Baroreflex Sensitivity During Psychological Stress

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Summary

The aim of this study was to analyse the changes of baroreflex sensitivity (BRS) and their relation to changes of heart rate and blood pressure in medical students during moderate psychological stress brought about by oral examination. The changes of BRS during the stress were compared with the changes during light physical exercise. Thirty three students were examined 30 min before and 30 min after the exam. Thirty-nine students of control group were examined at rest and during light exercise. Blood pressure was noninvasively recorded by Peňáz method at rate-controlled breathing (0.33 Hz). The BRS [ms/mm Hg] and BRS_f [Hz/mm Hg] were calculated by spectral analysis of spontaneous fluctuations of blood pressure and inter-beat intervals (IBI). BRS before examination (7.12 ms/mm Hg) was significantly lower than after the oral exam (8.77 ms/mm Hg, p<0.05). The difference between BRS in the test group after the oral exam and the control group at rest (10.78 ms/mm Hg) was not significant. BRS during light exercise (7.44 ms/mm Hg) corresponded to the value during psychological stress. The values of BRS_f did not change during psychological stress (before: 0.0182 Hz/mm Hg; after: 0.0182 Hz/mm Hg; after: 0.0188 Hz/mm Hg; and blood pressure were not found. A significant negative correlation (r = -0.404, p<0.05) between BRS_f and blood pressure were not found. A significant negative correlation (r = -0.404, p<0.05) between BRS_f and the increase of diastolic blood pressure during stress was observed. It is concluded that BRS_f remained constant during psychological stress and exercise, and differed essentially from that in hypertensive subjects.

Key words

Baroreflex sensitivity - Exercise - Psychological stress - Blood pressure - Heart rate

Introduction

The measurement of baroreflex sensitivity (BRS) in man was introduced by Smyth et al. (1969). The principle of the method is the determination of the slope of the line expressing the relationship between the phenylephrine-induced increase of systolic blood pressure and the increase of inter-beat interval caused by the baroreflex. Since then, this and other invasive and noninvasive methods have been used to measure BRS in various physiological and pathological states. The non-invasive spectral method is based on continuous blood pressure recordings according to Peňáz (1973). BRS corresponds to the gain of the system with spontaneous systolic blood pressure fluctuations as input and cardiac intervals fluctuations as the output and is expressed in ms/mm Hg (Robbe et al. 1987). In an early study, Mulder (1989) showed that BRS significantly decreases during a mental load.

During physical exercise, the absolute changes in heart rate elicited by a given change in pressure are remarkably similar to those in rest (Bevegard and Shepherd 1966). However, when these changes in heart rate are evaluated from the inter-beat interval changes, the data indicate a reduced gain for baroreflex control of heart rate. This indicates a correlation between BRS and heart rate during exercise.

On the other hand, a negative correlation between BRS and blood pressure was found by many authors if hypertensive subjects had not been excluded (Gribbin *et al.* 1971).

In our study, we measured the changes of BRS during psychological stress caused by final oral examination (pathological physiology) of medical students. The changes of BRS during psychological stress were also compared with changes during light physical exercise. Our main interest was to find if changes in BRS during psychological stress correlate with changes in the inter-beat interval or in blood pressure.

Psychosocial factors such as job strain, modulating baroreflex gain centrally may contribute to the development of human hypertension (Sleight 1991). For this reason we wanted to study if BRS plays a role in the blood pressure changes induced by psychological stress.

Subjects and Methods

Thirty three medical students (12 men and 21 women, between 20 and 25 years of age) were tested 30 min before an oral examination in pathological physiology and then 30 min after successfully passing the examination. The control group consisted of 39 students (all men) of the same age, tested 2 months later after the examination. All subjects gave their informed consent. At least 30 min before the test the subjects were resting and adjusting to the environment. Meanwhile, a photoplethysmographic transducer, Finapres Ohmeda, was mounted. The blood pressure signal was monitored for 10 min of rest. Three minute records were taken at the end of the resting period for further analysis. Then the subjects were instructed to breath in synchrony with a metronome at 0.33 Hz for 5 min. The tidal volume was not controlled, the subjects were instructed to adjust their breath depth to achieve full comfort. The last 3 min were recorded and analysed. The procedure was repeated 30 min after the examination. In addition, the subjects of the control group were asked to lift a load of 2 kg to a height of 1 m in synchrony with the metronome. These the breathing and lifting were synchronized at 0.33 Hz. The subjects exercised for 6 min and the last 3 min were analysed. From the non-invasive continuous blood pressure record the following parameters were computed and analysed: systolic and diastolic blood pressure (SBP and DBP, respectively) and inter-beat (pulse) interval (IBI).

Values of SBP, DBP and IBI were linearly interpolated at 2 Hz to ensure equidistant sampling in each time series. The baseline linear trend was removed from all signals. The power spectral densities and cross-spectral densities were calculated from the auto- and cross-correlation functions using the Hanning spectral window. The value of cross-spectral power density of pulse intervals and systolic blood pressure fluctuations [ms x mm Hg] was divided by the value of spectral power density of systolic blood pressure fluctuation [mm Hg x mm Hg] at 0.1 Hz. The obtained value, modulus, was considered the measure of BRS [ms/mm Hg] (Pagani et al. 1988). The coherence at 0.1 Hz, i.e. the degree of linear coupling between SBP and IBI fluctuations was calculated from both the spontaneous and the controlled breathing records of all subjects.

Baroreflex sensitivity can also be evaluated as heart rate change per change of pressure in $[Hz/mm Hg] BRS_f$. BRS_f was calculated according to the following formulas:

1. f[Hz] = 1/IBI[s].

2. dIBI = BRS x mm Hg.

3. f - df = 1/(IBI + dIBI).

Thus df = f - 1/(IBI + dIBI).

4. BRS_f = df/mm Hg.

The comparison between test and control data, between pre- and postexamination data as well as the comparison of data before and during exercise in control subjects were performed using the Wilcoxon test for paired data. The correlation was evaluated by means of least square linear regression analysis and expressed as a correlation coefficient. The data are presented as mean values \pm S.D. Statistical significance was accepted for values p < 0.05.

Results

Table 1A presents the mean values (\pm S.D.) of IBI, SBP, DBP, BRS and BRS_f obtained in the test group before and after the examination during controlled breathing rate as well as the correlation coefficients calculated among these parameters. The differences between mean values of IBI, SBP and DBP before and after the examination were statistically significant (p<0.01). BRS values before and after the examination were significantly different (p<0.05) but BRS_f was not changed. No significant sex differences were found in any of the variables.

The comparison of the data obtained after the exam with the resting data in the controls (Table 1B) showed the only significant difference in IBI (p < 0.05) indicating that IBI was not normalized within 30 min after the examination. In contrast, SBP, DBP, BRS and BRS_f changed only insignificantly. The significantly higher SBP during rest (p < 0.05) corresponds to the decrease in heart rate at unchanged mean blood pressure (Fig. 1). In the control group, exercise increased SBP (p < 0.01), DBP (p < 0.01), decreased IBI (p < 0.01) and BRS (p < 0.01), but did not change BRS_f.

In the control group during exercise the mean values of IBI did not significantly differ from the value obtained either before or after the examination in the test group. The mean values of systolic and diastolic pressure were higher (p < 0.05).

The correlation coefficients among the variables revealed a significant positive correlation between IBI and BRS in all situations, i.e. before and after the examination in the test group, at rest and during physical exercise in the control group. No correlation was found between BRS_f and IBI. Correlation between BRS and blood pressure, and between BRS_f and blood pressure were not found.

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Values of IBI, SBP, DBP, BRS, and BRS_f and the correlation coefficients between pairs of values obtained. Test group: 30 min. before and 30 min after examination. Control group: at rest and during exercise.

	IBI (ms)	SBP (mm Hg)	DBP (mm Hg)	BRS (ms/mm Hg)	BRS _f (Hz/mm Hg)	
Test group		Before examination				
Mean ± S.D.	608.7±96.8	122.9 ± 16.1	75.2±9.1	7.12 ± 3.30	0.0182 ± 0.0059	
SBP DBP BRS BRS _f	-0.051 -0.292 0.809** 0.241	0.657** -0.188 -0.208	-0.238 -0.118	0.731**		
		After examination				
Mean ± S.D.	677.3±101.1	112.7 ± 15.5	71.1 ± 8.9	8.77 ± 4.68	0.0182 ± 0.0070	
SBP DBP BRS BRS _f	-0.150 -0.369* 0.635** 0.151	-0.692** -0.312 -0.248	-0.106 0.109	0.829**		
Control group			At rest			
Mean ± S.D. SBP DBP	806.9±124.4 -0.007 -0.055	128.1±15.6 0.750**	73.5±8.4	10.78±6.57	0.0158 ± 0.0067	
BRS BRS _f	0.570**	0.152	0.122 0.136	0.826**		
Durin			During exercise			
Mean ± S.D.	674.8 ± 102.1	148.9 ± 29.5	88.1±11.2	7.11 ± 4.78	0.0144 ± 0.0054	
SBP DBP BRS BRS _f	-0.140 -0.253 0.522** 0.028	-0.691** -0.273 -0.244	-0.267 -0.214	0.619**		

Significance of correlation coefficients: * p < 0.05; ** p < 0.01.

Power spectra of IBI, SBP and DBP fluctuations are shown in Figures 2 and 3. The shape of spectra of the three variables in all experimental situations is similar. Only the modulus function calculated as the ratio of cross-spectral density of SBP and IBI fluctuations and power spectral density of SBP fluctuation at 0.1 Hz is lower before the examination in the test group and during exercise in the control group. We analysed the role of BRS and BRS_f on changes of blood pressure and heart rate elicited either by psychological stress or by exercise. The results are presented in Table 2. Positive correlation between BRS_f and difference in DBP indicates that high BRS_f attenuated the increase of diastolic blood pressure during psychological stress. Similar trends, but not statistically significant, were seen in changes of blood pressure elicited by exercise.



Fig. 1. The mean values $\pm S.D.$ of inter-beat interval (IBI) [ms], systolic and diastolic blood pressure (BP) [mm Hg], baroreflex sensitivity (BRS) [ms/mm Hg] and baroreflex sensitivity BRS_f [Hz/mm Hg] in the test group (T) before (B) and after (A) the examination, and in the control group (C) during rest (R) and exercise (E).

Table 2

Above: Values of BRS (after), BRS_f (after) obtained after the examination in the test group, difference of systolic blood pressure (Δ SBP_{b-a}), and of diastolic blood pressure (Δ DBP_{b-a}) before and after the examination and the correlation coefficients between pairs of values. Below: Values of BRS(rest), BRS_f(rest) obtained at rest in the control group, difference of systolic blood pressure (Δ SBP_{e-r}) and of diastolic blood pressure (Δ DBP_{e-r}) at rest and during exercise and the correlation coefficients between pairs of values.

		Test group (Psychological stress)					
	BRS (after) (ms/mm Hg)	BRS _f (after) (Hz/mm Hg)	ΔSBP _{b-a} (mm Hg)	ΔDBP (mm Hg)			
Mean ±S.D. BRS _f (after)	8.77±4.68 0.829**	0.0182 ± 0.0070	10.07 ± 11.84	4.17±8.25			
ΔSBP	0.057	-0.148					
ΔDBP	-0.280	-0.404*	0.655**				
		Control group (Exercise)					
	BRS(rest) (ms/mm Hg)	BRS _f (rest) (Hz/mm Hg)	ΔSBP _{e-r} (mm Hg)	ΔDBP_{e-r} (mm Hg)			
Mean ±S.D. BRS _f (rest)	10.78±6.57 0.826**	0.0158 ± 0.0067	23.37±14.58	14.87±8.03			
ΔSBP	-0.180	-0.229					
ΔDBP	-0.127	-0.183	-0.623**				

Significance of correlation coefficients: * p < 0.05; ** p < 0.01.



Fig. 2. Mean power spectra $(\pm S.D.)$ of IBI, SBP, and DBP fluctuations, modulus between IBI and SBP fluctuations (MOD) and coherence (COH) between IBI and SBP fluctuations before and after the examination.



Fig. 3. Mean power spectra $(\pm S.D.)$ of fluctuations of IBI, SBP, DBP, modulus between fluctuations of IBI and SBP (MOD) and coherence (COH) between IBI and SBP fluctuations at rest and during exercise.

Discussion

The introduction of techniques which allowed the measurement of intra-arterial blood pressure *via* portable recorders made it possible to study haemodynamic responses to emotional behaviour when the subjects examined were living in their usual environment. Such monitoring in medical students who underwent an examination in internal medicine revealed an increase of blood pressure observed several hours before the exam (Mancia 1986). In another subject playing poker for several hours, an increase of blood pressure was recorded throughout the game (Parati *et al.* 1986).

Since intra-arterial cannulation may involve emotional stress and may affect cardiovascular reflex control, the introduction of non-invasive blood pressure measurement by the Peňáz method brings forth obvious advantage (Hainsworth 1990, Wieling and Wesseling 1993). This technique has made it possible to estimate BRS by spectral analyses during mental arithmetics (Mulder 1989). Both values of BRS, at rest (12.1)ms/mm Hg) and during mental load (9.9 ms/mm Hg) were higher in Mulder's experiments than the BRS observed before and after examination in our study. However, the resting value of BRS in our control group was similar to that of Mulder. This comparison suggests that the emotional impact of mental arithmetic is probably lower than the real psychological stress brought about by the examination. The finding of Parati et al. (1986) that the emotional impact of mental arithmetic may vanish in time is another example demonstrating the limitations of laboratory stressors.

Our study of BRS and BRS_f during exercise confirmed the results of various studies in man

(Bevegard and Shepherd 1966, Fišer *et al.* 1992) and in the dog (Melcher and Donald 1981). During exercise, depending on the index of baroreflex function used (absolute changes in heart rate versus the R-R interval), different conclusions can be reached, with the former indicating a decrease and the latter an unchanged gain for the reflex. The same conclusion can be drawn from studying psychological stress.

BRS is decreased in patients with essential hypertension. Using the inter-beat interval and BRS we can also calculate BRS_f. Using spectral analysis in normotensive subjects, Pagani *et al.* (1988) found BRS to be 10 ± 2 ms/mm Hg, SBP: 133 ± 3 mm Hg, IBI: 828 ± 37 ms. The corresponding values in hypertensive patients were: SBP: 177 ± 9 mm Hg, IBI: 799 ± 27 ms and BRS 4 ± 1 ms/mm Hg. The corresponding BRS_f value was 0.0144 Hz/mm Hg for normotensive and 0.0062 Hz/mm Hg for hypertensive subjects. In another series of measurements (Siegelová *et al.* 1995), we found BRS to be 7.8 ± 3.8 ms/mm Hg in normotensives (121/74 mm Hg, 75.4 beats/min) and 4.7 ± 1.8 ms/mm Hg in hypertensives (146/88 mm Hg, 79.6 beats/min). Corresponding BRSf was 0.0122 Hz/mm Hg for normotensive and 0.0082 Hz/mm Hg for hypertensive individuals. In both studies, BRS_f in normotensives corresponded to the average values of our subjects. BRS_f of hypertensives was lower. This means that we can not compare healthy subjects under psychological stress and patients with essential hypertension. On the other hand, some values of BRS_f in our healthy subjects are in the range of hypertensives as is indicated by the S.D. of the BRS_f it can not be decided on the basis of our results, whether the subjects with low BRS_f are at a higher risk for developing hypertension. However, the negative correlation between BRS_f and stress-induced increase of DBP suggests that low BRS_f can contribute to the development of hypertension. The exaggerated response to stressors is a risk factor for the development of this disease (Kaplan 1995). Our data support the hypothesis that the exaggerated increase of blood pressure as a response to psychological stress can be the link between low baroreflex sensitivity and essential hypertension.

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