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Influence of External Periodic Stimuli on Heart Rate Variability in Healthy Subjects and in Coronary Heart Disease Patients

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Abstract—Frequency estimates of the heart rate variability (HRV) spectrum influenced by external periodic stimuli were studied in healthy subjects and patients with coronary heart disease (CHD). Sensory stimulation by periodic eye opening at a rate of 15, 10, 8, 6, or 5 times per minute, as well as spontaneous and controlled breathing at a rate of 15, 10, 8, 6, or 5 times per minute, was used. It was found that the spectral response to external periodic oscillations was determined by a frequency-dependent phenomenon, the maximal amplitude of heart rate variations being observed in the case of external stimuli at a frequency of 0.1 Hz. A resonance frequency in the 0.1-Hz range may be suggested to exist in the cardiovascular controls. Significant differences in the HRV frequency characteristics between CHD patients and healthy subjects were shown. CHD patients had a characteristic decline in HRV responses to external oscillations; the power of these responses did not depend on the frequency of external stimuli.

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INTRODUCTION

The functional parameters of the cardiovascular system, even under steady-state conditions, undergo various spontaneous fluctuations [1, 2], whose mechanism is very complex and is characterized by the presence of numerous periodic oscillatory processes at different frequencies, which may change under the influence of external factors [3].

Spectral analysis of short (2–5 min) rhythmograms is usually performed in two ranges of the heart rate variability (HRV) spectrum: the high-frequency (0.15–0.4 Hz) and low-frequency (0.04–0.15 Hz) ranges [4–6]. This is consistent with concepts that oscillations in these frequency ranges are of importance for actualizing the functional parameters of the autonomic control of cardiovascular activity and vascular tone [7, 8].

High-frequency variations in the heart rate are determined by its respiratory modulation and are a characteristic of the activity of the parasympathetic division of the autonomic nervous system. Low-frequency heart rate variations are, in the opinion of some authors [5, 8], a consequence of the influence of autonomic oscillations in the central link of the sympathetic division of the autonomic nervous system and are a marker of its activity. HRV values constructed on short rhythmograms are used for the assessment of the autonomic control of the heart rate both under experimental conditions [9, 10] and in clinical practice [10, 11]. The main argument in favor of studying short recordings of the electrocardiogram (ECG) *R–R* intervals is the fact that, during a comparatively short time for a certain position of a subject's body, the external conditions of the work of the cardiovascular system may be considered to be relatively stable.

When studying short *R-R* series and arterial blood pressure fluctuations, it is necessary to take into account that the cardiobaroreceptor system of control may determine a slow blood pressure and heart rate dynamics from several seconds to several minutes [3, 12, 13]. Anatomical and electrophysiological studies of the central mechanisms of the baroreflex have demonstrated a significant role of quick nerve centers and the spinal cord sympathetic nuclei in the formation of heart and respiratory rate variabilities [14]. The input channel of heart rate baroreceptor control is multiple afferent signals from baro- and chemoreceptors, respiratory neurons, and higher nerve centers. It has been established that the concordance of the work of the vasomotor and respiratory centers is characterized by a nonlinear dynamics [15–17]. On the whole, they work as a complex nonlinear time-adjusted system [13]. Thus, the cardiobaroreceptor control of heart rate may be regarded as a system with a complex neuronal network, where spontaneous oscillations in the output signal may be determined by the dynamics in the system itself and by input stimuli [18, 19]. It is evident that, in the mechanisms of formation of nonlinear high-frequency heart rate fluctuations, central connections between two oscillatory systems (the respiratory rate and the baroreflex control) determining the formation of a frequency-dependent phenomenon are of importance [20, 21]. A number of studies on experimental

Table 1. Clinical examination data on 11 patients with CHD

Parameters	
NYHA functional class (number of patients):	
I	5
II	5
III	1
IV	0
Coronary vessel damage (number of patients):	
0 vessels	2
1 vessels	4
2 vessels	4
3 vessels	1
Echocardiographic examination ($M \pm SD$):	
Left ventricular ejection fraction, %	45 ± 7
Left ventricular diastolic diameter, mm	56 ± 9
Left ventricular systolic diameter, mm	45 ± 9
Left ventricular residual fraction, %	23 ± 7

models have shown that low-frequency generation in response to an external signal may cause a resonance phenomenon that will be reflected in the baroreflex mechanism of cardiac activity control [16, 22]. The significance of the resonance frequency detected in the range of 0.1 Hz in the mechanism of baroreflex control has been confirmed in several experimental works [23, 24] in which the frequency responses in the baroreflex system were studied using rhythmic stimulation of the carotid sinus baroreceptors. However, the importance of other input stimuli (disturbances or perturbations) of the system in the formation of its output signal has not been studied yet. In most cases, in healthy subjects, frequency estimates of short recordings of ECG *R–R* intervals were analyzed [21, 23, 25], whereas the use of these records may be effective for determining the adjustments of the heart rate control system and its functional state.

The purpose of this work was to analyze frequency estimates of the HRV spectrum influenced by external periodic stimuli in healthy subjects and in patients with coronary heart disease (CHD).

METHODS

Eleven apparently healthy men (average age, 43 ± 9 years) and 11 male CHD patients (average age, 50 ± 5 years) were the subjects of the study. None of the healthy subjects were diagnosed as having cardiovascular pathology or other diseases. Preparation for the study included abstaining from smoking and drinking coffee for 2 h before the beginning of the experiment. All CHD patients were assessed for the state of the myocardial contractile function and the severity of atherosclerotic damage of the coronary arteries.

The clinical characteristics of the CHD group are shown in Table 1. This group did not include patients with diabetes mellitus associated with CHD. All the patients were given drug therapy, including angiotensin-converting enzyme, nitrates, β-adrenergic blockers, and diuretics. During the examination, all the patients were clinically stable and had been free from acute coronary attacks for one month. Before the inclusion into the study group, normal blood pressure and sinus rhythm were confirmed in all the patients. Coronary pathology was verified by the following instrumental methods: (1) 12-channel electrocardiography (a VSD-804 digital electrocardiograph, Volzhskie Peredovye Tekhnologii, Russia); (2) Doppler echocardiography (Image Point Hx, Hewlett-Packard, United States); (3) a bicycle ergometer test (EC 1200, Hellige, Germany); (4) Holter monitoring (Premier IV, Series 8500, Marquette Electronics Inc., United States); and (5) coronarography (Polydiagnost C, Philips, Netherlands).

All the subjects gave informed written consent to participate in the study.

The ECG was digitized at a frequency of 1000 Hz. The intervals between normal *QRS* complexes were identified automatically. The systolic and diastolic blood pressures (SBP and DBP, respectively) were recorded using a standard sphygmomanometer before and after the experimental stage, as well as after ECG recording was completed.

Healthy subjects and CHD patients were subjected to sensory stimulation by periodically opening their eyes at a rate of 15, 10, 8, 6, or 5 times per minute (0.25, 0.17, 0.13, 0.1, and 0.08 Hz, respectively). Control of the period of eye opening and closing was exercised by electronic metronome commands. Before the beginning of the study, all the subjects had an eye opening and closing training session.

Both groups were also examined under conditions of spontaneous and controlled breathing at a rate of 15, 10, 8, 6, or 5 respiratory movements per minute (0.25, 0.17, 0.13, 0.1, and 0.08 Hz, respectively). In the process, an ECG was recorded; the breathing period was set by an electronic metronome command. The controlled breathing depth and phase ratio corresponded to spontaneous breathing. Before the study, all the subjects were trained to breathe at the metronome command.

A 180-s period of ECG *R–R* series recording was chosen for all stages of the study. A break of 1–2 min was taken between the stages.

The criterion for selecting ECG *R–R* intervals for subsequent analysis was the absence of artifacts and marked noise. The spectral heart rate analysis involved the estimation of the total spectral power values, as well as the spectral power values in the high-frequency (HF, 0.15–0.4 Hz) and low-frequency (LF, 0.04–0.15 Hz) ranges. An *R–R* series was transformed on the basis of an autoregression algorithm; therefore, the HRV spec-

Table 2. Parameters of hemodynamics and the frequency ranges of the HRV spectrum at the baseline and for different eye opening periods in healthy subjects and patients with CHD

Parameters	Baseline	Eye opening frequency (times per minute)				
		15	10	8	6	5
Healthy subjects (11)						
R–R interval, s	0.84 (0.74–0.89)	0.79 (0.76–0.86)	0.83 (0.77–0.88)	0.79 (0.77–0.87)	0.80 (0.77–0.88)	0.81 (0.76–0.88)
SBP, mm Hg	128 (118–130)	130 (116–130)	130 (120–132)	132 (118–134)	128 (118–130)	133 (120–134)
DBP, mm Hg	84 (70–88)	82 (74–87)	82 (72–86)	84 (66–88)	82 (70–84)	86 (68–88)
LF range, ms^2	190 (78–326)	122 (63–305)	202 (82–256)	155 (64–329)	137 (79–343)	154 (72–363)
HF range, ms^2	310 (200–418)	253 (166–404)	306 (173–373)	250 (200–308)	264 (140–353)	279 (180–444)
Total power, ms^2	903 (808–1100)	863 (454–1397)	711 (478–1036)	731 (496–857)	892 (791–1107)	850 (493–1430)
Patients with CHD (11)						
R–R interval, s	0.82 (0.81–0.92)	0.83 (0.80–0.94)	0.81 (0.83–0.95)	0.87 (0.82–0.92)	0.85 (0.81–0.89)	0.87 (0.81–0.92)
SBP, mm Hg	125 (116–132)	128 (117–132)	126 (120–133)	129 (116–129)	127 (120–131)	128 (119–134)
DBP, mm Hg	80 (72–87)	78 (70–83)	83 (68–84)	77 (71–78)	80 (70–84)	82 (68–85)
LF range, ms^2	115 (46–165)	120 (30–190)	124 (38–167)	118 (42–164)	126 (32–155)	106 (42–134)
HF range, ms^2	117* (85–198)	108 (80–170)	113 (74–184)	128 (94–190)	111 (76–212)	120 (88–165)
Total power, ms^2	640 (384–780)	694 (435–880)	530 (320–824)	386 (280–920)	478 (348–830)	450 (356–790)

Note: The data are shown as *Me* (25–75%); * significant difference ($p < 0.05$) from apparently healthy subjects.

trum was obtained with the most significant frequency components of the heart rate (HR) fluctuations being reflected.

The data are shown as the median and first and third quartile values (corresponding to 25 and 75% of data) for the groups. The fit of the data to the normal distribution was tested using the Kolmogorov–Smirnov test. Since it was found that the data did not correspond to the normal distribution, further statistical calculations were made using nonparametric statistics tests, including Friedman analysis and Wilcoxon's paired comparison test. The intergroup statistical differences were assessed with the Kruskal–Wallis ANOVA method. Statistical analysis was performed using the Statistica 5 software package (StatSoft, Tulsa, United States). The admissible type I error was taken to be $p < 0.05$.

RESULTS

The average HRV spectral range power values, as well as the hemodynamic values, in healthy subjects and CHD patients during the periodic eye opening test are shown in Table 2. In this case, statistically significant intergroup differences in the averaged ECG *R–R* interval, arterial blood pressure, and the total and high-frequency spectral power of the HRV were absent. However, the groups differed significantly in the power of the low-frequency range of the HRV spectrum ($p < 0.05$); in CHD patients, the spectral power in this range was substantially lower.

A change in the eye opening period did not influence the average *R–R* interval values or the SBP and DBP levels in either group.

In healthy subjects, a change in the periodicity of blinking caused a significant increase in the HRV spectral power at the frequency of the input disturbance (Fig. 1). The value of this spectral power increase depended on the eye opening rate ($F = 26.8$, $p < 0.001$). In most healthy subjects (7 out of 11 examined), rhythmic opening of the eyes eight or six times per minute caused a peak increase in the HRV spectral power at the corresponding frequencies. In this case, the external

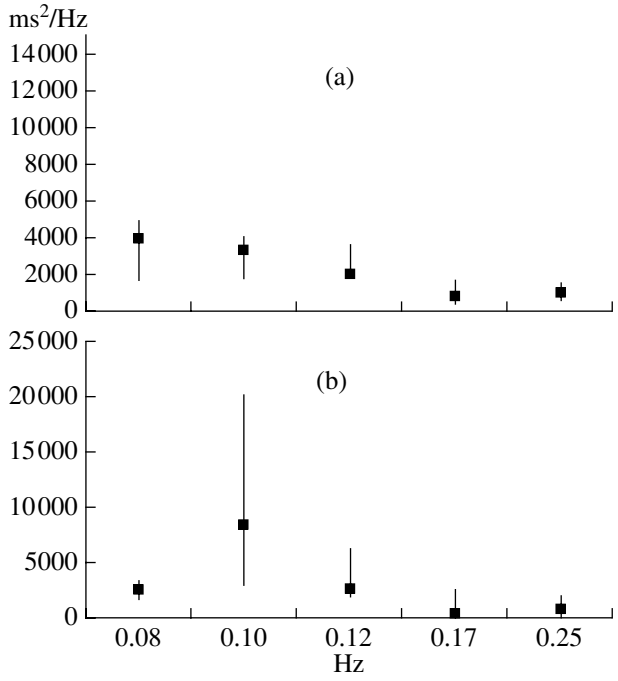

Fig. 1. Power spectral density of the frequency components of the HRV spectrum in healthy subjects (a) at a spontaneous eye opening frequency and (b) at controlled eye opening frequencies. The data are shown as *Me* (25%; 75%). Abscissa: the frequency components of the HRV spectrum (Hz). Ordinate: the values of the power spectral density of these components (ms^2/Hz).

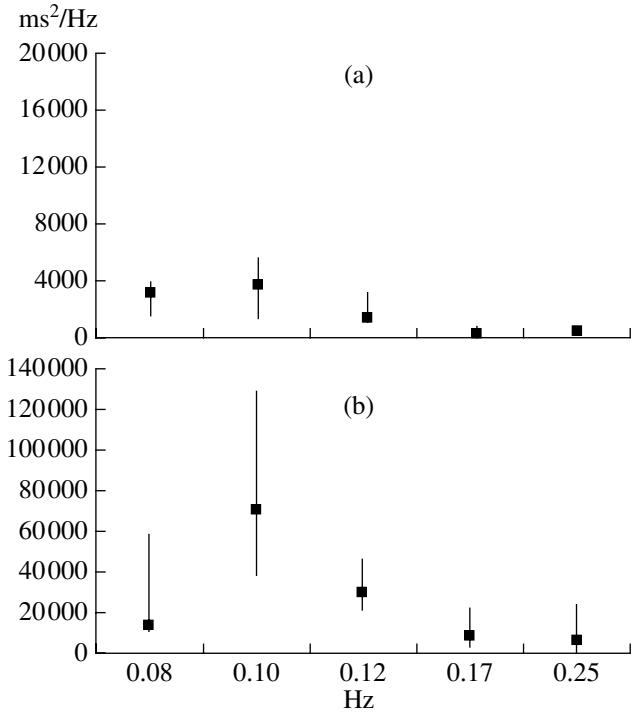

Fig. 2. Power spectral density of the frequency components of the HRV spectrum in healthy subjects (a) under the spontaneous breathing conditions and (b) at controlled breathing frequencies. See Fig. 1 for designations.

sensory stimulation at a rate of six times per minute caused the most marked response of the HRV spectrum (Fig. 1). In 4 out of 11 healthy subjects, opening of the eyes at 0.1 Hz (six times per minute) did not cause a response of the HRV spectrum at this frequency.

In CHD patients, periodic blinking did not cause changes in the activity of heart rate fluctuations.

Table 3 shows the average hemodynamic parameters and frequency ranges of the HRV spectrum in healthy subjects and patients with CHD during spontaneous and controlled breathing at a rate of 15, 10, 8, 6, and 5 respiratory movements per minute. No statistically significant intergroup differences were found in the averaged ECG *R–R* interval, arterial blood pressure, or total or HF HRV spectral power. However, statistically significant differences in the LF-range power of the HRV spectrum between healthy subjects and CHD patients were observed, the lower values being observed in those afflicted with cardiovascular pathology ($p < 0.05$).

It should be noted that in both groups a change in the period of controlled breathing was not associated with a shift in the level of the average ECG *R–R* interval or arterial blood pressure values.

In healthy subjects, controlled breathing at a rate of 15–5 inspirations per minute (0.25–0.08 Hz) caused the appearance of heart rate fluctuations at the respiratory rate, which could be detected by means of the spectral analysis of the ECG *R–R* series (Fig. 2). The respiratory rate determines the most pronounced modulating effect on the HRV of subjects without cardiovascular pathology ($F = 21.6$, $p < 0.001$). In this case, the maximal changes in the HRV spectrum were observed at a controlled breathing rate of six respiratory movements per minute (0.1 Hz) ($p < 0.05$) (Fig. 2). The results of the spectral analysis of the ECG *R–R* series showed a spectral power peak at 0.1 Hz and its harmonic (Fig. 2). Controlled breathing at a frequency different from 0.1 Hz also caused the appearance of heart rate fluctuations with a preset frequency, but the power of these spectral responses was considerably less ($p < 0.01$) compared to the peak with a breathing frequency of 0.1 Hz (Fig. 2).

The respiratory rate in healthy subjects influenced significantly the total HRV spectral power ($F = 25.2$, $p < 0.001$) and the LF ($F = 43.2$, $p < 0.001$) and HF ($F = 10.1$, $p < 0.05$) ranges (Table 3). It should be pointed out that breathing at a high rate (15 and 10 breaths per minute) was characterized by fewer specific changes in the total power of the HRV spectrum than controlled breathing at a low rate (8, 6, or 5 breaths per minute) ($p < 0.001$) relative to spontaneous breathing. The total frequency range power value was about the same in three periods of slow breathing.

Table 3. Parameters of hemodynamics and the frequency ranges of the HRV spectrum in spontaneous and controlled breathing in healthy subjects and patients with CHD

Parameters	Baseline	Eye opening frequency (times per minute)				
		15	10	8	6	5
Healthy subjects (11)						
R–R interval, s	0.83 (0.77–0.88)	0.82 (0.72–0.90)	0.80 (0.75–0.93)	0.80 (0.77–0.93)	0.81 (0.74–0.95)	0.81 (0.75–0.92)
SBP, mm Hg	130 (116–132)	130 (120–130)	127 (120–132)	129 (118–132)	128 (121–129)	131 (119–133)
DBP, mm Hg	80 (68–84)	80 (72–83)	77 (70–84)	81 (69–82)	82 (73–85)	83 (68–85)
LF range, ms^2	202 (82–265)	251 (127–1451)	340* (230–1109)	215 (143–682)	260* (152–649)	346* (209–921)
HF range, ms^2	306 (173–373)	237 (99–348)	263 (158–345)	920* (483–1520)	1307*# (820–2320)	537* (380–1416)
Total power, ms^2	892 (791–1107)	1052 (516–1584)	1290 (716–1917)	1760* (862–3035)	2240* (1294–3419)	1463* (1006–3832)
Patients with CHD (11)						
R–R interval, s	0.85 (0.82–0.94)	0.86 (0.83–0.94)	0.87 (0.85–0.90)	0.86 (0.81–0.91)	0.87 (0.80–0.90)	0.89 (0.83–0.93)
SBP, mm Hg	128 (120–130)	125 (119–130)	122 (121–133)	129 (118–129)	120 (116–130)	123 (120–132)
DBP, mm Hg	78 (74–81)	80 (68–80)	75 (70–83)	77 (66–80)	81 (72–82)	80 (71–86)
LF range, ms^2	109 (33–175)	138+ (44–218)	117+ (22–147)	56+ (21–152)	82+ (29–203)	134+ (49–314)
HF range, ms^2	110+ (70–188)	91+ (62–159)	91+ (64–97)	113+ (88–363)	172+ (102–385)	261+ (93–389)
Total power, ms^2	623 (402–722)	686+ (449–834)	421+ (251–712)	358+ (215–644)	460+ (368–1135)	932+ (418–1355)

Note: The data are shown as *Me* (25–75%); * significant difference ($p < 0.05$) from the case of spontaneous breathing; # significant difference ($p < 0.05$) from other controlled breathing periods; + significant difference ($p < 0.05$) from apparently healthy subjects.

The strongest statistically significant influences were observed in the LF HRV spectrum range during controlled breathing at a rate of six respiratory movements per minute ($p < 0.05$). Spontaneous breathing and controlled breathing at rates of 10, 6, and 5 respiratory movements per minute also influenced the power of the HF component of the HRV spectrum ($p < 0.05$). The strength of the influences did not depend on the period of controlled breathing (Table 3).

In CHD, controlled breathing caused heart rate fluctuations at a preset frequency; however, the breathing period did not influence significantly the value of HRV variations (Fig. 3). As shown in Table 3, neither the total HRV spectral power nor the LF and HF ranges exhibited significant differences during different periods of controlled breathing.

Comparison between CHD patients and healthy subjects showed intergroup differences in HRV. For example, in all the breathing periods, more marked responses in the HRV spectrum were observed in healthy subjects than in CHD patients ($p < 0.05$) (Figs. 2b, 3b). The total power and the LF and HF ranges of the HRV spectrum were also lower in patients with cardiovascular pathology ($p < 0.05$) than in apparently healthy subjects at all the respiratory rates (Table 3).

DISCUSSION

It follows from the above data that an input signal in the form of periodic sensory disturbances in the control system influences the mechanism of HRV formation during short-term recording of ECG *R–R* intervals. This indicates that, in addition to cardiobaroreceptor control in the central link of the heart rate control, there are mechanisms processing incoming information from other centers, in particular, from the thalamus and the cerebral cortex. In healthy subjects, the most significant power peak of the HRV spectrum in the case of this type of external stimuli was observed at frequencies of input stimulation of 0.1 and 0.12 Hz (six and eight times per

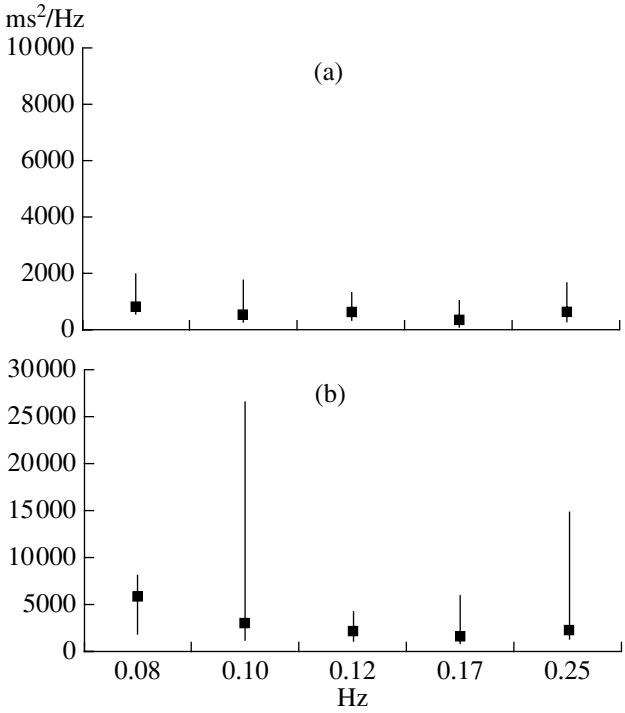

Fig. 3. Power spectral density of the frequency components of the HRV spectrum in CHD patients (a) under the spontaneous breathing conditions and (b) at controlled breathing frequencies. See Fig. 1 for designations.

minute, respectively). The maximum response corresponded to an external periodicity frequency of 0.1 Hz. CHD patients showed no changes in the power of the HRV spectral ranges in response to external disturbances with a different rate in the form of periodic eye opening.

It was also found that the spectral power in the LF range of the HRV spectrum in controlled breathing at a rate of six respiratory movements per minute (0.1 Hz) was characterized by the highest absolute value. Breathing in the LF range also determined a significant increase in the total power of the HRV spectrum, whereas the HF spectral range did not have significant differences under the conditions of the five controlled breathing frequencies used. Note that the averaged *R–R* intervals and arterial blood pressure were relatively constant. The results obtained agree with the data of other investigations [21, 26–28] in which it was shown that the most frequent HRV response was noted in controlled breathing at 0.1 Hz. One of the possible mechanisms of the origin of these oscillations is central interaction between two self-sustained oscillatory systems, such as nonlinear oscillations in the cardiobaroreceptor mechanism of control and breathing when the phenomenon of frequency coincidence is manifest: the respiratory rate coincides with the internal frequency of the baroreflex mechanism in the 0.1-Hz range, which causes the interaction between them to be significantly increased [29]. In a number of experimental works, an important role of baroreflex control in the interaction between the respiratory and heart rates and variation in arterial blood pressure has been suggested. For example, it was shown in [26] that the influence of the respiratory rate on the interaction between the heart rate and arterial blood pressure fluctuations is determined by a frequency-dependent phenomenon and does not depend directly on the activity of the sympathetic division of the autonomic nervous system. The respiration-dependent fluctuations in systolic blood pressure are more marked than those in the ECG *R–R* intervals, which testifies to the baroreflex mechanism.

Our study also demonstrated the importance of a frequency-dependent phenomenon in the formation of HRV in short sections of *R–R* interval recordings with external periodic disturbances. Moreover, the analysis of the frequency responses shows that external 0.1-Hz disturbances exert the most significant influence on the HRV formation. The data obtained may be considered an expression of the effect of a resonance response in the 0.1-Hz range in the quick mechanism of cardiovascular control influenced by low-frequency rhythmic breathing. The comparison of the data of experimental studies allowed us to put forward a hypothesis that a resonance frequency in the 0.1-Hz range exists in the system of cardiovascular control. A similar hypothesis was formulated in [23] and [24], where the baroreflex control was disturbed by rhythmically influencing the aortal nerve and the carotid sinuses. It was also established that the amplitude of the heart rate and blood pressure fluctuations significantly increased upon stimulation at 0.1 Hz in humans and 0.4 Hz in rats.

The above studies [23, 24] and the results of our works [30] do not agree with the existing concept that low-frequency heart rate fluctuations are determined by the activity of the sympathetic division of the autonomic nervous system [5, 8]. For example, in this study, a change in the amplitude of the 0.1-Hz heart rate oscillations was not correlated with the dynamics of the level of sympathetic activity. On the contrary, neither controlled breathing nor periodic eye opening at 0.1 Hz were accompanied by changes in the average HR or blood pressure, which evidences the constancy of the activity of the sympathetic division of the autonomic nervous system. However, similar external disturbances caused a significant increase in the peak power in the 0.1-Hz range in the HRV spectrum. This observation indicates a discrepancy between the dynamics of the power of the LF range of the HRV spectrum and the level of sympathetic influences on the heart rate. It was also shown that blocking β-adrenergic receptors does not influence the power of the LF range of the HRV spectrum [31, 32] or the amplitude of low-frequency HR fluctuations [33]. In addition, a decrease in the low HRV frequencies was observed during ischemic exercise in animals, despite the sympathetic heart rate stimulation [34]. However, many experimental studies have also demonstrated that sympathetic activity significantly influences low-frequency heart rate variations

[5, 8, 35]. Therefore, their nature is still not completely clear.

Our studies on the influence of controlled breathing on HRV in healthy subjects are consistent with and supplement results obtained by other researchers [20, 21, 36]. However, we were the first to study the influence of periodic sensory stimulation in the form of eye opening and controlled breathing in patients with CHD: we showed that the frequency responses in short sections of HRV recordings of CHD patients differed from those of healthy subjects. For example, eye opening six or eight times per minute was accompanied by a peak increase in the HRV spectral power in the range of these frequencies in healthy subjects but did not change the spectrum in CHD patients. However, controlled breathing caused a spectral power increase at respiratory rates of six and eight times per minute in both healthy subjects and CHD patients. However, the amplitude of respiratory fluctuations in the heart rate of CHD patients was considerably lower than in healthy subjects. Moreover, controlled breathing at a rate of six times per minute caused a peak increase in the spectral power in the 0.1-Hz range, which was not observed in CHD.

Interesting data that agree with our results were obtained in a study of type 1 diabetes mellitus patients [37]. The authors showed that, in diabetes, the amplitude of the ECG *R–R* interval oscillations in controlled breathing at 0.1 Hz was strongly correlated with the baroreflex activity in both groups studied.

It is possible that a complex interaction between respiration, the baroreflex mechanism of control, and the cardiovascular system may underlie the phenomenon discovered. In addition, there is an opinion that controlled breathing is associated with markers of injury in the baroreflex control of the cardiovascular system [37–39]. Interestingly, the effect of the respiratory rate on the baroreflex activity appears to be the functional state of the cardiovascular system, respiration at a rate of 0.15 Hz influencing the baroreflex in healthy subjects but not in CHD patients [39].

It was shown in studies using artificial models and in experimental studies [20, 24] of cardiovascular control that the magnitude of the response and its frequency in HRV were determined by the time delay in the baroreflex feedback loop, the feedback delay being more pronounced in CHD patients than in healthy subjects [40, 41]. These distinctions could form the basis for developing a method of noninvasive assessment of the degree of damage to the cardiovascular system [42, 43]. Since the data presented allow us to speak about differences in the state of the system of cardiovascular function control in CHD patients and healthy subjects, this explains the distinctions in frequency response characteristics between these groups in response to external disturbances. Additionally, it has been shown that CHD is associated with signs of baroreflex decline and HRV changes [44, 45].

On the basis of the results of the studies mentioned above and our own data, we may suggest that the initial power of the low-frequency component of the HRV spectrum and the decreased frequency response to respiration at 0.1 Hz in CHD patients are determined by an impairment of the baroreflex mechanism, which may improve the prediction of the stability of the state of CHD patients.

It must be noted that, in our study, spontaneous respiratory disturbances during the tests with periodic eye opening were not considered; therefore, we cannot assert that the eye opening procedure did not exert an effect on spontaneous respiration. The influence of spontaneous respiration on the power of the HRV spectral range was not assessed either. Moreover, we did not record the baroreflex activity under the spontaneous and controlled breathing conditions, nor was the degree of baroreflex dysfunction in CHD patients assessed.

CONCLUSIONS

Spectral analysis based on an autoregressive model shows that, when short rhythmograms are examined, the spectral response to external periodic oscillations is determined by a frequency-dependent phenomenon. In this case, the maximal amplitude of heart rate fluctuations is observed with external 0.1-Hz disturbances. These results lead us to suggest the existence of a resonance frequency in the 0.1-Hz range in the cardiovascular control that shows up in the low-frequency heart rate variations.

There are significant differences in the HRV frequency characteristics between CHD patients and healthy subjects. CHD patients typically exhibit decreased HRV frequency responses to external oscillations; the power of these responses also does not depend on the frequency of external disturbances.

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