Differences in the EMG pattern of leg muscle activation during locomotion in Parkinson’s disease

Giovanni Albani
Giorgio Sandrini
Gabriella König
Chantal Martin-Soelch
Alessandro Mauro
Riccardo Pignatti
Volker Dietz
Klaus L. Leenders

Department of Neurology, IRCCS Italian Auxology Centre, Piancavallo (Verbania) and Department of Neuroscience, University of Turin, Italy
a IRCCS C. Mondino Institute of Neurology, University of Pavia, Italy
b Institute of Neurology, University of Zürich, Switzerland
c Institute of Psychology, Basel, Switzerland
d Swiss Paraplegic Centrum, Balgrist Hospital, University of Zürich, Switzerland
e Department of Neurology, University of Groningen, The Netherlands

Reprint requests to: Dr Giovanni Albani
Department of Neurology, Istituto Auxologico Italiano, Piancavallo (Verbania), Italy
E-mail: giannialbani@libero.it

Accepted for publication: May 25, 2003

Summary

In this pilot study, EMG patterns of leg muscle activation were studied in five parkinsonian patients with (B1) and five without (B2) freezing. Gastrocnemius medialis (GM) and tibialis anterior (TA) activity was analysed, by means of surface electromyography (EMG), during treadmill walking at two different belt speeds.

Both groups showed reduced GM activity and an overactive TA at the lower speed compared with controls. Upon increasing the speed, the B2 patients showed a marked GM response (increment index 100%), while a moderate change was observed in the B1 group. Poor recruitment of the GM characterises parkinsonian gait in general; this pattern is much more marked in parkinsonian patients with freezing of gait, who show a loss of GM adaptation to variation of locomotion speed.

KEY WORDS: adaptation, disability, electromyography, freezing, gait, Parkinson’s disease.

Introduction

Loss of interlimb coordination, freezing and dynamic postural instability are the main manifestations of parkinsonian gait (1,2). Prolonged stance or double support phase (3), reduced lateral shift of the body mass over the stance limb (4), and decreased propulsive forces (5) are some of the main biomechanical observations. Gait analysis reveals a characteristic pattern of leg muscle activation in Parkinson’s disease (PD) – increased tibialis anterior (TA) activity during the swing phase and reduced amplitude of the electromyographic (EMG) response and poor modulation in the leg extensor muscles during the stance phase (6,7). Gait disorders in PD may be either prominent or, even in the advanced stages of the disease, mild. Certain clinical features are more likely to be associated with disturbed gait: rapid course of motor symptoms (8), poor response to dopaminergic therapy (9) and an infrequent presence of the tremor (10); disturbed gait also shows a positive association with sleep problems and with motor symptoms that started on the left side of the body (11).

The aim of this study was to evaluate whether the presence of freezing is correlated with changes in the EMG patterns of leg muscle activation during walking on a treadmill.

Materials and methods

In order to evaluate the adaptive response of agonist/antagonist muscles to an external stimulus (change in walking speed), this study focused, among the various parameters of gait (kinetic, kinematic, etc.), on EMG patterns of lower limb muscle activation.

Ten idiopathic PD patients, five with freezing (B1) and five without freezing (B2), were studied in comparison with seven normal subjects (mean age: 63 years, four males/three females) (Table I, over). The inclusion criteria for B1 patients were: a) presence of freezing; b) Hoehn & Yahr stage ≥3; c) history of falls; d) no evidence of dysautonomic involvement; e) absence of dystonia and dyskinesia; f) absence of dementia. The inclusion criteria for B2 patients were: a) Hoehn & Yahr stage <3; b) no history of falls; c) good response to levodopa (L-dopa). All patients presenting fluctuating motor performances were investigated during the “on” state. The inclusion criteria for B1 patients were: a) presence of freezing; b) Hoehn & Yahr stage ≥3; c) history of falls; d) no evidence of dysautonomic involvement; e) absence of dystonia and dyskinesia; f) absence of dementia. The inclusion criteria for B2 patients were: a) Hoehn & Yahr stage <3; b) no history of falls; c) good response to levodopa (L-dopa). All patients presenting fluctuating motor performances were investigated during the “on” state. Recordings were made while subjects were walking on a treadmill at belt speeds of 0.3 m/sec and 1.5 m/sec. One-minute recordings of walking in each speed condition were made, the impact of the right leg triggered the biomechanical and electrophysiological signals. The
various stride cycle parameters and EMG signals were calculated from the last twenty consecutive stride cycles of any given trial period. EMG signals were collected using surface electrodes placed over the left and right TA (TAL and TAR) and the medial head of the left and right gastrocnemius (GML and GMR). These muscles are very representative of the phases of the stride cycle and their EMG activity is very easily detected by surface electrodes, without any risk of cross-talk from adjacent muscles. The treadmill was placed over a force measuring platform, thus, the stance and swing phases of both legs could be determined on the basis of changes in the signal of the vertical vector of force, according to the presence (stance phase) or absence (swing phase) of foot pressure. The EMG and biomechanical records were amplified and, after rectification of EMG, transferred on-line to a computer system via an A/D converter sampling at 500 Hz.

Statistical analysis

Because of the small size of the group of subjects, we decided to use an analysis of variance with the exact H-test (non parametric test). Thereafter, a comparison of the groups was performed using the method of Conover, which is a non parametric test valid for small samples. For each speed condition, we analysed inter group differences in GM and TA activities in the agonist and antagonist phases of the stride cycle. To determine the index of increment (increment of activity of each muscle due to increased walking speed), we used the formula X-Y/(X+Y/2) X 100 (%), in which X and Y represent muscle activity at 1.5 m/sec and 0.3 m/sec, respectively.

Results

Walking at a belt speed of 0.3 m/sec

Our PD patients were found, in the stance phase, to show reduced activation of the GML (Fig. 1a), with the data of both the B1 (p<0.0001) and the B2 (p<0.0001) patients differing significantly from those of the controls (Fig. 1a). No differences were found between the B1 and B2 subjects. As regards the GMR, no significant differences emerged among the three groups (Fig. 1b). Conversely, in the swing phase, B1 patients presented greater TAL (p<0.0001) and TAR (p<0.0001) activity than controls. In the B2 patients, this pattern was found only in the TAL (p<0.0001), while TAR activation was not significantly different from that found in controls (Fig. 1c,d).

Walking at a belt speed of 1.5 m/sec

Reduced activity of the GM in parkinsonian patients versus controls was still evident when walking speed was increased, and involved both the GML (p<0.0001) and the GMR (p<0.0001) (Fig. 2a,b) in both groups of patients. However, the increment index (Table II) was different in the two groups: in the B1 patients, GM activity increased by only around fifty percent, as opposed to around one hundred percent in the B2 patients. In B1, TA activation at 1.5 m/sec did not differ significantly from that recorded at the lower speed, or from the level recorded in controls (Fig. 2c,d). The B2 patients showed a much more marked TA index increment than the B1 patients.

Table I - Clinical features of patients.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Duration PD (years)</th>
<th>Duration LD (years)</th>
<th>UPDRS (motor part)</th>
<th>H&amp;Y</th>
<th>Side more involved</th>
<th>Clinical form</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td>77</td>
<td>3</td>
<td>3</td>
<td>62</td>
<td>III</td>
<td>symmetrical</td>
<td>A-R</td>
</tr>
<tr>
<td>Case 2</td>
<td>71</td>
<td>11</td>
<td>10</td>
<td>38</td>
<td>III</td>
<td>left</td>
<td>A-R</td>
</tr>
<tr>
<td>Case 3</td>
<td>82</td>
<td>1</td>
<td>1</td>
<td>43</td>
<td>III</td>
<td>left</td>
<td>M</td>
</tr>
<tr>
<td>Case 4</td>
<td>66</td>
<td>15</td>
<td>10</td>
<td>74</td>
<td>IV</td>
<td>left</td>
<td>A-R</td>
</tr>
<tr>
<td>Case 5</td>
<td>68</td>
<td>14</td>
<td>13</td>
<td>49</td>
<td>IV</td>
<td>left</td>
<td>M</td>
</tr>
<tr>
<td>B2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td>66</td>
<td>7</td>
<td>4</td>
<td>24</td>
<td>II</td>
<td>symmetrical</td>
<td>M</td>
</tr>
<tr>
<td>Case 2</td>
<td>49</td>
<td>6</td>
<td>5</td>
<td>23</td>
<td>II</td>
<td>symmetrical</td>
<td>A-R</td>
</tr>
<tr>
<td>Case 3</td>
<td>70</td>
<td>2</td>
<td>1</td>
<td>19</td>
<td>II</td>
<td>symmetrical</td>
<td>A-R</td>
</tr>
<tr>
<td>Case 4</td>
<td>43</td>
<td>6</td>
<td>3</td>
<td>22</td>
<td>1.5</td>
<td>left</td>
<td>T</td>
</tr>
<tr>
<td>Case 5</td>
<td>46</td>
<td>4</td>
<td>3</td>
<td>36</td>
<td>2.5</td>
<td>left</td>
<td>A-R</td>
</tr>
</tbody>
</table>

Abbreviations: PD=Parkinson’s disease; B1/B2=with/without gait disorders; LD=levodopa; UPDRS=Unified Parkinson’s Disease Rating Scale; H&Y=Hoehn & Yahr stage; A-R=akinetic-rigid; T=tremor-dominant; M=mixed.
EMG patterns during locomotion in PD

Figure 1 - EMG activity of lower limbs at 0.3 m/sec.

Figure 2 - EMG activity of lower limbs at 1.5 m/sec.
Absence of co-contraction EMG pattern

Statistical analysis of the muscle activation values recorded during the antagonist phases of each muscle (swing phase for GM; stance phase for TA) revealed no significant difference between controls and B1/B2 patients. Indeed, in their antagonist phases, muscles were found to be similarly activated in all groups. Figure 3 (a,b), referring to one healthy subject, one B1 patient and one B2 patient, summarises the main findings of this study: reduced GM and enhanced TA activity versus controls in both speed conditions in B1, and only in B2 patients an increment of GM and TA activity upon increase of walking speed.

Discussion

Common patterns in B1 and B2

Instrumental gait parameters have sometimes been found to correlate with the clinical pictures of PD and parkinsonism. For example, some authors have demonstrated marked differences between the gait pattern of patients with vascular parkinsonism and that of patients with idiopathic PD (12). Compared with PD patients, patients with vascular parkinsonism have relatively preserved arm movements and show less flexion dystonic posture of the hip, knee, and trunk throughout the stride cycle.

The aim of this study was to identify electrophysiologically different patterns between parkinsonian patients with and without gait disorders by means of the EMG analysis of treadmill-induced walking.

First of all, our results confirm the presence of reduced GM activity during the stance phase in both B1 and B2 patients. This finding may be the result of impaired proprioceptive feedback from extensor load receptors (13). Facilitation of the gastrocnemius/soleus long latency stretch reflex at the end of the stance phase helps to compensate for ground irregularities and assists during the push-off phase (14). Its impairment may represent a basic distinction between normal subjects and PD patients, irrespective of the presence of a gait disorder. This weakness of extensor muscles with the increased activation of flexor muscles may explain the typical flexed posture of the parkinsonian patient.

Different patterns in B1 and B2

The new information emerging from this study concerns the difference in the response of B2 and B1 patients (marked and poor, respectively) to the increased treadmill speed. The parkinsonian patient with a gait disorder has lost the capacity to adapt to the variation of external stimuli, in our study represented by the increment of speed.

Parkinsonian patients with minor gait involvement showed a greater response, reflected in a higher increment of EMG activity (Table II).

TA overactivity could represent a compensatory response to the reduced GM activity that is no longer sufficient at higher locomotion speed. Other adaptive mechanisms, preserved in B2 patients, serve to ensure postural stability. From the clinical point of view these pathways are probably not dopaminergic, given the failure of L-dopa to improve temporal gait parameters (2).

The integration of sensory information at cortical level may play an important role in the control of this adaptive mechanism (15). Thus, parkinsonian patients with gait disorder behave like infants who do not yet process afferent inputs for integration with programmed leg muscle EMG patterns which are needed to achieve modulation and adaptation to the actual needs (16). Again considering this similarity of gait between parkinsonian patients and infants, it has also been postulated that an immature pattern may reappear in PD as a result of deficits in the neural circuits controlling locomotion (17). This pattern could reflect incorrect computation of the required force or defective memory for computed forces (18).

Overactivation of TA

The statistical analysis of the activity of each muscle in its agonist and antagonist phase of each step cycle did not reveal any significant differences among groups. The co-contraction phenomenon was not present because the TA was activated only in the swing phase, as in controls, and overactivity was not prolonged to the stance phase.

These data are in agreement with those of other studies, and indicate the presence of normal alpha/gamma co-activation in PD patients (19,20).

<table>
<thead>
<tr>
<th></th>
<th>B1 (Mean ± SD)</th>
<th>B2 (Mean ± SD)</th>
<th>Controls (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GML</td>
<td>62.5±31.0</td>
<td>106.3±52.8</td>
<td>81.6±28.2</td>
</tr>
<tr>
<td>GMR</td>
<td>43.3±22.2</td>
<td>91.3±38.0</td>
<td>94.2±55.4</td>
</tr>
<tr>
<td>TAL</td>
<td>9.8±16.3</td>
<td>28.7±30.8</td>
<td>84.9±81.6</td>
</tr>
<tr>
<td>TAR</td>
<td>0.5±30.6</td>
<td>28.0±19.8</td>
<td>71.5±76.9</td>
</tr>
</tbody>
</table>

* Increment of muscle activity upon increasing the speed of walking.

Abbreviations: GML=gastrocnemius medialis, left; GMR=gastrocnemius medialis, right; TAL=tibialis anterior, left; TAR=tibialis anterior, right; B1=parkinsonian with gait disorder; B2=parkinsonian without gait disorder.
EMG patterns during locomotion in PD

While these data need to be confirmed in more extensive studies, they suggest that the automatic spinal locomotor centre (21) is efficient in PD and allows the rhythm of the physiological phases of the gait cycle to be preserved, even though the proprioceptive system is impaired.

In conclusion, the loss of adaptation of GM and TA muscle activity to a new locomotion speed is a marker of the severity of the gait disability. It is possible that this difference in locomotor response to an external cue could contribute to the neurophysiological definition of PD patients with gait disturbances.

References


21. Grillner S. Interaction between sensory signals and central networks controlling locomotion in lamprey, dogfish and cat. In: Grillner S, Stein PSG, Stuart DG, Forssberg...
EMG patterns during locomotion in PD