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Short-Term Effects of Behavioral Treatment on Movement Initiation and Postural Control in Parkinson's Disease: A Controlled Clinical Study

*Viktor Müller, *Bettina Mohr, †Regina Rosin, *Friedemann Pulvermüller, †Friedemann Müller, and *‡Niels Birbaumer

**Institute of Medical Psychology and Behavioral Neurobiology, and †Department of Neurology, University of Tübingen, Tübingen, Germany; and ‡University of Padua, Department of Psychology, Padua, Italy*

Summary: In a controlled clinical study, we investigated the effects of behavioral treatment on postural and gait initiation problems in idiopathic Parkinson's disease (PD). Comparable groups of patients received behavioral therapy (experimental group, n = 15) and nonspecific psychological treatment (control group, n = 14) for 10 weeks. We monitored various variables reflecting properties of posture and gait initiation by using an optoelectronic motion analyzer (electronic movement analysis system, ELITE). A clinician blind to group membership of the patients assessed PD severity with the Unified Parkinson's Disease Rating Scale (UPDRS) before and after the treatment period. ELITE measures of postural stability and movement initiation revealed treatment-specific effects. In addition, UPDRS motor scores showed significant improvement only after behavioral treatment. We conclude that behavioral treatment in Parkinson's disease may improve motor disabilities in moderately advanced PD patients. **Key Words:** Parkinson's disease—Behavioral treatment—Optoelectronic movement analysis—Gait initiation—Posture.

Besides the main clinical features *tremor, rigidity*, and *bradykinesia*, Parkinson's disease (PD) causes problems in initiation and coordination of movements and abnormal postures. Gait and postural problems are so characteristic of PD that they are used as criteria for the diagnosis of this disease and for assessing its severity (1). *Postural abnormalities*, among others, include a stooped posture first described by Parkinson (2) as a "propensity to bend the trunk forward." In addition, PD patients suffer from a decline in postural stability of the trunk and particularly of postural fixation of the head (3). Additional postural abnormalities are the tendency to flex elbows and knees as well as the fingers. Postural problems may contribute to specific *gait problems* (4), which include the characteristic shuffling gait with small steps, poor trunk movement, and reduced or absent movements of the upper limbs (for example, arm swing) while walking. The initiation of locomotion is particularly difficult and frequently leads to the well-known symptom of arrested or "frozen" gait. Furthermore, an involuntary hastening in gait, propulsion (or festination), and retropulsion may occur (5–7). These postural and gait problems are among the most characteristic manifestations of PD.

The traditional treatment of motor disturbances associated with PD consists in the application of L-dopa or dopamine agonists. This medication often improves symptoms like bradykinesia and rigidity in the beginning, but the effects lessen over time (8). In addition, medications appear less effective in improving coordination problems during axial body movement, such as turning in bed and rotational movements of the trunk during locomotion (9). As a result, many medicated PD patients have problems in initiating locomotion, in turning around, in switching gaits, and in stopping locomotion.

Several studies have attempted to improve coordination of posture and locomotion in PD patients by special physical training programs in conjunction with drug treatment. Although some of these studies reported improvement in motor performance (10–12) as well as bet-

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Address correspondence and reprint requests to Dr. B. Mohr at Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen, Gartenstrasse 29, 72074 Tübingen, Germany.

ter performance of daily living activities after physical exercise (12–15), others did not find any improvement in motor abilities after intensive physical training (16,17).

It is well known that Parkinson patients are frequently able to improve their gait with strong voluntary effort, but when concentration weakens, they resume their usual shuffling and small-stepped gait (5). This observation demonstrates that gait can be improved without drugs such as L-dopa, but that a method or strategy must be found that maintains this better performance for a longer period. In addition, problems in motor performance of PD patients, especially difficulty in initiating and maintaining stepping, can frequently be positively influenced by external (visual or acoustic) or internal cues. PD patients have less difficulty walking on a striped floor (external visual cue) or when they are listening to a marching song (external acoustic cue). Gait initiation can also be facilitated when patients are silently speaking a command to move (internal cue). Furthermore, abnormalities in gait, posture, and other motor performances are strongly influenced by psychological variables, such as the emotional state of the patient or psychosocial stress (18,19). All of these features indicate that Parkinson patients are able to influence their motor performance positively for a short period without drugs. While psychological interventions (for example, behavior therapy) demonstrate a reduction in stress responses and improvement in social skills (20,21), results of controlled treatment programs aimed at improving motor skills by intensive (daily) training in motor strategies and relaxation in PD patients have not yet been reported. The purpose of this study was to evaluate the efficacy of a behavioral treatment program directed at initiation and control of movements by means of an electronic movement analysis system (ELITE camera system). The ELITE system enables the detection of very small changes in motor performance, especially in posture and gait.

According to Nutt and co-workers (4), normal walking requires the capacities (a) to stand upright, (b) to maintain balance, (c) to initiate stepping, and (d) to maintain rhythmic stepping. Although the so-called components of equilibrium (posture) (a and b) and locomotion (c and d) are aspects of walking that can be described separately, they are inseparable and strongly interwoven in gait. Because PD includes deficits related to all four of the aspects, however, a useful strategy appears to be to address each of the components separately during therapy and to measure each of them when evaluating therapy effects.

It was hypothesized that after 10 weeks of behavioral treatment with emphasis on training of motor skills and muscle relaxation, the posture and gait of PD patients would improve. In particular, it was expected that treated patients would show

1. Better postural control in the upright position
2. A reduction in forward bending during walking
3. A reduction in the time needed for initiating movements
4. Better coordinated movements while walking

However, because PD is progressive, it may appear unlikely that patients improve their performance relative to the pretreatment baseline without changing medication. Nevertheless, if therapy is effective, improvements can be expected *relative to normal symptom aggravation* seen in PD patients.

Therefore, two equivalent groups of PD patients were included in this study: one receiving behavioral treatment and the other receiving nonspecific treatment. Both groups were tested before and after treatment. In this design, a significant group × prepost interaction with either stronger behavioral improvements or smaller performance decline in the behavioral group compared with controls would indicate benefits of the behavioral treatment.

METHODS

Experimental Procedure

The study included an experimental group receiving behavioral treatment and a control group receiving nonspecific psychological and physical treatment. The nonspecific treatment group was included to control for nonspecific effects of the therapeutic intervention (placebo control group). Patients were randomly assigned to either group. Informed consent was obtained from all patients. Neither patients nor the physician who made the clinical ratings and ELITE recordings were informed about group membership. Patients were tested before and after the treatment period in order to assess possible changes caused by therapeutic intervention. Both kinds of treatment lasted for 10 weeks with two 1½-h sessions per week.

Treatment Groups

Patients in the *behavioral group* learned to use specific strategies in motor behavior (such as walking, standing in an upright position, getting up from a chair, turning in bed, and handwriting) in order to improve their initiation and coordination of movements. For example, they learned to use external cues (visual, acoustic, or tactile) during walking or when having a "freezing" episode. *Chaining* (dividing a complex movement into several simple movements with the application of positive reinforcement) was used as a behavior modification technique when training handwriting or walking a longer distance. In addition, *video feedback* and *behavior rehearsal techniques* were employed, especially when training gait. A *progressive muscle relaxation technique* (22) was applied for better control of movements, especially in stressful situations. In a *social skills training*, patients learned to use the recently learned strategies in problematic social situations. Activity schedules were used for the control of practicing the newly learned behavior. A detailed description of the behavioral treatment has been provided by Mohr et al. (23) and Strehl and Birbaumer (24). During treatment, no instructions explicitly addressed variables later quantified in the ELITE examination.

The control group received nonspecific treatment. Patients in this group were given information about the disease. Breathing exercise as well as physical exercises were applied and patients could discuss disease-related problems [see Mohr et al. (23) for a complete description].

Patients

A total of 29 patients with idiopathic PD participated in the study. The experimental or behavioral group consisted of 15 patients (three women), and the control group included 14 patients (six women). Patients in both groups were comparable for clinical and demographic variables, as well as IQ (Table 1). All patients were prescribed a combination of L-dopa with either a dopamine agonist (usually bromocriptine or lisuride) and/or a monoamine oxidase inhibitor. There was no change in medication at least 4 weeks prior to treatment. Medication intake remained unchanged during the 10-week treatment period. Average L-dopa doses were 300 mg/day in both groups. For each patient, gait recordings and clinical assessments before and after therapy were carried out at the same time of the day to exclude artifacts caused by motor fluctuations during the day. Patients were tested in on-phases, but several hours (at least 2 h and, on the average, 3 h) after drug intake to exclude peak-dose effects.

Patients suffering from depression, dementia, or other psychiatric disorders according to DSM-III-R (25) were excluded from the study. None of the patients had a history of alcohol or drug abuse nor any other significant physical illness.

Clinical Assessment

A physician blind to the hypotheses of the study conducted the neurologic evaluation before and after the treatment in both groups. The following rating scales were used for assessment of symptoms: Unified Parkinson's Disease Rating Scale (UPDRS) (26), including the modified Hoehn & Yahr Scale (1) and the Activities of Daily Living Rating Scale (27). For the assessment of depression, the Beck Depression Inventory (28) was applied.

TABLE 1. *Mean scores and standard deviation of demographic and clinical variables of both patient groups*

Measure	Experimental group (n = 15)	Control group (n = 14)
Age (SD)	62.7 (7.77)	61.5 (5.69)
IQ, WAIS-R (SD)	118.9 (14.75)	120.8 (15.02)
Years of formal education (SD)	10.6 (1.80)	10.1 (1.61)
Duration of illness (SD)	7.7 (3.77)	9.0 (4.84)

SD, standard deviation; WAIS-R, Wechsler Adult Intelligence Scale—Revised.

Gait Recordings

For analysis of posture and gait before and after treatment, an optoelectronic two-camera motion analysis system was used (ELITE motion analyzer; Bioengineering, Technology and Systems, Milan, Italy). To assess gait (posture and locomotion), horizontal and vertical changes in seven defined marker positions were recorded. Small light-reflecting dots were attached to the left lateral part of patients' head, shoulder, elbow, wrist, trochanter, knee, and ankle (Fig. 1).

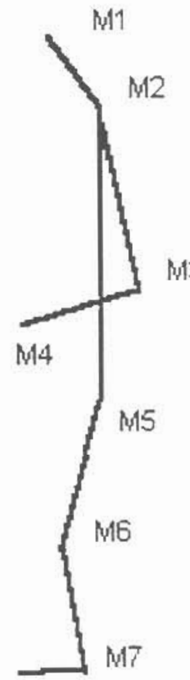

FIG. 1. Lateral aspect of a Parkinson patient showing the position of seven markers used for gait recordings with the ELITE camera system M1, zygomatic bone; M2, acromion; M3, elbow; M4, wrist; M5, trochanter; M6, knee; M7, ankle.

Patients were instructed to stand at a starting line and to begin walking immediately after an acoustic signal. The distance between patient and camera was 2.5 m. Patients had to walk a distance of 4 m parallel to the camera system at normal pace. This procedure was repeated 10 times (10 trials) for each patient.

The positions of the seven markers in space were traced by a video camera under infrared stroboscopic illumination. Marker positions were extracted automatically from the digitized video frames. The acoustic starting signal triggered a microcomputer recording the successive marker positions. Position data were recorded with a sampling rate of 100 Hz. Start position amplitude and duration of movements were calculated from position signals. Kinematic data were analyzed separately for each marker.

Data Reduction and Analysis

Comparisons between the experimental and control group were calculated for dependent variables defined as relevant with respect to the hypotheses (see the introduction): (a) As a measure of initial posture (*trunk position*), the angle of the trunk relative to the vertical was measured before movement onset. (b) Posture changes during walking were assessed using the position of the trunk by calculating the *delay* of the first change in the trunk position relative to its position at the start. A second measure of postural stability was the *maximal angle of trunk position* during walking minus its angle at the start. This variable reflects the angle by which patients bent forward relative to their starting position. (c) Initiation of walking was measured by two variables related to movements of the upper body. Variables, such as stride length and stride duration, related to movements of the lower limbs were not included in the analysis of gait initiation because they seem to be steady-state parameters of locomotion rather than parameters of movement initiation (29). In addition, these measures exhibited a large variance, even within each patient, making them inappropriate for documenting changes. Arm movements usually accompany walking and can be taken as a measure of the initiation of dynamic stabilization involved in the walking process. First, the time to *onset of arm flexion* relative to the starting signal was analyzed. Second, a similar measure, the time to *maximal arm flexion* after the starting signal was obtained. Both of these variables provide information on how long it takes to start movements necessary for holding balance while walking. (d) Movement coordination was assessed using the initiation time of arm flexion (A) subtracted from the initiation time of shoulder movement (B). Usually, arm flexion and shoulder movements occur almost simultaneously during walking (29). The ELITE system records shoulder movement as the earliest motion when a person starts walking. Therefore, shoulder movement is taken as a measure for movement initiation of the whole trunk. A large value for the time difference A − B indicates impaired coordination of walking movements.

Three-way analyses of variance (ANOVAs) were performed for each of the six dependent variables. The design of each analysis was group × prepost × trials, with the last two factors being repeated measures. Greenhouse–Geisser corrections were applied when appropriate. Two-way (prepost × trials) ANOVAs were calculated for each group separately to find differences in data obtained before and after treatment. To test the hypothesis that the experimental group benefited more (or declined less) after treatment relative to the control group, average prepost difference scores were calculated for each subject. These scores were used as the dependent variable in an unpaired t test with group as the independent variable. One-tailed tests were used because we had specific predictions about the direction of the outcome of the study.

RESULTS

Pretreatment Assessment

One-way ANOVAs were performed on all pretreatment data to verify that the two groups were comparable. The analyses revealed no significant differences between the two treatment groups' demographic variables and clinical ratings.

TABLE 2. *Mean scores and standard deviation of the Unified Parkinson's Disease Rating Scale and Beck Depression Inventory (BDI) before and after treatment*

Measure	Experimental group			Control group		
	Before	After	p	Before	After	p
Hoehn & Yahr (SD)	2.13 (0.67)	1.93 (0.42)	0.05	2.07 (0.33)	2.04 (0.36)	NS
Mental subsection (SD)	0.75 (0.82)	0.55 (0.56)	0.003	0.82 (0.69)	0.71 (0.59)	0.05
ADL subsection (SD)	1.06 (0.88)	0.89 (0.76)	0.002	0.88 (0.74)	0.85 (0.76)	NS
Motor subsection (SD)	1.37 (0.97)	1.18 (0.94)	0.003	1.24 (0.90)	1.23 (0.90)	NS
BDI	9.86 (6.61)	8.40 (5.03)	NS	10.9 (5.14)	9.00 (3.86)	NS

ADL, activities of daily living; NS, not significant; SD, standard deviation.

Beck Depression Inventory

A two-way ANOVA (group × prepost) revealed a significant main effect of prepost indicating improvement of depression scores after treatment for both groups [$F(1,27) = 5.23$, $p = 0.03$].

Unified Parkinson's Disease Rating Scale

Scores of all items were taken for calculation of treatment-specific effects in both groups. Means for each of the three subsections were calculated by dividing the sum scores of each subsection by the number of items administered. Three-way ANOVAs with the factors group × prepost × items were calculated for each subsection.

1. Subsection: Mentation, behavior, and mood. The analyses demonstrated a significant main effect of prepost [$F(1,27) = 16.55$, $p = 0.0004$]. Two-way ANOVAs revealed a significant difference between pretreatment data for the behavioral group [$F(1,14) = 12.92$, $p = 0.003$] and for the control group [$F(1,13) = 4.50$, $p = 0.05$].

2. Subsection: Motor examination. The analyses revealed a significant interaction of the factors group × prepost [$F(1,27) = 6.97$, $p = 0.01$]. Two-way ANOVAs showed a significant difference between pretreatment and posttreatment data for only the behavioral group [$F(1,14) = 12.8$, $p = 0.003$].

3. Subsection: Activities of daily living. The interaction of the factors group × prepost just failed to reach significance [$F(1,27) = 3.4$, $p = 0.07$]. Two-way ANOVAs indicated a significant difference between pretreatment and posttreatment data in only the behavioral group [$F(1,14) = 14.2$, $p = 0.002$).

4. Hoehn & Yahr Scale. There was a significant improvement after treatment only in the behavioral group [$F(1,14) = 4.1$, $p = 0.05$].

5. Activities of daily living (Schwab and England). There were no significant main effects or interactions.

ELITE

1. Initial posture (trunk position). Analysis of initial posture did not reveal any differences between groups before or after treatment, and the group × prepost interaction was far from significance. Thus, the angle of the trunk at the start relative to vertical essentially remained constant for both groups.

2. Forward bending during walking. There was an almost significant interaction between the factors group and prepost for the variable "bending delay" [$F(1,27) = 2.94$, $p = 0.09$]. In the control group, there was a reduction in the delay of the change in the trunk position relative to its position at the start—thus, bending occurred faster at the end of treatment (Fig. 2). Separate two-way (prepost × trial) ANOVAs revealed a significant decline in the control group [$F(1,13) = 4.9$, $p = 0.04$], but not in the behavioral group. A further comparison of average prepost difference scores showed that control patients had a greater prepost change in the onset of bending compared with the experimental group [one-tailed $t(27) = 1.71$, $p = 0.04$].

Most importantly, a three-way ANOVA revealed a significant interaction of the factors group × prepost for the variable "maximal angle of trunk position minus angle of trunk position at start" [$F(1,27) = 4.03$, $p = 0.05$] (Fig. 3). Again, this variable describes the angle of forward bending during a few walking steps. Separate two-way (prepost × trial) ANOVAs demonstrated a significant decline in the control group [$F(1,13) = 6.93$, $p = 0.02$], but not in the experimental group. Control patients evidenced stronger forward bending after treatment. A further comparison of average prepost difference scores confirmed that control patients had a greater prepost change in angle positions of the trunk compared with the behavioral group [one-tailed $t(27) = 2.16$, $p = 0.02$].

3. Gait initiation. As a measure of accuracy of movement initiation, the "time to onset of arm flexion relative to starting signal" revealed a significant prepost × group interaction [$F(1,27) = 4.45$, $p = 0.04$] (Fig. 4). Further comparisons of average prepost difference scores indicated that patients in the behavioral group showed faster onset of movements after therapy compared with the control group, where performance declined after therapy [one-tailed $t(27) = 2.11$, $p = 0.02$].

The "time to maximal arm flexion," another measure of adequate initiation of walking, revealed a marginally

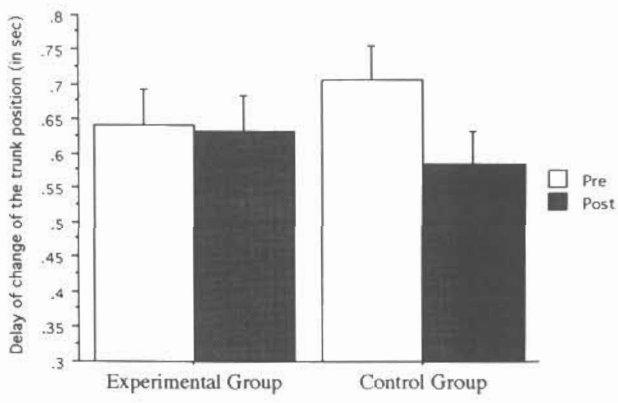

FIG. 2. Delay of change of the trunk position relative to its position at the start in both treatment groups before and after treatment. The interaction is marginally significant. Two-way analyses of variance revealed a significant decline in the control group, but not in the behavioral group.

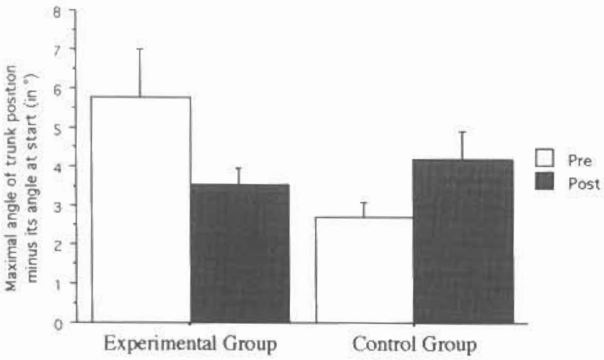

FIG. 3. Maximal angle of trunk position during walking minus its angle at start (forward bending) in both treatment groups before and after treatment. The interaction is significant. Two-way analyses of variance demonstrated a significant decline in the control group, but not in the experimental group.

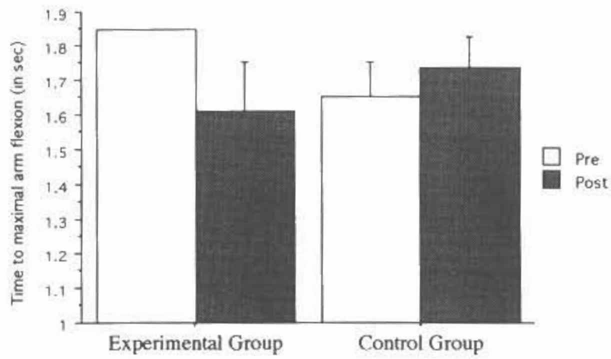

FIG. 5. Time to maximal arm flexion in both treatment groups before and after treatment. The interaction is marginally significant. A further comparison of prepost difference scores showed differential responses to therapy by the two groups.

significant interaction of group × prepost [$F(1,27)$ = 3.51, p = 0.07] (Fig. 5). This variable reflects the time that a patient needs for arm flexion, a movement accompanying walking. A further comparison of prepost difference scores showed differential responses to therapy by the two groups [one-tailed $t(27)$ = 1.86, p = 0.03].

4. *Movement coordination.* Coordination of movements of the upper limbs as reflected by the variable "initiation time of arm flexion minus initiation time of shoulder movement" showed an almost significant group × prepost interaction [$F(1,27)$ = 3.3, p = 0.08] (Fig. 6). This measure indicates how well arm movements are coordinated relative to the whole trunk when a patient starts to walk. Further comparisons of average prepost difference scores revealed no differential effects of treatment for the two groups. (Using the more stringent two-tailed tests, the majority of variables are still significant, except for the two variables "bending delay" and "time to maximal arm flexion," which were only marginally significant after two-tailed testing.)

DISCUSSION

The present results document changes in posture and gait related to behavioral treatment in PD patients. Equivalent groups of PD patients treated with behavioral and nonspecific methods, respectively, and tested before and after treatment, revealed the following: Clinical measures of motor performance (UPDRS motor examination) and general severity of PD (Hoehn & Yahr Scale) disclosed improvements in the behavioral group, but not in controls. A more fine-grained analysis of posture and gait by exact measuring of position and movement dynamics evidenced (a) no between-group differences in posture (trunk position before walking onset), (b) worsening of forward bending during walking in the control group over the 3-month interval, but not so in the be-

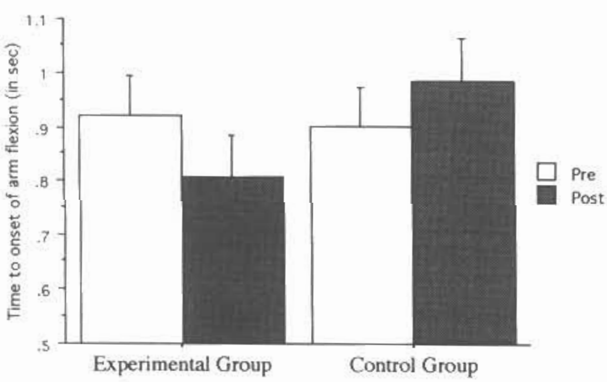

FIG. 4. Time to the onset of arm flexion relative to the starting signal in both treatment groups before and after treatment. The interaction is significant. There was a significantly faster onset of movements after treatment in only the experimental group and a performance decline in the control group.

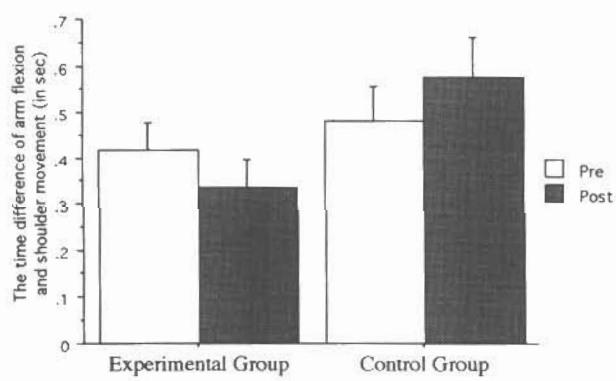

FIG. 6. The initiation time of arm flexion minus initiation time of shoulder movement in both treatment groups before and after treatment. The interaction is marginally significant. Further comparisons of average prepost difference scores revealed no differential effects of treatment for the two groups.

havioral group, and (c) quickening of gait initiation in the behavioral group, but no changes in controls. (d) There was only weak evidence, however, that deterioration of movement coordination in the control group was present, but not in the group receiving behavioral treatment. These data support a positive effect of behavioral treatment on clinical symptoms and motor performance of PD patients.

It is important to note that not only objective measures of position and postural dynamics produced evidence of positive effects of behavioral treatment, but that two clinical measures—UPDRS motor examination and Hoehn & Yahr Scale—also disclosed that changes were related to treatment methods. This finding supports the validity and clinical significance of results obtained with the objective measures.

Trunk bending is a major indicator of postural stability. As detailed in the introduction, bending forward while walking is one of the most characteristic features of PD. The severity of forward bending can be an indicator of the progression of the disease. It was, therefore, important that differential changes in forward bending could be observed for the two treatment groups. A nearly significant interaction of the group and prepost factors in the analysis of the delay of deviation in trunk position from its position at the start provides some evidence that performance deteriorated in the control group only. Further evidence comes from the significant decline found only in the control group. This suggests that behavioral treatment may contribute to a reduction in postural deterioration. The significant interaction of the factors group and prepost seen in the analysis of the degree of forward bending provides even more powerful evidence for a positive effect of behavioral therapy. Again, deterioration was found in the control group receiving nonspecific treatment, but no change was seen in the experimental group. It should be noted, however, that this variable revealed between-group differences before therapy (see Fig. 2). One may argue that performance of the control group before treatment was only accidentally better and that its decline merely reflects a return to baseline. Our view is that this is not very likely, because most of the other variables investigated provided at least some evidence for a better prepost relation in the experimental group compared with controls. The majority of the analyses of the control group patients' performance indicated a tendency to decline. It is very unlikely that an analysis leading to a significant result that is closely paralleled by significant or near-significant changes in related measures reflects an accidental change. Note, furthermore, that the patient groups did not exhibit significant differences with regard to various demographic and clinical variables. If several variables are being controlled for, it is impossible to exclude the possibility that well-controlled groups differ with regard to a small number of these variables. Taken together, both measures of delay and degree provide strong evidence that forward bending in PD can be improved by behavioral treatment. This is another strong argument for the use of behavioral therapy as a complement to the pharmacologic treatment of PD (24).

The worsening of forward bending in the control group observed over a period of only 10 weeks suggests that this measure is very sensitive to minimal behavioral changes and may be fruitful for clinical applications, in particular for the exact quantification of disease progression in PD patients.

The most pronounced evidence reported in this study, however, concerns the differential changes in movement initiation seen in the two treatment groups. Before therapy, both groups showed almost exactly the same time to onset of arm flexion. After therapy, initiation of movement improved in the experimental group, but declined in the control group. Further evidence for the beneficial effects on movement initiation comes from the almost significant interaction for the variable *time to maximal arm flexion*. Most importantly, planned comparisons showed different prepost treatment effects for the two groups. This result indicates that Parkinson patients' actual performance in movement initiation is significantly below what their competence would allow them.

The postural variable and the variable used for assessing motor coordination did not reveal any fully significant interactions. Therefore, conclusions about these measures cannot be drawn regarding differential changes in both patient groups. The lack of change in posture may be due to the fact that only mild-to-moderate cases were included in this study. Postural deficits were either absent or mild in these patients. Such changes may be obtained in patients in later stages of the disease. The lack of any significant changes in the variable measuring motor coordination may be due to the large variance in these data, suggesting that this variable may have had a low reliability. It should, however, be noted that all measures of coordination reflect the coordinated activity of large muscle groups and are therefore likely to exhibit large variation.

Some of the changes reported here are rather small, and one may argue that a change in movement initiation time of ~0.1 s or an increase of forward bending of 1° does not necessarily reflect a change that is relevant for the practical purposes of everyday life. However, it should be considered (a) that changes reported here were seen over the rather short period of 3 months, and (b) that many complex movements require a coordination of movements in the millisecond range and that a deterioration of ~100 ms is certainly substantial from this perspective. Thus, although the changes reported here are small, they may well have considerable consequences, especially if the changes observed continue over years.

Differential performance changes related to behavioral treatment are likely to result from the consequent use of processing resources still available in PD patients. We do not argue that the behavioral improvements indicate evidence of improvement in their neurologic condition. It may be more appropriate to state that behavioral therapy enables patients to make use of the competencies the disease has left them with.

On the basis of recent neurobiological research, however, it becomes more and more apparent that biochemical dysfunction (such as the inability of certain neurons to produce dopamine) is only one factor affecting the function of nervous tissue. Taub et al. (30) have shown that learned suppression of movements in animals after deafferentiation and in humans suffering from acute stroke is another relevant factor for the reduction of neurophysiologic activity and motor abilities. Taub and his colleagues even showed that behavioral training of acute-stroke patients that forced them to use the extremities affected by the stroke led to improved motor abilities. These and other data strongly support the view that what Taub calls learned nonuse is an important factor contributing to the reduction in nerve cell functioning and motor abilities in neurologic diseases. From this perspective, it appears likely that learned nonuse plays an important role also in the progression of PD. In this case, behavioral treatment could be an adequate method for surmounting learned nonuse and its negative effects. The present behavioral treatment may, therefore, improve the patients' conditions by saving them from additional neuronal dysfunction caused by learned nonuse. Note that behavioral treatment applied in this study included daily practice at home, so a higher activity level can be assumed for the behavioral group compared with the control group. To determine with certainty, however, whether learned nonuse plays a role in PD progression and whether it can be minimized by using behavioral techniques, additional studies are needed.

In conclusion, these data provide evidence that behavioral treatment can have significantly beneficial effects on movement initiation and forward bending during walking. Consistent with earlier studies (20,21), these results strongly suggest the use of behavioral therapy as a complement of the traditional treatment with L-dopa. However, additional studies are necessary in order to evaluate treatment-specific behavioral changes after a longer therapy period.

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