Medicina Sportiva, Vol. 9 (1): 28-32, 2005

CONTROLLED BREATHING AND HEART RATE VARIABILITY

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ABSTRACT

Purpose: The aim of this study was to compare SA HRV under conditions of spontaneous and controlled breathing with the use of a new SA HRV evaluation method.

Methods: The results of the short-term recording of spectral analysis (SA) of heart rate variability (HRV) in 21 healthy volunteers with additional vagal stimulation by means of controlled breathing are presented in this article. To a standard orthoclinostatic test (supine-standing-supine) three supine positions with controlled breathing (12, 9, 6 breaths/min) were added.

Results: Controlled breathing did not influence heart rate. With gradually decreasing breathing frequency (BF) and increasing breathing volume, a step by step increase in the average value of total spectral power (P_T) started. The highest value of P_T was found during the slowest BF (6/min). Also, the frequency of dominant fluctuations slowed down and a shift of spectral power from the HF zone towards the LF zone took place.

Conclusion: Because of these shifts, we were not able to use the usual interpretation of individual indexes as well as complex indexes of SA HRV.

Key words: heart rate variability, autonomic nervous system, spontaneous respiration, controlled respiration

Introduction

Spectral analysis (SA) of heart rate variability (HRV) is a non-invasive method enabling researchers to quantify the activity of the autonomous nervous system (ANS).

There are three main spectral components in the spectrum of short-term HRV recording: VLF (very low frequency, in our modification – 0.02 to 0.05 Hz) – its output is related to the thermoregulatory sympathetic activity of blood vessels, to the level of circulating catecholamines**,** or to oscillations in the renin-angiotensin system; LF (low frequency – 0.05 to 0.15 Hz) – reflects baroreflex activity on which sympathetic and parasympathetic activity participate; HF (high frequency – 0.15 to 0.50 Hz) – is influenced by efferent vagal activity only (1).

The spectral power of heart rate variability is influenced by many internal (age, health, gender, fitness, body position, etc.) and external factors (physical activity, daytime, season, quantity and quality of sleep, medication, fluid/food intake, alcohol, smoking, altitude, temperature, etc.). For example, the spectral power of HRV decreases during mental activity aging increases or some diseases on the other hand the regular exercise activity increases the spectral power of HRV (2, 3, 4).

Respiration also significantly influences the SA HRV results, especially its depth and frequency (BF), which cause periodic oscillations of heart rate (HR) – respiratory sinusoidal arrhythmia (RSA) (5). During this slowing down, the respiration frequency at 6 breaths/min, the RSA comes up to a maximum (6). An abnormal RSA is very often the first sign of ANS dysfunction in many diseases.

Slower RF produces a higher RSA and the power of the HF component increases as well (proof of increased vagal activity); on the other hand, with a faster respiration the HF component is reduced (7, 1, 4, 8). Slower than 0.15 Hz (less than 9 breaths/min) shift in the respiratory component to the LF zone, in which, next to the vagal activity also sympathetic activity, takes place (9, 10, 11).

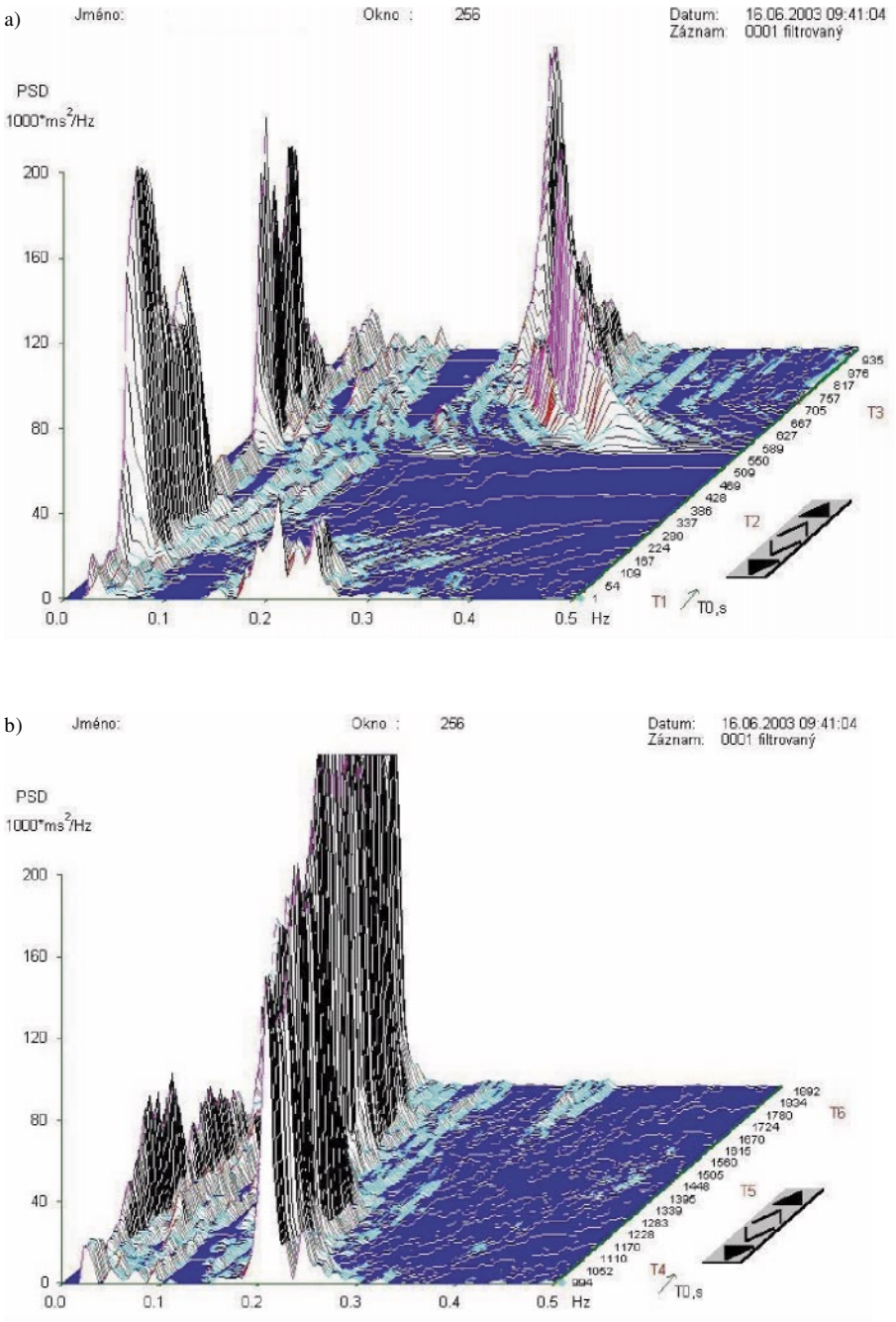
The aim of this study was to compare SA HRV under conditions of spontaneous and controlled breathing with the use of a new SA HRV evaluation method (3).

Materials and Methods

SA HRV evaluation by complex parameters

SA HRV evaluation by individual indexes is relatively difficult and very often relatively less conclusive and the antagonistic results can be found. The discrepancies in the testing results have prompted the authors to create a new evaluation method for SA HRV (3).

The authors worked under the assumption that the most important external factors influencing ANS function are aging and some diseases. Exercise intensity significantly influences HRV spectral power as well: gradually descending vagal activity is presented by gradually descending total spectral power and the power of individual components. The gradu-



T1 = supine (spontaneous breathing); T2 = standing (spontaneous breathing); T3 = supine (spontaneous breathing); T4 = supine (12 breaths/min); T5 = supine (9 breaths/min); T6 = supine (6 breaths/min)

Fig. 1. *Three-dimensional graph of SA HRV*
a) measured in positions P1 (T1), P2 (T2) and P3 (T3) during the orthoclinostatic test (supine-standing-supine) with spontaneous breathing;
b) measured in positions P4 (T4), P5 (T5) and P6 (T6) (supine-supine-supine) with controlled breathing (12, 9, 6 breaths/min)

al transfer of spectral power to slower fluctuations is proved by an increase in the VLF percentage and its ratio to LF and HF components (VLF/LF a VLF/HF) (2).

Due to previous findings for the new evaluation of SA HRV, the authors used all age dependent parameters obtained from the orthoclinostatic manoeuvre from 216 healthy volunteers aged 12 to 70 years (35.05±14.30) (12). Parameters decreasing with age, disease and exercise intensity are combined in the complex index of vagal activity (VA). Parameters increasing with age, disease and exercise intensity are joined in the complex index of sympatho-vagal balance (SVB) (2). By combining these two parameters, we acquired a so-called total score (TS) of SA HRV (which reflects all of the combined age dependent parameters). By relating the total score to calendar age we arrived at so-called functional age of ANS (3).

All complex indices are represented by point values from +5.0 to –5.0 points. Positive values mean high vagal activity and shift of the sympatho-vagal balance towards the vagus; negative values have the opposite meaning.

The new SA HRV evaluation method leads to a more unambiguous identification of less noticeable changes in the power spectrum; orientation in the complex indices of SA HRV is, in comparison to the standard parameters, easier, and the interpretation of results less complicated.

Methodology

We tested 21 volunteers aged 32.02 ± 12.57 years (7 male and 14 female). All subjects were healthy and free of medication except for women taking hormonal contraceptives. Of the subjects, 7 carried out regular aerobic physical activity; all subjects were non-smokers.

We measured HRV by means of the original hardware and software VarCor PF5 (13). The number of recordings of cardiac cycles was increased from 300 to 330 (14) because of the time data stationarity and the possibility of improving the filtration of multiple arrhythmias and artifacts. For the SA HRV calculation the standard number of 256 R-R intervals was used.

The fasting subjects were examined by means of an orthoclinostatic test in a quiet room in the morning. Subjects kept their eyes closed during the entire testing process. In each of the three positions (P) of the orthoclinostatic test (supine – P1, standing – P2, supine – P3), subjects stayed until 330 heart contractions had been recorded. ECG registration started 45 seconds after changing each position. Thus, all periods of measurements were carried out in a steady-state. To this standard orthoclinostatic test (supine-standing-supine) another three positions with controlled breathing were added P4 (supine, 12 breaths/min) P5 (supine, 9 breaths/min) P6 (supine 6 breaths/min) (Fig. 1). The breathing cycle during controlled breathing was divided into a 2:3 ratio (inspiration : expiration ratio). Thus during 12 breaths/min (0.2 Hz), one breathing cycle took 5 seconds (2s inspiration; 3s expiration); during 9 breaths/min (0.15 Hz) one breathing cycle took 6.66 seconds (2,66s inspiration; 3,99s expiration) and during 6 breaths/min (0.1 Hz) one breathing cycle took 10 seconds (4s inspiration; 6s expiration). Breathing was directed by the verbal instructions "breathe in, two, breathe out, two, three" in the case of 12 breaths/min from a compact disc (CD). A CD recording with an accurate breathing frequency was made with the help of Cool Edit software. The ECG record from the first position was not evaluated and this was only used for measurement standardization. The P2 and P3 positions (ANS reaction to orthostatic and clinostatic tests during spontaneous breathing) or the P2 – P4, P2 – P5 and P2 – P6 positions (ANS reaction to orthostatic and clinostatic tests during controlled breathing) were evaluated.

The following individual SA HRV parameters for statistical processing were chosen: total spectral power (P_T), spectral power of individual components (P_{VLF}, P_{LF}, P_{HF}), percentage of the individual components (%VLF, %LF, %HF) and ratios between individual components (VLF/HF, LF/HF, VLF/LF). In addition to these parameters, also complex indices of SA HRV (TS, VA, and SVB) were calculated.

Basic statistical characteristics (average and standard deviation) and the paired Wilcoxon test were calculated by means of the software programs SPSS and Microsoft Excel.

Results

BF was, during spontaneous breathing, between 13.81 ± 4.08 breath per minute.

SF, P_{VLF} and the VLF/HF ratio did not change significantly during spontaneous and controlled breathing (Table 1).

Average value of P_T increased with decelerating BF, but statistical significance was determined only at BF = 6/min.

P_{HF} and %HF were significantly higher within controlled breathing 12/min than within spontaneous breathing, whereas P_{LF} and %LF decreased. On the contrary, during controlled breathing 6/min P_{HF} and %HF decreased and P_{LF} and %LF increased. That is why the LF/HF ratio decreased and the VLF/LF ratio increased within controlled breathing 12/min and the LF/HF ratio increased and VLF/LF decreased within controlled breathing 6/min (Table 1).

All complex parameters (TS, VA a SVB) significantly increased during controlled breathing 12/min. whereas during controlled breathing 6/min all complex parameters decreased (Table 1).

Discussion

A strict standardization is necessary for SA HRV measuring due to many external and internal factors which can considerably influence HRV (7, 1, 4); that is why we tried measuring conditions in a maximally standardized manner (measuring approximately at the same times of day and minimizing sensual stimulations).

An important factor influencing RSA is the ratio between duration of expiration and inspiration. The RSA was larger in trials with short inspiration followed by long expiration than in trials with long inspiration followed by short expiration (15). That is why we kept the same expiration/inspiration ratio during all frequencies of controlled breathing (ratio 2:3). In further research projects it will be necessary to investigate the influence not only of various breathing frequencies but also investigate in detail the influence of different duration of inspiration and expiration on HRV.

Table 1. *Comparison of heart rate and SA HRV parameters during spontaneous and controlled breathing*

		spontaneous breathing	12/min	9/min	6/min
HR	$\overline{X}$	57.84	58.76 NS	58.44 NS	58.42 NS
	SD	6.19	6.85	7.28	7.07
P_T	$\overline{X}$	3182.26	3261.83 NS	4168.99 NS	5127.25 **
	SD	3018.42	2605.84	3719.68	3637.32
P_{VLF}	$\overline{X}$	209.46	211.70 NS	208.37 NS	188.34 NS
	SD	211.83	159.85	158.12	188.30
P_{LF}	$\overline{X}$	623.18	244.80 **	1046.31 *	3774.86 ***
	SD	824.45	238.55	963.16	2790.35
P_{HF}	$\overline{X}$	2349.62	2805.33 *	2914.30 NS	1164.04 **
	SD	2336.63	2476.22	2779.80	1142.96
%VLF	$\overline{X}$	11.08	9.51 NS	9.03 NS	5.96 *
	SD	10.28	9.05	8.91	6.34
%LF	$\overline{X}$	22.51	10.87 ***	25.62 NS	67.97 ***
	SD	17.68	11.20	10.34	19.60
%HF	$\overline{X}$	66.41	79.62 **	65.35 NS	25.53 ***
	SD	23.06	18.36	13.83	16.17
VLF/HF	$\overline{X}$	0.34	0.18 NS	0.18 NS	0.36 NS
	SD	0.69	0.32	0.25	0.55
LF/HF	$\overline{X}$	0.68	0.21 ***	0.45 NS	5.31 ***
	SD	1.28	0.34	0.30	4.52
VLF/LF	$\overline{X}$	0.76	1.18 **	1.41 *	0.11 **
	SD	0.81	0.82	5.03	0.22
VA	$\overline{X}$	0.17	0.67 **	0.56 *	-0.86 ***
	SD	1.65	1.36	1.44	1.61
SVB	$\overline{X}$	0.96	1.94 **	1.15 NS	-1.03 **
	SD	1.87	1.62	1.34	1.46
TS	$\overline{X}$	0.33	0.92 *	0.61 NS	-1.10 ***
	SD	1.79	1.73	1.66	1.80

$\overline{X}$= average value; SD = standard deviation; HR = heart rate; VLF = very low frequency; LF = low frequency; HF = high frequency; P_T = total spectral power; P_{VLF} = VLF power; P_{LF} = LF power; P_{HF} = HF power; %VLF, %LF, %HF = relative part of the individual component in total power; VLF/HF, LF/HF, VLF/LF = ratio of spectral power of separate components; VA = complex index of vagal activity; SVB = complex index of sympatho-vagal balance; TS = total score of SA HRV; NS = no significant difference; * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

In a previous study (16), we investigated the influence of HRV during controlled breathing – 12 breaths/min. It was found that controlled breathing did not influence the HR. In this study we found the same results and also during a follow-up decrease of BF (9 breaths/min and 6 breaths/min) the HR did not change.

Controlled breathing with a frequency of 0.2 Hz (12 breaths/min) did not change the P_T compared with spontaneous breathing and, rather than absolute changes of the HRV spectrum, a shift of spectral power towards faster fluctuations took place (decrease in P_{LF} and %LF and increase in P_{HF} a %HF). This result confirms the conclusions of Malik and Camm (7) – controlled breathing with a frequency between 0.20 and 0.30 Hz is characterized by the shift of sympathovagal balance towards the vagus. SA HRV evaluation by complex parameters unambiguously showed increased vagal activity within BF 12/min (16).

Vagal activity during BF 9/min was also higher (increased VA) than during spontaneous breathing, although the increase in VA was lower than during 12 breaths/min; the changes in P_{HF} and %HF were significant.

A significant increase in P_{LF} and %LF together with a decrease in P_{HF} and %HF during 6 breaths/min indicate a shift of spectral power from the HF band to the LF band. This shift indicates a decrease of vagal activity (Table 1). The same result was also found by Aguirre et al. (9) and Pitzalis et al. (11). According to these authors, during slower BF than 9/min, the respiratory wave is not only a reflection of vagal activity but is partly influenced by sympathetic activity. During deep breathing approaching 0.1 Hz, P_{HF} can not be used as an index of vagal activity (10).

A significant increase in P_T during 6 breaths/min corresponded with the slowing down of the dominant spectral power and its shift from the HF band to the LF band. Contribution of vagal activity to the LF component increased while the power of the HF component decreased. Thus, the conditions for evaluation by complex indexes did change and a significant increase in the LF/HF ratio was not a result of a sympathetic activity increase, just as a decrease in HF power is not an outcome of a vagal activity reduction. The LF/HF ratio, which is considered to be an index of sympathovagal balance, did lose this value within BF < 9/min. Due to the above we were not able to interpret individual spectral power indexes of HRV within BF < 9/min in the same way as within BF ≥ 9/min and we can not use complex indices either. Accordingly, BF control also seems to be a necessary part of ANS evaluation by the SA HRV.

Conclusion

The border between the HF zone (only vagal activity) and LF zone (mixed zone of sympathetic and vagal activity) is 0.15 Hz. A decrease in BF under 0.15 Hz causes principal difficulties during SA HRV interpretation and excludes to use complex indexes in SA HRV evaluation.

References

1. Stejskal P, Salinger J. Spektrální analýza variability srdeční frekvence. Základy metodiky a literární přehled o jejím klinickém využití. *Med Sport Boh Slov* 1996; 2: 33-42.
2. Stejskal P, Rechbergová J, Salinger J, et al. Power spectrum of heart rate variability in exercising humans: The effect of exercise intensity. *Sports Training Med. Rehab.* 2000; 10(1): 39-57.
3. Stejskal P, Šlachta R, Elfmark M, et al. Spectral analysis of heart rate variability: new evaluation method. *Gymnica* 2002; 32(2): 13-18.
4. Šiška E. Variabilita srdeční frekvence – možnosti využití ve výzkumu a v klinické praxi. *Československá psychologie* 2000; 44(3): 266-278.
5. Opavský J. Autonomní nervový systém a diabetická autonomní neuropatie, klinické aspekty a diagnostika. Praha, Galén, 2002.
6. Bernardi L, Porta C, Gabutti A, et al. Modulatory effects of respiration. Autonomic Neuroscience: *Basic Clin* 2001; 90: 47-56.
7. Malik M, Camm AJ. Heart rate variability. New York: Futura publishing company, Inc., 1995.
8. Šlachta R. Sledování závislosti hodnot ukazatelů spektrální analýzy variability srdeční frekvence na věku vyšetřovaných osob. Disertační práce, Universita Palackého, Fakulta tělesné kultury, Olomouc, 1999.
9. Aguirre A, Wodicka GR, Maayan C, Shannon DC. Interaction between respiratory and RR interval oscillations at low frequencies. *J. Auton. Nerv. Syst.* 1990; 29(3): 241-246.
10. Barron SA, Hemli J. Sinus arrhythmia during deep breathing is not all respiratory. *Electroencephalogr Clin Neurophysiol* 1995; 95(3): 82.
11. Pitzalis MV, Mastropasqua F, Massari F, et al. Effect of respiratory rate on the relationships between RR interval and systolic blood pressure fluctuations: a frequency-dependent phenomenon. *Cardiovasc Res* 1998; 38(2): 332-339.
12. Šlachta R, Stejskal P, Elfmark M, et al. Age and spectral analysis of heart rate variability. *Gymnica* 2002; 32(1): 59-67.
13. Salinger J, Stejskal P, Opavský J, et al. New diagnostic system, type VarCor PF6, for non-invasive assessment of the function of the autonomic nervous system. *Clin Autonom Res* 2003; 13(2): 125.
14. Stejskal P, Salinger J. Několik poznámek ke spektrální analýze variability srdeční frekvence. *Med Sport Boh Slov* 2003; 12(1): 40-41.
15. Strauss-Blasche G, Moser M, Voica M, et al. Relative timing of inspiration and expiration affects respiratory sinus arrhythmia. *Clin Exp Pharmacol Physiol* 2000; 27(8): 601-606.
16. Žujová E, Stejskal P, Jakubec A, et al. J. Respiration frequency and spectral analysis of heart rate variability. *Gymnica* 2004; 34(1): 43-47.

Received: August 17, 2004
Accepted: December 15, 2004

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