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The influence of exercise intensity on the power spectrum of heart rate variability

Renza Perini[1], Claudio Orizio[1], Giuseppe Baselli[2], Sergio Cerutti[3], and Arsenio Veicsteinas[1]

[1] Istituto di Fisiologia Umana, Dipartimento di Scienze Biomediche e Biotecnologie, Università di Brescia, via Valsabbina 19, I-25124 Brescia, Italy
[2] Dipartimento di Automazione Industriale, Università di Brescia, Brescia, Italy
[3] Centro di Teoria dei Sistemi, CNR, Dipartimento di Elettronica, Politecnico di Milano, Milan, Italy

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Summary. The power spectral analysis of R-R interval variability (RRV) has been estimated by means of an autoregressive method in seven sedentary males at rest, during steady-state cycle exercise at 21 percent maximal oxygen uptake (% $\dot{V}_{O_{2max}}$), SEM 2%, 49% $\dot{V}_{O_{2max}}$, SEM 2% and 70% $\dot{V}_{O_{2max}}$, SEM 2% and during recovery. The RRV, i.e. the absolute power of the spectrum, decreased 10, 100 and 500 times in the three exercise intensities, returning to resting value during recovery. In the RRV power spectrum three components have been identified: (1) high frequency peak (HF), central frequency about 0.24 Hz at rest and recovery, and 0.28 Hz, SEM 0.02, 0.37 Hz, SEM 0.03 and 0.48 Hz, SEM 0.06 during the three exercise intensities, respectively; (2) low frequency peak (LF), central frequency about 0.1 Hz independent of the metabolic state; (3) very low frequency component (VLF), <0.05 Hz, no peak observed. The HF peak power, as a percentage of the total power (HF%), averaged 16%, SEM 5% at rest and did not change during exercise, whereas during recovery it decreased to 5%–10%. The LF% and VLF% were about 50% and 35% at rest and during low exercise intensity, respectively. At higher intensities, LF% decreased to 16% and VLF% increased to 70%. During recovery a return to resting values occurred. The HF component may reflect the increased respiratory rate and the LF peak changes the resetting of the baroreceptor reflex with exercise. The hypothesis is made that VLF fluctuations in heart rate might be partially mediated by the sympathetic system.

Key words: Exertion – Heart rate control system – Power spectral analysis – Autonomic nervous system – Sympathetic system

Introduction

In resting conditions heart rate (f_c) shows regular fluctuations, which depend on oscillations in cardiovascular control systems (Hyndman et al. 1971; Sayers 1973; Akselrod et al. 1985).

The frequency content of f_c variability has been estimated by means of spectral analysis. Two peaks, corresponding to different rhythms in f_c variability, have been recognized. Agreement exists that one peak is centred at about 0.25 Hz and can be considered a quantitative estimation of the respiratory arrhythmia (Akselrod et al. 1981; Akselrod et al. 1985; Pomeranz et al. 1985). The amplitude of this peak has been suggested as an index of tonic vagal activity (Pomeranz et al. 1985; Pagani et al. 1986). The second peak has been recognized at a frequency of about 0.1 Hz and has been related to arterial pressure control mechanisms (Akselrod et al. 1981). An increase in sympathetic activity, as in the standing position (Pomeranz et al. 1985) or after passive tilting (Pagani et al. 1986), is associated with an increase in power in this band. The ratio between the power of the 0.1 Hz peak (low frequency, LF) and the power of the peak at respiratory frequency (high frequency, HF), i.e. LF:HF, has been suggested to be a useful index for estimating the sympatho-vagal balance (Pagani et al. 1986).

It is well known that during dynamic exercise f_c increases to a steady-state value because of both vagal withdrawal and augmented sympathetic activity. The relative role of the two drives depends on the exercise intensity (Robinson et al. 1966; Orizio et al. 1988). During recovery, a return to the pre-exercise level of activity in the two drives occurs within 5 to 8 min (Robinson et al. 1966).

Recently, a decrease in f_c variability and no correlation between LF power and sympathetic activity have been observed during muscular exercise (Arai et al. 1989). However, these authors have analysed data from cycle exercise with load increments during which the f_c was related linearly to the exercise intensity. Therefore, the analysis of f_c variability was necessarily performed on short periods of unsteady-state f_c values.

The absence in the literature of data on f_c variability during exercise at constant intensities, which is particularly suitable to the investigation of control mecha-

Offprint requests to: A. Veicsteinas

nisms (Wassermann et al. 1987), prompted the present study. The power spectral analysis of f_c variability was, therefore, performed during steady-state exercise at different intensities and during the corresponding recovery periods, with the purpose of estimating the influence of exercise intensity on power spectrum distribution of f_c variability.

Methods

Subjects. Seven sedentary males (mean age 23.7 years, SEM 0.8) volunteered for the study. The subjects were untrained medical students, familiar with laboratory tests and accustomed to the use of the cycle ergometer.

The maximal oxygen consumption ($\dot{V}_{O_{2max}}$) of each subject was measured by an open circuit method during cycle ergometer exercise by establishing a plateau of oxygen consumption ($\dot{V}_{O_2}$) with increasing exercise intensities (Åstrand and Rodahl 1986).

Exercise test. The subjects exercised on a cycle ergometer (Ergomedic 818 Monark, Sweden) at a pedal frequency of 50 rpm for 5 min. Exercise intensities were 50, 100 and 150 W (low, medium and high respectively). A series of trial runs was performed with the sole purpose of determining individual $\dot{V}_{O_2}$ during the last minute of exercise at each given intensity.

The actual experiments were carried out in the morning at the same time of day, in a room at constant temperature (20°–22° C). The sequence of exercise intensities was randomized.

With the subject seated on the cycle, the electrocardiographic signal was continuously recorded and observed on a monitor for 5 min at rest, during exercise and recovery. The occurrence of the R-wave in the ECG was detected by means of an electronic differentiator. A pulse coincident with the R-wave was fed on-line into a computer (AT-IBM, Armonk, USA) and f_c measurements were stored as the sequence of R-R intervals.

The f_c variability analysis. Off-line analysis of the data was performed on a DEC VAX-750 (Digital, Maynard, USA) minicomputer. The R-R interval variability (RRV) was estimated in each period of the task, i.e. at rest, during exercise and during recovery.

A linear trend was subtracted from the signal at rest to decrease the contribution of very slow random fluctuations. The initial transients of exercise and recovery, i.e. about 1 min, were discarded. In addition, the exponential trend was subtracted from the remaining signal. The series $\{t_i\}$, i.e. the R-R variability around the subtracted trend, was then obtained for each period. Variance of $\{t_i\}$ represents the total power of R-R variability and the root mean square of $\{t_i\}$ [rms(t_i)], quantifies the amplitude of f_c variability.

In order to compare the variability between the three periods and between the three exercise intensities, characterized by different mean R-R values, the R-R variability percentage ($RRV\%$) was calculated as:

$$RRV\% = \mathrm{rms}(t_i)/T \cdot 100$$

where T is the mean R-R value calculated before the detrend procedure.

The power spectral density of discrete series $\{t_i\}$ in each period was estimated by means of an autoregressive (AR) method, in which coefficients are estimated via the Levinson Durbin algorithm (Kay and Marple 1981). The validity of the model was checked by testing the whiteness of the prediction error (Anderson's test) (Kay and Marple 1981) and the choice of the optimal AR model order was performed by applying the Akaike Information criterion (Akaike 1970).

Individual spectral components were recognized automatically via a spectral decomposition procedure, which related each spectral component to a pair of conjugate poles in the AR model (Zetterberg 1978; Baselli et al. 1986). In order to classify the detected components, three bands were defined a priori: very low frequency (VLF), below 0.05 Hz; LF, between 0.05 and 0.15 Hz; HF, between 0.15 and 1.0 Hz. If two or more components were present in each band, the sum of their power and the mean frequency were considered as the power and the central frequency of that band, respectively.

Both absolute and normalized power, i.e. the percentage of the total power calculated as the sum of the power of the three components, and the central frequency of each component were calculated. The ratio between the power of the LF and the HF component was also calculated (LF:HF), as previously suggested by Pagani et al. (1986).

Data are presented as means and SEM. Variance analyses and t-tests were used to determine the significance of the differences. Differences were considered significant at $P<0.05$.

Results

The $\dot{V}_{O_{2max}}$ was 44.4 ml·min^{-1}·kg^{-1}, SEM 1.6. The oxygen demand at low, medium and high exercise intensity corresponded to 21.0% $\dot{V}_{O_{2max}}$, SEM 2.1%, 49.3% $\dot{V}_{O_{2max}}$, SEM 2.0% and 70.7% $\dot{V}_{O_{2max}}$, SEM 2.5%, respectively.

From an average resting value ($n=21$) of 812 ms, SEM 43, the R-R interval decreased to 614 ms, SEM 21, to 468 ms, SEM 9 and to 400 ms, SEM 11 during low, medium and high exercise intensity, respectively, and during the corresponding recovery was 818 ms, SEM 46, 682 ms, SEM 37 and 610 ms, SEM 24, respectively.

In Fig. 1 the mean RRV% is plotted as a function of relative exercise intensity. From a resting value of 8.9%, SEM 0.7, the RRV% decreased to 4.4%, SEM 0.4 at 50 W, to 2.2%, SEM 0.1 at 100 W and to 1.4%, SEM 0.1 at 150 W. During recovery from low and medium exercise, the RRV% returned to the pre-exercise value (8.8%, SEM 0.4 and 9.1%, SEM 0.7, respectively). After the high exercise intensity, however, it remained lower than the resting value (6.2%, SEM 0.7).

In Fig. 2 the power spectrum distribution of RRV at rest, during exercise and on recovery is shown for a representative subject. Note the different scales in the y axes, indicating that the total power of RRV decreases 10 times, 100 times and 500 times during the three exercise intensities in comparison to rest. At rest, three com-

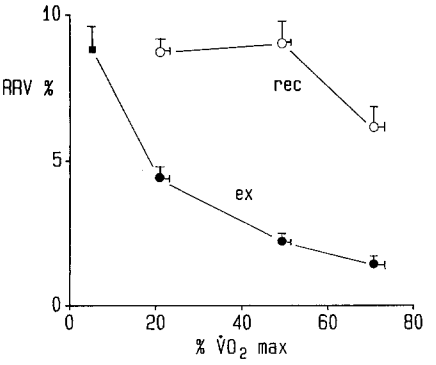

Fig. 1. Mean values and SEM of normalized R-R variability ($RRV\%$) as a function of relative exercise intensity (% $\dot{V}_{O_{2max}}$) during exercise (ex, ●) and recovery (rec, ○)

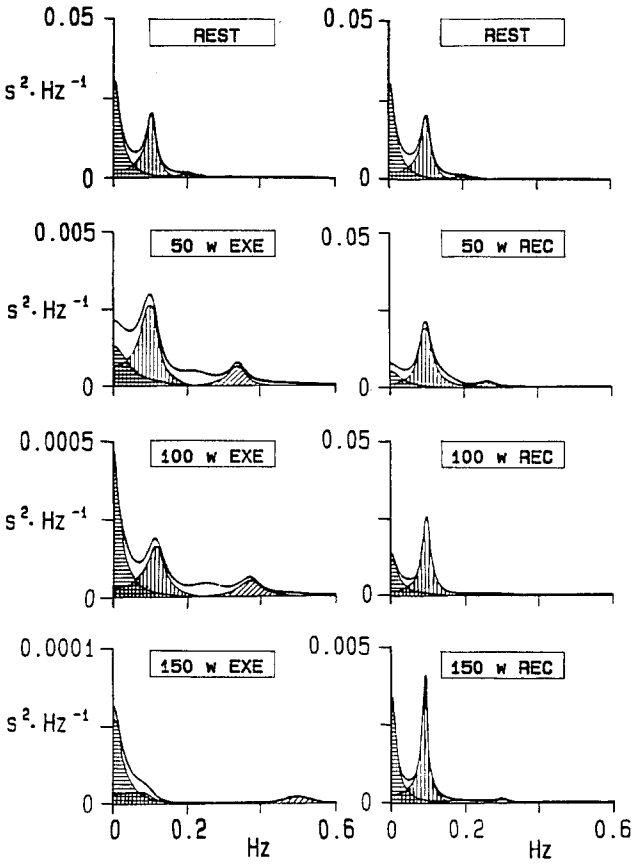

Fig. 2. Power spectrum distribution of R-R variability at *rest*, during the steady-state at three exercise (*EXE*) intensities and in the corresponding recovery (*REC*) period in a representative subject. Note the different scales on the *y axes*, decreasing with *EXE* intensity and increasing during *REC*. The dominant autoregressive spectral components are shown: *VLF* (≡), *LF* (IIIIII) and *HF* (////)

ponents can be recognized. An important portion of the total power is present below 0.05 Hz (VLF). However a peak cannot be recognized in this range. The second component is a high, sharp peak centred at 0.1 Hz (LF). Finally, a peak much lower in amplitude can be seen at about 0.20 Hz (HF).

Exercise caused some changes in the power spectrum distribution. The VLF component represents a substantial part of the spectrum at all exercise intensities. The LF peak is always recognizable in the spectrum, but is less sharp and lower as exercise intensity increases. The HF peak shifts towards the right as exercise intensity is increased. During recovery, independent of the exercise intensity, the power spectrum distribution is practically the same as at rest. The LF peak is particularly sharp however, especially after medium and high exercise intensity.

At rest, the central frequency of the VLF, LF and HF peaks had a mean value of 0.019 Hz, SEM 0.005, 0.10 Hz, SEM 0.004 and 0.24 Hz, SEM 0.007, respectively. During exercise, independent of the intensity, no VLF central frequency could be seen, and no statistical change was observed in the LF central frequency, being 0.099 Hz, SEM 0.005, 0.108 Hz, SEM 0.007 and

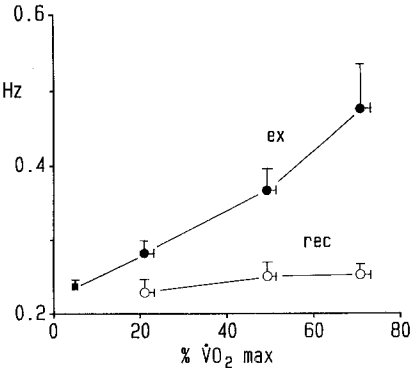

Fig. 3. Mean values and SEM of the central frequency of the high frequency peak as a function of relative exercise intensity (% $\dot{V}_{O_2max}$) during exercise (*ex*, ●) and recovery (*rec*, ○)

0.093 Hz, SEM 0.003 at low, medium and high intensity respectively. In Fig. 3 the mean value of the central frequency of the HF component is shown as a function of relative exercise intensity. It was 0.28 Hz, SEM 0.017 at 21% $\dot{V}_{O_{2max}}$; 0.37 Hz, SEM 0.029 at 49% $\dot{V}_{O_{2max}}$ and 0.48 Hz, SEM 0.058 at 71% $\dot{V}_{O_{2max}}$. During recovery, the central frequency of the HF component returned to pre-exercise values.

In Fig. 4 the average normalized power of the VLF, LF and HF components (VLF%, LF% and HF%) is plotted as a function of the percentage $\dot{V}_{O_{2max}}$ during exercise (top panel) and the corresponding recovery (bottom panel). At rest the dominant component is rep-

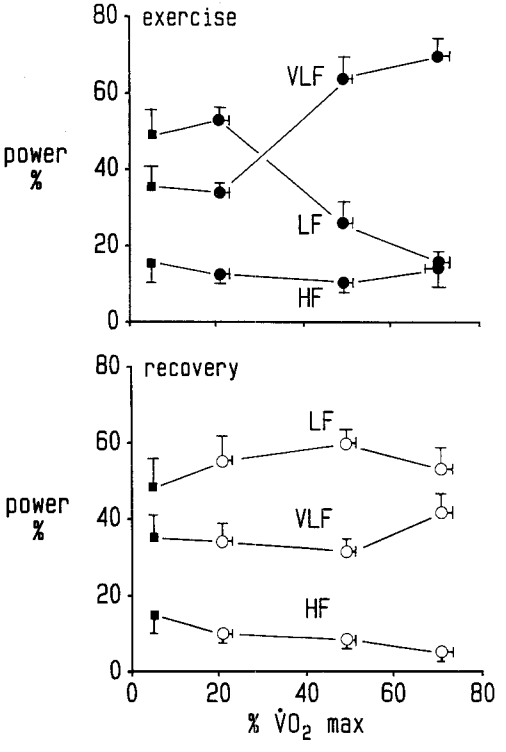

Fig. 4. Mean values and SEM of normalized power of very low frequency, low frequency and high frequency peak components (*VLF%*, *LF%*, *HF%*) as a function of relative exercise intensity (% $\dot{V}_{O_2}max$) during exercise and recovery

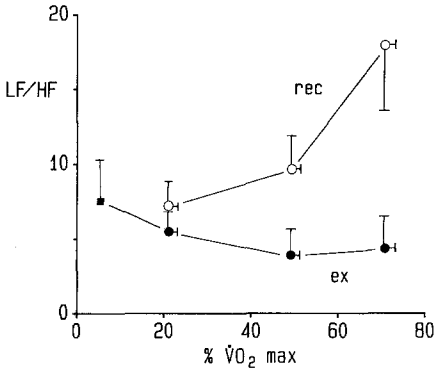

Fig. 5. The low frequency:high frequency peak (*LF/HF*) power ratio as a function of relative exercise intensity (% $\dot{V}_{O_2max}$) during exercise (*ex*, ●) and recovery (*rec*, ○)

resented by the LF peak, which is 48.9%, SEM 6.8% of the total power. The VLF component, on the other hand, is 35.5%, SEM 5.4% and the HF component 15.6%, SEM 5.2%.

At low intensity, no significant difference in the relative power of the components was found in comparison to rest. On the contrary, at higher exercise intensities a significant increase in the VLF component was observed, concomitant with a significant reduction in the LF peak. The relative power of the HF band did not change ($P > 0.05$).

During recovery the relative power distribution tended to return to pre-exercise values. The HF component, however, was about 40% lower than the rest value and the LF component tended to be higher, though not significantly.

The ratio LF:HF is plotted as a function of exercise intensity in Fig. 5. At rest it was 7.6 SEM 2.7. During exercise the ratio did not change significantly, but it was consistently lower than at rest, reflecting the marked reduction in LF peak power. During recovery the ratio returned to resting value, except following high exercise intensity when it increased about 2.5 times.

Discussion

Power spectral analysis of f_c variability has been proposed as a useful tool when evaluating autonomic control of the heart (Akselrod et al. 1981; Pomeranz et al. 1985).

In agreement with previous studies in man (Pomeranz et al. 1985; Pagani et al. 1986; Saul et al. 1988; Arai et al. 1989), we observed two main peaks in the RRV power spectrum at rest in the sitting position; one peak occurred at the respiratory frequency (HF) and a second one centred at 0.1 Hz (LF). The latter turned out to be the dominant component of the spectrum, reflecting the activity in the sympathetic system due to the orthostatic position. An increase in the LF band, in fact, has been observed after changing posture (Pomeranz et al. 1985) and after passive tilting (Pagani et al. 1986).

Even though a linear trend had been subtracted from the resting signal, in order to minimize the contribution of random fluctuations, about one-third of the total power was still found in the frequency range below 0.05 Hz (VLF). A similar power distribution in normal subjects has been described by Saul et al. (1988).

Dynamic exercise caused a marked decrease in the RRV, i.e. in the absolute power of the spectrum, as has been previously reported (Arai et al. 1989). In spite of a large difference in the absolute values of the R-R intervals the RRV% was found to decrease dramatically with increasing exercise intensity. During recovery RRV% returned to resting value, except after high exercise intensity (see Fig. 1).

Considering that the vagus affects the entire range of f_c fluctuations (Akselrod et al. 1981; Pomeranz et al. 1985), these findings reflect the reduction in vagal activity occurring during exercise and the restoration of vagal tone occurring at the end of the task (Robinson et al. 1966).

During exercise with increments in intensity up to levels of fatigue, no changes in the normalized values of LF and HF power with respect to rest were observed and no correlation between LF power and sympathetic activity was found (Arai et al. 1989). Our results, obtained from steady-state f_c values at constant intensity exercise, showed that only at low exercise intensity did no changes occur in the relative power of the three components with respect to rest. In contrast, at exercise intensities higher than about 30% $\dot{V}_{O_{2\,max}}$, a marked decrease in LF normalized power coupled to an increase in VLF% was found. The HF% remained unchanged.

The discrepancy between the data obtained by Arai et al. (1989) and by ourselves might be the result of the different experimental procedures used. Exercise with intensity increased in increments is characterized by a continuously increasing metabolic demand, which leads to continuously changing inputs to the cardiovascular centres. By contrast, during submaximal exercise of constant intensity steady-state conditions are reached (Wassermann et al. 1987). Therefore, due to the quite different control of f_c in these two situations, the data can scarcely be compared.

Moreover, Arai et al. (1989) performed the power spectral analysis on short periods (64 s), as f_c increased linearly with increasing exercise intensity. In contrast, we were able to analyse longer, relatively stable periods at each exercise intensity. This, coupled with the use of an AR method, should have improved the accuracy of spectral estimation, especially at very low frequencies (Kay and Marple 1981; Baselli et al. 1986).

In order to explain our findings, it is necessary to re-examine the mechanisms that are involved in f_c fluctuations. The HF peak has been considered a quantitative estimation of sinus arrhythmia (Pomeranz et al. 1985) which is affected by the respiratory rate and the tidal volume (Hirsch and Bishop 1981). With exercise, the HF central frequency shifted to higher values, being about double at high exercise intensity. This confirms that the HF peak reflected the respiratory rate, as observed in previous studies when respiration at a fixed

rate was imposed (Pomeranz et al. 1985; Pagani et al. 1986). The increase in ventilation and the rhythmicity of respiration occurring with exercise (Åstrand and Rodahl 1986) may be responsible for the maintenance of a constant normalized power in HF during exercise in comparison to rest.

It has been suggested that fluctuations in f_c at 0.1 Hz (LF) depend on the baroreceptor reflex activated by fluctuations in arterial pressure (Sayers 1973; Akselrod et al. 1981). To explain the origin of these fluctuations, a spontaneous rhythmic activity of vessel smooth muscle (Akselrod et al. 1985) and a rhythmic sympathetic discharge of central origin have both been hypothesized (Preiss and Polosa 1974; Gebber 1980). An involvement of the resonance properties of the feedback loops involved in the control of arterial pressure have also been suggested (Hyndman et al. 1971; Baselli et al. 1988). During exercise, one or more of these factors are changed. In fact, both somatic inputs from active muscles and the central command cause a bulbar resetting of the blood pressure control system (Korner 1971). In addition, peripheral resistance during exercise is regulated differently to that at rest. In particular, local metabolic control of muscular vessels occurs (Åstrand and Rodahl 1986).

Because of all these changes occurring during exercise, one would expect large modifications in LF rhythm. During low exercise the LF peak was less sharp than at rest, even if its normalized power did not change (Fig. 2). At an exercise intensity between 30% $\dot{V}_{O_2max}$ and 70% $\dot{V}_{O_2max}$, in addition to a further enlargement of the peak, a marked decrease in LF normalized power was found. Thus our results seem to confirm the hypothesis that changes in LF rhythm occur with exercise and that they are dependent on exercise intensity.

It is interest to note that 30% $\dot{V}_{O_2max}$ represents a threshold in the cardiovascular adjustment to exercise. In fact, at this exercise intensity increases in the activity of sympathetic (Galbo 1983; Orizio et al. 1988) and renin-angiotensin systems occur (Galbo 1983), indicating that additional mechanisms are involved in cardiovascular adjustment.

The observed decrease in the LF peak while increasing exercise intensity is coupled with an increment in the relative power of the VLF component, which represents the major part of the remaining RRV at the highest exercise intensities. While attempting to attribute a physiological meaning to this spectral component, it has to be considered that it is indeed impossible to eliminate completely the constant trend in the signal. This fact affects the estimation of the power in the VLF band and is of particular significance when the total power of the spectrum is much reduced, as during exercise at high intensity.

Saul et al. (1988), comparing at rest normal subjects and patients with congestive heart failure, suggested that the fluctuations in f_c below 0.04 Hz are related to slow respiratory fluctuations, probably being mediated by the sympathetic system. During exercise at medium and high intensities, the enhanced sympathetic tone coupled with the reduced vagal activity and the changes in respiratory pattern might, therefore, justify the persistance of the VLF component. It has to be taken into account, however, that f_c and in turn its fluctuations may also be affected by inputs from suprabulbar areas to the cardiovascular centre (Korner 1971) and humoral agents (Oberg 1976).

During recovery, after the initial fast f_c transient elapsed, we observed a power distribution similar to that during the pre-exercise period. These findings suggest that the mechanisms controlling the cardiovascular system and modified by exercise have returned to resting conditions within 5 min. After exercise at medium and high intensities, however, the tendency for HF% to be lower than at rest and for the LF peak to be particularly sharp may reflect the slow return of sympathetic activity to resting values, as previously observed, using a different approach, after an exercise intensity higher than about 30% $\dot{V}_{O_2max}$ by Perini et al. (1989).

In conclusion, our results are consistent with the literature that the power spectral analysis of f_c variability estimates the sympatho-vagal balance at rest and during recovery. During exercise this does not hold true, as proven by the negative correlation between the LF power and the exercise intensity beyond 30% $\dot{V}_{O_2max}$. Moreover, our results indicate that a not negligible portion of the power of RRV is in the very low frequency band and that this component might reflect, at least in part, the sympathetic activity. Finally, in the power spectrum distribution of f_c variability the entire control of the cardiovascular system can be discerned.

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