

Figure 4 Correlation between total UPDRS part 3 score and step length in darkness

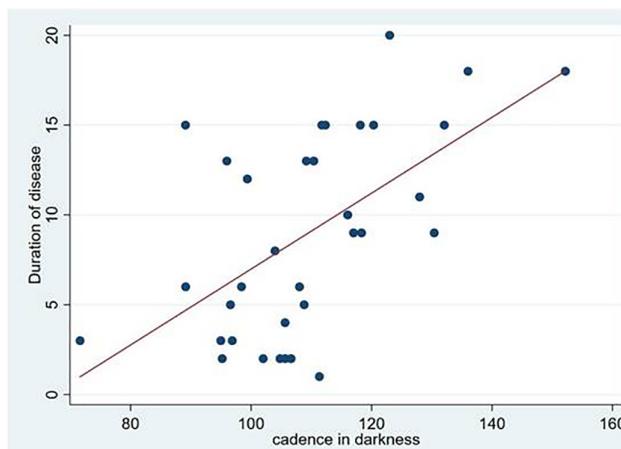


Figure 5 Correlation between duration of disease and cadence in darkness

between bright and dark situations were also compared between patients and controls.

Results: PD patients had significantly decreased normalized velocity ($p<0.01$), shorter stride length ($p<0.01$) and step length ($p<0.01$) but increase double support time ($p<0.01$) in the dark compared to the bright situation. In contrast, healthy controls showed significant increase normalized velocity ($p<0.01$) and cadence ($p=0.01$) with decreased double support time ($p=0.04$) in the dark situation. The percentage of differences in gait parameters between bright and dark situations were opposite in PD compared to controls.

Conclusions: Our study demonstrated that gait abnormalities were worsen in the dark compared to bright situations in PD, highlighting the important effect of light in these patients.

References: Pilgram L.M., G.M. Earhart, and K.A. Pickett, Impact of limiting visual input on gait: Individuals with Parkinson disease, age-matched controls, and healthy young participants. *Somatosens Mot Res*, 2016. 33(1): p. 29-34. Panyakaew P, Anan C, Bhidayasiri R. Visual deprivation elicits subclinical postural inflexibilities in early Parkinson's disease. *J Neurol Sci*. 2015;349(1-2):214-9. Martens KAE, Pieruccini-Faria F, Almeida QJ. Could Sensory Mechanisms Be a Core Factor That Underlies Freezing of Gait in Parkinson's Disease? *Plos One*. 2013;8(5).

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Neurophysiological features of the premotor cortex excitability parameters after a course of therapeutic transcranial magnetic stimulation in patients with Parkinson's Disease stage II living in the Zaporizhzhya region

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Objective: Modern principles of Parkinson's disease (PD) treatment don't include only pharmacotherapy, but also non-drug methods. Particularly, transcranial magnetic stimulation (TMS) is actively used in the treatment of PD. Neurophysiological parameters of the cerebral cortex are the sensory markers of its excitability and inhibition processes and reflect the prognosis of the possibilities of brain cells neuroplasticity.

Background: To determine changes of the premotor cortex excitability parameters in patients with PD stage II after a course of TMS treatment.

Methods: 45 patients with PD stage II according to Hoehn-Yahr were examined. Additionally to basic PD therapy, all patients were prescribed a course of therapeutic TMS sessions in C3, C4 and Cz zones of the brain with a pulse rate of 5 Hz for 8 minutes, 10 sessions per course. Patients were examined using neurophysiological study: determination of the amplitude and latency of the motor evoked potential (MEP), central motor conducting time (CMCT).

Results: Before TMS treatment, the MEP amplitude of the premotor cortex was 7.65 ± 1.21 μ V on the left and 8.36 ± 1.67 μ V on the right brain hemisphere. The latent period of MEP of the premotor cortex was 22.7 ± 0.9 ms on the left and 24.4 ± 1.01 ms on the right, while the sample with facilitation showed a decrease in the latent period of MEP to 17.2 ± 1.23 ms on the left and 18.6 ± 1.87 ms on the right. CMCT was 11.31 ± 1.12 ms on the left and 12.04 ± 0.89 ms on the right.

After the course of TMS there was a statistically significant ($p < 0.05$) increase in the MEP amplitude, which was 13.41 ± 1.45 μ V on the left and 15.76 ± 1.81 μ V on the right. The latent period of MEP significantly ($p < 0.05$) decreased both in classical MEP and was 19.3 ± 0.76 ms on the left and 19.9 ± 1.23 ms on the right, and in the sample with facilitation with decreasing latency of MEP by 12% on the left and 15% on the right. There was also a significant decrease in CMCT after a course of TMS up to 8.77 ± 1.27 ms on the left and 7.84 ± 1.07 ms on the right.

Conclusions: After a course of therapeutic TMS in the examined patients with Parkinson's disease stage II, there is a change in the excitability of the premotor cortex bilaterally, which is manifested by a significant increase in the MEP amplitude and a decrease in the latency of MEP and CMCT.

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Inter-Electrode Distance influences the phase-amplitude coupling (PAC) of Bipolar Subthalamic Local Field Potentials Recordings in Parkinson's Disease

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Objective: This study aimed to assess phase-amplitude coupling (PAC) between two local field potential (LFPs) signals captured from both wide- and close-spaced contact pairs (i.e. LFP03 and LFP12) in the subthalamic nuclei (STN), before and after Levodopa administration in Parkinson's disease (PD) patients.

Background: PAC reflects whether and how different frequency bands, spoken by different neuronal populations or basal ganglia networks, interact each other. Despite the correlation of β -phase - high frequency (200-500Hz) amplitude coupling with PD symptoms [1-8], whether PAC changes are consistent with different electrodes geometry/disposition is still not clear. Yet, no studies have systematically characterized differences between close- and wide-spaced electrode pairs in the subthalamic nucleus.

Methods: LFP12 and LFP03 were recorded from 20 PD patients. We evaluated phase-amplitude coupling between close- and wide spaced electrodes within STN.

Results: Before levodopa, LFP12 showed a strong β -HFO PAC, while after levodopa β -LFO coupling increased and β -HFO decreased in comparison to before levodopa. LFP03 also showed a marked β -HFO PAC before levodopa and after levodopa we found a decrease in β -HFO PAC in comparison to before levodopa condition. Moreover, we found PAC differing between LFP12 and LFP03. Either before and after levodopa LFP03 had lower level of β -LFO and β -HFO coupling than LFP12.

Conclusions: This study discloses the differences in PAC between LFP12 and LFP03 and reveals that levodopa increases β -LFO (15-45 Hz) coupling. low- β -low- γ PAC is a normal mechanism by which a pro-kinetic rhythm is controlled in the GPi [8], possibly accounting the increased β -LFO coupling found in STN following dopaminergic stimulation. This finding may have important implications for adaptive DBS (aDBS) in PD possibly suggesting novel electrophysiological control signals.