Neurophysiological analysis of vestibular system and postural abnormalities in Parkinson's disease

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INTRODUCTION

Pisa Syndrome (PS) is a postural abnormality, defined as a lateral trunk flexion of more than 10°, complicating late stage of Parkinson's Disease (PD). The pathophysiology is still unclear but vestibular system, whose role in postural control is well known, may be involved1.

Cervical Vestibular Evoked Myogenic Potentials (cVEMPs) are muscular potentials recorded in sternocleidomaistoid (SCM) muscles after intense bilateral acoustic stimulation. They are an expression of the ipsilateral pathway linking the VIII and XI cranial nuclei, and represent the neurophysiological correlate of the vestibulo-collic reflex. Recently, cVEMPs have been studied to evaluate vestibular function in several neurological conditions, notably Parkinson's disease (PD)2; currently, they have not yet been assessed in PD patients with PS (PDPS).

Here we used **cVEMPs to evaluate vestibular involvement in PD patients with and without PS, compared to a healthy controls group (HC).**

SUBJECTS AND METHODS

We enrolled 15 patients diagnosed with PD, 9 with PD and PS and 19 HC. They all underwent otoscopy and neurological examination. They were evaluated using Unified Parkinson's Disease Rating Scale (UPDRS) part II-III and Hoehn and Yahr scale (H&Y) by a movement disorders expert; then cVEMPs were recorded. Exclusion criteria included dementia (MMSE score< 24), improper neck movements that interfere with audiological assessment, middle ear diseases and hearing thresholds exceeding 50 dBnHL.

Differences in categorical and continuous variables were respectively calculated with the chi-square test and the one-way ANOVA test with Bonferroni correction. Correlations between clinical and electrophysiological data were calculated by Spearman correlation test.

	Parkinson's disease (n=15)	Pakinson's disease with Pisa Syndrome (n=9)	Control group (n=19)	
Age (y)	69.6 +/- 7.11	73.3 +/- 3.6	69.36 +/- 6.67	n.s.
Gender (M/F)	8/7	6/3	8/11	n.s.
Disease duration (y)	4.6 +/- 3.83	8.7 +/- 4.4	---	*p=0.03*
UPDRS II	6.2 +/- 3	12.4 +/- 2.8	---	*p<0.01*
UPDRS III	18.9 +/- 6.1	30 +/- 6.1	---	*p<0.01*

Table 1: clinical data

	Parkinson's Disease (n=15)	Parkinson's disease with Pisa Syndrome (n=9)	Control group (n=19)	
Presence of altered cVEMPs	5/15 (33%)	4/9 (45%)	1/19 (5%)	*p=0.03 (PD vs HC)* *p=0.01 (PDPS vs HC)* p= 0.49 (PD vs PDPS)
P23 latencies (ms)	15.54 +/- 24.15	15.83 +/- 5.07	16.07 +/- 8.97	n. s.
N23 latencies (ms)	24.15 +/-8.08	23.58 +/- 5.39	25.77 +/- 10.48	n. s
P23 amplitudes (mcV)	43.55 +/- 34.87	43.66 +/- 24.93	40.57 +/- 26.1	n. s.
N23 amplitudes (mcV)	-50.88 +/- 34.86	-50 +/- 38.36	-63.16 +/- 42.31	n. s.

Table 2: electrophysiological data

RESULTS

Groups **did not show differences in gender, and age** distribution *(table 1)*. PDPS group had a **longer disease duration** (p=0.03) and **higher** scores in **UPDRS II and III** (p<0.01) *(table 1)*.
cVEMPs were altered in 1/19 (5%) HC, in 5/15 (33%) PD patients and in 4/9 (45%) PDPS patients. **Absence of potential** *(fig. 1)*, the main cVEMPs alteration, was significantly **more frequent in PD and PDPS** patients than in HC (respectively p=0,03, p=0,01). On the other hand, even if PDPS group showed a higher percentage of patients with abnormal cVEMPs (*fig.2*), this was far from being statistically significant (p=0,49). There were **no differences in cVEMPs P13 and N23 latencies and amplitudes** among groups *(table 2)*.
Finally, **no correlation** was found between neurophysiological and clinical data.

Figure 1:

A. example of normal cVEMPs

B. unilateral absence of cVEMP.

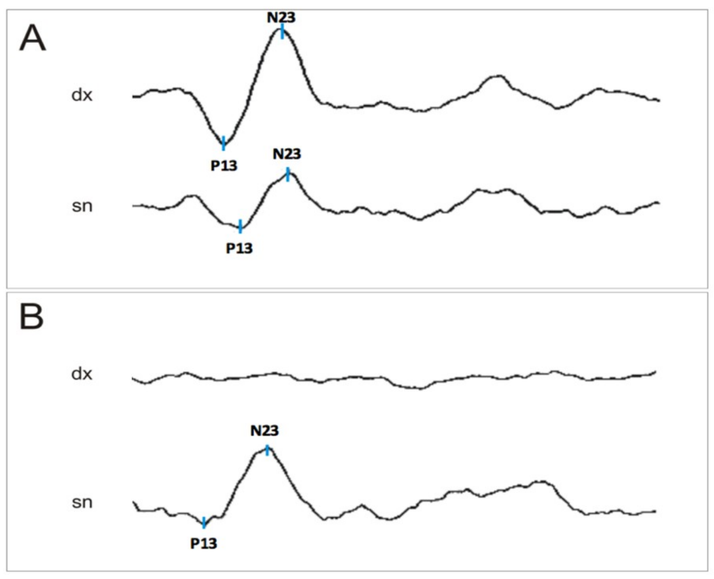
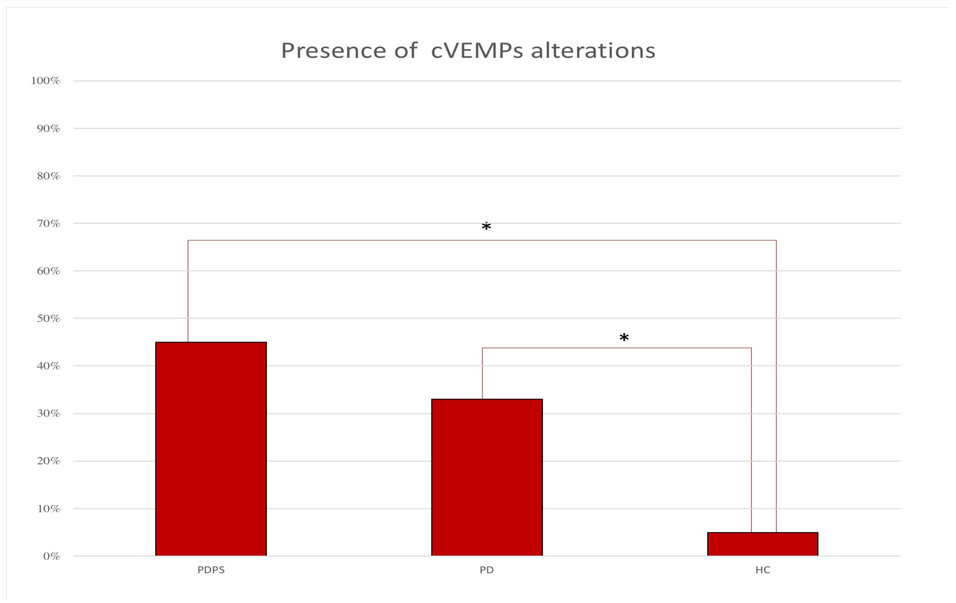


Figure 2:

Percentage of subjects presenting abnormal cVEMPs among groups.

**=p<0.05*



DISCUSSION

PD is a **neurodegenerative** disease, in which **α-synuclein** deposits are found not only in SNpc but also in most of brainstem structures, causing the impairment of multiple neurotransmettitorial systems. Of interest, it has been recently observed that **dopamine** is a **modulator of vestibular nuclei function3.**
According to these findings, **cVEMPs alteration** in PD patients seem to be a direct **marker** of brainstem neurodegeneration and vestibular dopaminergic dysfunction, which resulted to be **higher in patients with PS**.
Our study showed that **PD patients exhibit vestibular dysfunctions**, higher in patients with PS**,** at late stages of disease, such to suggest a role of vestibular system in the pathophysiology of this peculiar postural abnormality.

References:

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