ORIGINAL ARTICLE

Gait Analysis in Patients With Parkinson's Disease Off Dopaminergic Therapy

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ABSTRACT. Švehlík M, Zwick EB, Steinwender G, Linhart WE, Schwingenschuh P, Katschnig P, Ott E, Enzinger C. Gait analysis in patients with Parkinson's disease off dopaminergic therapy. Arch Phys Med Rehabil 2009;90:1880-6.

Objective: To compare time-distance, kinematic, and kinetic gait parameters in patients with idiopathic Parkinson's disease (PD) off dopaminergic therapy with a group of healthy control subjects.

Design: A group-comparison study.

Setting: Gait analysis laboratory.

Participants: Patients with PD (n=20) and healthy age-matched controls (n=20).

Interventions: Not applicable.

Main Outcome Measures: Time-distance, kinematic, and kinetic gait variables.

Results: PD patients walked slower with shorter stride-length, comparable cadence, and longer double support times. Kinematics showed a reduction of the range of motion in the hip, knee, and ankle joints. Maximum hip extension and the ankle plantar flexion were significantly reduced. Kinetic gait parameters showed reduced push-off ankle power and lift-off hip power generation. Strong correlations between these important body advancement mechanisms and the walking velocity were observed.

Conclusions: In addition to previously described dysfunctional kinematics, abnormal kinetic parameters play an important role in the characterization of gait in PD patients off therapy. Hence, these parameters could be used to document treatment effects of parkinsonian gait disorders.

Key Words: Biomechanics; Levodopa; Neurologic gait disorders; Parkinson Disease; Rehabilitation.

IDIOPATHIC PARKINSON'S DISEASE is a chronic, progressive neurologic disorder affecting approximately 1 in every 100 people over 65 years of age. Gait disorders are a hallmark of the condition and are associated with a loss of independence[1] and an increased incidence of falls.[2] Clinically, patients with PD tend to show a shuffling gait pattern with shortened stride length, reduced overall velocity, and increased stance phase durations.[3] Moreover, reduced or absent arm swing; reduced trunk rotation; and decreased amplitude of motion at the hips, knees, and ankles are characteristic of the PD gait.[4] Gait deficiencies in PD arise from a disturbance in the motor set function of the basal ganglia specifically involved in the regulation of movement amplitude.[5] Basal ganglia also appear to inhibit movements via direct and indirect pathways[6] and contribute to the regulation of postural alignment and axial motor control.[7,8] Visual cues are known to improve the gait of PD patients, possibly by bypassing the faulty basal ganglia. They may develop through the patient's ability to utilize visual feedback to regulate the movement amplitude, thus reducing reliance on kinesthetic feedback.[5]

Early studies investigating parkinsonian gait disorders used footswitches and stride analyzers to describe abnormalities in spatial-temporal parameters of gait (velocity, stride length, or cadence).[9,10] More recently, computer-assisted gait analysis has been adopted as an objective measurement tool to describe kinematic parameters.[11,12] Morris et al[13] and Lewis et al[5] described both the kinematic and kinetic features of parkinsonian gait and found reduced plantar flexion at toe-off and insufficient ankle power generation. However, only 1 study by Sofuwa et al[14] has compared quantitative spatiotemporal, kinematic, and kinetic gait analysis parameters of PD patients on dopaminergic therapy to a group of healthy elderly controls. To our knowledge, no equivalent study investigating PD patients off therapy has been published. However, such an approach may provide greater insight into the nature of the motor deficits caused by the disorder itself, as the medication might have confounding effects on force generation patterns.[5] Therefore, we set out to compare time distance, kinematic, and kinetic gait parameters of PD patients off therapy with a group of healthy control subjects to circumvent these limitations.

METHODS

Subjects

Patients. Twenty patients with idiopathic PD (13 men, 7 women) were recruited from the local movement disorders clinic at the Department of Neurology at Medical University of Graz, Austria. Inclusion criteria were PD according to UK Parkinson's Disease Society Brain Bank clinical diagnostic criteria[15]; age 50 to 80 years; dopaminergic medication; no concurrent neurologic, orthopedic, or other medical conditions affecting gait; absence of dementia based on prior cognitive assessment; Hoehn and Yahr stages 1 to 4 off therapy; and the ability to walk along a 12-m walkway at least 5 times without

List of Abbreviations

PD	Parkinson's disease
L-dopa	levodopa
ROM	range of motion

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assistance. Participants were asked to stop short-acting dopaminergic medications (standard preparations of L-dopa and short-acting dopamine agonists) at least 12 hours before the study and long-acting preparations (cabergoline, control release preparations of L-dopa) at least 24 hours beforehand, which induced a relative and clearly defined "off" state.[16] Two subjects had a history of rare freezing when walking and 3 had a history of festination, but we did not observe any freezing of gait during testing.

Controls. Twenty healthy subjects (10 men, 10 women) aged 50 to 80 years with no current neurologic, orthopedic, or other medical conditions affecting gait made up the control group. The control group was age matched to the PD group. Although the groups were not sex matched, there was no statistically significant difference in the leg lengths that could have influenced our results. The Mini-Mental State Examination scores did not differ between the groups.

All participants gave written informed consent according to the 1991 Declaration of Helsinki, and the study was approved by the local research ethics committee. Before gait analysis, all patients were examined according to the Unified Parkinson's Disease Rating Scale motor rating scale[17] and classified with the Hoehn and Yahr disability scale.[18] Details concerning demographics, clinical data, and medication for the patients are given in table 1.

Gait Analysis

Gait analysis was performed with the use of a 12-camera motion capturing system[a] and 4 force plates[b] mounted under the walkway. Marker arrangement, calculation methods, and model assumptions applied have been described in detail by Kadaba et al.[19] Calculations of kinematic parameters for the pelvis and the hip, knee, and ankle joints as well as kinetic parameters for the hip, knee, and ankle joint were performed by using the Vicon Clinical Manager.[a] Moment and power parameters were normalized to the weight of the patients. Power generation and absorption patterns in the sagittal plane were calculated and labeled according to the method described by Winter.[20] All patients walked at self-selected speeds along a 12-m walkway, and only steady-state walking was measured. For each patient, a minimum of 5 trials per limb providing a clear foot force plate contact were captured. From each trial, only 1 gait cycle was used to calculate the patient's mean trial. Pelvic kinematics and sagittal plane kinematic and kinetic parameters of the hip, knee, and ankle joints, as well as time-distance parameters (gait velocity, cadence, stride length, single and double support time) were used as outcome measures in this study.

Statistical Analysis

Time-distance parameters, kinematics, and kinetic data were compared with the values obtained from the control group by using unpaired Student *t* tests after testing for normal distribution. Bonferroni correction for multiple testing was applied, and *P* values of less than .001 were considered as statistically significant. A Pearson correlation coefficient was used to express the relationship between the velocity and the main propulsion generation variables. All analyses were calculated by using the Statistica 6.0[c] software.

RESULTS

Time-Distance Parameters

The PD group showed decreased walking velocity and stride length, whereas cadence did not differ from the control group (table 2). The double-limb support and the stance phase of gait were prolonged in the PD group.

Lower-Extremity Joint Angles

The ROMs were reduced at all lower-extremity joints in the PD group (table 3). PD subjects walked with a markedly increased pelvic tilt. Pelvic obliquity and transversal rotation did not differ between the groups. The extent of maximum hip flexion was comparable between the groups. In the control

Table 1: Demographic, Clinical, and Drug Details of PD Patients

Patient	Age	Sex	MMSE	Years Since Diagnosis	Side Most Affected	UPDRS III Off Therapy	Hoehn and Yahr Stage	Levodopa (DD, mg)	Dopamine Agonists (DD, mg)	Other PD Medication (DD, mg)
1	67	M	23	4	R	42	2,5	—	pra, 4.5mg	sel, 10mg
2	67	M	23	10	L	15	2	—	per, 3mg	—
3	65	M	21	4	L	49	2	450mg	—	—
4	66	F	26	11	R	38	2	300mg	rop, 21mg	sel, 10mg
5	76	M	20	1	L	35	2	300mg	—	—
6	59	F	28	8	L	52	3	700mg	pra, 4.5mg	sel, 10mg
7	73	M	27	10	L	39	2,5	800mg	pra, 6.0mg	—
8	76	M	28	3.5	L	37	2	500mg	pra, 3mg	—
9	67	M	24	5	L	49	2	—	pra, 3mg	—
10	70	M	26	10	L	42	2,5	300mg	pra, 3mg	sel, 10mg
11	71	F	27	5	R	35	2	150mg	pra, 3mg	—
12	63	M	24	10	R	30	2	300mg	pra, 3mg	sel, 10mg
13	75	F	27	1	R	32	2,5	150mg	pra, 1.5mg	—
14	52	M	30	3	R	31	2	—	rop, 15mg	—
15	60	M	29	5	L	57	3	400mg	rop, 12mg	—
16	74	M	22	5	R	51	2	150mg	cab, 3mg	—
17	56	M	28	7	R	45	2,5	300mg	cab, 4mg	sel, 10mg
18	57	F	27	7.5	R	31	2	150mg	cab, 4.5mg	sel, 10mg
19	66	F	24	9	L	32	2	600mg	pra, 4.5mg	—
20	76	F	28	6	R	15	1,5	100mg	pra, 2mg	—

Abbreviations: F, female; M, male; MMSE, Mini-Mental State Examination; UPDRS III, Unified Parkinson's Disease Scale–motor part; DD, daily dose; pra, pramipexole; per, pergolide; rop, ropinirole; cab, cabergoline; sel, selegiline; R, right; L, left.