DETERMINATION OF THE VAGAL THRESHOLD AND CHANGES OF IT'S USING

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Exercise intensity causes changes in the activity of both branches of the autonomic nervous system (ANS) as involved in cardiovascular system regulation. Reduction in vagal activity and an increase in sympatho-adrenal activity is associated with an increase in death risk from both cardiac and arrhythmic causes during exercise. The main aim of this work was to develop a simple mathematic algorithm for determination of critical exercise intensity, at which, if exceeded, the cardiovascular system starts to be influenced dominantly by rising sympathetic activity including catecholamine and a significant withdrawal in cardiac vagal activity (vagal threshold – T_{VA}) occurs. The testing group consisted of 10 volunteers (men). Their mean age was 27.24 ± 3.23 years and the mean value of their maximal oxygen uptake (VO_2max) was 50.24 ± 4.63 ml·kg^{-1}·min^{-1}. ANS activity was monitored by the microprocessor diagnostics system VarCor PF 7 and assessed by a non invasive spectral analysis (SA) of heart rate variability (HRV) method. The power of the high frequency component (P_{HF}) was calculated by integrating the area under the power spectral density curve in the frequency range from 0.15 to 0.5 Hz. Changes in autonomic cardiac regulation were assessed during walking in the steady state with exercise intensities ranging from 20 to 70% of maximal heart rate reserve (MHRR) on the treadmill. Each exercise intensity increases of about the 10% MHRR in a range from 20 to 70% MHRR led to a significant decrease in vagal activity. A designed mathematic algorithm for detecting the deflection point of the vagal activity during incremental exercise intensity revealed T_{VA} at $43.63 \pm 4.66\%$ MHRR. We can state that the designed algorithm for detection of T_{VA} enables an estimation of such a "safe" intensity, when the vagal activity is still preserved and sympathetic activity does not markedly rise up during exercise. The estimation of T_{VA} could be recommended especially for the exercise prescription for patients with both reduction HRV, and at risk for sudden cardiac death.

Keywords: Spectral analysis of heart rate variability, exercise intensity, vagal threshold, prescription of the training exercise.

INTRODUCTION

A sedentary lifestyle or physical inactivity is generally associated with the appearance of the most relevant chronic diseases. On the other hand, regular endurance exercise represents an effective preventive and therapeutic tool (Hilberg, 2008) against such diseases. However, the patients with cardiovascular diseases who practice exercise are exposed to certain risks resulting from insufficient heart function, an increased tendency to myocardial arrhythmias and also from changes in the bloodstream (ACSM, 2007). The level of intensity seems to be a crucial risk factor of sudden cardiac death or myocardial arrhythmias; therefore, its optimizing is a basic requirement for safe exercise.

The cardiovascular system is mostly controlled by autonomic activity through the activity of the sympathetic and parasympathetic pathways of the ANS, and their activity depends mainly on the intensity of the exercise (Arai et al., 1989; Casedei et al., 1995; Parekh & Lee, 2005; Stejskal et al., 2001; Yamamoto, Hughson, & Peterson, 1991). At low intensity, tachycardia occurred mainly due to a withdrawal of efferent vagal activity (Perini et al., 1990). Rising sympathetic activity together with the level of circulating catecholamine plays a major role in heart regulation at higher intensities (Breuer et al., 1993; Kluess, Wood, & Welsch, 2000). With the dominant increase in sympatho-adrenal activity, higher demands on compression heart work are associated therewith (Ganong, 1999), and thereby, increase in the risk of both heart and circulation failure are related. From the presented data it is clear that patients with a higher risk of sudden cardiac failure should exercise only at such an intensity as does not lead to a significant increase in sympatho-adrenal activity, but which enables changes in the vagal activity regulation of the cardiovascular system.

As optimal physical activity for healthy people is endurance activity with an intensity set between 60–70% of VO_2max being recommended (ACSM, 1990). In people with some health limitations, lower intensity exercise is recommended, e. g. hypertension patients should keep their exercise intensity within a range from 45 to 60% of VO_2max (Cléroux, Feldman, & Petrela, 1999; Izdeb-

ska et al., 2004). So far, published studies have brought us different results describing exercise intensity, from which the efferent cardiac vagal modulation disappears, and the activity of the heart will be mainly mediated by the sympatho-adrenal system. For example, Perini et al. (1990) and Orizio et al. (1989) presented an intensity of about 30 and 33% VO_2max. Hautala et al. (2003) described 40% VO_2max as the dividing intensity in vagal and sympathetic cardiac modulation. According to the studies of Achten and Jeukendrup (2003) and Nakamura, Yamamoto and Muraoka (1993), the target intensity is found at 50–60% VO_2max.

Heart rate variability (HRV) is generally accepted as a feedback marker of cardiac vagal and sympathetic activity (Akselrod et al., 1981; Task Force, 1996). Due to obesity, diabetes mellitus, hypertension or several cardiovascular diseases, there is significantly decreased HRV (Kuch et al., 2004; Matsunaga et al., 2004; Nolan et al., 1998; Shibata et al., 2002) which has been associated with higher risk of sudden death or myocardial arrhythmias (Schwarz, La Rovere, & Vanoli, 1992). On the contrary, regular endurance exercise leads to an increase in vagal cardiac activity and a decrease in sympathetic activity (increase in HRV) in healthy people as well as in patients who exercised during their rehabilitation process (Cornelissen & Fagard, 2005; Dixon, Kamath, McKartney, & Fallen, 1992; Fujimoto et al., 1999; Goldsmith, Bigger Jr., Steinman, & Fleiss, 1992; Mueller, 2007; Takeyama et al., 2000).

The spectral analysis (SA) of HRV is a non invasive method for the direct assessment of vagal cardiac activity and for the indirect evaluation of sympathovagal balance. It is known that exercise intensity decreases the vagal parameters of SA HRV (Stejskal et al., 2001). Therefore, the aim of this study was to identify the critical exercise intensity level which is linked with a very mild reduction of vagal cardiac modulation (vagal threshold – T_{VA}). A sophisticated determination of T_{VA} may allow us to prescribe "safe" exercise for patients with a higher risk of sudden cardiac death or myocardial arrhythmias.

METHODS

The testing group consisted of 10 men who were studying or working at the Faculty of Physical Culture (FPC), Palacký University in Olomouc. Each testing protocol and situation was clearly and precisely described to them. The study protocol was approved by the ethics committee of the Faculty of Physical Culture, and the subjects gave written informed consent. During this study, all measurements were performed in the laboratories belonging to the Department of Functional Anthropology and Physiology of FPC PU. The investigated

persons were instructed to keep an optimal regime, and vigorous physical activity was forbidden minimally for 48 hours before the testing. Further, the volunteers were not allowed to eat and drink coffee, tea, nor any substance, which could influence ANS activity, 2 hours prior to the measurement of ANS activity. All subjects were non smokers, and they were asked to come on an empty stomach for all measurements.

Before the testing, the volunteers had been investigated to preclude any medical or health limitations to perform the maximal exertion test. Usually in the morning, each tested person underwent an initial tests battery, which was performed 14 days before the start of the study in the exercise laboratory. Subjects underwent basic anthropometric measurements (height [cm], weight [kg]) and body composition was assessed using the bioimpedance method (In Body 720, South Korea). The measurement of HRV and oxygen consumption followed anthropometric measurements. The maximal running test was performed on a Lode Valliant treadmill (Netherlands). The test started with a warm up phase: 4 minutes (min.) at 8 $km \cdot h^{-1}$ and 10 $km \cdot h^{-1}$, respectively. Immediately after the warm up, the inclination of the treadmill increased from 0 to 5%, and speed remained at 10 $km \cdot h^{-1}$. Then the speed increased every minute by 1 $km \cdot h^{-1}$ till exhaustion. During the test, the subject breathed in a mask: ventilation and both O_2 and CO_2 exchange were analyzed by a gas ventilator ZAN 600 Ergo USB (Germany).

ECG data were collected during a standardized ortho-clinostatics maneuver of lying–standing–lying by the VarCor PF 7 system (Salinger & Gwozdziewicz, 2008), which requires for HRV analysis 256 artifacts free of subsequent R–R intervals for each position. Frequency domain analyses were performed according to the methods described by Salinger et al. (1998). The amplitude density of the collected signal was estimated using the fast Fourier Transform method with a partly modified Coarse-Graining Spectral Analysis algorithm (Yamamoto & Hughson, 1991). The power of the mean spectral component with high frequency (P_{HF}) was calculated by integrating the area under the power spectral density curve in the frequency ranges according to Salinger et al. (1998) with a result of: P_{HF} from 0.15 to 0.5 Hz.

Changes in autonomic cardiac regulation were assessed during walking in the steady state with exercise intensities ranging from 20 to 70% of MHRR on the treadmill. The value of the MHRR was calculated for each subject individually as a differential of the HRmax value, obtained within the framework of the maximal exertion test, and the HR rest value estimated as a mean HR in the third position (lying) during the investigation of HRV. The target HR was expressed as the HR value (HR_T = MHRR · [% exercise intensity / 100] + HR rest) within the range of ± 5 $beat \cdot min^{-1}$. The measurement of

both HRV and oxygen consumption lasted 5 minutes. The measurement itself followed a 5 minute warm up aimed at the achievement of HR_T. The investigations at 50, 60 and 70% MHRR coursed separately in different days. Only intensities of 20–30–40% MHRR were done by the subject all together during one measurement. The intensities were chosen randomly.

An algorithm of T_{VA} calculation based on load curve is described in this article. The result of a load curve measurement is the set of parameters $\{x_i, y_i\}$, $i = 1, 2,...n$, where y_i is the value of P_{HF} [ms^2] measured within load x_i [%]. In our ramp test schedule, the load is increased step by step at levels of 20%, 30%,...70%, and so on, in our case bringing a result of $n = 6$. The mathematics algorithm does not include resting P_{HF}.

The algorithm splits the measured set of parameters into two subsets, calculates a regression line in each subset and calculates the T_{VA} as a point of intersection of two regression lines. The first subset contains the parameters $\{x_i, y_i\}$, $i = 1, 2,...k$ and the second subset contains the parameters $\{x_i, y_i\}$, $i = k, k+1,...n$. The parameters with index k belong to both subsets.

The regression line

$$y = a_1 + b_1 x \tag{1}$$

with coefficients

$$b_1 = \frac{k\sum_{i=1}^{k} x_i y_i - \sum_{i=1}^{k} x_i \sum_{i=1}^{k} y_i}{k\sum_{i=1}^{k} x_i^2 - \sum_{i=1}^{k} x_i \sum_{i=1}^{k} x_i}, \quad a_1 = \frac{1}{k}\sum_{i=1}^{k} y_i - \frac{b_1}{k}\sum_{i=1}^{k} x_i \tag{2}$$

is calculated from values in the first subset. The regression line

$$y = a_2 + b_2 x \tag{3}$$

with coefficients

$$b_2 = \frac{(n-k+1)\sum_{i=k}^{n} x_i y_i - \sum_{i=k}^{n} x_i \sum_{i=k}^{n} y_i}{(n-k+1)\sum_{i=k}^{n} x_i^2 - \sum_{i=k}^{n} x_i \sum_{i=k}^{n} x_i},$$

$$a_2 = \frac{1}{(n-k+1)}\sum_{i=k}^{n} y_i - \frac{b_2}{(n-k+1)}\sum_{i=k}^{n} x_i \tag{4}$$

is calculated from values in the second subset. The T_{VA} is calculated as the point of intersection $[x_P, y_P]$ of lines (1) and (3), the resulting equations are

$$x_P = \frac{a_1 - a_2}{b_2 - b_1}, \quad y_P = a_1 + b_1 x_P = a_2 + b_2 x_P. \tag{5}$$

The algorithm calculates the value of index k, which splits the measured set into two subsets, by means of an iterative procedure. At the beginning of this iterative procedure, the index k is set for a starting value, i. e. $k = 2$, and the condition

$$b_2 < 0 \wedge a_2 + b_2 x_n > 0 \tag{6}$$

is tested. If the condition (6) is fulfilled, the iterative procedure is stopped and the value of index k is taken. If the condition (6) is not fulfilled, the index k is incremented, i. e. $k = k + 1$, the coefficients (4) are calculated again and the condition (6) is tested. The iterative procedure continues until the condition (6) is fulfilled. The meaning of this condition is that the regression line (3) has a decreasing slope and lies above zero level within the interval $\langle x_k, x_n \rangle$. It can be said that the goal of the iterative procedure is to find such a regression line (3) which does not cross the x axis.
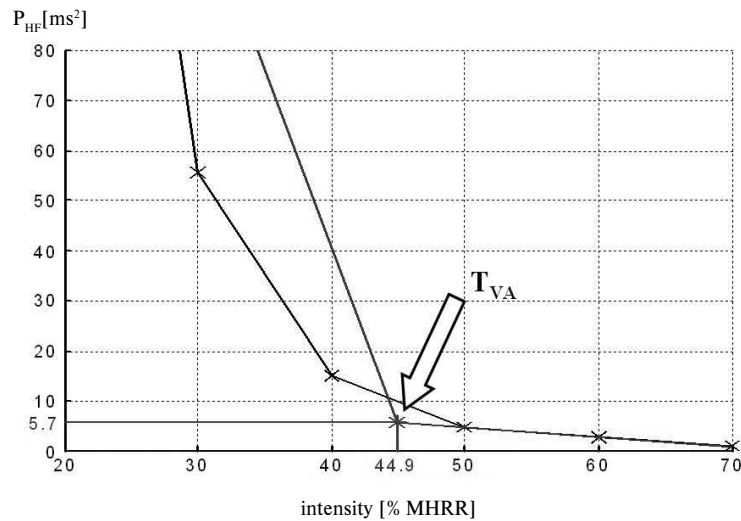
All statistical processes were performed in MS Excel 2003, Statistica 6.0 software and the algorithm for detection of the T_{VA} was created in MatLab 7.5.

RESULTS

The Wilcoxon test revealed significant differences in P_{HF} values between all assessed exercise intensities. Fig. 2 clearly shows that the highest reduction in P_{HF} values occurred up to 40% MHRR. On the contrary, minimal attenuation of P_{HF} values was detected at over 50% MHRR.

TABLE 1

Basic characteristics of the testing group

Age [year]	BMI [kg·m^{-2}]	Body Fat [%]	HRrest [beat·min^{-1}]	HRmax [beat·min^{-1}]	ANT$_R$ [beat·min^{-1}]	VO$_2$max [ml·kg^{-1}·min^{-1}]
27.24 ± 3.23	24.89 ± 1.81	9.39 ± 3.97	50.30 ± 2.21	193.0 ± 9.32	170.0 ± 8.82	50.24 ± 4.63

Legend: VO$_2$max – maximal oxygen uptake, HRrest – resting heart rate, HRmax – maximal heart rate, ANT$_R$ – respiratory anaerobic threshold, BMI – Body Mass Index.

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Fig. 1
Illustrated example of the vagal threshold assessment

Legend: % MHRR – percent of the maximal heart rate reserve, P_{HF} – high frequency power, T_{VA} – vagal threshold.

TABLE 2
Statistical differences of the P_{HF} values during different exercise intensity

Parameter [units]	R	20% MHRR	30% MHRR	40% MHRR	50% MHRR	60% MHRR	70% MHRR
HR [beat·min⁻¹]							
M	83.23	78.02	91.05	106.42	122.03	136.86	151.08
SD	5.90	4.13	2.75	3.67	4.42	6.51	6.80
P_{HF} [ms²]							
M	288.94	551.62	185.80	37.24	9.05	4.25	0.86
SD	402.36	421.37	189.63	34.18	5.06	2.54	0.52
P_{HF} [ms²] **p**	R vs 20* R vs 30* R vs 40** R vs 50** R vs 60** R vs 70**	20 vs 30** 20 vs 40** 20 vs 50** 20 vs 60** 20 vs 70**	30 vs 40** 30 vs 50** 30 vs 60** 30 vs 70**	40 vs 50** 40 vs 60** 40 vs 70**	50 vs 60* 50 vs 70**	60 vs 70**	

Legend: R – rest HR during standing position, P_{HF} – high frequency power, M – mean value, SD – standard deviation, % MHRR – percent of the maximal heart rate reserve, HR – heart rate, p – significant values (Wilcoxon test), *p ≤ 0.05, **p ≤ 0.01, vs – versus.

DISCUSSION

Cardiovascular adjustment in exercise represents a combination and integration of the following factors: central command, autonomic cardiac regulation, reflexes originating in the baroreflex, and circulating catecholamine (Aubert, Seps, & Beckers, 2003). Mechanisms of cardiac regulation apply their influence to increasing cardiac output during exercise, when demands of working muscles for oxygen and energy substrates delivery increase (Åstrand, Rodahl, Dahl, & Strømme, 2003). The increase in cardiac output is caused by heart rate acceleration and an increase in stroke volume during exercise. It is well accepted that decrease in efferent cardiac vagal activity allows an increase in cardiac output during low exercise intensity (Arai et al., 1989; Perini et al., 1990; Tulppo et al., 1998). In addition, the regulation effects of both the sympathetic activity and catecholamine are negligible for the heart activity at this moment. At low intensity, in humans, an increased sympathetic activity together with norepinephrine is responsible throughout the stimulation of α receptors for redistribution of blood flow away from the splanchnic area, the kidney, and resting skeletal muscles to the working muscles (Christensen & Galbo, 1983). At higher intensities, the vagal withdrawal came to be insufficient for further augmentation of the cardiac output, and thereafter, increments in cardiac output are attributed with the positive inotropic

Fig. 2
Dynamics of spectral measure P_{HF} within different exercise intensity
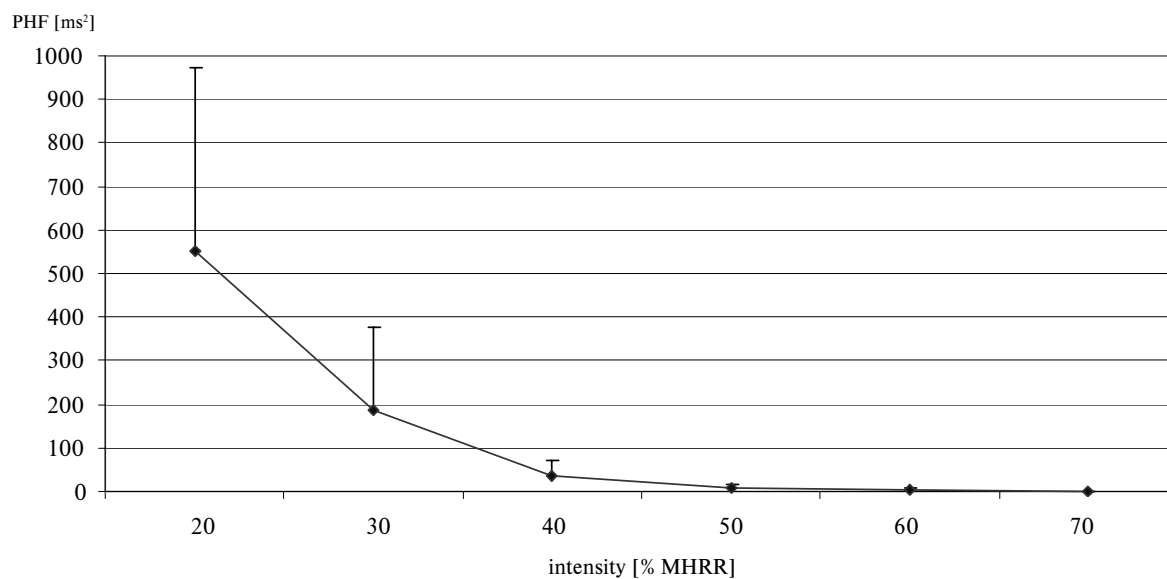
Legend: % MHRR – percent of the maximal heart rate reserve, P_{HF} – high frequency power.

TABLE 3
Level of the vagal threshold expressed by various parameters

Parameters	*M*	*SD*	*Max*	*Min*
INT [% MHRR]	43.63	4.66	49.73	37.10
HR [beat·min^{-1}]	112.06	9.30	132.97	100.18
P_{HF} [ms^2]	10.87	5.87	19.53	1.80

Legend: INT – exercise intensity, P_{HF} – high frequency power, % MHRR – percent of the maximal heart rate reserve, HR – heart rate, M – mean value, SD – standard deviation, Max – maximal value, Min – minimal value.

Fig. 3
3D Graphs of the SA HRV during different intensity
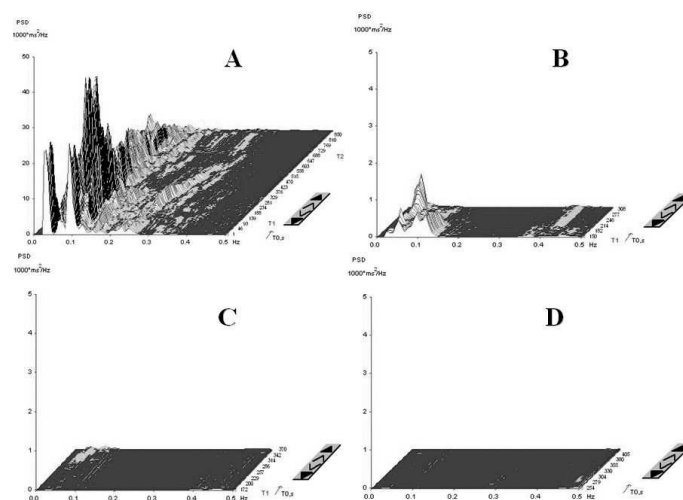
Legend: A – 20, 30, 40%, B – 50%, C – 60%, D – 70% maximal heart rate reserve.

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and chronotropic effects of increases in sympatho-adrenal activity (Breuer et al., 1993; Ganong, 1999; Kluess, Wood, & Welsch, 2000).

The conclusions associated with investigation of autonomic cardiac behavioral during exercise have used rather misleading data. In a relatively older study, Robinson, Epstein, Beiser and Braunwald (1966) described that an initial rise in HR connected with vagal withdrawal can reach a maximum increase of 30 beats·min^{-1}. This further HR increment has been ascribed to increased cardiac sympathetic activity. On the basis of the dynamics of the relative value of the very low frequency power during exercise, Perini et al. (1990) states that 30% VO_2max represents a threshold in cardiovascular adjustment when increases in the sympathetic activity during exercise occur. It is interesting to note the study by Hautala et al. (2003), who used the fractal method of HRV during exercise for analysis of R–R intervals, and investigated that sympathetic activity starts to dominate after vagal activity in the cardiac regulation at an intensity of 40% VO_2max. According to Tulppo et al. (1998), the vagal modulation disappears at the level of 50–60% VO_2max, whereafter the increase in heart activity is mainly mediated by sympathetic activity.

Cottin, Papelier and Escourrou (1999) and Warren Jaffe, Wraa and Stebbins (1997) together concluded that HRV is a valid technique for the non invasive assessment of vagal activity during exercise, but its validity as measure of sympathetic activity during exercise is equivocal. Therefore, P_{HF} as a good index of vagal activity (Task Force, 1996) was measured during walking in a steady state at that intensity level, which ranges from 20 to 70% MHRR in this study. Our results showed that each enhancement in the intensity by about 10% MHRR evoked a significant reduction in vagal activity. This study confirmed the previously published statement about a negative relationship between intensity and HRV (Arai et al., 1989; Parekh & Lee, 2005; Stejskal et al., 2001; Yamamoto, Hughson, & Peterson, 1991).

The dynamics of the P_{HF} clearly shows that the most pronounced attenuation in vagal activity during exercise occurred between 20–50% MHRR. Otherwise, the higher intensity led to another significant withdrawal in vagal activity, but these changes were not so obvious compared to changes in vagal activity observed at low intensity. According to our opinion, such a residual vagal activity at above 50% MHRR has neither marginal nor any regulatory effect on the heart action.

Several chronic diseases cause significant withdrawal in vagal cardiac regulation. It has been also revealed that such a decline in vagal activity has been associated with the electrical instability of the myocardium, malignant arrhythmias or ventricular fibrillations (Nolan et al., 1998; Schwarz, La Rovere, & Vanoli, 1992; Vanoli

& Schwarz, 1990). Therefore, exercise below T_{VA} may have a protective effect against the mentioned medical complications due to persisting vagal activity. On the other hand, exercise intensity over T_{VA} level induces a marked enhancement in sympato-adrenal system activity, which causes not only a rising risk of myocardial arrhythmia development, but also increases demands on the compression of the heart's work, which may lead to its failure in threatened patients.

In 2002, Shibata et al. came up with the idea of a new method to determine exercise intensity for obese women based on cardiac vagal activity. They suggested that exercise at the T_{VA} level represents a safe exercise intensity in the light of cardiac stress. Hence, T_{VA} may be recommended generally for people who might possess a lower cardiac sympatho-vagal balance. They have established the T_{VA} level at HR 114.6 ± 8.5 beats·min^{-1}. The HR value of our T_{VA} was almost equal to the HR value of T_{VA} of the last cited study. However, our tested subjects were about 13 years younger than volunteers in the study of Shibata et al. (2002), and therefore the relative load level in our case (with a mean value of T_{VA} 45% MHRR) was lower than in their study. From this point of view, we state that it is better to express exercise intensity in relative values (% VO_2max or % MHRR) than in absolute value of HR.

The relative small age dispersion (27.24 ± 3.23 years) can be considered as a limit to the applicability of this study because of the fact that an incremental increase in age causes a reduction in HRV, mainly due to a decrease in vagal activity (Finley & Nugent, 1995; Fukusaki, Kawakubo, & Yamamoto, 2000; Šlachta et al., 2002; Vallejo et al., 2004). It will be in the future very interesting to examine the relationship between T_{VA} level and age. Tulppo et al. (1998) have already published that exercise intensity at which instantaneous R–R interval variability disappeared and was not related to age.

CONCLUSIONS

In conclusion we can state that the monitoring of HRV by the SA HRV method enables us to assess changes in vagal cardiac regulation during walking in a steady state. In addition, each enhancement of the intensity by about 10% MHRR in a range from 20 to 70% MHRR evoked a significant decrease in vagal activity. Exercise intensity at around 45% MHRR may represent the mean level of T_{VA} in our measured subjects. The extension number of other age matched groups is a necessary condition for further application of T_{VA} within the prescription of a safe exercise level for patients with a reduction in ANS activity and an increased risk of cardiac complications.

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STANOVENÍ VAGOVÉHO PRAHU A MOŽNOSTI JEHO VYUŽITÍ
(Souhrn anglického textu)

Cílem této studie bylo navrhnout jednoduchý matematický postup, podle kterého by bylo možno stanovit takovou hraniční intenzitu zatížení, nad kterou se redukovaná vagová aktivita dále výrazně nemění a kardiovaskulární systém je dominantně řízen zvyšující se aktivitou sympatoadrenálního systému (vagový práh – T_{VA}). Testovaný soubor tvořilo 10 mužů ve věku $27,24 \pm 3,23$ let s hodnotou maximální spotřeby kyslíku $50,24 \pm 4,63$ ml.kg^{-1}.min^{-1}. Aktivita ANS byla hodnocena pomocí neinvazivní metody spektrální analýzy (SA) variability srdeční frekvence (HRV). Změny v autonomní kardiální regulaci byly posuzovány během chůze na běhátku v setrvalém stavu při intenzitách zatížení od 20 % do 70 % maximální tepové rezervy (MTR). Zvýšení intenzity zatížení o 10 % MTR v rozmezí od 20 % do 70 % MTR vedlo vždy k signifikantnímu snížení aktivity vagu. Navržený postup pro stanovení deflekčního bodu křivky závislosti P_{HF} na intenzitě zatížení, za kterým již P_{HF} výrazně neklesá, umožnil identifikovat T_{VA} na úrovni $43,63 \pm 4,66$ % MTR. Navržený algoritmus stanovení T_{VA} dovoluje odhadnout při tělesné práci „bezpečnou" intenzitu zatížení, při které je ještě zachována aktivita vagu a aktivita sympatiku se ještě výrazně nezvyšuje. Stanovení T_{VA} se může uplatnit zejména při preskripci intenzity zatížení v rámci programu pohybové aktivity u pacientů s redukovanou aktivitou ANS a se zvýšeným rizikem náhlé srdeční příhody.

Klíčová slova: spektrální analýza variability srdeční frekvence, intenzita zatížení, vagový práh, preskripce programu pohybové aktivity.

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