Quantitative Gait Analysis in Parkinson's Disease: Comparison With a Healthy Control Group

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ABSTRACT. Sofuwa O, Nieuwboer A, Desloovere K, Willems AM, Chavret F, Jonkers I. Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group. Arch Phys Med Rehabil 2005;86:1007-13.

Objective: To compare gait parameters in Parkinson's disease (PD) during the on-phase of medication cycle with those of healthy elderly control subjects.

Design: A group-comparison study.

Setting: Gait analysis laboratory of a university hospital.

Participants: Fifteen patients with PD and 9 healthy elderly controls.

Interventions: Not applicable.

Main Outcome Measures: Spatiotemporal, kinematic, and kinetic gait parameters.

Results: The PD spatiotemporal results showed a significant reduction in step length and walking velocity compared with controls. In the kinematics, the major feature of the PD group was a markedly reduced ankle plantarflexion excursion (at 50%–60% of the gait cycle). Most important, the kinetics showed reduced ankle push-off power and hip pull-off power. Unlike the control subjects, the patients with PD did not show any correlation between ankle generation (push-off) power and stride length (r=.19) or with gait speed (r=.29). Correction for walking velocity did not result in significant changes in the kinetics between the groups.

Conclusions: Reduced ankle (push-off) power generation and reduced hip flexion (pull-off) power persisted in PD gait despite being tested in the on-phase of the medication cycle. Lack of a correlation between ankle and hip power generation and walking velocity suggests that peripheral and central factors contribute to lack of forward progression. Patients with PD may benefit from intervention strategies that correct the kinematic and the kinetic gait components.

Key Words: Gait; Kinematics; Kinetics; Parkinson disease; Rehabilitation.

GAIT DISORDERS ARE COMMONLY observed in people with Parkinson's disease[1,2] (PD) and probably occur as a result of progressive loss of dopamine-producing cells of the substantia nigra compacta of the central nervous system. Absence of dopamine in the basal ganglia circuit ultimately results in the loss of gait automaticity. Clinically, people with PD usually have the hallmark features of slowness (bradykinesia),[2] cessation of movement (akinesia), or freezing of gait.[3] As the disease advances, these gait disorders become more pronounced, disabling the patients and severely limiting their quality of life.[4]

The reduction of stride length is considered the most prominent feature of PD gait and is often accompanied by lower walking speed and the tendency toward a longer duration in the double-support phase.[2,5,6] Other studies[7-10] have indicated that patients with PD are able to improve gait components, such as stride length and speed, if appropriate influences, like cues, are used.

Whereas most studies document the spatiotemporal changes of gait at baseline (no intervention level), few have focused on describing the kinematic and the kinetic parameters, especially in the on-phase of the medication cycle. Data, which include a statistical analysis of the kinetic changes during gait, are therefore lacking. However, 2 studies have described the kinematic and kinetic features of PD gait. Morris et al[9] reported on the gait parameters in 1 patient with PD during various testing conditions. The patient was able to correct the spatiotemporal and kinematic parameters through the introduction of an external cue but abnormality persisted in the kinetics, such as a reduced ankle power generation at push-off. Lewis et al[10] compared 14 PD patients with 14 age-matched controls and observed a reduced plantarflexion at toe-off and reduced ankle power generation at baseline. They also noted largely variable patterns of gait in the kinematics and kinetics. However, the statistical analysis of this part of their data was not presented. Therefore, the aim of our study was to compare statistically the spatiotemporal, kinematic, and kinetic parameters of PD gait in the on-phase of the medication cycle with the control values. The study also investigated whether gait speed contributed to some of the observed differences, because patients are expected to walk with reduced gait speed compared with controls. Furthermore, the relation between observed changes in spatiotemporal parameters and the reported kinetic changes in PD gait were analyzed.

METHODS

Participants

This study was part of a larger study in which 20 patients with PD and 10 healthy elderly control subjects were recruited. Informed consent was obtained according to the stipulations of the Declaration of Helsinki. For the purposes of this study, 15 patients with PD (11 men, 4 women) and 9 control subjects (3 men, 6 women) with comparable age, weight, and height (table 1) and with complete spatiotemporal, kinematic, and kinetic data sets were selected. The control subjects were selected

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Table 1: Group Characteristics

Characteristics	PD Group	Controls	*P* (*t* test)
Sex (F/M)	4/11	6/3	.054*
Age (y)	63.14±8.4	64.41±4.56	.787
Weight (kg)	68.0±13.5	73.89±10.46	.131
Height (cm)	162.7±5.8	163.9±8.3	.540

NOTE. Values are n or mean ± standard deviation (SD).
Abbreviations: F, female; M, male.
*χ^2 test.

Table 2: Spatiotemporal Variables

Variables	Controls	PD Group	*P*
Walking velocity (m/s)	1.19±0.11	0.94±0.21	.004*
Stride length (m)	1.24±0.10	1.03±0.16	.002*
Cadence (steps/min)	115.3±6.6	108.5±12.0	.133
Double support (%GC)	23.43±2.52	25.28±3.84	.212
Single support (%GC)	38.51±1.30	37.29±1.80	.090

NOTE. Values are mean ± SD.
Abbreviation: GC, gait cycle.
*Significant at $P<.05$.

from a population of healthy elderly people and were without sensory or other medical disorders affecting gait. The patients were diagnosed as having idiopathic PD and were referred by a consultant neurologist of the University Hospitals of Leuven. Patients were excluded if they had other neurologic problems; acute medical problems that could affect gait, such as visual or musculoskeletal defects; unpredictable off-periods (making stable testing difficult); and a score below 23 on the Mini-Mental State Examination (MMSE).[11] The mean age, height, and weight were 63.14±8.4 years, 162.7±5.8m, and 68.0±13.5kg, respectively, for the patients with PD and 64.41±4.56 years, 163.9±8.3m, and 73.89±10.46kg, respectively, for the control subjects. The mean illness duration for patients with PD was 11.25±3.8 years. They were mostly classified as stage II (n=7) and III (n=5) on the Hoehn and Yahr disease rating scale,[12] with an average of 2.6±0.6. The Hoehn and Yahr scale is a global measure of disease severity, identifying 5 disease stages (0–5), ranging from unilateral disease only to bedridden or wheelchair-bound mobility. In addition, rating of the disease severity using the motor section of the Unified Parkinson's Disease Rating Scale (UPDRS)[13] gave a group mean score of 16.1±6.4. The motor section of the UPDRS contains 14 items (speech, facial expression, tremor at rest, action tremor, rigidity, finger taps, hand movement, hand pronation and supination, leg agility, rising from the chair, posture, gait, postural stability, body bradykinesia). Each item is scored from 0 (normal) to 4 (unable to perform task). Statistically (Wilcoxon), there was no difference in MMSE scores of the PD and the control groups ($P=.735$).

Our study reports on the comparison of the more-affected side in PD patients with the left side in control subjects. The more-affected side was determined by questioning the patient. In 4 patients, the left side was the more-affected side but in 11 patients it was the right side.

Instrumentation and Procedure

Gait analysis was conducted using an 8 M-camera Vicon 612 data capturing system[a] set at 120Hz and 3 AMTI forceplates[b] mounted midway on an 8-m walkway. Retroreflective markers were placed on the specific anatomic points of the subjects' lower limbs, enabling 3-dimensional analysis during the gait cycle. These points were the anterior superior iliac spines, sacrum, midthighs, lateral malleoli, dorsolateral aspect of the foot between the second and third metatarsal heads, and on the calcaneus. Workstation and Polygon software[a] were used to manually define gait cycle events and to process kinematic and kinetic data. Another study[14] tested the procedure of manually defining gait cycle events and reported good interrater reliability with intraclass correlation coefficients, ranging from .88 to 1.0 in patients with PD. Joint internal moments (in Nm) and powers (in watts) were normalized with respect to the subject's body mass (in kilograms). The gait tests were performed during the on-phase of the medication cycle (1–2h after intake of their midday dose). Subjects were instructed to walk at their usual self-selected comfortable speed. Patients completed 3

Table 3: Kinematic Data for the PD and Control Groups

Joint	PD (deg)	Control (deg)	*P* (*t* test)
Ankle			
Initial contact	−0.50±2.91	−0.51±1.73	.99
Max dorsiflexion in stance	13.80±2.40	12.38±3.17	.226
ROM during push-off	19.29±4.70	24.05±2.84	.012*
Plantarflexion at toe-off	−5.49±4.51	−11.69±5.19	.005*
Max plantarflexion in swing	−7.74±4.54	−13.32±4.65	.008*
Max dorsiflexion in swing	6.46±2.75	4.83±1.68	.123
ROM in swing	14.20±4.14	18.15±5.17	.051*
Knee			
Extension/flexion			
Initial contact	7.52±4.39	6.71±3.25	.639
Max flexion in stance	17.11±3.53	17.81±5.74	.712
Max extension in stance	2.09±5.46	−1.27±5.61	.162
ROM in stance	15.01±5.40	19.08±6.80	.119
Flexion at toe-off	31.56±9.79	35.46±4.96	.280
Max flexion in swing	54.54±6.66	58.09±3.13	.149
Hip			
Extension/flexion			
Initial contact	36.14±8.47	40.06±6.57	.246
Max hip extension	−3.24±7.05	−5.68±5.67	.388
Flexion at toe-off	5.14±6.49	3.56±5.52	.549
Max flexion in swing	36.58±7.52	39.96±5.80	.261
Rotation			
Min	−15.10±16.49	−20.51±8.33	.372
Max	3.73±13.06	−0.45±10.50	.425
ROM	18.83±13.27	20.05±7.59	.804
Pelvis			
Tilt			
Min	12.90±8.88	15.38±3.77	.438
Max	16.94±5.63	17.94±3.64	.638
ROM	4.04±5.97	2.57±0.95	.474
Rotation			
Min	−4.87±4.67	−1.10±4.15	.059
Max	4.12±3.44	3.15±5.50	.600
Range of pelvic rotation	8.98±5.38	4.25±8.19	.100

NOTE. Values are mean ± SD.
Abbreviations: Max, maximum; Min, minimum.
*Significant at $P<.05$.

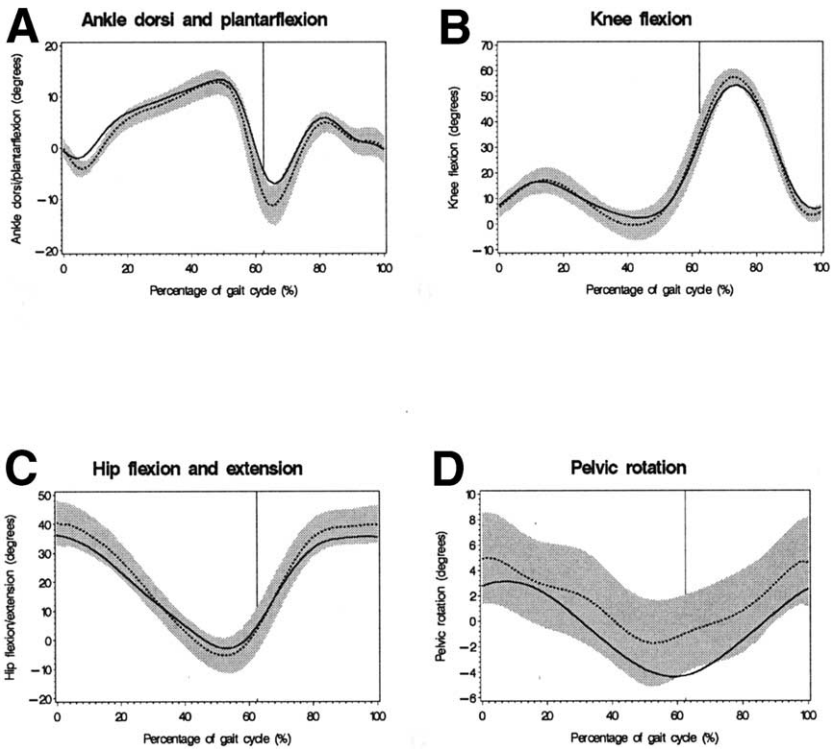

Fig 1. Kinematic data at the (A) ankle, (B) knee, (C) hip, and (D) pelvis across the gait cycle. Positive angles indicate joint flexion and dorsiflexion; negative angles represent extension and plantarflexion. The dotted line and shaded area represent the mean and ±1 SD, respectively, for the control group (n=9). The solid dark line represents the mean of the PD group (n=15). The vertical line represents point of toe-off (62.3% for controls, 62.4% for PD), distinguishing the stance phase of the gait cycle from the swing phase.

gait trials from which spatiotemporal, sagittal plane kinematics, and kinetics were obtained. From the 3 trials, we selected the one that had the most complete kinematic and concurrent kinetic data. For each subject, the mean of the 3 trials was obtained for the spatiotemporal data. In other studies,[15,16] good repeatability of 3 trials of normal walking has been reported. The selected kinematic and kinetic gait parameters used in the analyses were determined on the routine protocol of the gait analysis laboratory of the Pellenberg University Hospital, Leuven, Belgium.[17]

Statistical Analysis

We used an unpaired Student *t* test analysis to compare the mean spatiotemporal and mean kinematic data between patients with PD and control subjects. For the kinetic analysis, a multivariate model was used to study the kinetic pattern between the 2 groups with and without controlling for walking velocity (ie, a third variable—walking velocity—was included as a covariant in the multivariate model). An unstructured mixed model or a more simplified covariance matrix was used in SAS statistical analysis software, version 8.02,[c] for Windows. The results were expressed in estimated group means and standard error (SE) of the mean for each variable in the case of the multivariate models. The relation between the spatiotemporal variables and power generation in the lower limb during gait was calculated using the Pearson product moment correlation coefficient (r). Significance level for all analyses was set at α equal to .05.

RESULTS

Spatiotemporal Data

Table 2 shows the spatiotemporal variables of the PD and control groups. A significantly lower walking velocity ($P=.004$) and stride length ($P=.002$) was found for the PD group. They also appeared to have a lower cadence and spent more time in the double-support phase of gait, but these changes were not significant.

Kinematics

The kinematic differences between the 2 study groups were most pronounced at the ankle joint (table 3). The amount of maximal dorsiflexion observed in the stance and in the swing phase did not differ significantly between the 2 groups. Ankle range of motion (ROM) during push-off was significantly reduced in the PD group ($P=.012$)—that is, 19.8% lower than that of the control group. This resulted in marked reduction of plantarflexion at toe-off ($P=.005$). As a result, ROM in the swing phase was also reduced ($P=.051$). In the proximal joints, mean joint ROM was generally smaller in the patients with PD, but no significant differences were found. Kinematic data (fig 1) show that the average PD joint motion falls within the range of the control values. The timing of the plantarflexion excursion appears to occur slightly later than in the controls, but this was still within the range of the controls. Minimum (external) pelvic rotation was higher in the PD group but did not differ significantly from the controls ($P=.059$). In addition, mean pelvic ROM did not differ from that of the controls ($P=0.1$) (table 3). This is also depicted in the profile of the range of pelvic rotation motion, which remained within the range of the controls (see fig 1D). However, it appeared that the measured, more-affected side of the pelvis was always kept behind the less-affected side.

Kinetics

More differences between the groups were observed in the kinetics than in the kinematics (table 4). In the ankle joint of the PD group, there was a significantly lower (45%) moment at loading response ($P=.003$) and a reduction (15%) at mid and

Table 4: Kinetic Data for the PD and Control Groups

Joint	PD	Control	*P* (ANOVA)
Ankle			
Joint moment (Nm/kg)			
Moment at loading response	−0.08±0.01	−0.14±0.01	.003*
Max moment in mid and terminal stance	1.32±0.05	1.50±0.05	.021*
Minimum moment in preswing	0.05±0.01	0.06±0.02	.922
Power (W/kg)			
Absorption at loading response	−0.13±0.03	−0.12±0.04	.933
Generation at preswing	2.33±0.15	3.22±0.16	.001*
Knee			
Joint moment (Nm/kg)			
Max flexion moment in stance	−0.32±0.05	−0.40±0.05	.301
Max extension moment in stance	0.32±0.04	0.32±0.04	.952
Power (W/kg)			
Max power generated in stance	0.60±0.10	0.54±0.07	.627
Max power absorbed in late stance	−0.48±0.10	−0.90±0.12	.009*
Hip			
Hip joint moment (Nm/kg)			
Max extension moment in stance	0.71±0.12	0.83±0.12	.432
Max flexion moment in stance	−0.70±0.06	−0.94±0.07	.030*
Max abduction moment in stance	0.76±0.04	0.90±0.07	.097
Power (W/kg)			
Max power generated in stance	0.66±0.05	1.07±0.08	.001*
Max power absorbed in stance	−0.55±0.07	−0.82±0.08	.033*

NOTE. Values are estimated mean ± SE.
Abbreviation: ANOVA, analysis of variance.
*Significant at $P<.05$.

terminal stance phases ($P=.021$) compared with the control group. However, at loading response, ankle power absorption did not differ significantly from that of the controls. In the preswing phase (50%–60% of the gait cycle), the PD group also showed a significant reduction (28.2%) in ankle power generation ($P=.001$).

At the knee joint, the maximum flexion and extension moment generation in the stance phase did not significantly differ from that of controls. Both groups also showed no differences in maximum knee power generated in the stance phase. However, the PD group showed a reduction (43.7%) of the maximum power absorbed ($P=.009$) in the late stance phase (50%–60% of the gait cycle).

At the hip joint, maximum hip flexion moment (but not extension moment) was reduced (25.9%) in the PD group compared with the control group ($P=.03$). Also, a reduction (39%) of maximum hip power generated in the stance phase ($P=.001$) and a reduction (32%) of maximum power absorbed in the stance phase ($P=.03$) were observed. Figure 2 shows the mean kinetic profiles of the PD group in contrast with the normative range, as represented by the controls. The most notable difference was the decreased peak ankle power generated (at 57% of gait cycle) of the PD group, which was lower compared with the control group (see fig 2D).

Statistically controlling for the effect of walking velocity on the kinetics made no differences between the groups, except for the significantly reduced maximum hip flexion moment in the stance phase ($P=.03$), which was no longer statistically significant ($P=.09$) after correction for velocity.

No correlation was found between stride length or walking velocity and power generation at the ankle, knee, and hip joints in the PD group (table 5). In contrast, in the control group, ankle power generated at preswing correlated with stride length ($r=.69$, $P=.03$) and walking velocity ($r=.86$, $P=.002$).

It should be noted that, throughout the period of testing, no freezing of gait was observed in any of the subjects with PD.

DISCUSSION

This study describes the spatiotemporal, kinematic, and kinetic variables of gait in subjects with PD compared with control subjects, as measured in the on-phase of their medication cycle. Despite being tested during the on-phase, gait abnormalities were still observed. Although few kinematic changes were found, possibly because of large variability in the data, pronounced differences between PD and controls were present in the kinetic profiles.

Spatiotemporal

The key spatiotemporal findings in the PD group were consistent with most previous studies on spatiotemporal data in PD gait.[2,10,18-20] These studies also documented a tendency of longer duration in the double-support phase of the gait cycle and a normal cadence. In other studies,[21,22] a reduction of cadence values was reported. The fact that no significant difference was found in the double-support duration may indicate that patients were only moderately affected by PD. The clinical characteristics of the patient group confirm this supposition.

Kinematics

PD gait was characterized by reduced joint angular excursions. This was mainly true at the ankle joint but not at the knee or the hip joints. This finding is in agreement with other studies that have reported reduced ankle joint angular excursions of subjects with PD.[9,10] Most important, plantarflexion (but not dorsiflexion) was decreased in the PD group and consisted of a reduction in ROM during push-off and a reduction in plantarflexion at toe-off.

Kinetics

Abnormality in the pattern of the kinetics was more pronounced than in the kinematics. This discrepancy may be related to the fact that similar kinematic patterns can be produced by different underlying kinetics. At the ankle joint, the moment at loading response (dorsiflexion moment) was reduced in the PD group. This could occur if the ground reaction force vector remained close to the ankle joint. Hypothetically, this is possible if, at initial contact, the subjects with PD had limited hip flexion, inadequate knee extension, or absent heel strike. Although joint angular values at initial contact were highly variable, the PD group had a higher mean value of knee flexion (with consequent inadequate extension) and lesser hip flexion. As a result, a smaller heel rocker would ensue with a consequent flat-footed gait pattern. The feature of a flat-footed gait pattern in PD was also observed by Nieuwboer et al,[23] in a study of plantar force distribution in PD gait. Nieuwboer

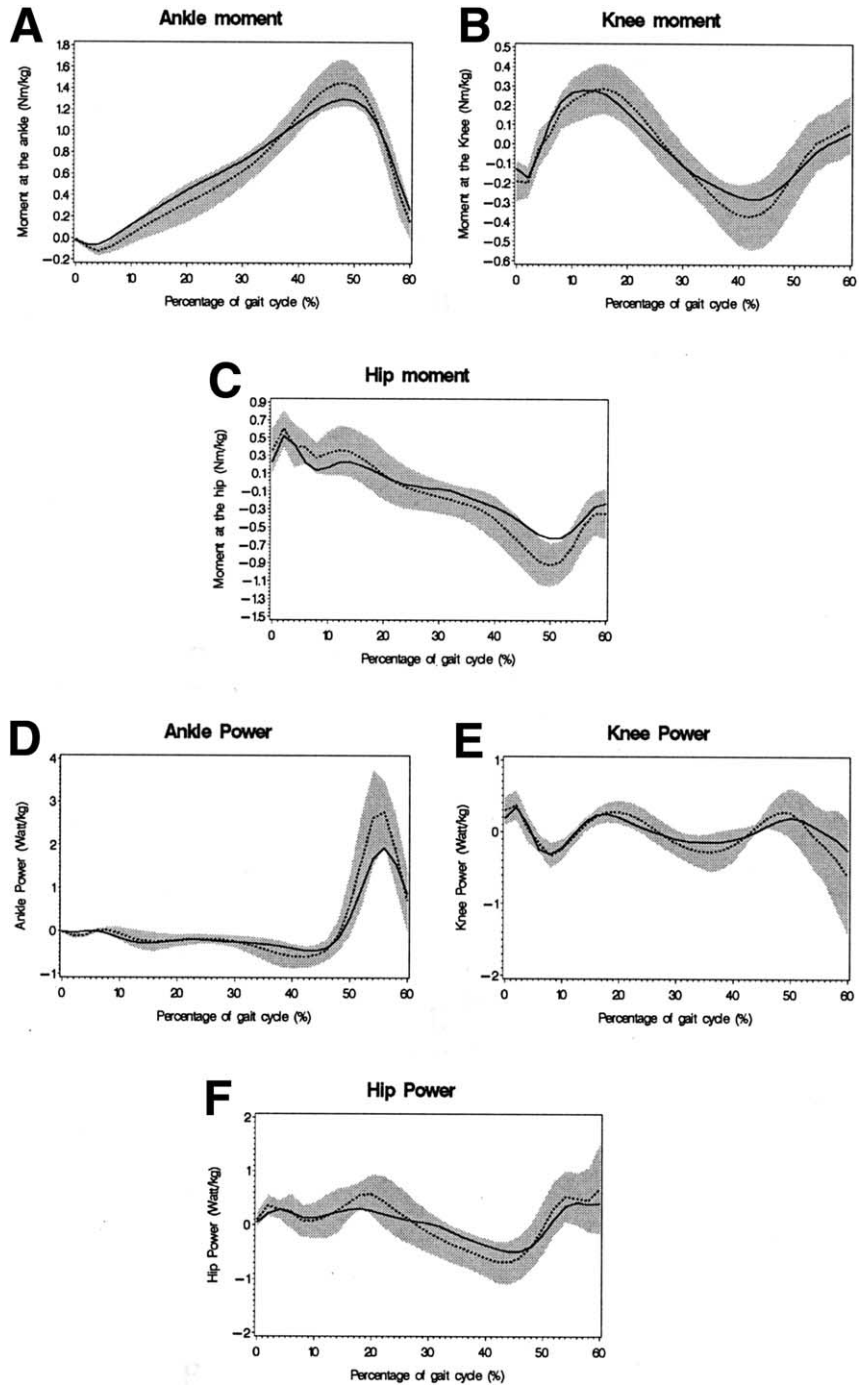

Fig 2. Kinetic data at the (A, D) ankle, (B, E) knee, and (C, F) hip during the stance phase only. Positive moment values indicate internal extension and plantarflexion moment; negative values represent flexion and dorsiflexion moments. Positive power values represent power generation; negative values represent power absorption. The dotted line and shaded area represent the mean and ±1 SD, respectively, for the control group (n=9). The solid dark line represents the mean of the PD group (n=15).

observed that subjects with PD increased the load at the midfoot when compared with control subjects.

A puzzling observation at the ankle joint is the normal ankle power absorption that was present with the aforementioned reduction of the ankle moment at loading response. One would expect that both are either reduced or increased. A plausible explanation of this contradiction is that, because the power absorbed is the product of the reduced moment times angular velocity, it is possible that the ankle angular velocity may have influenced the power absorbed.

As was observed in the plantarflexion ROM, the plantarflexors appeared to be more affected than the dorsiflexors. The kinetic data support this observation by showing a reduced maximal (plantarflexion) moment in mid and terminal phases (10%–50% of gait cycle). Further corroborating the alterations of the plantarflexors in PD gait, reduced ankle power was generated in the preswing phase (push-off power). This was supported by the kinematic data because we found a reduced ROM in push-off and reduced plantarflexion excursions at toe-off. The reduction in plantarflexion and push-off power is not a new finding. Other studies have shown that subjects with PD have a reduced third rocker (roll-off) at the terminal stance of gait[24] and underscaling of power generation at push-off,[8,9] with reduced amplitude of electromyographic activity in the

Table 5: Correlation Coefficients (Pearson) of Selected Gait Variables in PD and Control Groups

Group	Stride Length	Velocity
PD		
Ankle power generated at preswing	.19 (P=.49)	.29 (P=.28)
Max knee power generated in stance	.31 (P=.25)	.23 (P=.38)
Max hip power generated in stance	.21 (P=.43)	−.09 (P=.73)
Control		
Ankle power generated at preswing	.69 (P=.03)*	.86 (P=.002)*
Max knee power generated in stance	.09 (P=0.8)	−.47 (P=.19)
Max hip power generated in stance	.19 (P=0.6)	.57 (P=0.1)

*Significant at $P<.05$.

gastrocnemius muscle.[25] Furthermore, ankle power generation is believed to be the strongest predictor of step length in healthy elderly subjects, as was observed from a linear regression model used by Judge et al.[26] Judge suggested that increasing ankle plantarflexor power would likely increase the step length. Another study[27] found that ankle strength (and muscle power) correlated positively with gait velocity and stride length in older persons. Although the control group in our study also had a positive correlation between ankle power generated and stride length and velocity, the PD group did not show such a relation. Factors other than ankle power generation alone (push-off power) may therefore be responsible for the reduced step length. In the study of Lewis et al,[10] the question was raised whether patients with PD specify a normal stride length but fail to generate the required stride amplitude, or whether they specify a shortened stride length and regulate their movement around a smaller scale. It appears from the correlation analysis that no linear relation exists between stride length and peripherally reduced ankle power generation. Hence, in PD, reduced stride length may be determined by complex disease-related factors interacting with each other in a nonlinear fashion, such as deficient central specification, rigidity, and reduced muscle power.

At the knee joint, we found a lower power absorption in the late stance phase of the gait cycle (45%–60% of gait cycle). In a study of gait in healthy elderly subjects, Winter[27] related the normal power absorbed at this phase to the outcome of the reduction of the energy that was transferred from ankle push-off power. Also, Judge[26] interpreted this power absorption as the natural consequence of the transfer of energy generated by the ankle plantarflexors to the knee. This could mean, in the present study, that a reduced power generation (push-off) at the ankle would cause a reduced knee power absorption in the late stance phase.

The significantly reduced maximum hip flexion moment in the stance phase found in the PD group can occur because of inadequate hip extension, as shown in the kinematic data (decreased maximum hip extension and increased hip flexion position at toe-off). This will limit the progression of the trunk and therefore reduce the internal flexion moment as well as the hip power generation in the stance phase (50%–60% of gait cycle). Indeed, hip power generation in the stance phase (at 50%–60% of gait cycle) was significantly reduced in the PD group. In healthy people, hip power generation is required to accelerate the thigh and leg into the swing phase; therefore, its reduction would cause a decrease in the energy (or pull-off power) of the lower limb of the patients with PD. Morris et al[9] had expected to find an increased hip flexion power in the late stance phase as a compensatory strategy, which they found to be normal in their study on 1 PD subject. However, in this study, in case the inadequate push-off at the ankle is compensated for by reinforcing the pull-off at the hip level, this would be reflected in an increased hip (pull-off) power generation at the stance-swing phase transition. The opposite was apparent in the result of our study. Furthermore, the pelvic retraction of the more-affected side suggests that the propulsive capacity of the more-affected side was relatively deficient. It may therefore be concluded that the normal compensatory strategies were not fully exploited in patients with PD.

The typical slow gait in subjects with PD, as measured in comparison with controls in this study, may therefore be explained by the reduced ankle moment at loading response (tending to a flat-footed gait), the reduced ROM at push-off, and the reduced hip power absorption and generation in stance.

The differences between patients with PD and controls cannot be explained by the difference in gait velocity alone, because statistically controlling for gait speed did not result in important kinetic alterations, except for the increased maximum hip flexion moment in stance that was normalized after correction. This may mean that the hip flexion at initial contact was adequate when velocity was accounted for. Future investigation should be done using controlled velocity conditions to compare this finding.

A common striking feature of our data is the high variability in the gait pattern of the PD group and controls. Lewis[10] observed marked variability in subjects with PD. The fairly small sample size would have contributed to this variability. Therefore, the lack of statistical power may have influenced the difficulty of finding consistent trends. Intersubject variability of the healthy elderly subjects' data could also have influenced the differences between the 2 study groups. However, the control group's spatiotemporal and kinetic data were highly comparable to other healthy elderly controls in other studies,[28,29] with dissimilarity only present in maximum ankle push-off power of 6.04 ± 1.26W/kg,[27] compared with this study's 3.22 ± 0.16W/kg. Future studies need to increase the number of subjects and sex distribution within each study group, to allow subgroup analysis to be performed. Comparison between the more-affected side and the less-affected side is suggested. It is also suggested that electromyographic data be incorporated into future studies, to aid in interpreting joint moments and powers, as well as kinematic analysis in the coronal and transverse planes.

Implication for Therapy

Morris[9] studied the effect of visual cues on the gait of 1 patient with PD and found that the key spatiotemporal and kinematic parameters were adjusted toward the normal, but the kinetics remained abnormal, most notably the ankle plantarflexor power generation at push-off.

Our study did not find any correlation between ankle plantarflexor push-off power and stride length. In contrast, Scandalis et al[30] found that when patients with PD undergo calf resistance training, muscle strength increased to levels comparable to the normative levels. This gain in strength was found to translate into quantitative improvements in gait parameters such as stride length. It is suggested that further studies need to confirm the role of plantarflexors in the generation of stride length in PD gait and what effect the coupling of plantarflexor strength training and cueing will

have on gait. In contrast to the aforementioned feed-forward visual and auditory cueing mechanism, a feedback mechanism with a real-time visual or auditory display may be used, to elicit immediate gait adjustments.

CONCLUSIONS

The data of our study confirm that ankle plantarflexors are mostly affected in PD gait. In addition, hip flexors appear to be implicated in the abnormal gait pattern in PD. Walking velocity did not largely affect the results, which suggests that it is not the cause of the kinetic gait deviations found. Moreover, lack of correlation between stride length, gait velocity, and ankle and hip power generation suggest that central factors, as well as peripheral factors, are involved in the diminished gait parameters in PD. Patients may benefit from novel interventions that influence these factors and correct the gait abnormalities not only at spatiotemporal and kinematic levels but also at kinetic levels.

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Suppliers

a. Vicon Motion Systems Ltd, 14 Minns Estate, West Way, Oxford, OX2 0JB, UK.
b. Advance Mechanical Technology Inc, 176 Waltham St, Watertown, MA 02472.
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