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Parasympathetic reactivation after repeated sprint exercise

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¹Laboratoire de Recherche EA 3300 (APS et Conduites Motrices: Adaptations Réadaptations), Faculté des Sciences du Sport d'Amiens, Université de Picardie Jules Verne, France; and ²School of Exercise, Biomedical and Health Sciences, Edith Cowan University, Joondalup, Western Australia, Australia

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Buchheit M, Laursen PB, Ahmaidi S. Parasympathetic reactivation after repeated sprint exercise. *Am J Physiol Heart Circ Physiol* 293: H133–H141, 2007. First published March 2, 2007; doi:10.1152/ajpheart.00062.2007.—The purpose of this study was to examine the effects of muscular power engagement, anaerobic participation, aerobic power level, and energy expenditure on postexercise parasympathetic reactivation. We compared the response of heart rate (HR) after repeated sprinting with that of exercise sessions of comparable net energy expenditure and anaerobic energy contribution. Fifteen moderately trained athletes performed 1) 18 maximal all-out 15-m sprints interspersed with 17 s of passive recovery (RS), 2) a moderate isocaloric continuous exercise session (MC) at a level of mean oxygen uptake similar to that of the RS trial, and 3) a high-intensity intermittent exercise session (HI) conducted at a level of anaerobic energy expenditure similar to that of the RS trial. Subjects were immediately seated after the exercise trials, and beat-to-beat HR was recorded for 10 min. Parasympathetic reactivation was evaluated through 1) immediate postexercise HR recovery, 2) the time course of the root mean square for the successive R-R interval difference between successive 30-s segments (RMSSD_{30s}) and 3) HR variability vagal-related indexes calculated for the last 5-min stationary period of recovery. RMSSD_{30s} increased during the 10-min period after the MC trial, whereas RMSSD_{30s} remained depressed after both the RS and HI trials. Parasympathetic reactivation indexes were similar for the RS and HI trials but lower than for the MC trial ($P < 0.001$). When data of the three exercise trials were considered together, only anaerobic contribution was related to HR trial-derived indexes. Parasympathetic reactivation is highly impaired after RS exercise and appears to be mainly related to anaerobic process participation.

heart rate recovery; vagal-related indexes; autonomic activity; sprint interval training

REPEATED SPRINT (RS) training, characterized by recurring sessions of brief repeated bouts of supramaximal exercise, may be a time-efficient strategy for inducing metabolic adaptations in human skeletal muscle (15). Adaptations shown after a short time course of RS training include increased resting glycogen content (20), increased maximal activities of various enzymes involved in glycolytic (31) and oxidative energy provision (10, 20), an increased H⁺ buffer capacity (17, 20), and decreased cycling time-trial performance (10, 15, 20). As a result, RS training has been proposed as a viable alternative to classically prescribed submaximal endurance training (10, 17, 20).

Today, there are growing social and psychological reasons to encourage RS training within clinical populations. First, RS training is remarkably time efficient and more compatible with the Western world's time-poor modern lifestyle. Second, the

concept of RS training may also be more attractive than continuous exercise for sedentary individuals who have difficulty handling exercise sessions that are perceived to be of a long duration and of a monotonous nature. Third, the high level of muscular power needed to perform RS training stresses more type II muscle fibers, which comprise approximately one-half of the fibers within the thigh (vastus) and calf (gastrocnemius) muscles of most people and of which are not recruited during low-intensity exercise. Not surprisingly, RS training has been shown to result in maintenance of and even an improvement in muscular strength (17).

Although the effectiveness of RS training for maintaining and improving muscular performance is established (47), its influence on postexercise autonomic function is unknown. Knowledge of this effect, however, is critical for clinicians dealing with patients with certain disease states that may leave them more prone to adverse cardiovascular events. Indeed, sympathetic hyperactivity (4) or reduced cardiac vagal tone (3) after exercise may confer a poor cardioprotective background and underlie ischemic heart disease and the pathogenesis of malignant ventricular arrhythmias and sudden cardiac death.

To quantify parasympathetic reactivation after exercise, the time course of HR recovery (HRR) and HR variability (HRV) indexes have been used (7, 13, 22, 42, 45). The validity of these markers has been examined with the use of drugs that cause a parasympathetic blockade (i.e., atropine) (22, 45). The simplest and most used HRR index is the number of heart beats recovered within 60 s after the cessation of exercise (13). Fitting postexercise HRR to a first-order exponential decay curve has also been used (7, 42). Regarding HRV, it is the vagal-related indexes, such as the root mean square of successive differences of R-R intervals (RMSSD) or the power density in the high-frequency (HF) range obtained by spectral analysis, that are the most widely used methods (50). Finally, a new and simple temporal time-varying parasympathetic index has recently been proposed by Goldberger et al. (22). This index is derived from the time course of the RMSSD measured on successive 30-s segments (RMSSD_{30s}) over the recovery period.

The acute effects of a single exercise bout on HRV have been placed into long- and short-term categories. From 24 to 48 h after exercise, a rebound of parasympathetic activity, which seems independent of the exercise type undertaken (38), has often been described (18, 26). Concerning the short-term evolution of autonomic activity, an initial decrease in HRV and vagal-related indexes has been observed within minutes to hours after exercise (38, 39, 51). Moreover, vagal restoration

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has been shown to be more delayed after intense [80% peak oxygen uptake ($\dot{V}O_{2\text{ peak}}$)] than after moderate-intensity exercise (50% $\dot{V}O_{2\text{ peak}}$) (38, 39). Parasympathetic activity has also been shown to be more impaired after resistance exercises than with a moderate cycling effort (27). Nevertheless, in all of these previous studies, the absence of anaerobic metabolite measurements has made less clear the influences that muscular power engagement, anaerobic participation, and aerobic power level have on postexercise parasympathetic reactivation. For RS exercise, although $\dot{V}O_{2\text{ peak}}$ is sometimes reached (16), this is not always the case (21, 47); therefore, from a metabolic point of view, the aerobic exercise intensity could be considered to be "submaximal." Nevertheless, sprinting requires the development of very high muscular power, so that the exercise might also be considered to be "supramaximal" from a muscular and anaerobic point of view. As a result, the effects of RS on parasympathetic reactivation are difficult to predict.

The primary purpose of the present study was to quantify the time course of the parasympathetic reactivation after RS exercise and to observe the respective effects of muscular power engagement, anaerobic participation, aerobic power level, and energy expenditure on postexercise autonomic control. We used multiple HR-derived parasympathetic indexes measured after RS exercise and compared these with indexes obtained after exercises of 1) comparable levels of net energy expenditure to distinguish the specific effect of anaerobic process participation and muscular power engagement and 2) comparable anaerobic contribution to observe the explicit incidence of net energy expenditure. Finally, we used multiple linear regressions to show the respective consequence of each metabolic and mechanical exercise characteristic on postexercise autonomic regulation.

METHODS

Participants

Based on the assumption that a 8 ± 5 beat/min difference in HRR_{60} is meaningful (7, 13), we used Minitab 14.1 software (Minitab, Paris, France) to determine that a sample size of nine subjects was needed to provide a power of 80% with an alpha of 0.05. Although the evaluation of the incidence of RS exercise on autonomic function is especially crucial in sedentary individuals and patients, we preferred here to recruit moderately trained subjects because we expected that they would be more able to cope with this innovative experimentation ($n = 15$; age = 21.3 ± 3.1 yr, height = 178.1 ± 7.5 cm, body mass = 73.8 ± 10.4 kg, muscle mass = 30.7 ± 0.3 kg, body surface area = 1.91 ± 0.05 m²). All participants were routinely involved (6.6 \pm 2.7 h/wk) in various intermittent activities (soccer, handball, basketball, or tennis) and had no history or clinical sign of cardiovascular or pulmonary diseases. Muscle mass was estimated based on forearm and thigh calf girths and skinfolds (33), whereas body surface area was calculated according to the formula provided by Mosteller (37). Subjects were not taking prescribed medications and presented with normal levels of blood pressure and ECG patterns. The study conformed to the recommendations of the Declaration of Helsinki, and participants gave voluntary written consent to participate in this experiment, which was approved by the local ethics committee.

Experimental Design

Subjects performed, at the same time of day (± 1 h) over a 2-wk period, one graded aerobic test and three sessions of short exercise bouts. Each test was separated by at least 48 h. All tests were performed on an indoor synthetic track where ambient temperature

ranged from 18 to 22°C. The graded maximal aerobic test was performed first, followed by the three other tests in a random and balanced order for each subject. The exercise bouts consisted of a repeated sprinting bout (RS trial), a submaximal and continuous bout completed at comparable net energy expenditure to that of the RS bout (MC trial), and a high-intensity intermittent bout completed at a level of anaerobic energy expenditure comparable to that of the RS bout (HI trial). Subjects were familiarized with the exercise procedure before commencement of each test. Subjects were asked not to perform exercise on the day before a test and were asked to consume their usual last meal at least 3 h before the scheduled test time. All three of the experimental exercise bouts were preceded by a supervised and standardized warm-up consisting of 5 min of running at 45% of V_{IFT} (where IFT is intermittent fitness test, corresponding to a "maximal intermittent aerobic reference velocity," see below) along with a few athletic drills (i.e., skipping) and short bursts of progressive accelerations on the track. Exercise bouts began 2 min after this warm-up period.

Maximal graded aerobic test. Maximal aerobic performance of each subject was assessed with a 30–15 intermittent fitness test. This intermittent shuttle field test elicits $\dot{V}O_{2\text{ peak}}$ and has been shown to be accurate for individualizing intermittent shuttle running exercise (6). Moreover, this test has been shown to be reliable (intraclass correlation coefficient = 0.96) for the final running speed (V_{IFT}). The 30–15 intermittent fitness test consists of 30-s shuttle runs interspersed with 15-s passive recovery periods. For this test, velocity was set at 8 km/h for the first 30-s run, and speed was increased by 0.5 km/h for every 30-s stage thereafter. Subjects were required to run back and forth between two lines set 40 m apart at a pace that was governed by a prerecorded beep. The prerecorded beep allowed the subject to adjust their running speed within a 3-m zone placed in the middle and at each extremity of the field. During the 15-s recovery period, subjects walked in the forward direction toward the closest line (at either the middle or end of the running area, depending on where their previous run had stopped); this line determined where they would start the next run stage. Subjects were instructed to complete as many stages as possible, and the test ended when the subject could no longer maintain the required running speed or when subjects were unable to reach a 3-m zone in time with the audio signal on three consecutive occasions. The velocity attained during the last completed stage was determined as the subject's V_{IFT} . Respiratory gas exchanges were measured with an automated portable metabolic system (VO2000; Medgraphics, St. Paul, MN) (34). Before each test, the O₂ and CO₂ analysis systems were calibrated. $\dot{V}O_{2\text{ peak}}$ was defined as the highest oxygen uptake ($\dot{V}O_2$) attained in a 20-s period. Subjects were considered to have reached $\dot{V}O_{2\text{ peak}}$ if at least two of the following criteria were met: 1) respiratory exchange ratio was >1.1 , 2) a maximal HR (HR_{max}) was attained within 10 beats/min of the age-predicted maximum, and 3) volitional fatigue occurred (28).

RS exercise. RS exercise consisted of repeated 15-m sprints with 17 s of passive recovery (52). Subjects ran in the opposite direction after the 17-s rest. Total duration of the test was 6 min. Respiratory gas exchanges were measured during the test as previously described.

Equivalent RS net energy expenditure exercise. This trial was used to evaluate the influence of the muscular power engagement and/or anaerobic participation on postexercise parasympathetic reactivation. The "aerobic" intensity (% $\dot{V}O_{2\text{ peak}}$) of MC (moderate and continuous) exercise was similar to that measured during RS and corresponded to 65% of V_{IFT} (6). The exercise duration was determined a priori to achieve similar levels of net energy expenditure compared with the RS trial. Total calculated MC trial duration ranged from 368 to 501 s. As for other exercises, respiratory gas exchanges were measured during the test.

Equivalent RS anaerobic energy exercise. To examine the influence of net energy expenditure on parasympathetic reactivation, we attempted to compare the RS trial to that which occurred during a 12-min high-intensity intermittent exercise trial (HI trial). Our choice

of such an exercise trial of that duration was based on prior work showing that, at appropriate duration and intensity, HI exercise 1) is associated with lactate accumulation similar to that observed after RS exercise and 2) induces twice the net energy expenditure of a short RS exercise (>200 kcal) (2). The exercise consisted of 30-s runs at 90% of V_{IFT} [corresponding to ~105% of maximal aerobic velocity (6)] with intermittent 30-s passive recovery periods. During the 30-s exercise period, athletes were required to run back and forth over the 40-m area so that they covered the distance determined according to their V_{IFT} . Run interval pace was provided by a digital timer that produced a sound every 30 s from the start to the end of the exercise interval. After a 30-s rest, the subject ran again in the opposite direction. Respiratory gas exchanges were also measured during the test as previously described.

Blood lactate measurement. Three minutes after the end of each exercise set, a fingertip blood sample (5 μ l) was collected, and blood lactate concentration ($[La]_b$) was determined (Lactate Pro; Arkray, Kyoto, Japan) (44). The accuracy of the analyzer was checked before each test using standards.

Beat-to-Beat HR Analyses

Materials. An electrode transmitter belt (T61; Polar Electro, Kempele, Finland) was fitted to the chest of each subject as instructed by the manufacturer, after application of conductive gel. A Polar Electro S810 HR monitor was used to continuously record beat-to-beat HR during each exercise and each subsequent recovery phase (19).

Data treatment. All R-R series recorded by the S810 were extracted on an IBM-compatible personal computer with the processing program (Polar precision performance SW 4.03, Polar Electro). Occasional ectopic beats (irregularity of the heart rhythm involving extra or skipped heartbeats, i.e., extrasystole and consecutive compensatory pause) were visually identified and manually replaced with interpolated adjacent R-R interval values.

Postexercise HRR assessment. Beat-to-beat HR was analyzed during the 10-min recovery period immediately after each exercise. As soon as exercise was stopped, all subjects immediately sat passively on a chair placed adjacent to the track. Time duration between the end of exercise and sitting was <5 s. Particular attention to this detail was made because differences in body posture have been shown to result in different absolute HRR values (49). HRR was calculated by three ways. The first HRR index was defined as the absolute difference between the final HR observed at the end of exercise (mean of 5 s) and the HR recorded 60 s after (HRR_{60s} ; mean of 5 s) (7, 13). The second HRR index was calculated as proposed by Imai et al. (29), using a semi-logarithmic regression technique. The natural logarithm of heart beats during the initial rapid HR decrease (from the 10th to the 40th s) was plotted against the elapsed time of exercise, and a linear regression analysis was applied. The time constant of the short-time postexercise HR decay (T_{30}) was thus determined as the negative reciprocal of the slope of the regression line. HRR was further analyzed by fitting the 10-min postexercise HRR into a first-order exponential decay curve (7). An HR time constant (HRR_{τ}) was then produced by modeling the resultant 10-min of HR data using an iterative technique (Sigmaplot 10; SPSS Science, Chicago, IL) with the following equation: $HR = HR_0 + HR_{amp}e^{(-T/HRR_{\tau})}$, where HR_0 = resting (final) heart rate (HR), $HR_{amp} = HR_{max} - HR_0$, and T = time (s). A single exponential model was applied for all subjects because previously used models (i.e., biexponential) (41) did not significantly improve the gain of variance for the fit between modeled and measured HR data. HRR_{τ} and HR_{amp} were retained for statistical analyses.

Time-varying vagal-related HRV index. Although a progressive increase in the R-R interval is generally observed over the initial 5 min of recovery, on shorter scales (i.e., 15–60 s), the curve is piece-wise linear with superimposed oscillations. Thus a time-varying vagal-related index, the RMSSD, was calculated for each of the subsequent 30-s

segments of recovery ($RMSSD_{30s}$) (22). To smooth out transient outliers in the HRV plots (HRR vs. time in recovery), a median filter operation was performed in which each value was replaced with the median of the value as well as the preceding and following values. The first and last values were not median filtered (22).

Short-term resting HRV analysis. HRV analyses were performed on the last 5 min of the 10-min recovery period in the sitting-resting position, which ensures stability of the data. Although the respiratory rate is often controlled in HRV studies, we did not control respiratory rate in our participants because we did not want to perturb the natural return of HR to baseline. Nevertheless, respiratory rate was always in HF range (>0.15–0.50 Hz) and did not differ significantly during the last 5 min of recovery in the three conditions. Therefore, respiratory rate would have influenced the HRV indexes of each subject in the same way. The mean HR ($HR_{5-10min}$), the standard deviation of normal R-R intervals ($SDNN_{5-10min}$), the percentage of successive R-R differences >50 ms ($pNN50_{5-10min}$), and the root-mean-square difference of successive normal R-R intervals ($RMSSD_{5-10min}$) were calculated for the 5-min period. Power frequency analysis was performed sequentially with a fast-Fourier transform based on a nonparametric algorithm with a Welch window after the ectopic-free data were detrended and resampled. A fixed linear resampling frequency of 1,024 equally spaced points per 5-min period was used. The power densities in the low-frequency band (0.04–0.15 Hz) and the HF band (>0.15–0.50 Hz) were calculated from each 5-min spectrum by integrating the spectral power density in the respective frequency bands. The $SDNN_{5-10min}$, $pNN50_{5-10min}$, $RMSSD_{5-10min}$, $\ln HF_{5-10min}$ (natural-log of HF power density), the normalized HF power for the 5-min period [$HFnu_{5-10min}$; calculated as the HF/(low-frequency band + HF band) ratios], and the $HR_{5-10min}$ were retained for statistical analysis.

Energy Contribution to Each Exercise Bout

Total aerobic energy contribution. Total aerobic energy contribution for the trials was calculated as the sum of the $\dot{V}O_2$ measured during the exercise and the following 10 min plus the available oxygen stores assumed to be 2.3 ml O_2 /kg body mass (1).

Total anaerobic energy contribution. Total anaerobic energy contribution during the trials, expressed as oxygen equivalents, was calculated based on the estimated phosphocreatine and lactate contribution. The anaerobic energy contribution of phosphocreatine was estimated to be 37.0 ml O_2 /kg muscle mass (15a), whereas the anaerobic glycolytic energy contribution was calculated at 3.0 ml O_2 equivalents/kg body mass for each 1 mmol/l increase in $[La]_b$ above preexercise levels (15a), assumed to be 1 mmol/l.

Net energy expenditure during exercise. Net energy expenditure during exercise was obtained while converting total net oxygen cost of each exercise into kilocalories, assuming that the consumption of 1 liter O_2 in the human body yields 4.99 kcal, a value applicable only if the respiratory quotient (RQ) is 0.96. However, because the energy equivalent of 1 liter O_2 only varies from 4.68 to 5.05 kcal throughout the range of RQ values from 0.71 to 1.00, the minor effects of RQ would have been negligible (15a). The total net O_2 cost of each exercise trial was calculated as the sum of the total aerobic and total anaerobic energy contributions minus resting $\dot{V}O_2$, which was defined as the mean $\dot{V}O_2$ during the 10-min period preceding the maximal aerobic test (24).

Estimated mechanical power output. Mechanical power output during each of the exercise bouts was estimated from the formula given by di Prampero (15a), which takes into account the time of the exercise, the distance covered, and the body surface area of the individual. We also assumed a muscular efficiency of 25%.

Statistical Analyses

Data are presented as means \pm SE. The distribution of each variable was examined with the Kolmogorov-Smirnov and Shapiro-

Wilk normality tests. Because absolute HF power values were skewed, HF power density was transformed by taking its natural logarithm to allow parametric statistical comparisons that assume a normal distribution. A one-way ANOVA with Tukey's post hoc test was used to compare the variables of HRR_{60s} , T30, HRR_{τ} , HR_{amp} , $SDNN_{5-10min}$, $pNN50_{5-10min}$, $RMSSD_{5-10min}$, $\ln HF_{5-10min}$, and $HFnu_{5-10min}$, as well as the $HR_{5-10min}$ distributions between the three exercise bouts. For time-varying $RMSSD_{30s}$, a 3 (exercise trial) \times 20 (time) repeated-measures ANOVA was used to examine for main effects and/or interactions of intensity and time. When statistical significance was identified, a Tukey's post hoc test was used to further delineate differences between exercise trial or time. Multiple linear regressions were used to establish the respective relationships between parasympathetic indexes and exercise characteristics. Other polynomial regressions were rejected on the basis of importantly higher residuals. Adjusting calculations based on age and body mass index did not significantly change the outcomes. Moreover, because data were homogenous, we did not need to make adjustments to avoid overfitting. All statistical analyses were carried out with Minitab 14.1 software (Minitab, Paris, France) with the level of significance set at $P < 0.05$.

RESULTS

Maximal Aerobic Test

Mean $\dot{V}O_{2peak}$, HR_{max} , and $[La]_b$ were 51.6 ± 1.4 ml $O_2 \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$, 200 ± 2 beats/min, and 10.9 ± 0.9 mmol/l. Mean V_{IFT} was 18.9 ± 1.1 km/h.

Cardiorespiratory Measures During the Three Exercise Trials

All subjects successfully completed the 360-s RS and the 720-s HI exercise trials. The mean duration of the MC trial was 422 ± 21 s. As expected, mean $\dot{V}O_2$ was similar between RS and MC trials (78.4 ± 8.3 vs. $76.0 \pm 9.5\%$ $\dot{V}O_{2peak}$; $P = 0.18$) and significantly higher during the HI trial ($91.1 \pm 5.2\%$ $\dot{V}O_{2peak}$; $P < 0.001$). Mean HR for the RS trial ($87 \pm 2\%$ HR_{max}) was higher than for the MC trial ($80 \pm 3\%$ HR_{max} ; $P = 0.01$), whereas mean HR for the HI trial ($93 \pm 4\%$ HR_{max}) was greater than that for both RS and MC trials ($P < 0.001$).

Energy System Contribution and Energy Expenditure During the Three Exercise Trials

Mean (\pm SE) values of cumulated net $\dot{V}O_2$ during exercise, estimated total oxygen stores, measured $[La]_b$, $[La]_b$ O_2 equivalent, PCr O_2 equivalent, percent aerobic energy contribution, and percent anaerobic energy contribution to exercise during the trials are presented in Table 1. Mean (\pm SE) values for the net energy expenditure and total aerobic and anaerobic contribution to the RS, MC, and HI trials are summarized in Fig. 1. The net O_2 cost was marginally higher for the MC trial than for the RS trial ($P = 0.04$) but more than twice as high during the HI trial than during the RS and MC trials ($P < 0.001$). The difference in net exercise O_2 cost between the RS and MC trials was compensated for by the higher anaerobic energy contribution observed during the RS trial. As a result, the net energy expenditure was equivalent. Mean anaerobic energy contribution was comparable between the RS and HI trials and was significantly higher than the MC trial ($P < 0.001$). In addition, the net energy expenditure for the HI trial was more than double that of the RS and MC trials.

Table 1. Energy system contributions of the three exercise trials

	RS	MC	HI
Cumulated O_2 uptake, ml/kg	254.7 ± 8.8	$276.7 \pm 9.1^*$	$563.8 \pm 14.8^{*\dagger}$
O_2 stores, ml/kg	2.3 ± 0.0	2.3 ± 0.0	2.3 ± 0.0
$[La]_b$, mmol/l	10.9 ± 0.6	$3.5 \pm 0.2^*$	$11.6 \pm 0.5^\dagger$
$[La]_b$ O_2 equivalent, ml/kg	29.6 ± 1.7	$7.4 \pm 0.7^*$	$31.9 \pm 1.6^\dagger$
PCr O_2 equivalent, ml/kg	15.2 ± 0.2	15.2 ± 0.2	15.2 ± 0.2
%Aerobic energy contribution	85.2 ± 0.7	$92.4 \pm 0.3^*$	$97.3 \pm 0.4^{*\dagger}$
%Anaerobic energy contribution	14.8 ± 0.7	$7.6 \pm 0.3^*$	$2.7 \pm 0.4^{*\dagger}$

Values are means \pm SE. Estimated oxygen stores indicate myoglobin content. $[La]_b$, blood lactate concentration; PCr, phosphocreatine; RS, repeated sprint exercise trial; MC, moderate continuous exercise trial; HI, high-intensity exercise running trial. *Significant difference vs. RS ($P < 0.001$). \dagger Significant difference between HI and MC ($P < 0.001$).

Mechanical Power During the Three Exercise Trials

Figure 1, top right, illustrates the estimated mean power developed during RS, MC, and HI trials. Mean estimated power during RS was almost four times higher than the power developed during the MC trial and nearly three times higher than the power developed during the HI trial (11.4 ± 0.2 vs. 3.1 ± 0.1 and 4.6 ± 0.1 W/kg; $P < 0.001$). Power during the HI trial was also higher than during the MC trial ($P < 0.001$).

Parasympathetic Reactivation After the Three Exercise Trials

The time course of the R-R intervals after each exercise trial is illustrated in Fig. 2, and the HRR and HRV indexes are presented in Table 2. HRR_{60s} , T30, $SDNN_{5-10min}$, $pNN50_{5-10min}$, $RMSSD_{5-10min}$, $\ln HF_{5-10min}$, $HFnu_{5-10min}$, and $HR_{5-10min}$ were comparable across the RS and HI trials but were significantly lower than those shown in the MC trial ($P < 0.001$); no difference was observed across trials for HR_{amp} . HRR_{τ} was shorter after the MC exercise than after the RS trial ($P < 0.001$), and both were shorter than that shown after the HI trial ($P < 0.001$). Correlation coefficients of the regression line for T30 were 0.99 ± 0.01 for the three exercise trials. A significant time-by-exercise trial interaction was observed for $RMSSD_{30s}$ during the 10-min period after MC; however, post hoc analysis revealed that $RMSSD_{30s}$ remained constant and similar for the RS and HI trials (Fig. 3). Concerning HRR indexes, simple linear regression analyses demonstrated that the relationship was stronger between HRR_{60s} and T30 ($r = 0.84$, $P < 0.001$) than between HRR_{60s} and HRR_{τ} ($r = 0.68$, $P < 0.001$) or between T30 and HRR_{τ} ($r = 0.66$, $P < 0.001$). Relationships between HRR indexes and resting HRV indexes were all significant and moderate to strong ($0.53 < r < 0.81$). However, correlation coefficient values were higher for short-term than for long-term indexes (T30 vs. $\ln HF_{5-10min}$: $r = 0.81$, $P < 0.001$; T30 vs. $\ln HF_{5-10min}$: $r = 0.75$, $P < 0.001$; whereas HRR_{τ} vs. $\ln HF_{5-10min}$: $r = 0.53$, $P < 0.001$).

Relationship Between Parasympathetic Reactivation Indexes and Exercise Characteristics

Results from multiple linear regressions between HR-derived parasympathetic indexes and exercises characteristics are presented in Table 3. Only anaerobic process contribution

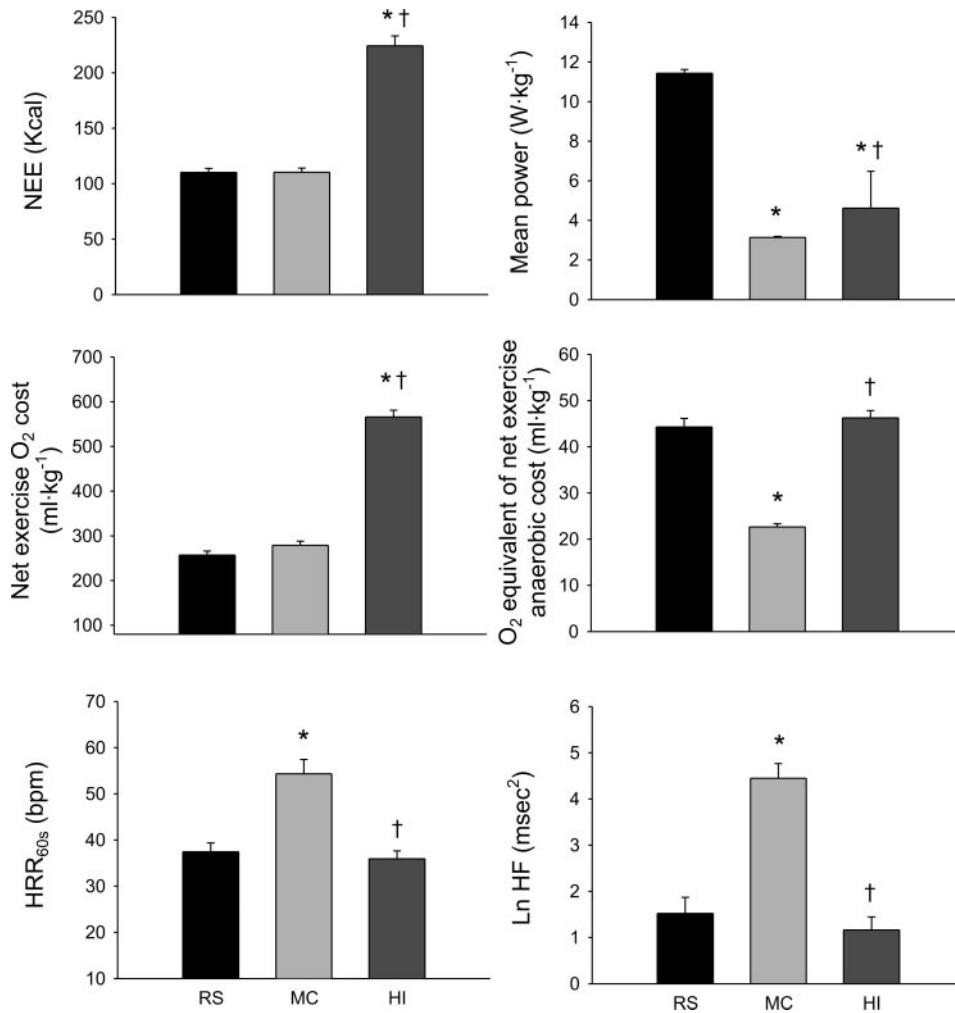


Fig. 1. Mean \pm SE net exercise energy expenditure (NEE), mean power, net O₂ cost, O₂ equivalent of net anaerobic energy cost, and parasympathetic reactivation indexes [number of heart beats recovered in 60 s after exercise cessation (HRR_{60s}) and high frequency (HF) power or R-R intervals] for each of the 3 exercise trials: repeated sprinting (RS), moderate continuous running (MC), and high-intensity running (HI) exercise. bpm, Beats/min. *Significant difference vs. RS ($P < 0.001$). †Significant difference between HI and MC ($P < 0.001$).

was significantly related to vagal-restoration indexes. Figure 4 presents the simple linear relationships between the T30 and O₂ equivalent for the anaerobic energy contribution ($r = 0.83$, $P < 0.001$).

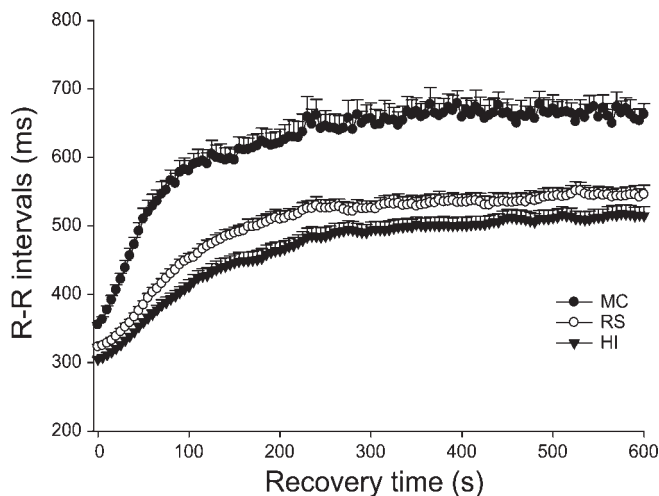


Fig. 2. Mean \pm SE R-R intervals during recovery from 3 different exercise trials: RS, MC, and HI exercise (HI). The R-R interval was calculated as the mean R-R intervals over the 5-s segment of interest. For the sake of clarity, symbols indicating significant differences vs. RS have not been added.

DISCUSSION

The aims of this study were to examine the time course of the parasympathetic reactivation using indexes of HRV after repeated sprint running and to quantify the postexercise autonomic regulation response in relation to exercise bouts of equal net energy expenditure, mean (aerobic) exercise intensity, muscular power, and anaerobic process participation. Results revealed that, compared with the MC trial of a similar energy expenditure, parasympathetic reactivation during the first 10 min of recovery was significantly more delayed after the RS exercise. Second, most of the vagal HR-derived indexes were similar after RS running than with the HI running bout of similar anaerobic energy release and double the energy expenditure. Thus we present novel data to suggest that anaerobic contribution and other factors associated with a high level of fast-twitch fiber recruitment (i.e., central stress command, catecholamine and sympathetic cotransmitter release, and lactate and H⁺ accumulation), rather than mean aerobic power or net energy expenditure, are of primary importance in determining the level of parasympathetic reactivation after repeated sprint running.

Postexercise Parasympathetic Reactivation After RS Exercise

Parasympathetic reactivation, i.e., the time course of RMSSD_{30s}, was highly impaired after the RS trial, so that the

Table 2. HR indexes of parasympathetic reactivation following each exercise trial

	RS	MC	HI
HHR _{60s} , beats/min	37.45 ± 1.92	54.34 ± 3.11*	35.94 ± 1.70†
T30, s	288.30 ± 19.90	144.11 ± 12.21*	295.20 ± 30.46†
HRR ₇ , s	74.47 ± 3.71	54.41 ± 6.39*	100.99 ± 7.02*†
HR _{amp} , beats/min	72.27 ± 2.76	76.54 ± 2.99	78.19 ± 1.94
HR _{5-10min} , beats/min	112.28 ± 2.60	91.67 ± 2.45*	119.51 ± 2.84†
SDNN _{5-10min} , ms	13.50 ± 1.13	37.28 ± 3.77*	12.03 ± 1.19†
pNN50 _{5-10min} , %	0.02 ± 0.01	1.63 ± 0.82*	0.00 ± 0.00†
RMSSD _{5-10min} , ms	4.96 ± 0.85	16.19 ± 2.93*	3.38 ± 0.35†
InHF _{5-10min} , ms ²	1.52 ± 0.35	4.45 ± 0.32*	1.16 ± 0.28†
HFnu _{5-10min}	0.19 ± 0.03	0.15 ± 0.01	0.22 ± 0.03

Values are means ± SE. HHR_{60s}, number of heart beat recovered in 60 s after exercise cessation; T30, time constant of short-time heart rate recovery; HRR₇, time constant of HR during the 10-min recovery; HR_{amp}, amplitude of HR during the 10-min recovery; HR_{5-10min}, mean HR; SDNN_{5-10min}, standard deviation of normal R-R intervals; pNN50_{5-10min}, percentage of successive R-R differences >50 ms; RMSSD_{5-10min}, root mean square of successive difference of R-R intervals; InHF_{5-10min}, high frequency power of R-R intervals; HFnu_{5-10min}, the normalized HF power. Subscript "5-10min" means calculated on the last 5 min of the recovery periods after each of the 3 exercise trials: RS, MC, and HI running. *Significant difference vs. RS ($P < 0.001$). †Significant difference between HI and MC ($P < 0.001$).

index did not change during the 10-min recovery period (Fig. 3). Although short-term autonomic regulation of HR after RS running has not been directly documented, the delayed parasympathetic reactivation that we have shown after RS exercise is consistent with what has been presented in the literature. Limited research in this area has revealed that the convalescence of the autonomic control of HR appears dependent on the exercise intensity. Indeed, HRR after moderate- to high-intensity exercise (80% $\dot{V}O_{2\text{ peak}}$) has revealed slower short-term restoration of vagal modulation than with low-intensity exercise (50% $\dot{V}O_{2\text{ peak}}$) (39, 51), even for isocaloric exercise trials (39). Moreover, in 2004, Mourot et al. (38) compared the effect of a hard interval training session (9 × 4 min at 79% $\dot{V}O_{2\text{ peak}}$, 1 min at 100% $\dot{V}O_{2\text{ peak}}$) with that of a constant load isocaloric session (48 min at 79% $\dot{V}O_{2\text{ peak}}$) and found that the return of parasympathetic activity during the first hour of recovery also appeared to be slower after the high-intensity interval training session. Although the exercise sessions in this study were completed at similar levels of total work, mean aerobic power was higher during the HI interval training session than during the MC exercise session, and anaerobic metabolites were not measured (38). Thus prior studies have not been definitive with respect to the influences that muscular power engagement, anaerobic participation, and aerobic power level have on post-exercise parasympathetic reactivation.

Effect of Muscular Power Engagement and Anaerobic Process Participation on Parasympathetic Reactivation

Parasympathetic reactivation was significantly lowered after the RS trial than that shown with MC exercise. Mean aerobic power and net energy expenditure were comparable between the RS and MC trials, whereas muscular power and anaerobic energy contribution were different. The present study design and the mathematical adjustments made enabled us to make a clear distinction between the factors that are of primary importance in determining parasympathetic reactivation level. Multiple linear regression analyses revealed, when considering

data of the three exercise trials together, that anaerobic contribution but not muscular power engagement is significantly related to all of postexercise parasympathetic reactivation indexes (Table 3 and Fig. 4). Therefore, we suggest that the impaired parasympathetic activity after RS exercise may be primarily related to the high anaerobic process participation and associated plasma metabolites. Although the incidence of lactate accumulation on postexercise autonomic regulation has still not been directly evaluated, the effect of high-intensity repeated muscular contraction has been recently examined. Heffernan et al. (27) found that a resistance workout (10-repetition maximum test) perturbed the parasympathetic reactivation of HR significantly more than did an aerobic exercise session (30 min cycling at 65% $\dot{V}O_{2\text{ peak}}$). Muscular power production during the RS trial in the present study was high enough to be compared with that commonly performed during resistance training, and the anaerobic contribution might have been even greater. This slowing of vagal restoration after resistance training or RS training may thus be related to the heightened sympathetic activity that occurs during exercise (43) and the persistent elevation of adrenergic factors (25) and local metabolites during recovery (epinephrine, norepinephrine, H⁺, lactate, P_i, etc.) (21, 47). For example, Perini et al. (40, 41) reported strong and significant linear relationships between HRR kinetics and norepinephrine plasma levels. Nevertheless, postexercise HRR is a multifaceted phenomenon. Various investigations have demonstrated complex interactions between the sympathetic and vagal systems with respect to HR regulation (30, 32, 35, 36), resulting in reduced (35) or amplified vagal stimulation (30, 32). As recently demonstrated by Sunagawa and coworkers (30, 35, 36), in the presence of sympathetic activation, HR response to vagal stimulation essentially depends on the type and site of adrenergic receptors most selectively activated under a given condition. For example, Sunagawa et al. showed that the dynamic HR response to vagal stimulation can be attenuated via activation of α -adrenergic receptors on the preganglionic and/or postganglionic cardiac vagal nerve terminals by high plasma norepinephrine

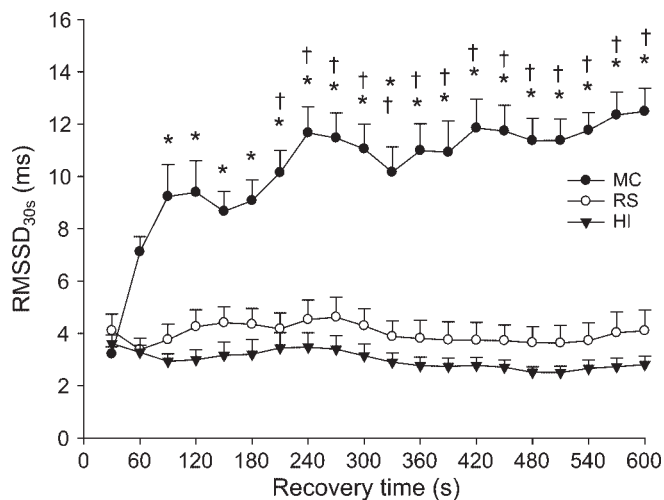


Fig. 3. Mean ± SE root mean square of successive difference of the R-R intervals measured on successive 30-s segments (RMSSD_{30s}) during the 10-min recovery period, as calculated for each of the 3 exercise trials: RS, MC, and HI exercise. *Significant difference vs. RMSSD_{30s} at the end of exercise ($P < 0.001$). †Significant difference vs. RS ($P < 0.001$).

Table 3. Relationships between postexercise parasympathetic reactivation indexes and mechanical and metabolic exercise characteristics

	Constant		Aerobic Contribution		Anaerobic Contribution		NEE		Muscular Power		Overall Relationship	
	β	<i>P</i>	β	<i>P</i>	β	<i>P</i>	β	<i>P</i>	β	<i>P</i>	<i>r</i> ²	<i>P</i>
HHR _{60s}	74.45 ± 4.92	<0.001	0.01 ± 0.01	0.97	-0.43 ± 0.22	0.05	-0.08 ± 0.07	0.27	-0.83 ± 0.61	0.19	0.60	<0.001
T30	-22.48 ± 31.16	0.48	0.03 ± 0.15	0.86	2.99 ± 1.33	0.03	0.62 ± 0.44	0.17	9.12 ± 3.78	0.02	0.73	<0.001
HRR _τ	8.59 ± 11.97	0.48	0.08 ± 0.06	0.19	1.16 ± 0.52	0.03	0.1 ± 0.17	0.96	-0.85 ± 1.47	0.57	0.57	<0.001
HR _{5-10min}	68.43 ± 6.87	<0.001	0.04 ± 0.03	0.26	0.63 ± 0.29	0.03	-0.01 ± 0.09	0.89	0.58 ± 0.83	0.49	0.55	<0.001
RMSSD _{5-10min}	29.64 ± 4.21	<0.001	-0.01 ± 0.02	0.73	-0.12 ± 0.18	0.49	-0.06 ± 0.06	0.2	-0.89 ± 0.50	0.09	0.47	<0.001
InHF _{5-10min}	7.88 ± 0.74	<0.001	-0.01 ± 0.01	0.49	-0.04 ± 0.03	0.24	-0.01 ± 0.01	0.22	-0.23 ± 0.09	0.06	0.65	<0.001

β -Coefficient values are means ± SE. NEE, net exercise energy expenditure. *P* values were from multiple linear regressions.

levels (35) but not by cardiac postganglionic sympathetic nerve stimulation (36). In contrast, elevated cardiac sympathetic nerve activity can augment the dynamic HR response to vagal nerve stimulation via activation of the postjunctional β -adrenergic cascade (30, 32). Unfortunately, in the present study, we did not measure any objective indexes of muscular power engagement (i.e., EMG measurement) or system stress (i.e., sympathetic muscle nerve activity, hormones, and muscle metabolite sampling) to assess objectively the level of contribution this may have had on HRV markers. Nevertheless, because high plasma norepinephrine levels have been repeatedly observed after high-intensity or sprint exercise (5, 40, 41), we put forth that the poor parasympathetic reactivation level observed after RS and HI exercise in the present study might be partly attributed to high sympathetic activity and associated metabolite persistence. Additionally, the potential presence of postexercise hypotension after acute exercise could have stimulated the arterial baroreflex, elevating sympathetic outflow and delaying the restoration of vagal tone. Changes in plasma volume have been reported to alter cardiac autonomic balance (48), and the occurrence of a plasma volume shift during the exercise trials in the present study could have altered the parasympathetic reactivation indexes.

Effect of Net Energy Expenditure on Parasympathetic Reactivation

All HR vagal-related indexes were different between the RS and MC trials, whereas these two exercises were equivalent with respect to their net energy expenditure. Moreover, a doubling of the net energy expenditure while a similar level of anaerobic energy contribution was maintained (RS vs. HI) was not associated with any further lowering of most indexes of parasympathetic reactivation (9 of the 10 indexes considered; Table 2). We also cannot exclude the possibility that our 10-min postexercise recording duration may have been too short to reveal differences between RS and HI in stationary HRV indexes. Nevertheless, these findings conform with the studies of Parekh and Lee (39) and Mourolet et al. (38), which have shown that the total work or energy expenditure is not the main factor influencing parasympathetic reactivation after exercise. It should be noted, however, that HRR_τ was significantly different between the RS and the HI trials, whereas HRR_{60s} and T30 were not significantly different. These differences, together with the moderate correlation coefficients that we found between short-term recovery indexes (HRR_{60s} and T30) and HRR_τ, confirm previous investigations that have shown that HRR_τ, in contrast to T30 or HRR_{60s}, is influenced by additional factor(s) and not only vagal modulation (29, 40–42).

When the effects of parasympathetic vs. complete autonomic blockade on T30 and HRR_τ were compared, Imai et al. (29) found that T30 was primarily mediated by vagal activity, whereas HRR_τ explained not only the prompt parasympathetic reactivation (rapid initial decrease in HR) but also the level of sympathetic activity (slow second decrease). Perini et al. (41) found that, in the absence of autonomic control in heart-transplanted recipients, the HR decrease began only after 50 s (which corresponds to the slow second phase) and was related to plasma norepinephrine clearance. Thus the present differences shown between our RS and HI trials suggest that HI exercise might be associated with a higher persistence of postexercise sympathetic activity compared with that shown with RS exercise, despite a similar time course of vagal activity restoration (similar HRR_{60s} and T30). This possible higher norepinephrine plasma concentration after the HI vs. the RS trial could in part be attributed to differences in exercise bout duration between the two exercises, as sprint times during the RS trial were almost 10-fold shorter than the intermittent runs during the HI trial. Exercise bout duration has been shown to

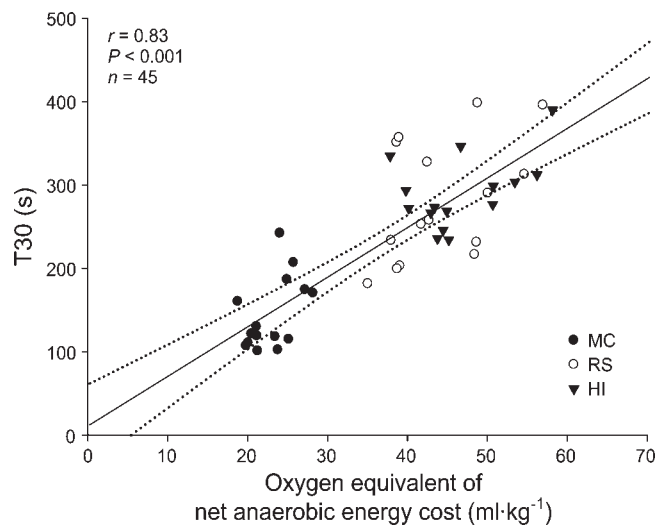


Fig. 4. Relationship between time constant of short-time heart rate recovery (T30; see MATERIALS AND METHODS) and oxygen equivalent of the net anaerobic energy cost of each of 3 exercise trials. Dotted lines represent 95% confidence intervals.

be essential in determining the level of muscle deoxygenation and consequently the level of anaerobic participation and plasma catecholamine accumulation (12).

Implications for the Clinical Use of RS Training

Although evaluation of the influence that RS exercise has on autonomic function is crucial in sedentary individuals and patients, we intentionally recruited moderately trained subjects for the present study because we expected that they would likely be able to cope better with this innovative experimentation. Trained subjects often display high HRR indexes (7), so the present results should be viewed with caution when inference is made as to what might occur in a sedentary subject. Whether individuals with low-activity levels show a similar response to RS exercise needs to be confirmed.

In summary, the present study has shown that short-term parasympathetic reactivation is impaired after repeated sprint running and that anaerobic contribution rather than muscular engagement and net (aerobic) energy expenditure appears to be of primary importance in determining the level of parasympathetic reactivation. This finding may be of particular importance for clinicians wishing to prescribe sprint training to clinical populations. Longitudinal studies that examine the effect of long-term repeated sprint training on autonomic control of the heart, parasympathetic reactivation, and cardiovascular risk prognostic are warranted.

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