Height and weight were measured before and after each test. Five skinfold measurements were taken (triceps, biceps, suprailiac, subscapular, and mid-thigh) with percent body fat computed using the Durnin and Womersley’s formula (11).

**Raw Cardiac Data Processing**

Signal processing was performed with LARY_CR, INRIA product developed in the scientific environment SCICOS-SCILAB (22). All time series (RR, BF, and Vt) were resampled at 8 Hz by interpolation of a third order spline function to obtain equidistant data. The RR time series were then prefiltered by band-pass Finite Impulsionnel Response filters (FIR) corresponding to HF and LF frequency bands, in order to reduce noise and obtain a merely mono-component signal in each band. The LF band was set between 0.04 and 0.15 Hz as recommended by the task force (29). The HF band was set between 0.15 and 1.5 Hz, depending on the subjects BF. Frequency, time and time-frequency methods were then applied to the RR time series in the different conditions. For a first overall comparison of the HRV response below and above PVT work rates, a frequency method, the fast Fourier transform (FFT) was applied on the RR time series. This spectral analysis method, assuming the signal is stationary, provides averaged frequency domain parameters but neglects time-varying phenomenons (9). To assess the behavior overtime of the CR parameters with the above PVT work rate conditions, a time-frequency method, the Smoothed Pseudo Wigner-Ville Distribution (SPWVD), and a time method, the complex demodulation (CDM), were jointly used (22). These methods are briefly described below.

**Spectral Analysis**

**Fast Fourier transform (FFT).** The FFT was computed on 3-min RR time series. The energy has been computed by integration of the power spectral density (PSD) in the LF and HF range as follows (9,10,29):

\[
LF = \sum_{f=0.04}^{0.15} PSD \cdot \Delta f \quad \text{and} \quad HF = \sum_{f=0.15}^{1} PSD \cdot \Delta f
\]

[1]

The measure of the energy contained in LF and HF bands is usually expressed in spectral energy unit (i.e., ms^2; 9,10,20). However, the LF and HF components can be normalized (9,10,20) and expressed in percent of the total short-term spectral energy (LF + HF) as follows:

\[
\text{LFn} = 100 \cdot \text{LF/(LF + HF)} \quad \text{and} \quad \text{HFn} = 100 \cdot \text{HF/(LF + HF)}.
\] [2]

**Smoothed pseudo Wigner-Ville distribution (SPWVD) and complex demodulation (CDM).** SPWVD and CDM have been already used in cardiovascular applications (14,21,22,28). They provide an instantaneous and continuous assessment of spectral parameters, allowing the spectral changes to be followed over time. They use the analytic signal of the RR series, i.e., its real and imaginary parts provided by the Hilbert transform (22). Of particular interest were the instantaneous amplitudes of the HF and LF RR components (IAmp_HF_RR and IAmp_LF_RR), the frequencies of the HF component (IF_HF_RR), and the phases between the HF component and the respiratory signal (IPh_HF_CR).

The CDM provides a continuous evaluation of the amplitude, frequency, and phase. RR time series were first filtered around the central BF in order to extract the envelope and phase. Because it was not possible to access the raw respiratory signal, the real modulation of breathing was not provided (22). However, because the K4 gives the instantaneous BF, a more classical approach was adopted by fixing the central frequency in a narrow band corresponding to the BF variations for each subject (14). The IPh_{HF_CR} was obtained by multiplying the RR phase (arctan(imagRR/realRR)) with a complex oscillator at the central BF.

The SPWVD provides a continuous evaluation of the amplitude and frequency, giving nearly “instantaneous” complex FFT spectrum for each beat, with a high resolution achieved by independent time and frequency smoothing. In this study, a spectrum of 1024 frequency values was obtained every 0.125 s. Furthermore, the SPWVD provides two indices, related to the signal noise (Inoise) and to the spectrum dispersion (Idisp), respectively, validating the measurements of instantaneous parameters even in conditions of reduced variability such as in the P_{+10} period (22).

**Statistical Analysis**

Nonparametric Wilcoxon paired test was used to analyze:

1. The effect of work rate on HRV components by comparing RR, HF_{RR}, LF_{RR} in absolute and normalized values between P_{30} and P_{+10}.

2. The time effect on HRV and breathing components during heavy exercise by comparing RR, BF, Vt, \text{lamp}_{LF_{RR}}, \text{lamp}_{HF_{RR}}, IF_{HF_{RR}}, and the HR/BF ratio (number of cardiac beats in one respiratory cycle) between the beginning and the end of the P_{+10} exercise test. These components were averaged over 1 min at the beginning and at the end of the P_{+10} exercise test. A linear regression test was used to describe the continuous time evolution of the P_{+10} exercise test parameters.

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I\textsubscript{PH\_HF\_CR} does not require any statistical analysis since the occurrence (or not) of sudden shifts over several seconds is only of interest.

### RESULTS

#### Blood Lactate Concentration

Measures of blood lactate concentration during exercise confirmed that: \( P\_30 \) and \( P\_10 \) lactate levels (2.26 ± 1.65 vs 11.35 ± 3.83 mmol·L\(^{-1}\), \( P < 0.001 \)) corresponded to work rates below and above anaerobic threshold, respectively (4 mmol·L\(^{-1}\)).

#### Effect of Work Rate on HRV

**Effect of work rate on HRV components.** As expected, mean RR was significantly lower during \( P\_30 \) compared with \( P\_10 \) (316 ± 15 ms vs 508 ± 40 ms, \( P < 0.001 \), Table 2).

All HRV components expressed in absolute values (LF, HF, and LF+HF) were significantly higher during \( P\_30 \) compared with \( P\_10 \) (all \( P < 0.001 \), Table 2 and Fig. 1).

When comparing normalized HRV values, LFn was found to be significantly higher and HFn significantly lower in \( P\_30 \) compared with \( P\_10 \) (80 ± 10% vs 11 ± 8% for LFn and 20 ± 10% vs 89 ± 8% for HFn, respectively, \( P < 0.001 \), Fig. 2). In addition, the balance between LFn and HFn was reversed from \( P\_30 \) to \( P\_10 \). This balance is further illustrated in Figure 1 where the \( P\_30 \) spectrogram shows a prevalence of LF compared with HF, whereas in \( P\_10 \) the spectrogram shows a prevalence of HF energy with no or little LF. Furthermore, the LF/HF ratio was always above 1 for \( P\_30 \) and less than 1 for \( P\_10 \) (4.92 ± 2.4 and 0.14 ± 0.12, respectively, \( P < 0.001 \), Fig. 2).

**Effect of exercise duration on HRV components during heavy exercise (\( P\_10 \)).** Between the beginning and the end of the heavy exercise bout, mean RR interval significantly decreased from 342 ± 15 ms to 317 ± 16 ms (\( P < 0.01 \); Table 3), the instantaneous HF frequency (IF\_HF\_RR) significantly increased (from 0.70 ± 0.18 Hz to 0.93 ± 0.31 Hz, \( P < 0.01 \), Table 3) whereas instantaneous HF and LF amplitudes (I\textsubscript{Amp\_HF\_RR}; I\textsubscript{Amp\_LF\_RR}) remained constant (Fig. 3). Furthermore, linear regression showed that both mean RR and IF\_HF\_RR changes were strongly correlated with time between the beginning and the end of the \( P\_10 \) exercise test (\( R^2 = 0.86 ± 0.04 \) for mean RR and \( R^2 = 0.73 ± 0.20 \) for IF\_HF\_RR). However, I\textsubscript{Amp\_LF\_RR} and I\textsubscript{Amp\_HF\_RR} were not correlated with time between the beginning and the end of the \( P\_10 \) exercise test (\( R^2 = 0.16 ± 0.16 \) for I\textsubscript{Amp\_HF\_RR} and \( R^2 = 0.08 ± 0.10 \) for I\textsubscript{Amp\_LF\_RR}).

**Effect of exercise bout duration on breathing during heavy exercise (\( P\_10 \).** Figure 3 shows a time representation of cardiorespiratory components during the heavy exercise test (\( P\_10 \)) for one subject between the 350th and the 1105th second. From top to bottom, the cardiorespiratory components were RR interval; instantaneous HF RR (dashed line, IF\_HF\_RR); breathing frequency (continuous line, BF); tidal volume; instantaneous HF RR amplitude (I\textsubscript{Amp\_HF\_RR}); and instantaneous LF RR amplitude (I\textsubscript{Amp\_LF\_RR}). BF (continuous line) increased significantly from 0.70 ± 0.18 Hz to 0.93 ± 0.31 Hz (\( P < 0.01 \), Table 3) in synchrony with IF\_HF\_RR (dashed line). However, Vt remained constant (see Table 3 and Fig. 3) and was not correlated with time between the beginning and the end of the \( P\_10 \) exercise test (\( R^2 = 0.22 ± 0.26 \)).

**Dynamic cardiorespiratory interactions during heavy exercise (\( P\_10 \).** From the beginning to the end of the exercise test, the phase between HR and BF (I\textsubscript{PH\_HF\_CR}) and RR did not change significantly.
remained constant. This result is strengthened by the very low instantaneous noise (Inoise) and dispersion (Idisp) levels as shown in Figure 4. Note that IPh_HF<sub>CR</sub>, Inoise, and Idisp can only be interpreted between the 350th and the 1105th second, corresponding to the adapted frequency parameters. The HR/BF ratio significantly decreased from 4.47 ± 1.23 to 3.42 ± 0.72 heart beats by breathing cycle (P < 0.01; Table 3).

**DISCUSSION**

**Effect of exercise rate on HRV.** This study showed an expected decrease of the overall HRV spectral energy between sub- versus supra-ventilatory threshold work rate. However, the typical reversed distribution of normalized spectral energy in LF and HF range between the two exercise intensities is an evidence of a qualitative change in cardiac control. In healthy subjects, it is known that every energy component of HRV decreases when exercise intensity increases (1,10,20). It is now also recognized that the decrease in LF and HF energy is mainly linked to vagal withdrawal (12,20). This study confirmed these results with HRV in LF and HF range (LF+HF) significantly different between P<sub>-30</sub> and P<sub>+10</sub> (166 ± 120 ms<sup>2</sup> vs 24 ± 16 ms<sup>2</sup>, P < 0.001). During heavy exercise (P<sub>+10</sub>) autonomic control of HR seems to be less effective compared with moderate exercise conditions (P<sub>-30</sub>). These results are in agreement with Casadei and coworkers (6,7) who showed the persistence of HRV during exercise in humans after complete ganglion autonomic blockade, which inhibits the autonomic control of HR. The authors gave evidence for a

<table>
<thead>
<tr>
<th>Mean Cardiorespiratory Components</th>
<th>Beginning (first minute, P&lt;sub&gt;-30&lt;/sub&gt;)</th>
<th>End (last minute, P&lt;sub&gt;+10&lt;/sub&gt;)</th>
<th>P</th>
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<tbody>
<tr>
<td>RR (ms)</td>
<td>342 ± 15</td>
<td>317 ± 16</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BF and IF_HF&lt;sub&gt;RR&lt;/sub&gt; (Hz)</td>
<td>0.70 ± 0.18</td>
<td>0.93 ± 0.31</td>
<td>&lt;0.01</td>
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<tr>
<td>Vt (L)</td>
<td>2.33 ± 0.69</td>
<td>2.31 ± 0.73</td>
<td>NS</td>
</tr>
<tr>
<td>IAmp_HF&lt;sub&gt;RR&lt;/sub&gt; (ms&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>2.50 ± 0.77</td>
<td>2.80 ± 0.66</td>
<td>NS</td>
</tr>
<tr>
<td>IAmp_LF&lt;sub&gt;RR&lt;/sub&gt; (ms&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>0.45 ± 0.15</td>
<td>0.39 ± 0.10</td>
<td>NS</td>
</tr>
<tr>
<td>HR/BF (heart beats by breathing cycle)</td>
<td>4.47 ± 1.23</td>
<td>3.42 ± 0.72</td>
<td>&lt;0.01</td>
</tr>
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Beginning corresponds to the first minute and end to the last minute of exercise bout. Low frequency ranges from 0.04 to 0.15 Hz, whereas high frequency ranges from 0.15 to 1.5 Hz, NS (non significant) stands for P > 0.05.
negligible cardiac control of nonneural mechanisms at rest (1% of total HRV), gradually increasing and becoming prevalent with severe exercise. In addition, studies with heart transplant recipients showed a reappearance of HRV during exercise (1,4). Although the HF spectral energy in heart transplant recipients is significantly lower than in control at rest, there is no significant difference between heart transplant recipients and controls during exercise (1,4). Because the breathing effect on RR time series cannot be mediated by vagal nerve in heart transplant recipients, it has been postulated that the observed HF variability resulted from a mechanical effect of breathing to the sinus node. We hypothesize that the remaining HRV in P+10 condition was linked to nonneural mechanisms including the hyperventilation induced above ventilatory threshold. This hypothesis is confirmed by the results obtained with the normalized HRV values. A comparison of LFn and HFn between P.30 and P+10, showed reversed results with large prevalence of LF variability (80%) during moderate exercise compared with HF and large prevalence of HF variability (89%) compared with LF during heavy exercise. In summary, a major increase in breathing rate combined with the disappearance of LF compared with LF during heavy exercise conditions.

The LF/HF ratio is supposed to reflect sympathovagal balance (23). This concept has been widely disputed and remains controversial (12). However, LF/HF ratio has been used in this study because it was always higher than 1 for P.30 and always smaller than 1 for P+10. This gradual switch of LF/HF ratio could be correlated to the overstepping of the ventilatory threshold, and could provide a reliable index of the ventilatory threshold detection from HRV. However, more endurance exercise tests under metabolic steady state conditions are necessary to verify the usefulness of LF/HF ratio as a reliable index for the determination of the ventilatory threshold.

Dynamic cardiorespiratory interactions during heavy exercise. The present study confirms the progressive decrease in RR interval that is linked to the VO2 slow component (2) and the increase in BF with Vt remaining constant (8,13) if HRV was under autonomic control. The increase in BF with Vt remaining constant should have induced a concomitant decrease in the amplitude of HF (10,16,18), and yet the HF amplitude remained constant. This particular point is in agreement with the hypothesis of a nonneural mechanism that is not time dependent (not dependent on BF). It could be the mechanical effect of breathing on the sinus node (17) generating the residual HF variability during heavy exercise as mentioned above.

The last main result concerns the phase between RR HF and BF oscillations, which is consistent during exhaustive heavy exercise. This result implies that central cardiovascular and respiratory centers remain synchronous during heavy exercise. Conversely, in chronic heart failure patients, the cardiorespiratory phase is not constant (21). Hence, at the end of the exercise test, just before exhaustion, HR/BF ratio tended toward three beats per breathing cycle, whatever the initial level. Could this particular ratio be a limit value before exhaustion during heavy exercise?

Lastly, a limiting factor for the interpretation of the data is the small number of subjects (only nine males and two females). Based on the small number of subjects, an interpretation of physiologic responses may not reflect changes that may be gender specific.

CONCLUSION

This study showed the prevalence of HF in contrast to LF during heavy exercise (work intensity above PVT), whereas the opposite distribution was observed during moderate exercise (prevalence of LF compared with HF). Therefore, HRV analysis allows distinguishing between sub- from supra-ventilatory-threshold exercise. Determinants of the residual HRV observed at this intensity are in all probability of nonneural origin. Among the plausible determinants, a mechanical effect of breathing rate to the sinus node is very conceivable. Finally, exercise duration at supra-threshold intensity does not alter the cardiorespiratory synchronization as evidenced by a constant cardiorespiratory phase.

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