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Features of brain activity in alcohol dependence in the task of inhibitory control

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ABSTRACT

Aim. To study neurophysiological correlates of inhibitory control to determine the features of inhibition processes in alcohol dependence.

Materials and methods. 77 patients with alcohol dependence were examined (42 men and 35 women) (F10. 2 according to ICD-10). Patients were examined using a test to assess inhibitory control – Go / No – Go. According to the task performance, patients were divided into two groups: group 1 – without inhibitory control impairments, group 2 – with impaired inhibitory control. During execution of test, electroencephalogram recordings were made according to the "10–20" system. The values of spectral power and coherence of θ-, α- and β-rhythms were analyzed. Statistical processing was carried out using nonparametric Mann – Whitney U-test and Wilcoxon W-test.

Results. In patients with impaired inhibitory control, there was a decrease in the spectral power of the α-rhythm in the frontal cortex ($p = 0.003$), whereas in patients without inhibitory control disorders – in the Central cortex ($p = 0.036$). Patients with impaired inhibitory control responded by increasing β-power to cognitive stimulus in the occipital ($p = 0.014$), left temporal ($p = 0.009$) and right temporal ($p = 0.008$) cortex, while patients without inhibitory control disorders showed an increase in β-power only in the occipital ($p = 0.007$) and left temporal ($p = 0.002$) cortex. According to coherence data, patients with impaired inhibitory control have greater involvement of brain structures during the "Go/No–go" test in all frequency ranges.

Conclusion. Patients with and without impaired inhibitory control have regional differences in changes in brain bioelectric activity during the "Go/No–go" test.

Key words: alcohol dependence, inhibitory control, electroencephalography, diagnostics.

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Conformity with the principles of ethics. All the participants of the study had signed the informed consent. The study was approved by the local Ethics Committee at Mental Health Research Institute, Tomsk National Research Medical Center of the Russian Academy of Sciences (Protocol No. 114 of 22.10.2018).

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Особенности мозговой активности при алкогольной зависимости в задаче на ингибиторный контроль

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РЕЗЮМЕ

Цель: изучить нейрофизиологические корреляты ингибиторного контроля для определения особенностей процессов торможения при алкогольной зависимости.

Материалы и методы. Обследованы 77 пациентов (42 мужчины и 35 женщин) с алкогольной зависимостью (F10.2 по МКБ-10). Пациенты обследованы с помощью теста для оценки ингибиторного контроля – Go / No – go. По результатам этого теста пациенты были разделены на две группы: группа 1 – без нарушения ингибиторного контроля, группа 2 – с нарушением ингибиторного контроля. Во время выполнения теста проводилась запись электроэнцефалограммы по системе 10-20. Анализировались значения спектральной мощности и когерентности θ- α- и β-ритмов. Статистическая обработка проводилась с применением непараметрического *U*-критерия Манна – Уитни и *W*-критерия Вилкоксона.

Результаты. У пациентов с нарушенным ингибиторным контролем происходило снижение спектральной мощности α-ритма во фронтальной коре головного мозга ($p = 0,003$), тогда как у пациентов без нарушений ингибиторного контроля – в центральной коре ($p = 0,036$). Пациенты с нарушенным ингибиторным контролем реагировали повышением β-мощности на когнитивный стимул в затылочной ($p = 0,014$), левой височной ($p = 0,009$) и правой височной ($p = 0,008$) коре, при этом у пациентов без нарушений ингибиторного контроля наблюдалось повышение β-мощности только в затылочной ($p = 0,007$) и левой височной ($p = 0,002$) коре. По данным когерентности у пациентов с нарушением ингибиторного контроля наблюдается большая вовлеченность мозговых структур во время выполнения теста Go / No – go во всех частотных диапазонах.

Заключение. Пациенты с нарушением и без нарушения ингибиторного контроля имеют региональные различия в изменениях биоэлектрической активности головного мозга в процессе выполнения теста Go / No – go.

Ключевые слова: алкогольная зависимость, ингибиторный контроль, электроэнцефалография, диагностика.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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INTRODUCTION

Understanding the psychological and neural processes that lead to alcohol-related disorders is an urgent task in both health care and neuroscience [1, 2].Current research has linked the inability to abstain from alcohol consumption, as well as maintain long-term remission, with impaired cognitive functions that regulate behavior, in particular, weakened executive control [1, 3]. Inhibitory processes are important components in controlling behavior [3]. Adaptive inhibitory functioning reflects the ability to stop a potential behavioral response to an external stimulus [4].

The Go/No-Go method is widely used for evaluating the processes of inhibition in higher-order executive functioning [5]. A number of studies have shown a decrease in response inhibition in patients with alcohol use dependence (AUD) to the No-go signal [1, 6, 7]. It is believed that alcohol consumption affects both the suppression of the response to the stimulus and its processing, which leads to the actualization of an erroneous response (response error) to the No–go signal [6, 7]. However, some researchers found no differences in the Go/No-Go task between patients with AUD and moderate drinkers in healthy people [6, 8]. A number of authors are inclined to believe that there is not always a deficit of inhibitory control in alcohol dependence [3]. Thus, the question of violation of inhibitory control in people with alcohol dependence is still open.

Recording and analyzing the bioelectric activity of the brain is one of the most accessible tools for studying the neurophysiological foundations of mental disorders [9, 10]. Spectral and coherent analysis of the main rhythms of the electroencephalogram (EEG) are the most widely used in research. It is believed that changes in the spectral characteristics (amplitude, power) of the EEG are associated with the neurotransmitter systems restructuring in the CNS [11]. Coherence of electrical signals is a quantitative indicator of the synchronicity of involvement of various cortical departments in any process [9].

The use of the method of assessing the bioelectric activity of the brain in psychiatry and drug therapy can expand the available data on the neurophysiological profile of patients with alcohol use dependence. However, despite the high availability and informative nature of EEG, studies of neurophysiological correlates of inhibitory control disorders in AUD are insufficient. Thus, the aim of our work was to study the neurophysiological correlates of inhibitory control to determine the features of inhibition processes in alcohol dependence.

MATERIALS AND METHODS

Materials. The study was conducted on the basis of the Department of the clinic of the Mental Health Research Institute (Department of addictive conditions) Tomsk national research medical center of the Russian Academy of Sciences, according to the Protocol approved by the local ethics Committee at the Mental Health Research Institute (Protocol No. 114 of October 22, 2018).

We examined 77 patients (42 men, 35 women, age 45 [38; 51] years) with a diagnosis of: mental disorders and behavioral disorders associated with substance use, alcohol dependence syndrome (F10. 2 according to ICD-10), after detoxification. Based on the literature data on various profiles of electrophysiological indicators in right-handed and left-handed people, right-handers were selected for the study group using a questionnaire of lateral characteristics. Diagnostic evaluation and clinical qualification of the disorder were performed using ICD-10 diagnostic criteria, patient anamnestic data, and a set of standardized psychometric tools. The inclusion criteria were verified diagnosis of an addictive disorder (alcohol dependence) according to ICD-10, voluntary consent to participate in the study, normal or adjusted to normal vision, and age 18–55 years. The exclusion criteria were the presence of chronic somatic diseases in the acute stage, epilepsy, severe organic brain damage, traumatic brain injuries of any severity, mental retardation, and refusal to participate in the study.

Methods. The Hamilton Anxiety Scale (HAM-A) and the Clinical Global Impression Scale (CGI-S) were used as psychometric tools. Data on the age of AUD disease, education, the number of hospitalizations and the amount of alcohol consumption were taken from the patient's medical history.

The inhibitor control was evaluated using a computerized Go/ No – go test (Fig. 1). Patients had to press the button when a green signal appeared on the computer screen (the Go signal) and not press the button when a red signal appeared (the No-go signal). The signals were presented in random order. The time of presentation of the signal was 500 ms, and the interval between signals was 800 ms.

Recording and evaluation of bioelectric activity of the brain was performed on a 16-channel Neuropoligraph EEG using the international system "10–20" (Fig. 2). The cutoff frequencies of upper and lower frequency filters were 30 and 1.5 Hz. The study procedure included recording the background EEG at rest with open eyes (1 min), after which the patients

performed a "Go/No – Go" test with simultaneous EEG registration. Artefact fragments were deleted from the EEG recordings. The signals were processed using fast Fourier transform, and the values of absolute spectral power (mV^2) and coherence for θ- (4–7 Hz), α- (8–13 Hz) and β- (14–30 Hz) rhythms were analyzed.

Statistical analysis. Statistical data processing was performed using the Statistica 10.0 program. Statistical data is presented in the form of *Me* [Q_1; Q_3]. Verification of agreement with the normal distribution law was performed using the Shapiro – Wilk test. The obtained data did not follow the normal distribution law. The nonparametric Mann – Whitney test was used to evaluate differences between two independent samples (group 1 vs. group 2) and the Wilcoxon test to evaluate differences between two related samples (rest EEG vs. EEG test). The differences were considered statistically significant at a significance level of $p < 0.05$.

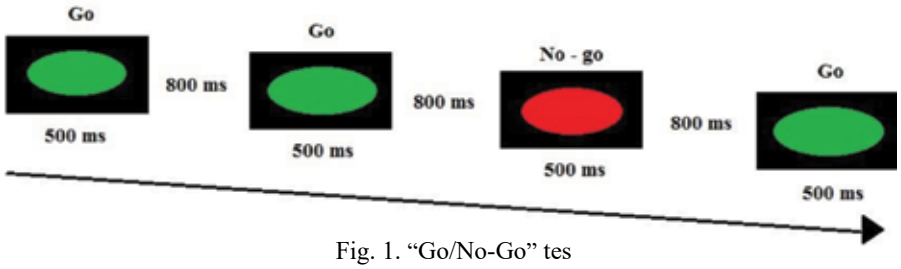


Fig. 1. "Go/No-Go" tes

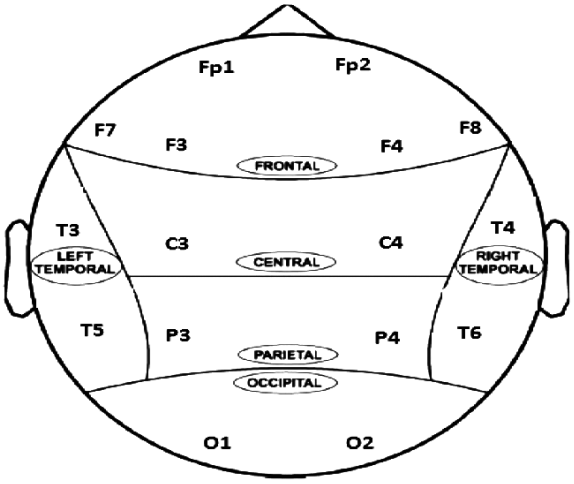


Fig. 2. Scheme of EEG

RESULTS

According to the obtained data of the Go/No-Go test, patients were divided into two groups: patients who did not make mistakes on the No-go signal (group 1) and patients with errors on the No-go signal (group 2) (Table 1).

Table 1

Answers to the "Go/No – Go" test

Characteristic	Group 1, $n = 30$	Group 2, $n = 30$	p
Number of errors per Go signal	10 [7; 13]	8 [3; 13]	0.092
Number of errors per No-go signal	–	4 [3; 7]	–
Reaction time (ms)	461 [455; 498]	489 [466; 500]	0.015

Median [Q_1; Q_3]; p – the level of statistical significance when comparing groups using the Mann – Whitney criterion.

Patients from group 2 have fewer errors on the Go signal compared to patients from group 1, but no statistically significant differences were found ($p > 0.05$). However, patients in group 2 had statistically significantly higher response times ($p < 0.05$).

Demographic and clinical characteristics of the studied groups of patients are presented in Table 2. There were no statistically significant differences in gender, age, level of education, duration of the disease, CGI-S scales (the average assessment of general health after detoxification corresponds to the indicator "moderate disorder") and HAM-A (symptoms of anxiety in both groups) ($p > 0.05$). Compared with patients from group 1 (without violation of inhibitory control), patients from group 2 showed a statistically significantly higher number of hospitalizations over the entire medical history ($p = 0.024$) and alcohol consumption (days per week) over the past 6 months ($p = 0.032$).

Table 2

Demographic and clinical characteristics of patients

Characteristic	Group 1	Group 2	p
Composition	18 men; 12 women	24 men; 23 women.	0.43; 0.35
Age (years)	44 [39; 50]	44 [36; 51]	0.915
Education (years)	13 [11; 14]	13 [11; 15]	0.501
Duration of the disease (years)	10 [4; 13]	9 [5; 12]	0.119
Number of hospitalizations	1 [1; 2]	3 [2; 4]	0.024
Amount of alcohol consumption in the last 6 months. (day per week)	2 [1; 4]	4 [2; 4]	0.032
CGI-S	4 [4; 5]	4 [4; 5]	0.522
HAM-A	19 [10; 25]	19 [12; 23]	0.702

Median [Q_1; Q_3]; p – the level of statistical significance when comparing groups using the Mann – Whitney criterion.

According to the background values of spectral power and coherence of α-, β- and θ-EEG rhythms, patients with errors for the No-go inhibitor signal did not differ significantly from patients without errors ($p > 0.05$). We were able to divide patients qualitatively only by changes in the EEG during the "Go/No – Go" test. Thus, an intragroup analysis of brain bioelectric activity rearrangements in group 2 patients revealed a statistically significant decrease in the spectral power of the α-rhythm in the frontal cortex ($p = 0.003$) and a decrease in α-coherence in Fp1–Fp2 ($p = 0.021$), T3–T4 ($p = 0.003$), Fp1–T3 ($p = 0.003$), T3–O1 ($p = 0.002$) and C3–O1 ($p = 0.034$) during the Go/No-Go test. Whereas in group 1 patients, there was a statistically significant decrease in the spectral power of the α-rhythm in the Central cortex ($p = 0.036$) and a decrease in α-coherence in Fp1–Fp2 ($p = 0.014$), C3–C4 ($p = 0.018$), T3–T4 ($p = 0.004$) and T3–O1 ($p = 0.012$) during the "Go/No – Go" test (Fig. 3).

Group 2 patients responded to a cognitive stimulus in the occipital ($p = 0.014$), left temporal ($p = 0.009$), and right temporal ($p = 0.008$) cortex with increased β-power. And with decreased β-coherence in Fp1–Fp2 ($p = 0.001$), F3–F4 ($p = 0.001$), F7–F8 ($p = 0.006$), C3–C4 ($p = 0.001$), P3–P4 ($p = 0.001$), T3–T4 ($p = 0.001$), T5–T6 ($p = 0.004$), FP1–T3 ($p = 0.045$), FP2–T4 ($p = 0.037$), T3–O1 ($p = 0.004$), T4–O2 ($p = 0.001$), and C3–O1 (v0.005). Patients from group 1 showed an increase in β-power only in the occipital ($p = 0.007$) and left temporal ($p = 0.002$) cortex, as well as a decrease in β-coherence in Fp1–Fp2 ($p = 0.001$), F3–F4 ($p = 0.002$), F7–F8 ($p = 0.009$), C3–C4 ($p = 0.003$), P3–P4 ($p = 0.04$), T3–T4 ($p = 0.002$), T3–O1 ($p = 0.02$), T4–O2 ($p = 0.02$) and C3–O1 ($p = 0.03$) (Fig. 4).

Changes in the slow-wave rhythm (θ-rhythm) in the studied groups of patients with AUD during the "Go/No – Go" test are shown in Fig. 5. Patients in group 2 had a statistically significant decrease in θ-rhythm coherence in Fp1–Fp2 ($p = 0.018$), F7–F8 ($p = 0.009$), T3–T4 ($p = 0.043$), Fp1–T3 ($p = 0.001$), Fp1–C3 ($p = 0.021$), T3–O1 ($p = 0.001$), T4–O2 ($p = 0.001$), C3–O1 ($p = 0.009$), and C4–O2 ($p = 0.004$) during the test. At the same time patients from group 1 showed a decrease in θ-rhythm coherence only in F7–F8 ($p = 0.026$), O1–O2 ($p = 0.007$) and T3-T4 ($p = 0.04$).

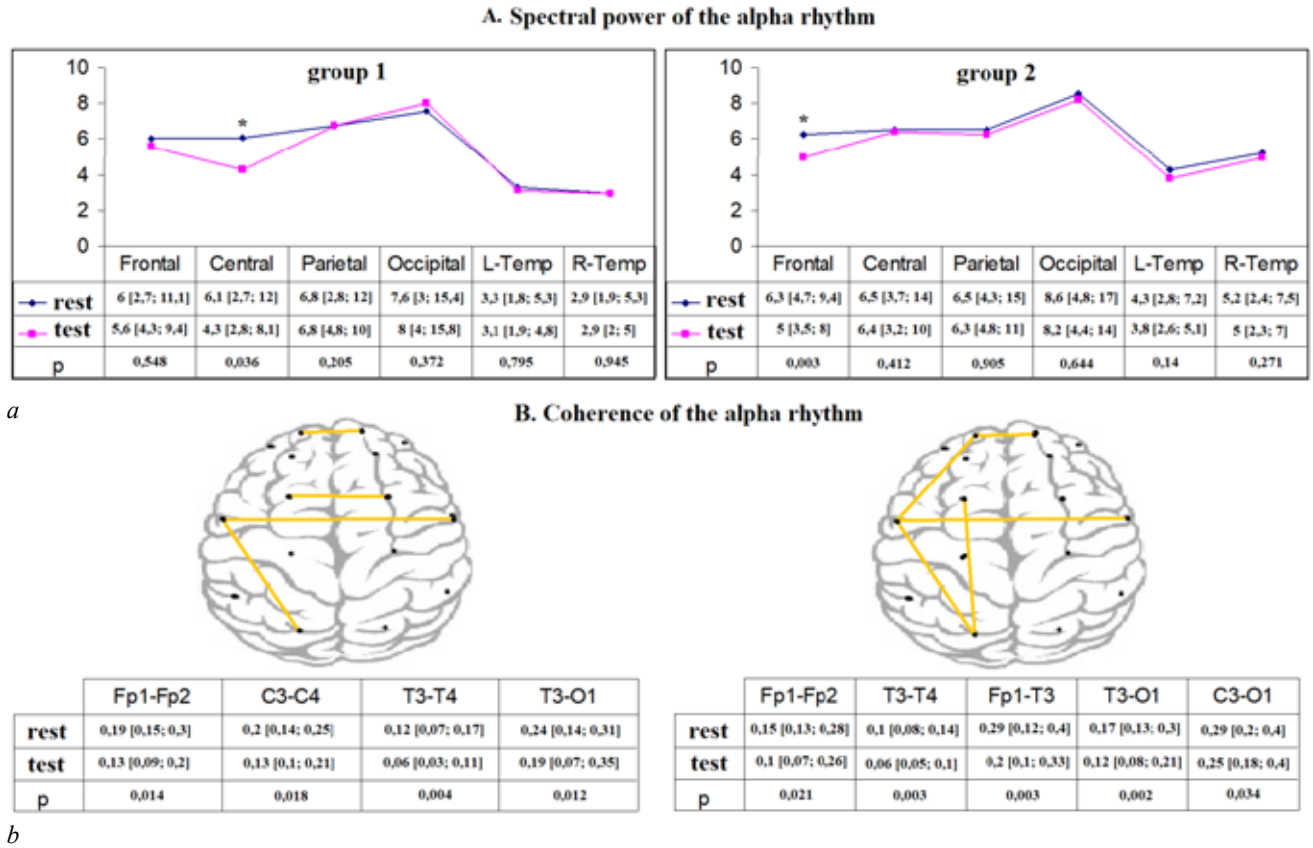


Fig. 3. Dynamics of EEG alpha rhythm during the "Go/No – Go" test,
Median [Q_1; Q_3]: * level of statistical significance when comparing groups using Wilcoxon criterion at $p < 0.05$

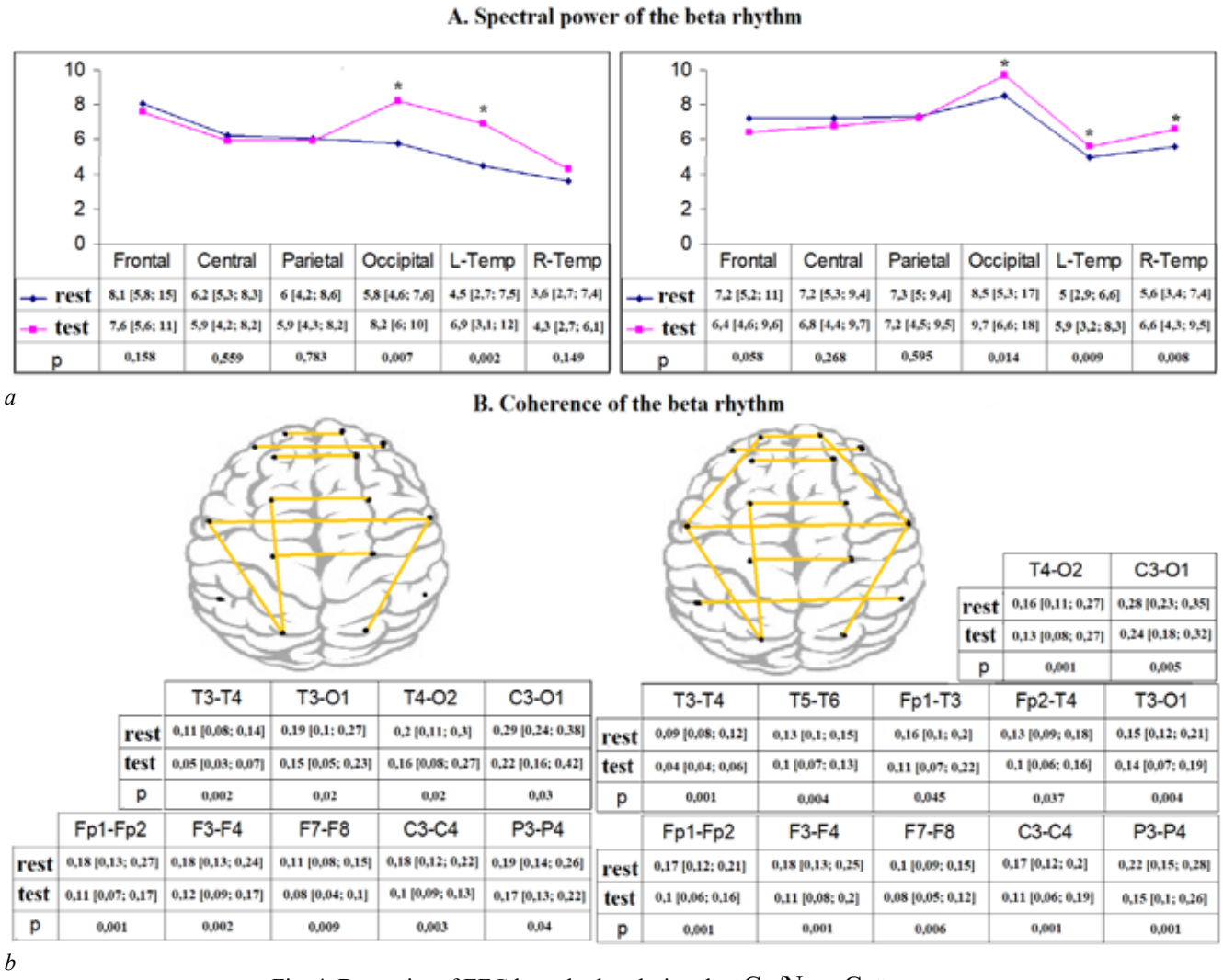
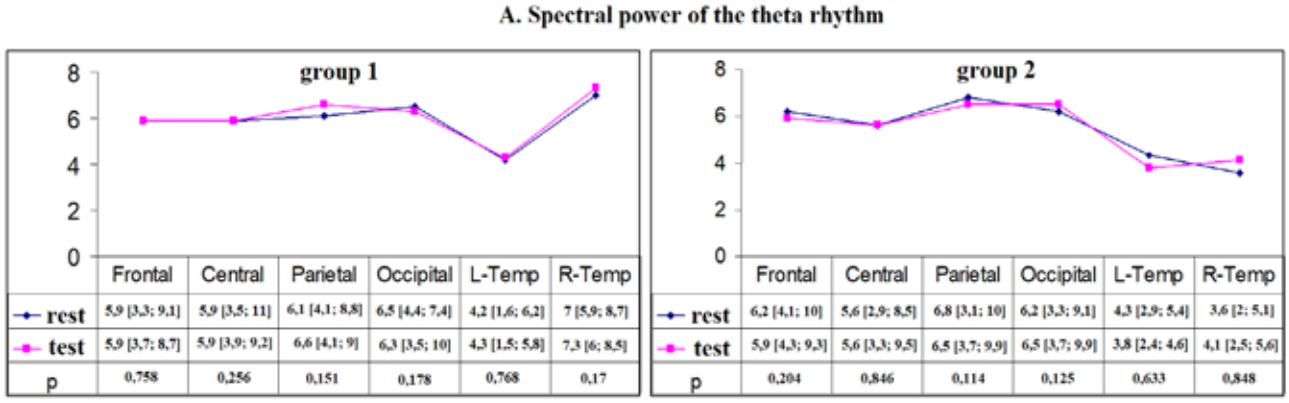


Fig. 4. Dynamics of EEG beta rhythm during the "Go/No – Go" test,
Median [Q_1; Q_3]; * level of statistical significance when comparing groups using Wilcoxon criterion at $p < 0.05$

DISCUSSION

In this study, behavioral characteristics of inhibitory control and its neural correlates were studied using the "Go/No – Go" method to determine the features of inhibition processes in AUD. The data obtained are consistent with the results of some studies using the cognitive "Go/No – Go" task in patients with AUD [4, 6, 12]. It was found that patients with impaired inhibitory control (group 2) had a more severe course of AUD than patients from group 1: more hospitalizations and alcohol consumption per week. Moreover, there is evidence that impaired inhibitory control complicates the symptoms of AUD and increases resistance to therapy [12].

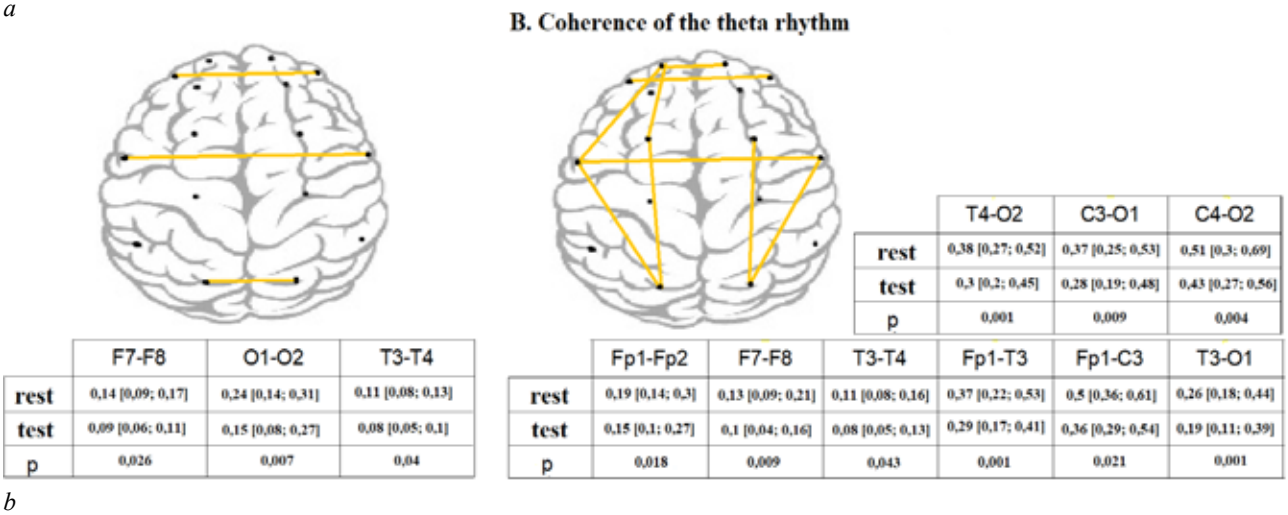
The results of the electrophysiological study revealed a number of important differences. First, in patients with impaired inhibitory control during the "Go/No – Go" test, there was a decrease in the spectral power of the α-rhythm in the frontal cortex, while in patients without impaired inhibitory control this occurred in the central cortex. As a rule, the weakening of inhibitory control is reflected in the form of a decrease in the prefrontal cortex activity [1, 13, 14]. Consequently, the observed decrease in α-activity during the Go/No-Go task in the frontal cortex may objectively reflect a lack of brain resources in suppressing the response to a stimulus. A decrease in the spectral power of the α-rhythm in the central cortex in patients without inhibitory control violations may reflect the processes of triggering behavior (suppression of the response to a stimulus) [13]. Secondly, we found an unusual increase in β-activity in the occipital-temporal cortex during the "Go/No – Go" task, and in patients with impaired inhibitory control, changes



	Frontal	Central	Parietal	Occipital	L-Temp	R-Temp
rest	5,9 [3,3; 9,1]	5,9 [3,5; 11]	6,1 [4,1; 8,8]	6,5 [4,4; 7,4]	4,2 [1,6; 6,2]	7 [5,9; 8,7]
test	5,9 [3,7; 8,7]	5,9 [3,9; 9,2]	6,6 [4,1; 9]	6,3 [3,5; 10]	4,3 [1,5; 5,8]	7,3 [6; 8,5]
p	0,758	0,256	0,151	0,178	0,768	0,17

	Frontal	Central	Parietal	Occipital	L-Temp	R-Temp
rest	6,2 [4,1; 10]	5,6 [2,9; 8,5]	6,8 [3,1; 10]	6,2 [3,3; 9,1]	4,3 [2,9; 5,4]	3,6 [2; 5,1]
test	5,9 [4,3; 9,3]	5,6 [3,3; 9,5]	6,5 [3,7; 9,9]	6,5 [3,7; 9,9]	3,8 [2,4; 4,6]	4,1 [2,5; 5,6]
p	0,204	0,846	0,114	0,125	0,633	0,848

a



	T4-O2	C3-O1	C4-O2
rest	0,38 [0,27; 0,52]	0,37 [0,25; 0,53]	0,51 [0,3; 0,69]
test	0,3 [0,2; 0,45]	0,28 [0,19; 0,48]	0,43 [0,27; 0,56]
p	0,001	0,009	0,004

	F7-F8	O1-O2	T3-T4
rest	0,14 [0,09; 0,17]	0,24 [0,14; 0,31]	0,11 [0,08; 0,13]
test	0,09 [0,06; 0,11]	0,15 [0,08; 0,27]	0,08 [0,05; 0,1]
p	0,026	0,007	0,04

	Fp1-Fp2	F7-F8	T3-T4	Fp1-T3	Fp1-C3	T3-O1
rest	0,19 [0,14; 0,3]	0,13 [0,09; 0,21]	0,11 [0,08; 0,16]	0,37 [0,22; 0,53]	0,5 [0,36; 0,61]	0,26 [0,18; 0,44]
test	0,15 [0,1; 0,27]	0,1 [0,04; 0,16]	0,08 [0,05; 0,13]	0,29 [0,17; 0,41]	0,36 [0,29; 0,54]	0,19 [0,11; 0,39]
p	0,018	0,009	0,043	0,001	0,021	0,001

b

Fig. 5. Dynamics of EEG theta rhythm during the "Go/No – Go" test,
Median [Q_1; Q_3]; * level of statistical significance when comparing groups using Wilcoxon criterion at $p < 0.05$

were observed in both hemispheres. These changes may be due to the fact that when inhibitory control is violated, there is an increased need to activate additional parts of the brain to perform cognitive function. Third, in terms of coherence parameters, we observed significant brain structures involvement in patients with impaired inhibitory control during the Go/No-Go test, and in all frequency ranges. This also confirms the conclusion that the functional activity of the cortex is deficient in patients with AUD with impaired inhibitory control.

CONCLUSION

In general, the results of this study allow us to supplement and improve the understanding of neural functioning in cognitive processes, especially inhibitory control in patients with AUD. Our study showed the ability of the EEG method to detect differences in the electrical activity of the brain during the Go/No-Go task among patients with AUD with or without impaired inhibitory control. A better understanding of the various correlates of alcohol-related behavior and neural effects on regulatory processes can help in the diagnosis of AUD, as well as in the creation of predictive criteria for pathological attraction to alcohol.

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Galkin S.A. – development of the research concept, neurophysiological examination of the sample, data analysis, drafting of the article. Peshkovskaya A.G. – design of the study. Roshchina O.V. – clinical and psychopathological examination of the sample. Kisel N.I. – clinical and psychopathological examination of the sample. Ivanova S.A. – final approval of the manuscript for publication. Bokhan N.A. – final approval of the manuscript for publication.

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