Physical therapy and Parkinson's disease:

A controlled clinical trial

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Article abstract—In a randomized, single-blind, crossover study, we evaluated physical disability in moderately advanced Parkinson's disease (PD) patients after 4 weeks of normal physical activity and 4 weeks of an intensive physical rehabilitation program. We used a timed motor task and a standard assessment of PD severity (the Unified Parkinson's Disease Rating Scale [UPDRS] with subscales for mentation, activities of daily living [ADL], and motor function) completed by an investigator blinded to the physical rehabilitation status of the patient. Following physical rehabilitation, there was significant improvement in the UPDRS ADL and motor scores, but no change in mentation score. During the 6 months following physical rehabilitation, patients did not regularly exercise, and the UPDRS scores returned to baseline. We conclude that physical disability in moderately advanced PD objectively improves with a regular physical rehabilitation program, but this improvement is not sustained when normal activity is resumed.

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Physical rehabilitation is used in conjunction with antiparkinson agents as a nonpharmacologic treatment for Parkinson's disease (PD). Physical therapy was particularly emphasized prior to the introduction of levodopa,[1,2] but its efficacy in PD has not been extensively studied, and most studies were unblinded and used only global assessments of physical or disease status.[3-6] We conducted a single-blind, crossover study to test whether a regular physical rehabilitation program improved clinical function as measured by a standardized PD rating scale. Since most physical therapy programs are short-term, we additionally assessed whether gains made after 1 month of physical therapy were maintained during the next 6 months with normal activities.

Methods. *Study design.* Consecutive PD patients with Hoehn and Yahr stage 2 or 3 who were not demented (Mini-Mental State Examination score greater than 27) and were without significant other medical or orthopedic conditions were invited to participate. This prospective, randomized, crossover study was divided into two 4-week periods separated by 6 months. Patients were randomized to the physical rehabilitation program during the first or second period. All patients were evaluated at baseline, following the 4-week control and physical therapy phases, and 6 months after completion of each phase. Medications were not changed during either phase, although medication adjustments were permitted during the 6-month interval between study periods.

Physical rehabilitation phase. The physical rehabilitation program was administered by licensed, experienced physical and occupational therapists. The program was a modification of that described by Wroe and Greer,[7] and consisted of a series of 69 repetitive exercises to improve range of motion, endurance, balance and gait, and fine motor dexterity. Exercise periods were 1 hour in duration and occurred three times a week for 4 consecutive weeks. The number of repetitions for each exercise was increased progressively throughout the rehabilitation period as endurance increased. Following the physical therapy phase, patients were instructed to continue the exercise program at home.

Control phase. During the control phase, patients maintained their usual level of physical activity without any specific exercise program or additional instruction. Following the control phase, patients were instructed to continue their usual activities.

Evaluations and statistical analysis. All patients were evaluated by a neurologist (C.L.C.) blinded to the patient study grouping, using the Unified Parkinson's Disease Rating Scale (UPDRS)[8] and timed finger taps.[9] Depression was assessed by the Geriatric Depression Scale (GDS).[10] All patients were evaluated at baseline, at the end of the 4-week phase (both the control and physical therapy phases), and 6 months following completion of each phase. Patients were examined in the morning at the peak-dose effect of their antiparkinson medication.

A nonparametric Friedman's ANOVA was employed to assess overall treatment effect. Post hoc analysis of specific effects was performed using a Wilcoxon signed rank statistic for the following measures: total UPDRS score, UPDRS scores for mental functioning, activities of daily living (ADL), and motor evaluation, as well as GDS score.

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Table. **Changes following rehabilitation program**

	Rehabilitation			No rehabilitation		
	Before	After	p*	Before	After	p*
Total UPDRS (SD)	39 (8)	31 (8)	0.002	37 (7)	35 (8)	NS
Mental subsection (SD)	2 (1)	2 (2)	NS	2 (1)	2 (2)	NS
ADL subsection (SD)	12 (4)	8 (3)	0.005	10 (4)	10 (3)	NS
Motor subsection (SD)	26 (6)	20 (7)	0.007	25 (8)	23 (9)	NS
Geriatric Depression Scale (range)	15 (0-31)	13 (0-32)	NS	15 (0-31)	14 (0-33)	NS

* With Bonferroni adjustment, alpha = 0.008.

A previous study of the factor structure of the motor subsection of the UPDRS demonstrated that the scale could be decomposed into separate evaluations of bradykinesia, rigidity, rest tremor, and action tremor.[11] The bradykinesia factor was the summation of midline (speech, facial expression, arising from a chair, posture, gait, postural stability, and body bradykinesia) and limb (right and left hands and feet) bradykinesia scores; the rigidity factor was the sum of the rigidity scores for all extremities and neck; the tremor factor was the sum of the tremor score for right and left side, and head and neck; and the postural/action tremor score was the score for postural or action tremor for the upper extremities.[11] To assess differential effects of physical rehabilitation on these factors, a Wilcoxon signed rank statistic was used to compare factor scores before and after each study condition.

Changes in the mean of the timed performance for finger tapping after rehabilitation and control phases were assessed with paired t tests.

Due to the large number of statistical analyses required in this study, we controlled for the increased likelihood of finding spurious significance by employing a Bonferroni adjustment for all analyses.

Results. Of the 18 patients initially enrolled in the study, two were excluded for missed visits during the program. Four women and 12 men completed the study. At study entry, the mean age was 66 years (SD 8), mean PD duration was 10 years (SD 7), median Hoehn and Yahr stage was 2.3 (range, 2 to 3), and median GDS score was 15 (range, 0 to 31). All patients received antiparkinson medications during the study period—anticholinergic agents (four patients), amantadine (nine patients), selegilene (nine patients), levodopa (13 patients, mean dose 696 mg, SD 435), and pergolide or bromocriptine (six patients, mean dose 15 mg, SD 6 in bromocriptine equivalents in which 1 mg pergolide is equivalent to 10 mg bromocriptine). Although there were no overall significant differences in dosage of any drug, during the 6-month period following active rehabilitation, levodopa therapy was initiated in one patient, bromocriptine in one patient, and amantadine discontinued in one patient.

Analysis of changes in UPDRS total score revealed a significant treatment effect (Friedman's ANOVA = 25, $df = 3$, $p < 0.001$). Post hoc comparisons of specific changes revealed that following physical therapy, patients significantly improved in total UPDRS and in the ADL and motor subsections (table). Examination of the factors constituting the motor section of the UPDRS showed that the rigidity and bradykinesia factors improved significantly following rehabilitation (bradykinesia factor: 18 [SD 5] versus 14 [SD 5], $p = 0.009$; rigidity factor: 7 [SD 2] versus 5 [SD 3], $p = 0.005$), while action and rest tremor factors did not change. Timed finger taps and GDS score did not change following physical therapy.

Six months following the rehabilitation phase, gains for total UPDRS and for the ADL and motor subsections returned to baseline.

Discussion. This study is the first to evaluate the effect of a systematic program of physical therapy on standardized measures of PD severity using a prospective, single-blinded, crossover design. Our results show that moderately disabled PD patients objectively improve following 4 weeks of regular physical exercise, but when active therapy is terminated, these improvements return to baseline after 6 months. Franklyn and Stern[12] also reported modest benefit from physical therapy. Gibberd et al,[13] however, found no short-term benefit in 17 PD patients attending a physical therapy program compared with matched PD control subjects attending physical therapy sessions but not actively participating. The discrepancies between these studies may be attributed to differences in the rigor of clinical assessment as well as in patient age, severity of PD, and concomitant medications.

The motoric gains we documented could be multidimensional in origin. Although most exercises in physical rehabilitation programs are not specifically directed toward increasing muscle bulk and strength, Palmer et al[4] found that grip strength improved following both karate training and stretching exercises in PD subjects. Practice can improve the speed of the movement,[14] and preparedness for a motor movement may be augmented with repetition.[15] Worringham and Stelmack[15] showed that PD patients improved their performance in simple reaction time when repeated trials were given. We do not feel that the improvement in our patients was the specific result of a practice effect since the rehabilitation program did not involve the actual tasks of the UPDRS. Furthermore, the selective improvement in certain fac-

tors constituting the UPDRS suggests that bradykinesia and rigidity benefit preferentially from rehabilitation. The lack of change in depression score or in the mentation subscale of the UPDRS argues against a significant role for psychological factors as the basis for the improvement seen following rehabilitation.

Pharmacologic explanations for the motor changes could include exercise-induced redistribution of blood flow away from the gastrointestinal system to the muscles, with a resultant alteration in medication absorption. However, if this occurred, levodopa absorption would be predicted to decrease with exercise and, in contrast to the findings of this study, worsen motor performance. Previous investigators have in fact shown that active exercise does not have a regular effect on plasma levodopa levels.[16] Furthermore, we examined patients on the day following the final day of physical therapy, when exercise-induced changes in blood flow would be expected to normalize.

The gains we documented were not long-standing once the physical rehabilitation program ceased. We were impressed that, although all patients received instructions to continue the exercises at home at the completion of the rehabilitation phase, every patient resumed a more sedentary life-style. This observation suggests that physical rehabilitation is not easily incorporated into a patient's life-style at home and that active physical therapy is better assured by an organized physical therapy program.

Although motor improvements were no longer apparent at the 6-month follow-up, we do not know if continued physical therapy would maintain the short-term improvement. On the one hand, an open, nonrandomized study found that significant improvement in step length and walking speed was sustained during 12 weeks of physical therapy.[3] However, Palmer et al[4] found that more than one-half their PD patients improved following 6 weeks of physical therapy, but only 25% continued to show improvement at 12 weeks despite continued therapy. We are currently examining this question in a long-term follow-up study. Additional studies evaluating whether other measures of daily functioning improve in PD patients following physical rehabilitation also need to be addressed.

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