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Increase in perceived stress is correlated to lower heart rate variability in healthy young subjects

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ABSTRACT. Emotional stress is a risk factor for cardiovascular disease. Decreased heart rate variability (HRV) is associated to increased mortality rates in certain heart diseases. Current study assessed the co-relation between perceived stress and HRV parameters. The correlation between psychological stress, measured by the perceived stress scale (PSS-14), and HRV parameters obtained during 5 min. at rest was evaluated. Data from 35 healthy young volunteers demonstrated a significant correlation between PSS-14 scores and Low Frequency-LF (ms²) by frequency domain HRV analysis. Other variables such as High Frequency and Standard Deviation of R-R intervals had also negative coefficients but did not have any significant correlation with PSS-14. No correlation between PSS-14 and sympathovagal balance parameters was found. Data interpretation demonstrated that increase in perceived stress was correlated to decrease in heart rate variability, which may point out an important mechanism in cardiovascular pathophysiology that should be further investigated.

Keywords: heart rate, psychological stress, cardiovascular diseases.

Aumento no estresse percebido correlaciona-se com diminuição da variabilidade da frequência cardíaca em indivíduos jovens saudáveis

RESUMO. O estresse emocional é um fator de risco para doença cardiovascular. A diminuição da Variabilidade da Frequência Cardíaca (VFC) está associada ao aumento da mortalidade em algumas doenças cardíacas. Este estudo teve como objetivo avaliar a correlação entre o estresse percebido e os parâmetros da VFC. Dessa maneira, avaliou-se a correlação existente entre o estresse percebido e os parâmetros da vFC. Dessa maneira, avaliou-se a correlação existente entre o estresse psicológico mensurado através da escala de estresse percebido (PSS-14) e parâmetros de VFC obtidos durante 5 min. ao repouso. Dados de 35 voluntários jovens e saudáveis demonstraram uma correlação significativa do PSS-14 com os valores de baixa freqüência- LF (ms²) na análise da VFC no domínio frequência. Outras variáveis, tais como alta freqüência e desvio padrão dos intervalos RR, também tiveram coeficientes negativos, mas não se correlacionaram com a PSS-14. Nenhuma correlação entre o PSS-14 e os parâmetros do balanço simpato-vagal foi encontrada. Portanto, a interpretação dos dados demonstra que o aumento no estresse percebido se correlacionou com a diminuição da variabilidade da frequência cardíaca, o que pode ser indicativo de um mecanismo importante na fisiopatologia cardiovascular que deve ser melhor investigado.

Palavras-chave: frequência cardíaca, estresse psicológico, doenças cardiovasculares.

Introduction

There are several acknowledged risk factors in cardiovascular diseases. Although many are additive and/or synergic, most are preventable (WHO, 2007). Among the risk factors, stress is a highly prevalent factor worldwide. Psychological stress, depression and anxiety may participate in the development of cardiovascular diseases (BRYDON et al., 2010). The cardiovascular system is in constant adaptation to physical and emotional stress and the integrity and harmonic work of the autonomic nervous system is essential to cardiovascular regulation and, consequently, to body homeostasis.

Heart rate variability (HRV) is a non-invasive way to access the integrity of the autonomic nervous system and to evaluate the sympathovagal balance. HRV is based on the acquisition of cardiac electrical signals and analysis of R-R intervals. Since short term recordings are relatively easy to collect in a clinical setting, it is an accepted and recommended method (KLEIGER et al., 2005).

Decrease in HRV is associated to increased morbidity and mortality rates in such conditions as

diabetes, coronary arterial disease and heart failure (GERRITSEN et al., 2001; ROY et al., 2013). In fact, acute emotional and mental stress has been associated to alterations in heart rate variability (DIKECLIGIL; MUJICA-PARODI, 2010; KUDO et al., 2014).

Due to the fact that HRV reflects cardiac autonomic control and that alterations in HRV are associated to cardiovascular mortality, a hypothesis may be raised that increased psychological stress levels in daily activities may be associated to alterations in HRV. Current assay assesses the correlation between HRV and perceived stress in healthy young volunteers.

Material and methods

Study design and subject

All procedures were previously approved by the local Research Ethical Committee (CEP-HUOL, approval #534/11) and followed the Brazilian Ministry of Health's resolution 196/96. Subjects' inclusion criteria comprised being older than 18 years; no prior history or diagnosis of chronic or psychiatric disease; absence of an active systemic disease; no hospitalization within a period of 3 months; no prior history or diagnosis of cardiac disease or use of drugs that would interfere with HRV; not currently using any electronic medical equipment (contraindication for bioelectrical impedance). Forty-two subjects were invited and data were collected from 40 individuals for further analysis. A total of 35 healthy young subjects (19 to 29 years old; 6 males) met the inclusion criteria. Since the data collected passed HRV standards for measurements (described in the next section), they were included in current study. Exclusions were due to poor signal acquisition in 2 cases; others were due to background artifacts that did not allow precise analysis of continuous 5 min. recordings even after subtracting the linear tendency using automatic filters, as described in the section below.

Heart rate variability assessment

Previous to HRV assessment, volunteers were informed they had to refrain from caffeinated beverages on the day of the exam; they should have a good night of sleep and avoid exhaustive physical exercises the day before the examination. All data were collected in the morning (8:00 to 11:30).

After the subjects rested for 10 min. in a supine position in a temperature-controlled room (21 to 24°C), the cardiac electrical signal was monitored by two electrodes positioned on the skin surface, or rather, one on the area beside the xiphoid process and another on the anterior axillary line at the fifth

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intercostal space. Both were connected to a heart rate monitor (RS800CX, Polar Electro Inc, USA) (RUHA et al., 1997) which measured and recorded R-R intervals within one millisecond resolution.

Data were transferred to a computer and the R-R intervals were processed to calculate HRV parameter with a Kubius HRV analysis software (Kuopio, Finland). HRV was analyzed in the frequency domain, using the area of greatest stability in R-R intervals corresponding to 5 min. of recordings (containing at least 256 consecutive beats) during spontaneous breathing. Frequency domains were analyzed by Fast Fourier Transform after subtracting the linear tendency using automatic filters. The domains were studied using low (LF: 0.04 to 0.15Hz) and high (HF: 0.15 to 0.4Hz) frequency ranges in spectral density (ms²), normalized units (n.u.) and the LF/HF ratio. So that the correlation of perceived stress and HRV could be better assessed, time domain parameters were calculated as follow: pNN50 - the percentage of intervals more than 50 ms different from preceding interval; SDNN-Standard deviation of all R-R intervals; rMSSD-Square root of the mean of the squares of successive R-R interval differences. Further, the R-R triangular index based on the plotted histogram was calculated.

Psychological stress assessment

Psychological stress was assessed by the Perceived Stress Scale (PSS-14) instrument (COHEN et al., 1983). The translated and validated version in Brazilian Portuguese was used by self-administration (LUFT et al., 2007). In brief, the questionnaire quantifies the level of stress perceived by the individual when facing stressful situations. The instrument is composed of 14 questions with 0-4 scores. Total score from the sum of questions may vary from 0 to 56 and the perceived stress is higher as scores increase.

Statistical analysis

Data are given as mean \pm standard deviation. Data normality was verified by the Kolmogorov-Smirnov test. Correlation of data was determined by the calculation of Spearman's correlation coefficient and p < 0.05 was considered a criterion for statistical significance.

Results and discussion

Table 1 summarizes clinical and anthropometric characteristics of the population and also average for the perceived stress scale.

Perceived stress and heart rate variability

 Table 1. Clinical and anthropometric characteristics of the population and Perceived Stress Scale Score (PSS).

	$Mean \pm SD$	Mean CI	
Age (years)	21.3±2.7	20.3-22.2	
BP _{systolic} (mmHg)	104.0 ± 9.1	107.1-109.9	
BP _{diastolic} (mmHg)	72.0 ± 5.8	70.0-74.0	
HR _{rest} (bpm)	65.5 ± 7.9	62.7-68.2	
BMI (kg m ⁻²)	22.3±1.9	21.6-22.9	
PSS-14	23.5 ± 7.2	21.1-26.0	

SD = Standard deviation; BP = Blood pressure; BMI = Body mass index; CI = 95% confidence interval.

Table 2 summarizes the HRV parameters analyzed in healthy volunteers at rest, in a supine position, during spontaneous breathing. Registered by the mean LF/HF ratio, sympathovagal balance was lower than 1.0 and suggested a parasympathetic predominance, probably due to the fact that most volunteers in the study were women.

Table 2. Summary of heart rate variability data.

	Mean±SD	Mean CI
TP (ms ²)	3150.8±2517.1	2286.2-4015.5
LF (ms ²)	794.8±579.7	595.7-994.0
HF (ms ²)	1507.9 ± 1783.3	895.3-2120.5
LF (n.u.)	41.08 ± 16.23	35.50-46.66
HF (n.u.)	58.92 ± 16.24	53.34-64.49
LF/HF	0.892 ± 0.801	0.617-1.167
SDNN	54.77±21.17	47.50-62.04
rMSSD	55.87±32.21	44.80-66.93
PNN50	32.46±23.05	24.54-40.38
Triangular index	13025 ± 3946	11680-14370

Total power (TP ms²) = the variance of RR intervals over the temporal segment; LF (ms²) = Power in low frequency range (0.04-0.15 Hz); HF (ms²) = Power in high frequency range (0.15-0.4 Hz); LF (n.u.) = LF power in normalized units; LF/HF = Ratio LF (ms²)/HF (ms²); SDNN = standard deviation of all normal to normal R-R (NN) intervals; rMSSD = Square root of the mean of the squares of successive R-R interval differences; pNN50 = the percentage of intervals differing more than 50 ms different from preceding interval; CI = 95% confidence interval.

Spearman's non-parametric correlation was calculated for HRV parameters, PSS-14 and BMI. Table 3 presents summarized data for Spearman's correlation coefficient and p value.

Table 3. Correlation coefficients (ρ) and p value for Spearman´s non-parametric correlation analysis.

-		PSS-14		BMI	
	ρ	р	ρ	р	
SSDN	-0.207	0.233	0.010	0.954	
rMSSD	-0.243	0.160	-0.030	0.864	
NN50	-0.173	0.320	-0.014	0.937	
PNN50	-0.175	0.314	0.044	0.801	
RRindex	-0.236	0.173	-0.030	0.866	
LF (ms ²)	-0.343	0.044^{*}	-0.062	0.723	
HF (ms ²)	-0.158	0.366	-0.102	0.560	
LF (n.u.)	-0.005	0.976	-0.107	0.539	
HF (n.u.)	0.007	0.969	0.109	0.533	
LF/HF	-0.007	0.969	-0.109	0.533	
ТР	-0.265	0.124	-0.035	0.843	

There was a significant correlation between PSS-14 scores and LF (ms²) ($\rho = -0.343$; p = 0.044). Other HRV variables failed to show any significant correlation even though they had negative Spearman's

coefficient (TP, $\rho = -0.265$; HF, $\rho = -0.158$; SSDN, $\rho = -0.207$; rMSSD, $\rho = -0.243$). LF/HF and LF (n.u.) did not correlate at all with PSS-14 and Spearman's ρ was very close to zero. No correlation was found between HRV parameters and BMI (Table 3). Although LF (ms²) had a normal distribution based on the normality test, the authors thought it was prudent to use a non-parametric test due to the fact that normality could not be assumed for all other HRV parameters (the analysis of data with Pearson's correlation coefficient leads to the same conclusion, r = -0.372, p = 0.028).

Data demonstrate a correlation between perceived stress level and HRV at rest, more precisely a negative correlation of LF (ms²) and PSS-14. Changes in LF (ms²) may be a consequence of both sympathetic and parasympathetic activity what could represent global alterations in HRV or a consequence of sympathovagal balance alterations. In current assay, there were no correlations between perceived stress and indexes of sympathovagal balance, such as in LF/HF ratio, LF (n.u.) and HF (n.u.). Moreover, other variables representing global HRV, such as TP and SSDN, also had negative Spearman's ρ similar to LF (ms²). These data favor the interpretation that psychological stress is correlated with decrease in overall heart rate variability.

Low HRV has been associated to increased cardiovascular mortality in several conditions such as diabetes mellitus and coronary heart disease (GERRITSEN et al., 2001; ROY et al., 2013). Increased psychological stress is also associated with several diseases including heart disease, such as left myocardial ischemia and angina (COHEN et al., 2007).

As previously shown, acute psychological stress is associated with increased sympathetic stimulation (DIKECLIGIL; MUJICA-PARODI, 2010; KUDO et al., 2014). In fact, a chronic sympathetic stimulation is present in some cardiovascular diseases (DICARLO et al., 2013; HERING; NARKIEWICZ, 2013). Previous studies also reported that chronic psychological stress may lead to morphological and physiological changes in neuroregulatory systems (PAPOUSEK et al., 2002). In current study, a negative correlation occurred between stress (assessed by the PSS-14) and LF (ms²). A sympathetic overstimulation that could be expected in chronic cardiac disease would show a high LF (ms²) and a high LF/HF ratio. That was not the case in the population in current assay since volunteers did not present a sympathetic predominance. It was not an unexpected situation. It was a consequence that all the subjects were healthy and young and not submitted to a protocol of acute mental or psychological stress. Contrastingly, based on the results of other HRV parameters (negative

correlation for PSS-14 and HF, TP and SDNN and no changes in HRV balance, i.e. no variation in LF/HF ratio), data were interpreted as an overall decrease in HRV correlated to increase in chronic psychological stress. It should be taken into consideration that this association was present in a range of PSS scores provided by healthy volunteers facing daily life stressors. It could be different if individuals were chronically stressed by experimental conditions. Furthermore, due to sample size it was not possible to analyze gender influence on the data. Although decrease in HRV in young and healthy volunteers may be only associative, it may indicate an important mechanism in cardiovascular disease development not previously reported.

It is possible that impaired (reduced) HRV may be an early consequence of stress induced neural and adaptation that endocrine may contribute throughout life towards the pathophysiology of cardiovascular disease. It is also possible that reduced HRV would precede the progression to sympathetic over activity if chronic stress persists. Further research in the field should be performed, including the assessment of hormonal status which may further clarify the association between reduced HRV and perceived stress and the application of a protocol design to a larger population that allows data sub-analyses by gender and assessment of HRV recovery from acute psychological stress.

Conclusion

Increase in perceived stress in healthy subjects was correlated to decrease in LF (ms²) without correlation to sympathovagal balance, suggesting an association between increase in perceived stress and decreased heart rate variability. The above may point out an important mechanism in cardiovascular pathophysiology that should be further investigated.

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