VISUAL EVOKED POTENTIALS IN PATIENTS WITH PARKINSON'S DISEASE

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SUMMARY : *Visual evoked potentials have been studied in many neurological disorders including Parkinson's disease after having been used in multiple sclerosis. In Parkinson's disease, characterized with the widespread deficiency of dopamine, prolongation of the P100 latency seems to be strictly related to dopamine insufficiency. But the exact localization of the lesion, whether it is due to retinal or anterior visual pathway impairment is still controversial. In this study the results with checkerboard pattern reversal stimulus were normal whereas the latencies with flash stimulus were found to be prolonged. We concluded that this finding was consistent with an impairment of the retinal function.*

Key Words : *Visual Evoked Potentials, Parkinson's Disease.*

INTRODUCTION

The measurement of the visually evoked cortical potential (VEP) is a widely used non-invasive technique for studying the visual system which has first been used as a reliable diagnostic tool in multiple sclerosis. The VEP latency delays detected in multiple sclerosis have been attributed to slowing of central conduction reflecting optic nerve demyelination. However, changes in the VEP have been found in a variety of other central nervous system disorders including Parkinson's disease (1, 2, 3, 6, 8, 10, 11, 12). Parkinson's disease, which is characterised with a widespread deficiency of dopamine in the central nervous system, is essentially a motor system disease (2). As there is neither involvement of visual pathways nor demyelinization, delayed VEP's have been attributed to dopamine insufficiency in multisynaptic visual system (1, 2, 3, 10, 11). On the other hand, dopamine is found in the interplexiform layer of the retina which makes it possible that retinal dopamine insufficiency can be responsible for the abnormality in the VEP (2, 10).

The present study deals with the analysis of VEP's evoked by flash and checkerboard pattern reversal (CBPR) stimulus in patients with Parkinson's disease, comparing them with the results of normal control cases at the same age group.

MATERIALS AND METHODS

Our sample consisted of 20 patients; 10 male and 10 female with Parkinson's disease who were under treatment with different combinations of levodopa, dopa decarboxylase inhibitors, anticholinergic drugs and bromocriptine. Their ages ranged from 55 to 80 (mean 64.6). Normal control subjects were 4 males and 6 females, ages ranging from 49 to 65 (mean 56.2). For each group the visual acuity was not less than 6/9 and there were no pathologies detected by the ophtalmoscopic examination. Recordings were made with Nihon Kohden Neuro-

pack II plus. Cases were all studied in the same dark room. For CBPR VEP's they were seated one meter apart from the monitor which consisted of 16 checks 4 cm square wide, changing from black to white with a frequency of 1/sec. For flash VEP's the distance between the generator and the eye was 30 cm. Stimulus with an intensity of 1, 2 joule was given every second. VEP's were recorded from each eye seperataly with surface electrodes, with the reference electrode placed on Fz and the active electrode placed on midline, 2 cm above the inion. Bandpass filters were set at 2-100 Hz. The analysis time was 500 msec and 256 sweeps were avaraged. P100 latency was accepted as the time interval between the stimulus and the peak of the major positive component. The average values of the left and right eyes of each subject in both Parkinsonian and normal control groups with CBPR and flash stimulus and the standart deviations were calculated. The results derived from the Parkinson and normal control groups with two different types of stimuli were compared with the Student-t test.

RESULTS

In both Parkinsonian and normal control groups, subjects entered the analysis with one eye, namely the one characterized by the longer latency for the two types of stimuli. In the patient group, latencies of the P100 ranged from 94 to 120 msec (mean 100.8 sec, SD : 7.93) with the CBPR stimulus. In the normal group it was 92 to 106 msec with a mean of 98.4 msec (SD : 3.86). When they were compared with the t-test no meaningful difference obtained between the patients and the normal cases (t = 0.137).

With the flash stimulus, P100 latency of the Parkinsonian patients ranged from 92 to 200 msec (mean 131.6 msec SD : 21.59). In normal controls it was 100 to 134 msec with a mean of 118.2 msec (SD : 10.76). When compared with the t-test there was a significant difference between the two groups (t=0.015).

The results have been summarized in Table 1.

P100 (msec)		P100 (msec)	
Parkinsonian patients		Normal control subjects	
CBPR-VEP	Flash-VEP	CBPR-VEP	Flash-VEP
94	115	96	124
100	130	98	112
102	140	92	100
106	122	102	106
120	140	100	110
90	140	96	120
100	125	98	134
100	125	96	128
90	102	100	126
88	92	106	122
104	115		
100	138		
110	150		
106	125		
110	150		
90	128		
100	130		
106	200		
100	135		
100	130		

Table 1 : P100 latencies recorded from the Parkinsonian patients and the normal control subjects with CBPR and flash stimuli.

DISCUSSION

Different results have been reported from VEP studies in Parkinson's disease. Independent studies reporting prolongation of the P100 latency in more than 50 % of the patients can be found (2, 3, 8, 12) whereas there are others reporting normal values (4, 10, 13). Different stimulating techniques have been used in these different studies. Flash, CBPR and grating VEP's recorded with different stimulus frequencies have been studied (2, 3, 6, 10, 11, 13). Electroretinographic (ERG) studies are also present (3, 5, 10). Because of the variability of results, VEP changes in Parkinson's disease is still of interest.

In our study flash stimulus has been compared with the CBPR stimulus.

With the CBPR stimulus, prolongation of the P100 latency in parkinsonian patients has been reported (1, 3, 6) as well as normal results (10, 12). Some authors believe grating VEP's to be more sensitive in detecting abnormalities (12). The explanation for this is not so clear but it is speculated that the processing of visual information related to checks and gratings corresponds to different functional organisation (12). However grating VEP's recorded with different spatial frequency of stimulation have given different results; higher frequencies being responsible for the VEP delays in Parkinson's disease (11).

In our study CBPR stimulus reversing with a frequency of 1/sec detected no VEP delays in Parkinsonian patients when compared with the age matched controls.

There are only a few studies concerning flash VEP's in Parkinson's disease (9, 13). One has failed to detect any abnormalities (13). The other dealing with hemiparkinsonism has demonstrated VEP delays recorded from the affected hemisphere (9).

In our study using flash stimulus with a frequency of 1/sec, prolongation of the P100 latency has been detected in parkinsonian patients when compared with the normal subjects.

Despite controversial results gathered from different studies using different stimulation techniques, existence of VEP abnormalities in Parkinson's disease is quite obvious, which is known to be due to dopamine insufficiency. The reduction of the latencies after dopamine precursor therapy can be shown as a proof of this (1, 6, 11). However the exact site of the lesion is still obscure. Some authors believe that dopaminergic neurons found in the interplexiform layer of the retina can be damaged by the disease process and the retinal lesion can be responsible for VEP delays (10). In experimentally dopamine depleted rats VEP following direct optic nerve stimulation has been found of normal latency whereas the flash evoked potential recorded directly from the optic nerve has been delayed; indicating that the abnormality responsible for the delay in VEP to a flash stimulus has been situated anterior to the optic nerve, most probably in the retina (10). However ERGs used to evaluate retinal dysfunction have given controversial results. One study has shown normal VEP's with the CBPR stimulus when pattern ERG has been abnormal and this has been interpreted as a proof of the retinal lesion (10). In another study VEP's have been found to be affected more than pattern ERGs and this has been accepted as a dysfunction of the central visual pathway, relatively independent of the abnormalities at the retinal level (3).

In our study CBPR VEP's, known to be mainly recorded from the macular fibers, have shown no abnormalities whereas flash VEP's recorded from the peripheral retina have been delayed in parkinsonian patients when compared with the normal subjects. This made us believe that the lesion could be at the retinal level. If the ERGs of the same patients had been recorded, it would have been easier to make further comments on this subject. So the next step should be studying simultaneous VEPs and ERGs to compare the results.

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