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Levodopa-induced alterations in speech rate in advanced Parkinson's disease

Miet De Letter[1], Patrick Santens[2], Marc De Bodt[3], Paul Boon[2] and John Van Borsel[1]

[1]Department of Oto-Rhino-Laryngology, Center for Speech and Hearing Disorders, Gent University Hospital ; [2]Department of Neurology, Gent University Hospital ; [3]Department of Oto-Rhino-Laryngology, Center for Communication Disorders, Antwerp University Hospital

Abstract

The effect of speech rate on overall intelligibility in Parkinson's Disease (PD) is still a matter of debate. A comparison of the results of previous studies on speech rate in PD is hampered by methodological differences. In this study, we evaluated the effects of levodopa on speech rate and on its variability in a standardized reading task. Twenty-five patients were studied before and after levodopa administration while reading a standardized text. In accordance with previous studies, no significant improvement of speech rate was found. Rather an increased variability of speech rate in the on-medication state could be demonstrated. It is possible that this increased variability may be the consequence of respiratory deficits due to levodopa-induced dyskinesia or an increase of dysfluencies. However, the effects of defective auditory feedback and disturbed executive function cannot be ruled out.

Key words : Speech rate ; Parkinson ; levodopa ; reading ; dyskinesia ; dysfluency.

Introduction

The speech deficits associated with Parkinson disease (PD) include monopitch, monoloudness, hypokinetic articulation, voice quality deficits, short rushes of speech and a variable speech rate (Darley, Aronson and Brown, 1969). Speech rate is generally expressed as the number of syllables during a defined time period. It is affected by a number of factors such as segment duration, variability between the duration of utterances, variability between the duration of consonants and vowels (articulation rate) and the pause time between the different utterances. It is generally accepted that speech timing is constrained by the physiology of the speech production system. The basal ganglia are supposed to regulate temporospatial aspects at the level of the motor cortex (Brown *et al.*, 1998, Goberman *et al.*, 2005). Moreover, psychomotor tonus and affect have an important influence on speech rate, which can be observed for instance in an increased speech rate during agitation (Braun *et al.*, 2004). Therefore structural, motivational and neurogenic components are implicated in the variation of speech rate.

The influence of dopaminergic treatment on speech rate in PD was studied in a limited number of reports. Intuitively, one would presume speech rate to increase following levodopa administration, due to decrease of akinesia and chest wall rigidity (Solomon and Hixon, 1993). However, the results of the initial reports were conflicting, probably as a consequence of the subjective nature of the evaluation. Wolfe *et al.* (1975) reported no speech rate changes after levodopa intake, while Rigrodski and Morrison (1970) noted significant improvement of "speech rate adequacy" after administration of levodopa. These reports were also based on single measurements of speech rate in the off- and on-condition. Fluctuations in speech rate in both conditions were not taken into account.

The effects of medical treatment on speech rate are of utmost importance to the speech therapist. If a clear effect can be demonstrated, this would influence diagnostic evaluation as well as the outline of the speech therapy program. Indeed, moments of optimal speech rate could be used to increase the awareness of the patient to these "reference episodes" and to urge the patient to integrate this adaptive speech rate in spontaneous conversation. As for other aspects of speech, external cueing will be necessary to obtain this integration.

This study aimed to investigate the influence of levodopa on the mean speech rate of patients with PD in a reading task. In addition we tried to pinpoint eventual fluctuations of speech rate with and without medication.

Methods

Participants

Twenty-five patients (8 women, 17 men) were included in this study. They all had a clinical diagnosis of "probable" idiopathic PD (Gelb *et al.*, 1999). All patients were in the advanced stages of PD and were treated with individualized medication

schemes resulting in highly variable dose regimens to obtain an "optimal treatment", as is characteristic in PD. All included patients had been treated with levodopa for a long time, most of them combined with other drugs, and had motor fluctuations with identifiable off- and on-periods. None of the patients had significant psychiatric or cognitive dysfunction that could interfere with the measurements. Moreover all patients were tested by means of a comprehensive neuropsychological test battery to exclude cognitive impairment severe enough to interfere with our evaluations. This battery included a general screening of cognition (Minimental State Examination), test of attention (Bourdon-Wiersma Test), memory (Verbal memory : Rey Auditory Verbal Learning Test ; visual memory : Benton Visual Retention Test), visuo-spatial functions (Clock Drawing Test) and executive functions (Controlled Oral Word Association Test, Stroop Color Word Test, Wisconsin Card Sorting Test). The dysarthria profile obtained from the Frenchay Dysarthria Assessment (Enderby, 1983) accorded with a classical hypokinetic dysarthria in all patients. There were no clinical or radiological (CT or MRI) signs suggestive of co-morbid neurological disease. Patients with deep brain stimulation were excluded because earlier studies have found that at least some patients with PD have an increase of speech problems following surgical treatment (Santens *et al.*, 2003).

Procedure

All patients were examined in both on- and off-conditions during the morning, so as to avoid effects of fatigue as much as possible. The procedure was the same in all patients. Anti-Parkinson medication was stopped for at least 12 hours to induce a practically defined off-condition, as suggested in the internationally accepted CAPSIT protocol (Defer *et al.*, 1999).

Each patient was asked to read a 182 syllables standardised passage ("The north wind and the sun") . We preferred a standardized reading text over spontaneous speech because of the well-known influence of dialect on speech rate (Robb *et al.*, 2004). The entire passage was recorded with a digital video-camera (Sony DCR-TRV420E). Speech analysis and video taking took place in a quiet room with low levels of background noise. After this reading task the regular morning dose of anti-parkinson medication was given. One hour later, during a practically defined on-condition as defined by the CAPSIT-protocol (Defer *et al.*, 1999), the entire procedure was repeated.

Data analysis

All obtained samples of both conditions were presented three times to the examiner (M.D.L.) in a randomized patient-by-patient order : 1) to time each text with a chronometer 2) to count the number of syllables that are read every ten seconds 3) to assess the presence and severity of dyskinesia and speech dysfluencies, as both are known as potential causes of decreased speech rate.The speech rate, timed in the on- and off-condition, was calculated and expressed as syllables/ min. The time of reading the entire text was divided in segments of ten seconds. The number of syllables uttered in each ten-second segment were calculated in both conditions. All comparisons between both conditions were performed using non parametric tests for related samples (Friedman). Post hoc analysis was done using the Wilcoxon signed rank test. All statistical analysis were performed in SPSS 12.0. P-values less than 0,05 were considered significant.

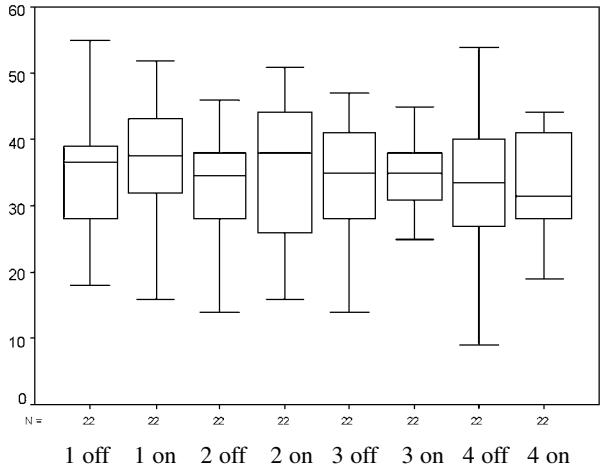

Fig. 1. — Box plot of speech rates in the four segments of ten-second each in the off- and on-condition.

Significant differences found between :
2 off – 2 on (p = 0,05)
1 on – 4 on (p = 0,02)
2 on – 3 on (p = 0,02)
2 on – 4 on (p = 0,01)

Results

The results of the speech rates in both off- and on-state, expressed as syllables/min, are shown in table 1. The presence or absence of dyskinesia and dysfluencies is also included this table. There were no significant differences between both states.

When comparing the segmented fragments of the entire passage, significant differences were found between segments of the on-state (Fig. 1), which point to a slower speech rate in segments 3 and 4 as compared to segments 1 and 2. No significant differences were obtained between segments in the off-state. When comparing the individual segments between the off- and on-states, only segment 2 displayed a significant increase in the on-evaluation.

Table 1

Speech rate, expressed in syllables/min and presence of dyskinesia and disfluencies in the individual subjects

Patients	Syllables/min		Dyskinesia		Disfluencies	
	OFF	ON	OFF	ON	OFF	ON
1	214,12	227,50	–	+	–	–
2	273,00	222,86	–	++	–	–
3	218,40	248,18	–	–	–	–
4	352,26	273,00	–	–	–	–
5	170,63	173,33	–	++	–	–
6	160,59	168,00	–	++	–	–
7	273,00	253,95	–	–	–	–
8	185,08	168,00	–	–	+(I)	++ (I)
9	210,00	185,08	–	–	–	+ (I)
10	260,00	287,37	–	–	–	–
11	145,60	153,80	–	+	–	+ (I)
12	253,95	232,34	–	–	–	–
13	232,34	237,39	–	–	–	–
14	195,00	232,34	–	–	–	–
15	198,55	280,00	–	+	+ (I)	–
16	242,67	242,67	–	–	+ (P)	+ (I+P)
17	133,17	162,99	–	–	+ (I)	+ (I)
18	280,00	295,14	–	–	–	–
19	202,22	188,28	–	+	–	–
20	136,50	122,70	–	–	–	–
21	242,67	214,12	–	–	–	–
22	168,00	195,00	–	–	–	–
23	104,00	165,45	–	+	–	–
24	253,95	242,67	–	+	–	–
25	242,67	237,39	–	–	–	+ (I)

Legend : – : absent ; + : present ; ++ : severe ; I : iterations ; P : prolongations.

Discussion

This study confirms previous reports on the effects of levodopa on speech rate in PD. No differences could be demonstrated in speech rate, expressed as the number of syllables per minute, between the off and on-states. However, we found a significant increase in speech rate in the early segments of the on-testing as opposed to the later stages of on-testing. This tendency was not found in the off-state. This suggests that speech rate is modified by the effects of levodopa in a more subtle way. Apparently speech rate varies little over the entire duration of reading a passage in the off-state, while significant variability was found during the same task in the on-state.

These findings can be interpreted in different ways. It has been suggested before that decreases in speech rate are mainly due to increases in pause time (Torp and Hammen, 2000).

In this study we preferred not to differentiate speech rate in articulation rate and pause time. We concentrated rather on the general outcome of speech rate in a reading task in order to obtain an "ecological speech rate". It is clear that this measure will be influenced by a number of variables, such as dyskinesia and dysfluencies. Therefore, a first hypothesis to explain the variability of speech rate in the on-state is an increased variability of pause time, as a consequence of disordered respiration due to levodopa-induced dyskinesias. Indeed, in this group of 25 patients, 9 suffered from dyskinesia during testing in the on-state (Table 1).

Secondly, the occurrence of dysfluencies (prolongations and iterations) might also contribute to an increase of pause time. It is well described that levodopa might induce dysfluencies in some patients (Louis *et al.*, 2002). In our group of patients, increase of dysfluencies in the on-state was found in 5 patients (Table 1).

Although we did not formally test the effects of dysfunctional auditory feedback, executive function and motivation, these factors cannot be excluded in the interpretation of our results. The role of

auditory feedback in self-monitoring of speech rate should not be underestimated. Clear defects in self-perception have been demonstrated for intensity, in that PD patients perceive their proper speech consistently as louder than the objective intensity measurements (Ho *et al.*, 1999). To the best of our knowledge, feedback defects for speech rate have not been described, although they cannot be excluded. Moreover, the impact of the well-known deficits in divided attention on different aspects of speech is equally unknown. Patients may be unable to maintain the stability of temporal aspects of speech during a complex task such as reading a text. The improvement of temporegulation by means of external strategies, such as metronomic regulation of speech, delayed auditory feedback and noise masking suggests indeed a role of defective executive function in the control of speech rate.

Finally, motivational factors can not be excluded. One might imagine an increase of motivation in the on-state leading to an initial improvement of speech rate, followed by a drop in later stages of text reading.

Therefore, it can be concluded that speech rate in PD is a complex phenomenon, which is modified by levodopa in a subtle way. Our results suggest that the presence of dyskinesia and dysfluencies may contribute to the variability of speech rate. We can hypothetically expect cognitive and mental dysfunction to contribute equally to this variability. Therefore, the physiology and pathophysiology of speech rate in PD, specifically the influence of dopaminergic treatment, requires further exploration.

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M. De Letter, MSc,
Department of Oto-Rhino-Laryngology,
Center for Speech and Hearing Disorders,
Gent University Hospital,
De Pintelaan 185,
B-9000 Gent (Belgium).
E-mail : miet.deletter@ugent.be.