



Research report

Parkinson's patients can rely on perspective cues to perceive 3D space



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ABSTRACT

3D perception, which is necessary for an optimal navigation in our environment, relies on 2 complementary kinds of cues; binocular cues allowing precise depth localization near the point of visual interest and monocular ones that are necessary for correct global perception of visual space. Recent studies described deficient binocular 3D vision in PD patients; here we tested 3D vision in PD patients when based on monocular cues (m3D).

Sixteen PD patients and 16 controls had to categorize visual stimuli as perceived in 2D (flat) or 3D (with depth). Both performance and response times were measured. EEGs were recorded to extract Visual Evoked Potentials. Effects of PD were tested by comparing psychometric and electrophysiological data obtained in controls and PD patients evaluated without dopaminergic treatment. Effects of Levodopa were tested by comparing data in PD patients with and without dopaminergic treatment.

We didn't find statistical differences between PD patients and controls' performance. Severity of PD (UPDRS III) in OFF condition is positively correlated with P1 amplitudes and latencies for both 2D and m3D stimuli. Levodopa administration didn't modify either PD patients' performances although it increases principal visual components latencies for both 2D and m3D stimuli.

Unlike binocular 3D vision, monocular 3D vision does not seem to get affected by PD. However given the correlation between severity of PD and VEPs' modifications, alteration of visual cortical processing might have nonetheless begun. PD patients reporting trouble in perceiving space must rely more on m3D cues present in the environment.

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1. Introduction

Although there is a wide variety of visual impairments in Parkinson's disease (PD) (Armstrong, 2011; Bodis-Wollner and Paulus, 1999; Sauerbier and Chaudhuri, 2013), three-dimensional (3D) visual perception of space roused some interest only recently in PD patients (Kim et al., 2011; Kwon et al., 2014; Lee et al., 2015; Sun et al., 2014). Yet, a decent 3D spatial vision is essential for optimal navigation and interaction with our environment. Objects project onto the two retinal images that are in 2 dimensions (2D)

but the brain is able to process those images in order to reconstruct their 3D properties. To achieve that, the brain uses two types of visual indices; on one hand, binocular cues that require both eyes, allow a quantitative and precise 3D perception close to the fixation point (stereopsis), and principally in near space. On the other hand, monocular cues, such as perspective, shading, relative size of objects... give rise to the same 3D perception whether they are viewed by one or both eyes, and allow a more qualitative assessment of 3D position and shape of objects located further away. Both kinds of 3D indices are integrated together in the brain to give rise to a coherent 3D perception (Howard and Rogers, 2002). Deficits in the cortical processing of 3D indices would result in a flat 2D perception of the world. Recently it has been shown that a high proportion of PD patients present impaired binocular stereopsis (Kim et al., 2011; Kwon et al., 2014; Lee et al., 2015; Sun et al., 2014) which raises the possibility that this trouble might have consequence for the interaction of PD patients with their environment.

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In this study, we addressed the question of whether monocular 3D (m3D) vision was also impaired in PD. We compared m3D vision in PD patients and control subjects with psychophysical methods as well as the underlying electrophysiological activities. The effect of Dopamine on m3D vision was also addressed.

2. Results

The demographic, clinical characteristics and principal statistical effects are shown respectively in [Tables 1 and 2](#).

2.1. Effect of PD

No differences were found between PD patients and controls in terms of performances ($p = 0.7$) or response times ($p = 0.7$) ([Fig. 1, Table 2a](#)). 3D perception based on monocular cues is not affected in our population of patients, which means that images with an imbedded perspective are correctly interpreted as being in 3D and are not perceived flat. We didn't find either any relationship between the severity of disease expressed by UPDRS III measured in OFF condition and performances ($p = 0.26$, [Table 2b](#)) or response times ($p = 0.9$, [Table 2c](#)). Although PD patients displayed normal performances and did not have cognitive impairment, PD patients had on average lower Mattis scores ($p < 0.05$, see [Table 1](#)) and we observed a correlation between the Mattis global score and performances ([Table 2b](#), $p = 0.04$). This relationship was mainly driven by the attention subdomain scores ([Table 2c](#), $p = 0.02$).

Consistently with psychophysical data, we observed no differences in the underlying electrophysiological signals between PD patients and controls whether in 2D ($p = 0.91$, 0.39 , 0.53 , 0.89 for P1 and N1 amplitudes, P1 and N1 latencies respectively) or 3D conditions ($p = 0.87$, 0.89 , 0.7 , 0.94 for P1 and N1 amplitudes, P1 and N1 latencies respectively) (see [Fig. 2a and b](#) and [Table 2d](#)). However, we did find that patients with higher UPDRS scores had, for both kinds of stimuli, higher P1 amplitudes (2D, $p = 0.052$; m3D, $p = 0.02$) and longer P1 latencies (2D, $p = 0.008$; m3D, $p = 0.002$) ([Table 2e](#)).

Furthermore, patients with higher attention sub-scores had longer P1 and N1 latencies ([Table 2f](#)) for both kind of stimuli ($p = 0.05$, 0.02 , 0.04 , 0.02 respectively for P1 latency for 2D and m3D stimuli, N1 latency for 2D and m3D stimuli).

2.2. Effect of treatment on 3D perception

While motor symptoms measured by UPDRS III sub score were greatly reduced with Levodopa administration ($p < 0.001$, see

[Table 1](#)), no differences were found between PD patients in OFF and ON conditions in terms of performances ($p = 0.73$) or response times ($p = 0.62$) ([Fig. 1, Table 2g](#)).

Although PD patients were as capable as controls to categorize 2D and m3D stimuli, we did observe a general slow-down in recorded cortical activities in patients in ON condition compared to OFF condition ([Fig. 2b](#) right part, [Table 2h](#)), concerning both responses to 2D and m3D stimuli ($p = 0.0001$, 0.005 , 0.03 , 0.005 respectively for P1 latency for 2D and m3D stimuli, N1 latency for 2D and m3D stimuli).

3. Discussion

In our study, PD patients preserved a normal depth perception in presence of monocular cues like perspective, although Kim and collaborators showed that PD patients had difficulty to perceive depth produced by binocular stereopsis (Kim et al., 2011).

Considering the very small statistical size effects observed, it is unlikely that we missed a potential effect of PD on monocular 3D perception because of an insufficient number of patients.

Rather, our results suggest that PD has a different effect according to the types of visual indices for 3D perception. This result is not surprising because selectivity for retinal disparity, which is responsible for binocular 3D perception, is present as early as primary visual cortex (see (Howard and Rogers, 2002)). PD could affect stereopsis processing there which is not the case for monocular 3D cues as they are processed much later in visual cortex (Howard and Rogers, 2002). Both kinds of cues are then combined in regions within the intra parietal sulcus (Durand et al., 2007) in order to allow the optimal estimation of object's 3D position and shape required for reaching movement and to perform correctly pre-shaping of the hand before grasping that object.

We found that patients with higher Mattis attention sub-scores tend to have better performances and longer components latencies: patients who were able to maintain more steadily their attention focused during the visual discrimination task obtained logically better performances. Coherently, it has been shown that a discrimination task requiring great attention effort was accompanied by increased N1 latencies (Callaway and Halliday, 1982). Our subjects did confirm after the experiments that they indeed found the discrimination task used here quite attention demanding.

We did not observe slower responses in PD patients as often reported in other studies; this is certainly due to the instructions given to the subjects. Because this 2D/m3D discrimination task was quite difficult, we emphasized more on the necessity of precise responses rather than on speed. Time needed to form a firm decision must have exceeded the motor slowdown observed in PD patients. Coherently, we did not observe either a decrease in response time in ON condition.

Although no modification in psychophysical results have been observed after Levodopa administration, we detected at the neuronal level a small increase of P1 (3.3 ms) and N1 (4.2 ms) latencies that was not specific to 3D vision but on the contrary seemed to reflect a general slow-down in visual processing. Other studies have measured VEP latencies in PD before and during Levodopa treatment; results are very variable depending on studies and seem to be dose-dependent, reflecting thus antagonizing effects of dopamine in visual system due to both excitatory and inhibitory influences (Bodis-Wollner et al., 1982; Yaar, 1980).

Patients included in this study were moderately affected by PD. Whereas we did not observe any relationship between psychometric data and severity of disease as measured by UPDRS part III sub score in OFF condition, we can't exclude the existence of a deterioration in m3D perception in patients with more severe motor symptoms. The correlation between both amplitude and latency

Table 1
Demographic and clinical data.

Demographic and clinical profiles	Controls	PD patients	Significativity
Gender (M/F)	11/5	11/5	n.s.
Age (years)	65.1 ± 8.5	65.8 ± 7.8	n.s.
Duration of disease (years)		7.6 ± 4.3	
UPDRS III			
OFF		22.3 ± 10.8	
ON		14.9 ± 10.6	ON/OFF***
Mattis total score	142.8 ± 2.3	140.5 ± 3.3	*
Attention	36.7 ± 0.4	36.4 ± 0.8	n.s.
Construction	6	6	n.s.
Memory	24.6 ± 1.3	24.1 ± 1.6	n.s.
Initiation	36.9 ± 0.5	35.6 ± 2.2	*
Conceptualization	38.6 ± 0.7	38.4 ± 0.9	n.s.
Mean Levodopa dose administrated (mg)		284.4 ± 92.3	

Mean values ± SD. * $p < 0.05$, *** $p < 0.001$.

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