Autonomic Recovery after Exercise in Trained Athletes: Intensity and Duration Effects

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ABSTRACT

SEILER S., O. HAUGEN, and E. KUFFEL. Autonomic Recovery after Exercise in Trained Athletes: Intensity and Duration Effects. *Med. Sci. Sports Exerc.*, Vol. 39, No. 8, pp. 1366–1373, 2007. **Purpose:** To investigate the effects of training intensity and duration, through a range representative of training in endurance athletes, on acute recovery of autonomic nervous system (ANS) balance after exercise. **Methods:** Nine highly trained (HT) male runners ($\dot{V}O_{2max}$ 72 ± 5 mL·kg·min^{-1}, 14 ± 3 training hours per week) and eight trained (T) male subjects ($\dot{V}O_{2max}$ 60 ± 5 mL·kg·min^{-1}, 7 ± 1 training hours per week) completed preliminary testing to determine ventilatory thresholds (VT_1, VT_2) and $\dot{V}O_{2max}$. HT performed four intensity-controlled training sessions: 60 min and 120 min below VT_1; 60 min with 30 min between VT_1 and VT_2 (threshold); and 60 min above VT_2 (6 × 3 min at 96% $\dot{V}O_{2max}$, 2 min of recovery). T also completed the interval session to compare ANS recovery between HT and T. Supine heart rate variability (HRV) was quantified at regular intervals through 4 h of recovery. **Results:** When HT ran 60 or 120 min below VT_1, HRV returned to pretraining values within 5–10 min. However, training at threshold (2.7 ± 0.4 mM) or above VT_2 (7.1 ± 0.7 mM) induced a significant, but essentially identical, delay of HRV recovery (return to baseline by approximately 30 min). In T, HRV recovery was significantly slower, with HRV returning to baseline by ≥90 min after the same interval session. **Conclusions:** In the highly trained endurance athlete, exercise for ≤120 min below the first ventilatory threshold causes minimal disturbance in ANS balance. ANS recovery is more rapid in highly trained than in trained subjects after high-intensity exercise. Further, the first ventilatory threshold may demarcate a "binary" threshold for ANS/HRV recovery in highly trained athletes, because further delays in HRV recovery with even higher training intensities were not observed.
Key Words: AUTONOMIC NERVOUS SYSTEM, PARASYMPATHETIC, HEART RATE VARIABILITY, OVERTRAINING, ENDURANCE

Elite endurance athletes endure very high training loads (frequency, duration, and intensity), which induce adaptive effects and stress reactions. The high frequency of training imposed ensures that these adaptive effects are cumulative. Unfortunately, incomplete recovery from frequent training can make the stress-related side-effects cumulative as well. The day-to-day distribution of training intensity may be a crucial variable to effectively balance positive adaptive and negative stress effects so that performance development is achieved without stagnation or overtraining (30). We have previously proposed that two basic patterns of training intensity distribution emerge from the research literature (22). The *threshold* model emerges from a number of short-term studies on untrained subjects demonstrating that training at the lactate threshold intensity induces significant physiological improvements (10,12, 14,15). The *polarized* training model describes observations from several descriptive studies quantifying intensity distribution in international-class rowers (25,26), Olympic-winning pursuit cyclists (21), international-class marathon runners (4), and elite junior cross-country skiers (22). These studies report that elite athletes actually perform approximately 75% of their training at intensities clearly below the lactate threshold, relatively little training at the traditional lactate threshold, and approximately 10–20% of their training at intensities clearly above the lactate threshold. This latter training is typically characterized by blood lactate concentration in the 6- to 10-mM range and heart rate exceeding 90% of HR_{max} (5,23,27).

We hypothesize that the similar day-to-day training distribution characteristics observed in different groups of elite endurance athletes reflect a *self-organizing* strategy that balances the adaptive signaling and potentially maladaptive stress-inducing components of the training load appropriately. Training distribution may self-organize around two key constraints that are generally accepted as important to success by endurance athletes: high overall training volume, and adequate exposure to race-pace or near-race-pace intensity in training. Achieving these goals without excessive training stress may tend to induce a specific pattern of intensity distribution. The combined intensity and duration of a training session would be expected to impact the

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magnitude of the stress response and the time required for recovery. One key aspect of this stress response is activation of the sympathetic arm of the autonomic nervous system (ANS) and a shift in autonomic balance. The quantification of heart rate variability (HRV) has become an established, noninvasive tool to investigate autonomic balance via its impact on beat-to-beat heart rate variation (2,20,29). Measurements during exercise have demonstrated that HRV differentiates exercise performed above and below the ventilatory threshold (8). Several studies have examined autonomic recovery after exercise using HRV; however, these studies have employed untrained or physically active subjects after relatively short exercise periods at nonindividualized intensities (1,13,17,28). To date, we have not identified studies that have compared ANS recovery as a function of training intensity and duration in highly trained athletes.

To more accurately characterize the acute stress response associated with typical training sessions of well-trained competitive runners, we have used HRV to quantify acute ANS recovery of the autonomic nervous system in highly trained endurance athletes, after exercise bouts that were carefully controlled within the three training intensity zones individually defined by their first and second ventilatory turn points (9). In addition, we have compared ANS recovery after a high-intensity training session in highly trained athletes with responses in trained recreational athletes.

METHODS

Seventeen subjects volunteered to participate in this study, which was approved by the ethics committee for the faculty of health and sport. All subjects provided informed written consent before participation. The nine highly trained subjects (HT) were all competitive orienteers (eight subjects) or distance runners (one subject) of high national performance standard (including a junior world champion, a Senior Nordic Countries champion, and a World Cup Orienteering overall winner) who often trained two times daily. As such, they represented a convenience sample of available athlete volunteers who met the performance level and daily training characteristics of interest. Data were collected during January and February, the precompetition preparation period for the national and World Cup orienteering season (March to October). The eight subjects in the trained group (T) were sports active males who trained regularly, either individually (jogging and strength training) or as members of team handball or soccer clubs. Their typical endurance training included both individual runs of approximately 60 min, as well as more high-intensity interval-type sessions in the form of game play.

The study consisted of two parts. In part 1, HT subjects performed four randomly sequenced training sessions varying in intensity or duration, followed by a 4-h collection of HRV data to quantify ANS recovery. In part 2, HT and T subjects' HRV recovery responses were compared after a high-intensity interval training session. An untrained control group was not included, because these normal training sessions performed by highly trained athletes would have likely represented exhaustive, and therefore noncomparable, efforts in untrained subjects.

Preliminary testing. Before the study, all subjects performed a treadmill test to voluntary exhaustion to determine baseline physiological characteristics and training intensity zone cutoffs. Before each test, subjects warmed up for 20 min by running at self-selected speed on a motorized treadmill (Woodway ELG 55, Weil am Rhein, Germany). The test was initiated at 7 $km \cdot h^{-1}$ and a constant treadmill elevation of 5%. After a 2-min stabilization period, treadmill velocity was increased by 0.75 $km \cdot h^{-1} \cdot min^{-1}$ until the subject was no longer able to sustain the required pace. When the subjects were visibly near exhaustion, within the final 30 s of treadmill running, they were presented a poster of a 15-point Borg scale (6) for the determination of RPE at maximal exertion (RPE_{peak}). Before testing, each subject read a standard explanation of the Borg RPE scale (6) and received verbal instruction on how it would be presented during the test.

Gas-exchange measurements were made continuously with an automated breath-by-breath system (Oxycon Pro, Jaeger BeNeLux, Breda, Netherlands), calibrated before each test according to the manufacturer's instructions. Gas-exchange measurements were used to quantify the first ventilatory threshold (VT_1), second ventilatory threshold (VT_2), and maximal oxygen consumption ($\dot{V}O_{2max}$). VT_1 was defined as the intensity at which an increase in $\dot{V}_E/\dot{V}O_2$ occurred without an increase in $\dot{V}_E/\dot{V}CO_2$. VT_2 was identified as the intensity at which $\dot{V}_E/\dot{V}CO_2$ also began to rise (9). $\dot{V}O_{2max}$ was defined as the highest average oxygen consumption measured during a 30-s period. Heart rate was recorded continuously via telemetry (Polar s810i, Finland) for the determination of heart rates corresponding to VT_1, VT_2, and $\dot{V}O_{2max}$. For the determination of blood lactate at peak exercise ($lactate_{peak}$), a blood sample was collected from a finger within 60 s after test cessation and analyzed with a portable device (LactatePro LT1710, Arkay KDK, Japan).

Training sessions. Preliminary test results were used to identify three training intensity zones: below VT_1, threshold ($\geq VT_1$ and $\leq VT_2$), and above VT_2. After preliminary testing, HT performed four intensity-controlled training sessions in randomized order for an approximately 4-wk period of training, where they replaced one normal training bout each week with a session in the laboratory. Because the HT subjects were all in preparation for their competitive season, effort was made to integrate these sessions into the normal training organization. Although these athletes often trained two times per day, the laboratory sessions performed for this study were always the first (or only) training session of the day and were always preceded by a minimum of 12 h of recovery.

The below-$VT_1$60 session consisted of 60 min of running on the treadmill (2–5% incline) at self-selected intensity

TABLE 1. Physical characteristics of the highly trained and trained groups.

	Highly Trained (N = 9)	Trained (N = 8)
Age (yr)	23 ± 5	27 ± 4
Weight (kg)	68.3 ± 8.3*	83.6 ± 8.7
Height (cm)	180 ± 6	184 ± 3
$\dot{V}O_{2max}$ (mL·kg·min^{-1})	72 ± 5*	60 ± 5
$Lactate_{peak}$ (mM)	10.0 ± 2.8	9.6 ± 1.8
RPE_{peak}	18 ± 1	18 ± 0.6
VT_1 (% $\dot{V}O_{2max}$)	77 ± 5	76 ± 4
VT_2 (% $\dot{V}O_{2max}$)	86 ± 5	84 ± 3
HR_{max}	189 ± 8	189 ± 9
Weekly training volume (h·wk^{-1})	14 ± 3*	7 ± 1

* $P < 0.05$ vs trained.

below VT_1 (55–65% $\dot{V}O_{2max}$, 10–15% $\dot{V}O_{2max}$ below VT_1). Oxygen consumption, blood lactate, and RPE were determined at 20 and 40 min to confirm appropriate intensity. The below-$VT_1$120 session consisted of running for 120 min at the same self-selected intensity, verified with continuous heart rate monitoring, RPE, and lactate measurements performed after 40, 80, and 120 min of exercise. This session was performed outdoors on a forest trail loop. The threshold session, performed on the treadmill, consisted of a 20-min warm-up below VT_1 intensity followed by 30 min of running within the previously established threshold zone (typically 80–85% $\dot{V}O_{2max}$) and a 10-min cool-down below VT_1 intensity. $\dot{V}O_2$, blood lactate, and RPE were monitored at 10 and 20 min of the threshold training period. The above–VT_2 intensity session, performed on the treadmill, consisted of a 20 min warm-up below VT_1 intensity, a 30-min interval period consisting of 6 × 3 min at a velocity eliciting 95–100% of $\dot{V}O_{2max}$ with 2 min of active recovery periods (the above-VT_2 portion of the session), and a 10-min warm-down below VT_1. Gas exchange was measured continuously during the second and fourth interval bouts, and RPE and blood lactate measurements were made at the end of these bouts to estimate the average intensity of the interval session. The total duration of the below-$VT_1$60, threshold, and above-VT_2 bouts was 60 min, and the below-$VT_1$120 session was 120 min in duration. The second group of trained subjects (T) performed only the above-VT_2 session, under identical conditions to those described for HT. Thirty minutes after the conclusion of each training session, subjects were asked to rate the overall stress, or intensity, of the session on a 10-point scale developed by Foster et al. (11). This posttraining index of the session stress is termed *session RPE*.

Heart rate variability measurements. Before each training session, subjects assumed a supine position on a medical examination bench in a quiet room for the determination of resting heart rate variability (HRV_{pre}). After heart rate had stabilized in the supine position, RR intervals were collected for a period of 5 min using a Polar s810i heart rate set to RR interval mode. For HT, HRV_{pre} values used for reference in each athlete were derived from pooled preexercise data for the four test sessions. After each training session, 5-min supine RR interval measurements were made beginning at 5, 15, 30, 60, 90, 120, 180, and 240 min after exercise. Food intake was controlled during the 4-h data-collection period such that subjects consumed a standard sequence of caloric replacement using foods they normally consumed. After the 30-min measurement, subjects consumed one banana and one half of a New Energy energy bar (~275 kcal). After the 60-min HRV measurement, subjects consumed one banana plus a slice of whole-grain bread (local bakery) with one slice of Norvegia mild cheese (~275 kcal). After the 120-min measurement, subjects consumed one banana and three whole-grain bread slices with cheese (~475 kcal). Water or an electrolyte replacement drink was consumed *ad libitum* during the 4-h postexercise period. Supine HRV measurements were collected during spontaneous breathing at all time points.

The time in milliseconds from one QRS peak to another QRS peak is defined as the RR interval. RR interval recordings were made with an accuracy of 1/1000th second, using a Polar s810i Polar heart rate monitor in RR recording mode. Data files were transferred to computer via infrared interface, visualized, and corrected for occasional erroneous RR interval signals using visual inspection and an error detection algorithm in the Polar Precision Performance Analysis program SW 4.0 (Polar Electro Oy, Kempele, Finland). This process was performed by the same investigator for all measurements. Corrected data files were then transported to a dedicated HRV analysis program (HRV Analysis Software, The Biomedical Signal Analysis Group, University of Kuopio, Finland). All subsequent HRV analyses were performed on 300-s data windows (24).

HR variability analysis was used to quantify mean heart rate (HR), mean RR interval, the root mean square of sequential deviations (RMSSD), and the percentage of all sequential RR deviations exceeding 50 ms (pNN50) in the time domain, plus HF power in normalized units (HF n.u.= HF/total power − VLF) × 100) and LF power in normalized units (LF n.u. = LF/total power − VLF) × 100) in the frequency domain (29). LF and HF power were determined

TABLE 2. Intensity characteristics of the training sessions performed by the highly trained (HT) and trained (T) groups.

Session	HR (% max)	$\dot{V}O_2$ (% max)	Blood Lactate (mM)	RPE	Session RPE
Below $VT_1$60 (HT)	68 ± 7	61 ± 0.7	1.0 ± 0.1	9.7 ± 0.4	2 ± 0
Below $VT_1$120 (HT)	68 ± 7	ND	1.0 ± 0.1	10 ± 0.4	2.4 ± 1.1
Threshold (HT)	88 ± 2	84 ± 0.7	2.7 ± 0.4	13.9 ± 0.5	5 ± 0.6
Above VT_2 (HT)	95 ± 3	96 ± 0.7	7.1 ± 0.7	17.2 ± 0.8	8.1 ± 1
Above VT_2 (T)	97 ± 2	95 ± 1	8.7 ± 1.6	17.5 ± 1.4	8.2 ± 0.5

ND, not determined—oxygen consumption not measured because session was performed outdoors. There were no significant differences in intensity between the two below-VT_1 sessions. All measures of physiological intensity and perceived exertion were significantly different ($P < 0.01$) among below-VT_1, threshold, and above-VT_2 sessions performed by HT. There were no significant differences in physiological responses to the above-VT_2 sessions between HT and T.

TABLE 3. Preexercise heart-rate variability values for highly trained (HT) and trained (T) subjects.

	Heart Rate (bpm)	RR Interval (ms)	RMSSD (ms)	pNN50 (%)	HF n.u. (%)	LF n.u. (%)
Highly trained (N = 9)	55 ± 11	1127 ± 210	103 ± 34	56 ± 16	48 ± 18	52 ± 18
Trained (N = 8)	60 ± 6	1011 ± 99	87 ± 55	37 ± 22	41 ± 17	59 ± 17

RMSSD, root mean square of sequential deviations; pNN50, percentage of RR intervals varying by more than 50 ms from previous interval; HF n.u., high-frequency heart-rate variability expressed in normalized units; LF n.u., low-frequency heart-rate variability expressed in normalized units. For highly trained subjects, the values above are pooled results of four preexercise measurements. Although all measures of heart-rate variability tended to be greater in highly trained subjects, the differences were not statistically significant.

using fast Fourier transform analysis. The low frequency (LF: 0.04–0.15 Hz) and high (29). All analyzed data were detrended to minimize the VLF contribution to the frequency spectrum.

Statistical analysis. Statistical analyses were performed using SPSS 14.0. All HRV data were normalized to a percentage of individual pre exercise values. The time course of HRV recovery as a function of preceding exercise intensity in HT was analyzed using a 4 (exercise bouts) × 8 (recovery time points) repeated-measures ANOVA. Comparison of HRV recovery in HT and T after above-VT_2 training was performed using a two-way ANOVA with repeated measures on time (two groups, eight time points) to compare the main effect of group on the time course of recovery. After determination of significant group and interaction effects, comparisons between the two groups at specific time points were made using independent-samples *t*-tests. A P value of <0.05 was considered statistically significant.

RESULTS

The subjects in the HT group trained on average (± SD) 14 ± 3 h·wk^{-1} and had a $\dot{V}O_{2max}$ of 72 mL·kg·min^{-1}. In comparison, the T group exercised 7 ± 1 h·wk^{-1} and had a $\dot{V}O_{2max}$ of 60 mL·kg·min^{-1}. The physical characteristics of the two groups are presented in Table 1. The training sessions were well controlled so that all subjects performed within the prescribed intensities for each training session (Table 2). Further, the intensity zones defined using ventilatory thresholds seemed to accurately demarcate three different training intensity zones based on lactate responses, RPE, and session RPE report from the subjects. Baseline preexercise values for time and frequency domain HRV indices in both HT (who performed four different training sessions) and T (who performed only the interval session) are presented in Table 3.

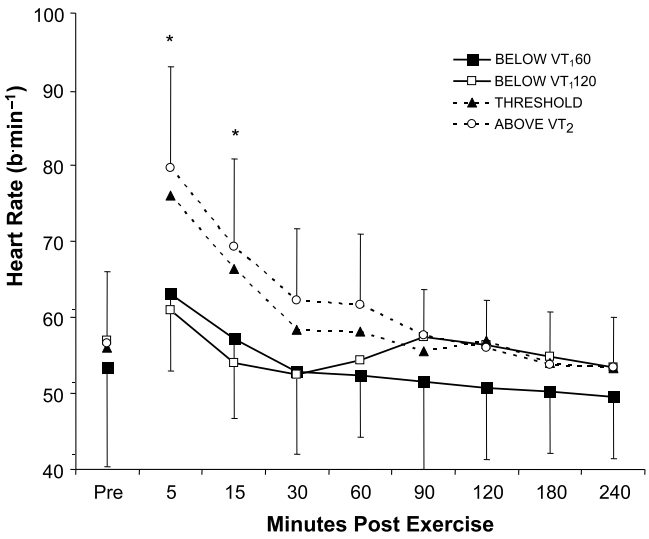

FIGURE 1—Heart rate recovery time course in highly trained subjects (HT, N = 9) after training bouts varying in intensity (below VT_1 vs threshold vs above VT_2, 60-min total bout duration) and duration (below VT_1, 60 min vs 120 min). * P < 0.05 for heart rate recovery after above-VT_2 bout vs below-$VT_1$60 and below-$VT_1$120 bouts.

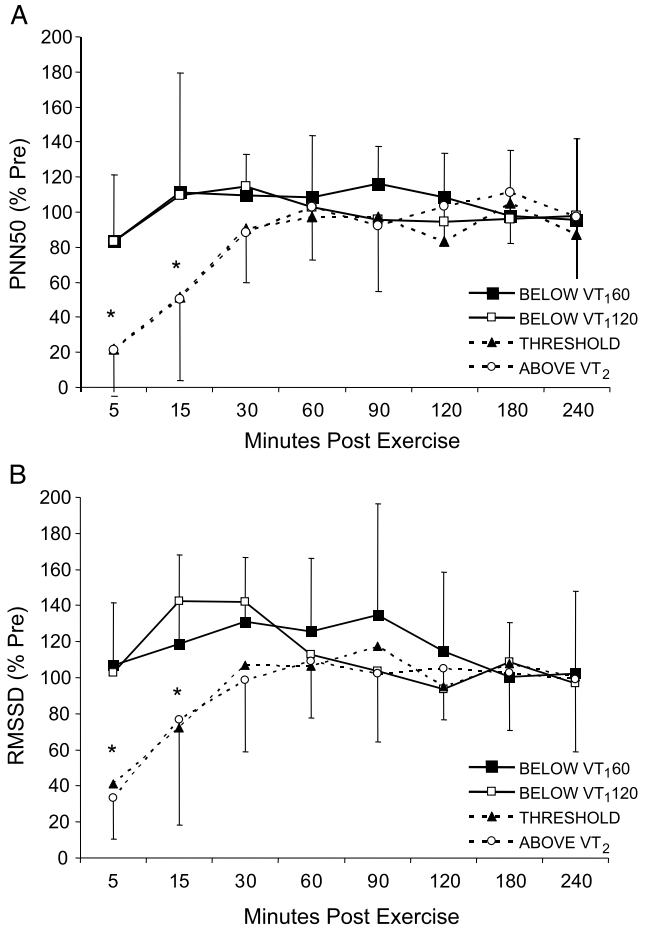

FIGURE 2—Time course of recovery of heart rate variability measured as percentage of RR exceeding 50 ms (PNN50, *panel A*) and root mean square of sequential deviations (RMSSD, *panel B*) in highly trained subjects (HT, N = 9) after training bouts varying in intensity (above VT_1 vs threshold vs above VT_2, 60-min total bout duration) and duration (below $VT_1$60 vs below $VT_1$120). * P < 0.05 for threshold and above VT_2 vs below $VT_1$60 and below $VT_1$120. *Error bars* are omitted from two of the groups for clarity.

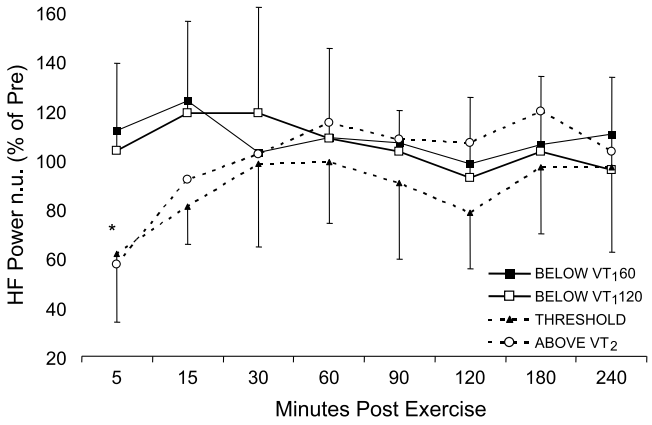

FIGURE 3—Time course of recovery of high-frequency HRV (parasympathetic tone) expressed in normalized units in highly trained subjects (N = 9) as a percentage of preexercise resting values after four exercise bouts differing in intensity and duration. * $P < 0.05$ for threshold and above-VT_2 exercise bouts vs below-$VT_1$60 and below-$VT_1$120 bouts. *Error bars* are omitted from two of the groups for clarity.

ANS recovery and exercise intensity/duration in HT athletes. The recovery time courses of resting heart rate after exercise bouts of different intensity are presented in absolute values in Figure 1. Time domain (Fig. 2A and B) and frequency domain (Fig. 3, high-frequency component) measures of HRV recovery during the 4-h postexercise period are expressed as a percentage of preexercise values Exercise for 60 or 120 min below VT_1 induced little or no delay in postexercise ANS recovery. HRV measures were already recovered to preexercise values by 5 min after exercise. There was a tendency toward a rebound increase in parasympathetic tone observed after the below-$VT_1$120 condition. In contrast, both threshold and above-VT_2 exercise conditions induced significant delays in the recovery of autonomic balance. However, no difference in the autonomic balance recovery time course between the threshold and above-VT_2 training intensity conditions was detected using HRV indices.

Comparison of HRV recovery after high-intensity training in HT and T subjects. Because the HRV recovery time-course of HT athletes to even high-intensity interval training was quite rapid, we also quantified HRV recovery after an identical interval training session in a group of recreational athletes (T) who were training at approximately 50% of the weekly training load of HT. On the basis of a comparison of physiological and RPE responses (Table 2), HT and T performed the above-VT_2 session at the same relative intensity. However, recovery of parasympathetic control after above-VT_2 exercise was significantly slower, by about 60–90 min, in T compared with HT (Fig. 4).

DISCUSSION

This study can be summarized with three key findings. The first key observation was an increase in autonomic stress in the HT athletes once the first ventilatory threshold intensity was exceeded. Recovery of parasympathetic tone was extremely rapid after up to 120 min of running below the first ventilatory threshold (below-$VT_1$60 and below-$VT_1$120 conditions). In contrast, when exercise was performed at intensity above the first ventilatory threshold (threshold and above-VT_2 conditions), ANS recovery was significantly delayed. One primary conclusion of this study is, therefore, that the first ventilatory threshold also seems to demarcate a clear threshold for ANS perturbation as measured using HRV recovery. Secondly, we did not observe any further delay in ANS recovery when interval training at 95% $\dot{V}O_{2max}$ was performed, compared with training at lactate-threshold intensity. Because HT athletes recovered quite rapidly from even above-VT_2 training, we added a second group of subjects who also trained regularly, but at only half the volume of HT. The third key finding of this study was that the more moderately trained subjects required two to three times longer (60–90 min) to reach the same level of parasympathetic recovery after above-VT_2 training as the HT subjects. We believe these findings are meaningful for understanding training optimization in highly trained endurance athletes.

The practical motivation for this study was our interest in optimal endurance training organization and the factors that may influence it. There is a growing body of data describing the organization of training intensity in high level endurance athletes (3,4,21,22,25,26). Those studies suggest that most elite athletes train in large volumes at intensities below the first ventilatory or lactate turn point (i.e., 60–70% $\dot{V}O_{2max}$), and in substantial volumes around or above the second ventilatory threshold (i.e., 88–95% $\dot{V}O_{2max}$), but comparatively little at intensities typically described as "lactate threshold" training (i.e., 75–85% $\dot{V}O_{2max}$). These observations run counter to the idea of concentrating training at the lactate threshold suggested by

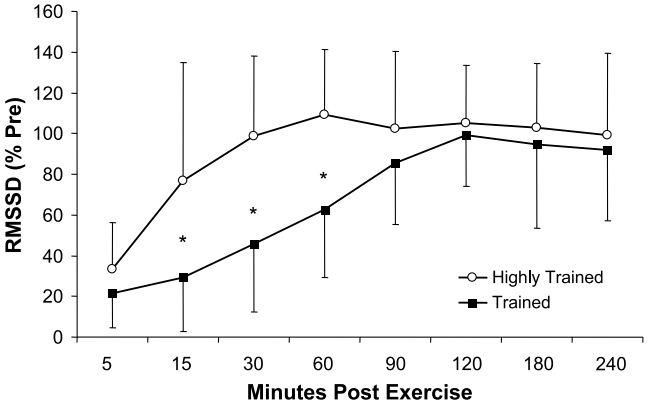

FIGURE 4—Comparison of recovery time course for HRV measured as root mean square of sequential deviations (RMSSD) in highly trained (HT, N = 9) vs trained (T, N = 8) subjects after an above VT_2 (near $\dot{V}O_{2max}$ intervals) exercise bout. * $P < 0.05$ for HT vs T. 95% Confidence intervals (upper and lower bounds) for percent recovery in HT and T at 15, 30, and 60 min of recovery were 15 min: HT 53–99%, T: 4–53,; 30 min- HT 75–122%, T 21–70%; 60 min- HT 86–133%, T 38–86%.

studies of untrained subjects. We therefore wished to quantify the stress-recovery response of the autonomic nervous system after training sessions performed within each of these three physiologically distinguishable intensity zones. HRV measured during exercise has been shown to differentiate exercise performed below and above ventilatory threshold (8). A number of previous studies have also used HRV analysis to evaluate recovery of sympathovagal balance after exercise. However, these studies have used untrained subjects, relatively short exercise periods (5–30 min), and/or intensities that were not individualized to the subjects' ventilatory or lactate thresholds (1,13,17,28). The results of the present study allow us to make some practical interpretations of how exercise intensity and duration impact acute autonomic recovery in the daily training of highly trained athletes.

Training for up to 120 min at 68% HR_{max} (61% $\dot{V}O_{2max}$) was easily tolerated by HT according to perceived exertion and HRV recovery. HRV indices of autonomic stress indicated essentially full recovery of parasympathetic control to preexercise levels at 5 min after exercise. The athletes controlled the pace of the below-VT_1 sessions in keeping with their own normal work intensity for this type of training. This intensity may be representative for as much as 75% of total training by successful athletes from several disciplines (4,21,22,25,26). Consistent with the present findings, a study of elite female rowers observed "minimal changes" (data not reported) in blood hormone (cortisol, growth hormone, epinephrine, norepinephrine) and immune-function measures in response to a 2-h training session performed at approximately 60% $\dot{V}O_{2max}$ (16).

Training at threshold intensity delayed HRV recovery compared with training below VT_1 intensity. We expected that HRV recovery would be further delayed (indicative of greater sympathetic stress) after above-VT_2 training (near $\dot{V}O_{2max}$ intervals) compared with threshold training. However, the HRV recovery time courses after these two training sessions were quite similar. These data suggest an essentially binary response in autonomic nervous system recovery in the HT athletes, with the first ventilatory threshold (VT_1) marking the threshold for delayed ANS recovery. Put in practical terms, an increase in blood lactate from approximately 1 to 3 mM during the training session was associated with a substantial delay in the ANS recovery time course. If HRV recovery provides a valid marker for an integrated stress response, we interpret these findings as being consistent with the way successful endurance athletes have been shown to organize their training. In the HT athletes, training sessions lasting up to 120 min, but performed below VT_1, likely induce a targeted increase (or maintained level) in DNA transcription at the muscular level without inducing a significant systemic stress response. We speculate that once training intensity exceeds VT_1, or the first lactate turn point, the positive signaling effects of the training bout on skeletal muscle are accompanied by a significantly greater systemic stress response. When regularly applied, monotonic training at lactate threshold intensity has been suggested to increase risk of overreaching or overtraining (7). Athletes may choose to 1) accumulate most of their training volume at intensities below this threshold and 2) perform most of the training sessions that activate this stress response at even higher intensities, to maximize the effects of training on cardiovascular performance, buffering capacity, and efficiency at near-racing velocities. Thus, in the high-performance endurance athlete performing a high training load (often typified by multiple daily training sessions), the autonomic system response pattern we observed would support a self-organization in the direction of polarization of day-to-day intensity organization away from a monotonic loading of threshold-intensity sessions, despite the fact that this type of loading has been consistently shown to stimulate performance improvements in untrained subjects (12,15) training 1 $h \cdot d^{-1}$ or less.

We believe this to be the first study comparing ANS recovery after exercise in two different groups of trained subjects differing in training volume and performance level. We found that the HT subjects had markedly accelerated ANS recovery after high-intensity exercise compared with a group of regularly active, well-trained subjects exercising approximately 7 $h \cdot wk^{-1}$. Measures of parasympathetic recovery after intense interval training were delayed by 60–90 min in the T subjects compared with the HT subjects. This difference was not attributable to relative differences in the exercise intensity. It was also not associated with differences in either acute RPE or session RPE responses between groups. Although these are cross-sectional data, they suggest that one aspect of the transition from trained to highly trained status is more rapid stress recovery. Rapid recovery may be critical to tolerating the typical twice-daily frequency of training observed among elite endurance athletes. However, whether this accelerated recovery is a long-term adaptation to frequent training, or an inherent characteristic of successful athletes, cannot be determined from this study.

There are limitations to the study and our interpretation of the results. The lactate threshold and high-intensity sessions were representative of what trained subjects would complete during normal training. However, it is likely that the HT subjects could have tolerated a longer exercise bout at threshold intensity and more accumulated work above VT_2 (more interval bouts at 95% $\dot{V}O_{2max}$). Making these sessions longer and representative of the most demanding sessions performed by these athletes might have revealed differences in the ANS recovery time course between threshold and interval sessions.

Another question that cannot be answered from this study is what duration of exercise must be performed above the first ventilatory/lactate turn point to induce the elevated autonomic stress observed. In the present study, 30 min of exercise performed between VT_1 and VT_2 intensity was sufficient to markedly delay autonomic recovery postexercise

in HT. Assuming that exercise duration and intensity act in concert to determine the magnitude of both adaptive signaling and systemic stress responses, simultaneously measuring both of these aspects of the training response as a function of duration and intensity should be a goal of future studies.

Finally, we did not measure other stress responses, such as plasma catecholamines, that are known to increase with increases in both exercise intensity and duration. We have not identified any studies that have simultaneously measured HRV and stress hormones during recovery from exercise. Plasma epinephrine and norepinephrine concentrations return to baseline levels within 15 min after 60 min of exercise at 70% $\dot{V}O_{2max}$ in well-trained athletes. In contrast, plasma cortisol remains elevated several hours after the same exercise bout (19). This suggests that the full recovery of HRV to baseline values should not be taken to correspond to a complete return to preexercise homeostasis. Ronsen and colleagues (18) observed an augmented neuroendocrine response to a second bout of exercise on a single day despite normalized plasma concentrations of all hormones before the second exercise session. Therefore, although we hypothesize that the integrated HRV recovery delay after exercise is indicative of the overall magnitude of the stress response induced, the time course of HRV recovery does not indicate full withdrawal of the systemic stress response. The specific relationship between HRV recovery and other stress response variables needs to be tested in well-trained subjects.

CONCLUSIONS

In the present study, we found that highly trained endurance athletes were characterized by rapid recovery of parasympathetic balance after exercise, independent of intensity. However, the first ventilatory threshold also demarcates a clear threshold for ANS recovery, with recovery of parasympathetic nerve discharge being significantly delayed after 30 min of training above VT_1 intensity. We did not observe a difference in ANS recovery time course between interval training at 95% $\dot{V}O_{2max}$ and training at the lactate threshold intensity, even though perceived exertion measures were significantly different. The present findings may give some insight into understanding how highly trained athletes organize the day-to-day distribution of training intensity.

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