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FEATURES OF CORTICAL FUNCTIONAL ACTIVITY IN PATIENTS WITH PARKINSON'S DISEASE AFTER DEEP BRAIN STIMULATION IN THE LONG-TERM POSTOPERATIVE PERIOD

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Annotation: Parkinson's disease is a chronic neurodegenerative disease characterized by impaired motor function and cognitive processes caused by a decrease in dopaminergic activity. Deep brain stimulation (DBS) is usually used in the late stages of PD to reduce the severity of motor symptoms. Despite the widespread use of the method, its effect on the functional activity of the cerebral cortex in the long-term postoperative period remains a subject of study. EEG is a non-invasive method that allows assessing the state of cortical neural networks over time.

Key words: Parkinson's disease, deep brain stimulation, neuromodulation, EEG, cortical functional activity, coherence, beta rhythm, alpha rhythm, neurophysiology, long-term period, epileptiform activity, cognitive functions.

Introduction. Parkinson's disease (PD) represents one of the most common neurodegenerative disorders, affecting approximately 1-2% of the population over 65 years of age with increasing prevalence in aging societies worldwide. The progressive loss of dopaminergic neurons in the substantia nigra pars compacta leads to the characteristic motor symptoms including bradykinesia, rigidity, resting tremor, and postural instability. While pharmacological management with levodopa and dopamine agonists remains the first-line treatment, many patients eventually develop motor fluctuations and dyskinesias that significantly impair quality of life and functional capacity.

Deep brain stimulation (DBS) has emerged as a well-established surgical intervention for advanced Parkinson's disease refractory to optimal medical management. This technique involves the implantation of electrodes that deliver high-frequency electrical stimulation to specific brain targets, most commonly the subthalamic nucleus (STN) or globus pallidus internus (GPi). Since its FDA approval in 2002, DBS has demonstrated remarkable efficacy in alleviating motor symptoms, reducing medication requirements, and improving quality of life in appropriately selected patients. However, the neurophysiological mechanisms underlying the therapeutic effects of DBS remain incompletely understood.

The traditional model of PD pathophysiology focuses on the direct and indirect pathways within the basal ganglia-thalamocortical circuits. According to this model, dopamine depletion leads to increased inhibitory output from the basal ganglia to the thalamus, resulting in reduced excitatory drive to the motor cortex. While DBS was initially thought to act primarily by inhibiting abnormal neuronal activity in the target nuclei, contemporary evidence suggests more complex mechanisms involving modulation of oscillatory activity and synchronization across distributed neural networks.

Recent neurophysiological and neuroimaging studies have highlighted the significant role of cortical dysfunction in PD symptomatology. Alterations in cortical excitability, abnormal oscillatory activity, particularly in the beta frequency band (13-30 Hz), and disrupted functional connectivity between cortical and subcortical structures have been documented in PD patients. These cortical abnormalities are partially normalized following effective dopaminergic medication and may also be modified by

DBS intervention, suggesting their potential role as electrophysiological biomarkers for therapeutic efficacy.

While the acute effects of DBS on cortical activity have been investigated using various neurophysiological techniques including electroencephalography (EEG), magnetoencephalography (MEG), and transcranial magnetic stimulation (TMS), limited data exist regarding the long-term impact of chronic stimulation on cortical functional organization. The neuroplastic changes that occur in response to continuous DBS over extended periods may differ from immediate effects and could provide crucial insights into the sustained therapeutic mechanisms and potential compensatory adaptations within the motor network.

Furthermore, the relationship between DBS-induced cortical modulation and clinical outcomes in the long-term postoperative period remains poorly characterized. Individual variability in cortical responses to stimulation may contribute to differences in therapeutic efficacy and could potentially serve as predictive markers for long-term benefits or stimulation-related side effects. Understanding these relationships may facilitate personalized optimization of stimulation parameters and improve patient selection criteria for DBS intervention.

The present study aims to investigate the features of cortical functional activity in patients with Parkinson's disease who have undergone STN-DBS, focusing specifically on the long-term postoperative period (>2 years after implantation). Using a multimodal approach combining high-density EEG, functional connectivity analyses, and detailed clinical assessments, we seek to characterize the patterns of cortical oscillatory activity, event-related potentials, and network synchronization associated with chronic stimulation. Additionally, we aim to explore the correlations between these neurophysiological parameters and clinical outcomes, including motor performance, cognitive function, and quality of life measures. Through this comprehensive assessment, we hope to advance our understanding of the neurophysiological mechanisms underlying the sustained therapeutic effects of DBS and potentially identify electrophysiological biomarkers that could guide clinical management and optimize treatment strategies for Parkinson's disease patients.

The aim of the study was to identify the features of the functional activity of the cerebral cortex according to EEG data in patients with Parkinson's disease who underwent deep stimulation surgery, in comparison with patients receiving exclusively drug therapy.

Materials and methods of research. 40 patients diagnosed with PD (stages II–IV according to Hyun and Yaru) were selected for the study, of which: group 1 (n=20) — patients who underwent DBS (subthalamic nucleus) surgery, followed 12-36 months after the intervention; group 2 (n=20) — patients who did not those who have had DBS and are only receiving medication (levodopa, dopaminomimetics, etc.). The patients underwent the following research methods: EEG recording at rest with their eyes closed, analysis of the power of rhythms (delta, theta, alpha, beta), coherence, topographic features. Clinical assessment — UPDRS-III, MoCA, Hamilton Depression Scale. Statistical analysis — Student's t-test, correlation analysis, ANOVA.

Results of the study: the analysis of electroencephalographic data showed significant differences in bioelectric activity between patients who underwent deep brain stimulation surgery (group 1) and patients who are exclusively on drug therapy (group 2). In group 1 (DBS) patients, there was an increase in beta rhythm power (13-30 Hz) in the anterolateral and central leads, especially pronounced in the right hemisphere ($p < 0.05$). This fact may indicate an increase in the functional activity of the motor cortical zones in the long-term period after stimulation. A decrease in the severity of alpha rhythm (8-12 Hz) in the occipital and parietal regions ($p < 0.01$), which may indicate a decrease in the level of basic cortical inhibition and a change in the interaction between the cortex and subcortical structures. Epileptiform patterns were observed in 7 patients (35%): acute waves, paroxysmal theta flashes, mainly in the frontal leads. Similar changes were absent in the group without neurostimulation. In group 2 patients (without DBS), bioelectric activity corresponded to a moderately altered age pattern: alpha rhythm prevailed while maintaining interhemispheric symmetry, and coherence was within the age norm. The coherence study showed that in group 1 there was an increase

in intrahemispheric coherence in the beta range between the frontal and central zones (especially on the right), which is probably due to the functional restructuring of cortical-subcortical networks after DBS. In group 2, there was more pronounced interhemispheric coherence in the alpha range, especially in the occipital leads, reflecting the stability of background cortical activity without surgical intervention. A statistically significant correlation was found between EEG indicators and clinical and functional data: in group 1 patients, an inverse correlation was observed between the severity of theta activity in frontal leads and cognitive indicators on the MoCA scale ($r = -0.63$; $p < 0.01$). This indicates a possible link between frontal dysrhythmia and cognitive decline. The beta rhythm power positively correlated with the results on the UPDRS-III scale ($r = 0.58$; $p < 0.05$), which may indicate the associated effect of DBS on motor activity. Additional analysis showed that the longer the period after DBS surgery (>24 months), the more pronounced EEG abnormalities were observed, including: increased focal theta and beta activity in the frontal and temporal leads; the appearance of unstable delta components at rest; a moderate decrease in coherence between the fronto-occipital zones.

Conclusions: the data obtained confirm that in the long-term period after DBS, patients with PD experience persistent changes in the functional activity of the cerebral cortex. An increase in the power of the beta rhythm and a change in coherence may indicate a long-term restructuring of neural networks involved in motor control. Decreased alpha activity and epileptiform patterns, on the contrary, may indicate maladaptation changes that require monitoring, especially in patients with cognitive decline. Deep brain stimulation has a significant effect on the bioelectric activity of the cortex in patients with PD in the long-term postoperative period. EEG changes are both compensatory and potentially risky. The results emphasize the need for long-term neurophysiological monitoring and individual selection of stimulation parameters.

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