intact (n = 9/10) whilst two groups (n = 17+14/34+13) underwent unilateral 6-hydroxy-dopamine (6OHDA) lesions of the medial forebrain bundle. One 6OHDA group was administered 10 mg/kg L-DOPA/day for two weeks. Animals were food restricted and drug-free throughout motor and operant testing.
In experiment 1, animals were assessed for their ability to retrieve pellets from staircase boxes using their ipsilateral and contralateral paws without pre-lesion exposure to the boxes. In experiment 2, animals were pre-trained on the staircase task and a lateralised choice reaction time task (CRTT), and their post-lesion performance on both compared.
6OHDA lesions decreased the number of contralateral pellets retrieved from the staircase boxes, as well as accuracy on the CRTT when responding to contralateral stimuli – something previous publications have proposed reflect impaired reward signalling. Surprisingly L-DOPA priming further impaired the CRTT deficit, and the rate with which animals without – but not with – pre-training learned to retrieve pellets from the staircase boxes. Preliminary data thus support the hypothesis that L-DOPA priming affects motor learning.

2.214
IMPAIRED CONSOLIDATION OF MOTOR LEARNING IN PARKINSON'S DISEASE

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Introduction: Although Parkinson's Disease (PD) patients benefit from motor rehabilitation in the short-term, benefits are not retained over the long-term. This is posited to be due to intact acquisition of motor learning, but impaired consolidation of motor learning into a longer-term state.
Consolidation requires training and/or post-training rest. Visuomotor adaptation was used to examine consolidation of motor learning in PD.
Previous work showed impaired 24 hour recall of visuomotor adaptation, which may be due to: (1) consolidation deficits *during* training; (2) consolidation deficits during the rest period *after* training; or both.
Objective: This study evaluated if PD patients show deficient consolidation *during* training while continuously on-task to preclude the influence of post-training rest.
Participants: Non-demented, medicated PD patients and age-matched controls.
Method: Participants adapted target-reaching movement to rotated visual feedback of their movement trajectory in an A1-B-A2 paradigm. Feedback was rotated by 30° counterclockwise during A, and 30° clockwise during B.
Task structure: 80 trials on A → 25 trials on B → 15 veridical feedback trials → 25 trials on A.
Results: Consolidation was indicated by: (1) *Anterograde interference:* slower rate of learning B as a result of interference from A and (2) *Savings:* faster re-learning of A at A2.
PD patients showed less anterograde interference from A1 to B, and less savings from A1 to A2 than controls.
Conclusions: PD patients show deficient consolidation of motor learning *during* training while continuously on-task. Hence previous findings of impaired 24 hour recall may be due to consolidation deficits *during* and/or *after* training.

2.215
THE ROLE OF THE CENTRAL CHOLINERGIC NEUROTRANSMITTER SYSTEM IN SLEEP DISORDERS IN PATIENTS WITH PARKINSON'S DISEASE

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Introduction: Sleep disorders such as excessive daytime sleepiness (EDS) and rapid eye movement (REM) sleep behavior disorder (RBD) significantly reduce quality of life in PD.
Research Objectives: The research was aimed to evaluate the potential of galantamine therapy for EDS and RBD in PD.
Methods: 26 patients (F/M=11/15, mean age is 66.6±7.8 years old) with PD having subjective complaints of sleep disorder were studied. All the patients were rated with following scales: MMSE, FAB, PDSS, ESS. 17 patients of this group underwent a polysomnography (PSG). Galantamine was given in addition to previous therapy 8 mg per day for 4 weeks, then 16 mg per day for 4 weeks and following up to 12 weeks 24 mg per day. In 12 weeks of galantamine therapy rerating of patients was carried out and PSG underwent.
Results: EDS was revealed in 22 patients (84.5%). Sleep structure disruption was observed in 16 (93%) of 17 patients (PSG). In 12 weeks of galantamine therapy re-examination of the patients showed significant decrease in intensity of daytime sleepiness ($p = 0.0006$), reduction in sleep fragmentation, improvement of sleep quality (PDSS scale results ($p = 0.0007$)), sleep efficacy (TST/TIB ($p = 0.0007$)). There were no behaviour disorders in REM sleep phase in patients treated by galantamine. Reduce of hallucination was also noticed: PDSS7 ($p = 0.001$) and cognitive disorders (MMSE ($p = 0.001$), FAB ($p = 0.001$)).
Conclusion: Significant decrease of daytime sleepiness and behaviour disorders in REM sleep, reduction in sleep fragmentation and improvement of sleep quality were noticed during galantamine therapy in PD.

2.216
THE NEW NEUROPROTECTIVE AGENT HEMANTANE REDUCES L-DOPA-INDUCED DYSKINESIA IN HEMIPARKINSONIAN RATS

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Objective: Chronic L-DOPA pharmacotherapy in Parkinson's disease is often accompanied by motor complications known as dyskinesia. Although the underlying mechanism of dyskinesia remains to be clarified, recent findings suggest that excitotoxicity, oxidative stress may play a role. Hemantane (H) (N-2(adamantyl)hexamethylenimine hydrochloride) – the NDMA-receptor antagonist, reduced oxidative stress, decreased motor disturbances, tremor in animal models.
The aim of the present study was to evaluate effect of (H) in L-DOPA-induced dyskinesia in rats.
Methods: Unilateral median forebrain bundle injection of 6-hydroxydopamine (12 mkg/4 mkl) was chosen as a model of Parkinson's disease. Three weeks after operation rats received a daily treatment with 10 mg/kg L-DOPA plus benserazide 15 mg/kg for four weeks. During this period about 50% of the rats gradually developed abnormal involuntary movements (AIMs) lasting for 2–3 h following each L-DOPA dose. AIMs were classified in four subtypes: locomotive dyskinesia, axial dystonia, orolingual dyskinesia and forelimb dyskinesia. For quantification of L-DOPA induced dyskinesia, rats were observed individually every 35 minutes from 35 to 140 minutes after the injection of L-DOPA. Treatment with H (10 mg/kg i.p.) and amantadine hydrochloride (A) (20 mg/kg i.p.) was initiated week prior to L-DOPA therapy. Then H was administered 5–10 min and A −100 min before L-DOPA.