Autonomic Cardiac Control in Athletes and Non-Athletes at Rest

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Summary

The effect of different types of physical training on heart rate variability was evaluated in 34 subjects, including ten aerobically trained athletes, seven anaerobically trained athletes, seven rugby players (mixed type training), and ten sedentary control subjects. All groups were age matched (18 – 26 years). The following measures of heart rate variability (HRV) were obtained from both a time analysis and a frequency analysis of 10 min at a resting heart rate: pNN50 = the number of pairs of adjacent normal RR intervals that differ by more than 50 ms in the entire recording and are divided by the total number of normal RR intervals; SDNN = the standard deviation of all normal RR intervals; and rMSSD = the square of differences between adjacent normal RR intervals. ECG tracings were recorded digitally in supine position and in standing position. After these tests, blood pressure was measured using an automatic inflation cuff. Resting heart rate was lower in aerobic and mixed type athletes compared to those subjects in the control group. Only aerobic athletes showed signs of increased vagal activity in the time domain compared with the control subjects (increased SDNN supine, increased rMSSD supine and standing, and pNN50 standing). In the frequency domain, aerobic athletes presented with both higher low frequency and high frequency power (HRV power spectral density integratedfrom 0.04 – 0.15 Hz and 0.15 – 0.4 Hz, respectively) in standing position and low frequency power in supine position compared to the control group. It can be concluded that heart rate variability is affected by chronic exercise, especially in endurance athletes. This suggests that aerobic exercising in particular can have beneficial effects on the cardiovascular risk profile.

Key Words

Heart rate variability (HRV), digital signal processing, physical training, aerobic and anaerobic athletes, power spectrum analysis

Introduction

Heart rate variability (HRV) is an established, non-invasive method for the assessment of autonomic influence on the heart [1]. Low HRV has been associated with increased mortality after myocardial infarction [2]. The autonomic nervous system consists of nerves that are concerned primarily with the regulation of bodily functions. These nerves generally function without a person's awareness. Autonomic nerves include the sympathetic and parasympathetic nerves that control heart rate, the force of cardiac contraction, and the state of constriction of blood vessels through the transmission of nervous impulses. Therefore, autonomic nerves play a pivotal role in the regulation of the cardiovascular system by ensuring optimal function during various healthy activities, as well as in mediating several types of cardiac disease. Long-term physi-

cal training influences cardiac rhythm: sinus bradycardia under resting conditions, as well as a slower increase in heart rate at any degree of submaximal oxygen uptake due to a shift of the sympathovagal balance towards vagal predominance [3,4]. Therefore, it can be expected that both the level and kind of exercise would be factors that influence HRV parameters in the general population. This study was performed to determine whether the type of training conducted by a young population differentiates between HRV parameters in athletic groups and those in the sedentary subjects.

Materials and Methods

Study Population

After giving their informed consent, four groups (all male) were selected and compared: ten endurance trained athletes (aerobic), seven static trained athletes (anaerobic), seven rugby players (mixed type), and ten subjects with a sedentary life style (control group). The athletes were of national competition level and trained between 6 and 9 hours a week. Ages ranged between 18 and 34 years, with no significant differences among the four groups.

Data Acquisition

The ECG signals of the subjects were recorded for 10 min in supine position and for the same period while standing. A/D converted at a rate of 1000 Hz (RR interval time resolution of 1 ms). After peak detection [5], a tachogram text file was created containing the consecutive RR intervals. The acquisition setup is shown in Figure 1.

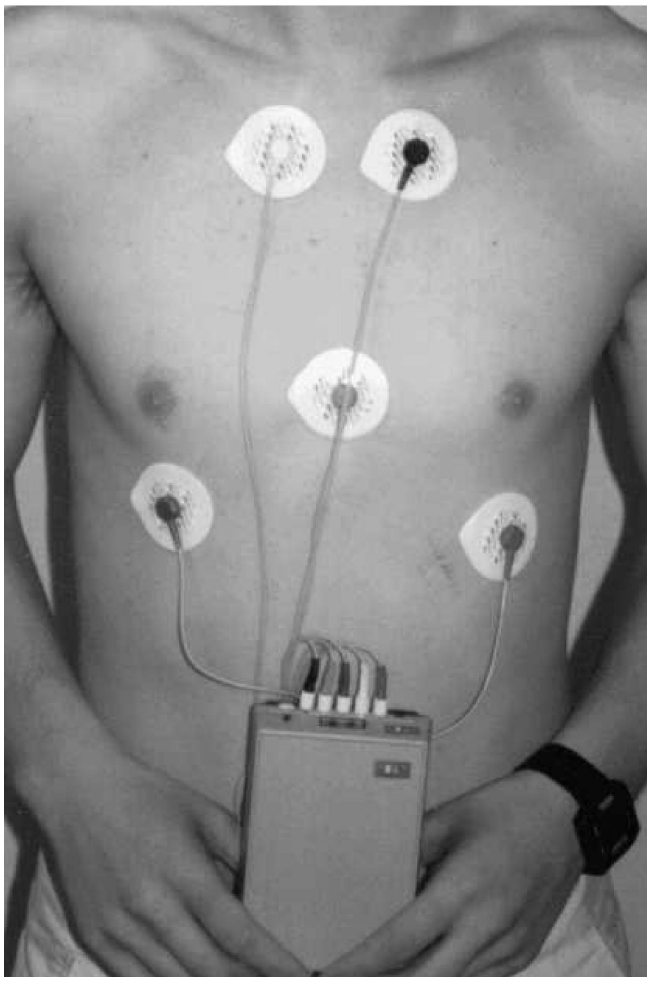

Figure 1. Data acquisition setup [5].

Data Analysis

Measurements of HRV in both the time and frequency domains were calculated in accordance with international standards [6]. In the time domain, measurements included mean NN and SDANN, rMSSD, and pNN50: mean NN = mean normal RR interval; pNN50 = the number of pairs of adjacent normal RR intervals that differ by more than 50 ms in the entire recording and are divided by the total number of normal RR intervals; SDANN = the standard deviation of all normal RR intervals; rMSSD = the square of differences between adjacent normal RR intervals; normal RR intervals = RR intervals, except for extrasytoles. The power spectral plot of HRV was derived from the tachogram after resampling at 2 Hz (in order to obtain equidistant points) and Fast Fourier Transform was computed by Hanning-windowing over 256 points (128 s) [7]. The total, low frequency (LF) and high frequency (HF) power, i.e., HRV power spectral density integrated from 0.04 – 0.15 Hz and 0.15 – 0.4 Hz, respectively, were calculated, as well as their ratios.

Statistics

The mean and standard deviations of data were determined. A logarithmic transformation was performed in case of non-normal distribution. ANOVA was used and corrected for multiple comparison testing by the least-significant difference (LSD) procedure using statistic software (SPSS, USA). Values were compared to the sedentary group. Significance is reported at the level of $P < 0.05$.

Supine

	Mean NN (ms)	SDNN (ms)	rMSSD (ms)	pNN50 (%)
Control	880.7 ± 263.8	69.7 ± 37.0	45.5 ± 26.8	21.8 ± 19.7
Aerobic	1103.4 ± 158.5*	97.9 ± 15.7*	73.5 ± 23.7*	40.1 ± 16.6
Anaerobic	842.3 ± 65.1	60.4 ± 20.7	38.7 ± 11.2	17.9 ± 19.7
Rugby	840.2 ± 204.7	55.0 ± 24.2	39.1 ± 25.7	19.2 ± 17.4

Standing

	Mean NN (ms)	SDNN (ms)	rMSSD (ms)	pNN50 (%)
Control	749.7 ± 165.6	65.4 ± 38.9	30.6 ± 16.9	10.5 ± 12.4
Aerobic	947.7 ± 108.8*	92.9 ± 30.9	47.2 ± 11.1*	22.4 ± 8.9*
Anaerobic	746.1 ± 45.5	50.4 ± 8.8	26.5 ± 4.7	6.1 ± 2.8
Rugby	750.1 ± 117.4	61.9 ± 27.8	29.6 ± 14.9	10.4 ± 11.1

*Table 1. Heart rate variability parameters in the time domain; Mean NN = mean value of all normal RR intervals; SDNN = the standard deviation of all normal RR intervals; and rMSSD = the square of differences between adjacent normal RR intervals, pNN50 = the number of pairs of adjacent normal RR intervals that differ by more than 50 ms in the entire recording and are divided by the total number of normal RR intervals. * = statistically significant difference with respect to the control group.*

Results

Basal conditions of the study population showed a lower heart rate in aerobic athletes (50 ± 4.6 vs 73 ± 14 beats/min in the control group). The time domain analysis (Table 1) also showed a higher mean NN in aerobic athletes, a higher SDNN and rMSSD in supine position, and a higher mean NN, rMSSD, and pNN50 in standing position. Analysis of the frequency domain variables (Table 2) revealed that aerobic athletes had significantly more total and LF power, a trend towards higher HF power in the supine position, as well as more total, LF, and HF power in the standing position. Rugby players showed a higher LF/HF ratio in the supine position. By comparing the during standing and in supine position, a significant increase in the ratio of LF to HF power was produced in all groups. Systolic blood pressure was higher in anaerobic athletes (147 ± 4.1 vs 134.6 ± 16.1 mmHg in the control group), although this was not considered significant.

Discussion and Conclusion

Performing an analysis of HRV may allow valuable insights into how the modulation of heart rate is affected by the autonomous nervous system [8]; in particular, activity in the HF spectrum (above 0.15 Hz) has been correlated with parasympathetic modulation. The results of this study show that aerobic athletes with a low resting heart rate show indications of increased parasympathetic modulation in both the time and the frequency domains. Stationary exercise (javelin and high jump) and mixed type activities (rugby) do not seem to have a significant influence on HRV. The mechanisms underlying the relationship between HRV and the training response are at present unclear. On the other hand, low frequency oscillations governing HRV are enhanced during orthostatic stress, leading to the speculation that humoral factors, such as circulating catecholamines, probably play a more dominant role than neural input. It should be stressed, however, that this study only

Supine

	ln (P_{tot})	ln (LF)	ln (HF)	LF/HF
Control	7.9 ± 0.8	6.88 ± 0.6	6.18 ± 1.01	2.4 ± 1.5
Aerobic	8.76 ± 0.36*	7.73 ± 0.49*	7.08 ± 0.68	2.15 ± 1.66
Anaerobic	7.82 ± 0.56	6.85 ± 0.51	6.07 ± 0.73	2.34 ± 1.0
Rugby	7.51 ± 1.11	6.80 ± 1.1	5.38 ± 1.63	5.0 ± 3.2*

Standing

	ln (P_{tot})	ln (LF)	ln (HF)	LF/HF
Control	7.84 ± 0.92	7.03 ± 0.86	5.54 ± 0.92	5.5 ± 3.2
Aerobic	8.71 ± 0.58*	7.78 ± 0.65*	6.35 ± 0.49*	4.8 ± 2.4
Anaerobic	7.65 ± 0.34	7.03 ± 0.37	5.38 ± 0.35	5.5 ± 1.7
Rugby	7.78 ± 0.86	7.18 ± 0.8	5.36 ± 0.79	6.7 ± 2.4

*Table 2. Heart rate variability parameters in the frequency domain. P_{tot}, LF, HF = HRV power spectral density integrated over the complete frequency range, from 0.04 – 0.15 Hz, and from 0.15 – 0.4 Hz, respectively. * = statistically significant difference with respect to the control group.*

involves subjects that are young (18 – 34 years of age) and male. The importance of these findings lies in the application of HRV for risk stratification in patients. The impact of age and gender on HRV is well known. Since HRV indices, especially from aerobic athletes, are different from those of sedentary subjects, it can be concluded that the type and level of training have to be taken into account along with the prognostic stratification of HRV. Furthermore, it can be hypothesized that physical activity has beneficial effects on the cardiovascular risk profile.

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