Quantitative Analysis of Ocular Movements in Parkinson’s Disease

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In the present study, oculomotor abnormalities were investigated in 24 patients with idiopathic Parkinson’s disease. Pursuit gain, slow and fast phase velocity of optokinetic nystagmus (OKN), saccade latency, amplitude and velocity, as well as vestibulo-ocular reflex (VOR) gain were quantitatively analyzed. Twenty-one out of 24 patients showed some abnormalities in their ocular movement. A reduction in pursuit gain, impaired OKN and a significantly increased saccade latency were observed in 11 patients. In 12 patients, hypometric saccade was seen, while a decrease in saccade velocity was observed in 10 patients. All patients registered normal values for VOR gain. Only one patient who had impaired smooth pursuit and decreased saccade velocity before treatment with a dopaminergic drug showed a remarkable improvement in these ocular abnormalities after treatment. These results suggest the possibility of dopaminergic control on these ocular movements.

INTRODUCTION

Parkinson’s disease, a syndrome of disordered motor performance, involves the basal ganglia. Recent studies have demonstrated the presence of various oculomotor abnormalities (1-6), but these data are contradictory in several aspects.

In the present study, a quantitative analysis of various ocular movements was performed using a computer system in 24 patients with idiopathic Parkinson’s disease to define the exact role of ocular movements in basal ganglia.

MATERIAL AND METHODS

Twenty-four patients (14 females and 10 males aged from 48 to 79, mean age 65 years) with a diagnosis of mildly affected idiopathic Parkinson’s disease were selected for the present study. The duration of the disease ranged from 0.5 to 13 years. Although two patients had never received dopaminergic drugs, the other 22 patients had been treated with such drugs at the time of EOG tests. For 3 of these 22 patients the drug administration was stopped at least 48 h before the EOG tests, and the EOG tests were carried out again after dopaminergic drugs had been administrated for two weeks. Ten normal subjects aged from 50 to 70 years, without auditory and vestibular abnormalities, were tested as controls.

The neuro-otological tests in this series included saccadic eye movement, smooth pursuit, optokinetic nystagmus (OKN) and vestibulo-ocular reflex (VOR). For smooth pursuit, the patients were asked to track a small laser spot moving in a sinusoidal pattern at a frequency of 0.3 Hz and a peak-to-peak amplitude of 20°. OKN was induced by stripes rotating at an angular acceleration of 1°/s² and an angular velocity between 0 and 100°/s. Saccades were elicited by a small laser spot moving step-wise with random amplitudes between 10 and 50°, and random jump intervals. VOR was induced by a chair rotating in a sinusoidal fashion at a frequency of 0.25 Hz and a peak-to-peak amplitude of 60°.

Horizontal eye movements were recorded with a DC-EOG. The analog data obtained
from the DC-EOG was digitized at a rate of 50 Hz for smooth pursuit and VOR, or 200 Hz for the other tests through a 12-bit analog-digital converter. Based on these data, pursuit gain, both slow phase velocity (spv) and fast phase velocity (fpv) of OKN, saccade latency, peak velocity and amplitude, and VOR gain were quantitatively calculated with a computer (MASSCOMP) system. In particular, saccade velocity and fpv-OKN were evaluated with the following exponential equation:

\[ \text{Velocity} = K (1 - \exp (-\text{Amplitude}/L)) \]

in which \( K \) and \( L \) are constant.

RESULTS

Twenty-one patients showed some abnormalities in their ocular movements and only 3 patients had normal ocular movements. Pursuit gain, spv-OKN, saccade latency, saccade accuracy and saccade velocity were significantly altered in Parkinsonian patients (Table I). In 11 patients, the values of pursuit gain were reduced less than 0.90 and revealed impaired OKN as compared with normal controls. Seven patients showed a decrease in fpv-OKN at an amplitude of 40°, and in 10 patients a decrease in the saccade velocity at an amplitude of 40° was seen. In 11 patients the saccade latency was increased, and 12 patients showed hypometric saccade. However, all patients had normal values of VOR gain.

In 2 of the 3 patients who were tested before and after the treatment, the dopaminergic drugs had no effect on ocular movement. On the other hand, the remaining patient showed significant improvement in the smooth pursuit and saccade velocity after the treatment (Fig. 1). However, in this case, the saccade latency did not change, either before or after treatment.

DISCUSSION

In an attempt to define the nature of the oculomotor abnormalities accompanying idiopathic Parkinson’s disease, we performed a quantitative analysis of ocular movement using a computer system. Previously published reports have suggested that Parkinsonian patients show a high incidence of oculomotor abnormalities (1–6), and that the most common abnormalities include prolongation of the saccade latency (1–5), hypometric saccade (1, 2, 5, 6) and impaired smooth pursuit (1, 3, 4, 5). In the present study, all the patients except 3 had some oculomotor abnormalities. Eleven patients showed prolongation of the saccade

Table I. Comparison of EOG data between Parkinsonian patients and controls

<table>
<thead>
<tr>
<th></th>
<th>Controls (n=10)</th>
<th>Parkinsonians (n=24)</th>
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<tbody>
<tr>
<td>Pursuit gain</td>
<td>0.97±0.07</td>
<td>0.88±0.24**</td>
</tr>
<tr>
<td>OKN: OAL (deg/s)</td>
<td>69.6±29.4</td>
<td>39.5±37.8**</td>
</tr>
<tr>
<td>Fast-phase velocity (deg/s)</td>
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<tr>
<td>Amplitude 10 deg</td>
<td>208.8±44.0</td>
<td>193.8±37.4*</td>
</tr>
<tr>
<td>Amplitude 40 deg</td>
<td>409.3±98.8</td>
<td>361.9±132.8*</td>
</tr>
<tr>
<td>Saccade latency (ms)</td>
<td>224.8±47.4</td>
<td>267.7±87.4**</td>
</tr>
<tr>
<td>Accuracy (%)</td>
<td>98.2±10.4</td>
<td>86.7±10.4**</td>
</tr>
<tr>
<td>Velocity (deg/s)</td>
<td></td>
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<tr>
<td>Amplitude 10 deg</td>
<td>200.7±39.4</td>
<td>201.0±35.2</td>
</tr>
<tr>
<td>Amplitude 40 deg</td>
<td>462.8±76.8</td>
<td>414.9±134.8**</td>
</tr>
</tbody>
</table>

** \( p<0.01 \), * \( p<0.02 \).
latency, and 12 patients demonstrated hypometric saccade. Furthermore, both impaired smooth pursuit and spv-OKN were noted in 11 patients. These findings are consistent with those of previously published reports (1–6). Therefore, it can be assumed that these findings are characteristic of Parkinson’s disease.

According to our results, both the saccade peak velocity and fpv-OKN for 10 patients were significantly reduced as compared with those of the normal subjects. Some authors have reported that the saccade velocity was decreased (1, 4, 5), while others did not find it to do so (2, 3, 6). Rascol et al. (1) pointed out that a wide age range of the controls or subjects investigated by some authors reporting normal values for the saccade velocity could have resulted in such oculomotor variability that differences between the means fall short of statistical significance. In any case, further investigations are in order.

Some authors have previously reported the efficacy of L-dopa for oculomotor abnor-
malities (1, 2). In these results, the saccade accuracy was significantly improved and both the smooth pursuit and saccade latency were moderately improved. Although we had only one case who was tested before and after the treatment with dopaminergic drugs, this case showed an improvement in oculomotor abnormalities. Hence, our results suggest the possibility of dopaminergic control of these ocular movements.

ACKNOWLEDGEMENT

We would like to thank Dr Hideo Shirai, Department of Neurology, Yamagata Prefectural Hospital for allowing us to examine his patients for this study.

REFERENCES


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