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Effect of Voluntary EEG α Power Increase Training on Heart Rate Variability

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Abstract—The following objectives were set out to study the effect of EEG α power increase training on the heart rate variability (HRV) as an index of the autonomic regulation of cognitive functions: (1) to establish the interrelation between a voluntary increase in the α power in the individual upper α band and the HRV and related characteristics of cognitive and emotional spheres; (2) to determine the nature of the relationship between the α-activity indices and HRV depending on the resting α-frequency EEG pattern; and (3) to study how the individual α-frequency EEG pattern is reflected in the HRV changes as a result of biofeedback training. Psychometric indices of cognitive performance and the characteristics of EEG α activity and HRV were recorded in 27 healthy men 18–34 years of age before, during, and after ten training sessions of a voluntary increase in α power in the individual upper α band with the eyes closed. To determine the biofeedback effect in the α power increase training, the data of two groups were compared: the experimental, with a real biofeedback (14 subjects), and the control, with a sham biofeedback (13 subjects). The follow-up effect of the training was assessed one month after its end.

The results showed that α biofeedback training increased the resting α frequency, improved cognitive performance, reduced psychoemotional stress, and increased HRV only in the subjects with a low baseline α frequency. In the subjects with a high baseline resting α frequency, the α biofeedback training had no effect on the resting α power and cognitive performance but reduced the HRV (judging by the pNN_{50} parameter). The positive correlation between the α peak frequency and HRV in subjects with initially low α frequency and the negative correlation in the subjects with a high baseline α frequency explains the opposite biofeedback effects on HRV in subjects with low and high α frequency.

From the theoretical standpoint, the results of this study contribute to understanding the mechanisms of heart–brain neurovisceral relationships and their effect on the cognitive performance. From the applied standpoint, they suggest that EEG biofeedback can be used for improving autonomic regulation in healthy subjects and the development of individual approaches to the development of the biofeedback technology, which can be used both in clinical practice for treatment and rehabilitation of psychosomatic syndromes and in educational training.

Keywords: HRV, EEG α activity, cognitive functions, biofeedback, sham biofeedback, effect of training

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INTRODUCTION

Studies of corticovisceral relationships in the regulation of the cognitive and emotional sphere have been relevant for more than 100 years [1]. At the beginning of the last century, I.P. Pavlov and his students proved that the cerebral cortex can affect the functioning of all autonomic organs and systems (circulatory, respiratory, etc.) and that the activity of visceral systems can change the functions of the cerebral cortex, including consciousness [2]. Fifty years ago, B. Hess assumed that the autonomic nervous system is not fully autonomous and automatic and includes both peripheral and central neurons [3]. After the discovery of the involvement of the central components in the regulation of autonomic functions [4], B. Hess assumed that the development of the technology of the simultaneous monitoring of the central and peripheral systems may help to identify the markers of integrity of corticovisceral processes in the regulation of autonomic functions. However, so far the search for adequate neurovisceral markers of cognitive performance continues. The development of information technologies of acquisition and processing of electrocardiographic (ECG) and electroencephalographic (EGG) signals showed that the most appropriate central and autonomic predictors of the mechanisms of psychosomatic coupling in cognitive human activity are EEG α activity and heart rate variability (HRV) [5, 6].

It was found that HRV, assessed by the ratio of low-frequency (LF) to high-frequency (HF) components of the interpeak distance range, increases during the performance of cognitive tasks due to both a decrease in the *HF* component [7–9] and an increase in the proportion of the *LF* component [7, 10–12]. According to Lin Tao et al., the greatest success in activities such as musical performance, which requires a high degree of self-control, is achieved by the musicians with the highest HRV values [13]. It is also believed that HRV increases after switching on the cognitive mechanisms of self-regulation during adaptation, for example, to normobaric hypoxia [14], but decreases in such states as attention deficit [15], the use of anabolic steroids by athletes [16], and in depression [17, 18]. It is important to note that the vector of changes in HRV depends on the emotional assessment of the cognitive activities performed [19] and the presence or absence of a feedback on the results [12]. Taking information on the practical use of HRV indices in clinical practice [20] and the data presented in the review by Reynard [21] as a basis, we can conclude that HRV may serve as an indicator of autonomic self-regulation of cognitive and emotional processes.

Other authors established the key role of α activity of the brain in the central regulation of cognitive functions [22, 23]. To date, a clear correspondence was found between the α-wave power in the individual upper α band and the efficiency of solving tasks requiring internal control of information processing [24], the use of short-term memory [25], or musical performance [26]; between the α spindle length and successful solution of semantic tasks [27] and creativity [28]; and between the individual α peak frequency in eyes closed resting condition and cognitive efficiency [29]. In other words, the activity of waves in the individual upper α band is a universal sign of an improvement in the regulation of cognitive processes by the central nervous system [22, 23]. However, the role of α activity in the regulation of autonomic systems is insufficiently studied, and the reported data are contradictory [30, 31]. It was shown that, as a result of transcendental meditation, the very-low-frequency component of HRV increases and α waves become more coherent within and between the hemispheres in a standard range of 8–12 Hz [30]. It was also shown that acupuncture cause first a power increase in the low-frequency α-band (8–10 Hz), simultaneously with an increase in the LF/HF ratio and then an increase in the power of the HF component of the HRV range [32]. Intermittent light simulation was shown to increase the frequency of α peak simultaneously with an increase in HRV [33]. Ryu and Myung showed that the depth of desynchronization of α waves and HRV increase as the cognitive load grows [31]. The authors of several studies showed that a more effective self-control of cognitive and psychomotor functions is associated with higher HRV values and a lower α-rhythm power in the range of 8–12 Hz [7, 11, 34]. Since the α-rhythm power in these studies was calculated with the eyes open, it can be concluded that the increase in HRV, observed simultaneously with the decrease in the α power, indicates enhancement of activation. However, the studies of psychophysiologists of the University of Rome, on the contrary, showed that the increase in HRV during a pleasant viewing of TV advertising was accompanied by a simultaneous increase in the α power in the low-frequency range, indicating a decline of activation [35].

The above results, despite the apparent discrepancy of the samples tested and the conditions of recording and analysis of EEG and ECG signals, agree in that they assume an association of HRV with different characteristics of α activity: frequency, power and width of the α band, and degree of desynchronization. Possible reasons for restrictive interpretation of these results could be, firstly, the lack of standards for measuring α activity reached on the basis of the empirical evidence of the functional role of these EEG waves rather than some theoretical assumptions or agreements [36]. (Just a few years ago α activity began to be assessed not only by the amplitude in a certain standard range, but also by the individual maximum α-peak frequency [22], the degree of the decrease of the α amplitude [37], and the width of the α band and characteristics of the α spindle [23]). Secondly, the discrepancy of the presented results may be due to the fact that the experimental conditions of the study (at rest or in cognitive loads, with the eyes open or closed) were not taken into account.

To study the relationship between the indices of α activity and HRV, it is reasonable to use the biofeedback technology, which makes it possible to arbitrarily modify a particular physiological parameter and then study the effect of this change on other psychophysiological characteristics. We have found only several papers describing the effect of training of a voluntary α activity increase on the parameters of autonomic regulation. For example, it was shown that, in patients with generalized anxiety disorder, 8–12 sessions of biofeedback training aimed at increasing the α-wave power led to an increase in cardio intervals and a decrease in the heart rate reactivity in response to stress [38, 39]. This effect was accompanied by a decrease in anxiety [38]. The biofeedback aimed at reducing the α power did not alter the heart rate's reactivity to the stressor [38]. Other early studies of the α-stimulating biofeedback showed no effect of the α-power increase training on the duration of cardio intervals [40, 41]. It should be also be mentioned that the authors of [42] showed that a single session of a biofeedback training aimed at increasing the α-rhythm power in the standard range of 8–12 Hz in healthy subjects resulted in an increase in HRV in terms of the standard deviation of the duration of cardio intervals [42].

Thus, on the basis of the papers cited above, it is still not possible to draw conclusions about the impact of changes in the α activity on HRV due to methodological ambiguities of measuring α waves of the activity, lack of adequate control, and individual α biofeedback protocols.

In our previous study of the effect of training of voluntary α power increase on the conscious and spirit, we developed a design of the experiment that restricted all the above-mentioned limitations [43]. It was found that the training of voluntary α-power increase in nine of fourteen subjects who used biofeedback and in five of thirteen subjects who increased α power using conventional techniques of self-control without biofeedback proved to be successful. These subjects were termed responders, and those who failed to increase the power of the range of interest were termed nonresponders. It was found that only the responders of the biofeedback training, who had a lower baseline level of α frequency, improved the indices of the performance of cognitive tasks after ten training sessions. However, in the nonresponders of this group, who had a higher baseline level of α frequency and, therefore, initially showed better performance of cognitive tasks, the level of α activity did not increase and the indices of cognitive functions did not improve. In the control group, neither responders nor nonresponders improved cognitive performance. An important result of this study was the finding that the ability of subjects to respond or not respond to the biofeedback training depends on the endophenotypic trait of the individual's resting α-peak frequency.

The purpose of this study was to investigate the effect of the training of a voluntary increase in EEG α power on HRV as an index of autonomic regulation of cognitive functions. To do this, the following objectives were set out: (1) to establish the biofeedback between a voluntary increase in the α power in the individual upper α band and the HRV and related characteristics of cognitive and emotional spheres; (2) to determine the nature of the relationship between the α-activity indices and HRV depending on the resting α-frequency EEG pattern; and (3) to study how the individual α-frequency EEG pattern is reflected in the HRV changes as a result of biofeedback training.

We believe that the results of this study will contribute to the theoretical understanding of the mechanisms of the heart–brain neurovisceral relationships and their effect on cognitive performance. From an applied perspective, this study will provide justification for using the EEG biofeedback to improve the processes of autonomic regulation in healthy subjects and develop individual approaches to the implementation of the biofeedback technology that can be used both in clinical practice for the treatment and rehabilitation of psychosomatic syndromes and in educational training.

MATERIALS AND METHODS

Twenty-seven healthy male subjects aged 18 to 32 years participated in this study on a voluntary paid basis. All participants were divided into two groups: the experimental group (14 subjects) was trained to increase α power using a biofeedback, and the control group (13 subjects) was trained to increase α power using only instructions or the so-called false biofeedback (explained below). The groups were balanced for age (in the experimental group, $MS = 19.5$, $SD = 0.4$; in the control group, $MS = 19.8$, $SD = 0.6$) and the occupational sector (university students, programmers, and security officers). To rule out cases of mental disease, drug use, and the presence of a family history of epilepsy, all subjects completed a specially designed questionnaire [44]. In addition, all subjects signed an informed consent letter, in which they agreed to participate in the study. After the experiment, those participants who passed all 11 training sessions received monetary compensation. The plans and procedure of the experiment were approved by the ethics committee at the Academic Council of the Institute of Molecular Biology and Biophysics, Siberian Branch, Russian Academy of Medical Sciences.

First of all, all subjects were subjected to psychometric and electrophysiological testing and one test biofeedback session. This session was necessary, first, to familiarize all participants with the methods used to increase α activity and, second, to determine the educational quotient.

The subjects of the experimental and control groups received eight true and sham biofeedback sessions, respectively. After this, they again were subjected to psychometric and electrophysiological testing and a test biofeedback session. One month later, this testing was repeated to assess the follow-up effect of biofeedback (Fig. 1 in [43]). The experimental scheme implied the analysis of the influence of the TIME factor (three levels: before, after, and one month after training), the BIOFEEDBACK factor (two levels: true vs sham), and the RESPONSE factor (two levels: responders vs nonresponders) and the effect of their interaction on the indices of cognitive function, psychoemotional stress, and characteristics of α activity and HRV. The introduction of the RESPONSE factor was determined by the fact that, in the previous study, in analyzing the response to the α-power increase training, we distinguished four subgroups of subjects: (1) responders of the experimental group, who used biofeedback to increase α power (RB), (2) nonresponders of the experimental group (NB), (3) responders of the control group, who did not use biofeedback to increase α power (RC), and (4) nonresponders of the control group (NC).

Psychometric characteristics of those types of cognitive functions that are sensitive to changes in the level of α activity were measured before, after, and one month after the end of training. They included the mental rotation task [45], the conceptual span task [27], and the creativity test [28]. The level of situational anxiety was measured using the Spielberger–Hanin test.

The electrophysiological testing included simultaneous recording of electroencephalographic (EEG),

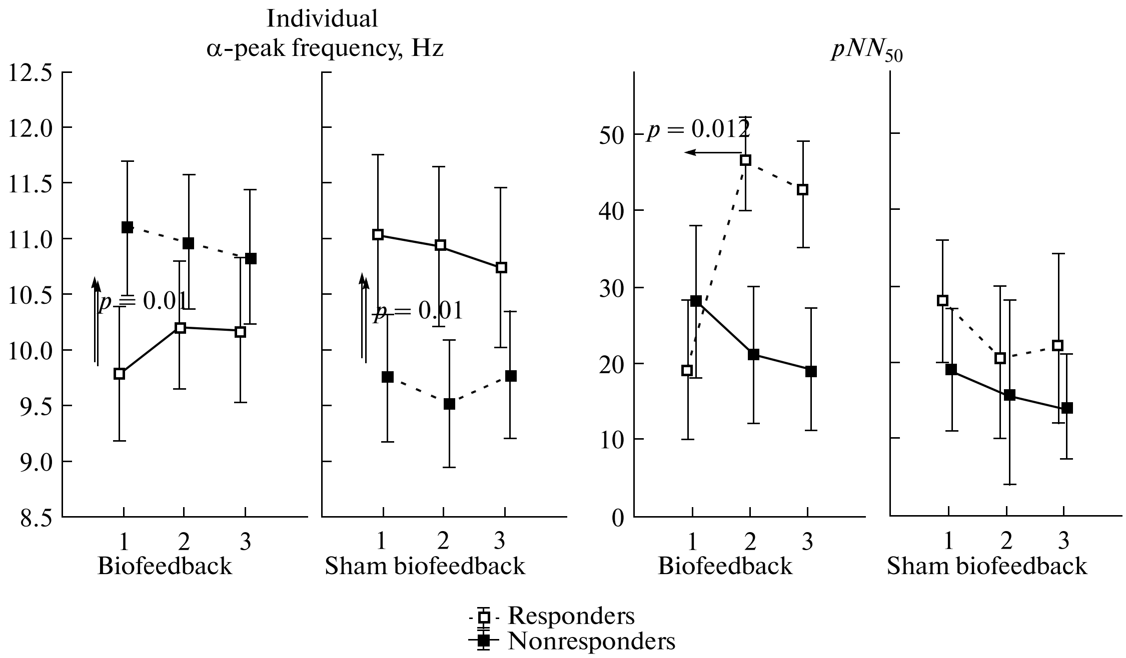

Fig. 1. Individual α-peak frequency (Hz) and heart rate variability (HRV) pNN_{50} (1) before, (2) immediately after, and (3) one month after the α-power increase training with and without the use of biofeedback (true and sham biofeedback, respectively) in the responders and nonresponders (subjects who responded and did not respond to the training, respectively).

electromyographic (EMG), and electrocardiographic (ECG) signals. EEG was recorded by monopolar arrangement, with the active electrode placed on the *Pz* site of the scalp and the reference electrode placed on the right ear. The *Pz* site was selected because the characteristics of α activity in the parietal-occipital region are the least variable and most stable in repeated measurements [46]. The ground electrode and active biopolar EMG electrodes were placed on the skin of the forehead. ECG was recorded by the conventional Einthoven's triangle [47]. EEG, EMG, and EKG signals were amplified and converted to a digital number with a frequency of 720 Hz by using the BOSLAB equipment and software (Komsib, Novosibirsk). Electrophysiological parameters were recorded before and after each training session with the eyes closed (5 min) and open (30 s) at rest and then during solving the arithmetic countdown task (E. Kraepelin test).

The frequency of maximum spectral peak, the width of an individual α band, and the degree of desynchronization were calculated by comparing EEG spectra recorded as described in [28] with the eyes open and closed. The boundaries of the band that was the target of the training were the frequency of the maximum peak (lower limit) + 2 Hz (upper limit). For example, if the frequency of the maximum peak was 10.2 Hz, the range extended from 10.2 to 12.2 Hz and was termed the upper α band.

The tonic tension of the forehead muscles was assessed using the integrated EMG power index, which was calculated as described by Merletti [48].

The interval between adjacent *R*-wave peaks (R–R interval) on ECG was taken as an interpeak distance (ms). The heart rate variability was assessed using the characteristics of both spectral analysis (low-frequency (LF, 0.04–0.15 Hz) and high-frequency (HF, 0.15–0.4 Hz) components and their ratio LF/HF) and the statistical temporal analysis (pNN_{50}, pNN_{10}, and *RSNN*) of cardio intervals according to the international classification [49].

After the psychometric and electrophysiological tests were completed, all subjects took a trial training session, which consisted in a simultaneous increase in the power in the upper α band and decrease in the integrated EMG power of the forehead muscles. The problem of increasing the EEG α amplitude using a biofeedback was solved simultaneously with the control of the EMG amplitude decrease to prevent the quenching of the EMG by artifact signals and reduce emotional stress [50]. Before each session, all subjects were given the same instructions on how the power in the upper α band can be arbitrarily increased to achieve the alpha state. Since the emergence of the alpha state feeling is associated with a complex of interdependent factors [51], all subjects were recommended for all sessions (both true and sham biofeedback) to use the behavioral strategies that are known to effectively increase the upper α band power. All subjects were familiarized with all the strategies and could

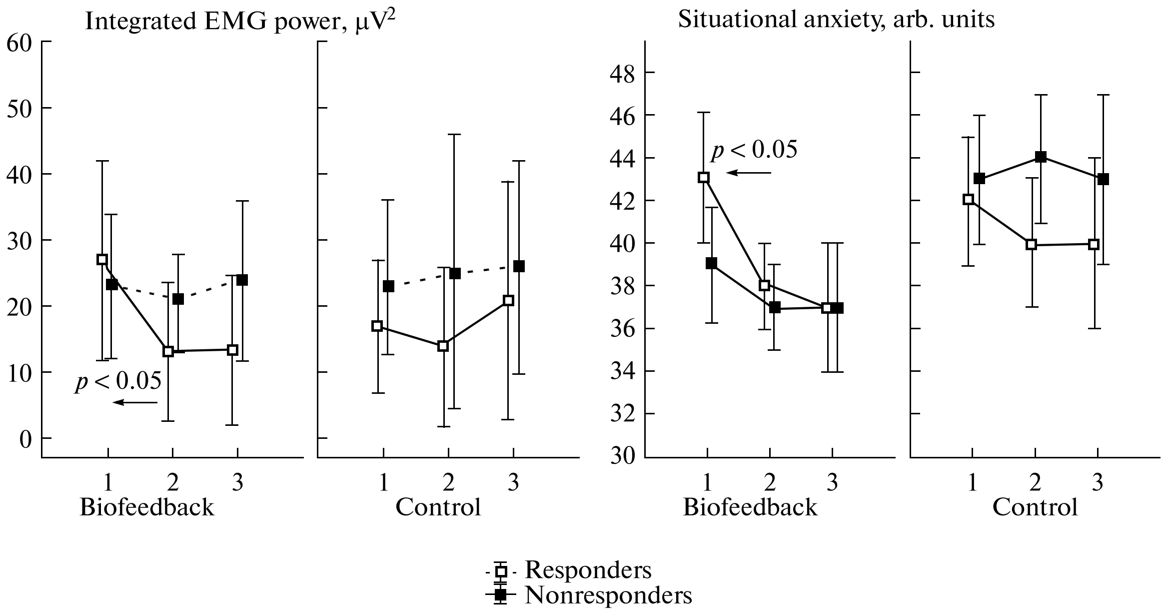

Fig. 2. Integrated electromyogram (EMG) power and situational anxiety indices (1) before, (2) after ten sessions, and (3) one month after the α-power increase training with and without the use of biofeedback (true and sham biofeedback, respectively) in the responders and nonresponders (subjects who responded and did not respond to the training, respectively).

evaluate their effectiveness during the trial biofeedback session.

In the biofeedback procedure, the EEG signal obtained from the *Pz* sensor, as mentioned above, was amplified, digitized at a rate of 720 times per second, and transformed by Fourier series to a spectrum. Then, every 100 ms, the power in the individual high α band was calculated, and this value was transformed on-line to a curve of the red color, the dynamics of which corresponded to changes in α power (hereinafter, the term "α power" will be used instead of the term "the power in the upper α band"). Similarly, the raw EMG signal was transformed into a curve of the green color, corresponding to the dynamics of the integrated EMG power, which indicated changes in the tonic tension in the forehead muscles. The mean values of α and EMG power at rest with the eyes closed were displayed on the monitor in the form of straight lines (thresholds). When the curve corresponding to the current α power exceeded the α threshold whereas the curve corresponding to the EMG power decreased below the EMG threshold, there was a beep (see Fig. 2 in [43]). The mean power values before the biofeedback session usually corresponded to the threshold level at which the feedback signal frequency was no less than 40% of the screen time [52]. The thresholds for each new biofeedback session were calculated and set anew in accordance with the resting α and EMG power before the session. All subjects were told to remember the feeling that accompanied the appearance of the sound and to try to reproduce them during the training. The subjects of the experimental group were instructed to try to hear this feedback signal during the sessions as long and often as possible, whereas and the subjects of the control group were told that they did not receive feedback signals and should not pay attention to the random sounds that were fed with the same frequency as in the experimental group. Both true and sham biofeedback training sessions were conducted with the eyes closed.

The training session consisted of six 3-min periods with breaks for self-reports about the methods and techniques of self-regulation that are used to increase the upper α band power. Immediately and one month after ten biofeedback sessions, all subjects again had psychometric and electrophysiological tests and the second trial biofeedback session, in which the subjects were asked to increase the α power without the aid of feedback during the last three 3-min periods.

The biofeedback session effectiveness was calculated using the software MatLab as the ratio of periods of successful training, in which the α power increased whereas the EMG power simultaneously decreased, to the total session duration [53].

The effect of biofeedback on the psychometric and electrophysiological characteristics was estimated by using analysis of variance (ANOVA) tests. The mean values of EEG parameters were statistically compared by ANOVA/MANOVA analysis using a personal comparison plan (*within-subjects design*) by the time factor (three levels: before (level 1), immediately after (level 2), and one month after (level 3) of the training course). Significant differences between repeated measurements were revealed using a posteriori comparisons (*post hoc*) and the Sheffe test. The significance of intergroup differences was estimated using Student's *t* test in the case of parametric samples and the nonparametric Wilcoxon and Mann–Whitney tests in the case of samples of variables that did obey normal distribution.

RESULTS

ANOVA analysis of the effect of the α power increase training on HRV parameters showed a significant relationship between TIME × GROUP × RESPONSE factors ($F_{(2, 39)} \geq 4.27$, $p \leq 0.03$) only for two variables: LF/HF and pNN_{50}; the other parameters studied did not change significantly. The relationship between TIME × GROUP × RESPONSE factors means that, after the training, HRV parameters of responders and nonresponders of the experimental and control groups changed in different directions. For example, after ten biofeedback training sessions, the LF/HF ratio and the pNN_{50} value in the RB subjects (responders, biofeedback), who managed to increase the α power using biofeedback, increased ($t \geq 5.17$, $p \leq 0.02$), whereas in the control group of responders (RC), who did not use feedback to increase the α-wave power, these HRV characteristics did not change ($p > 0.05$) (Fig. 1). Importantly, after ten biofeedback training sessions, the LF/HF and pNN_{50} parameters increased only in the RB subgroup, whereas in the NB group they decreased, which is reflected in the significant interactions of the factors TIME (two levels: before and after ten sessions) and RESPONSE (two levels: RB vs NB) in the group that used biofeedback ($F_{(1, 31)} \geq 3.16$, $p < 0.007$). In the group of subjects who did not use feedback (sham biofeedback), the mean values of LF/HF and pNN_{50} parameters did not change. One month after the end of ten training sessions, changes in HRV did not retain in any group studied. At the same time, the indices of emotional stress (situational anxiety, heart rate, and the integrated EMG power of forehead muscles) decreased in the biofeedback responders (RB) but did not change in NB and all subjects of the control groups (RC and NC) ($F_{(2, 39)} \geq 5.15$, $p < 0.001$) (Fig. 2). Thus, only a successful training of α power during ten biofeedback sessions led to an increase in HRV accompanied by a lower emotional stress, and this effect remained for one month.

To determine the nature of the relationship between the parameters of α activity and HRV as a function of α-frequency pattern at rest, we compared the mean values of psychometric parameters, characteristics of α activity, integrated EMG power of forehead muscles, and the duration and variability of cardio intervals at rest with the eyes closed in the high (NB and RC) and low (RB and NC) α-peak levels (see the table). It can be seen that the psychometric parameters (response time and fluency in solving divergent thinking and memorization accuracy problems) in the biofeedback responders (RB) and sham biofeedback nonresponders (NC) were lower than in the biofeedback nonresponders (NB) and in the control group of responders (RC). The mean subgroup values of individual α-band width in the responders was greater than in the nonresponders ($t \geq 4.42$, $p < 0.048$). Other individual baseline characteristics of α activity, EMG, interpeak distance, and HRV at rest did not differ within the four groups compared (table). Interestingly, the correlation between the baseline frequencies of the maximum peak and HRV with respect to pNN_{50} was positive in RB and NC groups but negative in NB and RC groups (Fig. 3).

These results indicate that (1) the difference in the α-peak resting frequency can serve as a predictor of getting a subject in the group of responders and nonresponders in the voluntary α-power increase training and (2) the correlation between α frequency and HRV is positive in subjects with lower α frequencies and negative in subjects with higher α frequencies.

To establish how the changes (log%) in pNN_{50} activity are associated with the changes in pNN_{50} and LF/HF indices as a result of biofeedback, we performed multiple regression analysis. It was found that the sign of correlation is determined by the interaction of factors GROUP (true biofeedback vs sham biofeedback) × ANSWER (responders vs nonresponders) (*Beta* ≥ –0.39, $t(24) \leq -2.14$, $p < 0.04$). This means that changes in the maximum α-peak frequency and pNN_{50} of responders and nonresponders are opposite in different subgroups: responders who trained α-power increase by using biofeedback, pNN_{50} and α-peak frequency changed unidirectionally ($r = 0.89$; $p = 0.042$), whereas in the nonresponders they changed in opposite directions ($r = -0.87$; $p = 0.058$) (Fig. 4). Interestingly, in the biofeedback group (section RB in Fig. 3), none of the responders showed a decrease in the α-peak frequency or HRV, whereas four out of five nonresponders of this group showed a decrease in the α-peak frequency and no change or increase in the pNN_{50} value (Fig. 3, section NB). This effect was not observed in the sham biofeedback group (Fig. 3). In addition, the wider the individual baseline α-band, the greater was the increase in α power reached by responders after ten biofeedback training sessions ($r = 0.72$, $p = 0.002$). The magnitude of change in HRV characteristics after ten biofeedback sessions was correlated with the magnitude of change in the α-band width ($r \geq 0.88$; $p \leq 0.05$); however, but this correlation was not observed in the control group without feedback.

Thus, our results suggest the existence of a close relationship between the indices of α activity and HRV, the nature of which varies depending on the α-frequency pattern of EEG and the ability to train voluntary modification of α waves.

DISCUSSION

In the 1980s, it became clear that, in addition to practical importance, biofeedback technology is an effective tool to study the heart–brain relationship

Mean values and standard errors of the mean (SEM) of baseline values of psychometric and electrophysiological measurements in subjects who increased (responders) and did not increase (nonresponders) α power by using the biofeedback training or psychic self-regulation techniques (sham biofeedback)

Measured parameter		Biofeedback			Sham biofeedback		
		Responders (RB) ($n = 9$)	Nonresponders (NB) ($n = 5$)	Significance of differences RB vs NB	Responders (RC) ($n = 4$)	Nonresponders (NC) ($n = 9$)	Significance of differences RC vs NC
Response time in the test "Mental rotation task" test, ms		5883(345)	5202 (530)	$p = 0.048$	5221(652)	5603 (731)	*ns*
Fluency in solving creative problems, arb. units		18.15(2.77)	37.18(6.72)	$p = 0.005$	29.62 (4.81)	16.01(4.52)	$p = 0.006$
Memorization accuracy, arb. units		41.78 (7.45)	49.56 (6.89)	*ns*	42.00(7.68)	35.33(4.42)	$p = 0.033$
Internality index on the locus of control scale, abr. units		10.77(3.45)	5.40 (3.87)	$p = 0.008$	5.16(1.66)	11.00(4.35)	$p = 0.004$
Maximum peak frequency, Hz		10.3(1.0)	11.7(0.1)	$p = 0.011$	11.5(0.1)	9.4(0.06)	$p = 0.004$
α-Band width, Hz		6.5(0.5)	5.2(0.4)	$p = 0.021$	7.5(0.8)	6.2(0.6)	$p = 0.049$
Power in α band (the training target), μV^2		20(5.5)	26(6.7)	*ns*	20(5.8)	27(7.2)	*ns*
Integrated EMG, μV^2		23.35(2.69)	17.35(3.61)	*ns*	16.45(3.30)	14.80(3.05)	*ns*
Interpeak distance, ms		718(89)	678 (111)	*ns*	739(109)	837(101)	*ns*
HRV	(LF/HF)	0.76(0.22)	1.12(0.19)	*ns*	1.21(0.23)	0.81(0.21)	*ns*
	pNN_{50}	19.5(6.7)	29.0(5.6)	*ns*	27.2(4.45)	19.2(5.09)	*ns*

Note: For explanation of abbreviations, see "Methods."

[39, 54, 55]. Despite the fact that these studies were performed in the pre-computer era and were limited by the possibilities of analysis of HRV and individual α-activity indices, the results of those studies demonstrated parallel changes in the α-wave power and interpeak distance as a result of biofeedback training. The results of our study, performed with the use of modern high-precision computer technology, agree with the estimates of trends of parallel changes in indices of autonomic (HRV) and central (EEG α activity) regulation of cognitive functions found in earlier studies [54, 55]. Similarly to Agnigotri [39], we found that, as a result of α-frequency biofeedback training, the interpeak distance increases with a decrease in anxiety and emotional stress.

The advantage of assessing not only the interpeak distance but also HRV is that this measurement makes it possible to estimate not only the state of the cardiovascular system but also, according to the vast majority of studies, one of the fundamental physiological properties of the human body—the autonomic regulation [9]. On the basis of this fact, it can be postulated that the training of a voluntary increase in the EEG power in an individual upper α band leads to an increase in the characteristics of the central (α activity) and autonomic (HRV) regulation.

A possible explanation for the fact that HRV increased as a result of training of the high-frequency α power are the data obtained by Plaschke et al. [56], who showed that the amplitude of this range is more sensitive to changes in the bloodstream than the amplitude of the low-frequency α range [56]. In addition, Shaw provided evidence that an increase in the lower α-band using biofeedback is a suitable tool for stimulating conditions such as meditation or hypnosis, whereas self-control management can be learnt only by training the upper α-band, also known as the sensorimotor rhythm [57].

The success of this training depends primarily on the presence or absence of feedback (biofeedback). In the absence of feedback, neither the α activity nor HRV change. In the group of subjects who trained an increase in α power without the aid of feedback, the α-band width range and HRV did not change. In addition, an increase or decrease in HRV is determined by α-frequency waves, an endophenotypic property of central regulation. For example, in the subjects with a lower baseline α-frequency (i.e., those who performed cognitive tasks with a greater strain) [53], HRV indices increased, whereas in the subjects with an initially higher α efficiency, whose initial efficiency in performing cognitive tasks was higher [27], the upper α-power increase training did not increase but, conversely, decreased HRV parameters such as pNN_{50}. This phenomenon of opposite changes in HRV and α frequency can be explained on the basis of the assumption of Koch et al. [58] that α activity is a modulator of the vascular response amplitude, increasing

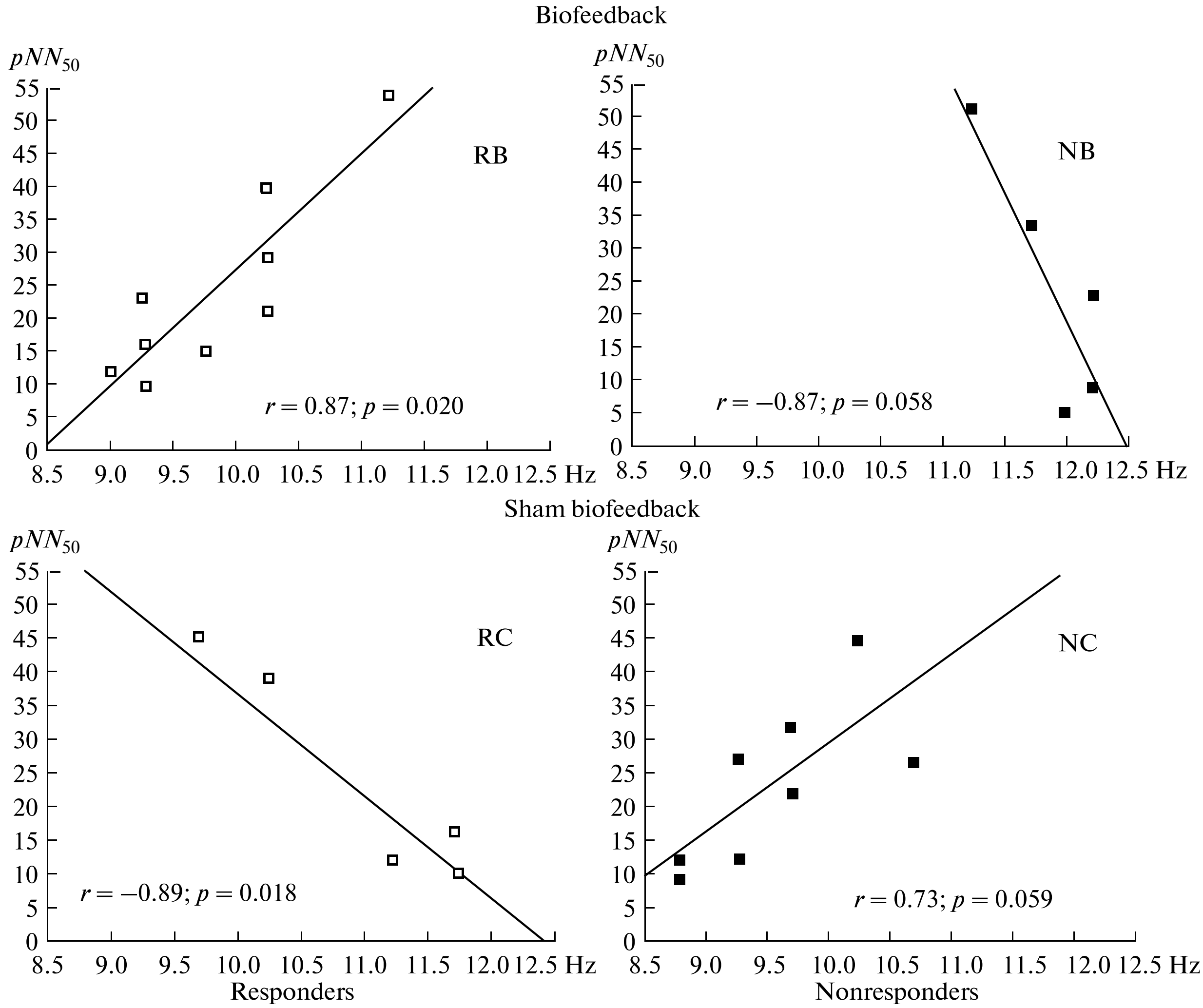

Fig. 3. Relationship between the resting heart rate variability (HRV, ordinate axis) and the maximum α-peak frequency (abscissa axis) in subjects who showed (responders, RB and RC) and did not show (nonresponders, NB and NC) an increase in resting α power as a result of the α-power increase training with and without the use of biofeedback (true and sham biofeedback). Pearson's correlation coefficients and their significance are shown in the corner of each square.

simultaneously with the vascular response at low frequencies and decreasing when vascular response increases at high frequencies. In addition, our results showed that the group of biofeedback responders included those subjects whose baseline indices of cognitive performance were lower are consistent with the conclusion made from the study of the effect of solving mathematical problems on HRV [10]. Those subjects who experienced difficulties in solving problems (i.e., required self-control), showed an increase in HRV in parallel with an increase in the α-wave power; however, neither the α-wave power nor HRV changed in those subjects who solved problems fluently [10]. In our case, HRV increased in those subjects who learned to control the amplitude of their α waves. The more the subject was convinced that he can control his behavior and emotions (according to the Locus of Control test results), the greater was the increment in α power and the α-band width in which the activation of the brain and HRV are observed ($r \geq 0.76$, $p \leq 0.021$), which were reached by the subject after the biofeedback training [43]. This pattern was not observed in the subjects of the control group, whose α training included the conventional psychomotor self-regulation techniques. Interestingly, we found no follow-up effect of the α-power increase training both with and without biofeedback on the resting indices of α-activity and HRV. However, as noted in our previous study [43], in all subjects (both responders and nonresponders), the biofeedback training abolished the decrease in the resting α power, observed in response to solving arithmetic problems, and this effect was retained one month after the end of the biofeedback training. This fact suggests that those subjects who learned to voluntarily increase the α power can

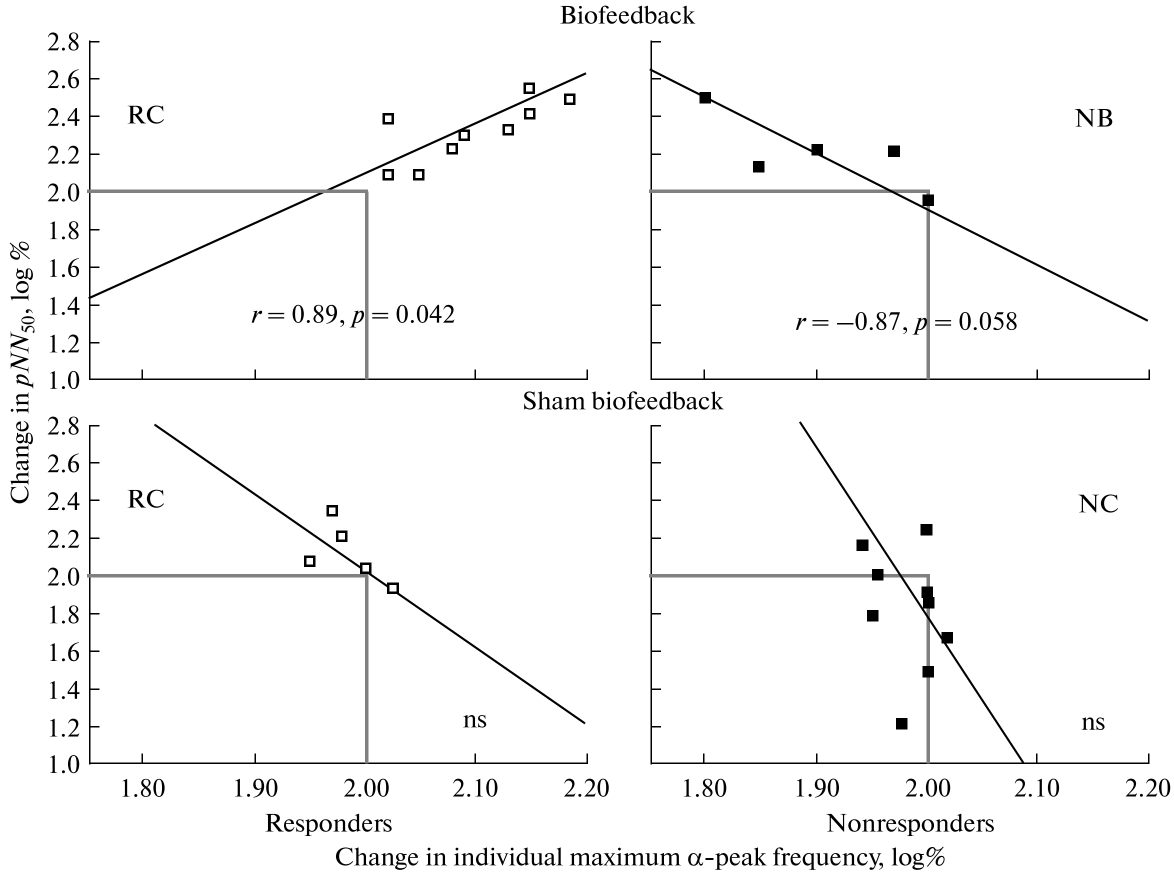

Fig. 4. Relationship between the changes occurred as a result of ten training sessions in the heart rate variability (pNN_{50}, ordinate axis) and the maximum α-peak frequency (individual maximum α-peak frequency, abscissa axis) in the groups of subjects who showed (responders, RB and RC) and did not show (nonresponders, NB and NC) an increase in resting α power as a result of the α-power increase training with and without the use of biofeedback (true and sham biofeedback). Pearson's correlation coefficients and their significance are shown in the lower corner of each square. Gray thick lines limit the range of log% = 2, which means the absence of changes (> 2, increase; < 2, decrease).

voluntarily reduce their psychoemotional stress in cognitive load.

The results confirming the conclusion that the increases in HRV self-control indices and α activity are correlated were obtained in the previous study of the effect of an interpeak distance increase training using a biofeedback game [59]. Such training, aimed at increasing the interpeak distance, increased the level of individual α-activity indices and the LF/HF ratio, whereas a self-regulation training with the aid of conventional computer games influenced neither the indices of EEG α activity nor HRV [59]. The term biofeedback emphasizes the fact that, regardless of which control index was chosen for training, the central and autonomic nervous systems find the same mechanisms of integration and restoration of the balance [60].

In discussing the physiological mechanisms underlying the effect of the biofeedback training on HRV, we encountered difficulties. On the one hand, the most popular is the postulation that an increase in LF/HF in terms of the sympathetic–parasympathetic balance means a decrease in the contribution of the parasympathetic regulation to the processes of autonomic support of mental activity [12]. On the other hand, the assessment of HRV methodologically implies neither the assessment of hormonal influences on the heart, nor the individual approach to establishing the criteria of low- and high-frequency components of interpeak distance range, nor the analysis of the pacemaker component (i.e., heart rate) in determining the total power of HRV range. This is why relative values of LF and HF become floating [61]. In this regard, to draw conclusions about the nature of changes in HRV after the biofeedback training, further studies on the role of hormonal influences (e.g., the pituitary-adrenal axis hormones) on the central and autonomic regulation of mental function are required.

This was the first study to demonstrate that the effect of biofeedback depends on individual and typological properties of the central nervous system. For example, we found that the only baseline difference in all four groups of subjects, revealed as a result of the α-power increase training, was the level of the maximum α-peak frequency, whereas the other physiological indices (EMG, HRV, and power and activation of α waves) remained the same (see the table). However, despite this fact, the correlation between the α-wave frequency and HRV in these groups is different (Figs. 3, 4) and depends on the α-peak frequency: positive in the low-frequency subjects and negative in the subjects with higher baseline values of α frequency. This finding suggests that, on the one hand, the results of biofeedback training can be predicted on the basis of analysis of an individual α-peak frequency and, the other hand, the effectiveness of biofeedback is itself a predictor of endophenotypic features of the psychophysiological profile. First observed by us in music students [53], the prognostic value of biofeedback was again confirmed in this study.

There is no doubt that the discovered correlation between the indices of α activity in the brain and HRV contributes to the theory of corticovisceral regulation and understanding individual mechanisms of realization of subcortical–stem effects on the cerebral cortex. However, this study did not include investigation of endocrine regulation, the most important component of corticovisceral interactions that should be the subject of future specific studies.

Thus, on the basis of the results of our study, it can be postulated that the use of biofeedback for training a voluntary increase in the upper α-band power is more effective than the use of conventional self-regulation techniques.

CONCLUSIONS

1. Training of a voluntary increase in individual upper α-band power by using biofeedback not only led to an increase in α activity but also reduced EMG indices of psychoemotional stress and increased the duration and level of variability of interpeak distances in 9 of 14 healthy subjects with an initially low individual α-peak frequency. In the subjects with an initially high α frequency, changes in the level of EMG and α activity were not observed, whereas the pNN_{50} parameter decreased.

2. Training of a voluntary increase in individual upper α-band power by using psychic self-regulation techniques without feedback signal does not change the emotional stress indices, the α-peak frequency and width, the degree of activation, and heart rate variability.

3. At rest, the correlation between the individual α-peak frequency and the heart rate variability parameter pNN_{50} is positive in the subjects with initially low α-frequency EEG pattern and negative in the subjects with initially high α-frequency EEG pattern.

4. The change in the heart rate variability, coherent with the change in the characteristics of cognitive and psychoemotional activity and α-activity indices testifies to the important role of neurovisceral relationships in the regulation of mental processes.

5. Individual α-frequency EEG pattern determines the efficiency of the influence of α EEG biofeedback training on the changes in the heart rate variability indices, which gives reason to predict the results and develop individual approaches to the biofeedback technology realization, which can be used in clinical practice for therapy and rehabilitation of psychosomatic syndromes and in educational training.

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