Heart Rate Variability: the Involvement of Breathing Pattern (chest breathing, abdominal breathing) and Anxiety.

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***Abstract*- The relationship of heart rate variability to thoracic and abdominal components of breathing pattern, as well as to slow paced respiration, was studied in healthy volunteers with different level of anxiety. Subjects with high anxiety showed lower levels of heart rate variability, slow respiration increased the low frequency component of HRV in both groups, chest and abdominal respiration reduced HRV.**

***Keywords*- heart rate variability, breathing pattern, paced respiration**

I. INTRODUCTION.

It's well known that heart rate variability (HRV) is a basic homeostatic parameter and some changes in this parameter, especially its decreasing, can be a marker of possible sudden death at patients with heart diseases [1]. Also, it's known that abdominal respiration has a positive effect on the organism, and pranayama (respiration used in yoga system) is considered as physiologic one with great catabolic effect. There are even some methods of breathing training for increasing HRV.

Otherwise, it's known that elevated anxiety of one person associates with autonomic disorders and psycho-autonomic syndrome, described in last century by germane authors [2] and developed ulterior by F. Vein and his school. This syndrome is considered as basic concept in present science of autonomic disorders.

Interaction between thoracic and abdominal components of the respiratory pattern also exhibits some interest [3]. Experimental studies on healthy people and patients which show the involvement of respiratory pattern and its variants (abdominal respiration, chest respiration, hyperventilation) are very few.

The purpose of this study is to analyze heart rate variability at different forms of breathing pattern and in relation to anxiety level in healthy volunteers.

II. METHODS.

The subjects were selected by screening with State-Trait Anxiety Inventory (STAI) Spielberger. 12 subjects (6 males, 6 females, mean age 20.59±0.43) formed the group with low anxiety (15-30 points), and 13 subjects (6 males, 7 females, mean age 20.49±0.38) formed the group with high anxiety (more than 45 points). ECG and pneumotachogram were recorded using MP35 unit from BIOPAC Systems. The recordings were performed in following conditions: spontaneous breathing (3 min), spontaneous breathing with limited thoracic movements (by belts) i.e. "abdominal" respiration (3 minutes), spontaneous breathing with limited abdominal movements (by belts) i.e. "chest" respiration (3 minutes) and paced breathing (guided by metronome) with breathe rate 6 per minute (3 min). Simultaneously, end-tidal fraction CO_2 was continuously measured by the capnograph MEC-2000 in order to maintain it at constant values.

From ECG, heart period was calculated as interval between two successive R waves, fast Fourier transform applied to calculate spectral power of RR intervals and following values of HRV were calculated: power spectral density (PSD), absolute and normalized high-frequency (HF) spectral power, absolute and normalized low-frequency (LF) spectral power (normalized LF and represent the relative value of each power component in proportion to the total power minus the VLF component) and LF/HF ratio.

Statistical analyses: *t*-Tests were used to assess the statistical significance of differences for HR power and its components in different conditions. Values are means ± SE unless otherwise stated.

III. RESULTS.

Subjects with high anxiety had lower values of PSD, as shown in fig.1, and this difference was described in all 4 tests under different conditions of breathing, as mentioned before. However, inside the group with low anxiety, the PSD didn't show any important difference between periods of recording. The opposite effect is seen in the group with high anxiety, PSD being decreased during abdominal and chest respiration, and increased during 6 per minute paced breathing.

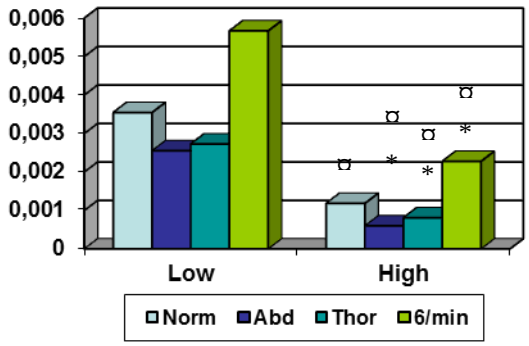

Fig. 1. Spectral power density in subjects with low (in the left) and high (in the right) anxiety during spontaneous, abdominal, thoracic and metronome-guided breathing (* - p<0.05 for the comparison inside the group, ¤ - p<0.05 for the comparison between the groups).

The main reason for low values of PSD in high anxiety group will be probably shorter RR interval (847±13 ms in low anxiety group, 670±11 in high anxiety group), since the increased heart rate leads to smaller fluctuations in RR interval. Also, PSD had decreased during both abdominal and chest respiration, although heart rate didn't change.

LF was slightly elevated in subjects with high anxiety

(41.74±2.3 vs. 18.06±1.28, fig. 2). During abdominal and chest respiration, the values of LF didn't differ neither within the group nor between the groups. During paced respiration, LF had increased in both groups, and this situation reflects not an increased sympathetic discharge, but an increased possibility for the same sympathetic neural outflow to modulate the heart rate.

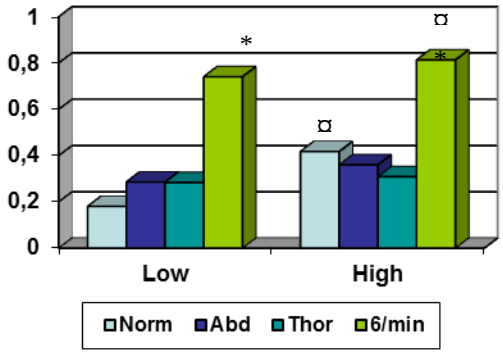

Fig. 2. LF in subjects with low (in the left) and high (in the right) anxiety during spontaneous, abdominal, thoracic and metronome-guided breathing (* - p<0.05 for the comparison inside the group, ¤ - p<0.05 for the comparison between the groups).

HF wasn't significantly different between spontaneous breathing and chest /abdominal breathing, but decreased significantly in paced respiration in both groups (fig. 3). In low frequency respiration (< 9 breaths/min), respiratory frequency and low frequency overlap, and the RR variability is modulated by both parasympathetic and sympathetic nervous system, and both will increase the LF component of the HRV, leaving HF at low levels.

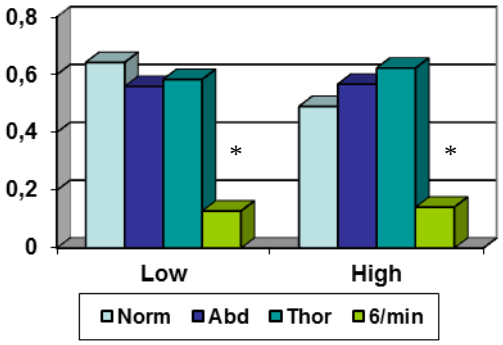

Fig. 3. HF in subjects with low (in the left) and high (in the right) anxiety during spontaneous, abdominal, thoracic and metronome-guided breathing (* - p<0.05 for the comparison inside the group, ¤ - p<0.05 for the comparison between the groups).

LF/HF ratio followed the same changes as LF, was the same during thoracic and abdominal breathing, and markedly increased during slow breathing in both groups (fig. 4). No differences were found in LF/HF ratio between the groups.

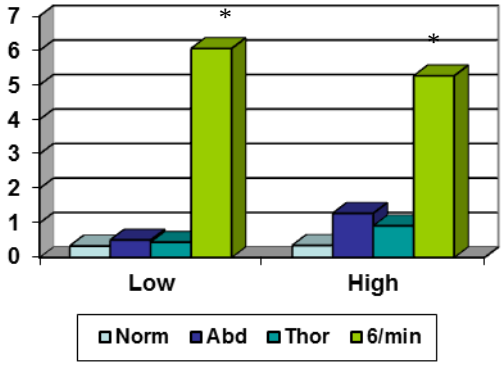

Fig. 4. LF/HF ratio in subjects with low (in the left) and high (in the right) anxiety during spontaneous, abdominal, thoracic and metronome-guided breathing (* - p<0.05 for the comparison inside the group, ¤ - p<0.05 for the comparison between the groups).

IV. CONCLUSIONS.

1. The subjects with anxiety present lower values of power spectral density, due to reduced mean RR interval
2. Slow rate respiration (6/min) increases the LF component of HRV, since LF component in this case is modulated by both sympathetic and vagal activity.

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