ORIGINAL RESEARCH PAPER

Physiology

COMPARATIVE STUDY OF HEART RATE VARIABILITY AT REST BETWEEN SEDENTARY AND NON-SEDENTARY MALES

KEY WORDS: Heart Rate Variability, Exercise, Sympathetic & Parasympathetic System.

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ABSTRACT

The present study was aimed to determine the effects of exercise training on heart rate and measures of heart rate variability associated with vagal cardiac modulation and to quantify the relationship between changes in these measures. 30 individuals who exercised at least 5 days/week for 45 min/day or more were compared with age matched sedentary controls. The resting HR was recorded in Lead II using Medicaid Students Physio Pac in supine position for 5 minutes. Due care was taken to remove factors which could interfere with results. There was a relative bradycardia in the non-sedentary group at rest as compared to the sedentary group ($p<0.001$). Time domain indices of HRV, mean RRI, SDNN, and RMSSD were significantly enhanced in the non-sedentary group ($p<0.001$). Frequency domain indices of HRV, LFn.u., HFn.u., LF/HF ratio were higher in the non-sedentary group than sedentary group but the difference was not significant ($p>0.05$). These results suggest that the adaptive responses of the cardiovascular system to regular physical activity appear to include a reduction in sympathetic (SNS) activity and an increase in parasympathetic (PNS) activity during rest, which may contribute to the reduction in mortality associated with regular exercise.

INTRODUCTION

Regular physical exercise is an important factor which affects the indices of cardiovascular function and all causes of morbidity/mortality. However, there are, apparently, additional and independent benefits of the regular practice of physical exercise and the improvement of the level of aerobic condition. Heart rate (HR) is modulated primarily by the direct activity of the autonomic nervous system (ANS), specifically through the sympathetic and parasympathetic branches over the sinus node auto- rhythmicity, with predominance of the vagal activity (parasympathetic) at rest, that is progressively inhibited since the onset of the exercise. The HR behavior has been widely studied during different conditions and protocols associated to the exercise. HRV is a non-invasive method used to obtain valuable data concerning physiological changes that occur in the response to physical activity. Heart rate variability (in time and frequency domain) reflects oscillations in the heart cycle duration over time and is generally considered the measure of regulatory influences, mainly of the activity of ANS to regulate the function of the cardiovascular system. HRV parameters are relevant in the analysis of stress that the body experiences during training and to increase insight into physiological recovery after training. Previous findings have identified the potential use of HRV for recognizing healthy and diseased states since the vagal-mediated HRV indices were inversely associated with several risk factors for diabetes, glucose intolerance, insulin resistance, central obesity, dyslipidemia and hypertension[1]. However, while HRV was largely applied to predict sudden cardiac death, diabetic neuropathies and in assessing disease progression[2,3], recent studies demonstrated the application of HRV in exercise training. Their findings supported the use of HRV as a marker to reflect the cardiac modulation of the sympathetic and vagal component of the ANS[4], and suggested that monitoring indices of HRV may be useful for tracking the time course of training adaptation/maladaptation in order to set optimal training loads that lead to improved performances[1,4-6]. The present study examined the possible role of HRV in sports physiology and its use in assessing the chronic effects of physical exercise on the autonomic nervous system, especially of the parasympathetic component, identifying possible changes on the cardiac vagal tone.

SUBJECTS AND METHODS:

In a cross-sectional design, the study included 60 subjects. 30 individuals in the age group of 20-40 years who exercised at least 5 days/week for 45 min/day or more were compared with 30 age matched sedentary controls after their written consent was obtained. A detailed history and thorough physical examination were carried out of all the subjects to exclude any other condition that is known to produce autonomic neuropathy or alter heart rate. The details of the procedure were described to the subjects so that they were without any anxiety at the time of the tests. Tests were done after an overnight fast. Data acquisition was carried out at least 24hrs after the last bout of exercise training. It was deemed necessary that ambulatory recordings were made on a non-exercise day. Calm and relaxed environment was provided while carrying out the test. The subjects were connected to Medicaid Physio Pac machine via ECG limb leads and heart rate and ECG was recorded in lead II in supine position for 5 mins. HRV from the recorded ECG was analyzed by using Kubois software version 2.1. All the values were presented as mean ± standard deviation. The data was statistically analyzed using Students Unpaired t test. P value of less than 0.05 was considered to be statistically significant.

RESULTS:

The mean age of subjects of the non-sedentary group was 28.10±6.10, and that of sedentary group 29.93±5.95; the mean height (in cms) of the subjects of non-sedentary group was 173.01±6.08 and that of sedentary group was 170.83±6.16; and the mean weight (in kgs) of subjects of the non-sedentary group was 74.57±7.67, and that of sedentary group was 71.13±4.64; thus, excluding the variables in autonomic tone occurring with age and BMI. There was a relative bradycardia in the non-sedentary group at rest as compared to the sedentary group($p<0.001$). The results of different time and frequency domain indices of HRV are given in the table below.

Table no 1: showing comparison of age, height, weight, heart rate and time and frequency domain parameters of Heart rate variability between sedentary and non-sedentary males

Parameter of HRV	Non sedentary subjects (cases)	Sedentary subjects (controls)	P-value
Age	28.10±6.10	29.93±5.95	0.24(NS)
Height (cms)	173.01±6.08	170.83±6.16	2.01(NS)
Weight (kgs)	74.57±7.67	71.13±4.64	2.01(NS)
Heart rate	66.76±7.01	84.06±5.31	<0.001*
Time domain indices:			

Mean R-R interval	0.856±0.10	0.696±0.04	<0.001*
SDNN	0.040±0.015	0.022±0.02	0.003*
RMSSD	31.88±13.53	21.82±3.96	0.002*
Frequency domain indices:			
LF(n.u)	76.11±61.91	65.03±8.65	0.33(NS)
HF(n.u)	41.75±47.46	33.01±9.12	0.32(NS)
LF/HF	2.97±2.23	2.15±0.73	0.06(NS)

Footnote: *significant; p-value<0.05 is considered significant; NS-not significant; unpaired T-test applied

Time domain indices of HRV, mean RRI, SDNN, and RMSSD were significantly enhanced in the non-sedentary group (p<.001). Frequency domain indices of HRV, LFn.u, HFn.u, LF/HF ratio were higher in the non-sedentary group than sedentary group but the difference was not significant (p>0.05).

DISCUSSION:

The present study was aimed to determine the effects of exercise training on heart rate and measures of heart rate variability associated with vagal cardiac modulation and to quantify the relationship between changes in these measures. Our study showed that training increased HRV at rest and decreased heart rate at rest. The resting heart rate was significantly lower in the non-sedentary group as compared to sedentary controls.

This result supports the current theory that exercise training can alter neuro-regulatory control over the heart. It also supports the notion that bradycardia after exercise training is partially associated with increased vagal modulation. However, a cross sectional analysis does not allow us to conclude that training was responsible for such adjustments of the ANS as our study did not take into consideration the level of aerobic conditioning and the autonomic function of the subjects prior to training. Uusitalo et al.[7] and Bonaduce et al.[8] after longitudinal studies, noted a reduction of resting HR, even though significant changes in autonomic indicators were not seen.

Exercise training results in significant increases in RR interval and HF power. These changes are influenced by study population age. The smaller effect size for HF and weak relationship between HF and RR interval suggest factors additional to increased vagal modulation are responsible for training bradycardia.

Time and frequency domain analyses of heart rate variability (HRV) provide a non-invasive method to evaluate the autonomic regulation of heart rate. Low levels of HRV are related to risk of sudden cardiac death and are associated with numerous other cardiac events such as: heart disease, heart failure, diabetes, hypertension, asymptomatic left ventricular dysfunction, and myocardial infarction. In these disease states, as well as in the general population, increased sympathetic drive is associated with arrhythmia formation and sudden death[9]. Conversely, interventions that reduce sympathetic activity and/or increase parasympathetic activity have been shown to protect against lethal arrhythmias[10].

Exercise has been proposed as a possible antiarrhythmic intervention in humans[11]. Experimental data in the dog suggest that exercise eliminates the incidence of ventricular fibrillation in previously susceptible animals via enhanced baroreceptor control and vagal modulation[12], represented by increased HRV after endurance exercise. Perini et al.[13] observed no changes in HF and LF at rest in seven men and eight women over 70 years old after an eight-week training period. Lerma et al [14] reported higher HF values in sportswomen compared to sportsmen in the same condition. Other authors report significant differences between sedentary and athletic subjects, with higher total power and HF in athletes, but no difference in LF and ratio LF/HF[15], although in this study the sample comprised both men and women.

Cross-sectional differences between athletes and controls support the notion of increased parasympathetic and/or decreased sympathetic drive in endurance-trained individuals. Specifically, the resting bradycardia observed in endurance-trained athletes is commonly accompanied by augmented markers of cardiac vagal modulation[16,17]. A recent Meta-Analysis[18] reports a higher HF in trained subjects but they do not differ for gender or age. Longitudinal data are less consistent, and a recent review[19] suggested that further, controlled, studies using larger cohorts should be carried out to clarify the effect of exercise in HRV.

A training-induced increase in parasympathetic tone, measure by HRV, has been documented in longitudinal studies in patients with congestive heart failure[20], young patients with hyper- tension[21], in normal, middle-aged subjects[22], and in cross-sectional analyses of healthy subjects[23]. However, other investigators found no change in parasympathetic tone after exercise training in cross-sectional[24] or longitudinal studies of young subjects[25]. To our knowledge, there have been no prior longitudinal studies of older subjects, which are of particular interest due to the known age-related reduction in HRV. Ekblom et al.[26] compared young subjects before and after 5 weeks of training by comparing the chronotropic response during exercise with atropine or propranolol blockade and concluded that training induced bradycardia is due to a reduced chronotropic response to sympathetic activity and an increase in parasympathetic tone at rest. Cross-sectional studies using a different methodology to determine autonomic balance have either supported Ekblom et all.[26] or shown only a decrease in the intrinsic heart rate without any change in autonomic control[27]. In contrast, a recent study concluded that training-induced bradycardia was due only to enhanced vagal tone because there was no significant change in intrinsic heart rate with training[28].

CONCLUSION:

Our findings suggest that training induces a resting bradycardia accompanied by increased cardiac vagal modulation in healthy individuals indicating that physical activity is a safe, non-pharmacological approach to favorably altering ANS function and thereby provides a cardio protective role. But, due to wide-spread discrepancies in research methodologies (e.g., training duration and intensity) and lack of standardized measurements of HRV during exercise, consensus has yet to be reached regarding the effects of exercise on HRV.

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