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Central cholinergic neurotransmitter system - the role in sleep disorders in patients with Parkinson's diseaseI.V. Krasakov¹, I.V. Litvinenko²¹*The Center of Extrapramidal Disorders, The Nikiforov Russian Center of Emergency and Radiation Medicine,*²*Department and Clinic of Neurology, Military Medical Academy named after S.M. Kirov, Saint Petersburg, Russia*

To evaluate the potential of Galantamine therapy for EDS and RBD in PD patients, we studied 26 patients at the age of 66.6 ± 7.8 years old with the 3rd stage of PD having subjective complaints of sleep disorders. All the patients were rated with the following scales: MMSE, FAB, PDSS, ESS. 17 patients of this group underwent a complex polysomnography (PSG) evaluating the sleep efficacy (TST/TIB \times 100%), sleep latency (LS); REM cycle was also studied for behaviour disorders. Galantamine was given in addition to previous therapy 8mg per day for 4 weeks, then 16mg per day for up to 12 weeks. EDS was revealed in 22 patients (84.5%). In 16 (93%) of 17 patients who had had PSG done a sleep structure disruption as its fragmentation, decrease in sleep quality as increasing the wake time during sleeping and, therefore, reduction of its efficacy were determined. In 12 weeks of treatment patients showed reliable decrease in the intensity of daytime sleepiness (ESS scale results ($p=0.0006$)), reduction in sleep fragmentation, sleep quality (improving the PDSS scale results ($p=0.0007$)), sleep efficacy (TST/TIB ($p=0.0007$)). Against background of the given therapy there were no behaviour disorders in REM sleep revealed. Improvement in hallucination intensity was also noticed: PDSS7 ($p=0.001$) and cognitive disorders (the MMSE ($p=0.001$) and FAB ($p=0.001$) scales). Galantamine therapy induces a significant decrease in daytime sleepiness intensity and behaviour disorders in REM sleep, reduction in sleep fragmentation and improvement in sleep quality.

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Issues of clinical and neurophysiological diagnostics of narcolepsy with cataplexy

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Introduction: Suggestive anamnesis of imperative sleepiness with sudden falls is often insufficient to prove narcolepsy with cataplexy. Diagnosis is supported by multiple sleep latency test (MSLT), where average sleep latency is shortened under the 8 minutes and SOREM is present in at least 2 tests. Because of ambiguous results, MSLT must be repeated in some cases to definitely confirm narcolepsy.

Methods: 31 patients suspected of narcolepsy with cataplexy were examined, which involved: all-night polysomnography with subsequent MSLT during the next day, HLA class II typification, MRI of the brain, psychological as well as psychiatric examination and laboratory screening of excessive sleepiness in order to exclude secondary cause of narcolepsy.

Results: According to the medical history, narcolepsy with cataplexy was presumed in 31 patients. Diagnosis was confirmed by the first MSLT in 25 subjects (80.6%), and by repeated MSLT in 4 subjects (12.9%). MSLT was repeated in those cases, where REM-sleep was not present in at least 2 tests during the first MSLT. Average sleep latency was shortened under 8 minutes in all patients during the first MSLT. Diagnosis was reassessed to depression with pseudocataplexy in 2 patients (6.5%) after the second MSLT.

Conclusion: Discrepancy between clinical and neurophysiological findings in diagnostics of narcolepsy with cataplexy may appear. This severe diagnosis should be clearly verified, most likely by the repeated MSLT, in case that examination of CSL hypocretin level is not available. Depression was the most common condition imitating the symptoms of narcolepsy.