Dopa-sensitive and Dopa-resistant gait parameters in Parkinson’s disease

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Summary

Quantitative analysis of gait was performed in 20 parkinsonians before and 1 h after the acute administration of L-Dopa in order to discriminate between the Dopa-sensitive and the Dopa-resistant kinematic gait parameters. The stride length and the kinematic parameters (swing velocity, peak velocity) related to the energy were Dopa-sensitive. The improvement of the bent forward posture by L-Dopa may explain the stride length increase. Temporal parameters (stride and swing duration, stride duration variability), related to rhythm, were Dopa-resistant. Experimental data argue for the importance of force control in maintaining the posture. The stride length variability, possibly related to the variability of force production shown to exist in parkinsonians was not significantly improved by L-Dopa. In Parkinson’s disease different hypotheses might explain the inexorable aggravation of gait disorders along the course of the disease: (1) an advancing disorder of coordination between postural control and locomotion, (2) if some gait parameters like stride length and kinematic parameters are Dopa-sensitive, the others are Dopa-resistant and thus may involve other mechanisms than dopamine deficiency.

Introduction

The clinical and therapeutical evaluation in Parkinson’s disease requires reliable methods. Most simple tests of motor performance in parkinsonians give variable and often good results hiding the marked motor handicap in such patients (Johnels et al. 1989). This could be related to the fact that complex motor behaviors are more altered than simple behaviours in the disorders of the basal ganglia (Schwab et al. 1954; Traub et al. 1980).

The quantitative analysis of gait is a reliable method with good reproducibility and well correlated to the clinical motor observation of the patient. However, few quantitative studies of the locomotion in parkinsonians have been performed up to now (Knutsson 1972; Murray et al. 1979; Stern et al. 1983; Blin et al. 1990a) and the effect of L-Dopa on gait parameters is poorly acknowledged (Knutsson and Martensson 1971).

Studies on long term treatment in Parkinson’s disease have shown that the akinesia, rigidity and tremor improvement with L-Dopa seem to be constant all along the course of the disease (Bonnet et al. 1987). Reversely, cognitive disorders (Pillon et al. 1989), falls (Koller et al. 1989), freezing (Weinrich et al. 1988) and posture reflexes impairment (Klawans 1986) do not remain Dopa-sensitive. The features which continue to respond to L-Dopa are thought to be purely dopaminergic while the latter may involve other mechanisms than dopamine deficiency (Klawans 1986; Bonnet et al. 1987).

Globally, gait disorders are thought to be inexorably aggravated along the course of the disease (Barbeau and Roy 1976; Klawans 1986; Bonnet et al. 1989).

The aim of this study was (1) to study, by means of a quantitative analysis, the modifications of the various temporal and kinematic parameters of locomotion induced by L-Dopa in parkinsonians and (2) to discriminate between the Dopa-sensitive and the Dopa-resistant gait parameters.

Patients and methods

Twenty parkinsonian patients, 11 women and 9 men between the age of 50 and 85 years (average age: 69.2), 1.50–1.85 m tall (average height: 1.71), weighing between
37 and 88 kg (average weight: 64.5 kg), were included in the study. All were referred to the department of neurology for predominant gait disorder. The diagnosis of idiopathic Parkinson's disease was confirmed by clinical and neurological examination. The course of the disease ranged from 1 to 17 years and L-Dopa had been administered since the onset. According to the Hoehn and Yahr classification (1967) there were 4 patients in stage I, 3 patients in stage II, 6 patients in stage III, and 7 patients in stage IV. There were "freezing" episodes in 5 patients, dystonia in 3, dyskinesia in 4 and "on-off" episodes in 2.

Procedure and recording
Recording was carried out in 2 sessions: first, in the morning in patients fasting overnight, before administration of L-Dopa and without treatment for at least 12 h, and second, 1 h after the intake of 200 mg L-Dopa + 50 mg benzerazide (Modopar 250*) according to Esteguy's protocol (1985). Each patient was his own control. At each session the patient was asked to walk to a point 10 meters away (active walking). The circumstance of the registration remained constant throughout the study. The same experimenter always gave the same instructions.

The locomotor parameters were automatically recorded by means of an apparatus designed by Bessou et al. (1989). This device measures the longitudinal displacement of both feet during locomotion, and functions as follows. Each foot is attached to a separate string which is pulled as the patient walks. The string unwinds to the required length by means of a gearing-down pulley system. Movement is recorded in the form of an electric signal via a pulley linked to a potentiometer. Data were recorded at 75 Hz, then filtered using a FIR filter with a 33-point window and cut-off at 10 Hz. The calibration of the device defines the volt/meter coefficient to be used in computing spatial and velocity data. This apparatus does not necessitate any special walkway or specific illumination, and can be used to record natural locomotion without any discomfort for the subjects. Furthermore, all patients were familiar with the apparatus. The recording sequence (10 meters) allows one to evaluate both the intra- and interindividual variability (the fluctuation in performances of Parkinson patients). In fact, the advantages of this apparatus have been well demonstrated and this device has been previously validated (Bessou et al. 1989; Blin et al. 1990a,b,c).

Spatio-temporal data (stride length, stride, stance, swing and double support duration) and kinematic data (sequence velocity, swing velocity and peak velocity) on locomotion were calculated. We also computed the variability index as defined by Gabell and Nayak (1984) for the stride length and stride duration. This index (coefficient of variation) allows the assessment of intra-individual spread in the sequence. With increased velocity after L-Dopa administration, we also studied the respective role of the increase in the stride length and of the reduced stride duration. To modify the gait velocity, two complementary strategies are available: either increase the stride length or decrease the stride duration.

Statistical analysis
The effect of L-Dopa on the different parameters was evaluated using the non-parametric Wilcoxon signed-rank test. Correlations between demographic data and the L-Dopa effect were evaluated using the Spearman rank correlation coefficient (Siegel 1956).

### Table 1

<table>
<thead>
<tr>
<th>Parkinsonians</th>
<th>Before L-Dopa (n = 20)</th>
<th>After L-Dopa (n = 20)</th>
<th>Before vs. after L-Dopa</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Velocity (m/sec)</td>
<td>0.45</td>
<td>0.2</td>
<td>0.57</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>0.57</td>
<td>0.27</td>
<td>0.69</td>
</tr>
<tr>
<td>Stride duration (sec)</td>
<td>1.28</td>
<td>0.2</td>
<td>1.24</td>
</tr>
<tr>
<td>Swing duration (sec)</td>
<td>0.4</td>
<td>0.08</td>
<td>0.41</td>
</tr>
<tr>
<td>Stance duration (sec)</td>
<td>0.88</td>
<td>0.16</td>
<td>0.78</td>
</tr>
<tr>
<td>Swing velocity (m/sec)</td>
<td>1.38</td>
<td>0.45</td>
<td>1.66</td>
</tr>
<tr>
<td>Peak velocity</td>
<td>2.02</td>
<td>0.68</td>
<td>2.42</td>
</tr>
<tr>
<td>Double support duration (sec)</td>
<td>0.23</td>
<td>0.08</td>
<td>0.19</td>
</tr>
<tr>
<td>Relative double support duration (sec)</td>
<td>36.4</td>
<td>9.8</td>
<td>29.6</td>
</tr>
<tr>
<td>Stride duration variability</td>
<td>5.48</td>
<td>5.2</td>
<td>4.61</td>
</tr>
<tr>
<td>Stride length variability</td>
<td>7.62</td>
<td>5.45</td>
<td>6.57</td>
</tr>
</tbody>
</table>
Results

The various spatio-temporal and kinematic parameters are reported in Table 1 for patients with active walking before and after L-Dopa intake. In all patients there was a significant improvement of the velocity with L-Dopa (average increase of 38%). Spatial and kinematic parameters improved after L-Dopa administration. Reversely, stride and swing durations were not significantly modified. The analysis of the velocity variations showed that the improvement (average improvement of 38%) corresponds mainly to the increase of the stride length (95%) and with a lesser extent to a decrease of stride duration (5%). The variability of stride length and duration were not significantly modified by L-Dopa.

No correlation was found between L-Dopa effect and the patient’s age, disease duration or L-Dopa treatment duration.

Discussion

This study deals with a “L-Dopa test” (Esteguy et al. 1985) on the parkinsonian gait and allows us to discriminate between the Dopa-sensitive and the Dopa-resistant kinematic gait parameters.

Dopa-sensitive parameters

The efficiency of L-Dopa is predominant on the stride length. Several hypotheses may account for these findings.

In Parkinson’s disease, the motor programs are preserved in the selection of muscles and in the timing of their activation. Reversely, the production of the strength required is altered (Marsden 1982, 1984). This is particularly displayed in ballistic movements suggesting the concept of “activation (energizing) impairment” (Hallett and Khosbin, 1980). The parameter related to the energy (e.g., the stride length) is Dopa-sensitive and the improvement by L-Dopa may lead to an increased swing velocity and peak velocity. Parkinson patients are hypokinetic and hypometric. L-Dopa improves stride length and as it does not significantly modify stride duration, it increases velocity parameters (swing velocity and peak velocity). A plausible explanation of the effect of L-Dopa may be a more powerful activation of each muscle group.

Parkinson’s disease causes a typical, bent posture: “propensity to bend the trunk forward” (Parkinson 1817). This bent forward posture can be, at least partially, improved by L-Dopa. Knuttson and Martensson (1971) gave an obvious illustration of this correction after L-Dopa administration. We suggest that this effect, through biomechanical modifications, may explain the stride length increasing. However, this hypothesis requires quantitative confirmation.

Dopa-resistant parameters

Temporal parameters (stride and swing duration, stride duration variability), related to rhythm, were Dopa-resistant.

Posture reflexes (Scholz et al. 1987; Dietz et al. 1988) and anticipation reflexes (Traub et al. 1988) are impaired in Parkinson disease. All these disorders cannot be induced by neuroleptics in healthy volunteers. This argument suggests their independence to dopaminergic systems (Dietz et al. 1988). Experimental data argue for the importance of force control in maintaining the postural reflexes and locomotion (Mori 1987). The variability of strength has been suggested to be one of the factors of the motor disorder observed in parkinsonians (Sheridan et al. 1987). Strength control is variable in parkinsonians (Stelmach et al. 1988, 1989; Muller and Abbs 1990). The stride length variability, possibly related to this variability of force production was not significantly improved by L-Dopa. Thus, the locomotor parameter representative of the force control is Dopa-resistant.

In Parkinson’s disease different hypotheses might explain the inexorable aggravation of gait disorders along the course of the disease: (1) an advancing disorder of coordination between postural control and locomotion (Johnels et al. 1989), (2) if some gait parameters like stride length and kinematic parameters are Dopa-sensitive, the others are Dopa-resistant and thus may involve mechanisms other than dopamine deficiency.

References


