Medicina Sportiva
Med Sport 17 (4): 171-175, 2013
DOI: 10.5604/17342260.1081273

HEART RATE VARIABILITY IN MEN SUBJECTED TO 30 HOURS OF EXERCISE AND SLEEP DEPRIVATION

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Abstract

Introduction: Heart rate variability (HRV) is considered to be a factor determining condition of the heart, especially its fatigue and possible overtraining. Impact of ultra-endurance exercise, additionally involving sleep deprivation, on HRV is poorly investigated so far.

Objective: To evaluate whether prolonged physical activity combined with complete sleep deprivation might alter the cardiac autonomic control in healthy young men.

Methods: Indices of HRV (HRV SDNN, pNN50, rMSSD and LF/HF ratio) were assessed in 11 endurance trained men before (trial B) and after (trial A) 30 hours of prolonged exercise combined with complete sleep deprivation using infrared sensor fitted in BioCardio Mouse (Poland).

Results: The values of heart rate (HR) were significantly higher and the HRV time domain indices were significantly lower in trial A as compared to the results obtained in trial B (pNN50 10.6 ± 2.3 ms vs.18.3 ± 2.0 ms, $P < 0.001$; rMSSD 45 ± 6 ms vs.66 ± 6 ms, $P < 0.05$ and SDNN 53 ± 7 ms vs.74 ± 5 ms, $P < 0.01$, respectively). The LF/HF ratio was significantly lower in trial A than in trial B (1.12 ± 0.11 vs. 1.38 ± 0.11, $P < 0.05$).

Conclusions: Ultra-endurance exercise (30-hours adventure race) combined with complete sleep deprivation seems to affect cardiac autonomic response (lower rMSSD, pNN50, SDNN and LF/HF ratio), thus reflecting a sustained sympathetic outflow during recovery from prolonged exercise. The new, simple and cheap BioCardio Mouse delivered consistent and reliable results of several factors determining HRV. Automatically generated reports are easy to follow and raw results can easily be used in everyday monitoring of athletes.

Key words: *heart rate variability, ultra-endurance exercise, sleep deprivation*

Introduction

It was demonstrated, that endurance trained people have beneficial adaptive changes in the cardiovascular system resulting from regular physical activity. These changes occur both in the heart (increase in stroke volume and in the myocardial contractility, intrinsic heart rate reduction, left ventricular hypertrophy) and in the peripheral vessels, mostly in skeletal muscles (decrease in total peripheral vascular resistance). It is known, that endurance training increases parasympathetic activity and decreases sympathetic activity directed to the heart at rest [1]. Some authors believe that the resting bradycardia observed in endurance trained individuals is a consequence of pacemaker cell alterations and intrinsic heart rate reduction [2, 3]. However, most of spectral analysis studies support the theory that endurance training increases parasympathetic activity [1,4-6].

Among the different available noninvasive techniques used as markers of autonomic modulation of the heart, heart rate variability (HRV) is considered the best for evaluation of the sympatho-vagal balance at the sinoatrial level [7]. In sports medicine HRV is used especially in monitoring a state of fatigue that could result in overtraining [8]. It was also described as a valuable factor applied for evaluation of effectiveness of altitude training [9]. Changes in HRV are present during phenomena disturbing homeostasis, from physiological like exercise to pathological, meaning most diseases [10].

Breuer et al. [11] found that HRV decreases exponentially as exercise intensity increases. This was due to a progressive decrease in parasympathetic activity during light and moderate exercise. Martinmäki et al. [12] and Kaikkonen et al. [13] reported that the increased intensity of exercise resulted in slower recovery of high frequency power computed from R-R interval data as well as lower levels of high frequency power when compared to low intensity exercise. It should be noted, that the high frequency power (0.15-0.40 Hz at rest and 0.15-1.0 Hz during exercise) is reflecting vagal effects on the heart. It was also demonstrated, that sleep deprivation just like any other exercise or stress induce autonomic imbalance and that this condition makes the HRV indices worse i.e. decreased the high frequency spectra and increased the sympathetic activity measured by HRV indices [14-16].

In the literature, a few reports are available on HRV either during prolonged exercise alone [17,18], or sleep deprivation without exercise [19,20]. Impact

of prolonged activity, especially ultra-endurance exercise additionally involving sleep deprivation, on HRV during the immediate recovery period is poorly investigated so far.

Ultra-endurance exercises become more popular in recent years, many running or cycling events, and especially adventure races, last longer than 24 hours and involve at least one night of sleep deprivation. Exercising in such an extreme form severely affects function of the heart and it is currently a hot topic, whether ultra-endurance is safe for health or could it lead to cardiac damage or fatigue phenomena.

Thus, the aim of this study was to evaluate autonomic cardiac control after the cessation of prolonged activity (30-hours` simulated adventure race) combined with complete sleep deprivation by using the HRV indices. The best and the most reliable tool for measurement of HRV is a full ECG analyzed by sophisticated software and statistics, so its application in sports field is limited. Perfect solution would be the simple device like a watch heart rate monitor or infrared sensor, which would still deliver pretty reliable and reproducible results. Therefore, in the present study we used simple, commercially available computer mouse with built in infrared sensors recording several indices determining HRV.

Methods

Participants

The study was performed with 11 men aged 31±2 years with BMI 22.2 ± 0.3 kg/m^2 (body mass 71 ± 1 kg; height 179 ± 2 cm) and $\dot{V}O_2$max 56±4 ml/kg/min. They were recruited from endurance trained amateur athletes with experience in ultra-endurance running, orienteering and adventure racing and found to be in good health. All subjects gave their informed consent to participate in the study. The investigation conformed with the principles outlined in the Declaration of Helsinki and was approved by Ethics Committee of Warsaw Medical University, permission number KB/213/2010.

Exercise protocol

Exercise protocol was a 30-hours' long simulated adventure race and was performed in the field. Subjects were trekking, mountain biking, running, kayaking and roller skating in 7 stages with short breaks for change of equipment and feeding between stages. Proper hydration and nutrition was thoroughly monitored by means of body mass and urine osmolality. During the whole 30 hour period subjects were not allowed to get any sleep.

Heart rate variability was assessed at rest in comfortably sitting conditions using infrared sensor fitted in the side of computer mouse (BioCardio Mouse, Poland). Data collection took approximately 5 minutes by simply resting with right hand frozen on computer mouse and thumb located on infrared sensors. All the tests were carried out in the laboratory under similar environmental conditions (23–24°C and 40–50% humidity) between 10:00 and11:00 a.m. Measurements were performed on two occasions in random order, one as control before (trial B) and second within 60 minutes after (trial A) 30-hours of prolonged exercise combined with complete sleep deprivation.

For evaluation of HRV standard deviation of average R-R intervals (SDNN), proportion in % of successive R-R intervals different by more than 50 ms (pNN50) and time domain square root of the mean squared difference of successive R-R intervals in ms (rMSSD) as well as low and high frequency power spectral components were chosen.

Statistical analysis

Statistical analysis was performed with Statistica 6.0 software. Data are presented as means ± SE, distribution of results was analyzed by Shapiro-Wilk`s test and accordingly paired t-test or Wilcoxon test were used to compare results. Level of significance was set at $P < 0.05$.

Results

The prolonged 30-hours' simulated adventure race combined with complete sleep deprivation significantly increased HR, decreased R-R intervals and reduced HRV time-domain indices.

The values of pNN50, rMSSD and SDNN were significantly lower in trial A compared to the control trial B. The values of HRV frequency-domain indices showed no significant differences between the trials, whereas LF/HF ratio significantly decreased from 1.38±0.11 in trial B to 1.12±0.11 in trial A, ($P < 0.05$).

The comparison between the HRV time-domain indices in the two trials is illustrated in Table 1. Examples of original reports of one subject in trial B and in trial A are presented in Fig.1 and 2.

It should be noted that the results obtained with BioCardio Mouse were consistent and reproducible, there were neither hardware nor software problems during the data collection.

Table 1. *Comparison of heart rate and heart rate variability time-domain indices before (trial B) and after (trial A) 30 hours of prolonged exercise and sleep deprivation; *P < 0.05, ** P < 0.01, *** P < 0.001*

	HR [bpm]	R-R interval [ms]	SDNN [ms]	PNN50 [ms]	RMSSD [ms]
Trial B	67±2	913±32	91±20	18.3±2.0	66.5±6.0
Trial A	74±2**	819±24**	55±6**	10.6±2.3***	45.4±5.9*

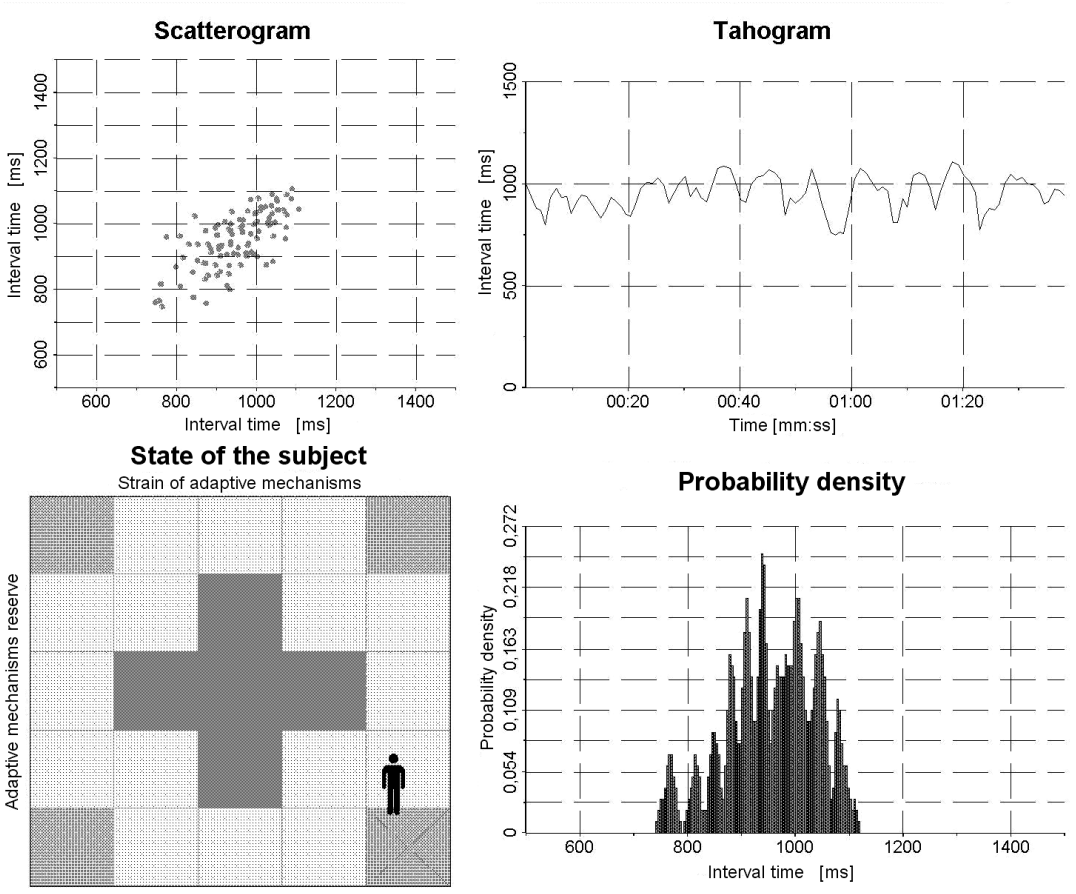

Fig. 1. *Original report of one subject in control trial B (before 30 hours of prolonged exercise and sleep deprivation)*

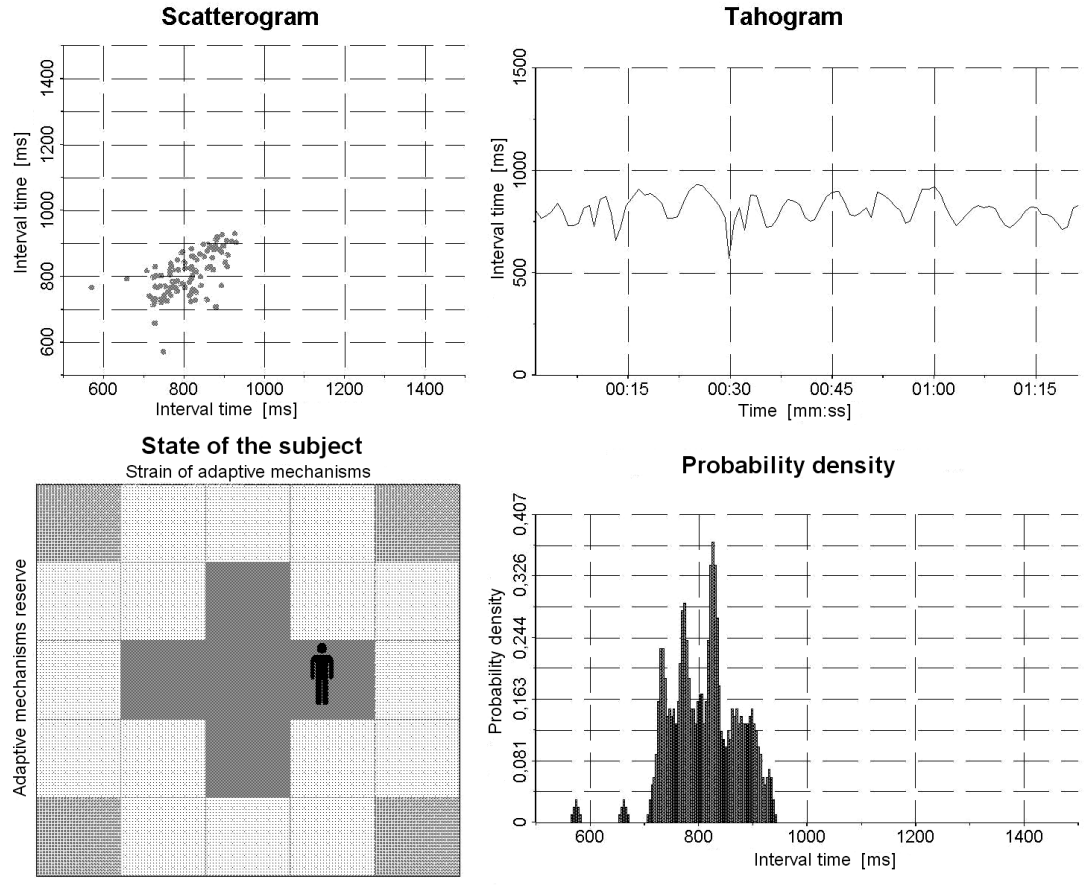

Fig. 2. *Original report of one subject in trial A (after 30 hours of prolonged exercise and sleep deprivation)*

Discussion

The present data showed that in healthy young men prolonged, 30-hours moderate intensity exercise combined with complete sleep deprivation decreased both HRV time-domain indices reflecting vagal modulation (pNN50, rMSSD, SDNN) and LF/HF ratio.

The alterations in HRV time-domain indices are consistent with the findings of Stewart et al. [21] who reported decreases in time-domain parameters of the R-R interval and increases in LF/HF ratio during 60-minute of recovery from prolonged exercise. The authors concluded that decreased rMSSD and total power of R-R intervals reflects reduced parasympathetic cardiac modulation, while the increased LF/HF ratio suggest sustained sympathetic outflow during recovery from prolonged strenuous exercise. The authors also point to the fact that the sustained sympathetic activity may influence depolarization and repolarization of the ventricles, since the mean QT interval returned to pre-exercise values after 24 hours.

Based on previous studies [12,13], it may be speculated that the low LF/HF ratio, observed in trial A of this study, resulted from slower recovery of high frequency power computed from R-R interval data or lower levels of high frequency power due to the increased intensity of exercise.

It has been generally accepted that HRV is a useful parameter for the quantification of autonomic nervous function, especially to reflect changes in cardiac-autonomic regulation. HRV time-domain indices, such as pNN50, SDNN and rMSSD as well as high frequency power spectral component (0.15-0.5 Hz) provide information related to parasympathetic activity. The low frequency power (0.01-0.08 Hz) is regulated by both sympathetic and parasympathetic nervous systems [21-24].

Several studies demonstrated that a combination of physical or mental stress and chronic sleep deprivation aggravate autonomic imbalance and magnesium deficiency-related vasomotor abnormality, which subsequently develops cardiovascular events [25-29]. There are only a few studies examining physiological strain of multi-day, prolonged ultra-endurance exercise and sleep deprivation. These studies demonstrated multi-factorial fatigue process, cardiac dysfunction, expansion in plasma volume, impaired thermoregulatory processes and elevation of phagocyte-related immune cell numbers [25-27]. It was also demonstrated that sleep deprivation just like exercise induce autonomic imbalance, and that this condition makes the HRV indices worse [14-16].

Adventure racing, used in the present study, was a combination of several endurance disciplines, including running, biking, orienteering, kayaking, with complete sleep deprivation (trial A). The values of heart rate and HRV time-domain indices obtained in trial A were significantly lower than those obtained before exercise (trial B).

From the literature it is known that at low to moderate exercise intensity an increase in heart rate is mediated primarily by parasympathetic withdrawal, and that sympathetic activity increased initially at 60% of $\dot{V}O_2$max [30]. It is also known that heart rate recovery to resting levels can take one hour after light or moderate aerobic exercise [31], four hours after long-duration exercise [32], and up to 24 hours after intense or maximal exercise [33]. The authors suggest that it depends on the interaction between factors such as exercise intensity [34], cardiac autonomic modulation, and the level of physical fitness [35-37].

A delayed heart rate recovery may be a reflection of autonomic imbalance resulting from a reduction in vagal tone or an exaggerated sympathetic activation [38, 39]. Some authors demonstrated that athletes, who had higher vagal activity, had faster heart rate recovery than non-athletes [40,41].

Thus, it seems likely that the delayed heart rate recovery and lower HRV time-domain indices, observed in the current study, after 60 minutes of recovery from ultra-endurance moderate exercise combined with complete sleep deprivation may reflect sympathovagal imbalance at the sinoatrial level resulting from reduced parasympathetic cardiac modulation and/or sustained sympathetic modulation of heart rate during recovery.

The results may contribute to a better understanding of the dynamic nature of autonomic nervous function as well as the consequences of ultra-endurance exercise.

Declaration of interest

The authors report no conflicts of interest.

Acknowledgements

The study was financed by the grant of Ministry of Education Nr IV.B.01 60/2011/PW-PB.

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Accepted: November 28, 2013
Published: December 20, 2013

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