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Ultra Short-Term Heart Rate Recovery after Maximal Exercise: Relations to Aerobic Power in Sportsmen

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Abstract

The main aim of the study was to investigate whether different levels of aerobic power influence heart rate (HR) responses during the first minute of recovery following maximal exercise in athletes. Thirty-two young male soccer players were recruited for the study during the final week of their training prior the competition. Following the maximal exercise on treadmill the participants were placed supine for 60 s of HR recording. The time between exercise cessation and the recovery HR measurement was kept as short as possible. At the end of exercise (*i.e.*, the start of recovery), HR was similar in both trials. At both 10 s and 20 s of recovery period, the players characterized by high aerobic power (> 60 ml/kg/min) revealed significantly lower HR as compared to their sub-elite counterparts (< 50 ml/kg/min; $P < 0.05$). No differences between the groups were found at later stages of the analyzed post-exercise HR. The data suggest that the athletes characterized by high aerobic capacity could be better adapted to maximal exercise with faster recovery HR immediately following an exercise test. These results generally suggest that the aerobic power along with autonomic modulation might have played a role in the ultra short-term cardiovascular responses to all-out exercise.

Key Words: soccer players, Vo_{2max}, tredmill, autonomic control

Introduction

Heart rate recovery (HRR) is the rate at which the heart rate (HR) decreases (*i.e.*, the time taken for HR to recover) following a moderate to heavy exercise in response to a combination of parasympathetic activation and sympathetic withdrawal (2, 15, 16, 20). HRR is known to change in response to acute and seasonal changes in training load (3, 19, 29). However, there are no clear data indicating whether HRR is a sensitive measure of autonomic control and, in particular, whether HRR can be used as an index representing the body's capacity to respond to training. Aerobic fitness is another variable that could influence HRR response. Short and Sedlock (24) showed that throughout the recovery period the group of trained athletes with superior aerobic capacity had a consistently lower HR as compared to untrained subjects, but there is still no clear explanation of this phenomenon. HRR after maximal exercise has been neither studied in elite athletes characterized by superior aerobic fitness nor compared it with athletes characterized by lower aerobic capacity. The evaluation of the post-exercise HRR can be quantified by different methods, including the absolute difference between the final HR at exercise completion and HRR recorded following 60 s of recovery (HRR_{60s}) (5), first 30 s of HRR *via* semi-logarithmic regression analysis (T30) (12), or the time constant of the HR decay obtained by fitting the post-exercise HRR by a

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first-order exponential decay curve (18). Concerning the fact that initial HRR progresses in a decreasing monoexponential fashion and seems to be predominantly influenced by parasympathetic function it has been suggested that short-term indexes (*i.e.* T30 and HRR_{60s}) could be considered as markers of cardiac parasympathetic tone in human studies (6, 9) Yet, there are no data describing the time course of 10-20 seconds of recovery of HRR immediately following the exercise cessation (*i.e.*, corresponding to the ultra short-term HRR [UST-HRR]). Assessing UST-HRR responses to the maximal exercise tasks could be of particular interest for monitoring recovery during soccer training due to the fact that the average duration of recovery in soccer matches is less than 20 s (17). Within the present study we tested the hypothesis that the differences in the level of aerobic power would influence heart rate responses during the 10-s intervals of the first minute of recovery following the maximal exercise in soccer players.

Materials and Methods

Participants

Thirty-two healthy and young male soccer players were recruited from two soccer squads during the spring half-season of 2007. All participants requesting a pre-competition medical examination with exercise-stress testing at the exercise physiology lab, who were participating in consistent training for the past 2 years and who were between 20 and 22 years of age, were candidates for inclusion in the study. The exclusion criteria were: [1] a history of heart diseases, [2] a musculoskeletal dysfunction, [3] known metabolic disease, [4] use of any performance enhancing substance within the past 14 days, [5] smoking, or [6] an impaired response to stress test. All participants gave their informed consent and volunteered to participate in the study with the approval of the institutional IRB. Eight weeks prior to the testing all subject followed a similar soccer-specific training program controlled by a certified physical conditioning coach. Participants were classified into two groups based on the level of aerobic fitness. Specifically, they were classified either as elite (VO_{2max} > 60 ml/kg/min) or sub-elite (VO_{2max} < 50 ml/kg/min). All participants were fully informed verbally and in writing about the nature and demands of the study as well as the known health risks. They completed a health history questionnaire and also were informed that they could withdraw from the study at any time, even after giving their written consent. All but six of the initial subject pool met the criteria and completed the testing. The final compositions of the groups were 12 players in the elite and 14 players in the sub-elite group.

Experimental Procedures

Physiological measurements were performed during the final week of preparatory training for competition. Twenty-four hours prior to the experiment, the participants were asked neither to participate in any prolonged exercise nor to drink alcohol and/or caffeine beverages. They reported to the physiology laboratory at 10 a.m. after a rest of between 10 and 12 h. Prior to the experimental session body mass, body height and percentage of body fat from skinfold thickness were determined. The skinfold thickness at seven sites was obtained using a caliper (Harpenden Skinfold Caliper, British Indicators Ltd., Burgess Hill, West Sussex, UK). The skinfold sites were triceps, subscapula, mid-axillary, anterior suprailiac, chest, abdomen, and thigh. The landmarks were identified and measured according to Wilmore and Behnke (28) with the median of three measurements used to represent skinfold thickness. Percentage of body fat was determined according to equations of Jackson and Pollock (13) (SEE = 8.8%). Thereafter, the subjects were instrumented for VO_{2max}, ECG and telemetric HR assessment. Exercise test was performed according to individualized ramp protocol to symptom-tolerated maximum using a treadmill system (Trackmaster TMX425C, Full Vision, Inc., Newton, KS, USA). Gas-exchange data were collected throughout the exercise test using a breath-by-breath respiromonitor system (Vacu-med CPX, Ventura, CA, USA) with VO_{2max} defined as the highest VO_2 achieved during the test. The guidelines of the Bentley and co-workers were followed to confirm that VO_{2max} was achieved (1). The electrocardiogram was continuously recorded using a 12-stress analysis system (Custo-Med EC1000, Ottobrunn, Germany), while HR was also recorded using a HR monitor at beat-to-beat interval (Polar S810, Polar Electro Oy, Kempele, Finland).

Immediately upon exercise cessation, the participants were placed supine with the face mask on for 60 s of HR recording. The time between exercise cessation and the recovery HR measurement was kept as short as possible. The ECG tracing was reviewed for correct identification of all beats. HR during the first minute of recovery was measured at 10 sec intervals *via* both HR monitor and ECG, attempting to correctly identify all beats. The mean of the two readings with a coefficient of variation below 15% was used in the study. HR decrease during the recovery phase was also quantified as a percent HR decrease from the peak exercise HR (100%) during the first minute of recovery. Due to the fact that the HRR following exercise typically progresses in a decreasing monoexponential fashion (5), we further quantified

Table 1. Physical and physiological characteristics of the subjects

	Elite athletes (n = 12)	Sub-elite athletes (n = 14)
Age (years)	20.5 ± 0.5	20.6 ± 0.6
Height (cm)	185.5 ± 5.8	184.1 ± 4.4
Body mass (kg)	77.0 ± 7.1	76.0 ± 5.1
Body fat (%)	8.5 ± 1.8	9.3 ± 1.5
Maximal oxygen uptake (ml/kg/min)	62.2 ± 2.0	46.7 ± 2.3*

Values are means ± SD. * Indicates significant difference between elite and sub-elite athletes at $P < 0.05$.

Table 2. Heart rate (HR) responses during the study

	Elite athletes (n = 12)	Sub-elite athletes (n = 14)
HR peak (b/min)	200 ± 4	198 ± 5
HRR at 10 sec (b/min)	193 ± 4	197 ± 5*
HRR at 20 sec (b/min)	189 ± 3	193 ± 4*
HRR at 30 sec (b/min)	184 ± 4	184 ± 4
HRR at 40 sec (b/min)	178 ± 4	176 ± 4
HRR at 50 sec (b/min)	171 ± 5	170 ± 3
HRR at 60 sec (b/min)	164 ± 5	162 ± 4

Values are means ± SD. Abbreviations: HRR-heart rate recovery. * Indicates significant difference between elite and sub-elite athletes at $P < 0.05$.

HR recovery by analyzing the first 30 s of recovery HR *via* semilogarithmic regression analysis and calculated the short-term time constant (T30) (12). The laboratory end-point committee evaluated all HRR responses in a blinded fashion and end points were determined by unanimous decision. The athletes were familiar with the testing procedures as part of their regular training process. A week prior to the testing, the subjects performed a 10 min familiarization trial on the treadmill along with passive recovery in the supine position for 60 s. To ensure that the testing environment was appropriately controlled, the laboratory was kept as quiet as possible during all recovery measurements. The testing room was maintained at 21.4 ± 1.0°C and 25.3 ± 3.2% relative humidity.

Statistical Analysis

The data were expressed as means ± SD. Statistical significance was assessed using Student's *t*-test for independent samples. One-way analysis of variance with repeated measures was used to test the differences between subjects' responses over time.

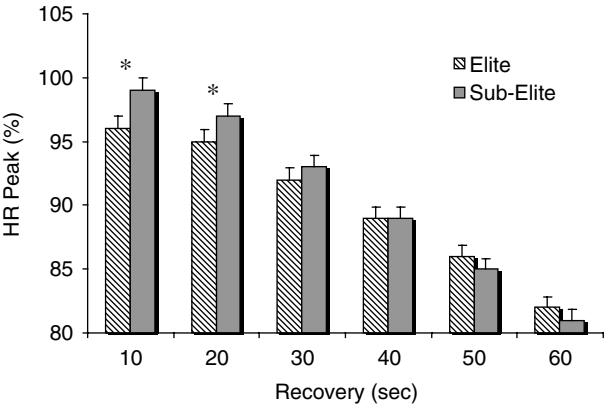

Fig. 1. Heart rate decrease during recovery quantified as percent heart rate decrease from the peak exercise heart rate (HR peak). * Indicates significant difference between elite and sub-elite athletes at $P < 0.05$.

Where significant differences were found, the Tukey test was employed to identify the differences. *P* values less than 0.05 were considered statistically significant. The data were analyzed using the statistical package SPSS, PC program, version 14.0 (SPSS Inc., Chicago, IL, USA).

Results

The subjects had a mean age 20.5 ± 0.6 years, mean body mass 76.5 ± 6.0 kg, and mean height 184.6 ± 5.0 cm (Table 1). The time between the exercise cessation and undertaking supine body position was similar for both groups of subjects (5.0 ± 1.5 s and 5.3 ± 1.4 s for the elite and sub-elite groups, respectively; $P > 0.05$). Table 2 shows the results of the HRR measurements. HR recorded at the end of exercise (*i.e.*, the start of recovery) was similar. Elite group revealed a significantly lower HR than in their sub-elite counterparts for both the 10 and 20 s of recovery period ($P < 0.05$). However, no differences between the groups were found at later stages of the analyzed HRR. Similar results were found when %HR dropouts were compared between the groups (Fig. 1). The 1-min beat-to-beat HRR with semilogarithmic transformation is shown in Fig. 2. The short-term time constant (T30) was not different between the groups (134.6 ± 32.8 s and 140.9 ± 48.6 s for the elite and sub-elite groups, respectively; $P > 0.05$).

Discussion

To our knowledge, this is the first study that has directly revealed the influence of aerobic capacity on the ultra short-term post-exercise HR in athletes. The results obtained in this study generally suggest that

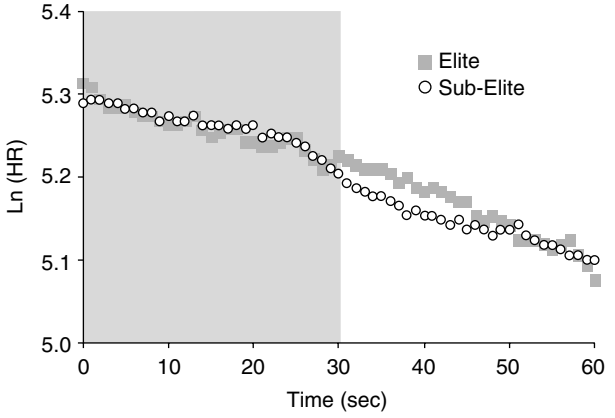

Fig. 2. Natural log-transformed heart rate (HR) with data averaged across participants during recovery following the maximal exercise in elite (n = 12) and sub-elite (n = 14) athletes. Gray shading represents initial 30-s period from which T30 is calculated. For the sake of clarity, error bars have not been added.

the soccer players with higher aerobic fitness could have lower HRR at 10 and 20 s following the maximal exercise, as compared with their counterparts characterised by lower VO_{2max}. The results suggest that the aerobic fitness along with the autonomic modulation might play a role in the ultra short-term cardiovascular responses to all-out exercise.

UST-HRR has not been previously reported in athletes. However, according to the work rate analysis, average time to recover following a high intensity running bouts is less than 15 s in ball games (7). Therefore, a faster ultra short-term cardiovascular response (*i.e.* lower HRR at a given time during the recovery period) reflects a positive adaptation to exercise training and, possibly, a superior capacity in endurance events (4). Therefore, it could be postulated that the athletes characterized by lower HRR during first 20 s post-exercise could be better adapted to maximal exercise due to several possible mechanisms, such as the restoration of parasympathetic tone, changes in plasma volume, or accumulation of metabolic factors (14). In the present study we found that the recovery HR at 10 and 20 s following the exercise is accelerated in athletes with VO_{2max} > 60 ml/kg/min. Several authors have shown that the endurance-trained athletes could have faster heart rate responses after the cessation of exercise, as compared with sedentary individuals (3, 8, 19). These findings could be explained by increased parasympathetic tone in trained athletes, along with withdrawal of sympathetic stimulation that contributes to the deceleration of HR after the cessation of exercise. Yamamoto *et al*. (29) found a relationship between an increase of parasympathetic tone and accelerated HRR following endurance training. However, there are no comparasions between HRR in athletes with similar training status. In the present study both the elite and sub-elite players were engaged in similar training program 8 weeks before the exercise testing. Therefore, it is likely that the changes in autonomic control of HR induced by training could be similar in two groups. As a result, the level of aerobic fitness (VO_{2max}) *per se* could induce changes in HRR and, therefore, the athletes with higher VO_{2max} could be more prone to rapid decrease in HR during ultra short-term recovery. This is in line with previous results (8) where post-exercise HR was quicker in the individuals characterized by higher aerobic capacity, although the underlying physiological mechanisms remain to be elucidated. Possible mechanisms of HR deceleration in athletes with high VO_{2max} during ultra short-term recovery that are not related with autonomic control, could be due to rapid changes in maximal left ventricular (LV) performance with increased ejection fraction and myocardial contractility (22), plasma volume disturbances with increased LV filling (11), myocardial lactate and/or nitric oxide metabolism (10), changes in gene expression (27) which requires further investigation.

The rapid recovery of HR following a moderate-to-heavy exercise may be an important mechanism in preventing excessive cardiac work, which could also have important implications for athletic training. Several investigators have shown that the endurance-trained athletes have faster HR responses after the cessation of exercise as compared with sedentary subjects (8, 19, 24). Of importance here could be that the parasympathetic activation is considered to be the main mechanism underlying exponential cardio-deceleration following the exercise (26). Different markers of HRR kinetics seems to be regularly used in both athletes and non-athletes as indicators of cardiovascular autonomic function (6). It has been suggested that short-term HRR indices (such as T30 and HRR_{60s}) could be considered as noninvasive indexes of cardiac parasympathetic outflow (12, 16). Since T30 and HRR_{60s} appear to be similar in the two groups tested in the present study, one could hypothesize that the parasympathetic activity assessed by the above mentioned markers could not be influenced by the differences in aerobic power. In contrast, the first ultra rapid HRR phase is found to be related to aerobic fitness of elite and non-elite soccer players. In line with our findings, Buccheit and co-workers (6) suggested that parasympathetic system is not the unique determinant of HRR indexes, as well as that the recovery in HR after all-out exercise could be mediated by intrinsic, neural, and humoral factors (26). Pierpont and Voth (21) revealed the time constants for parasympathetic reactivation of 44 s, as well as 65 s for sympathetic withdrawal, underlining other mecha-

nisms of HRR control. Other factors contributing to HRR following a physical activity have been thought to be slower changes in the stimuli to metaboreceptors and baroreceptors accompanying clearance of metabolites and delayed elimination of body heat and catecholamines (14). Taken together, these findings suggest that HRR kinetics may be influenced by both endurance conditioning and genetics, which certainly requires further investigations.

While our study revealed a set of rather novel data, it was also constrained by several limitations. First, one could question the selection of subjects since soccer players could not be proper subjects for addressing the stated research question, because they are not typical endurance trained athletes. The aerobic system is the main source of energy provision during soccer match-play, and the average values of VO_{2max} for elite soccer players generally fall toward the center of the continuum of the maximal oxygen consumption of elite athletes of different specialization (25). Indeed, VO_{2max} of sub-elite players (*i.e.*, the values below 50 ml/kg/min) suggest a poor training condition, thus making the comparison performed between trained and active (if not almost sedentary) subjects. A relatively small number of the tested subjects could also lead to an overestimation of the differences between the elite and sub-elite groups. Moreover, it is well known that the endurance training affects HRR (2, 3) and although we selected subjects with similar training program during the 8 weeks prior to the experiment, data with pretesting the absolute and relative workloads are lacking. Passive recovery mode applied in the present study has been frequently used in the past (14, 23), yet it does not reflects the real sport situation with jogging or walking during recovery. Finally, Bosquet *et al*. (4) highlighted numerous factors that can affect HR measurement, such as the emotional state, digestion, noise, infection, pharmacological and non-pharmacological substances known to influence the autonomic nervous system. Therefore, minimizing the influence of these variables may decrease the error of measurement. Finally, the present study is only a descriptive one that does not address any of the possible physiological mechanism underlying the observed phenomenon. Further investigation should focus on the relationship between the short-term recovery HR and acute changes in training volume and intensity, particularly in the top-level endurance athletes (*e.g.* long-distance runners, triathletes). Future research would be greatly improved if some biochemical markers of anaerobic metabolism, circulating cateholamines, or plasma volume were measured immediately following the maximal exercise.

In conclusion, the athletes characterized by high aerobic capacity could be better adapted to maximal exercise by faster HRR immediately following an exercise test, particularly over the first 10 and 20 s. Measuring UST-HRR over short intervals on a regular basis, particularly after all-out exercise under controlled conditions, may enable coaches to easily monitor how athletes are responding to exercise and, therefore, facilitate the training optimization. The results of the present study generally support the theories of coordinated interaction of mechanical, humoral and autonomical control during exercise short-term recovery and, conversely, does not support employing exercise HRR as index of vagal function alone.

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