

Jaw movement dysfunction related to Parkinson's disease and partially modified by levodopa

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Abstract

Objectives—To test the hypotheses that Parkinson's disease can differentially produce deficits in voluntary and rhythmic jaw movements, which involve different neuronal circuits, and that levodopa treatment improves specific components of the motor deficit.

Methods—Patients with idiopathic Parkinson's disease and control subjects were tested on a series of jaw motor tasks that included simple voluntary movement, isometric clenching, and natural and paced rhythmic movements. Jaw movements were measured by changes in electromagnetic fields and EMG activity. Patients with Parkinson's disease with fluctuations in motor responses to levodopa were tested while off and on.

Results—During the off state, patients with Parkinson's disease were significantly worse than the control subjects on most tasks. The deficits included a decrease in amplitude and velocity during jaw opening and closing, aberrant patterns and low amplitude of EMG activity during clenching, and low vertical amplitude and prolonged durations of occlusion during rhythmic movements. No decrements were found in the amplitude of voluntary lateral jaw movements or the frequency of rhythmic movements. During the on state, improvements occurred in the patterns and level of EMG activity during clenching and in the vertical amplitude and duration of occlusion during rhythmic movements, although a significant decrement occurred in the lateral excursion of the jaw.

Conclusions—Parkinson's disease affects the central programming of functionally related muscles involved in voluntary and rhythmic jaw movements and levodopa replacement influences only certain aspects of jaw movement, most likely those requiring sensory feedback.

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Orofacial motor abnormalities have long been recognised in Parkinson's disease.¹⁻³ Included in most textbook descriptions of Parkinson's disease is hypokinesia of the muscles of facial expression and a reduced eye blink rate, resulting in a mask-like face.⁴ Many patients

also have difficulties in the production of clear speech⁵ and with the automatic clearing of the throat or swallowing.³ Although the same peripheral structures are involved in various oral motor acts, such as speaking, swallowing, or chewing, distinct basal ganglia circuits may be used to generate the various motor patterns.⁶ The neural circuits involved in voluntary movement of the mandible may be different from those used for force production whereas those circuits regulating chewing may include portions of circuits for voluntary movement and force as well as additional circuits to process specific sensory input. A major basal ganglia circuit is the projection from the globus pallidus and substantia nigra reticulata, via the thalamus, to the primary motor cortex, the supplementary motor cortex, and the premotor area. Animal studies suggest that these cortical areas help to control the initiation, direction, force, and internal guidance of voluntary movements.⁶⁻⁸ A circuit that may influence rhythmic jaw movements is a small direct projection from the substantia nigra reticulata to brain stem neurons, which have connections with the trigeminal motor complex.^{9,10}

Little information is available on how Parkinson's disease affects the motor control of the jaw. Connor and Abbs¹¹ showed that patients with Parkinson's disease had decreased peak velocities and increased durations in a visually guided vertical jaw movement task. Abbs and associates³ reported that patients with Parkinson's disease were unable to sustain a steady four to five second isometric force using jaw closing muscles. However, the changes in velocity and force occurred in patients receiving dopamine replacement, which may have produced a motor deficit because of dyskinesia.⁴ Karlsson and coworkers¹² studied patients both on and off levodopa treatment and showed that levodopa increased the duration of the chewing cycle and the opening and closing velocities during peanut chewing.

The present study examines the effects of Parkinson's disease and the effect of dopamine replacement on voluntary jaw movements, jaw clenching, and rhythmic jaw movements. A battery of jaw motor tasks was used to discern possible involvement of different neural circuits (for example, voluntary jaw movements that likely involve connections with the motor cortical areas versus rhythmic movements that may include connections with brain stem neurons), differences among various variables of movement (for example, velocity, amplitude, or force), and differences between internal and

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external signals to organise rhythmic jaw movements. By testing patients with Parkinson's disease with major fluctuations in their motor response to levodopa (the on-off phenomenon), we could determine whether Parkinson's disease, as manifested during the off state, affects particular types of jaw motor control and whether acute levodopa treatment, during the on state, is useful for all types or only specific classes of jaw motor deficits.

Subjects and methods

SUBJECTS

We tested eight patients (six men and two women with a mean age of 53.7 years) with idiopathic Parkinson's disease (average duration nine years) and 11 control subjects (nine men and two women with a mean age of 54.4 years) who were free of signs of neurological disease. Routine examinations of the orofacial region were made and included an examination of intraoral structures; palpation of the temporomandibular joints, muscles of mastication, and associated muscles and tissues; and an assessment of the range of movement of the mandible and any accompanying pain or joint sounds. All subjects had most of their normal dentition and no signs of malocclusion. Two controls and two patients with Parkinson's disease had condyle displacement during the opening phase of the movement, but no evidence of pain and tenderness in the region of the jaw muscles and joints. All patients with Parkinson's disease showed some reduction in the range of normal jaw movement.

Patients were specifically selected because they displayed an on-off oscillating clinical response to levodopa and did not show signs of oral-buccal-lingual dyskinesia. For the data collection, the patients with Parkinson's disease had refrained from all medication for 10 to 12 hours and showed posture and locomotion disturbances when they entered the laboratory. During their off state, the patients with Parkinson's disease reported that they had considerable difficulty with activities of daily living, were unable or barely able to walk, and several reported having difficulty chewing or eating an entire meal. Their motor disability during the off state ranged from stage 2.5 to 5.0 on the Hoehn and Yahr scale.¹³ After being tested in the off state, the patients took their usual morning oral dose of carbidopa-levodopa (Sinemet). Within one hour, they were independent in ambulation and most other activities; their disability according to the Hoehn and Yahr scale ranged from stage 2.0 to 2.5, with an average improvement of 1.4 from the off state. At the end of the test session, the patients took their routine doses of other medications (including Sinemet-CR, selegiline hydrochloride, pergolide mesylate, and bromocriptine mesylate). All subjects gave informed consent for the procedures used in this study.

JAW POSITION AND MUSCLE ACTIVITY

Three dimensional measurement of mandibular movement was achieved with a model K6 Kinesiograph (MyoTronics, Seattle, WA). A

small magnet was glued to a point below the lower central incisors and changes in the electromagnetic fields were detected by magnetometers mounted on a lightweight set of eyeglass frames worn by the subject. The kinesiograph was interfaced with a microcomputer for data display and analyses.

Recordings of the EMG activity of the right and left temporalis and masseter muscles were obtained with bipolar, silver-silver chloride EMG electrodes placed in standardised positions on the skin over each pair of muscles. For the jaw clenching task, the EMG activity was differentially amplified, bandpass filtered (30 to 500 Hz), and full wave rectified.

EXPERIMENTAL DESIGN AND PROCEDURES

Patients with Parkinson's disease were evaluated in two one-hour test sessions. The first test session was done while the patients were in the off state and the second test session began one hour after the patients took their usual oral dose of carbidopa-levodopa (the on state). A single test session was used for most control subjects because no difference was found between the first and second test session for three subjects who were tested in two one-hour sessions with an hour of rest between sessions and because the performance of the control subjects was not significantly different from the results of a pilot study, which showed no difference between two separate one-hour sessions.

The jaw motor tasks were performed while the subject sat upright on a chair. Three types of jaw motor tasks were given—simple voluntary jaw movements, brief isometric molar clenching, and natural and paced rhythmic movements—and were done in the same sequence for all subjects. All tasks began and ended with intercuspal contact. The subjects were given simple verbal instructions and allowed to practice the tasks two to three times. Each task involved several trials with about 30 to 60 seconds of rest between trials; no information was given to the subject regarding their performance.

The voluntary jaw movements included normal vertical opening and closing of the mouth, opening the mouth as wide as possible, vertically opening and closing the mouth as fast as possible, and right to left horizontal jaw movements. Changing the conditions of the task—for example, from normal opening to opening wide or fast—differentially activates various muscle groups.¹⁴ Measurements were made on the amplitude, velocity, and variability of the jaw trajectory. For the isometric jaw clenching task, the subjects were required to bite on a piece of gauze using their molar teeth. Due to the wide range in age and clinical status of the patients with Parkinson's disease, the level of force during clenching was not specified. Each subject performed a series of three two second maximal effort jaw clenches, with one second of rest between each clench. The series was repeated three times with one minute of rest between each series. Measurements were made of the pattern, sequence, and amplitude of muscle activity.

The natural and paced rhythmic jaw movements were evaluated by unilateral gum chewing. The cadence of the natural versus the paced chewing provided information on internal timing regulation versus the ability of the subject to follow a rhythmic external signal. For the natural chewing, the subjects were instructed to first soften a piece of gum by chewing and then to chew the gum in their habitual manner on the right side for 60 seconds and, after a one minute rest, to repeat the task for another minute. Paced rhythmic jaw movements at various frequencies were included to disclose differences in jaw kinematics and velocity.¹⁵ The paced rhythmic jaw movement task involved making vertical jaw movements with tooth contact in time with a one, two, or three second auditory signal. Increasing the frequency of the paced movement also provided an indication of fatigue, as more work was done at the higher frequencies than at 1/s. The paced rhythmic movements were done for one minute and then repeated after a one minute rest. About 10 seconds after the subject began chewing, 10 seconds of data

were collected and another set of data was collected during the last 10 seconds of each one minute trial for both the natural and paced chewing tasks. A comparison between the first and last 10 seconds of data provided another measure of fatigue. Data were collected on the rate and variability of the rhythmic movements, the vertical amplitude, and the duration of occlusion, although accurate measurements of occlusion could not be made for the 2/s and 3/s paced conditions.

STATISTICAL ANALYSIS

Statistical analyses were performed separately for the results from each task. Student's *t* test was used to compare the differences between the control subjects and patients with Parkinson's disease. Differences in the performance between the off state (non-drug condition) and the on state (dopamine replacement condition) for each task were analysed using a repeated measures analysis of variance (ANOVA), followed by post hoc Student's *t* tests. The criterion for significance was set at $P < 0.05$.

Figure 1 Kinematics of three normal opening and closing jaw movements for a control subject (A) and two patients with Parkinson's disease (B). The control subject's traces were from two test sessions separated by one hour of rest; the data for the patients with Parkinson's disease were collected during the off and on states. The vertical plane represents the maximal vertical opening between the start position and the change between opening and closing phases of the cycle; the horizontal plane represents velocity during opening (traces on the right side) and closing (left side) of the cycle. The closing phase begins when the opening velocity returns to zero. During both off and on states, the patients had lower vertical amplitude and decreased velocity than the controls. During the on state, the opening amplitude increased for one patient (P03) and the opening velocities increased for both patients (P03 and P05). The midline is indicated by the small vertical arrow.

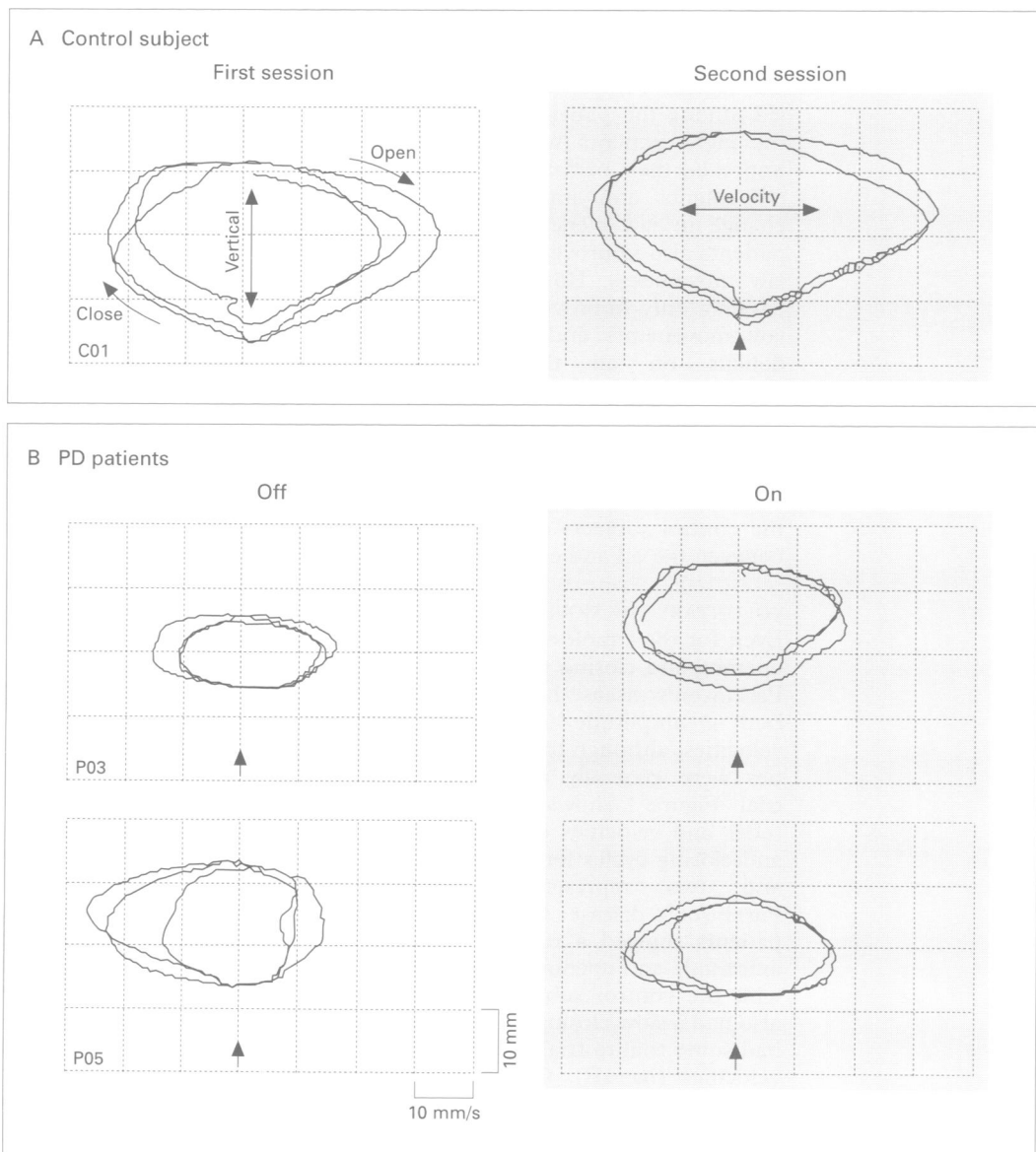
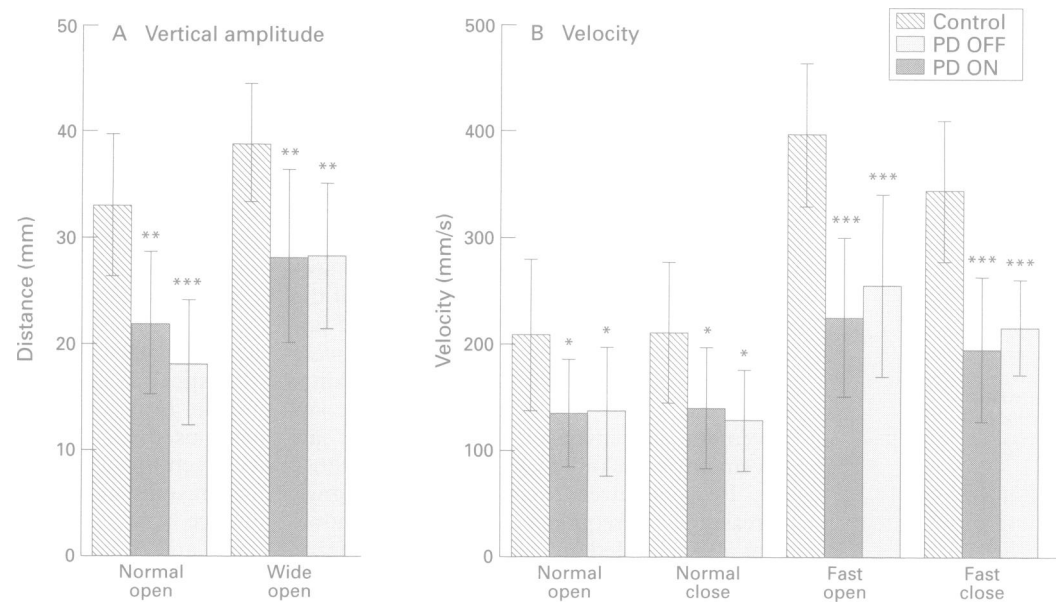


Figure 2 Means (SD) (error bars) of vertical amplitude (A) during normal opening and wide opening, and of the velocity (B) during normal and fast opening and closing. During off and on states, the patients with Parkinson's disease had a reduced vertical amplitude for both normal opening and wide opening tasks and slower velocities during normal and fast opening and closing than the controls. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$ v controls.



Results

During the off state, patients with Parkinson's disease experienced some difficulty doing most of the jaw motor tasks, including one patient who was unable to move his jaw laterally and two who were unable or declined to do the paced chewing at 3/s. In the on state, the degree of improvement or decrement varied among the patients and the tasks. Six of the eight patients with Parkinson's disease improved their performance on rhythmic jaw movements and improved or showed no change in jaw clenching, and two of these patients also improved their vertical voluntary jaw movements. However, one of the eight patients only improved his voluntary vertical jaw movements and one had performance deficits on all the behavioural tasks. Unexpectedly, most patients reduced their ability to move the jaw laterally during the on state. In this section, we describe for each task the differences between the patients with Parkinson's disease during the off state and the control subjects and then the differences between the off and on states.

VOLUNTARY JAW MOVEMENTS

Even for the simplest voluntary movement of opening and closing the mouth, patients with Parkinson's disease had restricted movement, both in amplitude and opening and closing velocities, although the amplitudes and velocities were generally consistent from trial to trial. Figure 1 shows examples of the amplitudes and velocities of three normal opening and closing cycles for a representative control and two representative patients with Parkinson's disease. During the off state, the patients showed a reduced maximal vertical amplitude and opening velocity in comparison with the control subjects, one patient (P03) also had a slow closing velocity, and one (P05) had some trial to trial variability in his closing velocities (fig 1B). Comparing the on state with the off state, one patient (P03) increased his maximal vertical opening and the opening and closing velocities, although they were still

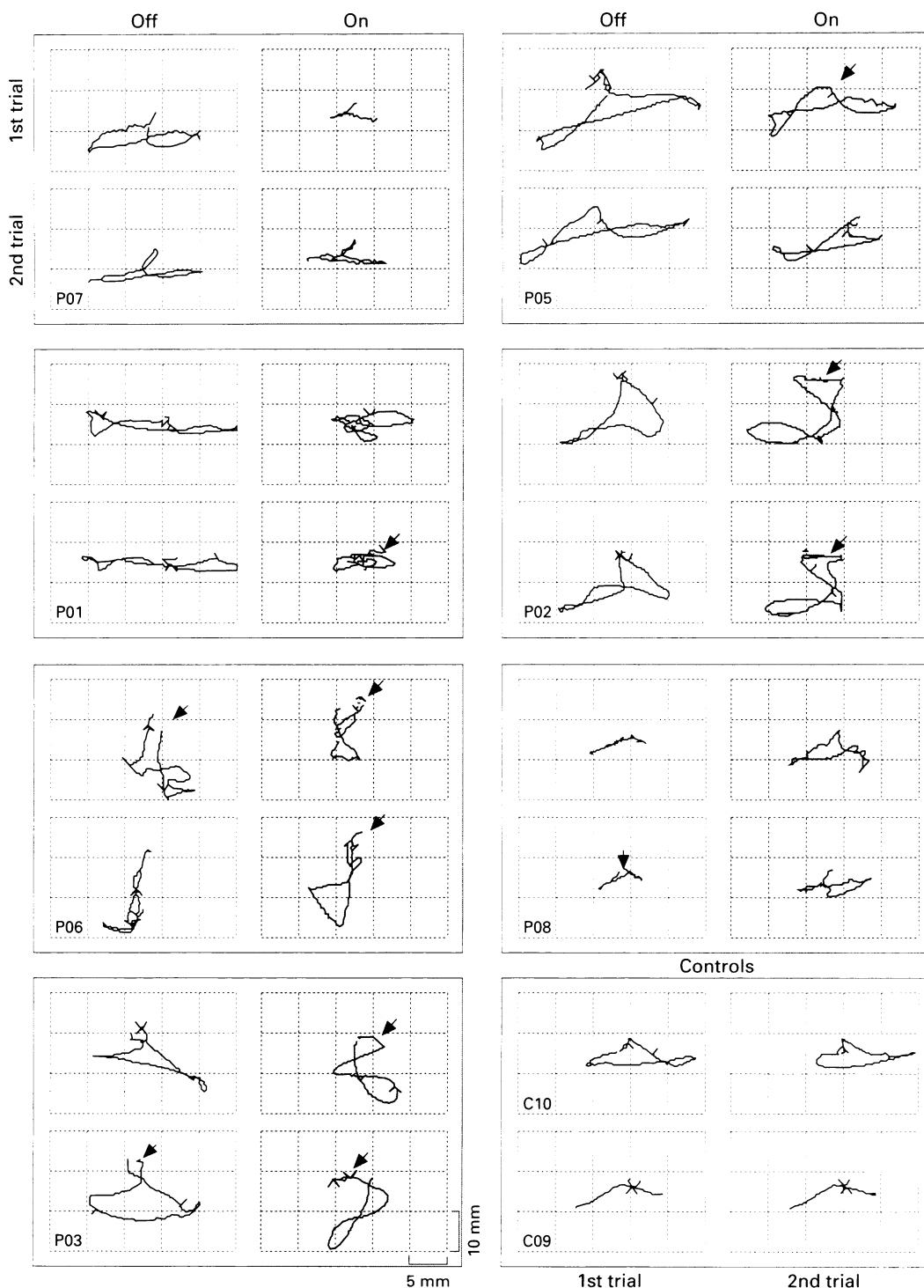
worse than the control's performance. The other patient (P05) slightly reduced his maximal vertical opening and closing velocity, but slightly increased his opening velocity.

Patients with Parkinson's disease were significantly worse than the control subjects for maximum vertical amplitudes and velocities for normal opening and closing, opening wide, and opening fast tasks (fig 2). During the off state, the maximal vertical amplitude during normal opening by patients was about 30% lower than the amplitude of the control subjects. During the on state, four patients had a 5% to 15% increase in vertical amplitude compared with the off state but three had a 30% to 61% decrease in amplitude. The average vertical amplitude during the on state was significantly ($P < 0.001$) lower than the control condition but was not significantly different from the off state. For the opening wide task, there was little change between the off and on states, with the vertical amplitudes for both off and on states being significantly less ($P < 0.01$) than the controls.

The maximum velocities during normal opening and closing were significantly ($P < 0.05$) slower for patients with Parkinson's disease during the off state than for the control subjects (fig 2B). The average opening and closing velocities did not change during the on state compared with the off state. When making fast vertical jaw movements, the control subjects increased their opening and closing velocities by about 50% in comparison with the normal situation. The patients with Parkinson's disease also increased their velocity by about 42% but they were still significantly ($P < 0.001$) slower than the control subjects for both the opening and closing phases. No significant differences were found between the off and on states for the opening and closing velocities or for the normal versus the fast conditions.

The lateral movement of the jaw was included in the test battery because it was a movement that the subject did not make routinely, so it required conscious control. The task required several practice trials by both

Figure 3 Frontal plane view of the kinematics of lateral jaw movements of seven patients with Parkinson's disease (one was unable to move his jaw laterally during the off state) and two representative control subjects. For the patients, the first and second trials are shown for both off and on states; for the controls, the first and second trials are shown for each subject. Although the kinematics between trials were similar, considerable variability occurred between patients. Five patients with Parkinson's disease (P07, P01, P06, P03, and P05) had less total right to left lateral excursion during the on state than during the off state. Some subjects required a final lateral movement to achieve intercuspal occlusion (arrow).



groups and one patient was unable to make lateral jaw movements during the off state (this patient's data were not included in the statistical computations). Variability in the vertical and the right to left horizontal amplitudes of the movement was evident among the controls and patients with Parkinson's disease (fig 3). However, all except one patient (P06) had consistent trial to trial kinematics, and all except two patients (P06 and P08) had symmetric right to left movements. The right to left distances averaged 18.7 mm for the controls and 15.4 mm for patients and were not significantly different.

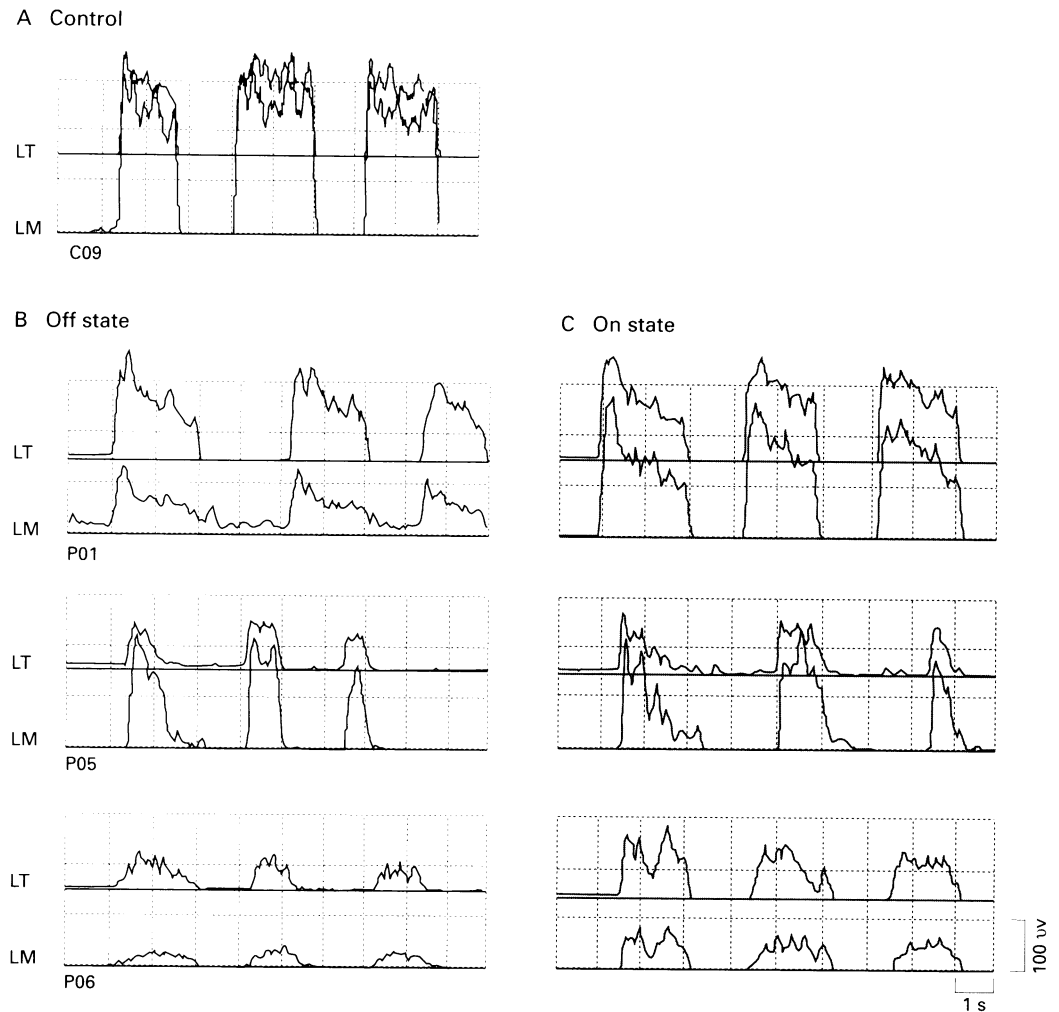
During the on state, the total right to left excursions decreased by about 6.1 mm, which

resulted in a significant ($P < 0.05$) difference between off and on states. Six patients with Parkinson's disease were worse during the on state than during the off state, including two with reduced total lateral excursions of more than 70% (for example, the average for patient P07 went from 15 mm to 6.6 mm total excursions). For several patients, the kinematics of the lateral movement during the on state were also more complex than during the off state and often included difficulties in achieving final intercuspal occlusion (fig 3: P01, P06, P03, and P02).

ISOMETRIC JAW CLENCHING

During the off state, none of the patients with

Figure 4 Rectified EMG activity of the left temporalis (LT) and masseter (LM) muscles during three isometric jaw clenches for a representative control subject (A) and three patients with Parkinson's disease during the off (B) and On (C) states. In the representative control subject (A), the left masseter muscle had greater activity than the left temporalis muscle, which was similar for half of the control subjects whereas the reverse was true for the rest of the controls. During the off state, none of the patients had a normal pattern of EMG activity (subject P01 had an EMG pattern that was most similar to the control pattern, whereas P06 was the least similar). During the on state, an improved pattern of EMG activity is represented by patients P01 and P06, whereas subject P05 represents little change.



Parkinson's disease had a pattern of EMG activity to clenching that completely resembled the EMG pattern of control subjects, although two patients had activity in one of the four muscles recorded that resembled the control condition (fig 4B, P01). The pattern of EMG activity for each two second episode of clenching by the control subjects consisted of a sharp onset of activity for each muscle that quickly reached peak amplitude and then gradually decayed until the verbal signal to relax, after which there was sharp, synchronous termination of activity, followed by an absence of activity during the rest phase (fig 4A). During the off state, patients with Parkinson's disease exhibited a variety of patterns of EMG activity to clenching (fig 4B). The abnormal EMG patterns included no clear onset or offset of activity, very low amplitude of activity in one or more of the muscles, a slow rise to peak amplitude, a rapid decay of activity, brief (< 1 s) sustained activity, and failure to return to baseline during the rest period.

During the on state, five of the eight patients showed improvements in the pattern of EMG activity, two had minimal change, and one was worse. Although no consistent pattern of improvement was evident among the patients with Parkinson's disease, improvements included an absence of inappropriate activity during the rest phase (fig 4C, P01), an increased duration of activity (fig 4C, P05), and a more well defined onset and ter-

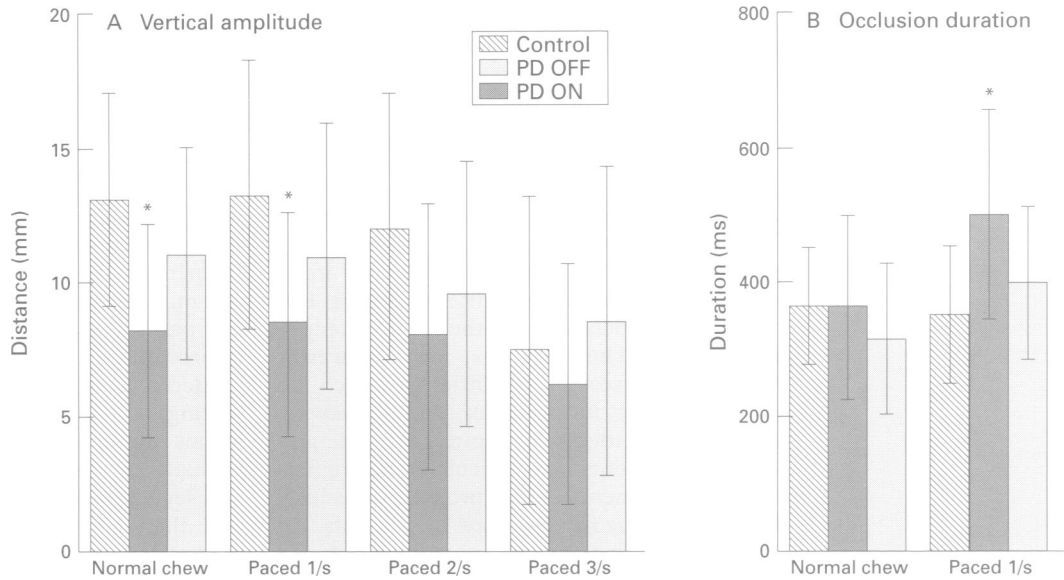
mination of activity (fig 4C, P06). The absence of EMG activity during the rest phase corresponds with the absence of involuntary jaw movements (mandibular dyskinesia) during the on state. The average peak amplitude of EMG activity (120 (SD 34) μV) of the jaw closing muscles during clenching for patients with Parkinson's disease while in the off state was lower than the average (215 (SD 102) μV) for control subjects, but they were not significantly different. During the on state, the average peak EMG activity increased by about 28% but was not significantly different from the off state.

RHYTHMIC JAW MOVEMENTS

All subjects were easily able to chew the gum for one minute and to make the paced movements at 1/s and 2/s. Several controls and patients with Parkinson's disease reported that the movements at 3/s were fatiguing and that they had problems maintaining the frequency; two patients declined to perform the 3/s paced movement. However, no significant differences were found in the cycle frequency or the vertical amplitude of the opening cycles between the first and last 10 seconds of the one minute trial of normal chewing or for any of the paced movements. Consequently, the first and last 10 seconds of data were combined for the present analysis.

The mean vertical amplitude of the opening phase was significantly less ($P < 0.05$) for

Figure 5 Mean (SD) of the vertical amplitude (A) and the duration of occlusion (B) during normal chewing and paced jaw movements (no measurements of occlusion were made for the 2 and 3/s paced tasks). During the off state, the vertical amplitude was significantly reduced during normal chewing and the 1/s paced condition and the duration of occlusion were significantly increased during the 1/s paced task. * $P < 0.05$.



Patients with Parkinson's disease during the off state than for the control subjects for normal chewing and the 1/s paced movement (fig 5A). The average vertical amplitudes during the 2/s and 3/s paced movement were also lower than the controls but were not significantly different. During the off state, the duration of the occlusal phase during normal chewing was similar to the control subjects, but was significantly ($P < 0.05$) longer than the control condition during the 1/s paced movement (fig 5B).

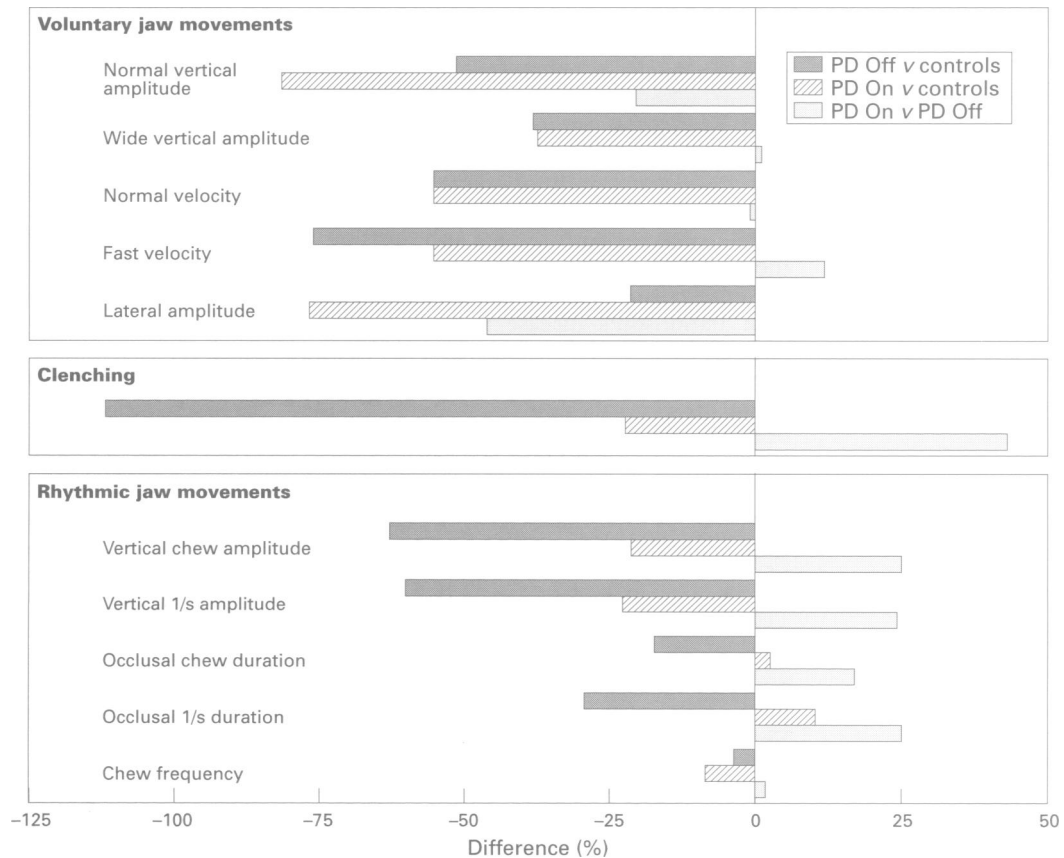
The vertical amplitude during the normal chewing and the 1/s paced movement increased slightly more than 4 mm between the off and on states. This resulted in a signifi-

cant ($P < 0.05$) difference between the off and on states but no significant difference between the on state and the controls (fig 5A). The duration of occlusion for the 1/s paced movement during the on state was also similar to the control condition. In addition, the two patients who were unable to do the 3/s paced movement during the off state, had an appropriate frequency and vertical amplitudes during the on state.

Discussion

The present study shows that Parkinson's disease affects more variables of motor control of the jaw than indicated by previous reports^{3 11 12}

Figure 6 Percentage difference between the control subjects and the patients with Parkinson's disease during the off state and between the off and on states for variables of the jaw motor tasks. Parkinson's disease was associated with deficits in all categories except the frequency of rhythmical jaw movements. Levodopa treatment (on state) produced improvement in the amplitude of EMG activity during clenching and in the opening amplitude and duration of occlusion during the rhythmical jaw movements, but caused a decrease in the amplitude of the lateral jaw movement and, in a few patients, a decrease of vertical amplitude during normal opening.



and that dopamine replacement seems to target only specific jaw motor deficits (fig 6). During the off state, Parkinson's disease is associated with large deficits in several variables of voluntary jaw control, jaw clenching, and rhythmic jaw movements. Most of the voluntary jaw movements were of low amplitude and slowly executed, similar to the characteristic bradykinesia of voluntary limb movements,⁴ the EMG activity was abnormal in all patients with Parkinson's disease during isometric jaw clenching, and the vertical amplitude during chewing was low. However, Parkinson's disease had little influence on cycle frequency during either normal chewing or paced movements during the off state and only a few patients had difficulties making lateral jaw movements or had prolonged occlusal durations during rhythmic jaw movements. An oral dose of levodopa (the on state) produced improvements, in comparison with the off state, in the jaw clenching and in the vertical amplitude and duration of occlusion during rhythmic movements. A few patients also improved the velocity of their fast voluntary jaw movements. However, levodopa treatment also resulted in a decreased amplitude of lateral jaw movements and, in a few patients, the vertical amplitude during normal jaw opening.

A limitation of the present study was the variability between patients of the motor impairments, which was most likely related to the unique features of the disease process and the variability of the therapeutic benefits of dopamine replacement. Patients with Parkinson's disease displayed a wide range of performance on each task during both the off and on states. For example, most patients had normal lateral jaw movements during the off state but one patient was completely unable to move his jaw laterally. The changes in performance related to levodopa ranged from one patient who achieved improvements on all tasks to another who was worse on all tasks. The individual differences were not correlated with the severity or duration of the disease, the degree of improvement of the Hoehn and Yahr scores between the off and on states, or the patients' age.

Some performance deficits of the patients with Parkinson's disease may be related to temporomandibular disorders (TMDs) because of their limited range of vertical and lateral movements and the abnormal pattern of EMG activity. Miller¹⁴ described neuromuscular changes of TMD, including decreased EMG activity during clenching, increased duration of the masticatory discharge, and spastic discharges. Although the EMG activity of the patients with Parkinson's disease included some EMG characteristics of TMD, none of the patients showed symptoms of tenderness and pain to palpation of their mandibular and cervical muscles, which is most often the initial symptom of the TMD.¹⁶ Further, there is little evidence to support the idea that variables of jaw movement and EMG activity are good predictors of TMDs in asymptomatic subjects.¹⁷

Peak dose dyskinesia can account for per-

formance decrements.⁴ Because the patients with Parkinson's disease were specifically selected to be free of cranial-cervical dyskinesias, they did not show any oral-buccal-lingual dyskinesia during the on state. Further, during the on state of isometric jaw clenching, the absence of masseter and temporalis EMG activity during the rest phase corresponds with the absence of involuntary jaw movements. Even the patient with decrements on all the jaw motor tasks had no obvious dyskinesia. In this patient, levodopa resulted in considerable improvement in posture and locomotion, but hindered her fine hand movements, such as writing, which may correspond to her deficits in jaw motor control. In this discussion, we evaluate the influence of Parkinson's disease and levodopa on the various jaw motor tasks and then consider how levodopa treatment may affect jaw motor control, while acknowledging that for a few patients other unknown mechanisms may be operating.

Disease or drug induced alterations of specific neural circuits may account for performance decrements during the off state or between off and on states. The voluntary jaw movement tasks used in the present study were designed to test the pallidothalamocortical connections with different cortical motor areas,^{6,7} which then project to interneurons of the gigantocellular reticular formation near the trigeminal motor nucleus.^{18,19} In the present study, Parkinson's disease resulted in deficits in the velocity and vertical amplitude of voluntary jaw movements made on command, movements that are likely regulated by motor areas of the cortex. Connor and Abbs¹¹ also have noted that Parkinson's disease results in decrements in the velocity:amplitude ratios of visually guided jaw movements, but not for similar movements made during speech. These investigators¹¹ suggested that the decrement during the visually controlled task was because the task was unfamiliar or unnatural, whereas the jaw movements made during speech were embedded in a natural sequence. The simple task of voluntary opening and closing of the mouth, used in the present study, was obviously a familiar task, which suggests that the deficit was probably in the cortical control of voluntary movement. This idea is supported by the similar deficits for normal opening versus wide opening and for normal versus fast opening and closing, though the different tasks activated different combinations of muscles and sensory feedback.¹⁴ Although patients with Parkinson's disease could increase their amplitude and velocity of jaw opening, their level was consistently below that achieved by control subjects. These results are consistent with the recent findings for arm movements²⁰ and postural adjustments²¹ of an inability of patients with Parkinson's disease to scale up the magnitude of a movement when increased speed or force is required.

Not all voluntary jaw movements were affected by Parkinson's disease or unaffected by levodopa. The amplitudes of the lateral jaw movements during the off state were not dif-

ferent from control subjects, but during the on state, were significantly reduced. This was just the opposite of the levodopa effects on vertical jaw movements. We initially expected that all voluntary jaw movements would have reduced amplitudes and that less practiced movements would have a greater deficit than the familiar movements. The lateral jaw movement task was initially not easy for the patients to execute, as they all required several practice trials. Consequently, the lateral jaw movement task may have involved learning whereas the vertical movement tasks were already well learned motor patterns. If the basal ganglia provide a mechanism to suppress unwanted movements during the acquisition of a movement,²² then performance of the lateral movement should have been worse during the off state than that of the control subjects, which was not the case. Another possible explanation is that the lateral and vertical jaw movements involve different neural circuits. For example, lateral jaw movements have a different cortical representation than the jaw closing representation in the motor cortex,^{23,24} which may also occur for the other motor cortical areas.

The negative effects of levodopa were also not expected. During the on state, patients with Parkinson's disease showed mild to severe reductions in the amplitude of the lateral jaw movements, even though levodopa was associated in these patients with improvements in various aspects of rhythmic movements and jaw clenching. The negative effect of levodopa on a specific task is consistent with negative effects of levodopa for lateral head movements²⁵ and postural adjustments.²¹ Further investigation will be required to decide whether the deficit induced by levodopa in lateral jaw movements is related to variables in the acquisition of the motor task, particularities of the neuronal circuits, or other reasons.

The isometric jaw clenching task was included as another task involving the pallido-thalamocortical circuit, particularly those connections with the primary motor cortex, which animal studies have shown to be important in regulating force.²⁶ A deficit in force production and maintenance is suggested by the low level of EMG activity during isometric jaw clenching by patients with Parkinson's disease, as the EMG activity during isometric clenching is linearly related to the total tension force developed by the muscles.²⁷ Reduced force production by patients with Parkinson's disease has been noted in tasks involving the upper extremities²⁸ and the orofacial muscles of the lip, tongue, and jaw.³ In the present study, all patients showed some abnormal EMG activity during isometric clenching, although the peak amplitude of EMG activity in patients with Parkinson's disease was not significantly different from the controls due to the high variability between patients. Some of this variability may have occurred because the instructions did not specify the amount of force to be reached, slight differences in the EMG electrode placement, and variations in facial dimensions and size of the jaw closing muscles.¹⁴ However, these factors were min-

imised by comparing the same patient during the off and on states, which showed that Parkinson's disease influenced both the pattern and amplitude of EMG activity during clenching. Levodopa treatment produced mixed effects, including improved onset and offset of EMG activity and large increases in EMG amplitude, although a few patients had decreases in amplitude. Although single unit activity of neurons in the basal ganglia is poorly correlated with EMG activity,²⁹ a reduction in thalamocortical activation may prevent the motor cortex from generating appropriate commands.⁷

Rhythmic jaw movements that occur during mastication are mainly controlled by brain stem neurons. The basal ganglia can influence rhythmic jaw movements through direct projections from the substantia nigra reticulata to parvocellular reticular neurons of the brain stem,^{9,30} which form direct connections with neurons controlling the jaw closing muscles.¹⁰ Parkinson's disease influenced the vertical amplitude and the durations of occlusion of internally (self induced) and externally paced rhythmic movements, suggesting either that the tasks involved similar circuits for internal and external control or that Parkinson's disease does not influence the internal versus the external control of a movement. Although internal control versus synchronisation with an external signal may involve separate neuronal circuits,⁸ there is also evidence from animal experiments that parts of the striatum, outside the motor region, are activated by both internal and external stimuli before a motor response.^{31,32}

Levodopa increased the vertical amplitude and decreased the occlusion duration enough that there was no significant difference between the on state and the control subjects. Karlsson *et al*¹² noted similar findings for peanut chewing during off and on states. The similarity of the results between gum chewing, vertical paced movements, and chewing peanuts is remarkable, as the size, shape, and hardness of the food are known to influence the pattern of chewing movements, the accompanying EMG activity, and the force-time curves.^{33,34}

Because both the jaw clenching and the rhythmic movements tasks benefited from levodopa, they may share important common features, such as access to sensory stimuli. Achieving appropriate force levels during clenching or chewing depends on feedback from receptors in the periodontal region of the dentition, the temporal-mandibular joint, and the muscles and ligaments,¹⁴ although animal studies have shown that muscle spindles or temporal-mandibular joint receptor afferents do not influence the frequency of rhythmic jaw movements.³⁵ The dopaminergic circuits may participate in the processing of sensory information or in the regulation of access of sensory input to appropriate motor centres.² Patients with Parkinson's disease show deficits in tests of orofacial sensory function and sensorimotor integration,² although they also exhibit hypersensitivity to cutaneous stimuli as part of a

perioral reflex³⁶ and alterations of various trigemotrigeminal or trigemofacial reflexes.³⁷ Thus the dopamine induced improvements of clenching may result from better processing of somatosensory stimuli within the brain stem reticular formation.

The results of this study support the idea that Parkinsonism does not alter just one type of jaw movement but affects several variables of voluntary and automatic movement, confirming the conclusions of animal studies.^{38 39} Parkinson's disease seems to affect behaviours that are probably controlled by ascending connections with various cortical areas and descending projections to the brain stem. However, the present data support a limited role for dopamine replacement in motor control of the jaw. The dopaminergic system may impact the general motor control of the limbs and posture in various ways but, for jaw motor tasks, the dopaminergic influence seems restricted to a few tasks, possibly those involving a particular type of sensory feedback.

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