James, David V ORCID logoORCID: https://orcid.org/0000-0002-0805-7453, Barnes, A J, Lopes, P. and Wood, D M (2002) Heart rate variability: response following a single bout of interval training. International Journal of Sports Medicine, 23 (4). pp. 247-251. doi:10.1055/s-2002-29077

Official URL: http://www.ncbi.nlm.nih.gov/pubmed/12015624
DOI: http://dx.doi.org/10.1055/s-2002-29077
EPrint URI: https://eprints.glos.ac.uk/id/eprint/114

ORIGINAL ARTICLE: TRAINING AND TESTING

David V.B. James, Anthony J. Barnes, Philippe Lopes and Dan M. Wood

Heart rate variability: response following a single bout of interval training

Running title: Heart rate variability following exercise

DVB James (⌧) A Barnes and DM Wood
Cheltenham and Gloucester College of Higher Education
Leisure and Sport Research Unit
Francis Close Hall
Swindon Rd
Cheltenham
Gloucestershire
GL50 4AZ
U.K.

P Lopes
University of East London
Dept of Health Sciences
Stratford Campus
Romford Road
Stratford
London
E15 4LZ
U.K.

Tel: 01242 543295
Fax: 01242 543283
email: djames@chelt.ac.uk
⌧Author to whom all correspondence should be addressed

Abstract

We investigated the effect of exercise on heart rate variability by analysing the heart rate power spectrum prior to, and 1 and 72 h following, an interval training session. Subjects initially performed a graded test to exhaustion to determine maximal oxygen uptake ($\dot{V}O_{2max}$) and the running speed at which $\dot{V}O_{2max}$ was first attained (v$\dot{V}O_{2max}$). The training session was completed on a separate day and comprised six 800 m runs at 1 km.h^{-1} below v$\dot{V}O_{2max}$. Prior to the training session (pre), 1 h following the training session (+1 h), and 72 h following the training session (+72 h), subjects sat quietly in the laboratory for 20 min whilst breathing frequency was maintained at 12 breath.min^{-1}. Cardiac cycle R-R interval data were collected over the final 5 min of each 20 min period and analysed by means of autoregressive power spectral analysis to determine the high frequency (HF) and low frequency (LF) components of heart rate variability. Heart rate was higher, and the standard deviation of the R-R intervals was lower, at +1 h than for pre or +72 h ($P<0.05$). The HF and the LF components of heart rate variability were also lower ($P<0.05$) for +1 h than for pre or +72 h when the data were expressed in ms^2. However, no changes in the LF:HF ratio were observed, and the changes in the HF and LF components disappeared when the data were expressed as a fraction of the total power. Whilst these findings illustrate the importance of controlling the timing of exercise prior to the determination of heart rate variability, the time course of the post-exercise heart rate variability response remains to be quantified.

Key words

Overload - autonomic nervous system - respiratory sinus arrhythmia

Introduction

The examination of heart rate variability (HRV) in the frequency domain is a non-invasive technique that has been used to assess autonomic nervous system influences on the heart [(13)]. Recently HRV has been studied in athletes, both during normal training and detraining [(3)] and during a period of overtraining [(14)]. It is likely that the assessment of the autonomic nervous system in athletes will increase over the next few years, since the autonomic nervous system is known to be important in the aetiology of the overtraining syndrome [(10); (8)].

It is generally assumed that recovery following a single bout of running exercise is normally complete after 72 h of recovery, and previous studies suggest that this is the case [e.g.,(6)]. However, performance has rarely been assessed during the days of recovery following a single bout of running exercise, with the exception of a marathon run [(12); (11)]. Not surprisingly after such an extreme running overload, these studies demonstrated prolonged fatigue that extended beyond 72 h. Physiological changes that would be expected to decrease performance capability have been demonstrated to recover within 72 h following a more ‘moderate’ single bout of exercise. These physiological changes include muscle glycogen [(1)] and plasma volume [(9)]. These studies provide a solid basis for the approach taken in the present study, which attempted to characterise the response of heart rate variability following a single bout of interval training at a point of definite fatigue and recovery [(6)].

Very few studies have examined the response of HRV following a single bout of exercise. Furlan and colleagues [(3)] studied untrained subjects 1, 24 and 48 h after a 30 min bout of high intensity exercise, whereas Bernardi and colleagues [(2)] studied trained runners 0.5, 24 and 48 h after a 46 km trail run. Both groups found that the power of the LF and HF components of the HRV power spectrum and the LF:HF ratio were elevated in the hour following exercise but had returned to pre-exercise levels 24 h after the exercise bout. There was, however, an important methodological difference between the two studies: whereas Furlan accounted for changes in the total spectral power in their analysis by expressing the LF and HF powers relative to this total power as well as in absolute units (ms^2) and analysing changes in both relative and absolute powers, Bernardi made no attempt to account for changes in total power.

The HRV response following a single bout of exercise may differ between trained and untrained subjects. However, the exercise bout studied by Bernardi et al. [(2)] differs from that which individuals would routinely perform in training due to its length (few athletes would routinely perform a 46 km run) and the fact that it was completed at an altitude of 2500 m. In the present study we investigated the HRV response of trained individuals following a single bout of interval exercise similar to that which many athletes might routinely perform in training. To evaluate the impact of changes in total power on this response we performed our analyses with and without correcting for changes in the total spectral power.

Materials and Methods

Eight trained male sports students (mean (SD): age 22 (2) years; height 1.81 (0.08) m; body mass 79.1 (8.1) kg; $\dot{V}O_{2max}$ 53.6 (4.4) $ml.kg^{-1}.min^{-1}$) volunteered to take part in the study after being informed of the nature of the study and the potential risks. In accordance with the requirements of the Institution's ethics committee, all subjects gave written informed consent and completed a medical history and health questionnaire. Subjects were involved in training for a variety of sports but they all included interval training sessions similar to the one they performed in this study as part of their regular training.

Each subject visited the laboratory on three occasions. Visits 1 and 2 were separated by 7 days and visits 2 and 3 were separated by 3 days (exactly 72 h). On their first visit subjects completed a graded running test to exhaustion. The second visit included 20 min of quiet sitting, followed by an interval training session (TS), an hour of recovery and rehydration, and a further 20 min of quiet sitting. On their final visit subjects completed only the 20 min of quiet sitting. Subjects were requested not to eat or drink anything other than water in the final 4 h before each visit and to perform no exercise, beyond normal lifestyle activities, between the second and third visits to the laboratory (see figure 1). In the final hour prior to each visit to the laboratory, subjects were instructed to abstain from consuming any fluid.

INSERT FIGURE 1 ABOUT HERE

Both the graded test and the TS were performed on a motorised treadmill (Ergo ELG 70, Woodway, Weil am Rhein, Germany) with the gradient set at 0%. The starting speed for the graded test was selected to ensure that exhaustion was reached in ~10 min with speed being increased by 0.75 km.h^{-1} every 45 s. This test was used to determine $\dot{V}O_{2max}$, the speed at which $\dot{V}O_{2max}$ was first attained (v$\dot{V}O_{2max}$), and maximum HR (HR_{max}). Expired air was collected continuously over ~45 s periods and the highest $\dot{V}O_2$ attained was taken as $\dot{V}O_{2max}$.

The TS comprised six 800 m runs at 1 km.h^{-1} below v$\dot{V}O_{2max}$ separated by 3 min recovery periods. Each subject was weighed before and after the TS and the change in body mass was calculated. The equivalent fluid volume was then determined and the subject was required to consume this fluid immediately on completion of the TS. This rehydration strategy has been used in previous studies and has been shown to be effective at restoring plasma volume [(4)].

Throughout the graded test and the TS subjects wore a chest strap and heart rate (HR) was measured by short range telemetry (Vantage NV, Polar Electro Oy, Kempele, Finland). Throughout the graded test subjects wore a nose clip and breathed through a large, broad flanged rubber mouthpiece (Hans Rudolf, Kansas, USA) fitted to a low-resistance (inspired <3 cmH_2O and expired <1 cmH_2O at 350 $L.min^{-1}$) breathing valve (Cranlea, Birmingham, UK) of negligible volume (90 ml). A 150 L Douglas bag was connected to the expired side of this valve via a 1.5 m length of light weight Falconia tubing (3.5 cm internal diameter) (Cranlea, Birmingham, UK). A whole number of breaths were collected and the collection was timed using a digital stopwatch.

Expired fractions of O_2 and CO_2 were measured using a paramagnetic O_2 analyser and an infrared CO_2 analyser (1440 series, Servomex, Crowborough, UK). Bottled nitrogen was used to set the zero for both analysers, fresh (outside) air was used to set the span for the O_2 analyser, and a gravimetrically prepared mixture (4% CO_2, 16% O_2, balance N_2; Cryoserve, Worcester, UK) was used both to set the span for the CO_2 analyser and to check the linearity of the O_2 analyser. All gas mixtures were first saturated (Nafian tubing (Omnifit, Cambridge, UK) in water) and then cooled to 5 °C (Bühler PKE3, Paterson Instruments, Leighton Buzzard, UK) before they entered the gas analysers. Gas volume was measured using a dry gas meter (Harvard Apparatus Ltd., Edenbridge, UK), which was calibrated and checked for linearity throughout the typical collection volume range using a 3 L calibration syringe (Hans Rudolf, Kansas, USA).

During the tests at 1 hour prior to the TS (T1), 1 hour following the TS (T2) and 72 hours following the TS (T3), subjects sat quietly for 20 min and controlled their breathing frequency (BF). BF was set at 0.20 Hz (12 $breath.min^{-1}$), with each breath comprising 2 s of inspiration and 3 s of expiration. Subjects wore a chest strap consisting of two electrodes and a transmitter

(Polar Electro Oy, Kempele, Finland) and the data were transmitted directly to a PC via an interface (Advantage, Polar Electro Oy, Kempele, Finland). R-R interval data were collected over the final 5 min of the 20 min period and stored for subsequent analysis (Precision Performance 2.1, Polar Electro Oy, Kempele, Finland). The data were initially filtered using median and moving average based methods to minimise artifacts in the ECG signal. (Normally such artifacts are a result of the wireless transmission system which may be influenced by an external electromagnetic field.) The mean and standard deviation of the R-R intervals were then calculated and a power spectrum analysis was undertaken (using autoregressive modelling with a fixed model order of 18).

The HRV power spectrum can be divided into three frequency bands: high frequency (HF), low frequency (LF) and very low frequency (VLF) ((3); (13)). For the HF component, which is synchronous with respiration, a frequency band of 0.16 to 0.24 Hz was selected (BF was controlled at 0.20 Hz). For the LF component, which typically ranges between 0.03 and 0.15 Hz and is normally observed at ~0.1 Hz, a frequency band of 0.04 to 0.16 Hz was selected. Finally, for the VLF component, a frequency band of 0.00 to 0.04 Hz was selected. For the HF and LF components power was expressed both in ms^2 and in normalised units. The normalisation procedure involves dividing the HF or LF power (ms^2) by the total spectral power minus the VLF component (also in ms^2) and the result is therefore a dimensionless ratio. The use of normalised units is thought to minimise the influence of changes in total power on the HF and LF powers [(13)].

Differences between T1, T2 and T3 were evaluated using repeated measures analysis of variance and Newman-Keuls post hoc tests at the 0.05 alpha level. As the data for the spectral parameters were positively skewed (prior to normalisation), these data were transformed via a natural logarithmic function prior to analysis. This is consistent with the approach of Bernardi and colleagues [(2)] who, having found that the data for the spectral parameters were skewed, transformed the data with a log transformation. Data for T1, T2 and T3 are presented as mean (68% confidence interval) as it is not possible to 'back-transform' a log transformed standard deviation into the original measurement units. The presentation of data as mean (68% confidence interval) is consistent with the normal convention of presenting data as mean (one standard deviation).

Results

The TS was a heavy overload for the subjects, as shown by their heart rate response (Figure 2). The mean (SD) heart rate at the end of each bout increased from 177 (5) to 193 (5) beats.min^{-1} over the course of the six 800 m bouts. These figures should be compared with the maximum HR of 198 (5) beats.min^{-1} obtained during the graded test. The TS elicited heart rates of between 87 % and 98 % of maximum.

INSERT FIGURE 2 ABOUT HERE

Data for resting HR, the standard deviation of R-R intervals (RRSD), and spectral parameters of HRV are presented in Table 1. No differences were observed between T1 and T3 for any of the variables ($p>0.05$). However, some changes were observed between T1 and T2 that were then reversed between T2 and T3. Resting HR increased by 8 beats.min^{-1} between T1 and T2 and decreased by 12 beats.min^{-1} between T2 and T3. The RRSD showed an opposite pattern, decreasing by 18 ms between T1 and T2 and increasing by 26 ms between T2 and T3. Both LF and HF power decreased (by 1679 and 695 ms^2 respectively) between T1 and T2 and increased (by 2878 and 444 ms^2 respectively) between T2 and T3. Since total power also decreased between T1 and T2 and increased between T2 and T3 (by 2617 and 3969 ms^2 respectively), when the LF and HF powers were expressed in normalised units no changes were observed ($p>0.05$). The LF:HF ratio was not significantly altered at T2 relative to T1 and T3 ($p > 0.05$).

INSERT TABLE 1 ABOUT HERE

Discussion

The TS used in the present study provides a tolerable but heavy overload [(4)] and results in impaired performance 1 h following exercise [(6)] despite rehydration, normalised core temperature, and normalised blood lactate concentration ($[La^-]_B$) at rest [(5)] in similarly trained subjects. It is also thought to be a realistic TS - one that athletes would regularly undertake as part of their training programme.

The finding of increased HR 1 h following the TS is consistent with previous studies [(3);(4)]. Analysis of HRV in the time domain through the standard deviation of R-R intervals showed a decrease in HRV 1 h following the TS. Whilst this change in HRV is suggestive of a change in autonomic activity, it is impossible to attribute the change to parasympathetic or sympathetic influence. For example, the fact that a smaller variability was found in conjunction with a higher heart rate is likely to be partly a consequence of the reduced baroreflex influence on heart rate adjustment within a heart beat as the RR interval decreases. By examining HRV in the frequency domain it is possible to partition the effect of the parasympathetic and sympathetic nervous system. The HF component of the HRV power spectrum is centred at the BF and has been used as a non-invasive, indirect measure of cardiac parasympathetic tone [(7)]. In contrast, the low frequency component is thought to reflect slow oscillations of the arterial pressure variability signals (at ~0.1 Hz) and has been used as an indirect measure of cardiac sympathetic tone [(3)]. To ensure no overlap between the two components it is necessary to keep BF quite high. If BF is uncontrolled, or controlled at a low rate, overlap can occur between the HF and LF components of the power spectrum making it difficult to reliably determine the power of each component [(13)]. Our use of a controlled BF of 0.20 Hz (12 breath.min^{-1}) in the present study is consistent with the findings of Strano and colleagues [(13)] which suggest that BF should be maintained at between 12 and 15 breath.min^{-1} (0.20 and 0.25 Hz).

In the present study, LF, HF and total power (ms^2) were reduced 1 h following the TS but both had returned to the pre-TS level at 72 h. No changes were observed in the LF:HF component and the changes in LF and HF power disappeared when the data were expressed in normalised units. Furlan and colleagues [(3)], who studied untrained individuals, also found that LF, HF and total power (ms^2) were reduced 1 h following an exercise bout. However in these untrained subjects the LF:HF ratio was increased 1 h following exercise and the changes in the HF and LF powers were still present when the data were expressed in normalised units. The changes in HF power were in the same direction regardless of whether the data were expressed in ms^2 or in normalised units but when the LF power was expressed in normalised units an increase was observed from pre to 1 h post exercise. Whether the different findings relate to the training status of the subjects, the severity of the overload, or some other factor is unclear. Whilst Furlan and colleagues do not give any data regarding the subjects' physiological characteristics, it is likely that they differed somewhat from our subjects who had a $\dot{V}O_{2max}$ of 53.6 ml.kg^{-1}.min^{-1} and were trained. Importantly, our subjects were used to undertaking training sessions similar to the TS they performed in the present study. Furlan and colleagues do give some information about the exercise bout that was undertaken by their subjects. It comprised a graded test to exhaustion followed by 4-6 repetitions and the total exercise time was ~30 min. No information is presented on these repetitions and the physiological responses are not described. It is difficult, therefore, to determine whether the exercise was more or less severe than the TS that our subjects performed. Although Furlan failed to control breathing frequency it is unlikely that overlap between the LF and HF components would have presented a major problem in their study as the breathing frequency adopted by their subjects, and thus the central frequency for the HF component, was ~0.30 Hz.

In the study by Bernardi and colleagues [(2)], their well-trained subjects were subjected to a very severe overload at altitude. Their findings, however, were similar to the findings in the present study. Thirty minutes after the overload, the LF and HF components of the power spectrum were both reduced, although the HF component was reduced further than the LF component such that the LF:HF ratio increased. Bernardi and colleagues reported their LF and HF powers in ms^2. Neither total power nor the power for the VLF component was reported and therefore it is impossible to determine how the LF and HF powers would have changed in response to the overload had they been expressed in normalised units.

Whilst we can only speculate about the time course of the changes over the 72 h period in the present study, the study by Bernardi et al. [(2)] might shed some light on this issue. At the 24 h time point following the overload, both the LF and HF components of the power spectrum returned to baseline (pre-overload) values. We chose to examine the point of most extreme disturbance (1 h post-TS) in the present study as a preliminary investigation. A further development would be to track the changes between 1 and 72 h post-TS. As the time course of the HRV response following exercise remains to be established the results of studies assessing HRV in exercising populations should be interpreted with caution.

Conclusion

The present study has characterised the changes in heart rate variability that occur in trained individuals following an exercise bout that such individuals would regularly undertake as part of their training programme. The findings illustrate the importance of controlling the timing of exercise prior to the determination of heart rate variability. Further studies are required to investigate the time course of the post-exercise heart rate variability response for a given exercise bout. In addition, it would be of interest to investigate the influence of the severity of the exercise overload on the post-exercise response. An understanding of the post-exercise heart rate variability response is necessary if this measure is to be used in the monitoring of athletes in general and the process of overtraining in particular.

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Legend

Figure 1: Schematic of experimental protocol

Figure 2: Mean heart rate during the final stages of each exercise bout for the interval training session.

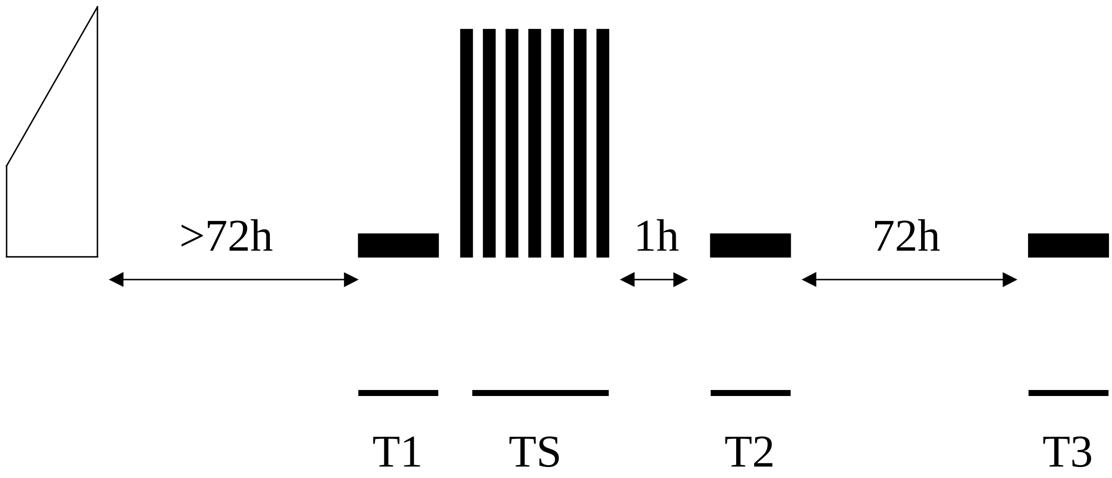
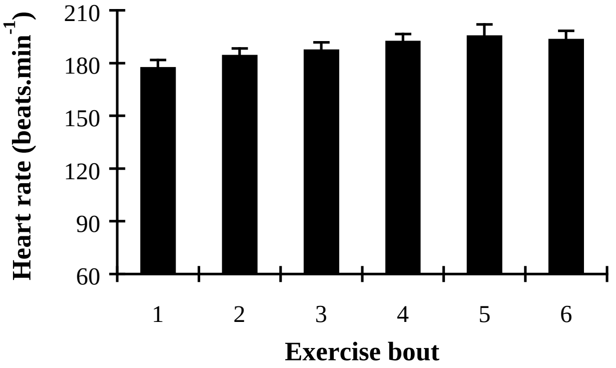

>72h
1h
72h
T1
TS
T2
T3

Figure 2


Heart rate (beats.min^{-1})
210
180
150
120
90
60
1
2
3
4
5
6
Exercise bout

Table

Table 1. Heart rate and heart rate variability parameters prior to and at 1 and 72 h following the training session.

	Time point			P value
	T1	T2	T3	(ANOVA)
HR, beats.min^{-1}	69 (61-77)	77 (64-90)	65 (59-71)	0.002
RRSD, ms	75 (62-88)	57 (33-81)	83 (60-106)	0.011
Total power, ms^2	6257 (4416-8130)	3831 (1162-7322)	7800 (3654-12504)	0.010
LF power, ms^2	3640 (2151-5223)	1961 (621-3621)	4839 (2224-7533)	0.004
HF power, ms^2	886 (145-1580)	191 (20-482)	635 (150-1256)	0.008
LF:HF ratio	14 (2-25)	21 (7-34)	14 (4-25)	0.233
LF power (nu)	0.82 (0.65-0.99)	0.93 (0.89-0.97)	0.88 (0.78-0.97)	0.228
HF power (nu)	0.18 (0.01-0.35)	0.07 (0.03-0.11)	0.12 (0.03-0.22)	0.244

T1, 1 h prior to the training session; T2, 1 h post: T3, 72 h post; HR, heart rate; RRSD, standard deviation of R-R intervals; LF, low frequency component of HRV power spectrum; HF high frequency component; nu, normalised units. Data are presented as mean (68% confidence interval).