Autonomic adaptations to intensive and overload training periods: a laboratory study

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ABSTRACT

PICHOT, V., T. BUSSO, F. ROCHE, M. GARET, F. COSTES, D. DUVERNEY, J. R. LACOUR, and J. C. BARTHÉLÉMY. Autonomic adaptations to intensive and overload training periods: a laboratory study. Med. Sci. Sports Exerc., Vol. 34, No. 10, pp. 1660–1666, 2002. Purpose: Looking for practical and reliable markers of fatigue is of particular interest in elite sports. One possible marker might be the autonomic nervous system activity, known to be well affected by physical exercise and that can be easily assessed by heart rate variability. Methods: We designed a laboratory study to follow six sedentary subjects (32.7 ± 5.0 yr) going successively through 2 months of intensive physical training and 1 month of overload training on cycloergometer followed by 2 wk of recovery. Maximal power output over 5 min (Plim5'), VO2peak and standard indices of heart rate variability were monitored all along the protocol. Results: During the intensive training period, physical performance increased significantly (VO2peak: +20.2%, P < 0.01; Plim5': +26.4%, P < 0.0001) as well as most of the indices of heart rate variability (mean RR, Ptot, HF, rMSSD, pNN50, SDNNIDX, SDNN, all P < 0.05) with a significant shift in the autonomic nervous system toward a predominance of its parasympathetic arm (LF/HF, LFnu, HFnu, P < 0.01). During the overload training period, there was a stagnation of the parasympathetic indices associated to a progressive increase in sympathetic activity (LF/HF, P < 0.05). During the week of recovery, there was a sudden significant rebound of the parasympathetic activity (mean RR, HF, pNN50, rMSSD, SDNN, all P < 0.05). After 7 wk of recovery, all heart rate variability indices tended to return to the prestudy values. Conclusion: Autonomic nervous system status depends on cumulated physical fatigue due to increased training loads. Therefore, heart rate variability analysis appears to be an appropriate tool to monitor the effects of physical training loads on performance and fitness, and could eventually be used to prevent overtraining states. Key Words: AUTONOMIC NERVOUS SYSTEM, HEART RATE VARIABILITY, FATIGUE, OVER-REACHING

The analysis of heart rate variability is an efficient tool to evaluate the autonomic nervous activity and its sympatovagal balance (1,24). This tool has gained increasing popularity these last few years in the field of exercise (2,6,8,9,12,15,18,21–23,27,31).

A physical exercise bout determines instantaneous modifications of the autonomic nervous system activity. Heart rate variability progressively decreases all along an incremental exercise bout, leading to a progressive shift of the sympatovagal balance where the sympathetic activity takes over the progressively withdrawing parasympathetic activity (2,31). The recovery of the initial heart rate variability level after an exercise session needs a few minutes up to 24 h, depending on the intensity of the physical exercise (7,9).

Furthermore, regular physical training induces a long-term increase in heart rate variability resulting from changes in the autonomic nervous system basal activity and balance. One of the proposed explanations is a higher parasympathetic tone, which can be evidenced by an increase of the high-frequency peak of heart rate variability (6,12,23). Indeed, endurance athletes have a lower resting heart rate compared with sedentary people (6,23). In addition, a linear relationship between VO2peak and the parasympathetic indices of heart rate variability has been shown (12). Although these transversal studies have demonstrated differences in heart rate variability between athletes and sedentary subjects, longitudinal studies have however hardly shown any significant modification in heart rate variability after a training program despite a significant increase of VO2peak (10,17,18).

Training programs of elite athletes are usually built with the repetition of training cycles composed of high training load periods followed by shorter resting periods (7,29). The training periods induce fatigue states (over-reaching) (4,13) followed by an increase of the physical capacity after the recovery period (supercompensation) (7,21). The lack of an appropriate recovery period and/or the accumulation of too
intensive training periods can result in an overtraining syndrome characterized by an advanced fatigue state, which can seriously compromise the competitive season of the athlete (13,14). Overtraining induces a long-lasting imbalance in autonomic nervous system activity (13,15,27), which is probably the excessive form of the previously described adaptation. In overtrained subjects, this imbalance is characterized by a dramatic predominance of the parasympathetic or the sympathetic activity, depending on the type of overtraining (13,15).

Thus, the prevention of overtraining syndrome remains a priority in intensive training in elite athletes (13). Many markers have already been proposed to assess fatigue in athletes (3,14,25,26,30) to avoid the emergence of an overtraining syndrome. The resting heart rate still remains the most used noninvasive index. However, the magnitude of variations of this parameter is tight and quite difficult to appreciate due to multiple interacting factors (14). In a previous study, we described the decrease of heart rate variability associated to physical fatigue during a training cycle of national-level, middle-distance runners (21). This field study led to the idea that heart rate variability indices could be used to monitor training and prevent overtraining states.

The goal of the present study was to assess the potential use of heart rate variability analysis as a biomarker in the control of the impact of successive increasing training loads on fitness and performance. Thus, we designed a laboratory protocol with sedentary subjects who underwent a first period of intensive training immediately followed by an overload training period and, finally, a recovery period. Training loads and physical performances of the subjects were all precisely quantified and correlated to the evolution of the nocturnal heart rate variability all along the intensive training period, the overload training period, and the recovery period.

MATERIAL AND METHODS

Subjects

Six sedentary men were included in the study (age 32.7 ± 5.0 y, weight 83.5 ± 12.6 kg, and height 1.82 ± 0.08 m). They were free of any known cardiac abnormalities, and none of them were on any cardiovascular medications. They were all volunteers and provided written informed consent. The protocol was approved by the local IRB.

Experimental Protocol

A schematic representation of the protocol is presented Figure 1. Further explanation and description of the training protocol can be found in the paper of Busso et al. (4).

The experimental protocol was composed of five successive periods. A reference nonactive period, a height-week intensive training period, a 1-wk recovery transition period, a 4-wk overload training period, and a 2-wk recovery period.

Reference period (W0). Two weeks before starting training, maximal oxygen consumption (VO_2peak) was measured in all subjects with a stepwise incremental maximal cycle ergometer test (Monark Model 818, Stockholm, Sweden). After collecting the data in a resting state, the subjects started pedaling at 100 or 150 W for 5 min, depending on their initial estimated level. The power was then incremented every 2 min by 20 or 30 W until they reached exhaustion. Expired gases were collected in a polyethylene bag (HP Production, Saint-Etienne, France) during the last 30 s of each 2-min period. The gas composition was measured with a paramagnetic analyzer for O_2 (Servomex Series 1440, Crowborough, UK) and an infrared analyzer for CO_2 (Datex Normocap, Helsinki, Finland). The VO_2peak corresponded to the value measured during the last step just before the subjects reached exhaustion. On another day, the maximal power that the subjects could maintain during 5 min (Plim5') on a cycloergometer (Monark Model 829E) was measured. This test constituted the performance level of the subjects and was reevaluated all along the protocol. Also, on a different day, the heart rate variability indices were calculated using a 24-h Holter recorder to establish the basal autonomic nervous system status.

Intensive training period (W1 to W8). The training period covered 8 successive weeks. The subjects performed three training sessions per week (from Monday to Friday), followed by 2 d of rest (Saturday and Sunday). Each training session included four successive steps: 1) 10-min warm-up, cycling at a low intensity; 2) a maximal effort test, Plim5'; 3) 15-min low-intensity cycling recovery; and 4) four bouts of 5-min cycling at 85% of Plim5', each separated by a 3-min active recovery period, cycling at a low intensity. A Holter recording was performed twice a week, on Thursday night, after a training session, and on Sunday night after the 2 d of rest. We calculated the heart rate variability indices for each recording.

Transition recovery period (W9). During the week of transition, 2 d were dedicated separately to a Plim5' test and a VO_2peak test. On another day, a Holter recording was also performed.

Overload training period (W10 to W13). The transition period was followed by a 4-wk overload training period in which the weekly training workload was multiplied by a factor of 5/3 compared with the previous intensive training period. On Monday, Wednesday, and Friday: the

FIGURE 1—Description of the training protocol. HRV, heart rate variability measurement; VO_2, maximal oxygen consumption test.
same training sessions than during the intensive training period. Tuesday and Thursday: 10 min of low-intensity cycling warm-up, five bouts of 5 min at 85% of Plim5′ each separated by 3 min of low-intensity cycling active recovery. Saturday and Sunday: rest. Two Holter recordings were made each week during the 4 wk, one recording beginning on Thursday evening and one beginning on Sunday evening, after the 2 d of rest.

**Recovery period (W14, W15).** During the 2 wk after the 4 wk of overload, the subjects reduced their physical exercise volume to exercise tests only. During the first week, they performed three Plim5′ tests on separated days, and during the second week, they performed two Plim5′ tests and a VO2peak test on different days as well. A Holter recording was performed during week 14 (W14) on a different day from the tests, and an additional recording was performed 7 wk later, after the end of the overload period (W21).

**Heart Rate Variability Analysis**

The heart rate variability was measured with 24 h Holter monitoring (Stratascan 563, Del Mar, Irvine, CA). Each RR interval was validated before the analysis. Then, we performed the heart rate variability analysis over the night periods to avoid variations originated by daily environmental factors. The mean RR interval, time domain indices and the Fourier transform indices of heart rate variability were standardized as previously described in the literature (24).

These indices of heart rate variability are assumed to represent the autonomic nervous system activity. The particular variables issued from the time domain analysis represent the parasympathetic activity (the percentage of differences between adjacent normal RR intervals more than 50 ms: PNN50, the square root of the mean of the sum of the squared differences between adjacent normal RR intervals: RMSSD) and the sympathetic activity (the standard deviation of all normal RR intervals: SDNN, the standard deviations of the mean of all normal RR intervals for 5-min segments: SDANN, and the mean of the standard deviation of all normal RR intervals for all 5-min segments: SDNNIDX) (24). In the frequency analysis, the total power of the spectrum (Ptot) indicates the global autonomic nervous system status; the VLF is notably an index of the activity; the low frequency expressed in absolute (LF) and in normalized value (LFnu = 100.LF/(Ptot - VLF)) represents the parasympathetic arm simultaneously to an autonomic nervous system equilibrium toward a predominance of the parasympathetic arm; and the high frequency expressed in absolute (HF) and in normalized value (HFnu = 100.HF/(Ptot - VLF)) represents the vagal vous system status; the VLF is notably an index of the regulation of the renin-angiotensin system, the thermoregulation, and parasympathetic activity; the high frequency of the spectrum expressed in absolute (HF) and in normalized value (HFnu = 100.HF/(Ptot - VLF)) contains both sympathetic and parasympathetic activities; and the LF/HF ratio represents the autonomic nervous system balance (1,24). Nocturnal mean heart rate (HR) was also calculated all along the study.

**Statistical Analysis**

The data were calculated and analyzed with the software MatLab 5 (Math Work Inc.®,) and SStview 4.5 (SAS Institute®,) on a Macintosh G3. The variables were compared using repeated ANOVA measures, and, when significant, further analysis was performed using paired t-test. P-value was taken as significant when equal or less than 0.05. This test was chosen to better fit the practical interest of the measure, i.e., significant differences between following weeks.

**RESULTS**

**Intensive training period.** The mean value of the VO2peak for the six subjects increased significantly from +20.2% during the 2-month training period (Table I). Similarly, the Plim5′ demonstrated a significant increase of +26.4% (Table I).

The progressive modifications of the mean RR and of the indices of heart rate variability are illustrated in Figures 2 and 3. The 2-month intensive training period demonstrated an increase in the mean nocturnal RR from 0.92 ± 0.13 s to 1.08 ± 0.11 s, which represents a decrease in the mean nocturnal heart rate from 66.2 ± 8.8 bpm to 56.5 ± 5.6 bpm (P < 0.001) (Fig. 4). The time domain analysis of heart rate variability showed a significant increase of the pNN50, rmSSD, and SDNNIDX indices during the training period (from 10.0 ± 12.3 to 18.3 ± 8.4%, from 40.8 ± 26.7 to 68.3 ± 18.9 ms, and from 62.7 ± 18.8 to 92.7 ± 21.1 ms, respectively; all P < 0.001). The Ptot, HF, and HFnu indices of the frequency analysis increased significantly (from 3390 ± 1046 to 5115 ± 2084 ms².Hz⁻¹, from 386 ± 368 to 747 ± 465 ms².Hz⁻¹, and from 30.8 ± 15.6 to 36.7 ± 11.9%), respectively; all P < 0.05), whereas the LF/HF ratio and LFnu decreased (from 4.0 ± 2.1 to 2.4 ± 1.1, and from 64.6 ± 15.3 to 58.4 ± 11.6%, respectively; both P < 0.01).

Globally, there was a progressive increase of both the parasympathetic and sympathetic indices with a shift of the autonomic nervous system equilibrium toward a predominance of the parasympathetic arm simultaneously to an increase of the aerobic performance during the 2-month intensive training period.

**Overload training period and the after recovery period.** The mean nocturnal RR did not show further increase during the overload period (from 1.08 ± 0.11 to 1.03 ± 0.15 s, NS), and demonstrated a significant further in-
increase during the week of in comparison with W13 (from $1.03 \pm 0.15$ to $1.11 \pm 0.15$, $P < 0.05$), corresponding to a concomitant decrease of $-4.1$ bpm in heart rate (Figs. 2 and 4).

There was a statistical stagnation with a clear visual tendency to decrease for most of the parasympathetic indices of heart rate variability ($\text{pNN50}$, $\text{mSSD}$, $\text{SDNN}$, HF; all NS) during the overload period, followed by a significant increase of these indices during the recovery period (from $16.9 \pm 8.4$ to $19.7 \pm 11.1$%, from $60.3 \pm 24.6$ to $76.9 \pm 40.2$ ms, from $111.9 \pm 29.5$ to $125.9 \pm 36.0$ ms, and from $732 \pm 475$ to $798 \pm 629$ ms$^2$/Hz$^{-1}$), respectively; all $P < 0.05$) (Figs. 2 and 3). The LF/HF ratio representing the sympathovagal balance significantly increased during the overload period (from $2.4 \pm 1.4$ to $2.7 \pm 1.4$; $P < 0.05$) and significantly decreased during the week of recovery (from $2.7 \pm 1.4$ to $2.4 \pm 1.4$; $P < 0.05$) (Figs. 2 and 3). Globally, the autonomic nervous system balance shifted toward a predominance of its sympathetic arm during the overload period and bounced back toward a predominance of its parasympathetic arm during the recovery week.

We can already notice that most indices demonstrated their maximal variation during the third week of overload training. As a matter of fact, the fourth week did not follow exactly the trend of the first 3 wk of overload training, constituting an eventual transitory period.

**Measurements 7 wk after the end of the protocol.** When measured 7 wk after the end of the protocol, the nocturnal mean RR and heart rate variability were at an intermediate value between the basal and maximal values reached in the previous weeks. They still remained significantly different from the basal values for most of them (HR, RR, $\text{pNN50}$, $\text{mSSD}$, $\text{SDNN}$, $\text{VLF}$, $\text{HF}$, $\text{LFn}$, $\text{HFnm}$, and LF/HF; all $P < 0.05$) though. Especially, the LF/HF ratio, $\text{LFnm}$, and $\text{HFnm}$, representative of the sympathovagal balance, were still close to the values obtained during the week of recovery, where the maximal rebound level was reached by the subjects.
The subjects demonstrated a significant progressive increase of heart rate variability and a significant decrease of the mean resting heart rate all along the 2 months of intensive training. These modifications corresponded to a progressive significant increase of the parasympathetic drive of the autonomic nervous system. These results are different from previous studies suggesting that aerobic training does not increase heart rate variability (10,17,18). Only Seals and Chase (22) have found a moderate increase of the standard deviation of heart rate after a training program lasting 30 wk. As the training durations in these studies were similar or longer than ours, the intensity of training, which was higher in our study, appears to be the parameter inducing the modifications in the autonomic regulation. Usually, the training loads used in similar protocols are set between 40 and 80% of VO₂peak measured at baseline. In our study, the training load was around 90% of VO₂peak, and was repeatedly reevaluated each week to keep up with that intensity level to determine a progressive adapted increase of the training load for each subject. Thus, this longitudinal study confirms the parallel increase of parasympathetic activity and VO₂peak, that was only suggested by transversal studies (12,18,23). Moreover, it strengthens the importance of the participation of the parasympathetic drive in the bradycardia observed in subjects with high aerobic abilities as well (6,23).

During the overload training, the heart rate variability analysis demonstrated a nonsignificant decrease of the global autonomic nervous system tone with an imbalance toward a predominance of sympathetic activity. This was counterbalanced by a rebound in the parasympathetic activity during the week of recovery. In a recent study concerning elite middle-distance runners, we also evidenced a progressive decrease of the parasympathetic activity during 3 wk of over-reaching, a decrease that was greater than in the present study and that was also followed by a rebound of this parameter during the resting week (21). The more intensive training workload might explain the more pronounced decrease of heart rate variability during the over-reaching period. In the present study, in spite of the high training load, the subjects did not fall into an overtraining state that could impair their physical capacities for many weeks or months. Nevertheless, they presented mild symptoms of nervousness as well as mood state or sleeping disturbances that disappeared in the days after the end of the overload period. These evolutions were followed by a rebound in heart rate variability, reflecting an over-reaching state. This affirmation is reinforced by the fact that the calculated time to recover on the last week of the overload period was only 3.6 d (4). After greater workloads, as in elite athletes, time to recover from an over-reaching period may vary from 8 to 23 d (5,19,20).

The results concerning the fourth week of overload training remain to be discussed because some heart rate variability indices started to be reversed. Several explanations can be proposed. During the fourth week, the first measurement of heart rate variability was made on Friday, after the last overload training session, and the second one was made...
on Sunday, after 2 d of recovery. We expected to observe a further decrease in heart rate variability. However, the subjects were ending 4 months of a stressful heavy protocol, and we can suppose that they felt a strong relief at that moment, and, consequently, the sleep quality may have been improved as well as the heart rate variability (28). One explanation might therefore be a feeling of premature entrance in recovery. In a previous study (21), the results obtained with the middle-distance runners did not show such an interruption in heart rate variability decrease. The context was different though because these athletes were following an annual training program composed of monthly repeated training cycles.

Seven weeks after the last training session, the heart rate variability indices of the autonomic nervous system tended to return to their basal values while still at an intermediate level. Importantly, the autonomic nervous system balance as assessed by the LF/HF ratio still showed a strong predominance of the parasympathetic drive. In such a population, the effects of training, as measured by autonomic nervous system indices, were still significant 7 wk after the end of the protocol. These results may have potential impact on the short-term effects of training programs.

Interestingly, our protocol confirmed some of the main aspects of aerobic training and generally accepted training principles (15,29), namely supercompensation based on profound transient spontaneously reversible cycles of homeostasis disequilibrium and added quantification. In the first period of intensive training, there was a quick and large increase of heart rate variability and $\overline{V}O_2$peak. During the second period, the subjects were already pretrained, and the large increase in their training load, assessed to induce a transient decrease in heart rate variability, appears to be the drive for the following supercompensation period. These data, added to the study concerning the elite athletes (21), suggest that the higher the autonomic nervous system activity, the more one will be able to shift the homeostasis regulation and, consequently, induce a secondary rebound in physical performance. Studies on overtraining are difficult to manage as all measurements need to be repeated at least once a week during the athletic season. For that reason, previous studies mainly focused on the description of specific periods before or during overtraining as well as on short longitudinal periods of programmed overtraining (11,14,16,21,27).

Heart rate variability analysis overcomes these practical difficulties because they are easy to use, therefore allowing an easy conduction of long-term prospective studies. Indeed, the analysis of the autonomic nervous system activity, which seems to be a crucial parameter in over-reaching and overtraining syndrome (13,15,21,27), could be helpful in finding optimal target values to optimize over-reaching as well as to assess threshold values beyond which there would be a risk of overtraining. Moreover, such an analysis could determine whether the parasympathetic and sympathetic types of overtraining are two different forms of overtraining or are two consecutive stages of overtraining.

In conclusion, this study has clearly demonstrated that a short period of 2 months of intensive training is able to increase both heart rate variability and $\overline{V}O_2$peak in sedentary subjects. These results underline important modifications of the autonomic nervous system global activity and equilibrium. Also, this study firms up the previous results concerning the interaction between an overload training period and an autonomic nervous system activity rebound. This could be helpful to better understand the physiological basis of overtraining states and, thus, be used for its prevention. Prospective studies have now to be done in top level athletes to determine whether it is possible to find heart rate variability thresholds between normal training fatigue and overtraining fatigue. Such parameters should prove to be efficient tools—noninvasive and easily used in free living situation—to follow up autonomic nervous system activity, and thus physical fitness all along the training periods. Further applications could include the monitoring of physical fatigue at the workplace, the benefits of rehabilitation programs in patients, as well as the effects of leisure activities or physical active lifestyles.

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