Effects of Levodopa on Finger and Articulatory Movements in Parkinson's Disease

Michèle Gentil, Claire-Lise Tournier, and Pierre Pollak

Centre Hospitalier Universitaire, Clinique Neurologique, BP 217, 38043 Grenoble cedex 9 (France)

Abstract: The objective of this study was to determine the efficiency of levodopa on finger and articulatory movements in Parkinson's disease (PD). The production of forces generated by the upper and lower lips, tongue, right and left forefingers was estimated under two conditions 1) without levodopa, 2) with levodopa, in 14 patients suffering from PD. Motor disability was also assessed in both conditions. Moreover, 14 healthy subjects served as control subjects. The beneficial effect of levodopa on finger movements was observed as indicated by an improvement in motor scores and production of forces. In contrast, the production of forces of articulatory organs was either not improved or aggravated by levodopa. These results suggest that cerebral non dopaminergic lesions are probably responsible for the impairments of parkinsonian speech.

INTRODUCTION

The efficacy of levodopa is not uniform on all parkinsonian features. Akinesia, tremor and rigidity are in general improved by levodopa while axial symptoms and in particular dysarthria do not respond well to levodopa (1). The variable responsiveness of parkinsonian motor symptoms to levodopa raises the possibility that certain symptoms are closely related to dopaminergic lesions, and others mainly involve non dopaminergic system lesions. To study this hypothesis the production of articulatory and forefinger forces was examined as well as global motor function in patients with fluctuating PD. The sensitivity of the orofacial and limb systems to levodopa would suggest the involvement of dopaminergic lesions.

METHOD

Fourteen patients (mean age 58 ± 7 years) and fourteen healthy subjects (56 ± 7 years) participated in this study. All patients were treated with levodopa. The mean duration of their symptoms at the time of examination was 9 ± 3 years. Unmedicated patient’s speech impairment was classified as "moderately impaired" with regard to perceived speech intelligibility. Antiparkinsonian medications were withheld overnight and the initial tests started after at least 10 hours medication free interval. Immediately before the first tests, overall motor disability of patients was assessed by means of the Unified Parkinson's disease Rating Scale (UPDRS) motor examination. After a first series of force tasks without levodopa, Madopar 125 was given at the dose which usually allowed the patients to turn "on". Ten minutes after the occurrence of a stable improvement of at least 30%, the same motor tests and force tasks as previously were carried out.

Load-sensitive cantilevers (Neuro Logic Incorporated, Bloomington/Indiana, USA) were used to sample compression forces generated by the upper and lower lips, and tongue. The cantilever slid along a jaw yoke that was encapsulated in a moldable dental impression block and placed between the molars. For testing the forefinger forces, the transducer was installed on the right and on the left of a tabletop which was fixed to a dental arm-chair. The subject rested his arm on the tabletop, with his forefinger extended on the strain gage device and his other fingers loosely curled up. A two-channel storage oscilloscope provided visual feedback. The subject was given the instruction to generate forces from baseline "as rapidly and as accurately as possible " in response to the target signal which appeared on the screen. The rapid phase of force increase to reach the target was followed by a stabilization to the target for the duration of the hold phase. The target force levels used in the present study included 0.25, 0.5, 1 and 2 newtons (N) corresponding to forces presumably involved in speech production. For each structure, 10 consecutive contractions were obtained at each of the 4 target force levels. This study was approved by the Grenoble University Hospital Ethics Committee.

RESULTS

Motor Disability: The mean motor scores of unmedicated and medicated patients were 35.5 and 14.6 respectively. So, motor performance of medicated patients was increased by 59%. On the contrary, speech impairment assessed by UPDRS item 18 was not improved.

Mean Reaction Times: Reaction time was defined as the time interval from the time the target signal appeared on the screen until the force had reached 10% of peak force. Table 1 shows the mean reaction times and standard deviations across force levels for articulatory organs and forefingers in unmedicated and medicated patients as well as in control
subjects. The mean reaction time of control subjects was always shorter than that of patients. The mean reaction time in producing articulatory forces was significantly different ($F = 11.85, p < 0.01$) and greater for medicated than unmedicated patients. Concerning forefingers, no significant difference was found between both medicated and unmedicated patients. Moreover, a greater variability as indicated by greater standard deviations was observed for patients in comparison with control subjects. This variability was greater for articulatory organs than for forefingers in both control subjects and patients.

**TABLE 1. Mean reaction times and standard deviations**

<table>
<thead>
<tr>
<th>Structures</th>
<th>Control Subjects</th>
<th>Unmedicated patients</th>
<th>Medicated patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Articulatory organs</td>
<td>561 (182)</td>
<td>611 (279)</td>
<td>645 (296)</td>
</tr>
<tr>
<td>Forefingers</td>
<td>586 (145)</td>
<td>612 (221)</td>
<td>605 (178)</td>
</tr>
</tbody>
</table>

Standard deviations are in brackets. Values in milliseconds

**Peak Rate of Force Change:** Peak rate of force change was taken from the maxima of the first derivative of force ($df/dt$ max). In general, for both subjects and patients we remarked an increase in the peak rate of force change with the target level, and greater values for articulatory organs. Whatever structure, the peak rate of force change of control subjects was greater than that of patients. Concerning the latter, there was no significant difference for articulatory organs between medicated and unmedicated patients, but significant differences for forefingers, medicated patients having higher values of the peak rate of force change ($p < 0.05$). Table 2 presents values of the peak rate of force change at 0.25 N and 2 N target level for articulatory organs and forefingers in unmedicated and medicated patients.

**TABLE 2. Peak rate of force change at 0.25 N and 2 N target level in unmedicated and medicated patients**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Target 0.25 N</th>
<th>Target 2 N</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Articulatory organs</td>
<td>Forefingers</td>
</tr>
<tr>
<td>Unmedicated</td>
<td>3.40</td>
<td>1.98</td>
</tr>
<tr>
<td>Medicated</td>
<td>3.17</td>
<td>2.94</td>
</tr>
</tbody>
</table>

Values in N/s

**Hold Phase:** Control subjects were very close to the target, and this precision was steady, as indicated by low standard deviations. In patients, forefingers were more precise than articulatory organs, namely significant differences in precision were revealed by two sample t-test between articulatory organs and forefingers in medicated as well as in unmedicated patients, at any target level. Moreover, medicated patients held the force closer to the target level than unmedicated patients but with a greater variability.

**CONCLUSION**

In concert with clinical motor disability, levodopa improved forefinger force production on the whole, and mostly we noted an increase in rate of force development and improvement in precision of the hold phase. This agrees with other works showing that medication influences both strength and rate of force development (2). On the opposite, the orofacial motor system did not respond to levodopa in the same way, dysarthria was not improved as well as the most of parameters relative to articulatory organ forces. This supports the hypothesis that orofacial system deficits in PD involve other mechanisms and could result principally from non dopaminergic lesions. Attempts to correlate the motor symptomatology to treatment should allow to gain a greater knowledge of the pathology of the motor system.

**REFERENCES**