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Original Article

Quantitative Assessment of Gait Bradykinesia in Parkinson's Disease Using a Portable Gait Rhythmogram

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To quantify gait bradykinesia during daily activity in patients with Parkinson's disease (PD), we measured movement-induced accelerations over more than 24 h in 50 patients with PD and 17 age-matched normal controls, using a new device, the portable gait rhythmogram. Acceleration values induced by various movements, averaged each 10 min, exhibited a gamma distribution. The mean value of the distribution curve was used as an index of the "amount of overall movement per 24 h". Characteristic changes were observed in both the gait cycle and gait acceleration. During hypokinesia, the gait cycle became either faster or slower. A number of patients with marked akinesia/bradykinesia showed a reduced and narrow range of gait acceleration, *i.e.*, a range of floor reaction forces. The results suggest that assessment of the combination of changes in gait cycle and gait acceleration can quantitatively define the severity of gait bradykinesia.

Key words: Parkinson's disease, gait disorders, portable gait rhythmogram

Akinesia and bradykinesia are core symptoms of Parkinson's disease (PD) and are caused by progressive loss of melanin-containing neurons in the substantia nigra, locus coeruleus, and other brain structures [1, 2]. Akinesia is defined as poor movement generation, whereas bradykinesia represents slowness of executed movements [3–5]. For proper assessment of the response to dopamine replacement therapy, it is desirable to measure patient movements in daily activities and to identify and quantify akinesia/bradykinesia related to these movements. However, quantitative estimation of motor symptoms in daily life is difficult. In fact, the available pathophysiological data have been obtained from experimentally designed tasks, including tasks of tracking a moving index [5] or tasks related to reaction time [6]. For quantitative assessment of akinesia/bradykinesia in daily activities, we focused on parkinsonian gait disorders [7, 8], since gait movements are represented by a few simple parameters such as cycle (cadence: steps per minute),

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stride and floor reaction forces [7–12].

We developed a new long-term monitoring device, the portable gait rhythmogram (PGR), which measures acceleration induced by limb and trunk movements and gaits in daily life [8]. The main features of the device are: 1) it is capable of long-term monitoring (> 40 h), 2) it is designed to allow identification of rhythmical gait movements from the voluntary limb and trunk movements or artifacts.

In the present study, 2 aspects of daily activities were analyzed using the PGR. First, we calculated an index that reflects overall movements within 24 h estimated from accelerations induced by various movements executed during daily life. The PGR is designed to interpret decreased acceleration as poverty or slowness of movements. Second, we filtered out non-gait related accelerations. Using data of the gait cycle and acceleration, we determined the characteristic changes in gait cycle and floor reaction forces. By comparing these parameters, we attempted to construct a new test that allows quantitative assessment of akinesia/bradykinesia in the daily activities of PD patients.

Materials and Methods

Subjects. The study subjects were 50 patients with PD (mean age, 70.3 ± 7.6 years, mean $\pm$ SD, 30 men and 20 women, Table 1). They represented all patients admitted to Tokyo Medical University Hospital between June 2009 and March 2010 who could walk unaided and showed no peak-dose dyskinesia during their 'on' time. They included 8 patients with Hoehn and Yahr stage I, 14 with stage II, and 28 with stage III. The duration of illness was 4.5 ± 3.6 years. The clinical status was determined using the Unified Parkinson's Disease Rating Scale (UPDRS) motor score 'on' state [13]. We also studied 17 age- and height-matched normal controls (mean age, 64.7 ± 4.5 years, 8 men and 9 women). Matching for age and height was based on the finding that gait cycle and floor reaction forces are influenced by these 2 parameters [7, 8, 18].

Informed consent was obtained from all subjects. All procedures were conducted in accordance with the guidelines of the Ethics Committee of our institution. Statistical analysis was performed using the student's *t*-test. A p value of < 0.05 denoted the presence of a significant difference.

Measurement of acceleration induced by various movements during daily activities. The PGR is a small device (size, $8 \times 6 \times 2$ cm, weight, 80 g) that three dimensionally measures (a_x, a_y, a_z) accelerations associated with voluntary limb and trunk movements and accelerations induced by step-in and kick-off during gait (Mitsubishi Chemical Group Science and Technology Research Center, Inc., Tokyo, Japan) [8]. The PGR is attached to the patient's waist, and records the above signals at a sampling rate of 10 ms. The data are automatically stored in a microSD card. When recording is completed, the absolute value of acceleration vectors ($a; a^2 = a_x^2 + a_y^2 + a_z^2$) is calculated and graphically displayed on the PC. A fully-charged PGR can achieve 40 consecutive hours of recording. All recordings in the present study were performed for more than 24 h and data of 24 h were analyzed.

Evaluation of "amount of overall movements per 24-h". After continuous recording for more than 24 hrs, the accelerations induced by all movements were averaged every 10 min of recording, and the data were displayed graphically (Fig. 1). Based on the assumption that the curve fits the gamma distribution, the mean value of the distribution was calculated mathematically. The gamma distribution is defined as the following formula.

$$f(x) = x^{k-1} e^{-x/\theta} / \Gamma(k) \theta^k \text{ for } x > 0$$

The mean value was named the "*amount of overall movements per 24-h*", representing an index of akinesia/bradykinesia.

Quantitative analysis of gait disorders

1. Identification of acceleration induced by gait movements. The acceleration vectors caused by stepping can be distinguished from those by other voluntary limb and trunk movements or by artifacts, based on the algorithm of *pattern matching* [8]. Since the acceleration wave induced by gait motion is similar in shape in each patient, the correlation is tested mathematically between the concerned waves and template. Using this method, we identified all steps that were performed during the 24 h in each individual. The peak-to-peak interval was automatically detected to calculate the duration of one gait cycle. We also measured the amplitude of the gait acceleration. Based on these parameters, we documented the characteristics of the gait cycle and the floor reaction

Table 1 Characteristics of patients classified according to gait disorder pattern

Patient number	Age (yrs)	Sex	Duration of illness (yrs)	Hoehn Yahr	UPDRS III	UPDRS III 26-31	Amount of overall movements per 24-h	Off step cycle (sec)	Regression slope in gait acceleration
Mild gait disorder									
1	54	M	3	III	23	5	0.78	1.13	0.68
2	73	F	0.5	I	11	3	0.60	1.15	1.25
3	73	M	2	III	27	9	0.57	1.11	0.70
4	68	F	4	III	24	8	0.57	1.12	0.81
5	83	M	4	I	12	6	0.54	1.23	0.93
6	68	M	4	III	30	9	0.53	1.09	0.76
7	67	F	4.5	II	19	7	0.53	1.14	1.31
8	72	M	2	II	31	5	0.46	1.11	0.69
9	76	M	4	II	16	1	0.46	1.24	1.24
10	56	F	3	III	18	8	0.46	1.16	1.31
11	72	M	3.5	III	29	8	0.45	1.17	1.03
12	73	F	10	I	10	2	0.44	1.15	0.94
13	74	F	2	II	35	7	0.43	1.17	1.06
14	67	M	3.5	I	30	7	0.42	1.16	1.32
15	78	F	10	III	23	4	0.38	1.10	0.90
16	82	F	5	III	17	3	0.35	1.10	1.18
17	74	M	4	III	12	5	0.31	1.11	1.21
18	79	M	2	III	43	12	0.28	1.14	1.18
19	80	M	10	III	46	9	0.22	1.20	0.84
Moderate gait disorder									
20	64	F	13	III	21	6	1.10	1.30	0.80
21	63	M	5	III	29	8	0.68	0.99	0.42
22	65	M	1.5	III	26	7	0.66	1.00	0.54
23	63	F	0.5	II	23	5	0.59	1.35	0.90
24	65	M	1.5	III	26	7	0.49	1.02	0.69
25	64	M	2	II	10	3	0.48	1.42	1.13
26	71	M	9	III	24	8	0.43	1.29	1.07
27	63	F	1	I	21	8	0.41	1.39	1.13
28	56	M	0.5	II	11	4	0.40	0.99	1.08
29	70	F	3	III	13	5	0.39	1.04	0.69
30	65	M	1.5	I	10	2	0.39	1.32	1.26
31	75	M	12	III	28	5	0.36	1.30	1.34
32	70	F	5	II	30	5	0.35	1.01	1.44
33	70	F	6	II	21	5	0.33	0.97	1.11
34	77	M	3	I	14	7	0.32	1.26	1.15
35	65	F	3	III	15	7	0.31	1.25	1.32
36	70	F	3	I	9	5	0.31	1.01	1.05
37	75	M	3	III	23	9	0.30	1.29	1.44
38	78	F	2	III	31	12	0.29	1.40	1.15
39	82	M	3	III	32	9	0.24	1.26	1.12
40	70	F	2	III	17	8	0.23	1.03	1.29
Moderate gait disorder									
41	78	M	9	II	11	1	0.52	1.24	2.47
42	80	M	9	III	21	7	0.36	1.12	1.76
43	65	M	2	III	13	6	0.35	1.11	1.54
44	59	M	4	II	15	5	0.31	1.20	1.65
Severe gait disorder									
45	63	M	6	II	16	5	0.43	1.39	2.35
46	72	F	3	II	13	8	0.39	1.06	1.73
47	52	F	5	III	6	4	0.36	1.04	1.91
48	77	M	18	III	36	12	0.35	1.30	1.49
49	79	M	3	II	29	6	0.26	1.03	1.95
50	79	M	4	III	34	9	0.24	1.29	1.70

Hoehn Yahr, Hoehn and Yahr stage; UPDRS, Unified Parkinson's Disease Rating Scale. Mild gait disorder group: patients who walked with normal rhythm and force, Moderate gait disorder group: patients who walked with slow or fast rhythm or with reduced forces, Severe gait disorder group: patients walked with both abnormal rhythm and reduced force. Patients were arranged according to the amount of overall movements per 24-h.

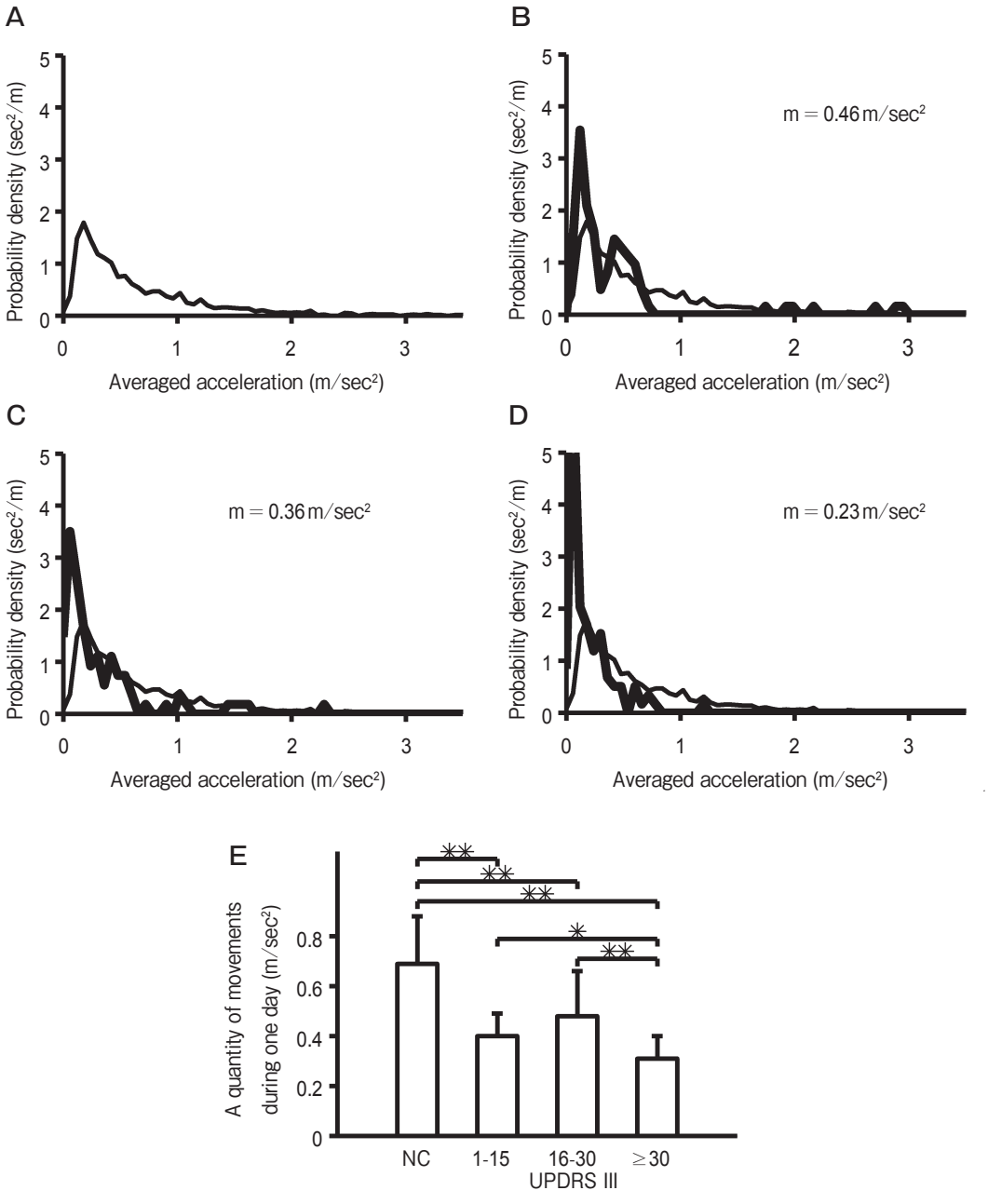

Fig. 1 Probability distribution of the averaged acceleration per 10 min, obtained from continuous recording for more than 24 h. (**A**) The summed average for 17 normal subjects. (**B**–**D**) Thin lines: distribution curve for 17 normal controls, bold line: distribution curve for a PD patient. Patient #8 in **B**, Patient #31 in **C**, and Patient #40 in **D**. The mean values of the distribution for Patients #8 in **B**, #34 in **C**, and #40 in **D** were less than the intersubject mean-1SD of the normal controls. (**E**) Quantity of movements in one day of patients with UPDRS 1–15, 16–30, and ≥ 30. NC shows that of normal controls. $^{*}P < 0.05$, $^{**}P < 0.01$.

force by the following 2 methods.

2. Estimation of changes in gait cycle duration during hypokinesia. The duration of the gait cycle was plotted against all motion-induced accelerations averaged each 10 min (Fig. 2). Thus, the graph shown in Fig. 2 describes the relationship between the gait cycle and the *amount of overall movements*. Two possibilities were considered for low physical activity: *1*)

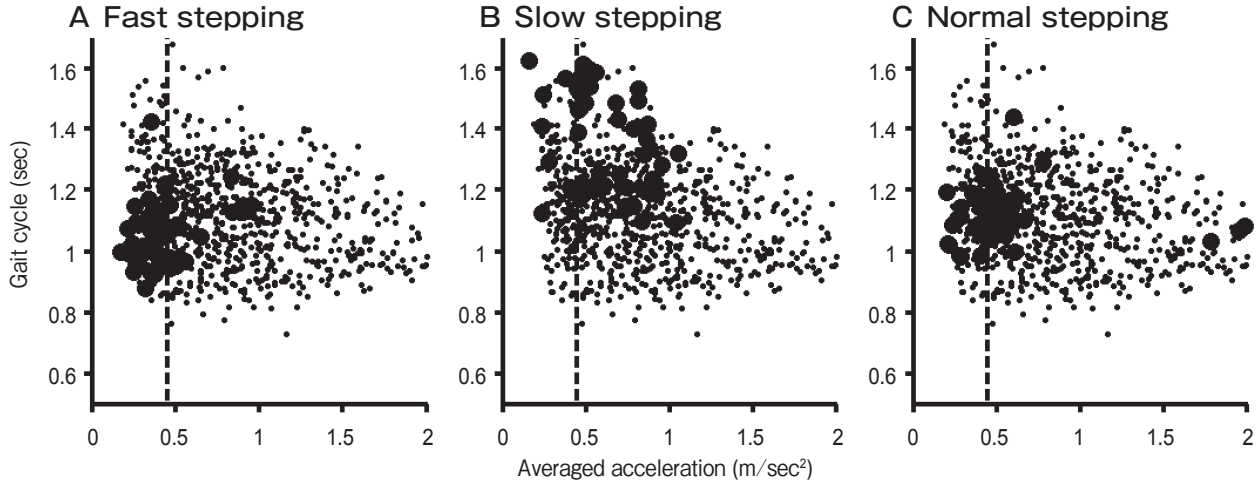

Fig. 2 Relationship between all motion-induced accelerations and gait cycle durations. Motion-induced accelerations and gait cycles were averaged every 10 min and plotted. The averaged values represented all steps during the 24 h. The plot includes the data of 17 normal controls (small circles) and those of PD patients (large circles). The mean of the gait cycle was calculated within a range of the lower 10% of motion-induced accelerations ($\leq 0.45\,m/sec^2$, vertical dotted line). (**A**) Example of '*Fast stepping during hypokinesia*' (Patient #49). The gait cycle was fast while the averaged motion-induced acceleration was low. (**B**) Example of '*Slow stepping during hypokinesia*' (Patient #45). The gait cycle was slow while the averaged motion-induced acceleration was low. (**C**) The gait cycle was normal while the averaged motion-induced acceleration was low (Patient #8).

the patients did not move voluntarily, or *2*) the patient could not move due to akinesia/bradykinesia. Therefore, this graph allows approximate estimation of changes in the gait cycle during hypokinesia. Data collected over a short walking period (less than 50 steps) were excluded due to the large variance.

3. Estimation of floor reaction forces. Since gait acceleration correlates with the floor reaction forces, we measured gait acceleration as an index of the floor reaction forces. The gait-induced accelerations and duration of gait cycles were averaged for each 10-min recording (Fig. 3). The graph shown in Fig. 3 indicates that changes in the gait acceleration correlate with the gait cycle duration. The averaged logarithmic values showed normal distribution and the slope of the regression line was calculated using principal component analysis.

Results

Amount of overall movements per 24-h. We first analyzed the accelerations induced by all voluntary movements (all motion-induced accelerations) from the 24-h data recorded by the PRG. Fig. 1 shows the probability distribution of the 10-min averaged accelerations. Fig. 1A provides the summed average obtained from 17 normal control subjects. Compared with the distribution curve of normal controls, that of each PD patient was shifted to the left, indicating poverty of movement (Patient #8 in B, Patient #31 in C, Patient #40 in D). The mean value of the distribution curve, termed the "*amount of overall movements per 24-h*", was calculated with the assumption that the curve fitted gamma distribution. The intersubject mean value of the *amount of overall movements per 24-h* was $0.69 \pm 0.19\,m/sec^2$ in normal controls. The a*mount of overall movements per 24-h* was $0.46\,m/sec^2$ in Patient #8 (Fig. 1B), $0.36\,m/sec^2$ in Patient #31 (Fig. 1C), and $0.23\,m/sec^2$ in Patient #40 (Fig. 1D). When the *amount of overall movements per 24-h* was less than the intersubject mean-1SD in normal controls ($0.50\,m/sec^2$), it was defined as significant change; 38 of 50 patients showed a significant decrease in activities. In Fig. 1, Patient #8 (Fig. 1B), Patient #31 (Fig. 1C) and patient #40 (Fig. 1D) exhibited significant decreases in movement activities.

The *amount of overall movements per 24-h* varied according to the severity of PD. Patients were classified into 3 groups based on the UPDRS-III: patients with scores of 1–15, those with scores of 16–30, and those with scores ≥ 30. The *amount of overall movements per 24-h* was $0.40 \pm 0.09\,m/sec^2$ in patients with

scores of 1–15, $0.48 \pm 0.18\,m/sec^2$ in patients with scores of 16–30, and $0.31 \pm 0.09\,m/sec^2$ in patients with scores ≥ 30 (Fig. 1E). The *amount of overall movements per 24-h* for patients with scores ≥ 30 was significantly lower than that of patients with scores of 1–15 ($p < 0.05$) and that of patients with scores of 16–30 ($p < 0.01$).

Gait cycle during hypokinesia. Next, we examined the relationship between activities and gait cycle. All motion-induced accelerations and gait cycle duration were averaged each 10 min and plotted (Fig. 2). The averaged values obtained from all steps achieved per 24 h were plotted. The results of 17 normal controls were also plotted (small circles, Figs. 2A, B and C). In the normal controls, the gait cycle duration did not change significantly with the degree of physical activities. On the other hand, several patients (large circles in Figs. 2A and B) showed characteristic changes in the gait cycle during the time when all motion-induced accelerations were reduced, presumably during hypokinesia.

To estimate these changes quantitatively, the mean gait cycle was calculated within a range of the lower 10% of all motion-induced accelerations (within a range of $0.45\,m/sec^2$). The intersubject gait cycle was 1.16 ± 0.08 sec in normal controls. Some PD patients walked with a gait cycle duration exceeding the intersubject mean $\pm$ 1SD of the controls. Two paradoxical changes were observed. Twelve patients (Patients #21, 22, 24, 28, 29, 32, 33, 36, 40, 46, 47, 49) walked using a fast step cycle when all motion-induced accelerations were low (Fig. 2A, Patient #49). This type of gait was termed '*Fast stepping during hypokinesia*'. In contrast, 15 patients (Patients #20, 23, 25, 26, 27, 30, 31, 34, 35, 37, 38, 39, 45, 48, 50) walked using a slower cycle (Fig. 2B, Patient #45), termed '*Slow stepping during hypokinesia*'. The other 23 patients walked with a normal step cycle while the activity decreased (Fig. 2C, Patient #8).

Gait acceleration as an index of floor reaction forces. Fig. 3 shows the relationship between gait acceleration and gait cycle. Both parameters were recorded continuously for more than 24 h, and then the average value for each 10 min was computed. Fig. 3A shows the results of a normal control, and Figs. 3B–D show those of PD patients with the corresponding regression line (thick line). The regression line, obtained from the summed average of 17 normal controls, is also shown (thin line).

In the control subjects, walking with a slower cycle was associated with a decrease in acceleration. The slope of the linear regression line in logarithm was 1.20 ± 0.29 in normal controls. Several PD patients showed characteristic changes. The amplitude of the gait acceleration decreased in some patients (Patient #24 in Fig. 3B, Patient #32 in Fig. 3C, and Patient #49 in Fig. 3D). Furthermore, the range of gait acceleration was narrow, and thus the slope was steeper (Figs. 3C and D). The slope of the regression line exceeding the intersubject mean + 1SD (1.49) of the controls was defined as significant deviation, which was observed in 10 patients (Patients #41, 42, 43, 44, 45, 46, 47, 48, 49, 50). In Fig. 3, the slope of the regression line was 0.69 in Patient #24 (Fig. 3B), 1.44 in Patient #32 (Fig. 3C), and 1.95 in Patient #49 (Fig. 3D). Thus, patient 49 in Fig. 3D can be considered part of the significant deviation group.

Combination analysis of gait parameters: rhythm and force. Fig. 4A shows the relationship between the pathophysiological changes in gait cycle and the floor reaction forces. In this graph, the abscissa represents a decrease in the floor reaction forces, while the ordinate represents gait cycle changes during hypokinesia. The *z* axis represents the number of patients. Patients were classified into three groups: *the mild gait disorder* group comprised patients with a normal gait cycle and floor reaction forces (open bars, n = 19, 38%), *the moderate gait disorder* group comprised patients in whom one of the parameters was abnormal (gray bars, n = 25, 50%), and *the severe gait disorder* group comprised patients in whom both parameters were outside the normal range (solid bars, n = 6, 12%). The Table shows the clinical profiles of the three groups of patients. The UPDRS-III values were 24.0 ± 10.4, 19.8 ± 7.3, and 22.3 ± 12.3 for the *mild*, *moderate* and *severe groups*, respectively. The respective UPDRS-III 26-31 values were 6.2 ± 2.8, 6.2 ± 2.3, and 7.3 ± 2.9. For UPDRS-III 26-31, the score tended to be higher in the *severe group*, albeit statistically insignificant (Fig. 4B).

The *amount of overall movements per 24-h* was also compared among the groups. The number of patients who showed significant changes in the *amount of overall movements per 24-h* was 12/19 (63.1%) of the patients from the *mild group*, 20/25 (80.0%) of the *moderate*

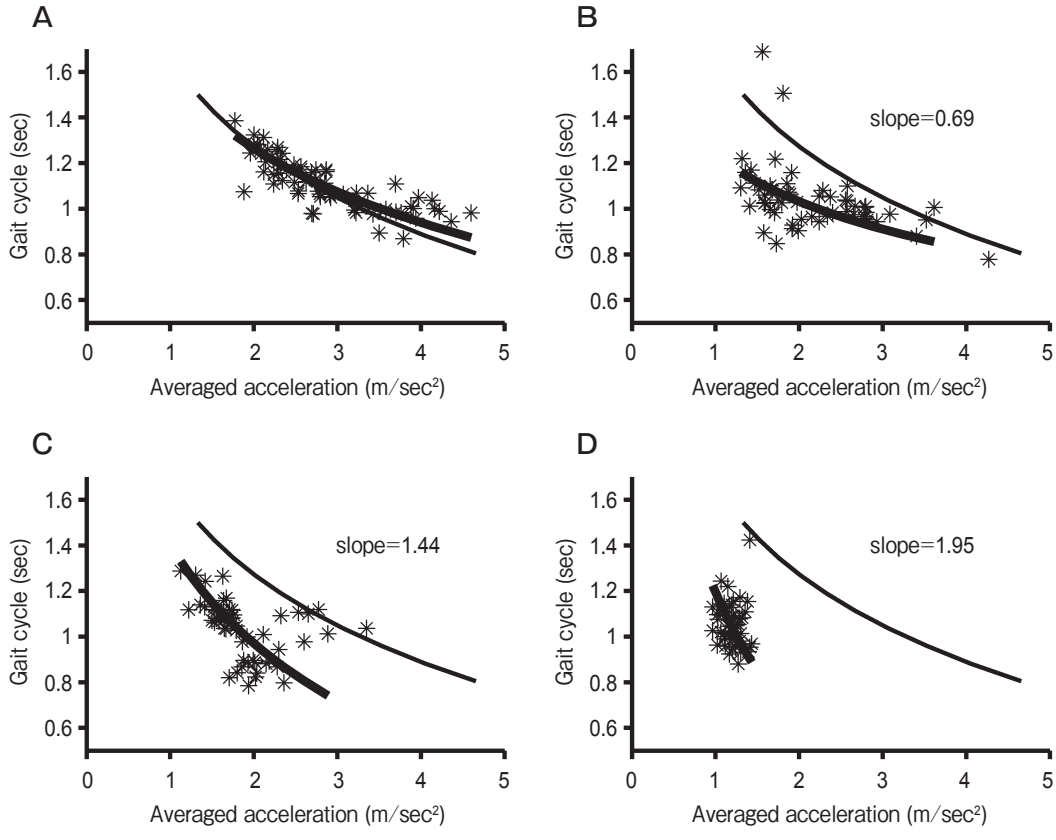

Fig. 3 Relationship between gait acceleration and gait cycle duration. Both parameters were recorded continuously for more than 24 h and averaged every 10 min. (**A**) Data of a representative normal control. (**B**–**D**) Data of a representative PD patient (large circles) with the corresponding regression curve (bold line). Patient #24 in **B**, Patient #32 in **C**, and Patient #49 in **D**. *Thin line*: the regression line obtained from the summed average values of 17 normal controls (A, B, C, and D). The slope of the regression line of Patient 49 in **D** exceeded the intersubject mean + 1SD of the normal controls.

group, and 6/6 (100.0%) of the *severe group*. On the other hand, the values of the *amount of overall movements per 24-h* were $0.46 \pm 0.12\,m/sec^2$, $0.42 \pm 0.18\,m/sec^2$ and $0.33 \pm 0.07\,m/sec^2$ for the *mild*, *moderate*, and *severe groups*, respectively (Fig. 4C). There was a significant difference between the *mild* and *severe* groups ($p < 0.01$) and between the *moderate* and *severe* groups ($p < 0.05$).

Discussion

Actigraphy has been used to monitor movements and daily activities [14]. Although the extent of poverty of movements (akinesia) can be roughly estimated using this method, the slow nature of the executed movements (bradykinesia) cannot be estimated by actigraphy. The present study was designed to quantify the degree of akinesia/bradykinesia in the daily lives of PD patients. Using the long-term monitoring system of PGR, we calculated the amount of daily movements as an index of the poverty of movements, and differentiated the nature of gait bradykinesia by identifying changes in the gait cycle and gait acceleration (*i.e.*, the floor reaction force).

Quantitative index for akinesia/bradykinesia. Using the PGR, we measured the amount of overall movement, similar to the parameters measured by actigraphy [14]. In the present study, we also calculated a quantitative index for intersubject comparisons. The acceleration values averaged each 10 min showed a mathematical gamma distribution. Thus, we calculated the mean value of the distribution, *i.e.*, the *amount of overall movements per 24-h*. The mean value should decrease in the presence of both

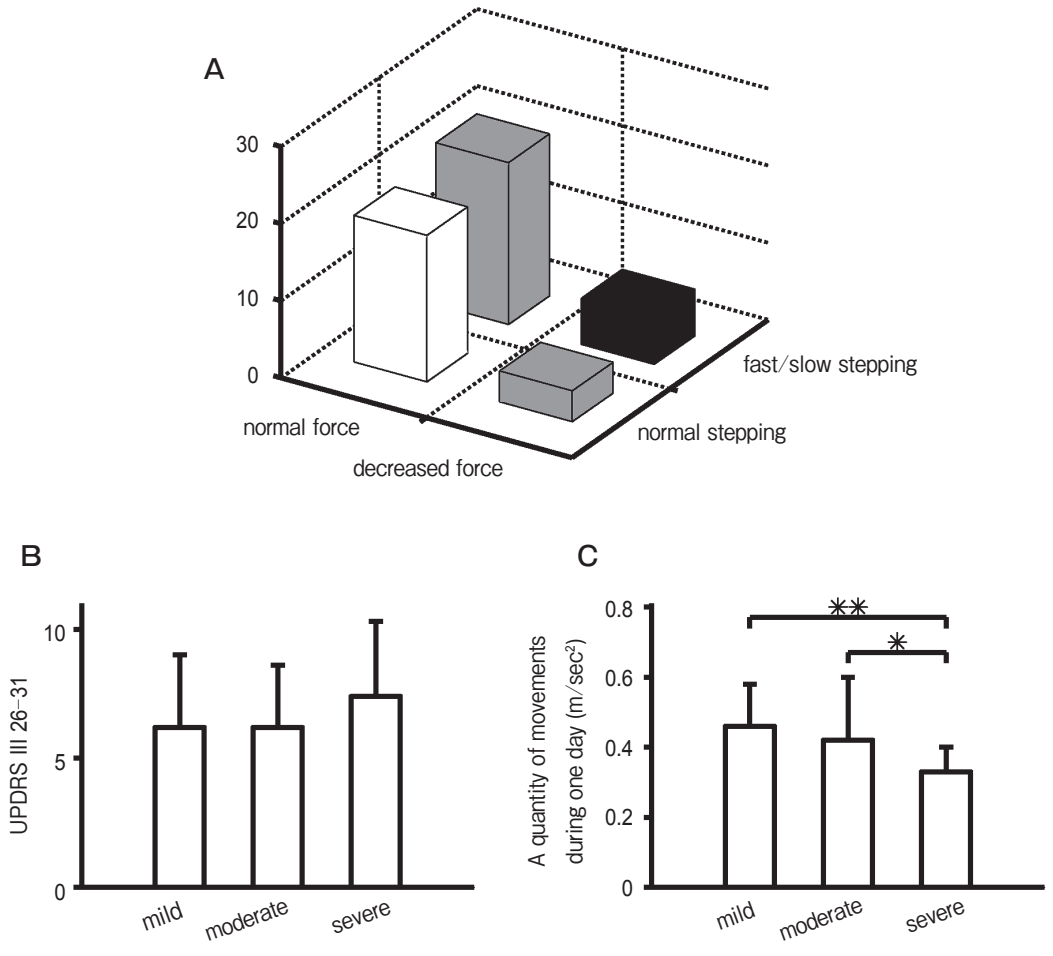

Fig. 4 (**A**) Relationship between changes in the gait cycle and acceleration. The *z* axis represents the number of patients. When both parameters were normal (open bars), the patients were considered to have *mild gait disorder*. When each parameter was abnormal (gray bars), the patients were considered to have *moderate gait disorder*. When both parameters were beyond the normal range (solid bars), the patients were considered to have *severe gait disorder*. (**B**) The mean UPDRS-III 26–31 value of the *mild, moderate and severe gait disorder* groups. (**C**) The *amount of overall movements per 24-h* for each group. $^{*}p < 0.05$, $^{**}p < 0.01$.

akinesia and bradykinesia. Advanced UPDRS-III values are associated with a significant decrease in the *amount of overall movements per 24-h*, as shown in Fig. 1E. The *amount of overall movements per 24-h* was significantly low in patients with UPDRS-III ≥ 30, indicating prominent poverty of movement (akinesia) in these patients. It appears that there was no significant difference between the UPDRS 1–15 and 16–30 groups, since UPDRS-III assesses not only akinesia, but other motor symptoms such as resting tremor or difficulties in motor performance.

Assessment of the *amount of overall movements per 24-h* should take into consideration 2 issues: *1*) contamination with involuntary movements and *2*) inter-day variability in activity. Involuntary movements should result in overestimation of accelerations. Resting tremor, occurring periodically at 5–6 Hz, was easily excluded using our analysis algorithm. However, dyskinesia is difficult to identify since it occurs irregularly and in a variety of forms. Severe disturbances of the gait parameters, associated with a high *amount of overall movements per 24-h*, could reflect dyskinesia. On the other hand, differences in daily activities could also influence the movement-related accelerations. Strenuous physical activity, such as exercising or doing housekeeping chores, should

increase acceleration. Thus, during monitoring with the PGR, patients should be advised to engage only in their usual and routine daily activities, and to refrain from any unusual activities. Practically, these 2 problems cannot be avoided when measuring acceleration and, accordingly, patients should be advised to use a diary to record daily activity, which could be used during data analysis.

Changes in gait cycle during hypokinesia. To explore changes in the gait cycle during hypokinesia, we examined the relationship between the gait cycle and all motion-induced acceleration. Both parameters were averaged each 10 min. Reductions in all motion-induced acceleration could represent 2 possibilities: 1) a sedentary position and lack of movement, and 2) the inability to move due to hypokinesia. In the present study, we assessed the approximate changes in gait cycle during hypokinesia. The results showed 2 opposite changes: '*Fast stepping during hypokinesia*' and '*Slow stepping during hypokinesia*'. In our previous report [8], we measured changes in gait cycle during 24 h and showed changes toward a faster rhythm mainly in patients who walked with short-stepping, festination or freezing of gait (FOG) [8]. Each gait parameter could cause shortening of the gait cycle. Patients walking with a short stride compensate by increasing cadence, resulting in a fast gait rhythm [9–11]. Furthermore, festination was characterized by the gradual shortening of one gait cycle, whereas FOG was defined as an abrupt decrease in the gait cycle [8]. On the other hand, our data also showed a shift to a slower rhythm in patients with marked bradykinesia and instability [8]. Prolongation of the stepping cycle was due to 2 main deficits: 1) marked slowness in the swinging of the lower extremities, and 2) protective walking to compensate for marked instability. Taken together with previous findings, the present results indicate that gait bradykinesia is not uniform. Two types of direct disorders that can change automatic movement cycles in an opposite manner appear to occur during hypokinesia in PD.

Defective production of floor reaction forces. It has been postulated that PD patients can only execute movements of different amplitudes at a single, slow, velocity and cannot increase their movement velocity [15]. The peak EMG activity that can be generated in a muscle burst is limited in PD patients [16]. We previously reported decreases in the reciprocal muscle activities of the tibialis anterior and gastrocnemius muscles in PD patients [7]. Consistent with these observations, the present results showed that the range of gait acceleration, *i.e.*, the range of floor reaction forces, was narrow. Under these conditions, PD patients were obliged to produce a particular degree of force and, consequently, disturbance in their ability to change force production. This change seems to be the mechanism underlying the monotonous impression of walking seen in PD patients.

Classification of gait disorders: rhythm or force? Disordered gait cycle and the floor reaction force appear to be the main pathomechanisms of the parkinsonian gait, since animal experiments showed that loss of output from the basal ganglia elicits changes in the cycle and muscle activities [17]. In these studies, manipulation by electrical stimuli resulted in a slower gait cycle and weaker muscle activity. Therefore, there is a need for a method that can quantitatively estimate changes in the gait cycle and floor reaction force to determine the nature of parkinsonian gait disorder. In the present study, we were able to classify our patients into 3 groups based on the type of disturbance in gait parameters: *the mild gait disorder* group comprised of patients who walked with a normal rhythm and force, *the moderate gait disorder* group comprised of patients who walked with a slow or fast rhythm or with reduced forces, and *the severe gait disorder* group comprised of patients who walked with both an abnormal rhythm and reduced force. The present classification correlated with the extent of decrease in the *amount of overall movements per 24-h*. For UPDRS-III 26-31 (gait and posture items), the score tended to be higher in the *severe* group.

The present study included patients with mild posture disorders, since they could walk unaided throughout the day. In these patients, the UPDRS gait and posture items were mildly affected. The narrow distribution might explain the lack of significant difference in the score of UPDRS 26–31. The same sampling bias in the PGR testing is also applicable to a few numbers of patients in the *severe gait disorder* group.

Compensatory parkinsonian walking. Our results showed no significant difference between the *mild* and *moderate gait disorder* groups. One possible explanation for this could be compensatory adjustments that can maintain gait performance when only

one gait parameter, *e.g.*, cycle or force, is affected. This argument is based on the finding of compensatory efforts commonly seen in patients with various gait disorders during walking [7, 18]. Thus, PD patients could improve gait performance by reinforcement of the intact gait control mechanisms, which results in preserved activities. In agreement with this notion, previous studies reported that patients with a short stride due to decreased kick-off forces tried to increase their cadence (steps/min) by shortening the gait cycle [9–11]. Under these conditions, the UPDRS-III gait and posture parameters or the *amount of overall movements per 24-h* would not be severely disturbed in patients with the *moderate gait disorder* compared to those with the *mild gait disorder*. Taken together, the present results suggest that latent motor disturbances could be unexpectedly advanced even in patients with mild impairment of various parameters of the UPDRS-III gait and posture. The present classification using PGR could be helpful in the early detection of such latent gait disorders.

In conclusion, continuous recording of daily lives using our newly developed PGR seems to allow the detection of elementary and primary deficits in gait motor control. Therefore, assessment of changes in both the gait cycle and gait acceleration could be employed to quantitatively define the severity of gait bradykinesia, and hopefully be used for fine tuning dopaminergic medications.

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