[F(1.81, 11.17)=5.95, p = 0.005], naming [F(1.81, 18.51)=5.61, p = 0.007], and semantic verbal fluency [F(1.81, 151.55)=15.52, p < 0.001]. On the contrary, there were significant improvement on verbal memory [F(1.91, 402.36)=9.87, p < 0.001] and reduction on false alarm in memory recognition [F(1.74, 17.19)=5.18, p = 0.01]. Post hoc analyses showed that all significant changes occurred from baseline to 1-year postoperation.

Conclusions: Details of postoperative cognitive changes will be discussed, including the possible mechanisms of memory improvement the performance of our cohorts in semantic fluency. The results also provided information that allowed better patient education among Chinese-Cantonese patients.

References: Højlund A, Petersen MV, Sridharan KS, Østergaard K. Worsening of Verbal Fluency After Deep Brain Stimulation in Parkinson's Disease: A Focused Review. Computational and Structural Biotechnology Journal 2017; 15: 68-74 Tang V, Zhu CXL, Chan D, Lau C, Chan A, Mok V, Yeung J, Poon WS. Evidence of improved immediate verbal memory and diminished category fluency following STN-DBS in Chinese-Cantonese patients with idiopathic Parkinson's disease. Neurological Sciences 2015; 36: 1371-1377.

1738

Heart Rate Variability and Cognitive Impairment in Parkinson's Disease

C. Terroba-Chambi, D. Vigo, M. Merello (Caba, Argentina)

Objective: To evaluate the association between the heart rate variability (HRV) and cognitive impairment in PD patients.

Background: Emerging evidence suggests an association between cognitive impairment and autonomic dysfunction in Parkinson's disease (PD). However, there is lack of information between the relationship of HRV and changes in cognition in this population.

Methods: Nineteen consecutive PD patients without dementia in Hoehn & Yahr stage 2.1±0.2 mean (SD) enrolled in a prospective study of Fear of Falling and falls were assessed by short-term HRV analysis and blood pressure measures during postural changes. EKG were recorded during 5 minutes in resting (RS) and standing (SS) states. Time domain (Mean RR, SDNN, RMSSD), frequency domain (VLF, LF, HF, LF/HF) and non-linear (DFA α1, DFA α2, SampEn) parameters were calculated. Data in RS and SS and its difference (DS) were analyzed. Cognitive performance was evaluated by MoCA, Frontal Assessment battery [FAB], Trail making test [TMT-B], Digit symbol modality test [DSMT], phonemic fluency and Stroop word-color test [SCWT].

Results: Eleven patients (57%) were classified as mild cognitive impairment (PD-MCI) by MoCA. The mean (SD) of age and illness duration were 70.9 ± 6.5 and 8.1 ± 4.7 years respectively. There was no difference in age, gender, illness duration, blood pressure, LED or any MDS-UPDRS scores between groups with or without MCI; MCI-group showed significantly higher LF/HF-SS ratio (p=0.032), lower RMSSD-DS (p=0.003) and lower SampEn-DS (p=0.032) than non-MCI group. The poorer performance in multiple cognitive domain tests was significantly associated with HRV indexes: the phonemic fluency scores with reduced overall HRV (RMSSD-SS p = 0.016; SDNN-RS p = 0.039; SDNN-SS p = 0.016); decreased baroreflex function (LF-SS p = 0.029; HF-RS p = 0.017; HF-SS p = 0.037) and SampEn-SS p = 0.049); MoCA test was associated to lower RMSSD-DS (p=0.003) and higher DFA α2-SS (p=0.044); SWCT was associated to higher values of DFA α2-SS (p=0.026) and DFA α2-DS (p=0.024). No significant differences were found between groups with or without MCI or any independent cognitive test and blood pressure measures during postural changes.

Conclusions: Reduced HRV reflecting decreased parasympathetic activity independently of orthostatic hypotension is significantly associated with PD-MCI. Considering the high rate of conversion of PD-MCI to PDD its role as predictive biomarker should be explored in future studies.

1739

Vascular white matter involvement in Idiopathic Normal Pressure Hydrocephalus: association with cognitive and motor impairment

M. Todisco, P. Vitali, B. Minafra, F. Avantaggiato, R. Zangaglia, N.G. Pozzi, R. Ceravolo, C. Pacchetti (Pavia, Italy)

Objective: To explore the relationship between vascular subcortical encephalopathy, ventricular dilation and clinical severity in idiopathic normal pressure hydrocephalus (iNPH).

Background: The frequent co-occurrence of iNPH and vascular white matter lesions (WML) has been well described in previous literature. Although the clinical impact of WML on iNPH severity is generally acknowledged, the relation between these two conditions is still under investigation.

Methods: 52 consecutive patients (33 males, aged 77,7 +/-4) diagnosed with iNPH following the International NPH Consultant Group Guidelines were enrolled from our Movement Disorders clinic. Clinical history concerning cardiovascular risk factors was collected for each subject. Participants underwent complete neurologic evaluation, coupled with standardized motor (iNPH gait and balance scales) and cognitive (MMSE and MoCA test) assessment, and 3 Tesla brain MRI for standardized measurement of WML and NPH (Fazekas scale, Evans' index). Subjects were subsequently divided into two groups, depending on radiological severity of vascular disease: Group 1 (23 subjects, Fazekas score 5-6, corresponding to moderate/severe vascular involvement), and Group 2 (22 subjects, Fazekas score 0-4, absent/mild vascular involvement).

Results: Statistical analysis revealed a significant correlation between motor and cognitive performance (p=0.028, R=0.31), no correlation between ventriculomegaly (evaluated through Evans' index - EI) and WML, no association between EI and disease severity. Patients with moderate/severe vascular disease showed worse cognitive and motor performance than patients with mild or absent vascular involvement (p=0.013 and p = 0.02 for cognitive and motor performances, respectively), despite no evidence of different prevalence of cardiovascular risk factors.

Conclusions: Our study confirms the role of vascular white matter disease as a major determinant of both motor and cognitive impairment severity in patients with iNPH. The degree of vascular involvement is not entirely related to cardiovascular risk factors, suggesting a specific etiology, directly related to iNPH. As WML does not correlate with EI values, more complex hypotheses than plain mechanical compression should be taken into consideration in future studies, possibly involving the glymphatic system.

1740

Role of Alzheimer's Disease Genetic Risk Variant rs9331896 in Cognitive Decline in Parkinson's Disease

T. Tropea, L. Baratta, K. Maddy, M. Guo, J. Rick, V. Van Deerlin, J. Trojanowski, D. Weintraub, A. Chen-Plotkin (Philadelphia, PA, USA)

Objective: To investigate whether susceptibility loci for Alzheimer's disease (AD) are associated with rates of longitudinal cognitive decline in Parkinson's disease (PD).

Background: Parkinson's disease is the second most prevalent neurodegenerative disease. In addition to cardinal motor symptoms, the majority of PD patients experience significant cognitive impairment long-term. Cognitive impairment can arise and progress at any point in the disease course, while also varying widely in severity between individuals. At autopsy, the presence of co-morbid AD pathology – beta-amyloid plaques and tau neurofibrillary tangles – correlates with more severe cognitive outcomes, such as dementia, in PD. However, the role of common AD-risk genetic variants in the development of cognitive decline and dementia in PD is poorly understood.

Methods: 151 non-demented patients with established, idiopathic PD patients were followed for 1-9 years (mean 2.92 years). A linear mixed-effects model was used to test for associations between long-term cognitive decline (total Montreal Cognitive Assessment (MoCA) or Mattis Dementia Rating Scale-2 (DRS-2) scores) and AD-risk genotype at 19 single nucleotide polymorphisms (SNPs) previously identified by genome-wide association studies of AD. The association of clusterin protein expression in cerebrospinal fluid (CSF) on cognitive function was assessed via linear regression. Models were adjusted for APOEgenotype, sex, age, and baseline MoCA score.

Results: The risk allele at rs9331896, whose nearest gene is CLU, previously associated with increased risk for developing AD, also predicts rate of longitudinal cognitive decline in PD. In addition, a lower level of clusterin protein in CSF was associated with cognitive impairment cross-sectionally and cognitive decline longitudinally.

Conclusions: The AD-risk genotype at rs9331896 and lower CSF clusterin protein expression is associated with a faster rate of cognitive decline in PD. Future studies to validate these findings in additional PD cohorts are warranted.