Tapering for Marathon and Cardiac Autonomic Function

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Key word
- endurance performance
- cardiac autonomic activity
- heart rate recovery
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- parasympathetic reactivation

Abstract

The purpose of this study was to investigate changes in post-exercise heart rate recovery (HRR) and heart rate variability (HRV) during an overload-tapering paradigm in marathon runners and examine their relationship with running performance. 9 male runners followed a training program composed of 3 weeks of overload followed by 3 weeks of tapering (−33±7%). Before and after overload and during tapering they performed an exhaustive running test (T_{lim}). At the end of this test, HRR variables (e.g. HRR during the first 60 s; $HRR_{60\,s}$) and vagal-related HRV indices (e.g. $RMSSD_{5-10\,min}$) were examined. T_{lim} did not change during the overload training phase (603±105 vs. 614±132 s; P=0.992), but increased (727±185 s; P=0.035) during the second week of tapering. Compared with overload, $RMSSD_{5-10\,min}$ (7.6±3.3 vs. 8.6±2.9 ms; P=0.045) was reduced after the 2nd week of tapering. During tapering, the improvements in T_{lim} were negatively correlated with the change in $HRR_{60\,s}$ (r=−0.84; P=0.005) but not $RMSSD_{5-10\,min}$ (r=−0.21; P=0.59). A slower HRR during marathon tapering may be indicative of improved performance. In contrast, the monitoring of changes in HRV as measured in the present study (i.e. after exercise on a single day), may have little or no additive value.

Introduction

Reduced training load (tapering) during the preparation for an important competition aims to minimize fatigue without restricting the positive training effects. The overload-tapering paradigm is characterized by an initial increase in training load for a time period of several weeks, followed by a reduction of training load during a time period of 1–4 weeks [39,40,54]. Taper-induced performance increase is generally greater when the taper phase is preceded by an overload period with increased training load (up to 50%). Maintaining high training intensities as well as a high frequency (>80% of normal) during the taper phase seems to be important [9,39,40,54]. Training intensity seems to be the key factor for optimized performance prior to a main competition [54]. Race preparation according to current tapering recommendations can lead to performance gains from 2 to 9% [9,54]. To date, except for competition results, there is no practical and reliable method for measuring the effect of tapering on the athletes' training status, fatigue and performance.

Searching for a minimally invasive and minimally disturbing method to optimize preparation for peak performance has always been a matter of interest in exercise physiology and sports medicine [7]. The autonomic nervous system (ANS) function plays an important role in the training responses and in the functional adaptations occurring from a given training stimulus [7,37].
The mono-exponential decrease in heart rate after maximal exercise is primarily modulated by the ANS, and short-term post-exercise heart rate recovery (HRR) can therefore be used as a marker of cardiac parasympathetic outflow [29]. Heart rate variability (HRV) measurements are also well-accepted procedures for the assessment of the cardiac vagal function [12,16]. In this context, HRV monitoring has been proposed as a valuable tool for detecting the complex changes in ANS activity in athletes [10,11,37,48].
Consequently, either post-exercise HRR, resting-HRV, or post-exercise HRV have been suggested as indirect markers of cardiac autonomic control and may offer practical and simple ways of quantifying the physiological effects of training [8,16,17,24,32]. The ability of these indices to predict endurance performance in the field and

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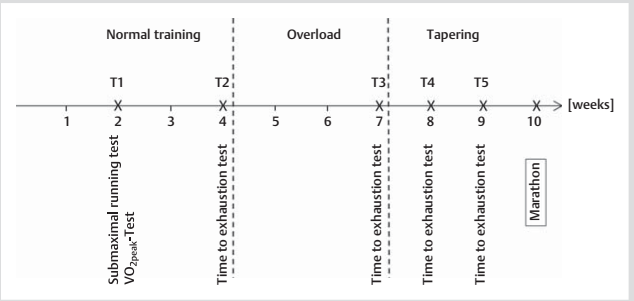

Fig. 1 Study design. Testing at time point T1 was composed of a "submaximal running test" and a $\dot{V}O_{2peak}$ test. At T2, T3, T4 and T5, the athletes performed an identical "Time to exhaustion test" as well as heart rate recovery (HRR) and heart rate variability (HRV) assessment.

to monitor positive or negative adaptations could help in the design of individualized HRR- or HRV-guided training programs for athletes [10,31,44].

However, the relationship between training load, fatigue, performance and changes in HR-derived indices [25,26,35,38,42,55–57] has led to conflicting results. Hedelin et al. [26] reported, in an overtrained cross-country skier, reduced competition performance and decreased profile of mood states, along with an increased cardiac parasympathetic modulation. In contrast, total power spectral density of HRV was decreased in 5 overtrained female endurance athletes undergoing heavy training over a 6 to 9-week period [57]. In another study, Hedelin et al. [25] reported no significant change in HRV in 9 overexerted canoeists after increasing training load by 50% over a 6-day training camp. These discrepancies between results are probably due to the differences in methodology [45] or protocols, such as the nature of the overload period, and/or the number and performance level of the athletes involved in the different studies.

At rest or while exercising at moderate intensity, aerobically-trained athletes have a greater cardiac parasympathetic activity compared to untrained subjects [18,50,51]. In moderately-trained athletes, Buchheit et al. [15] reported a positive relationship between changes in performance and parasympathetic reactivation following an 9-week training program. In contrast, well-trained elite athletes have been reported to exhibit decreased vagal-related HRV indices following a large-volume training program [28]. Iwasaki et al. [30] and Manzi et al. [37] have reported an inverted U-shaped relationship between training load and vagal-related HRV indices in marathon runners. Pichot et al. [42] showed that HRV may follow changes in training load in untrained athletes, with reduced training load being associated with increased vagal-related HRV indices.

Few authors have investigated the relationship between reduced training load and changes in cardiac parasympathetic activity. Atlaoui et al. [2] found during a tapering regime in highly-trained swimmers a positive correlation between resting cardiac parasympathetic activity and performance. Le Meur et al. [34] divided a group of trained male triathletes into a normal training and an intensified training group who performed 3 weeks of overload followed by 1 week of taper. The overreached triathletes showed an increase in cardiac parasympathetic activity, whereas performance in an incremental performance test had decreased. However, these responses were reversed during the taper. Despite its potentially high practical interest, the relevance of HR-derived indices for monitoring changes in fatigue and/or performance in athletes during an effective training program is still unclear. To improve our understanding of the usefulness of these non-invasive markers for monitoring training adaptations, we investigated changes in running performance and parasympathetic reactivation following maximal exercise in response to an overload-tapering paradigm in well-trained marathon runners. Therefore, the aims of the present study were to (1) examine the respective changes in HRR and HRV indices during a 3-week overload followed by a 3-week tapering period in well-trained runners and (2) assess the possible relationships between these indices and running performance (time to exhaustion). Based on previous research, it was hypothesized that, during the preparation for a marathon, post-exercise parasympathetic reactivation would be slower after a 3-week overload period but increased during a subsequent 3-week taper period [2,42].

Methods

Subjects

11 well-trained marathon runners satisfied the inclusion criteria (male, personal best marathon performance under 3 h, no injuries in the last 3 months, absence of clinical signs or symptoms of infection, absence of cardiovascular diseases or injuries and a minimum weekly training dose of 5 running sessions) and gave written informed consent to the study, which was approved by the internal review board of the Swiss Federal Institute of Sport and was performed in accordance with the ethical standards of the IJSM [23]. Throughout the normal training phase, the athletes had 6–7 running sessions per week (training volume of 8.2 ± 1.4 h). Each runner had a history of at least 5 years of training for running competitions. During the study period, one runner dropped out due to injury and another one due to illness. Therefore, 9 subjects (34.6 ± 5.7 years; 180 ± 9 cm; 69.0 ± 6.3 kg) completed all measurements.

Experimental design

An outline of the study design is shown in ○ **Fig. 1**. Athletes completed a 10-week study period before participating at the Lucerne Marathon in Switzerland. They started with a 4-week training phase with record of their "usual" training regimen (normal training). In the subsequent overload phase, training load was increased for 3 weeks by 23 ± 10%. During the overload phase, each runner had one additional 1-h high intensity running session per week and the long run was prolonged by 30 min. During the tapering phase, the number of high intensity training sessions was kept similar to that of the previous (overload) phase, while the training volume was reduced gradually (1 session less per week and reduction in the average duration of the other sessions). The mean reduction in volume between the overload and tapering phase was 33 ± 7%. During the 10-week study period, athletes kept a training log and a history of health status and nutrient intake. The athletes had to record session-RPE within 30-min of finishing their workout [20]. A series of different tests was completed at different time points (T1-T5, ○ **Fig. 1**). At T1, athletes performed a submaximal running test and a maximal oxygen uptake ($\dot{V}O_{2peak}$) test. These tests were used to calculate the running speeds for the subsequent performance tests. At T2, T3, T4 and T5, the athletes performed a time to exhaustion test (T_{lim}) to examine performance changes during the study period and post-exercise HRR and parasympathetic reactivation was analysed for 10 min.

The runners were advised to maintain the same preparation procedure (e.g. habitual nutrition plan; warm-up; hydration) before each testing day. Preceding all tests, the runners abstained from alcohol and caffeinated beverages and refrained from medium intensity (48 h) and heavy training (24 h) prior to all testing days. The athletes completed their test at the same hour (±1 h) between 09:00 and 16:00 h to avoid possible circadian influences on the parameters. Room temperature (18–19 °C) and humidity (38–40 %) were kept constant for all tests.

Training intervention

As recommended [9, 39, 40, 54] during tapering, the reduction in load was mainly due to the decrease in volume partially counterbalanced by the increase in intensity. Intensity of training and the number of workouts per week were kept nearly at the same level as during the overload phase to ensure positive effects of the taper and a possible increase in performance [40, 49, 54]. The training log was sent weekly with an Excel-sheet by e-mail to the main investigator for analysis. Feedback with light modifications to the training program was then provided by the investigators. Training loads from week 10–7 prior to the marathon were averaged to one mean value "normal training". Training loads from week 6–4 prior to the marathon were averaged to one mean value "overload". During the taper phase, summarized loads of the first week were recorded as "TP1" value and similarly as "TP2" for the 2nd week. The last tapering week was recorded without the load corresponding to the marathon race (i.e., 6 days adjusted to 7 by linear extrapolation).

Submaximal running test (T1)

The subjects started with a general standardized warm-up for 5 min at 8 $km \cdot h^{-1}$. Next, a blood sample was drawn from the earlobe and analysed for pre-testing lactate ([La]). The runners then continued to run at 9 $km \cdot h^{-1}$ for 5 min followed by resting for 30 s. This procedure was repeated with 11 $km \cdot h^{-1}$, 13 $km \cdot h^{-1}$ and 15 $km \cdot h^{-1}$. During the 30-s rest, a blood sample was taken from the earlobe for [La] measurement, and RPE was indicated by the subject using a scale from 6 to 20 [5]. [La] was analysed with a standard analyser (Hitado Super GL; Dr. Müller Gerätebau GmbH, Freital, Germany) using 10-µl open-end capillaries. Respiratory gas exchanges were measured breath-by-breath during the entire submaximal running test (Jaeger Oxycon Pro; Jaeger, Hoechberg, Germany). Average O_2 consumption of the last 3 min of each running stage was used for the calculation of the individual relationship between oxygen uptake and running speed, as usually performed in our laboratory [58].

The analyser was calibrated before each use with 2 samples of a known concentration. Calibration procedures were performed before each test, according to the manufacturer's recommendations and also for the location of the laboratory at 950 m above sea level. The respiratory analysis system was calibrated first using a gas of known O_2 and CO_2 concentrations and then using ambient air, with partial O_2 composition being assumed to be 20.9 %. Calibration of the turbine flow-meter of the Oxycon Pro was performed with an automated program.

$\dot{V}O_{2peak}$-Test (T1)

15 min after cessation of the submaximal running test, the subjects performed a $\dot{V}O_{2peak}$ test on a treadmill. Starting at 7 $km \cdot h^{-1}$ running velocity was increased by 0.5 $km \cdot h^{-1}$ every 30 s until voluntary exhaustion [19]. During the tests, gas exchange data were collected continuously and recorded as means for every 30 s. The following variables were measured and analysed: oxygen uptake ($\dot{V}O_2$), respiratory exchange ratio (RER), ventilation ($\dot{V}E$), breathing frequency (BF), maximal lactate ($[La]_{max}$) and maximal velocity. $\dot{V}O_{2peak}$ was defined as the highest mean $\dot{V}O_2$ value obtained for any continuous period of 30 s. Maximal HR (HR_{max}) (Suunto dual belt; Helsinki, Finland) was defined as the highest value during the test.

Time to exhaustion test at 95 % $v\dot{V}O_{2peak}$ (T2_T5)

In this test, the athletes ran at an individual running speed corresponding to 95 % of the velocity associated with $\dot{V}O_{2peak}$ ($v\dot{V}O_{2peak}$) until exhaustion. $v\dot{V}O_{2peak}$ was calculated by the individual relationship of oxygen uptake and running speed by extrapolation of the 4 values recorded at submaximal speeds (9, 11, 13 and 15 $km \cdot h^{-1}$) and the $\dot{V}O_{2peak}$ recorded during the incremental test as described previously [58]. Of interest is that this protocol is the one recommended by Swiss Olympics and used in most Swiss elite athletes. The testing protocol was planned with a running time that should not exceed 20 min for not interfering with the training program. As T_{lim} at $v\dot{V}O_{2peak}$ is related to the anaerobic threshold [3] and endurance time plotted against velocity exhibits a hyperbolic shape, it can be predicted that running time to exhaustion at 95 % $v\dot{V}O_{2peak}$ should be around 10±5 min in well-trained runners. The subjects started running at 60 % $v\dot{V}O_{2peak}$ and ran continuously for 10 min on the treadmill followed by 30 s of rest (for blood sampling and RPE collection). They then continued running for eight min at 80 % $v\dot{V}O_{2peak}$. After 5 min of rest, the subjects then performed a maximal running test to exhaustion at 95 % $v\dot{V}O_{2peak}$. $\dot{V}O_{2peak}$ was defined as the highest 30 s-mean $\dot{V}O_2$ value obtained at exhaustion. Running energy cost (EC, $ml \cdot min^{-1} \cdot km^{-1}$) was calculated from the $\dot{V}O_2$ of the last 5 min during the stage of 60 % $v\dot{V}O_{2peak}$. In all sessions, the subjects were encouraged to perform to their best effort and required to immediately sit for 10 min at T_{lim} test cessation. The time duration between the end of exercise and sitting was less than 5 s.

Resting HR and HRV

The runners sat down and rested for 10 min in a quiet environment in the laboratory prior to the T_{lim} test. For both HR and HRV measurements, the first 2 min of recording were disregarded due to lack of stability, and the last (stable) eight minutes of the resting phase were analysed. While this 8-min period for HRV analysis is slightly longer than that usually employed in the literature (i.e., 5-min), the recording length is unlikely a major issue when dealing exclusively with time-domain HRV indices as in the present study [22]. Additionally, if we consider the possible small fluctuations in ANS activity over time, a longer recording period may reflect a more accurate representation of the actual cardiac autonomic activity. The Suunto T6 watch has recently shown good validity compared to a mobile ECG-system [59]. Standard custom software from the company was used to generate average values for HR and RR-intervals, which were exported into an Excel file. HRV was analysed with Kubios HRV (Version 2.0, University of Kuopio, Finland).

Post-exercise HRR and parasympathetic reactivation

RR intervals were recorded, and HRR was assessed during the recovery period after cessation of the T_{lim} test and analysed as follows: (1) the first 30 s (from the 10th to the 40th s) of HRR via semi-logarithmic regression analysis (T30) as proposed by Imai et al. [29]; (2) the time constant of the HR decay (HRRτ) by fitting

	Normal training	Overload	Taper 1st week	Taper 2nd week
training load	1826 ± 293	2227 ± 460 ##	1938 ± 241 ^^	1686 ± 227 ^^ *
T_{lim} (s)	603 ± 105	614 ± 132	618 ± 132	727 ± 185 # ^ *
$\dot{V}O_{2peak}$ ($ml \cdot min^{-1} \cdot kg^{-1}$)	59.5 ± 2.9	60.3 ± 4.1	58.9 ± 2.8	60.4 ± 2.6
$\dot{V}E_{peak}$ ($l \cdot min^{-1}$)	143.0 ± 18.2	141.8 ± 15.4	137.8 ± 19.0 #	139.4 ± 18.0
BF_{peak} (min^{-1})	57.6 ± 7.7	57.0 ± 8.5	56.0 ± 9.5	55.6 ± 7.6
$[La]_{peak}$ ($mmol \cdot l^{-1}$)	6.8 ± 2.3	6.4 ± 2.4	5.8 ± 1.8 #	5.6 ± 1.5 ##
EC 60 % $v\dot{V}O_{2peak}$ ($ml \cdot min^{-1} \cdot km^{-1}$)	201.9 ± 11.8	200.4 ± 11.4	195.1 ± 14.9	202.8 ± 12.8 *
HR_{Rest} (bpm)	52.7 ± 9.0	52.9 ± 10.5	50.9 ± 10.1	52.3 ± 7.9
HR_{peak} (bpm)	178.0 ± 13.0	177.7 ± 12.3	176.3 ± 12.5	179.6 ± 11.3 *
$HR_{60\,s}$ (bpm)	119 ± 21.3	118.9 ± 22.6	115.7 ± 21.9	123.4 ± 19.6 *
$HR_{600\,s}$ (bpm)	88.2 ± 10.6	88.2 ± 11.0	88.8 ± 12.4	91.9 ± 12.0
$HR_{5-10\,min}$ (bpm)	89.0 ± 11.3	89.1 ± 11.1	88.1 ± 12.9	93.0 ± 11.7 *
$HRR_{60\,s}$ (bpm)	59.0 ± 12.3	58.8 ± 12.9	60.7 ± 13.6	56.1 ± 11.4 *
HRR_{600s} (bpm)	89.8 ± 10.9	89.4 ± 9.1	87.6 ± 8.2	87.7 ± 9.7
T30 (s)	126.6 ± 52.5	115.4 ± 49.8	116.9 ± 62.5	126.6 ± 69.1
HRRτ (s)	55.0 ± 16.2	54.7 ± 21.4	53.7 ± 18.2	55.9 ± 17.9
$RMSSD_{Rest}$ (ms)	63.5 ± 24.4	67.7 ± 34.2	66.8 ± 29.2	64.5 ± 29.7
$RMSSD_{5-10\,min}$ (ms)	8.1 ± 2.7	8.6 ± 2.9	8.1 ± 3.7	7.6 ± 3.3 ^

Table 1 Performance and heart rate recovery variables with respect to the different training phases.

Values are mean ± SD. T_{lim}: running time to exhaustion (s) at 95 % of the velocity associated with $\dot{V}O_{2peak}$. $\dot{V}E_{peak}$: peak ventilation; BF_{peak} = maximum breathing frequency. $[La]_{peak}$: peak lactate. EC: energy cost. HR_{peak}: peak heart rate; $HR_{60\,s}$: heart rate 60 s after exercise. $HR_{600\,s}$: heart rate 600 s after exercise. $HRR_{60\,s}$: number of heart beats recovered within 60 s after exercise. $HRR_{600\,s}$: number of heart beats recovered within 600 s after exercise. T30: semi-logarithmic regression analysis from the 10th to the 40th s of heart rate post exercise. HRRτ: time constant of heart rate recovery. RMSSD: square root of the mean squared differences between successive RR-intervals

#: P < 0.05, ##: P < 0.01 for differences with normal training

^:P < 0.05, ^^: P < 0.01 for differences with overload

*:P < 0.05, **:P < 0.01 for differences with taper 1st week

the 10-min post-exercise HR recovery into a first-order exponential decay curve; and (3) the absolute difference between the final 5-s averaged HR at test completion and the HR recorded at 60 s and 600 s during recovery ($HRR_{60\,s}$, $HRR_{600\,s}$). Post-exercise HRV was assessed as described previously [21]. Calculated values were (1) the time course of RMSSD on successive 30-s segments ($RMSSD_{30\,s}$) (moving window) and (2) the RMSSD from the 5th to the 10th min during seated recovery ($RMSSD_{5-10\,min}$). While we acknowledge that the period of analysis differed in length between resting (i. e., 8 min) and post-exercise HRV (i. e., 5 min) conditions, this is unlikely to affect to present results since those data were not compared directly. RMSSD reflects parasympathetic activity of the cardiac autonomic nervous system [53] and is therefore frequently used to monitor changes in ANS response to training [46]. To standardize testing conditions (i. e., to avoid breathing perturbations), participants were not allowed to speak or drink during the 10-min recovery period. Respiratory rate was spontaneous for practicality during field-based measurements, and because there is little difference in parasympathetic-related HRV indices during controlled or spontaneous breathing [4]. Importantly also, RMSSD has much greater reliability than other spectral indices [1], particularly during 'free-running' ambulatory conditions [41].

Statistical analysis

Data in the text and in the tables are presented as mean ± SD. Each variable was tested with one-way repeated measure analysis of variance (RM ANOVA) completed by post-hoc Tukey test to locate statistical differences (SigmaPlot 11.0; Systat Software, Inc, San Jose, CA). Two-way repeated measures trials [normal training; overload; TP1; TP2] x time [20 × 30-s windows] ANOVA was used for the analysis of $RMSSD_{30\,s}$. Differences were considered statistically significant when $P \leq 0.05$. Pearson correlation coefficients were calculated to test for significant associations between performance, HRR and HRV parameters. Pearson correlations (r, 90 % confidence limits, CL) between per cent changes in HRV, HRR, training load, performance and related variables were calculated for each training phase.

Results

Marathon results

Eight runners successfully completed the marathon at the end of the third taper week. 6 athletes reached their personal best time. The average time was 169.5 ± 9.9 min. One athlete was forced to drop out halfway due to gastrointestinal disturbances.

Training intervention

Training load data are shown in **Table 1**. The compliance to training guidelines was satisfying as shown by the per cent changes in training load (23 % increase during overload; reduction compared to the overload period was 12 % (TP1), 22 % (TP2) and 64 % during the last tapering week).

$\dot{V}O_{2peak}$ Test (T1)

The $\dot{V}O_{2peak}$ was 60.9 ± 2.8 $ml \cdot min^{-1} \cdot kg^{-1}$, $v\dot{V}O_{2peak}$ was 17.7 ± 1.0 $km \cdot h^{-1}$, $[La]_{max}$ was 5.7 ± 1.5 $mmol \cdot l^{-1}$ and HR_{max} was 182 ± 13 bpm. Calculated individual speed parameters were 10.7 ± 0.6 $km \cdot h^{-1}$ for the 10-min stage at 60 % $v\dot{V}O_{2peak}$, 14.2 ± 0.8 $km \cdot h^{-1}$ for the 8-min stage at 80 % $v\dot{V}O_{2peak}$ and 16.9 ± 1.0 $km \cdot h^{-1}$ for the final phase at 95 % $v\dot{V}O_{2peak}$.

T_{lim} test at 95 % $v\dot{V}O_{2peak}$ (T2_T5)

Table 1 shows the results of the repeated T_{lim} test for each training period (T_{lim}, HR_{peak}, $\dot{V}E_{peak}$, BF_{peak}, $[La]_{max}$). HR_{peak} did not change during overload (P = 0.995) and at TP1, but an increase was found at TP2 (P = 0.024). A significant time vs. percent

change effect was observed for T_{lim} between normal training and TP2 (P=0.019), overload and TP2 (P=0.035), as well as between TP1 and TP2 (P=0.044).

Post-exercise HRR and parasympathetic reactivation

All HRR and HRV values are presented in **Table 1**. A significantly slower HRR was found at TP2 compared with TP1. Average HR from the 5th to the 10th min during exercise recovery was higher (P=0.037) in TP2 than in TP1. HRR_{60s} (P=0.017) was significantly lower at TP2 than TP1. $RMSSD_{5-10\,min}$ was reduced (P=0.045) at TP2 compared with the overload measurement. Parasympathetic reactivation ($RMSSD_{30}$) for most subjects had a peak between the first and third min of the recovery phase (**Fig. 2**). A significant $RMSSD_{30}$ measure vs. time interaction was reported with differences located between 90s – 120s between overload and TP2 (**Fig. 2**).

Changes between variables

In the present study, there was no significant association between training load and any other recorded variable during overload and tapering. Of interest is the very large and negative relationship between the changes in HRR_{60s} and T_{lim} from the end of the overload training to the 2nd week of tapering (r=−0.84, 90% CL (−0.50;−0.96); P=0.005) (**Fig. 3a**). A very large correlation was also observed between the changes in HRRτ and in T_{lim} for the period between overload and TP2 (r=0.69, 90% CL(0.17;0.91); P=0.039) (**Fig. 3b**). There was, however, no significant correlation between $RMSSD_{5-10min}$ and either training load (r=−0.09, P=0.82) or T_{lim} (r=−0.21, P=0.59). There was no significant relationship when considering the other variables.

Discussion

The main findings of this study were:

1. Indices of parasympathetic reactivation were sensitive to training load manipulation, as evidenced by the significant decreases in HRR and RMSSD observed during the tapering period.
2. Performance was only significantly improved (e.g. increase in T_{lim}) after the second week of taper, and these changes were very largely correlated with those in HRR. In contrast, changes in performance did not correlate with changes in post-exercise vagal-related indices.

Our results support the view that in endurance athletes known to have a high parasympathetic activity [18,50,51], an efficient tapering phase is associated with decreased parasympathetic activity and/or increased sympathetic tone [28,30,37,46], as evidenced here by the slower HRR indices/lower RMSSD values. These findings suggest that enhanced performance is not necessarily associated with faster HRR [32,33]. In endurance athletes, a more balanced sympathovagal activity seems to be favourable to aerobic performance [28,30,37].

Training intervention

In the present study, the 18% improvement in T_{lim} during tapering clearly shows that the latter was efficient. Tapering aims to reduce fatigue while maintaining fitness during the last weeks prior a main competition [39,40,54]. In these lines, the improved recovery of overload-induced fatigue during the second tapering week could partly explain the significant increase in T_{lim} at the end of the tapering phase.

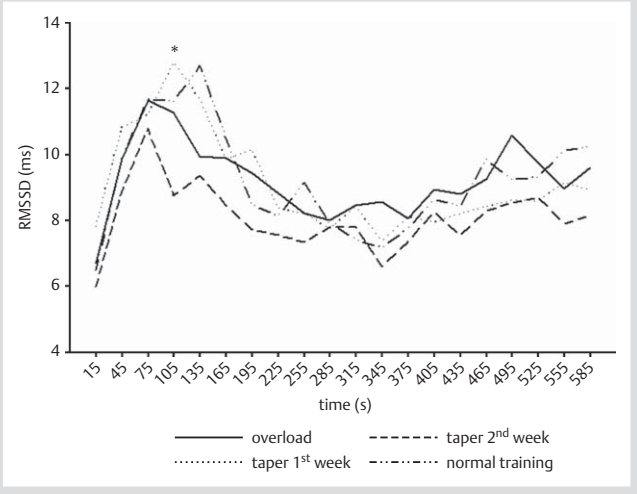

Fig. 2 Root mean square of successive differences of RR-intervals measured on successive 30-s segments ($RMSSD_{30s}$) during the 10 min recovery period after running to exhaustion at 95% of the velocity associated to $\dot{V}O_{2peak}$. Values of repeated trials (normal training, overload, taper 1st week, taper 2nd week) are plotted without SD for clarity. * P=0.05 for group vs. time interaction between overload and taper 2nd week (90–120s)

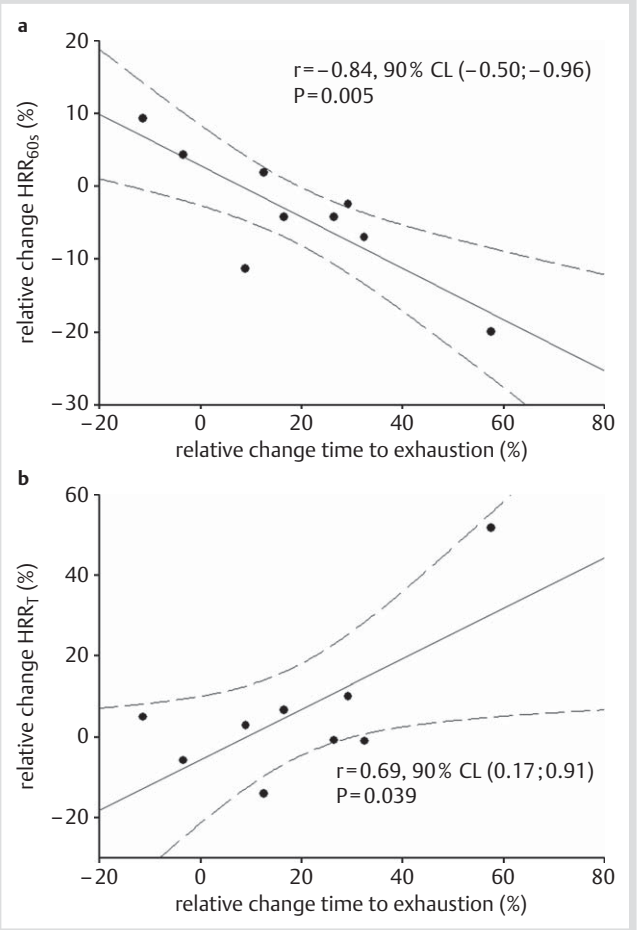

Fig. 3 **a** Relationship between the relative change in running time to exhaustion (T_{lim}; s) and relative change in heart rate recovery during the first 60s (HRR_{60s}; bpm) during the first 2 weeks of tapering. **b** Relationship between the relative change in running time to exhaustion (T_{lim}; s) and relative change in the time constant of the heart rate decay (HRR_T; s) during the first 2 weeks of tapering.

Post-exercise HRR during overload and tapering

Post-exercise HRR is induced by a combination of sympathetic withdrawal and parasympathetic reactivation [47]. Central neural control causes the parasympathetic system to be inhibited in direct proportion to sympathetic activation [43]. In the present study, we did not observe any significant change in HRR during the overload period, which contrasts with the conclusions by Daanen et al. [17], where HRR was reported to be related to changes in training load. These results also contrast with the findings from Borresen and Lambert [6], who found a decreased HRR with an increase in training load. However, the authors speculated that their subjects had reached a state of short-term overtraining, which is improbable in the present study given their running performance. In contrast, present results support the idea of Lambert et al. [32] and Lehmann et al. [36], where a faster HRR can be observed with acute fatigue (overload).

Following the second week of taper however, there was a significant reduction in HRR, which is consistent with the results reported by Houmard et al. [27], who reported a slower HRR after a 10-day reduction of training load. While additional physiological measures (e.g., drugs, muscle nerve activity) would be required to draw definitive conclusions, this suggests that either sympathetic activity increased and/or that post-exercise parasympathetic reactivation was reduced after 2 weeks of tapering. Possible explanations for this altered sympathovagal balance include changes in training intensity distribution related to the tapering phase: while high volumes of moderate intensity exercise may induce rapid increases in post-exercise parasympathetic activity (within 24 h), high-intensity exercise generally leads to prolonged reduction in parasympathetic activity (48–72 h) [52]. To our knowledge, the present study is the first to report that this reduction in HRR may be associated with an improvement in T_{lim} during tapering in endurance athletes. In line with this observation, was also the very large correlation between changes in HRRτ and T_{lim} during the taper (**Fig. 3b**). In fact, only one subject exhibited both an increased HRR_{60s} and an increase in performance during tapering, whereas all of the athletes had a decreased HRR_{60s} along with an increase in performance (**Fig. 3a**). One may then speculate that a more balanced ANS activity (inferred by a slower HRR) is actually favourable for marathon performance [46]. Since T_{lim} was longer after the second week of tapering, it could be argued that the changes observed in HRR were more related to changes in relative exercise intensity, exercise duration, and/or energy contribution, rather than ANS activity per se. While we cannot rule out the possibility that the longer time to exhaustion was associated with greater sympathetic activity (higher central command, greater peak HR), which could have, in turn, lowered parasympathetic reactivation, post-exercise blood lactate was actually lower after the second week of taper, which would be expected to be related to faster, not slower HRR. In fact, parasympathetic reactivation has been shown to be largely correlated with muscle metaboreflex activation and associated systems stress metabolites accumulation in the blood, with the greater the anaerobic contribution, the slower the post-exercise parasympathetic reactivation [13]. It is also worth noting that exercise intensity (which remained the same throughout the different testing sessions, i.e., 95% of $v\dot{V}O_{2peak}$), instead of exercise duration, is likely the stronger determinant of cardiac parasympathetic reactivation [52]. Importantly, there was no difference in any of the other potential confounding factors between each testing session (e.g., nutrition status, peak ventilation, peak breathing frequency), which increases the confidence in the interpretation of the observed changes. The use of a standardized submaximal exercise (with respect to both duration and intensity [14]) might nevertheless allow a better examination of the changes in ANS activity per se, which should be the focus of further research. We nevertheless believe that, irrespective of the underlying mechanisms responsible for this reduced HRR following tapering, these changes are likely of interest for practitioners, who may use HRR as an indirect measure of training adaptation.

Post-exercise parasympathetic modulation during overload and tapering

It is well known that long-term aerobic training increases parasympathetic activity and reduces sympathetic activity at rest and during submaximal exercise [11, 12]. The short-term effects of endurance training likely mirror a dose-response relationship between training load and HRV components in recreational marathon runners [37]. It was found that increased training loads during a 6-month training period were related to a shift toward a sympathetic predominance in supine resting position. In an earlier study with world-class rowers, performance required adaptations in the neural regulation of the cardiovascular system that were the opposite of those brought about by moderate-intensity training [28]. After the second tapering week, $RMSSD_{5–10min}$ was lower and $RMSSD_{30s}$ was reduced between 90 and 120 s of recovery (**Table 1**, **Fig. 2**). These results confirm the data collected in elite rowers [46], and suggest a reduction in post-exercise parasympathetic reactivation at the end of the taper, as already inferred from the HRR results. Interestingly also, the lack of association between T_{lim} and HRV changes support previous observations showing that the respective link between HRR, HRV, training load and performance differ [12].

Conclusion

The present results show that in well-trained marathon runners, the significant increase in running performance following tapering was associated with slower HRR. Therefore, fitness improvements may not always be associated with increased HRR (or conversely). The present results suggest that the interpretation of changes in HRR should be made in relation to the specific training phases of an endurance program (e.g. base training; tapering). One may also recommend using HRR – and for practical reasons the simplest variable, HRR_{60s} – instead of post exercise HRV for monitoring acute or short-term changes in cardiac autonomic function, and possibly performance capacity during taper. Further studies on HRR and HRV indices comparing endurance, power-sprint, glycolytic (anaerobic) or intermittent athletes during tapering are required to confirm the present results.

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