J Rehabil Med 2001; 33: 266–272

THE EFFECT OF A HOME PHYSIOTHERAPY PROGRAM FOR PERSONS WITH PARKINSON'S DISEASE

Alice Nieuwboer,[1] Willy De Weerdt,[1] René Dom,[2] Mieke Truyen,[3] Luc Janssens[4] and Yvo Kamsma[5]

From the [1]Department of Rehabilitation Sciences of the Faculty of Physical Education and Physiotherapy, [2]Department of Neuroscience and Psychiatry of the Faculty of Medicine, [3]Biostatistical Centre of the Faculty of Medicine of the Katholieke Universiteit Leuven, Belgium, [4]Institute of Technology, Groep T, Leuven, and [5]Department of Human Movement Sciences of the University of Groningen, The Netherlands

The purpose of this study was to evaluate the effect of a home physiotherapy program for persons with Parkinson's disease. Thirty-three patients took part in the study using a within-subject controlled design. Functional activities including walking and carrying out transfers were measured at home and in the hospital before and after a 6-week baseline period, after 6 weeks home physiotherapy and after 3 months follow-up. Spatiotemporal and plantar force variables of gait were determined with video and pododynography. Treatment provided by community physiotherapists consisted of teaching cueing and conscious movement control 3 times a week. The study revealed that patients had significantly higher scores on a functional activity scale after treatment in the home setting and to a lesser degree in hospital, a result, which was partly sustained at follow-up. However, duration of the transfer movements, spatiotemporal and plantar force variables were not significantly improved except for stride length. The results support application and development of the treatment concept and highlight that physiotherapy aimed at improving function in Parkinson's disease is best provided in the home situation.

Key words: Parkinson's disease, rehabilitation, physiotherapy, gait, effect.

J Rehabil Med 2001; 33: 266–272

Correspondence address: Alice Nieuwboer, Department of Rehabilitation Sciences, Faculty of Physical Education and Physiotherapy, Katholieke Universiteit Leuven, Tervuursevest 101, BE-3001 Leuven, Belgium. E-mail: alice.nieuwboer@flok.kuleuven.ac.be

(Accepted April 12, 2001)

INTRODUCTION

The effectiveness of physiotherapy in alleviating functional activities of people with Parkinson's disease (PD) has been subject to limited scientific scrutiny. This may be due to the fact that the intrinsic value of physiotherapy has been underestimated in comparison with the benefits of medication. Moreover, we found that the side effects of drug use complicated scientific evaluation (1).

A number of studies showed a significant short-term effect on function after programs of general exercise administered in either the home situation (2), as group training (3–5) or as individual hospital-based treatment (6). However, contemporary authors (7, 8) put greater emphasis on specifying the concepts behind rehabilitation in PD outlining two possible paradigms. The first is aimed at overcoming the physical limitations secondary to inactivity and disuse. In this context recent research has highlighted the potential for improving cardiorespiratory fitness (9, 10), trunk muscle strength (11) and spinal mobility (12) through specific training in mild to moderately affected patients. The second approach intends to tailor rehabilitation to insights into the specific nature of basal ganglia deficits (7, 13, 14). It is generally accepted that basal ganglia pathways control the automatic execution of movement generated in cortical motor areas (15). The core deficit underlying brady- and akinesia is the inability to drive motor output internally in well-learned sequential and complex tasks (16). Hence, physiotherapy methods may be able to compensate for the loss of the internal motor generator through the allocation of attention and external reference points (7, 13, 14). Numerous studies on the effect of verbal, visual and auditory cues established short term benefits on the quality of gait (17–20) and on overcoming initiation difficulties and freezing (21, 22). Some preliminary work indicated that implementing cues, cognitive planning and breaking down complex tasks into their component parts enhanced functional motor performance in individuals with considerable disease duration (13, 14).

While these studies indicate the potential for more optimal motor output, the most burning question for clinical practice is whether these strategies will transfer to conditions where sensory cues and attention are not available or otherwise engaged. The relevance of this issue came out of a series of experiments on training locomotion with visual cues and attention (18). Improvement of gait speed acquired through practice yielded no carry-over on performance the following day. Likewise, offering secondary cognitive tasks showed interference with the improved quality of gait. Both the continued dependence on external control and the so-called 'inflexibility of motor behaviour' often associated with PD (15) underscore the importance of rehabilitation of everyday actions in the very environment in which they usually take place.

Taking these findings together illustrate the need for further exploration of the value and feasibility of treatment modalities in line with the second rehabilitation paradigm. The present study is

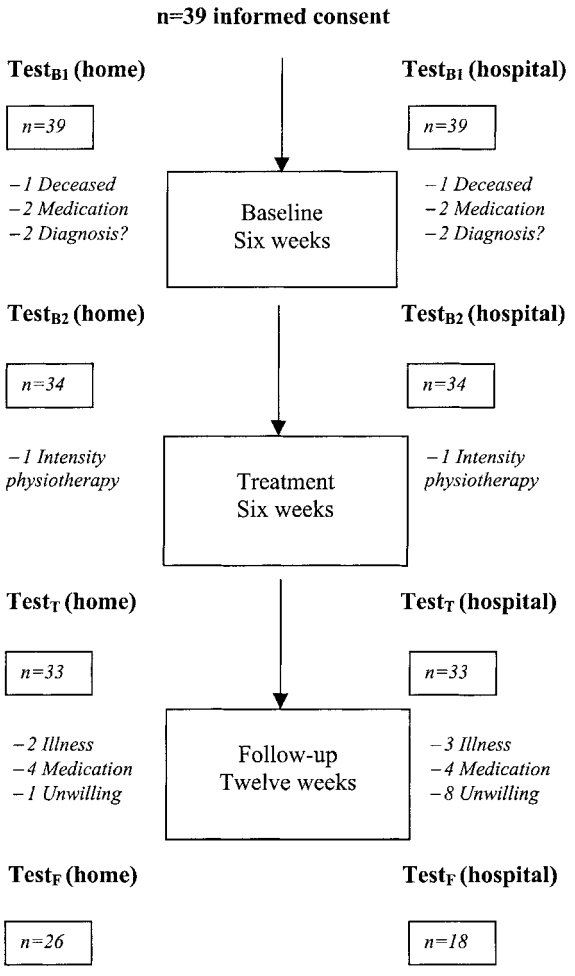

Fig. 1. Overview of study procedure and dropout.

meeting this need, as its primary objective is to investigate the effectiveness both at home and in a hospital environment of home physiotherapy providing external compensation methods.

METHODS

Study design

We used a within-subject controlled design accounting for the problems of heterogeneity and within-patient variability inherent in this population (1). After admission into the trial patients entered a baseline period of 6 weeks in which they did not receive physiotherapy (Fig. 1). Concurrent measurements of the main outcome variables took place in the home and the hospital within the same week, at onset and at completion of the 6-week baseline period (test$_{B1}$ and test$_{B2}$). Parallel measurements in both settings were repeated after a 6-week rehabilitation program (test$_T$) and a follow-up period of 3 months (test$_F$). Total duration of the study period was 6 months.

Intervention

Physiotherapy treatment aimed to reduce the specific difficulties experienced during functional activities rather than improving movement speed. Therefore, treatment was provided in the home setting. Treatment principles consisted of cueing, conscious control, biomechanical compensation and repetition in different circumstances to enhance the quality of gait, chair rising, bed mobility and the occurrence of gait blocks. For training bed mobility strategies described by Kamsma et al. (13) were adopted, involving structured sequences of movements adapted to each individual's needs. Treatment for rising from the chair entailed strategies to reposition the center of mass in relation to the base of support to compensate for slow trunk flexion and insufficient horizontal momentum (23). Gait aspects were trained using visual and auditory cues and self-instruction to improve step length, foot roll-off, posture, initiation, turning and freezing (7, 13, 18, 20, 21). Therapists decided for themselves which cue or instruction was most beneficial and would be included in the program. A therapist of choice provided 3 treatment sessions of 30 minutes a week for 6 weeks. Thirty-two physiotherapists participated in the study with an average age of 39 years (range 24–58 years) and a mean professional experience of 16.2 years (range 2–26 years). To ensure a standardized treatment approach:

1. Therapists received an instruction booklet and video outlining the rationale and execution of treatment.
2. The researcher checked adherence to the trial guidelines and treatment principles during a visit and aided in setting the specific goals for each patient.
3. Therapists had to record the frequency, duration and treatment strategies used, enabling post-trial exclusion if treatment did not meet the requirements of the study.

Subjects

Patients regularly attending a university hospital outpatient clinic for movement disorders volunteered to partake in the study. They were resident in the community within a radius of 1.5 hours' driving distance from hospital and selected from a cohort of 275 persons with PD over a period of 18 months. We recruited patients with a diagnosis of idiopathic PD according to accepted research criteria (24), with a stable medication regimen, with hindrance of functional disability, without current engagement in physiotherapy, without dementia (>23, Mini-Mental State Examination (MMSE) (25)), without acute other medical problems and with significant periods of the day free of severe dyskinesias and unpredictable off-periods.

Procedures

At entry into the study we classified patients into Hoehn & Yahr grades (26) in both "on" and "off" phases. The MMSE (25) documented the level of cognitive functioning. As a measure of disease severity we employed the Unified Parkinson's Disease Rating Scale (UPDRS) (27) at onset (test$_{B1}$), after treatment (test$_T$) and after follow up (test$_F$). Patients also completed the Dutch version of the 30-item Geriatric Depression Scale (28) (GDS-30) before and after therapy.

To control for the confounding effects of medication, subjects had to adhere strictly to their usual medication schedule. We reminded patients by telephone to comply with this measure before the day of testing. At each test we verified whether the regimen still applied and had been complied with. Also, we recorded the time of medication intake and standardized the timing of tests accordingly. Tests took place during the "on"-period, between 1 and 2.5 hours after taking the last L-dopa dose. Three times during each test patients rated themselves as having been in the "on", "wearing off" or "off" phase during the previously performed part of the test. These "on"–"off" ratings enabled post-hoc inclusion of data obtained during "on" only. We defined the "on" phase as the typical "peak dose" level of motor performance when the action of medication is considered optimal. We defined the "off" phase as the typical "end of dose" level of motor performance when the action of medication is strongly decreased or absent. "Wearing off" was considered as the unstable period in which patients are changing from "on" to "off".

Outcome measures

As the primary outcome measure we used an activity scale in both settings, which we had tested earlier for internal consistency, inter-rater and test–retest reliability (1). This 10-item instrument (range of scores 0–40) involved scoring the effectiveness of functional activities on a scale from zero to four in the following areas:

1. Initiating gait and turning, scoring the occurrence of hesitation, festination and freezing (2 items on gait akinesia).
2. Rising from and sitting down into a chair, scoring difficulties with controlling the center of body mass and the need for arm support (2 items on chair transfers).
3. Rolling and transferring in and out of bed, scoring difficulties with

axial mobility and achieving an adequate position in bed (3 items on bed mobility).
4. Handling the bed covers as well as moving in and out of bed, scoring difficulties with performing complex movement and double tasks (3 items on bed mobility with cover).

The same tester administered the activity scale in both the hospital and the home using standard instructions and test circumstances. In the hospital we recorded performance on the items of the activity scale on video. After completion of the trial, we randomized the order of the tests yielding a blind scoring procedure.

Additionally, the tester measured stride length and speed of sit to stand, supine to sit and gait using two-dimensional videotaping in the hospital. Video camera placement (two cameras) was standardized and optimized to avoid parallax error and increase measurement resolution. The cameras stood perpendicular to the plane of motion and were calibrated using a frame of reference behind the measurement target. For gait analysis patients walked on a 6-meter walkway at their normal comfortable speed. We calculated gait velocity and stride length from the markers attached to the shoes of the patient using custom-made software. Two independent testers established the inter-tester reliability of the procedure (ICC = 0.99). However, the same tester carried out the data processing for the purpose of this study. We also recorded the duration of a controlled protocol of chair and bed rises. Subjects stood up from a chair without armrests upon a verbal signal keeping the arms folded across the chest. The tester adjusted the seat height in relation to the length of the shank and the placement of the feet obtaining an angle of approximately 80° between lower legs and horizontal. In a similar fashion patients were asked to get up from supine lying to sitting from a bed of 40 cm height using their preferred method. The settings were standardized for subsequent measurements. During gait analysis we also recorded temporal data of the two most central strides of the walking trajectory and the plantar force distribution of the foot with a portable pododynography system (29). Patients wore standardized shoes available in different sizes with pressure sensitive insoles as part of the system. Each sole contained 64 pressure sensors. Sampling frequency was 50 Hz lasting 10 seconds. A detailed description of the measurement materials and calculation methods was given in a previous publication (29). For this study, we calculated the impulses or the plantar force/time products for the heel, mid-foot, forefoot and toes regions. We averaged 2 successful trials of the timed sit to stand and supine to sit movements and of 2 consecutive gait cycles for statistical analysis.

Data analysis

We calculated UPDRS scores by averaging scores from multiple body parts, in case of asymmetry of the most affected side. Results of the activity scale were summed into a total score and 4 sub-scores (gait akinesia, chair transfers, bed mobility and bed mobility with cover) as proposed in a previous investigation (1). The distribution of activity scale scores justified a parametric approach to data analysis. Missing values occurred as a result of technical failure, dropout at follow-up and random occurrence of "wearing off" or "off" phases. Considering the repeated measures design and the problem of missing values we used a linear mixed statistical model. The calculation of regression lines for each individual as well as for the entire group, inherent to this technique, corrected for missing values (30). We fitted the models using maximum likelihood estimations with the SAS PROC MIXED (31). Because of the exploratory nature of the study we did not correct for multiple testing. To investigate the occurrence of dropout at follow-up we performed logistic regression analysis. Age was included as a confounding variable in the regression models and interaction effects with age were checked but found insignificant ($p > 0.05$). To test if patients' mood changed after treatment we employed McNemar and Wilcoxon signed rank tests. We considered patients as either not depressed (GDS-30 score between 0 and 10) or depressed (11–20 mild depression, 21–30 severe depression) (28).

RESULTS

Subjects

Thirty-nine patients entered into the study. During the first 12 weeks 6 patients dropped out or were excluded from the trial (Fig. 1). One patient died due to heart failure. Two developed additional neurological symptoms possibly indicating non-idiopathic PD. Two subjects required adjustments of medication and one patients' rehabilitation program did not fulfill the intensity criteria of the study. We were left with 33 patients, who completed the baseline and treatment periods. At 6 months follow-up 7 patients dropped out from the tests because 2 patients became ill from other medical conditions than PD, 1 was unwilling to cooperate and 4 required alterations of their medication regimen. A further 8 patients dropped out for the hospital follow-up, as one patient developed acute thrombosis between the home and hospital tests and 7 were unwilling to cooperate because of the traveling involved, leaving a total of 18 participants.

The 33 patients who completed the first 3 months of the trial were 21 males and 12 females mainly of Hoehn & Yahr grade II.5 ($n = 15$) and III ($n = 16$) during the "on" phase as demonstrated in Table I. In the "off" phase 21 persons were in grade IV. Mean Mini Mental Scale score was 26.9 (range 23–30). Table I summarizes the daily medication regimens patients were taking during the study period. The most frequently concurring medical problems affecting mobility were chronic back pain ($n = 6$) and peripheral arthroses ($n = 6$). Three patients had had previous hip fractures. Five patients reported cardiovascular problems and 3 chronic pulmonary disease in addition to PD.

Sixteen patients suffered from recurring "on"–"off" fluctuations during the day as measured by the UPDRS. During actual testing 7 patients in the hospital and 4 in the home experienced a random change of their "on" phase. Sixteen subjects reported to have choreiform movements for some time during the day. The level of dyskinesias experienced during test sessions was mild to moderate in 5 patients and marked in 3 cases. No dystonia was observed.

Effect of treatment on the activity scale scores

Table II shows the results of the linear mixed model on the functional activity scale in the home and hospital setting estimating the size and significance of the effects of the baseline, treatment and follow-up periods. A mild improvement of activity scale scores was apparent during the baseline period at home, significant for gait akinesia ($p = 0.04$), bed mobility with manipulating the cover ($p = 0.03$) and total function ($p = 0.02$). None of the baseline increments were significant in the hospital setting. After the treatment period, activity scale scores improved significantly in all areas at home ($0.0001 < p < 0.004$). The size of these effects was estimated to vary from 0.65 to 1.95 points on the scale for the different functional activities. Total activity scale scores at home increased with 5.2 points implying an increment of 21.5% of the mean initial score. In the hospital significant improvements were found for gait akinesia, chair transfers and total activity scale scores after treatment ($0.0001 < p < 0.007$). Changes of bed mobility with and without cover were either borderline or not significant

Table I. *Clinical characteristics of the patients* (n = *33*)

	n		Mean (range)
Hoehn & Yahr	"on"	"off"	
II	2	0	
II.5	15	1	
III	16	10	
IV	0	21	
V	0	1	
Age (years)			66.2 (49–81)
Disease duration (years)			12.1 (4–25)
Sex			
Female	12		
Male	21		
Daily dose of medication (mg)			
Levodopa	33		640.7 (200–1000)
Bromocriptine	6		22.5 (15–30)
Pergolide-mesylate	12		2.3 (0.75–3)
Amantadine-HCl	17		161.8 (100–200)
Selegeline-HCl	11		7.7 (5–10)

($p = 0.05$ and $p = 0.29$). Overall effect size was smaller in the hospital. Total activity scale scores enhanced with 2.28 (8.6%) points on the scale.

Because a consistent "baseline effect" was detected, albeit not significant in most cases, the impact of therapy was further explored assuming that this effect would recur during the treatment period. After subtraction of baseline increments the scores remained significant for chair transfers ($p = 0.04$), bed mobility without and with cover ($p = 0.03$ and 0.002) and the total activity scale ($p = 0.0008$) after treatment at home. In the hospital chair transfers continued to display a significant effect ($p = 0.002$).

Looking at the effect of treatment after three months follow-up revealed that the estimated change of activity scale scores was smaller compared with that of immediately after treatment. However, both at home ($p < 0.0007$, 12.6%) and in the hospital ($p = 0.03$, 5%) total activity scale scores still showed a significant difference compared with the second baseline measurement.

Whereas Table II displays the effects predicted by the statistical model, Fig. 2 depicts the actually observed mean total scores at the four tests in both environments. From this figure it is apparent that at baseline patients performed overall better in the hospital than in the home situation, differences, which did not reach statistical significance ($p = 0.09$). A continued improvement is shown between $test_T$ and $test_F$ in the hospital, which was not predicted by the linear mixed model. To explore this discrepancy the considerable dropout of particularly the hospital evaluations was further investigated. Logistic regression exposed that patients with lower total activity scale scores at baseline and after treatment in the home situation had a significantly greater chance to drop out at follow-up with *p*-values ranging from 0.02 to 0.03 and intercepts from 3.2 to 4.1. From the hospital scores predictions were almost significant ($p = 0.08$ and 0.09, intercept = 3.5 and 3.8). This outcome justifies the use of the linear mixed approach in correcting for inflation of the results due to dropout of the worst cases.

Effect of treatment on additional variables

Table III summarizes the effects on spatiotemporal gait parameters as predicted by the statistical model. At the first baseline evaluation patients walked with a mean velocity of 0.87 meter/second (±0.23), mean stride of 0.99 meter (±0.23), mean cadence of 103.8 steps/minute (±12) and mean double support phases of 26.6% (± 6.2). Baseline instability was not meaningful. A significant treatment effect ($p = 0.004$) was recorded for stride length which was estimated to increase with 0.09 meter after therapy (9%). The net effect of this improvement on speed was counteracted by an almost significant reduction of cadence with 3.7 steps per minute ($p = 0.08$) resulting in a moderate velocity increase of 0.035 meter/second. Double support

Table II. *Estimate effects (β), the standard errors (s.e.) and the significance (p) of the activity scale scores in the home and the hospital. The number of subjects (n) varied due to dropout and to random occurrence of "off" or "wearing "off" as rated by the patients themselves during performance of various parts of the activity scale*

	Baseline effect ($test_{B2} - test_{B1}$)		Treatment effect ($test_T - test_{B2}$)		Follow-up effect ($test_F - test_{B2}$)	
	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*
Home (range)	*n* = 31–33		*n* = 31–32		*n* = 25–26	
Gait akinesia (0–8)	0.45 (0.21)	0.04	0.65 (0.21)	0.004	0.16 (0.24)	0.52
Chair transfer (0–8)	0.05 (0.23)	0.84	0.99 (0.29)	0.002	0.45 (0.27)	0.11
Bed mobility (0–12)	0.44 (0.29)	0.15	1.57 (0.34)	0.0001	1.27 (0.32)	0.0005
Bed mobility +cover (0–12)	0.61 (0.27)	0.03	1.95 (0.26)	0.0001	1.32 (0.44)	0.006
Total score (0–40)	1.61 (0.64)	0.02	5.16 (0.60)	0.0001	3.04 (0.79)	0.0007
Hospital (range)	*n* = 26–31		*n* = 26–32		*n* = 15–17	
Gait akinesia (0–8)	0.01 (0.03)	0.66	0.08 (0.03)	0.007	0.06 (0.03)	0.06
Chair transfer (0–8)	0.14 (0.19)	0.46	1.26 (0.21)	0.0001	0.48 (0.27)	0.09
Bed mobility (0–12)	0.21 (0.34)	0.55	0.54 (0.26)	0.05	0.63 (0.34)	0.08
Bed mobility +cover (0–12)	0.30 (0.28)	0.29	0.39 (0.36)	0.29	0.44 (0.36)	0.23
Total score (0–40)	0.75 (0.48)	0.13	2.28 (0.61)	0.001	1.53 (0.65)	0.03

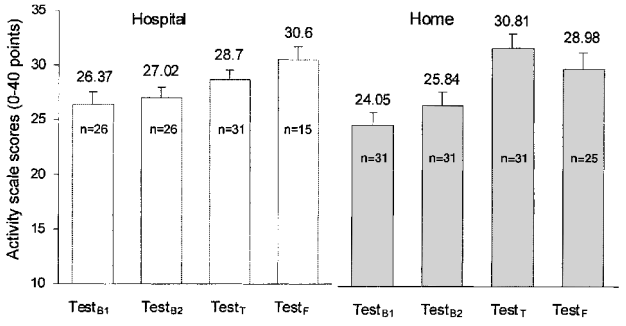

Fig. 2. Means and standard errors of the total activity scale scores in the hospital and home setting at baseline (test$_{B1}$ and test$_{B2}$) after treatment (test$_T$) and after follow-up (test$_F$).

duration decreased after treatment but not significantly. Effects on roll-off of the foot as reflected by the plantar force distribution patterns are not represented in Table III rendering insignificant results. At follow-up the improvement of stride length disappeared and was no longer meaningful when compared with baseline levels (test$_T$ – test$_{B2}$). Unlike the predicted decrease of stride length at follow-up, an actual mean increase of 0.025 meter was measured, resembling the discrepancy between estimated and actual results for total function in the hospital discussed earlier. Results of logistic regression analysis confirmed that patients with a better gait profile were more likely to remain in the study at follow-up. Shorter stride lengths after the baseline (intercept = 4.5, $p = 0.04$) and treatment periods (intercept = 8.2, $p = 0.03$) significantly predicted dropout. Patients' mean duration of rising from a chair being 2.46 second (±0.68) at onset of the study did not significantly alter throughout the study period nor did the duration of moving from supine to sit ($\bar{X} = 10.5 \pm 5.1$ second).

Non-specific effects

Before treatment 19 patients were considered not depressed and 14 mildly depressed as indicated by the Geriatric Depression Scale (28) (GDS-30). After treatment 4 previously (mildly) depressed patients became not depressed and in one patient the opposite change occurred, a result, which was not significant ($p = 0.18$). Alterations of the mean GDS-30 score were also not meaningful ($p = 0.12$).

Patients presented with a mean UPDRS total score ("on" phase) of 50.5 (±10.8) at baseline. We found a significant ($p = 0.02$) mean improvement of 2.4 points of UPDRS scores after treatment, 4.8% of the initial score, which returned to baseline levels at follow-up. Effects on the subscales of the UPDRS were only significant for the ADL-section (part II) which got better immediately after treatment ($p = 0.03$, $\beta = -1$) but deteriorated at follow-up.

DISCUSSION

Rehabilitation professionals need to know whether providing specific physiotherapy to functionally impaired individuals with PD is worthwhile. From earlier studies the appropriateness of teaching conscious control to replace the disrupted automatic motor planning emerged (13, 14, 18). The current study supports the use of this rehabilitation concept as implementing such treatment at home led to more effective performance of functional activities in patients with a mean disease duration of 12 years. Clinically relevant improvement was demonstrated on a specific activity scale (8.6–21.5%) which was confirmed by the UPDRS (4.8%). The impact is modest compared with that of medication, established in similar populations to be 37% on function, 35% on gait velocity and 30% on stride length (32). However, in view of the presence of significant functional disability, possible improvement from physiotherapy may be a meaningful addition to pharmacological treatment.

We propose that one of the mechanisms underlying the improvement may stem from the conscious activation of motor cortex overriding the loss of basal ganglia function. Recent research suggested that over-activity in the unaffected cerebellar and lateral premotor routes may signify an adaptive mechanism through which patients can use sensory or attentional guidance to overcome their movement disorders (33). A second mechanism may be that repeating the functional movements in themselves is beneficial, especially when under the influence

Table III. *Estimate effects (β), standard errors (s.e.) and the significance (p) of the spatiotemporal gait variables. The number of subjects (n) varied due to dropout and to random occurrence of "off" or "wearing off" as rated by the patients themselves during performance of various parts of the activity scale*

	Baseline effect (test$_{B2}$ − test$_{B1}$)		Treatment effect (test$_T$ − test$_{B2}$)		Follow-up effect (test$_F$ − test$_{B2}$)	
	β (SE)	p	β (SE)	p	β (SE)	p
Hospital	$n = 26$		$n = 26$–32		$n = 14$	
Velocity (meter/second)	0.027 (0.02)	0.13	0.035 (0.02)	0.12	0.01 (0.03)	0.61
Stride length (meter)	0.03 (0.02)	0.11	0.09 (0.004)	0.004	0.03 (0.03)	0.29
Cadence (steps/minute)	0.11 (1.76)	0.95	−3.7 (2.0)	0.08	−1.18 (1.91)	0.25
Double support phase (%)	1.7 (0.9)	0.06	−1.5 (1.0)	0.16	−0.2 (0.74)	0.79

$n = 39$ informed consent.

of cues the execution of functional activities occurs in the most optimal way.

An interesting and novel finding was that the effect was most convincing in the very context in which training took place. Improvement of functional activity scores at home were more than twice those observed in hospital. In our view, this discrepancy may be explained by a better retention of treatment strategies within the actual learning-context. Motor learning in healthy subjects is generally regarded as highly task-specific but evidence on context-specificity is more limited (34). Over and above these general learning issues, the intrinsic features of basal ganglia deficits and associated cognitive change may clarify the moderate transfer to a different environment. PD-patients' inability to internally drive motor output results in a striking sensitivity to the external and mental conditions under which motor behavior is performed. Inasmuch as this factor is used to its advantage in therapy it may also assume limitations where generalization is concerned. The cognitive dysfunction in non-demented patients with PD in terms of shifting appropriate attention to new demands and activating memory processes may exacerbate these difficulties (35). We therefore interpret the context-specific results as an integral part of the disease which may prove hard to redress as patients have lost the ability to internally self-generate their maximal motor output. For clinical practice this underscores the relevance of providing rehabilitation in the home situation as well as practicing a variety of tasks in different conditions and circumstances.

The weaker results found in hospital may also be due to the methodological limitations of this study. The use of video-technology permitted retrospective randomization of tapes and blind scoring in hospital. Both for practical reasons and to create a familiar test atmosphere no equipment was used at home where awareness of the test order may have created observer bias.

The improvements found essentially indicate that patients had less specific difficulties in accomplishing the activities trained, i.e. turning with less hesitation and freezing, rising from a chair without falling back and achieving a comfortable position in bed. Increments of the UPDRS ADL-section rather than of the motor examination part confirm that the benefits of therapy occur at activity rather than at impairment level of the ICIDH hierarchy (36). No meaningful change occurred of the duration of test movements demonstrating an acceleration of performance and accentuating the contents-specificity of the effects. The isolated improvement of stride length without carry-over to gait speed and roll-off of the foot provides further evidence of specificity. Training was focused on normalizing the typical gait deficits of reduced stride length and roll-off of the foot rather than on producing a faster gait as such. Moreover, during evaluation patients were asked to walk with normal speed and were not reminded of the contents of therapy. The trend towards a reduced mean cadence supports the view that patients utilize stepping rate as an adaptive mechanism for the fundamental loss of stride length (18). Although speed did not improve, it can be argued that patients' gait normalized in terms of the stride/cadence relationship. However, normalization did not reflect itself in an alteration of plantar force distribution. This is in agreement with other studies (37, 38) in which correction of kinematic and spatiotemporal variables coincided with persistent abnormalities of the kinetics of gait in response to visual cues. The fact that force production at the foot remained abnormal despite intervention raises questions as to the exact working mechanism of cues and self-instruction. More research into this area is called for clarifying how this mechanism may be exploited to greater benefit. In agreement with findings on general exercise (4, 6) the effects of intervention subsided 3 months after therapy but performance of the trained activities in both settings continued to be significantly better with the exception of stride length. This result was obtained while statistically correcting for dropout. A need for adjustment of medication and a waning commitment to the trial were the most frequent reasons for dropout. Patients with poorer motor performance ran a higher risk of dropping out. The illustrated vulnerability of the population in relation to the demands of the study imposes limitations on future study design. The size of the effects, the heterogeneity of PD in general and the drug side effects inherent to prolonged disease argue against the use of a control group in studies constrained by limited numbers of available patients. The present design provided a within-subject controlled design. Baseline results were not always stable. Several explanations must be explored in this context. A learning effect is conceivable but unlikely considering that when six repeated tests of the same functional activities were carried out in one day no such effects were evident (1). Baseline improvement might have been caused by patients' raised expectations and enhanced psychological well being, factors that were suggested to constitute part of the effect of rehabilitation in PD in other studies (10, 13). However, no evidence of positive mood changes was found. Future studies should allow more frequent repeated measures to enable accurate prediction of the slope of baseline effects taking into account the possibility of dropout of the most vulnerable patients.

The patients included in this study were without severe fluctuations and cognitive decline. They displayed a willingness to go through multiple measurements sometimes requiring considerable travel. The implied cooperation of the population projects onto the generalization of the results. On the other hand rehabilitation was provided by a sample of average rather than specialized community therapists, a notion, which stresses the pertinence of the outcome.

This within-patient controlled trial provides some new insights into the applicability and value of physiotherapy addressing activities, instrumental to preserving independence in advanced PD. Stimulating reliance on contextual stimuli and conscious control seem effective as a compensatory mechanism to improve function, an effect, which is partly sustained at 3 months follow-up. However, the limitations imposed by basal ganglia dysfunction on the capacity for permanent learning also emerge as limited transfer occurs to a different context. The specificity of the effects in terms of context and contents has to

be taken on board by the rehabilitation profession and by those who refer patients to the service. Therapists have to advance the paradigm identifying methods to promote generalization and to address the specific effects of cues.

ACKNOWLEDGEMENTS

We gratefully acknowledge the contribution of the Research Council of the Katholieke Universiteit Leuven and the Fund of Scientific Research-Flanders and the patients who cooperated so willingly with this study.

REFERENCES

1. Nieuwboer A, De Weerdt W, Dom R, Bogaerts K, Nuyens G. Development of an activity scale for individuals with advanced Parkinson's disease: reliability and "on–off" variability. Phys Ther 2000; 80: 1087–1096.
2. Banks MA, Caird FI. Physiotherapy benefits patients with Parkinson's disease. Clin Rehabil 1989; 3: 11–16.
3. Formisano R, Pratesi L, Modarelli FT, Bonifati V, Meco G. Rehabilitation and Parkinson's disease. Scand J Rehabil Med 1992; 24: 157–160.
4. Comella CL, Stebbins GT, Brown-Toms N, Goetz C. Physical therapy and Parkinson's disease: a controlled clinical trial. Neurology 1994; 44: 376–378.
5. Dam M, Tonin P, Casson S, Bracco F, Piron L, Pizzolato G. Effects of conventional and sensory-enhanced physiotherapy on disability of Parkinson's disease patients. Adv Neurol 1996; 69: 551–555.
6. Patti F, Reggio A, Nicoletti F, Sellaroli T, Deinite G, Nicolettit Fr. Effects of rehabilitation therapy on Parkinsonian disability and functional independence. J Neurol Rehabil 1996; 10: 223–231.
7. Morris ME, Iansek R. Characteristics of motor disturbance in Parkinson's disease and strategies for movement rehabilitation. Hum Mov Sci 1996; 15: 649–669.
8. Cutson TM, Cotter Laub K, Schenkman M. Pharmacological and nonpharmacological interventions in the treatment of Parkinson's disease. Phys Ther 1995; 75: 363–373.
9. Canning CG, Alison JA, Allen NE, Groeller H. Parkinson's disease—an investigation of exercise capacity, respiratory-function and gait. Arch Phys Med Rehabil 1997; 78: 199–207.
10. Bridgewater KJ, Sharpe MH. Aerobic exercise and early Parkinson's disease. J Neurol Rehabil 1996; 10: 233–241.
11. Bridgewater KJ, Sharpe MH. Trunk muscle training and early Parkinson's disease. Physiotherapy Theory Pract 1997; 13: 139–153.
12. Schenkman M, Cutson TM, Kuchibhatla M, et al. Exercise to improve spinal flexibility and function for people with Parkinson's disease: a randomized, controlled trial. J Am Ger Soc 1998; 46: 1207–1216.
13. Kamsma YPT, Brouwer WH, Lakke JPWF. Training of compensational strategies for impaired gross motor skills in Parkinson's disease. Physiotherapy Theory Pract 1995; 11: 209–229.
14. Yekutiel MP, Pinhasov A, Shahar G, Sroka H. A clinical trial of the re-education of movement in patients with Parkinson's disease. Clin Rehabil 1991; 5: 207–214.
15. Marsden CD, Obeso JA. The functions of the basal ganglia and the paradox of stereotaxic surgery in Parkinson's disease. Brain 1994; 117: 877–897.
16. Cunnington R, Iansek R, Bradshaw JL, Phillips JG. Movement-related potentials in Parkinson's disease: presence and predictability of temporal and spatial cues. Brain 1995; 118: 935–950.
17. Bagley S, Tunnicliffe N, Kelly B, Turnbull GI, Walker JM. The effect of visual cues on the gait of independently mobile Parkinson's disease patients. Physiotherapy 1991; 77: 415–420.
18. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease: normalization strategies and underlying mechanisms. Brain 1996; 119: 551–568.
19. Thaut MH, McIntosh, Rice RR, Miller RA, Rathbun J, Brault JM. Rhythmic auditory stimulation in gait training for Parkinson's disease patients. Mov Dis 1996; 11: 193–200.
20. Behrman AL, Teitelbaum P, Cauragh JH. Verbal instructional sets to normalise the temporal and spatial gait variables in Parkinson's disease. J Neurol Neurosurg Psych 1998; 65: 580–582.
21. Nieuwboer A, Feys P, De Weerdt W, Dom R. Is using a cue the clue to the treatment of freezing in Parkinson's disease. Physiotherapy Res Int 1997; 2: 125–134.
22. Burleigh-Jacobs A, Horak FB, Nutt JG, Obeso JA. Step initiation in Parkinson's disease: influence of levodopa and external sensory triggers. Mov Dis 1997; 12: 206–215.
23. Hughes MA, Weiner DK, Schenkman ML, Long RM, Studenski SA. Chair rise strategies in the elderly. Clin Biomech 1994; 9: 187–192.
24. Ward CD, Gibb WR. Research diagnostic criteria for Parkinson's disease. Adv Neurol 1990; 53: 245–249.
25. Dick JPR, Guiloff RJ, Stewart A, Blackstock J, Bielawska C, Paul EA. Mini-mental state examination in neurological patients. J Neurol Neurosurg Psychiatry 1984; 47: 496–499.
26. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. Neurology (Cleveland) 1967; 5: 427–442.
27. Fahn S, Elton RL & UPDRS Committee. Unified Parkinson's disease rating scale. In: Fahn S, Marsden CD, Goldstein M, Calne DB, eds. Recent developments in Parkinson's disease, Vol. 2. Florhem Park, New York: Macmillan Health Care Information; 1987. p. 153–163.
28. Emanuels-Zuurveen ES, Brouwer WH, Lakke JPWF, Bowhuys AL. Physical symptoms of Parkinson's disease and the score on the Beck's Depression Inventory. Tijdschr Gerontol Geriatr 1991; 22: 134–137.
29. Nieuwboer A, De Weerdt W, Dom R, Peeraer L, Lesaffre E, Baunach B. Plantar force distribution in patients with Parkinson's disease: a comparison between patients and normal controls. Scand J Rehabil Med 1999; 31: 185–192.
30. Verbeke G, Molenberghs G.Linear mixed models in practice. A SAS-oriented approach. Lecture notes in statistics 126, New York: Springer-Verlag; 1997.
31. Littell RC, Milliken GA, Stroup WW, Wolfinger RD. SAS system for mixed models, 1st ed. Cary: SAS Institute Inc.; 1996.
32. O'Sullivan JD, Said CM, Dillon LC, Hoffman M, Hughes AJ. Gait analysis in patients with Parkinson's disease and motor fluctuations: influence of levodopa and comparison with other measures of motor function. Mov Dis 1998; 13: 900–906.
33. Samuel M, Caballos-Baumann AO, Blin J, Uema T, Boecker H, Passinham RE. Evidence for lateral premotor and parietal over-activity in Parkinson's disease during sequential and bimanual movements. A PET study. Brain 1997; 120: 963–976.
34. Schmidt RA, Lee TD. Conditions of practice. In: Motor control and learning: a behavioral emphasis, 3rd ed. Champaign: Human Kinetics; 1999. p. 285–321.
35. Dubois B, Pillon B, Malapani C, Deweer B, Verin M, Partiaud A. Subcortical dementia and Parkinson's disease: what are the cognitive functions of the basal ganglia. In: Wolters ECh, Scheltens P, eds. Mental dysfunction in Parkinson's disease, 1st ed. Dordrecht: ICG Publications; 1994. p. 195–207.
36. ICIDH-2 International Classification of Impairments, Activities and Participation. Beta-1 draft for field trials. 1997.
37. Forssberg H, Johnels B, Steg G. Is Parkinsonian gait caused by a regression to an immature walking pattern? Adv Neurol 1984; 40: 375–379.
38. Morris ME, McGinley J, Huxham F, Collier J, Iansek R. Constraints on the kinetic, kinematic and spatiotemporal parametres of gait in Parkinson's disease. Hum Mov Sci 1999; 18: 461–483.