NOTES AND DISCUSSION

The Temporal Control of Repetitive Articulatory Movements in Parkinson’s Disease

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Recent clinical data indicate that internal cueing mechanisms required for the triggering of movement sequences are impaired in Parkinson’s disease (PD). Nevertheless, most PD subjects produce maximal syllable repetition rates similar to those observed in healthy control individuals during oral diadochokinesis tasks. There is some evidence that tremor oscillations may pace repetitive movements in Parkinsonians giving rise to hastening phenomena. Conceivably, the performance of PD patients in syllable repetition tasks thus reflects a specific timing deficit, i.e., articulatory hastening. It is the aim of the present study to investigate the contribution of speech hastening to oral diadochokinesis in the presence of internal and external cues. By means of an optoelectric movement analysis system, the displacements of the lips during repetitions of the syllable /pa/ were recorded in two akinetic-rigid PD individuals. Subjects were asked to synchronize labial diadochokinesis to sequences of periodic acoustic stimuli (2.5–6 Hz). One of the PD patients showed speech hastening, i.e., he produced repetitions of 8 to 9 Hz whenever stimulus frequencies exceeded 4 Hz. The other Parkinsonian adequately matched the stimulus frequencies required. However, she achieved a higher diadochokinesis rate in the matching task than under the instruction to repeat “as fast as possible.” Thus, the presence of an external cue improved performance. In conclusion, our data indicate two deficits of the temporal control of repetitive articulatory gestures in PD: speech hastening and impaired self-paced sequencing. These two pathomechanisms may allow to reconcile the controversial findings on oral diadochokinesis in PD reported so far.
INTRODUCTION

Dysfunctions of the various components of the central motor system, e.g., corticobulbar tracts, basal ganglia, cerebellum, may give rise to dysarthric deficits (e.g., Darley, Aronson, & Brown, 1975). Oral diadochokinesis tasks, i.e., reiteration of a given syllable “as fast as possible on a single breath,” are a widely used clinical tool to assess the functional capacities of the articulatory system. Subjects with cerebellar atrophy, Friedreich’s ataxia, spastic dysarthria, or Huntington’s chorea show decreased syllabic rates, the inverse of syllable duration (Ziegler & von Cramon, 1986; Ludlow, Connor, & Basich, 1987; Gentil, 1990; Ziegler, Hartmann, Hoole, & von Cramon, 1990; Ackermann, Hertrich, & Hehr, 1995). In contrast, the performance of most patients suffering from Parkinson’s disease (PD) is similar to that observed in control subjects (Ludlow et al., 1987; Ackermann et al., 1995).

Bradykinesia, i.e., slowness of movement, is a well-recognized feature of limb motor dysfunctions in Parkinson’s disease (PD) which has been documented in the orofacial system as well (Caligiuri, 1987; Forrest, Weismer, & Turner, 1989). At first glance the finding of “unimpaired” syllable repetition rates seems to be at odds with the concept of bradykinesia. Slowed motor execution should give rise to prolonged movement times of articulatory gestures and, thus, to lengthened syllable durations. Yet, there is evidence that PD patients produce normal syllabic rates at the expense of movement amplitude. Caligiuri (1989) compared sequences of repetitions of the syllable /va/ at 3 to 5 Hz and 5 to 7 Hz and found hypokinesia, i.e., reduced range of movement, with faster oral diadochokinesis in PD subjects.

Considering these data one might assume unimpaired temporal control of oral diadochokinesis in PD. However, Parkinsonian patients may exhibit specific difficulties with the sequencing of repetitive movements in terms of hastening phenomena (Delwaide & Gonce, 1988). For example, PD subjects performing finger taps in response to a periodic sound signal often produce rates of 5 to 6 Hz as soon as the frequency of the external pacemaker exceeds 2.5 Hz (Nakamura, Nagasaki, & Narabayashi, 1978). Hastening has been documented during syllable repetitions as well. Logigian, Hefter, Reiners, and Freund (1991) asked PD patients to perform syllable repetitions at frequencies ranging from 1.2 to 6.3 Hz. Their measurements of syllable duration at the acoustic speech signal revealed speech hastening in five out of seven subjects. Kinematic data such as measurements of movement range and velocity were not obtained by these authors.

Given the above findings we hypothesize that normal performance during oral diadochokinesis tasks in PD not necessarily indicates preserved control of rapid alternating movements but, rather, might reflect articulatory hastening. If this suggestion holds true, the diadochokinesis rate achieved under the instruction “as fast as possible” must correspond to the frequency of articulatory hastening observed during syllabic repetitions in response to ex-
ternal pacing signals. Moreover, decreased movement amplitudes have to be expected under these conditions. To investigate this hypothesis we recorded labial movements in two PD patients by means of an optoelectric analysis system under self-paced and externally cued conditions.

PATIENTS AND METHODS

Both PD patients suffered from an akinetic-rigid type of their disease (KBA: male; age, 55 years; disease duration, 16 years; HWA: female; age, 74 years; disease duration, 10 years) and presented with the typical constellation of Parkinsonian dysarthria, i.e., monotonous speech of reduced loudness concomitant with articulatory inaccuracy in terms of mumbling utterances (Darley et al., 1975). These two subjects were selected because they showed an oral diadochokinesis rate at the upper (KBA) and at the lower limit of the normal range (HWA), respectively. It was expected that the fast oral diadochokinesis rate of the former patient might be due to articulatory hastening. Therefore, a control subject (first author = CON1: male; age, 38 years) who could produce syllable repetitions up to about 8 Hz was assigned as a match to patient KBA. Subject HWA was included in order to assess whether hastening contributes to rather slow syllable repetition rates in PD. A 79-year-old female (CON2) served as a control for patient HWA. Both controls had normal development of speech and language functions and had never suffered from cerebral disorders or diseases of the cranial nerves.

All subjects had to perform repetitions of /pa/ at various frequencies in response to periodic sound signals presented by earphones for the duration of 10 sec. The following matching frequencies were tested: 2.5, 3, 3.5, 4, 5, and 6 Hz. In addition, subjects were asked to produce syllable repetitions “as fast as possible on a single breath” (FAST mode) and at a self-paced comfortable diadochokinesis rate (COMFORT mode). Stimulus trains were generated using the Computerized Speech Lab (Kay Elemetrics; USA) and recorded on a DAT tape. Five trials of each of the eight conditions (six paced, two unpaced) were recorded (a total of 40 trials per subject). Matching frequencies were presented in ascending order, following the FAST and the COMFORT modes.

An optoelectric movement analysis system (ELITE; Milano, Italy) with two infrared cameras was used to track lip movements at a sampling frequency of 100 Hz. Reflective markers were attached to the nasion, upper and lower lips as well as the left corner of the mouth. During repetitions of /pa/, the lower lips perform the major part of the opening and closure gestures. Therefore, analysis was restricted to lower lip trajectories. To avoid onset effects, the first two syllables of each trial were discarded from analysis. The subsequent 11 syllable productions (numbers 3 to 13) were analyzed and provided the data base for the calculation of kinematic parameters. Using custom signal processing software the following measures were obtained.
from each syllable: duration, vertical displacement of the lower lip in the sagittal plane, peak velocity during the opening gesture. Over the eleven syllable productions per trial the median was computed for each of these three parameters. The medians of the five trials in each condition were averaged.

RESULTS

Figure 1 shows the performance of each individual under self-paced conditions (FAST and COMFORT mode) and in response to external stimuli. Both controls as well as subject HWA adequately matched all stimulus frequencies up to 6 Hz. In the FAST mode this patient’s performance dropped to a rate of 4.6 Hz. In the COMFORT condition she achieved a similar frequency (4.8 Hz). In contrast, patient KBA showed a shift in his repetition rate to 8±9 Hz when pacing rate exceeded 4 Hz. The gap between the matching and the produced frequencies was 0.4 Hz at the 4 Hz, 3.1 Hz at the 5 Hz, and 2.8 Hz at the 6 Hz condition. Under the instruction “as fast as possible,” subject KBA achieved a rate of 8.3 Hz (Fig. 1).

At repetition rates exceeding 3 Hz, both PD patients showed reduced displacements of the lower lips as compared to the respective control subject (Fig. 2). Articulatory “undershooting” was most pronounced at matching frequencies of 5 and 6 Hz. Reduced peak velocity of the opening gestures paralleled the decrease in movement amplitude. During syllable repetitions at a rate of 6 Hz both PD individuals already had reduced their lip gestures to a similar degree as the control subject CON1 in the FAST mode, i.e., at about 8 Hz. It is noteworthy that patient HWA who adequately performed the matching task in terms of speech tempo exhibited reduced displacement range under these conditions as well.

CONCLUSIONS

The kinematic data of the present study obtained from two Parkinsonian dysarthrics during syllable repetitions revealed speech hastening in one subject (KBA) and impaired self-paced timing in the other individual (HWA). Patient HWA performed at the lower limit of the normal range—as determined in healthy adults—when asked to perform oral diadochokinesis “as fast as possible” (HWA, 4.6 Hz; normal range: 4.4–7.1 Hz; Ackermann et al., 1995). However, she adequately matched stimulus frequencies up to 6 Hz in the presence of an external pacemaker. Animal and clinical studies indicate a deficient internal cueing mechanism required for the triggering of predictable movement sequences in PD (Georgiou, Iansek, Bradshaw, Phillips, Mattingley, & Bradshaw, 1993). The finding of decreased diadochokinesis rate in the absence of an external pacemaker is compatible with this model.

The impaired performance in the FAST mode as compared to syllable
Fig. 1. Syllable repetition rates produced by two patients with Parkinson’s disease (KBA, HWA) and by two healthy control subjects (CON1, CON2) during a matching task (required frequency, 2.5 to 6.0 Hz; external cueing) and under two self-paced conditions (F, “as fast as possible;” C, “at a comfortable speed;” internal cueing). Each value represents the averaged medians of five trials. (A) Patient KBA, control subject CON1. (B) Patient HWA, control subject CON2.

Repetitions in response to an external pacemaker does not seem to be an unspecific age-dependent effect. Subject CON2, a 79-year-old female control, adequately matched periodic acoustic signals up to 6 Hz and performed oral diadochokinesis “as fast as possible” at the same frequency. It cannot be excluded that the elderly control might have produced still higher externally paced repetition rates. However, subject CON2 would have to match
A repetition rate of about 7.5 Hz in order to achieve a similar "gap" between self-paced and externally cued oral diadochokinesis as patient HWA. In the absence of articulatory hastening, this frequency of alternating lip movements seems to be quite unlikely in elderly untrained speakers.

As a group, PD subjects show normal syllable rates in repetition tasks. A few individuals, however, may present with either decreased or accelerated speech tempo in comparison to healthy controls. For example, Ludlow and co-workers (1987) measured a lowered oral diadochokinesis rate under the instruction "as fast as possible" in three out of twelve PD patients. Other authors observed in single PD individuals a frequency of repetitive articulatory gestures exceeding the normal range (Ziegler et al., 1990; Ackermann, Gröne, Hoch, & Schönle, 1993; Ackermann et al., 1995). The results of the present study indicate that PD subjects do not represent a homogeneous group with respect to the pacing of repetitive articulatory gestures. The two documented pathomechanisms, i.e., speech hastening and impaired self-paced repetitions, may allow to reconcile the findings of slowed and increased diadochokinesis rates in PD.
Conceivably, the hastening phenomena observed in PD are due to a release of tremor oscillations pacing repetitive movements (Ackermann et al., 1993). Hertrich, Ackermann, Ziegler, and Kaschel (1993) documented a rhythmic modulation of pitch amounting to about 8 Hz in a PD patient with voice tremor and speech hastening. Thus, the observed shift to 8 Hz during speech hastening in patient KBA is compatible with the notion that tremor release provides the pathophysiological basis of speech hastening.

As compared to the control subjects, both PD patients produced smaller movement amplitudes (hypokinesia) of the lower lips concomitant with decreased peak velocity (bradykinesia). Even adequately matched syllable trains showed reduced articulatory excursions. These findings confirm former studies indicating, first, a trade-off between demands on speech tempo and movement amplitude (Caligiuri, 1989; Ackermann & Ziegler, 1991) and, second, the presence of bradykinesia (Forrest et al., 1989) in PD. Conceivably, the reduced peak velocity simply is due to the “undershooting” of articulatory gestures. Assuming a constant acceleration during movement execution reduced displacement must result in decreased maximum velocity.

In contrast to PD, other dysarthrias due to central motor disorders give rise to lowered rates of syllable repetitions (Ackermann et al., 1995). Thus, the question arises why patients with spastic, ataxic or choreic speech disorders do not produce normal speech rate at the expense of movement amplitude. Moreover, spastic dysarthria is characterized by slowed speech tempo in the presence of hypokinesia (Vogel & von Cramon, 1983; Ziegler & von Cramon, 1986). In the latter case the slowed transmission of the motor signal to the bulbar nuclei may account for the inability to accelerate syllable repetitions. A similar mechanism—at a level prior to the corticobulbar tracts—can be assumed for cerebellar dysfunctions as well. Transient inactivation of the cerebellar nuclei in nonhuman primates during prompt arm movements results in delayed triggering of the precentral cortex and, thus, delayed muscle innervation (Meyer-Lohmann, Hore, & Brooks, 1977). Assuming transmission of motor signals via cerebellum—motor cortex—corticobulbar tracts, it is quite conceivable that any disorder of this loop impedes acceleration of syllable repetitions.

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