Nocturnal heart rate variability following supramaximal intermittent exercise

Running title: Heart rate variability following supramaximal exercise

Submission Type: Original investigation

Hani AL HADDAD, Paul B. LAURSEN, Said AHMAIDI and Martin BUCHHEIT.

1 Laboratoire de Recherche, EA 3300 «APS et Conduites Motrices : Adaptations Réadaptation», Faculté des Sciences du Sport, Université de Picardie Jules Verne, F-80025 Amiens, France.

2 School of Exercise, Biomedical and Health Sciences, Edith Cowan University, Joondalup, WA, Australia.

Address for correspondence:
Hani AL HADDAD,
Laboratoire de Recherche Adaptations Réadaptations (APS et conduites motrices)
Faculté des Sciences du Sport
Allée P. GROUSSET
80025 AMIENS CEDEX 1
France
Tel : +333.22.82.89.36
Fax : +333.22.82.79.10
Email : alhaddad.hani@gmail.com
Abstract

**Purpose:** To assess the effect of supramaximal intermittent exercise on long-term cardiac autonomic activity, inferred from heart rate variability (HRV). **Methods:** Eleven healthy males performed a series of two consecutive intermittent 15-s runs at 95% $V_{IFT}$ [i.e., speed reached at the end of the 30-15 Intermittent Fitness Test] interspersed with 15-s active recovery at 45% $V_{IFT}$ until exhaustion. Beat-to-beat intervals were recorded during two consecutive nights (habituation night and 1st night) before, 10 min before and immediately after exercise, as well as 12 (2nd night) and 36 h (3rd night) after supramaximal intermittent exercise. HRV indices were calculated from the last 5 min of resting and recovery periods, and the first 10 min of the first estimated slow wave sleep period. **Results:** Immediate post-supramaximal exercise vagal-related HRV indices were significantly lower than immediate pre-supramaximal exercise values ($p<0.001$). Most vagal-related indices were lower during the 2nd night compared with the 1st night (e.g., mean RR intervals, $p=0.03$). Compared with the 2nd night, vagal-related HRV indices were significantly higher during the 3rd night. Variables were not different between the 1st and 3rd nights; however we noted a tendency (adjusted effect size, aES) for an increased normalized high frequency component ($p=0.06$ and aES=0.70) and a tendency towards a decreased low frequency component ($p=0.06$ and aES=0.74). **Conclusion:** Results confirm the strong influence of exercise intensity on short- and long-term post-exercise heart rate variability recovery and might help explain the high efficiency of supramaximal training for enhancing indices of cardiorespiratory fitness.

**Keywords:** Cardiac autonomic system, vagal-related HRV indices, autonomic recovery, cardiovascular adaptation, high-intensity exercise.
Introduction

Repeated intermittent supramaximal exercise training is an efficient training strategy for increasing both central and peripheral components of cardiorespiratory fitness.\(^1,2\) For example, sprint interval training has been shown to enhance central factors such as maximal oxygen uptake and stroke volume,\(^2\) as well as peripheral components including resting muscle glycogen content, oxidative capacity, and buffering capacity.\(^1\) Another aspect that could be related to these enhancements that occur with supramaximal training could be the large perturbation of short and long-term post-exercise autonomic function.\(^3\) However, this response has been less described.

Heart rate (HR) variability (HRV) represents a simple, accessible and non-invasive tool to investigate the short- and long-term effects of exercise on cardiac autonomic nervous system function.\(^3,4\) Beat-to-beat HR variations in the high-frequency ranges are thought to be related to parasympathetic activity under respiratory influences, whereas low-frequency oscillations are linked to both sympathetic and parasympathetic outflow.\(^3\) It is also now believed that overnight sleep measurements over a number of days are appropriate for the tracking of changes in HRV following exercise.\(^6\) Indeed, the monitoring of slow wave sleep episodes has been proposed as a relevant recording period for the assessment of autonomic activity, since it offers a steady state free from body movements and environmental disturbance, a regular respiration frequency (spontaneously in the high frequency range) and heart rate, as well as a more pronounced vagal tone compared with other sleep episodes or awake recordings.\(^6\)

Comparison of post-exercise HRV over a continuum of exercise intensities suggests that anaerobic participation might be the main factor influencing short-term post-exercise cardiac autonomic function (i.e., in the early minutes of recovery).\(^3\) For example, Buchheit et al.\(^3\) recently showed that short-term post-exercise autonomic activity was impaired to the same degree following two different supramaximal exercise training programs that contained similar levels of anaerobic contribution; either short term repeated sprint training or more prolonged ‘aerobic’ high-intensity interval training.\(^3\) The long-term effects of submaximal or maximal exercise on autonomic activity have also been reported. These studies have shown that, following the initial decrease in the early hours after exercise, at least 24 to 72 hours may be required for HRV parameters to return to pre-exercise values.\(^3,4,7-10\) Nevertheless, there is limited data available describing the cardiac autonomic response following supramaximal exercise. Niewiadomski et al.\(^10\) examined the time course of HRV indices after two successive Wingate exercises (all-out 30-s efforts).\(^10\) Although results suggested a comparable effect on the time-course of long-term autonomic activity compared with a 30-min submaximal exercise bout [80% \(\dot{V} O_2\)peak (i.e., peak oxygen consumption)], the supramaximal exercise bout used in this study (total exercise time: 2 x 30 s = 1 min) is not one that would be typically used in an athletic training program (i.e., > 8 to 10 min). Consequently, a more prolonged or repeated supramaximal exercise stimulus might induce a greater perturbation of autonomic activity (i.e., delayed recovery).

In attempt to gain insight into the mechanisms underlying the high efficiency of supramaximal training, we investigated the short and long-term changes in HRV indices observed immediately after a bout of supramaximal exercise training and during sleep over a two-night trial. We hypothesised that HRV, representative of cardiac autonomic function, would be perturbed for several days after exercise cessation.
Methods

Participants

Eleven healthy males (age 21.5 ± 2.5 y, stature 1.78 ± 0.06 m, mass 74.3 ± 7.9 kg, participating in 2 to 5 hours of physical activity per week) volunteered for the study after providing written informed consent. All participants were familiar with exercise testing and supramaximal intermittent training and none were taking medication. The study conformed to the ethical guidelines outlined in the Declaration of Helsinki and was approved by the local human research ethics committee.

Study overview

Participants exercised on two separate occasions. First, they performed an incremental intermittent aerobic test (30-15IFT) to determine peak oxygen uptake (\( \dot{V}O_2 \) peak) and a reference velocity (\( V_{IFT} \)) for the ongoing exercise session. Second, at least 5 days later they performed a supramaximal intermittent running exercise. Ambulatory HR recordings were carried out on the two nights preceding the supramaximal intermittent exercise (SIE) bout and on the two nights following the exercise day. All participants were asked to consume a last light meal at least 3 h before each test session and to refrain from smoking and consuming drinks containing caffeine.

Graded maximal aerobic test

Participants performed the 30-15 intermittent fitness test (30-15IFT) (30-s of running interspersed with 15-s of passive recovery) which is a graded intermittent shuttle field test aimed at assessing a reference maximal speed for intermittent running prescription. The test started at 8 km.h\(^{-1}\) and velocity increased linearly from 0.5 km.h\(^{-1}\) every stage (45-s) until exhaustion. The last fully completed stage was defined as the participants’ \( V_{IFT} \). This field test has been showed to be accurate for individualizing intermittent shuttle exercise.

Supramaximal intermittent exercise

Supramaximal intermittent exercise consisted of 15-s periods of running at 95% of \( V_{IFT} \) (corresponding to an intensity of approximately 120% of the minimal velocity that elicits \( V_{O2max} \) determined during a continuous graded test, interspersed with 15-s of active recovery at an intensity corresponding to 45% \( V_{IFT} \). Participants performed two SIE sessions until exhaustion (S1 & S2) separated by 10 min of passive recovery in the sitting position. Participants were verbally encouraged and asked to run for as long as possible. Participants were considered exhausted when they could not accomplish the required distance.

Cardiorespiratory measurements

During the 30-15IFT, respiratory gas exchange values were measured breath-by-breath using a portable metabolic system (Cosmed K4b\(^2\), Rome, Italy). Before each test, the O\(_2\) and CO\(_2\) analyzers were calibrated using both ambient air and a gas mixture of known O\(_2\) and CO\(_2\) concentrations. The calibration of the turbine flowmeter of the K4b\(^2\) was carried out using a 3-L syringe (Quinton Instruments, Seattle, WA). \( \dot{V}O_2 \) peak was defined as the
highest $\dot{V}O_2$ attained in two consecutive 20-s periods. Criteria used to determine whether or not $\dot{V}O_2\text{peak}$ had been attained included: 1) a respiratory exchange ratio > 1.1, 2) a lactate concentration > 9 mmol.L$^{-1}$, and 3) attainment of theoretical maximal HR (220-age) ± 10 bpm.

**Blood lactate concentration**

Three minutes after the end of the 30-15IFT and after each SIE series, a fingertip blood sample (5 µL) was collected to determine the blood lactate concentration ([La]) using a portable analyzing system (Lactate Pro, Arkay Inc, Japan).

**Heart rate measurements**

Heart rate was continuously recorded (s810 heart rate monitor, Polar Electro, Kemple, Finland) for 10 min before and after the SIE in the seated position. Sleep recordings were made two nights before [one night for habituation and the following night for baseline, 1st night] and two nights after SIE [2nd night and 3rd night]. To improve signal quality, a conductive gel was applied to the belt’s electrodes before participants went to bed. Recorded data were downloaded on a computer using compatible Polar software (Polar Pro trainer 5.00.100, Polar Electro, Kemple, Finland). All irregular heartbeats were visually identified and replaced with interpolated adjacent RR interval values.

**HRV analysis**

Short-term perturbations of the autonomic nervous system were assessed via HRV indices calculated during the (stationary) last 5 min of the 10 min rest period before the SIE (SIE-10) and compared to those values collected during the last 5 min of the 10 min post-exercise recovery (SIE+10; i.e., from the 5th to the 10th min). Although breathing frequency is often controlled in HRV studies, we chose not to control this in our participants as we did not want to perturb the natural return of HR to baseline. Nevertheless, respiratory rate was always in the high frequency range (> 0.15 Hz).

Long-term autonomic nervous system recovery was assessed via evening HRV indices. As previously reported, HRV was specifically analysed during very quiet periods of sleep, which we estimated to be a slow wave sleep episode via the overnight RR interval trend. Following the recommendations of Brandenberger et al., analyzed RR data sets were selected on the basis of the following criteria (confirming the supposed ‘slow wave sleep episode’, Figure 1): 1) the first 10 min of the first low and regular HR episode lasting at least 15 min, 2) the lowest standard deviation of RR intervals (SDNN) throughout the period of interest, 3) a round Poincaré plot (i.e., a round cluster of points in RR intervals plotted as a function of the previous one) and 4) a low inter-beat autocorrelation between successive RR intervals (i.e., correlation coefficient of the Poincaré plot < 0.5).

Time-domain HRV indices computed were mean RR intervals (mRR), SDNN and the root mean square of successive differences in RR intervals (RMSSD). FFT proposed by Polar software was conducted to analyze spectral power density in the low (LF) (0.04 Hz - 0.15 Hz) and in the high (HF, >0.15Hz - 0.40 Hz) frequencies as well as their ratio LF/HF. The normalized HF ratio was also calculated as $HF_{nu} = HF / (LF+HF)$. mRR, RMSSD and HF power were considered as indexes of parasympathetic activity. LF power can be considered as an index of sympathetic activity, but given its parasympathetic component, it should be used with care. Because the absolute values of spectral parameters are highly correlated to variance (corresponding to total power), focusing on the fractional distribution
of power independently of the absolute values of variance is also necessary. The LF/HF ratio is considered to reflect sympathovagal balance, and the normalized high frequency component (i.e., HFnu) is considered to represent the parasympathetic component of the sympathovagal balance.5

Statistical analysis

For all analyses, normality of the distribution was checked with the Kolmogorov-Smirnov goodness-of-fit test. Homogeneity of variance was verified by a Levene median test. A logarithm transform (Ln) was applied for HF and LF absolute values to obtain a normal distribution. A Student’s t test was conducted to compare data from SIE-10 and SIE+10. To compare data obtained during different nights of sleep, a one-way analysis of variance (ANOVA) for repeated measures with a Tukey’s post hoc test was performed. Pearson’s r was calculated to examine correlations between cardiorespiratory and HRV parameters. Results for short-and long-term recovery are presented as means ± standard errors of the mean (SE) in absolute values and as a percentage of the pre-exercise period (SIE-10) or night (1st night). All statistical analyses were performed using Sigma Stat software (Sigma Stat 3.11, Systat software inc., San Jose, Calif., USA), with significance set at an alpha level of 0.05. If no significant effects were observed, but a tendency towards significance (p<0.1) was apparent, then effect sizes (ES) and adjusted ES (aES) were calculated for percentage and absolute values, respectively. ES and aES formulas are as follow:

$$ES = \frac{m_a - m_b}{\sqrt{(SD^2_a)(n_a - 1) + (SD^2_b)(n_b - 1)}} \frac{1}{(n_a + n_b - 2)}$$

$$aES = \sqrt{1 - r}$$

where b stands for before, a for after, m for group mean, SD for standard deviation, n for group subject size, r for correlation coefficient between before and after data. The scale proposed by Cohen was used for interpretation. aES were used for all parameters, except for data expressed as a percentage of 1st night values, where ES were calculated. The magnitude of the difference was considered either small (>0.2), moderate (>0.5), or large (>0.8). If there was at least a moderate (a)ES (>0.50), but the statistical power was low, the likelihood of a Type II error was noted.

Results

Mean \( \dot{V}_O_2 \) peak and \( V_{IFT} \) were 52 ± 3.4 ml.kg\(^{-1}\).min\(^{-1}\) and 19.6 ± 0.9 km.h\(^{-1}\), respectively. Total SIE duration was 10 min + 1 min 30 s (6 min + 48 s ± 4 min 48 s for S1 and S2, respectively), total distance covered during both series was 2330 ± 435
m. Mean \([\text{La}]\) concentration was 13.9 ± 2.4 mmol.L\(^{-1}\) and 12.15 ± 1.6 mmol.L\(^{-1}\) for S1 and S2.

Figure 2 illustrates short-term autonomic nervous system perturbation following SIE, with values expressed as a percentage of those measured for SIE-10. Global HRV index (i.e., SDNN \(p<0.001\)) was significantly reduced and all vagal-related HRV indices were significantly depressed \((p<0.001)\) at SIE+10, when compared to SIE-10. LF/HF ratio was also significantly increased immediately after SIE \((p<0.001)\).

Long-term absolute HRV recovery indices during the three nights are reported in Table 1. Figure 3 shows changes in mRR, SDNN, LnHF and LF/HF, expressed as a percentage of the 1\(^{st}\) night. Compared to the 1\(^{st}\) night, parasympathetic-related HRV indices were significantly decreased or tended to decrease during the 2\(^{nd}\) night (mRR, \(p=0.03\); HFnu, \(p=0.06\) and aES=0.73; LnHF, \(p=0.06\) and aES= 1.49). Also a sympathetic predominance was noted in the sympathovagal balance where LF/HF ratio expressed as percentage of the 1\(^{st}\) night was significantly increased \((p=0.03)\). On the 3\(^{rd}\) night, most parasympathetic- and sympathetic-related indices returned to baseline values, whereas some indices tended to reach higher (vagal-related) and lower (sympathetic-related) values compared to the 1\(^{st}\) night. We also noted a tendency for an increased HFnu on the 3\(^{rd}\) night compared with the 1\(^{st}\) night \((p=0.06\) and aES=0.70). Compared to the 2\(^{nd}\) night, vagal-related HRV indices were significantly higher on the 3\(^{rd}\) night (mRR, \(p=0.02\); RMSSD, \(p=0.04\)) or tended to be higher (LnHF, \(p=0.06\) and aES=0.75; HFnu, \(p=0.06\) and aES=1.04). We also noted a shift towards a parasympathetic predominance in the LF/HF ratio (i.e., expressed as percentage of the 1\(^{st}\) night; \(p=0.03\)). The sympathovagal balance expressed as a percentage of the 1\(^{st}\) night increased significantly on the 2\(^{nd}\) night \((p=0.03)\) and decreased significantly on the 3\(^{rd}\) night \((2^{nd}\) night vs. 3\(^{rd}\) night, \(p=0.02)\).

There was no correlation between HRV indices on the 2\(^{nd}\) night and VO\(_2\)Peak or \([\text{La}]\). However, significant correlations were found between \([\text{La}]\) and parasympathetic indices measured on the 3\(^{rd}\) night (e.g., \([\text{La}]\) vs. RMSSD, \(r=0.63\) and \(p=0.04\); vs. LnHF, \(r=0.63\) and \(p=0.04\)).

**Discussion**

The present study is the first to show the short- and long-term response of cardiac autonomic nervous system function following a bout of supramaximal intermittent exercise. Because supramaximal training has been shown to markedly impair post-exercise cardiac autonomic function,\(^3\) and because such exercise has been reported as being effective at enhancing cardiovascular fitness,\(^1,2,14\) we hypothesised that HRV indices would be perturbed for several days after exercise cessation. Using non-confounded HRV recordings taken immediately after exercise and during selective quiet periods of sleep, we observed a significant reduction of vagal-related HRV indices and a shift in sympathovagal balance toward a sympathetic predominance at 10 min and 12 h after SIE. All HRV indices had returned to pre-exercise levels by the 36 h post-SIE measurement period.

**Short term recovery (within 10 minutes)**

Five minutes after the supramaximal intermittent exercise, all parasympathetic-related indices (e.g., RMSSD) were significantly decreased compared to immediate pre-exercise values, whereas those representative of sympathetic activity were increased (i.e., LF/HF).
(Figure 2). It is worth noting that while HRV offers a qualitative marker of cardiac parasympathetic regulation, care should nevertheless be taken when interpreting sympathovagal balance indices (i.e., LF/HF), because the LF component reflects some unknown interactions between sympathetic and parasympathetic inputs. However, these results are in accordance with previous studies that have reported comparable levels of depressed vagal-related HRV indices (and conversely increased sympathetic-related ones) after either submaximal or supramaximal exercise. Indeed, HRV has been shown to be impaired for 5 to 10 min following 30 to 46 min at 79% \( \dot{V}_O_{2\text{max}} \) for 1 h subsequent to six 3-min intermittent runs performed at the running speed associated with \( \dot{V}_O_{2\text{max}} \) for 9 min after a maximal graded test, for 10 min after a 30-s all-out Wingate test and for 5 min following repeated sprint running.

The strong perturbations of post-exercise parasympathetic activity observed here might be related to the supramaximal nature of the exercise and associated anaerobic participation and system stress metabolic accumulation. Indeed, the [La] values measured in the present study (13.9 ± 2.4 mmol.L\(^{-1}\) and 12.15 ± 1.6 mmol.L\(^{-1}\) for S1 and S2) are comparable to those recently shown under similar exercise conditions, after which parasympathetic reactivation was significantly impaired. In this study, supramaximal exercises inducing a high anaerobic participation (10.9 ± 0.9 mmol.L\(^{-1}\)) were associated with a low level of parasympathetic reactivation, whereas exercise of moderate intensity (3.5 ± 0.2 mmol.L\(^{-1}\)) induced a faster level of parasympathetic reactivation. This finding is consistent with other studies that have investigated post-exercise autonomic activity following submaximal exercise. Indeed, minimal disturbance of HRV indices have been shown following 60 and 120 min of submaximal exercise completed at an intensity below the first ventilatory threshold (55-65% \( \dot{V}_O_{2\text{max}} \); post exercise [La] = 1 ± 0.1 mmol.L\(^{-1}\)), following 25 min at 50% \( \dot{V}_O_{2\text{max}} \) and following 5 min at an intensity lower than 30% \( \dot{V}_O_{2\text{max}} \). These findings combined suggest that anaerobic participation, rather than mean aerobic power and exercise duration, is the most likely determinant of short-term autonomic activity after exercise. Other possible contributors to the perturbation of autonomic state after exercise (i.e., over sympathetic activity) include activation of muscle metaboreflex, oxygen debt repayment and core temperature regulation; however these variables were not monitored in the present study.

**Long term recovery (12 and 36h after exercise)**

This study is the first to investigate the long-term effect of supramaximal exercise on nocturnal HRV using an exercise stimulus representative of a training session typically used by athletes. During the first night following supramaximal exercise (i.e., 2\(^{nd}\) night), parasympathetic-related indices were significantly decreased (mRR) or tended to be lower (LnHF) compared to the pre-exercise night (i.e., 1\(^{st}\) night) readings (Table 1). Sympathovagal balance expressed as percentage of the 1\(^{st}\) night was significantly increased, suggesting a sympathetic predominance. During the 3\(^{rd}\) night of recording (i.e., 2\(^{nd}\) night after exercise), parasympathetic indices were increased (i.e., RMSSD, mRR) or tended to increase (i.e., LnHF) compared with 2\(^{nd}\) night indices. Absolute HRV indices measured on the 3\(^{rd}\) night were comparable to those measured during the pre-exercise night, suggesting that cardiac autonomic function had returned to baseline levels; however a shift towards parasympathetic predominance in the sympathovagal balance was noted (i.e., LF/HF, Table 1 and Figure 3). We also found a correlation between [La] and parasympathetic-related indices measured the second night after exercise. These results are consistent with previous diurnal recordings performed after either submaximal or supramaximal exercises.

After submaximal or maximal exercise, autonomic activity has been shown to return
to basal values within 48 h after 30 min of high-intensity exhaustive exercise,\textsuperscript{4} after 30 to 46 min at 79\% $\dot{V}O_{2}\text{max}$\textsuperscript{9} and within at least 72 h following six 3-min intermittent runs performed at $\dot{V}O_{2}\text{max} -1\text{ km.h}^{-1}$.\textsuperscript{8} Interestingly, within the same time period (48 h), Hautala et al.\textsuperscript{7} observed a rebound of parasympathetic activity (i.e., increased HF power density) after 75 km of cross-country skiing. In the present study, while we noted a tendency for a shift in sympathovagal balance toward parasympathetic predominance (higher HF ratio aES = 0.70), a similar rebound effect was not as clearly apparent, probably due to an insufficient exercise load, or due to our participants’ training status. Even if exercise intensity was well individualised (prescribed at a percentage of their $V_{\text{FIT}}$), it is worth noting the important inter-individual differences in the autonomic nervous system response observed (inferred from high SE values, i.e., 3\textsuperscript{rd} night LF/HF = 56.9 ± 14.0). These inter-individual responses might be due to distinct training backgrounds,\textsuperscript{25} and may explain why, from a global observation, no marked rebound of parasympathetic activity was observed. Nevertheless, the present HRV response, which occurred in response to a total exercise duration of less than 10 min on average, is comparable with those described in the literature following prolonged submaximal exercise.

Although a control trial using continuous moderate intensity exercise would have helped draw definitive conclusions regarding the influence of exercise intensity on cardiac autonomic function, the present results nevertheless suggest that supramaximal exercise is an efficient stimulus for perturbing long-term cardiac autonomic activity. Regarding the long-term HRV recovery following supramaximal exercise, our results are comparable with those reported by Niewiadomski et al.\textsuperscript{10} after two all-out 30-s Wingate efforts. However, since two Wingate exercises represent a very limited training stimulus (1 min),\textsuperscript{10} we aimed to investigate the effects of a longer and more practical training session on long-term autonomic recovery. We hypothesised that a longer supramaximal exercise training stimulus would induce a greater perturbation of autonomic activity (i.e., delayed recovery). Present results do not support such a hypothesis since HRV values were already comparable to pre-exercise values during the night 36 h following exercise. This finding could indicate that exercise duration does not significantly influence post-exercise autonomic recovery, with exercise intensity being the more likely key factor. Indeed, correlations showed that a high [La] response was related to the greater level of vagal-related indices during long-term recovery (i.e., 36 h later). The degree of body’s homeostasis perturbation through system stress metabolite accumulation (e.g., lactate), as well as exercise-induced plasma volume expansion,\textsuperscript{11} might be responsible for the long-term changes in HRV indices observed. For instance, elimination of plasma catecholamine, which is known to occur within at least 24 h,\textsuperscript{26} might be responsible for triggering sympathetic activity during the early hours after supramaximal exercise and help explain the delayed long-term recovery of HRV observed. The tendency to observe a shift toward parasympathetic predominance during the 3\textsuperscript{rd} night can also be related to an increase in plasma volume,\textsuperscript{27} which could have activated baroreflex-mediated parasympathetic activity (and conversely, inhibited sympathetic activity). The physiological determinants of long-term autonomic nervous system recovery have not been extensively investigated, but normalisation of blood pressure or baroreflex-mediated control of plasma volume expansion consecutive to exercise dehydration might also be involved.\textsuperscript{28}

**Practical applications**

Recent studies have reported autonomic nervous system status to be an important factor governing the training response. This is because assessment of autonomic nervous system function effectively includes important information concerning acute and chronic physiological processes that occur before, during and after aerobic exercise training. Our present results confirm that (nocturnal) heart rate variability monitoring can be used as a
simple non-invasive tool to evaluate, at the individual level, the autonomic/homeostatic impact of a given exercise type. This information might be of interest to practitioners’ intent on monitoring the response of specific training prescription. For instance, Iellamo et al.\textsuperscript{29} assessed cardiac autonomic nervous system evolution (i.e., diurnal recording) in world class athletes throughout a 9 month training period and showed heart rate variability measures to follow training loads and performance. Along the same line of thinking, Hautala and co-workers\textsuperscript{30} suggest that daily assessment of autonomic nervous system activity could serve as an indicator of a recovered physiological condition prior to aerobic training. Compared to a ‘classic’ training program (i.e., without heart rate variability feedback), the heart rate variability-guided athletes displayed significantly superior improvements in maximal oxygen uptake and physical performance. Since a 10-minute supramaximal exercise bout might not induce higher autonomic nervous system disturbances compared with long and moderate exercises as described in the literature \textsuperscript{4,7-10}, its use as a time-efficient alternative training method should be considered by coaches and exercise practitioners alike.

Conclusion

Via non-confounded heart rate variability recordings (i.e., immediately after exercise and during selective quiet periods of sleep), we have shown that a single supramaximal exercise bout, lasting no more than 12 minutes, can perturb the autonomic nervous system for about 36 hours. This confirms the strong influence of exercise intensity on short- and long-term post-exercise heart rate variability recovery and might help explain the high efficiency of supramaximal training for enhancing indices of cardiorespiratory fitness.

Acknowledgments

The authors would like to thank the participants who graciously volunteered their time for the study. We also would like to thank M. Pierre-Marie Leprêtre for his assistance.
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Table 1 Evolution of heart rate variability (HRV) indices over the three nights after supramaximal intermittent exercise

<table>
<thead>
<tr>
<th></th>
<th>1st night</th>
<th>2nd night</th>
<th>3rd night</th>
<th>p (Anova)</th>
</tr>
</thead>
<tbody>
<tr>
<td>m RR (ms)</td>
<td>1179.0 ± 48.3</td>
<td>1095.7 ± 47.3*</td>
<td>1195.5 ± 54.4#</td>
<td>0.02</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>49.6 ± 3.0</td>
<td>44.2 ± 9.8</td>
<td>48.3 ± 4.1</td>
<td>0.17</td>
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<tr>
<td>RMSSD (ms)</td>
<td>56.3 ± 5.0</td>
<td>46.7 ± 4.6</td>
<td>62.5 ± 7.3#</td>
<td>0.04</td>
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<tr>
<td>LnHF (ms²)</td>
<td>6.90 ± 0.17</td>
<td>6.49 ± 0.18†</td>
<td>7.00 ± 0.79‡</td>
<td>0.09</td>
</tr>
<tr>
<td>HFnu</td>
<td>0.61 ± 0.05</td>
<td>0.52 ± 0.04†</td>
<td>0.68 ± 0.05‡</td>
<td>0.06</td>
</tr>
<tr>
<td>LnLF (ms²)</td>
<td>6.41 ± 0.21</td>
<td>6.36 ± 0.17</td>
<td>6.12 ± 0.21</td>
<td>0.31</td>
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<tr>
<td>LF/HF</td>
<td>85.5 ± 22.7</td>
<td>106.8 ± 17.2</td>
<td>56.9 ± 14.0</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Values are mean ± SE. mean RR interval (mRR), standard deviation of RR intervals (SDNN), the square root of the mean squared differences of successive RR intervals (RMSSD), log-transformed high frequency power (LnHF), normalized unit in high frequency range (HFnu), log-transformed low frequency power (LnLF) and sympathovagal balance (LF/HF). HRV was analysed during sleep for the night before (1st night) and two nights after the supramaximal intermittent exercise (2nd night and 3rd night). *: significant difference vs. 1st night; #: Significant difference vs. 2nd night; †: differences vs. 1st night with adjusted effect size considered as moderate (>0.5); ‡: differences vs. 2nd night with adjusted effect size considered as moderate (>0.5).
Figure 1 - A: Nocturnal HR evolution and selected HR periods during the estimated slow wave sleep episode (10-min period between lines) where HRV indices were calculated (selection criteria based on Brandenberger et al.6). B: Poincaré plot for the chosen episode shown in A.
**Figure 2** - Relative changes from immediate pre-exercise values to immediate post-exercise values. Results are presented as mean (±SE) for mean RR interval (mRR), standard deviation of RR intervals (SDNN), square root of the mean squared differences of successive RR intervals (RMSSD), natural-logarithm of high frequency component (LnHF) and sympathovagal balance (LF/HF). *: significant difference from pre-exercise values $p<0.001$. 
Figure 3 - Changes in HRV indices throughout the three consecutive nights (before supramaximal intermittent exercise (1st night), during the first (2nd night) and second (3rd night) post supramaximal intermittent exercise nights. Data are mean (±SE) for mean RR interval (mRR), standard deviation of RR intervals (SDNN), natural-logarithm of the high frequency component (LnHF) and sympathovagal balance index (LF/HF). *: significant difference vs. 1st night; #: significant difference vs. 2nd night. *, #, p<0.05. Effect size considered as moderate (>0.5) †: compared to 1st night; ‡: compared to 2nd night.