

Relation between heart rate variability and training load in middle-distance runners

VINCENT PICHOT, FRÉDÉRIC ROCHE, JEAN-MICHEL GASPOZ, FRANCK ENJOLRAS, ANESTIS ANTONIADIS, PASCAL MININI, FRÉDÉRIC COSTES, THIERRY BUSSO, JEAN-RENÉ LACOUR, and JEAN CLAUDE BARTHÉLÉMY

Laboratoire de Physiologie, Université de Saint-Etienne, FRANCE 42055; Département de Médecine Interne, Hôpitaux Universitaires de Genève, SWITZERLAND 1211; Département de Statistiques, Université Joseph Fourier, Grenoble, FRANCE 38041; and Laboratoire de Physiologie, Université Lyon I, Lyon, FRANCE 69921

ABSTRACT

V. PICHOT, F. ROCHE, J. M. GASPOZ, F. ENJOLRAS, A. ANTONIADIS, P. MININI, F. COSTES, T. BUSSO, J. R. LACOUR, and J. C. BARTHÉLÉMY. Relation between heart rate variability and training load in middle-distance runners. *Med. Sci. Sports Exerc.*, Vol. 32, No. 10, pp. 1729–1736, 2000. **Purpose:** Monitoring physical performance is of major importance in competitive sports. Indices commonly used, like resting heart rate, $\dot{V}O_{2\max}$, and hormones, cannot be easily used because of difficulties in routine use, of variations too small to be reliable, or of technical challenges in acquiring the data. **Methods:** We chose to assess autonomic nervous system activity using heart rate variability in seven middle-distance runners, aged 24.6 ± 4.8 yr, during their usual training cycle composed of 3 wk of heavy training periods, followed by a relative resting week. The electrocardiogram was recorded overnight twice a week and temporal and frequency indices of heart rate variability, using Fourier and Wavelet transforms, were calculated. Daily training loads and fatigue sensations were estimated with a questionnaire. Similar recordings were performed in a sedentary control group. **Results:** The results demonstrated a significant and progressive decrease in parasympathetic indices of up to -41% ($P < 0.05$) during the 3 wk of heavy training, followed by a significant increase during the relative resting week of up to $+46\%$ ($P < 0.05$). The indices of sympathetic activity followed the opposite trend, first up to $+31\%$ and then -24% ($P < 0.05$), respectively. The percentage increasing mean nocturnal heart rate variation remained below 12% ($P < 0.05$). There was no significant variation in the control group. **Conclusion:** This study confirmed that heavy training shifted the cardiac autonomic balance toward a predominance of the sympathetic over the parasympathetic drive. When recorded during the night, heart rate variability appeared to be a better tool than resting heart rate to evaluate cumulated physical fatigue, as it magnified the induced changes in autonomic nervous system activity. These results could be of interest for optimizing individual training profiles. **Key Words:** AUTONOMIC NERVOUS SYSTEM, FOURIER TRANSFORM, WAVELET TRANSFORM, FATIGUE

In competitive sports, improved performance is often effected by alternating prolonged periods of intensive training and shorter periods with relative rest to avoid an overtraining state. Insights into physiological adaptations to exercise have been used to assess, predict, and improve the level of physical performance, as well as to prevent overtraining. Indeed, long-term training has been found to reduce contrasting results: improvement, as well as depression of physical performance. To optimize training profiles, including training intensity and duration, several variables have been monitored. Relationships between physical activity and different hormonal levels have been established, such as testosterone (36), luteinizing hormone (5), adrenocorticotrophic hormone (23), growth hormone (35), serotonin (41), and other biological parameters such as iron (2).

Variations in mean heart rate and in catecholamine levels observed during changes in performance have suggested a strong interaction with the autonomic nervous system (9,17,18,20,23–25,39). Among its representative parameters, resting heart rate has been promoted as a marker for fatigue, an increase being associated with increased physical activity and a decrease with recovery (9,18). However, although sometimes reported as significant, changes in resting heart rate are somewhat limited. The differences only reach a few beats per minute, which can be statistically significant but of limited practical value due to their dependence from numerous environmental factors. These changes depend on the autonomic nervous system equilibrium; thus, other representative parameters of autonomic tone, such as heart rate variability, should also reflect physical fatigue. Short-term decreases in heart rate variability after physical exercise have been well documented and shown to last at least 24 h (12,14). However, the long-term effects of physical training on heart rate variability have been questioned; in some studies, heart rate variability of trained subjects was shown not to differ from that of untrained subjects (7,26,31); these long-term effects of training have been analyzed

comparing subjects before and after training protocols (3,26,32). Only one cross-sectional study has demonstrated a positive correlation between exercise capacity assessed by maximal oxygen consumption and heart rate variability (19).

Careful monitoring of physical fatigue could provide two immediate interests: first, improving our knowledge of fatigue by giving insights into its underlying mechanisms, and, second, identifying information that could directly be applied to improve exercise training. Thus, we designed a prospective longitudinal study to correlate heart rate variability, training load, and physical fatigue during a full standard training cycle composed, as commonly used in such programs, of consecutive high-intensity training and resting periods. We chose to perform the analysis of the autonomic nervous system using time and frequency analysis of heart rate variability (30,33), which is noninvasive with good reproducibility (15,16,21,33).

MATERIALS AND METHODS

Subjects. Seven male middle-distance runners, aged 24.6 ± 4.8 yr (mean weight 65.2 ± 4.0 kg, mean height 1.79 ± 0.05 m) were included in the study. They had been practicing this sport activity for more than 3 yr and were ranked at the national level in France. A control group was composed of eight healthy sedentary male students (mean age 20.9 ± 0.8 yr, mean weight 72.7 ± 7.1 kg, and mean height 1.80 ± 0.03 m); they did not practice any physical training activity. All subjects were free of any known cardiac abnormalities, and none of them were on any cardioactive medications. They were all volunteers and provided informed consent. The protocol was approved by the local IRB.

Training protocol. The training program of the athletes was composed of training cycles of 4 wk duration with the first 3 wk (W1, W2, and W3) consisting of exhaustive training sessions, and the fourth week (W4) of light training sessions only, allowing recovery. During the 3 wk of intensive training, they performed 6–10 training sessions a week. They were asked to avoid coffee and alcohol intake the day preceding the recordings.

We monitored the athletes during a whole 4-wk cycle. Each athlete had his electrocardiogram recorded twice a week using a 24-h electrocardiogram ambulatory Holter device (Del Mar, Irvine, CA). Each week, one of the two recordings began 1 h after an intensive training session and the other 1 h after an extensive training session. The weekly results were then calculated as the average of these two recordings.

Training load and fatigue scoring. We chose to monitor the daily and weekly training loads in a simple way by analyzing the training program and the fatigue sensation of the athletes. To evaluate the weekly training load, we defined four different types of training sessions, to which a score was given, according to training intensity. First, daily training load scores were based on the activity chart recorded each day by each athlete, with the help of his coach: 0 for resting periods; 1 for endurance training; 2 for sprint, muscular, or extensive training sessions; and 4 for maximal

exhaustive training session, namely intensive training. Then, weekly training loads were calculated as the sum of the daily scores.

The subjects were asked to fill a questionnaire in which they estimated their feeling of fatigue after each training session, on a daily basis, plotting it on a visual scale between 0 and 10, 0 corresponding to the lack of fatigue sensation and 10 to the maximal fatigue sensation. Then, the sum of the scores was calculated for each week, giving a representative of the impact of the amount of work of each period on fatigue.

Control group. The ECGs of the control subjects were recorded twice a week using the ambulatory Holter system. They were asked to avoid intensive physical activity during the 4-wk recording period and to avoid coffee and alcohol intake the day preceding the recordings. They also listed their daily activity on a questionnaire.

ECG data analysis. The electrocardiographic Holter system (Stratascan 563, Del Mar) allowed us to extract the RR intervals list with a precision of 0.008 s. Each RR interval was validated before analysis. We chose to analyze only the night periods to avoid variations that could be introduced by differences in physical activity or by the environment. The beginning and the end of the sleeping periods were read from the activity questionnaires and verified from the 24-h RR intervals plots.

Mean heart rate and time domain analysis. On each recording, we calculated on a continuous 4-h period, between 0:00 and 4:00 a.m., the mean heart rate (beats per minute, bpm) and the following indices of heart rate variability: the percentage of differences between adjacent normal RR intervals more than 50 ms (PNN50); the standard deviation of all normal RR intervals (SDNN); the square root of the mean of the sum of the squared differences between adjacent normal RR intervals (RMSSD); 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).

Fourier analysis. The fast Fourier transform indices were calculated on sets of 256 consecutive RR intervals during the night periods. The power spectrum indices were calculated as recommended by the task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (33). The high-frequency peak of the spectrum (HF, 0.15–0.40 Hz) is known to represent parasympathetic activity; the low frequency (0.04–0.15 Hz) represents both parasympathetic and sympathetic activities. Additional calculations included: the very low frequency power (0 to 0.04 Hz); the ratio LF/HF, which represents an evaluation of the autonomic nervous system balance (sympathetic/parasympathetic); the normalized low and high frequency power (LFnu and HFnu) as $100 \cdot \text{LF} / (\text{total power} - \text{VLF})$ and $100 \cdot \text{HF} / (\text{total power} - \text{VLF})$, respectively; and the total frequency power (Ptot).

Wavelet analysis. Unlike Fourier, Wavelet transform is devoted to the analysis of nonstationary signals (1,13,30). Thus, there is no prerequisite regarding the stability of the

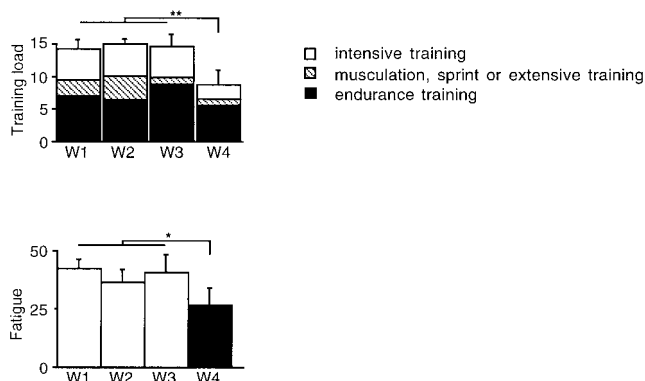


Figure 1—Training load quantification of different types of trainings (upper part) and fatigue sensation indices of the athletes (lower part), plotted against the 4-wk training cycle. The three first bars (W1, W2, and W3) represent the results obtained during the first 3 wk of heavy training period; the last bar (W4) represents the results obtained during the relative resting period.

frequency content along the signal analyzed. This analysis is devoted to the extraction of characteristic frequencies, contained along a signal which, in this case, was composed by consecutive intervals between RR interval series.

The decomposition of a signal by Wavelet transform requires a Ψ function adequately regular and localized, named Mother function. Starting from this initial function, a family of functions is built by dilatation and translocation, which constitutes the so-called Wavelet frame.

The analysis amounts to sliding a window of different weights (corresponding to different levels) containing the Wavelet function, all along the signal. The calculation gives a serial list of coefficients named Wavelet coefficients, which represent the evolution of the correlation between the signal f and the chosen Wavelet at different levels of analysis (or different ranges of frequencies) all along the signal f (29).

In our analysis, we used the Daubechies 4 Wavelet transform. For each record, the Wavelet coefficients were calculated on sets of 256 RR intervals, giving seven separate levels of analysis named 2, 4, 8, . . . 128. Then, we calculated the variability power, level by level, as the sum of squares of the coefficients. Thus, we obtained, for each recordings, the variability power for each level.

The sum of Wavelet power coefficients at levels 2, 4, and 8 ($HF_{Wavelet}$), approximately corresponds to the Fourier high frequencies (an index of parasympathetic activity); Wavelet power coefficients at levels 16 and 32 ($LF_{Wavelet}$) roughly corresponds to the Fourier low frequencies; Wavelet power coefficients at levels 64 and 128 ($VLF_{Wavelet}$) to the Fourier

very low frequencies; and the ratio $LF_{Wavelet}/HF_{Wavelet}$ to the Fourier ratio. The low and high frequencies indices can also be calculated in normalized units, as it is described in the Fourier analysis. The total frequency power was calculated as well ($P_{totWavelet}$).

Statistical analysis. Comparisons were conducted for each group, in two different ways: first a comparison was established on raw values of the different variables. Then, to eliminate the dispersion of the results due to different individual basal values at week 1, we used that first week as the 100% reference value. In that case, all parameters during the second (W2), third (W3), and fourth (W4) weeks were estimated with respect to the 100% reference values.

The mathematical analyses were performed on a Power-Macintosh using MatLab®, Statview®, and Super-ANOVA®. Data were compared using a two-factor ANOVA, weeks and subjects. Results were presented as means \pm standard deviations (mean \pm SD); they were considered as significant when P value was less than 0.05.

RESULTS

Athlete Group

Training load and fatigue. The weekly training load (Fig. 1, top) was relatively constant during the first 3 wk (all $P = NS$) with a mean of 14.80 ± 3.07 . The 40% decrease in the training load monitored during the last week (score: 8.80 ± 4.81) was statistically significant when compared with the first 3 wk ($P < 0.01$).

The scoring of subjective physical fatigue made by the runners on the visual scale, according to their own sensation (Fig. 1, bottom), reached a significant statistical difference between the first 3 wk and the last week ($P < 0.05$). There was a 32% decrease in the mean scores from the first 3 wk to the last week (39.53 ± 9.18 and 26.60 ± 12.89 , respectively).

Heart rate. The mean raw nocturnal heart rate progressively increased from W1 to W3, and the difference (3.74 bpm) reached statistical significance at W3; there was a significant decrease (5.85 bpm) in this parameter during the resting week (W4) as compared with W3, and a nonsignificant decrease as compared with W1 and W2 (Table 1). The relative values (Table 2) followed the same trend. A progressive increase of 9% from W1 to W3 ($P < 0.05$) and a 11% decrease from W3 to W4 ($P < 0.05$).

Time domain analysis. Time domain indices of heart rate variability showed a progressive decrease during the first 3 wk, followed by a marked increase at W4. When compared in terms of raw values (Table 1), none of the

TABLE 1. Raw values of time domain analysis on nocturnal periods for the athletes.

		Week 1 (Mean \pm SD)	Week 2 (Mean \pm SD)	Week 3 (Mean \pm SD)	Week 4 (Mean \pm SD)	
HR	bpm	46.39 \pm 4.88	48.26 \pm 6.78	50.13 \pm 6.99	44.28 \pm 5.12	*, †, ***
PNN50	%	26.83 \pm 6.79	22.54 \pm 6.20	20.38 \pm 6.61	26.34 \pm 4.83	
SDNN	ms	167.8 \pm 51.5	155.2 \pm 33.2	148.5 \pm 43.5	190.0 \pm 67.7	††
RMSSD	ms	22.10 \pm 22.33	13.65 \pm 12.28	13.65 \pm 17.44	32.76 \pm 49.95	
SDANN	ms	74.01 \pm 23.51	75.45 \pm 18.92	68.38 \pm 25.05	84.55 \pm 30.08	
SDNNIDX	ms	138.6 \pm 46.3	119.0 \pm 35.0	116.8 \pm 37.8	152.7 \pm 61.7	††

* $P < 0.05$ between week 1 and 3; *** $P < 0.001$ between week 3 and 4; † $P < 0.05$ between week 2 and 4; †† $P < 0.05$ between week 3 and 4.

TABLE 2. Relative values of time domain analysis on nocturnal periods for the athletes.

	Week 1 (Mean \pm SD)	Week 2 (Mean \pm SD)	Week 3 (Mean \pm SD)	Week 4 (Mean \pm SD)	
HR	100.00 \pm 0.00	103.58 \pm 12.01	109.05 \pm 7.90	97.95 \pm 6.70	*, **
PNN50	100.00 \pm 0.00	85.41 \pm 31.96	74.93 \pm 25.68	98.98 \pm 18.70	*, †
SDNN	100.00 \pm 0.00	100.33 \pm 20.14	89.89 \pm 20.75	108.12 \pm 18.93	
RMSSD	100.00 \pm 0.00	82.98 \pm 46.21	60.62 \pm 31.29	120.17 \pm 74.99	†
SDANN	100.00 \pm 0.00	115.37 \pm 69.90	98.57 \pm 51.54	109.94 \pm 26.75	
SDNNIDX	100.00 \pm 0.00	93.64 \pm 17.69	85.70 \pm 17.59	105.03 \pm 19.21	†

* $P < 0.05$ between week 1 and 3; ** $P < 0.01$ between week 3 and 4; † $P < 0.05$ between week 3 and 4.

decreases from W1 to W3 reached statistical significance; the increase observed at W4, compared with W3, was significant for SDNN and SDNNIDX (both $P < 0.05$). When compared in terms of relative values (Table 2), only PNN50 demonstrated a significant decrease at W3 (-25% , $P < 0.05$); the increase observed at W4, compared with W3, was significant for PNN50, RMSSD, and SDNNIDX ($+24\%$, $+59\%$ and $+19\%$, respectively; all $P < 0.05$).

Fourier Analysis

Raw values. Analyzed as raw values, two indices of heart rate variability demonstrated a significant increase from W3 to W4 (Table 3), Ptot, and VLF (both $P < 0.05$), whereas the increase of LF was significant between W2 and W4 ($P < 0.05$). Interestingly enough, LFnu and HFnu did not show any significant change.

Relative values (Fig. 2). When compared with the W1 values expressed as 100% reference values, statistically significant variations were observed for the parameters representative of parasympathetic drive (HF and HFnu), which demonstrated a significant decrease from W1 to W3 (-41.06% , $P < 0.05$ and -23.55% , $P < 0.01$; respectively). From W3 to W4, the HF and HFnu showed a significant increase ($+46.52$ and $+19.99$; respectively, both $P < 0.05$). The sympathetic LF index did not show any variation across the 4 wk; when expressed in normalized units, LFnu demonstrated a significant increase from W1 to W3 ($+31.56$, $P < 0.05$) and a decrease from W3 to W4 (-24.53 , NS). The relative LF/HF ratio, representative of the sympathico-vagal balance, showed a significant increase from W1 to W3 ($+75.29$, $P < 0.05$), followed by a significant decrease from W3 to W4 (-67.31 , $P < 0.05$).

Wavelet Analysis

Raw values (Table 4). The total power of Wavelet analysis (Ptot_{Wavelet}), demonstrated a consistent decrease from W1 to W3 (NS) and an increase from W3 to W4 ($P < 0.05$). The LF_{Wavelet} and the HF_{Wavelet} showed a nonsignif-

icant decrease from W1 to W3, followed by a significant increase from W3 to W4 ($P < 0.05$).

Relative values (Fig. 3). Relative values demonstrated a significant decrease in the total power of the heart rate variability, from W1 to W3 (-26.83% , $P < 0.05$), followed by a significant increase from W3 to W4 ($+36.23\%$, $P < 0.01$). The indices representing parasympathetic drive (HF_{Wavelet} and HFnu_{Wavelet}) showed a significant decrease from W1 to W3 (-34.69% , $P < 0.05$ and -11.35% , $P < 0.001$; respectively). From W3 to W4, the HF_{Wavelet} and HFnu_{Wavelet} demonstrated an increase ($+41.22\%$, $P < 0.05$ and $+7.13$, NS; respectively). The sympathetic LF index showed a nonsignificant decrease from W1 to W3 (-13.49 , NS), followed by a significant increase from W3 to W4 ($+22.55\%$, $P < 0.05$). When expressed in normalized units, the LFnu_{Wavelet} demonstrated a significant increase from W1 to W2 ($+17.75\%$, $P < 0.05$) and from W1 to W3 ($+23.87\%$, $P < 0.01$), followed by a significant decrease from W3 to W4 (-19.69% , $P < 0.05$). The relative LF/HF_{Wavelet} ratio, representative of the sympathico-vagal balance, showed a significant increase from W1 to W3 ($+43.85\%$, $P < 0.01$), followed by a nonsignificant decrease from W3 to W4 (-31.56% , NS).

Relationship between fatigue, training loads, and heart rate variability. None of the indices of the heart rate variability were significantly correlated with the estimated training loads and the fatigue scores.

Control group

Their results are shown in Tables 5, 6, and 7. None of the heart rate variability indices, either expressed in raw or in relative values, showed any significant variation.

DISCUSSION

Our data confirm the impressive autonomic changes that occur with 3 wk of intensive physical training, consisting in a global and progressive decrease in heart rate variability

TABLE 3. Raw values of HRV Fourier analysis on nocturnal periods for the athletes.

		Week 1 (Mean \pm SD)	Week 2 (Mean \pm SD)	Week 3 (Mean \pm SD)	Week 4 (Mean \pm SD)	
Ptot	s ²	0.0101 \pm 0.0057	0.0078 \pm 0.0050	0.0078 \pm 0.0044	0.011 \pm 0.0083	*
VLF	s ²	0.0051 \pm 0.0024	0.0043 \pm 0.0021	0.0042 \pm 0.0020	0.0054 \pm 0.0022	*
LF	s ²	0.0022 \pm 0.0017	0.0016 \pm 0.0011	0.0019 \pm 0.0013	0.0027 \pm 0.0023	†
HF	s ²	0.0026 \pm 0.0028	0.0018 \pm 0.0026	0.0016 \pm 0.0021	0.0034 \pm 0.0051	
ratio		1.36 \pm 1.07	1.28 \pm 0.69	1.78 \pm 0.99	1.45 \pm 0.91	
LFnu		49.79 \pm 18.22	51.70 \pm 12.81	58.50 \pm 13.42	53.75 \pm 15.64	
HFnu		48.44 \pm 16.94	45.51 \pm 11.52	39.12 \pm 12.79	45.44 \pm 15.30	

* $P < 0.05$ between week 3 and 4; † $P < 0.05$ between week 2 and 4.

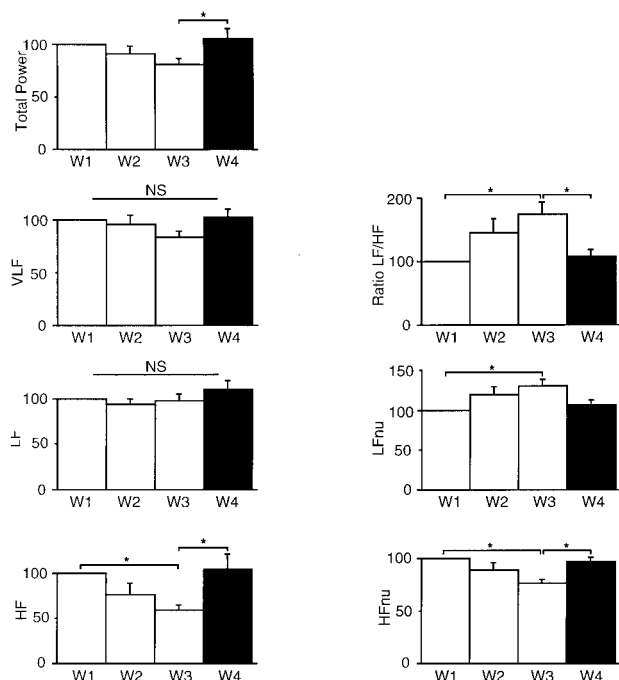


Figure 2—Relative indices of Fourier analysis of heart rate variability on the night periods during the 4-wk training cycle. The three first white bars (W1, W2, and W3) represent the results obtained during the first 3 wk of heavy training period; the black bar (W4) represents the results obtained during the relative resting period. Week 1 has been taken as the 100% reference value for each subject and each variable.

and in a tendency toward a progressively lower parasympathetic and higher sympathetic drives. Conversely, during the recovery week an abrupt compensation, mainly marked by a dramatic increase in global heart rate variability, associated with a relative increase in the parasympathetic drive and with a decrease in the sympathetic drive was observed.

The decrease in resting morning heart rate, which also reflects autonomic nervous system equilibrium, has largely been recognized as an index of physical fatigue (17,22); however, some authors have not confirmed this relation (8,38). The main problem with this variable lies in its relatively low amplitude of variation, in addition to its dependence on numerous other factors during its measurement, mainly mental stress and, more globally, any environmental condition. In contrast, the advantages of heart rate variability indices lie in their magnifying variations in autonomic nervous system activity, which makes them more usable and more reliable. Furthermore, night recordings, as already suggested in previous studies (18,23), enhance the reliability of the measurements by ensuring some indepen-

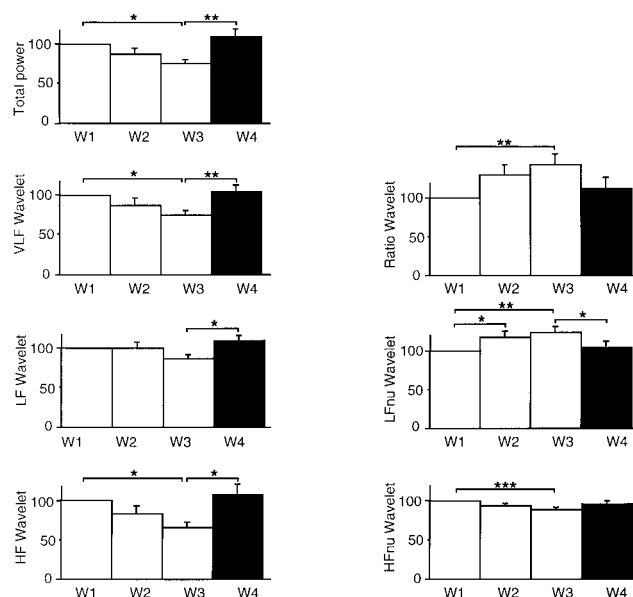


Figure 3—Relative indices of Wavelet analysis of heart rate variability on the night periods during the 4-wk training cycle. The three first white bars (W1, W2, and W3) represent the results obtained during the first 3 wk of heavy training period; the black bar (W4) represents the results obtained during the relative resting period. Week 1 has been taken as the 100% reference value for each subject and each variable.

dence from environmental factors and allows better discrimination of the changes in autonomic nervous system equilibrium. In our study, the mean night heart rate, one of the indices of fatigue most frequently and most easily used by the athletes, showed a variation of only 5–6 bpm between the heavy training period and the resting period, which, although significant, represented only ~10% increase. This could explain why Verde et al. (39) described a similar phenomenon as the one we observed in measuring mean heart rate, without reaching statistical significance. At the same time, some parameters measured by frequency analysis of heart rate variability demonstrated up to 40% variation. Thus, the analysis of heart rate variability using Fourier or Wavelet transform gave a larger scale, which should be less prone to measurement errors than the mean heart rate. The stability of these measurements from day to day in normal subjects has been demonstrated (15,16,21,33). It is of note that, when comparing weekly data, Wavelet transform indices gave an even slightly better separator power than Fourier transform indices. In addition, a better separation of heart rate variability status between consecutive weeks was observed when using values normalized to the

TABLE 4. Raw values of HRV Wavelet analysis on nocturnal periods for the athletes.

		Week 1 (Mean ± SD)	Week 2 (Mean ± SD)	Week 3 (Mean ± SD)	Week 4 (Mean ± SD)	
Total power	s ²	6.83 ± 4.25	5.11 ± 3.43	5.07 ± 3.35	8.36 ± 6.60	*, †
VLF _{Wavelet}	s ²	1.64 ± 0.81	1.39 ± 0.80	1.30 ± 0.67	1.75 ± 0.80	
LF _{Wavelet}	s ²	1.79 ± 1.00	1.46 ± 0.78	1.43 ± 0.77	2.02 ± 0.99	*, †
HF _{Wavelet}	s ²	3.40 ± 3.15	2.27 ± 2.39	2.33 ± 2.48	4.59 ± 5.65	*, †
Ratio _{Wavelet}		0.71 ± 0.35	0.81 ± 0.31	0.84 ± 0.28	0.77 ± 0.38	
LfnU _{Wavelet}		39.29 ± 12.15	43.23 ± 9.90	44.26 ± 10.44	40.80 ± 14.63	
HfnU _{Wavelet}		60.71 ± 12.15	56.77 ± 9.90	55.74 ± 10.44	59.20 ± 14.63	

* $P < 0.05$ between week 3 and 4; † $P < 0.05$ between week 2 and 4.

TABLE 5. Raw values of time domain analysis on nocturnal periods for the control group.

		Week 1 (Mean \pm SD)	Week 2 (Mean \pm SD)	Week 3 (Mean \pm SD)	Week 4 (Mean \pm SD)	
HR	bpm	56.35 \pm 6.69	54.34 \pm 5.55	54.94 \pm 5.58	56.54 \pm 4.98	NS
PNN50	%	19.89 \pm 9.58	20.94 \pm 9.89	19.46 \pm 9.06	19.70 \pm 10.06	NS
SDNN	ms	123.0 \pm 33.3	133.6 \pm 37.9	123.0 \pm 25.5	125.5 \pm 32.89	NS
RMSSD	ms	74.85 \pm 33.33	79.35 \pm 34.08	73.16 \pm 27.49	77.07 \pm 36.63	NS
SDANN	ms	58.66 \pm 20.10	63.23 \pm 19.73	56.15 \pm 15.19	56.70 \pm 16.82	NS
SDNNIDX	ms	100.5 \pm 27.7	107.6 \pm 33.0	99.8 \pm 25.0	104.1 \pm 30.0	NS

first recording of each subject considered as the 100% reference value (Fig. 2 and 3); this implies that each subject had his own autonomic nervous system equilibrium level in absolute values but demonstrates a highly similar percentage of variation when compared with others, given a cumulated training load.

The significance of these changes was reinforced by the stability of the heart rate variability indices of the control group over a 4-wk observation period. In addition, the reproducibility of heart rate variability analysis has been previously established (15,16,21), which emphasizes the fact that our observations are unlikely to be due to chance.

Uusitalo et al. (37) have also found an increase in the resting LF in endurance athletes at the end of a 6- to 9-wk overtraining protocol, but not during 6–9 wk of normal training. Their study showed an increase in sympathetic tone when athletes reached an overtrained state, although their supine resting heart rate did not change. They concluded that decreased heart rate variability could be an indicator of impending fatigue. Our protocol confirms this tendency for the sympatho-vagal balance, although our subjects did not follow a specific overtraining protocol but their usual intensive training program. However, a limitation of our study resides in the fact that we did not evaluate the performance of the athletes by an extra physical exercise, because we were not allowed to interfere with their regular training cycle. Nevertheless, the 4-wk training program was designed to allow them to reach an overreached state. As a matter of fact, the maximal difference in mean resting heart rate that we observed, between the resting week and the third week of training, reached only 6 bpm and none of the subjects manifested overtraining symptoms (Table 1). Conversely, Dressendorfer et al. (9) observed an increase of 10 bpm after a 20 d overtraining program. As heart rate differences are small, and as heart rate variability seems to give more contrast to them, it could be that the measurement introduced by heart rate variability indices, applied to training protocols, could, better than heart rate, reveal threshold values that athletes should not cross to avoid reaching an overtrained state. However, we cannot hypothesize the evo-

lution of the sympatho-vagal balance if the training had been prolonged at its highest intensity beyond 3 wk; more particularly, we don't know if we would have reached an overtrained state (17,22). From the foregoing, it would seem to be important that the reference week have a relatively standard training load during future studies.

The delay between the measurement of the autonomic nervous system variables and the last bout of exercise is of importance in the assessment of cardiac autonomic nervous system activity, because it is well established that heart rate variability is decreased following an exhaustive exercise (12). The recordings were performed, twice a week, after a training session, and averaged so as to obtain a representative value that could be comparable from week to week. Because there was a continuous decrease in heart rate variability over the 3 wk of heavy training, the changes that we observed were not only dependent from the training load of the day preceding the night recording, but they obviously also reflected a cumulative effect of fatigue all along the training cycle. The changes in heart rate variability could hardly be attributed to changes in training load, as its intensity did not vary during the first 3 wk (Fig. 1). On the whole, the interpretation of changes in heart rate variability, or of any other measurement of autonomic nervous system activity, should always take in account previous physical exercise sessions, performed the last day as well as the last weeks, and also make comparisons with basal resting values.

Different backgrounds in cyclical variations in training intensity should determine different autonomic nervous system adaptations. At the time of our protocol, the level of training of each athlete was already very high, as they had been practicing this activity for many years and were close to the beginning of the competition season. Conversely to middle-distance runners, marathon runners usually have a lower training intensity, as well as a much more regular training load throughout the year. This would probably result in less pronounced contrast in heart rate variability from one week to another. In older subjects, it appears from the limited data published, where measurements were per-

TABLE 6. Raw values of HRV Fourier analysis on nocturnal periods for the control group.

		Week 1 (Mean \pm SD)	Week 2 (Mean \pm SD)	Week 3 (Mean \pm SD)	Week 4 (Mean \pm SD)	
Ptot	s ²	0.0059 \pm 0.0026	0.0070 \pm 0.0033	0.0061 \pm 0.0026	0.0064 \pm 0.0034	NS
VLF	s ²	0.0037 \pm 0.0016	0.0045 \pm 0.0022	0.0039 \pm 0.0018	0.0039 \pm 0.0020	NS
LF	s ²	0.0013 \pm 0.0006	0.0013 \pm 0.0006	0.0013 \pm 0.0006	0.0014 \pm 0.0007	NS
HF	s ²	0.0009 \pm 0.0006	0.0011 \pm 0.0007	0.0009 \pm 0.0005	0.0010 \pm 0.0008	NS
Ratio		2.06 \pm 1.37	1.76 \pm 0.98	1.89 \pm 0.92	2.10 \pm 1.36	NS
LFnu		60.81 \pm 12.11	58.69 \pm 11.84	60.90 \pm 9.69	61.29 \pm 12.74	NS
HFnu		36.57 \pm 12.11	39.54 \pm 12.25	36.41 \pm 9.68	36.39 \pm 12.98	NS

TABLE 7. Raw values of HRV_{wavelet} analysis on nocturnal periods for the control group.

		Week 1 (Mean ± SD)	Week 2 (Mean ± SD)	Week 3 (Mean ± SD)	Week 4 (Mean ± SD)	
Total power	S ²	3.24 ± 1.67	3.85 ± 2.02	3.44 ± 1.75	3.31 ± 1.85	NS
VLF _{wavelet}	S ²	1.07 ± 0.53	1.27 ± 0.67	1.15 ± 0.63	1.03 ± 0.47	NS
LF _{wavelet}	S ²	1.01 ± 0.56	1.28 ± 0.71	1.15 ± 0.58	1.04 ± 0.53	NS
HF _{wavelet}	S ²	1.16 ± 0.75	1.30 ± 0.80	1.14 ± 0.69	1.24 ± 0.89	NS
Ratio _{wavelet}		1.03 ± 0.33	1.09 ± 0.36	1.11 ± 0.38	1.03 ± 0.29	NS
LFnu _{wavelet}		49.25 ± 9.62	50.95 ± 8.42	51.91 ± 9.51	48.96 ± 6.85	NS
HFnu _{wavelet}		50.75 ± 9.62	49.05 ± 8.42	48.09 ± 9.51	51.04 ± 6.85	NS

formed only once before and after the training protocol, that a continuous training load could yield similar results, with a global increase of heart rate variability and a predominant increase of the parasympathetic arm (28,32,34). This leads to our hypothesis that, in young subjects, repeating consecutive high- and low-intensity training periods could result in a progressive cumulated increase in, mainly, parasympathetic activity, which has been shown to be directly correlated to higher $\dot{V}O_{2max}$ values (19).

Many studies have dealt with the ability of other physiological indices to assess physical fatigue. However, the measurements were often technically difficult to obtain and the results were frequently controversial. Serum lactate level after exercise (10,11) was shown to be dependent on the dilution volume, the variations of its clearance, the time of blood sampling, and the measurement techniques, which lead to questionable and inconstant results. In the same manner, $\dot{V}O_{2max}$ was also found to be an ineffective index of fatigue (6,20,27). A mathematical modeling of the variations of physical performance with training to estimate a fatigue level using alterations of iron cycle parameters in runners (2) and hormonal concentrations in weightlifters has been proposed (4,5). A significant decrease in testosterone was observed during intensive training periods, followed by an increase during the following low-intensity training period (36). It was also shown that, in well-conditioned sub-

jects, this parameter was able to recover, with an overshoot phenomenon (40). The magnitude of the LH response to a testosterone decrease with intensive training appeared to be associated with fatigue status (5). Of note, we observed that these changes presented the same trends as the autonomic nervous system activities. As byproducts of sympathetic nervous activity, nocturnal urinary concentrations of dopamine, adrenaline, and noradrenaline have been shown to progressively decrease with cumulated physical training (23), which is consistent with our data.

Heart rate variability, thus, appears to be a potentially good indicator of cumulated training load in middle-distance runners, which could help to plan training programs. To benefit from such an individualized monitoring, each athlete should be given the ability to perform his or her own nocturnal heart rate variability measurement, which is recognized to have a good reproducibility. This could result in a better-individualized training profile and may be of help in the prevention of overtraining states.

The authors express their gratitude to the athletes and to the trainers of the athletic club of Coquelicot de Saint-Etienne, and Club 42, for their collaboration to the study.

Address for correspondence: Dr. Jean-Claude Barthélémy, Laboratoire de Physiologie, CHU Nord - Niveau 6, F-42055 Saint-Etienne, Cedex 2, France; E-mail: JC.Barthelemy@univ-st-etienne.fr.

REFERENCES

- AKAY, M., L. LANDESBURG, W. WELKOWITZ, Y. M. AKAY, and D. SAPOZNIKOV. Carotid-cardiac interaction: heart rate variability during the unblocking of the carotid artery. In: *Interactive Phenomena in the Cardiac System*, S. Sideman and R. Beyar (Eds.). New York: Plenum Press, 1993, pp. 365–372.
- BANISTER, E. W., and C. L. HAMILTON. Variations in iron status with fatigue modelled from training in female distance runners. *Eur. J. Appl. Physiol.* 54:16–23, 1985.
- BOUTCHER, H., and P. STEIN. Association between heart rate variability and training response in sedentary middle-aged men. *J. Appl. Physiol.* 70:75–80, 1995.
- BUSO, T., K. HÄKKINEN, A. PAKARINEN, et al. A systems model of training response and its relationship to hormonal responses in elite weight-lifters. *Eur. J. Appl. Physiol.* 61:48–54, 1990.
- BUSO, T., K. HÄKKINEN, A. PAKARINEN, H. KAUHANEN, P. V. KOMI, and J. R. LACOUR. Hormonal adaptations and modelled responses in elite weightlifters during 6 weeks of training. *Eur. J. Appl. Physiol.* 64:381–386, 1992.
- COSTILL, D. L. *Inside Running*. Indianapolis: Benchmark Press, 1986, pp. 123–132.
- DE MEERSMAN, R. E. Heart rate variability and aerobic fitness. *Am. Heart J.* 125:726, 1993.
- DEREVENCO, P., E. FLOREA, V. DEREVENCO, J. ANGHELS, and S. SIMU. Some psychological aspects of overtraining (Einige physiologische Aspekte des Übertrainings). *Sportartzt Sportmed.* 18:151–161, 1967.
- DRESSENDORFER, R. H., C. E. WADE, and J. H. SCAFF. Increased morning heart rate in runners: a valid sign of overtraining? *Physician Sportmed.* 13:77–86, 1985.
- FOSTER, C., A. C. SNYDER, N. N. THOMPSON, and K. KUETTEL. Normalization of the blood lactate profile in athletes. *Int. J. Sports Med.* 9:198–200, 1988.
- FRY, R. W., R. A. MORTON, and D. KEAST. Overtraining in athletes: an update. *Sport. Med.* 12:32–65, 1991.
- FURLAN, R., S. PIAZZA, S. DELL'ORTO, et al. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovasc. Res.* 27:482–488, 1993.
- GRAPS, A. An introduction to wavelets. *IEEE Comput. Sci. Eng.* 2:50–61, 1995.
- HAYASHI, N., Y. NAKAMURA, and I. MURAOKA. Cardiac autonomic regulation after moderate and exhaustive exercises. *Ann. Physiol. Anthropol.* 11:333–338, 1992.
- HOHNLOSER, S. H., T. KLINGENHEBEN, M. ZABEL, F. SCHRÖDER, and H. JUST. Intraindividual reproducibility of heart rate variability. *Pace* 15:2211–2214, 1992.

16. HUIKURI, H. V., K. M. KESSLER, E. TERRACALL, A. CASTELLANOS, M. K. LINNALUOTO, and R. J. MYERBURG. Reproducibility and circadian rhythm of heart rate variability in healthy subjects. *Am. J. Cardiol.* 65:391–393, 1990.
17. ISRAEL, S. About problems of overtraining from the internal medicine and physiological performance point of view (Zur Problematik des Übertrainings aus internistischer und leistungsphysiologischer Sicht). *Medizin Sport* 16:1–12, 1976.
18. JEUKENDRUP, A., M. HASSELINK, A. SNYDER, H. KUIPERS, and H. KEIZER. Physiological changes in male competitive cyclists after two weeks of intensified training. *Int. J. Sports Med.* 13:534–541, 1992.
19. KENNEY, W. L. Endurance training increases vagal control heart rate. In: *Exercise Physiology: Current Selected Research*, C. O. Dotson and J. H. Humphrey (Eds.). New York: AMS Press, 1988, pp. 59–65.
20. KINDERMANN, W. Overtraining: expression of a vegetative false increase (Das Übertraining: Ausdruck einer vegetativen Fehlsteuerung). *Dtsch. Z. Sportmed.* 37:138–145, 1986.
21. KLEIGER, R. E., J. T. BIGGER, M. S. BOSNER, et al. Stability over time of variables measuring heart rate variability in normal subjects. *Am. J. Cardiol.* 68:626–630, 1991.
22. KUIPERS, H., and H. A. KEIZER. Overtraining in elite athletes: review and directions for the future. *Sport Med.* 6:79–92, 1988.
23. LEHMANN, M., H. DICKHUTH, G. GENDRISCH, et al. Training–overtraining: a prospective, experimental study with middle-and long-distance runners. *Int. J. Sports Med.* 12:444–452, 1991.
24. LEHMANN, M., E. JAKOB, H. H. DICKHUTH, U. KORSTEN-RECK, and J. KEUL. Sympathetic activity in relation to performance diagnostics, training and overtraining. *Int. J. Sports Med.* 9:390–391, 1988.
25. LINDSAY, F. H., J. A. HAWLEY, K. H. MYBURGH, H. H. SCHOMER, T. D. NOAKES, and S. C. DENNIS. Improved athletic performance in highly trained cyclists after interval training. *Med. Sci. Sports Exerc.* 28:1427–1434, 1996.
26. MACIEL, B. C., L. GALLO, J. A. M. NETO, E. C. L. FILHO, J. T. FILHO, and J. C. MANÇO. Parasympathetic contribution to bradycardia induced by endurance training in man. *Cardiovasc. Res.* 19:642–648, 1985.
27. NOAKES, T. D. *The Lore of Running*. Cape Town: Oxford University Press, 1986, pp. 1–535.
28. PANTON, L. B., J. E. GRAVES, M. L. POLLOCK, et al. Relative heart rate, heart rate reserve, and VO₂ during submaximal exercise in the elderly. *J. Gerontol.* 51:M165–M171, 1996.
29. PERCIVAL, D. P. On estimation of the wavelet variance. *Biomedika* 82:619–631, 1995.
30. PICHOT, V., J. M. GASPOZ, S. MOLLIEUX, et al. Wavelet transform to quantify heart rate variability and to assess its instantaneous changes. *J. Appl. Physiol.* 86:1081–1091, 1999.
31. REILING, M. J., and D. R. SEALS. Respiratory sinus arrhythmia and carotid baroreflex control of heart rate in endurance athletes and untrained controls. *Clin. Physiol.* 8:511–519, 1988.
32. SEALS, D. R., and P. B. CHASE. Influence of physical training on heart rate variability and baroreflex circulatory control. *J. Appl. Physiol.* 66:1886–1895, 1989.
33. TASK FORCE OF THE EUROPEAN SOCIETY OF CARDIOLOGY AND THE NORTH AMERICAN SOCIETY OF PACING AND ELECTROPHYSIOLOGY. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation* 93:1043–1065, 1996.
34. TAYLOR, J. A., J. HAYANO, and D. R. SEALS. Lesser vagal withdrawal during isometric exercise with age. *J. Appl. Physiol.* 79:805–811, 1995.
35. URHAUSEN, A., H. GABRIEL, and W. KINDERMANN. Impaired pituitary hormonal response to exhaustive exercise in overtrained endurance athletes. *Med. Sci. Sports Exerc.* 30:407–414, 1997.
36. URHAUSEN, A., T. KULLMER, and W. KINDERMANN. A 7-week follow-up study of the behaviour of the testosterone and cortisol during the competition period in rowers. *Eur. J. Appl. Physiol.* 50:528–533, 1987.
37. UUSITALO, A. L., A. J. UUSITALO, and H. K. RUSKO. Endurance training, overtraining and baroreflex sensitivity in female athletes. *Clin. Physiol.* 18:510–520, 1998.
38. UUSITALO, A. L., A. J. UUSITALO, and H. K. RUSKO. Exhaustive endurance training for 6–9 weeks did not induce changes in intrinsic heart rate and cardiac autonomic modulation in female athletes. *Int. J. Sports Med.* 19:532–540, 1998.
39. VERDE, T., S. THOMAS, and R. J. SHEPARD. Potential markers of heavy training in highly trained distance runners. *Br. J. Sport Med.* 26:167–175, 1992.
40. VIRU, A. The mechanism of training effects: a hypothesis. *Int. J. Sports Med.* 5:219–227, 1984.
41. WILSON, W. M., and R. J. MAUGHAN. Evidence for a possible role of 5-hydroxytryptamine in the genesis of fatigue in man: administration of paroxetine, a 5-HT re-uptake inhibitor, reduces the capacity to perform prolonged exercise. *Exp. Physiol.* 77:921–924, 1992.