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PLANTAR FORCE DISTRIBUTION IN PARKINSONIAN GAIT: A COMPARISON BETWEEN PATIENTS AND AGE-MATCHED CONTROL SUBJECTS

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ABSTRACT. This study aimed to ascertain whether roll-off of the feet during gait was essentially different in patients with Parkinson's disease from that of elderly control subjects. Twenty-two patients, belonging mainly to Hoehn & Yahr grades III and IV, and 30 elderly people participated in the study. Plantar force distribution data were collected of two consecutive strides using pressure-sensitive insoles as part of the pododynograph system. Results showed that when correcting for gait speed and sex differences, patients with Parkinson's disease walked with significantly lower relative peak forces at the forefoot and heel and increased load at the midfoot. The onset of peak forces indicated slower load acceptance on the heel and early forefoot loading which was confirmed by a reduced amplitude of the centre of force along the length of the foot compared with healthy controls. Roll-off was significantly reduced in patients with Parkinson, a feature which was specific for the disease rather than a result of reduced gait speed alone.

Key words: Parkinson's disease; gait; plantar force; foot strike; walking speed.

INTRODUCTION

One of the typical features of gait in patients with Parkinson's disease (PD) is that the overall movement pattern remains more or less normal, except for the markedly reduced angular displacements in hip, knee and ankle joints (18, 27). Slowness of gait with reduced stride length and shuffling also characterise Parkinsonian walking (3, 4, 25, 27, 31). When comparing gait performance in patients with PD during both preferred and fast speed conditions, with and without visual cues, Morris et al. (25) found that patients had specific difficulties with enlarging their stride length. Also, it was demonstrated that the stride-cadence relationship was similar to that of normal controls but that patients had preset their stride length at a lower level (24). Hence, basal ganglia dysfunction seems to interfere with the normal tonic discharge in cortical motor neurons, resulting in an underestimation of the required movement amplitude for each gait cycle (24–26). Blin et al. (4) showed that the variability of stride length was more marked in patients with PD than in normal controls. These findings are consistent with the increased variability of shape found in the tibialis anterior and gastrocnemius muscles' EMG profiles during gait (23). Dietz et al. (7) established that when controlling for gait speed differences, the amplitude of gastrocnemicus EMG activity was reduced in patients, while tibialis anterior activation was similar compared with controls. These problems may be an expression of the general bradykinetic features of an impaired rate of force production (30) and more variable motor unit firing (12); or may reflect secondary neuromuscular dysfunction due to disuse. Knutsson & Mårtensson (18) showed that phasic activations of the lower limb muscles followed the normal pattern in Parkinsonian gait, but that the periods of relaxation in between were reduced. This was interpreted as rigidity, a possible further contributing factor to the pathogenesis of gait bradykinesia in PD.

Analysis of the plantar force distribution in normal subjects indicated that average loading of the forefoot was approximately three times that of the heel in barefooted young individuals (1). Peak pressures were not reached until 25% of stance phase at the heel and 80% at the forefoot. Plantar pressure displayed the highest peaks at the central heel, central forefoot and hallux regions, whereas the lowest values occurred at the midfoot (29). Furthermore, it was found that increasing walking speed not only increased plantar peak pressures but altered the pattern significantly (29). Higher peak pressures were recorded in the heel and medial forefoot and lower values were seen in the midfoot in contrast to

slow speed gait. After adjusting for speed differences, foot loading of older and younger adults was largely similar, except for the smaller contact times, peak forces and decreased impulses in the elderly (16). Wearing shoes was found to produce a more even distribution of force over the foot (1).

Despite the fact that abnormal foot placement and toe clearance may be factors which enhance the risk of falling (19), limited data are available on this aspect of gait in PD. Both Knutsson & Mårtensson (17) and Murray et al. (27) described diminished toe elevation at heel strike and foot clearance during swing phase. Time between the heel and the ball of the foot hitting the floor was found to be shorter in patients with PD compared with control subjects, indicating a typical flat-footed gait (28). Hughes et al. (13) discovered that simultaneous heel and forefoot strike occurred in 16% of controls and in 50% of medicated patients with PD. Hitting the floor with the forefoot first, which was not seen in control subjects, was apparent in 19% of patients on medication and 32% after placebo. Koozekanani et al. (20) found in 2 patients with PD a normal appearance of two peaks of ground reaction forces, representing the onset of mid-stance and push-off, but a reduced magnitude compared with normal values, especially for the second peak. Ueno et al. (32) established from five patients with severe shuffling gait that the double-peaked force curve was replaced by a single narrow peak.

Abnormal foot strike was modified by L-dopa treatment, albeit not to normal levels (10, 13). In addition, rehabilitation may be required for the management of this gait disorder, using visual, attentional and auditory rhythmic cues (2, 15, 22, 26). The aim of this study is to gain insight into the nature of abnormal foot roll-off in PD by comparing patients' plantar force distribution patterns during gait with a normal age-matched control group. Furthermore, it addresses whether alterations of foot loading are related to gait speed alone or indicate a fundamental deficit of PD.

METHODS

Subjects

Patients were recruited for gait analysis if they had a diagnosis of idiopathic PD confirmed by a consultant neurologist and presented with functional motor problems including a deterioration of gait. Patients were excluded if they had: (1) severe cognitive problems (<24 score on the Mini Mental Scale (6)); (2) other acute medical problems which would influence gait; (3) severe dyskinesias (>2 score on the UPDRS) (8); and (4) unpredictable off-periods. Twenty-two patients took part in the study, 13 men and 9 women; age range 51–81 years, mean 66, SD = 7.5. Data from thirty control subjects were used from a previous experiment (8 men and 22 women, age range 59–73 years, mean 65.5, SD = 3.5) (9). The mean weight of patients (72.7 kg, SD = 12.4) was highly similar to that of controls (73.4 kg, SD = 11.1). Disease duration varied from 4 to 19 years ($\overline{X}$ = 11.7, SD = 4.6). Twelve patients (54%) fell into Hoehn & Yahr grade IV during "on" (8). Seven (32%) were categorised into grade III and three (14%) into grade II. Patients were on a stable medication regimen, which in most cases involved a combination of Levodopa, Dopamine-receptor agonists and/or Selegiline. The control subjects lived in residential homes and were free of other medical problems that could impede their walking ability. They did not need walking aids. The patients were also screened for disabling conditions and specific foot abnormalities. Two patients had had hip and pelvic fractures more than one year ago. As a measure of general mobility, the activity levels of patients and controls were categorised according to Imms & Edholm (14). Twelve patients (54%) were classed as housebound. Six (28%) were able to go outdoors with some limitations and 4 (18%) had unlimited mobility outdoors. This pattern was quite different for the control group, of which 20 (67%) were classed as having unlimited outdoor mobility despite living in residence. Two control subjects (6%) were housebound and eight had limited outdoor activity (27%).

Procedure and measurement equipment

Prior to gait registration, demographic data, clinical information and body weight measurements were registered. Patients continued to take their normal medication regime. They were measured during the stable "on"-period, one to two hours after taking their morning or afternoon dose. Measurements during the "off"-state were not undertaken, as freezing and starting problems might have hindered gait analysis. Patients walked along a 6-metre long and 0.6-metre wide walkway. Gait velocity in the healthy group was determined over a 10-metre distance using a stopwatch, while video recordings were used for patients. Connelly et al. (5) calculated high within-rater and between-rater reliability of using a stopwatch method at two different days in a frail and elderly population (ICC's ranging from 0.78–0.93). Mean stride length was measured with video in patients and calculated from speed and cadence in control subjects. Both patients and controls wore the same standardised shoes which were available in different sizes. They were asked to walk the required distance with the pododynograph (PDG) system at a freely chosen comfortable speed. The PDG system measured plantar pressure and temporal data. It consisted of two pressure-sensitive insoles worn in the shoe. Each insole contained 64 pressure sensors. Spatial resolution differed from 1 sensor per 2.25 to 3.6 cm^2, according to the size of the insoles used (sizes 35–37, 38–40, 41–43). Each sensor had its own calibration file. Sampling frequency was 50 Hz during 10 seconds. The insoles were connected to a portable recording module, which was fixed to each patient's torso, allowing maximal freedom of movement. Several practice trials were carried out to familiarise the patient with the procedure and the equipment in order to achieve a natural rhythm of gait. Data of two consecutive trials were transferred to a computer for further analysis.

Data analysis

Results are based on the averages of two consecutive strides in the middle of the walkway covered during two trials. Temporal

Table I. *Temporal gait characteristics of the right foot, mean difference between left and right peak force (Dif-PF) and impulse (Dif-IMP), symmetry quotient and p-values of Wilcoxon rank sum tests (W) or t-tests (t). NS stands for not significant $p > 0.05$.*

	Patients		Controls		
	Mean	(SD)	Mean	(SD)	*p*-value (*t*-test or W)
Speed (m/s)	0.74	(0.23)	1.12	(0.18)	0.0001 (t)
Stride time (s)	1.19	(0.25)	1.07	(0.08)	NS (W)
Stride length (m)	0.75	(0.28)	1.07	(0.24)	0.0001 (t)
Stance phase (%)	68.7	(3.5)	64.1	(1.8)	0.0001 (t)
Swing phase (%)	31.3	(3.5)	35.9	(1.6)	0.0001 (t)
Double support (%)	17.5	(3.3)	14	(1.5)	0.0001 (t)
Dif-PF (% BW)	29.4	(35.6)	1	(18.3)	0.0005 (W)
Symmetry (%)	74.7	(15.9)	88.5	(10)	0.0012 (t)
Dif-IMP (% BW × s)	11.8	(15)	0.5	(8.6)	0.002 (W)
Symmetry (%)	76.6	(15)	89.6	(10.7)	0.0014 (t)

data, i.e. double limb support, stance and swing phase, were expressed as a percentage of total stride time. Pressure data were transferred into force values and normalised with respect to body weight (%BW). To account for the asymmetry of weight-bearing found in patients (see Results), the force data were expressed as a percentage of the total mean force on each foot. Two relative force variables were calculated: (1) peak forces; and (2) impulses, or the force/time products (%BW × sec). In addition, the time when peak force was reached was calculated as a percentage of the stance phase. These parameters were computed for the total foot as well as for the following plantar regions: heel, midfoot, forefoot and toes. For the total force data, a difference score was calculated between the right and left foot as well as a symmetry ratio. This ratio was calculated to be below 1, depending on whether the right or the left had the highest value and was expressed as a percentage. The position of the center of force was projected onto the longitudinal axis of the foot at every 10% of the stance phase. All statistical analyses were performed using the SAS system. Temporal gait characteristics and forces during stance on the total foot were compared between patients and controls using unpaired t-tests or Wilcoxon signed rank tests when the data were not normally distributed. An analysis of covariance was used to analyse force distribution in the various foot zones with group as the main effect and speed and sex as covariates. Speed rather than stride was used, because in an earlier study this factor was found to be an important determinant of the roll-off pattern of the foot in healthy individuals (29). Moreover, stride and speed proved to be highly correlated variables ($p < 0.0001$) in both healthy and patient groups. The sex covariate was included because the groups were ill-matched for sex and a clear impact of this factor on roll-off was established in this study. Age and stage of the disease rendered insignificant results and were excluded from the multivariate model. The progression of the centre of force was analysed with a random effects model ANOVA, using a mixed procedure, allowing for the difference in variability between patients and controls (21).

RESULTS

Temporal gait characteristics and asymmetry

Because findings on the temporal data were highly similar between both feet, only results of the right foot are given in Table I. It shows that patients walked with significantly slower speed and smaller strides than control subjects. Total stride time did not differ substantially between groups. Within the gait cycle, relative time spent in the stance and double support phases was longer, whereas the swing phase was shortened in patients. These changes were statistically significant for both the left and the right foot.

Analysing the difference of total peak forces as well as total impulses on both feet showed that patients had a much more asymmetrical distribution of weight with more weight borne on the left foot (Table I). This overall difference was found while 15% of patients favoured their right over their left leg as did 43% of control subjects. Mean differences between the left and right were significantly smaller for the control group, although large standard deviations point to individual exceptions. Expressing the amount of symmetry as a ratio, independent of bias to one side, revealed that patients' ratios approximated 75%, differing significantly from those of controls (approaching 90% for both force variables, $p < 0.01$). Fig. 1 represents the advancement of the centre of force over both feet showing only small differences between right and left for both groups. Analysing the significance of these differences using a random effects procedure did not show significant interaction between time and group (95% CI = [−0.003; 0.009], $p = 0.31$) implying no relevant difference in

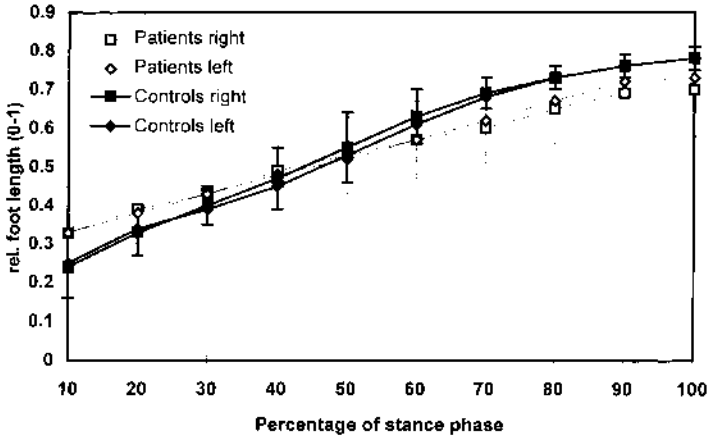

Fig. 1. The mean progression of the centre of force of patients and controls at 10 time points within the stance phase. Error bars indicate the standard deviations.

asymmetry of the progression of the centre of force between groups.

Relative peak forces at the four foot zones

Results of the ANCOVA showed that the confounding variables of sex and speed explained part of the differences found between patients and controls. Detailed information can be obtained from the authors on request. In Fig. 2, the distribution of the adjusted mean peak forces at the four designated foot zones is given. Variability indicated by the standard errors was relatively similar for both patients and controls. The figure shows that the difference in peak force between the groups was significantly lower in patients at both forefeet (25.7% BW at the left, 18.9% BW at the right), the right heel (15% BW) and the right toe area (10.1% BW). The most striking finding, however, was the increase of peak forces at the midfoot regions of both feet. Adjusted mean peak forces indicated an increment of 1.5 and 1.9 times the normal values of left and right midfoot, respectively. This result was obtained while a significant interaction was found between speed and group at the left forefoot ($p < 0.001$). Further analysis revealed that for subjects

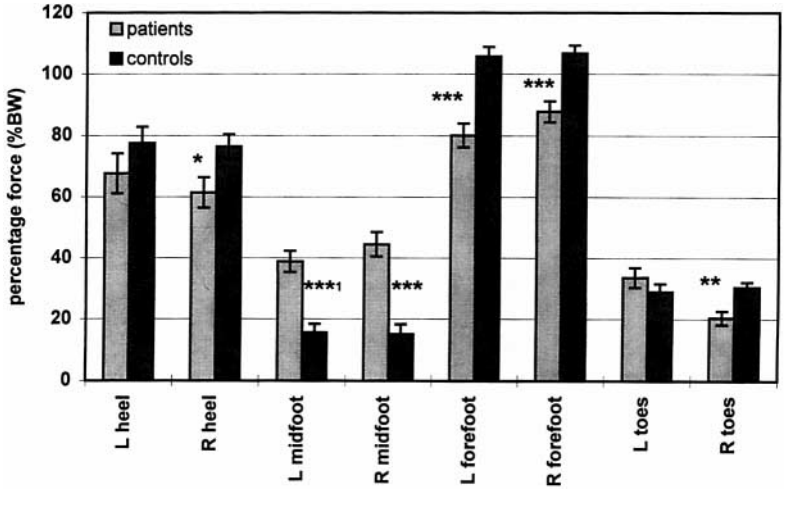

Fig. 2. The adjusted mean values of relative peak force at the four foot zones in patients and control subjects (* $\leq$0.05, ** $\leq$0.01, *** $\leq$0.001, ^{***1} $\leq$0.001 for high speed group only). Error bars indicate the standard errors.

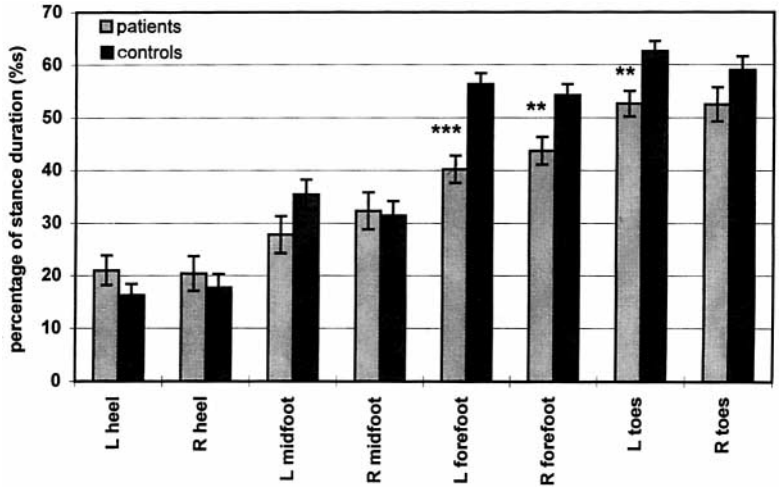

Fig. 3. The adjusted mean values of timing of peak force occurrence at the four foot zones in patients and control subjects (** $\leq$0.01, *** $\leq$0.001). Error bars indicate the standard errors.

with a higher speed than the median (0.99 m/s), group effect was highly significant ($p = 0.0005$, 95% CI = [−47.1; −15.2]). For subjects walking slower than 0.99 m/s, the group effect was an almost significant factor ($p = 0.07$, 95% CI = [−36.6; −25.3]).

Relative time to peak forces at the four foot zones

The analysis revealed that the covariate speed was a particularly significant factor in determining the onset of peak forces. Despite this, significant group differences emerged. Peak forces in controls occurred significantly later at both forefoot areas (16% of the left and 11% of the right stance phase) and at the left toe zone (10%), as can be seen in Fig. 3. Peak forces at the heel and the right midfoot tended to appear later in patients but these changes were not significant.

Relative impulses at the four foot zones

Fig. 4 displays the results of impulse distribution after correction for sex and speed. A slightly different pattern

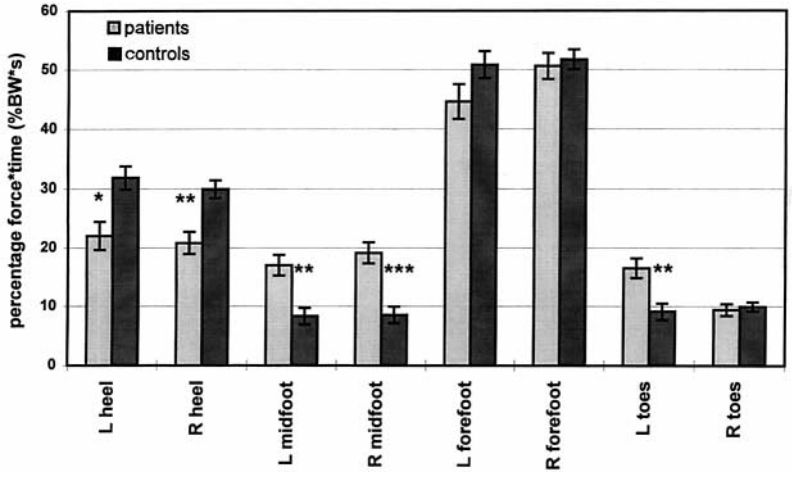

Fig. 4. The adjusted mean values of relative impulse at the four foot zones in patients and control subjects (* $\leq$0.05, ** $\leq$0.01, *** $\leq$0.001). Error bars indicate the standard errors.

of group differences was found compared with peak force. Patients showed significantly decreased impulses at the heel areas, a finding which was less pronounced with peak force. The magnitude of these group differences was 9.8% BW on the left and 9.1% BW on the right. Also, impulse values at the forefeet were reduced in patients but not significantly so. In agreement with earlier results, the most important finding was the increase of load on both midfoot regions. Patients showed increments of 1 and 1.2 times that of normal values. A significantly larger mean impulse was found at the left toe area in patients, which was not replicated on the right side.

Progression of the centre of force

Fig. 1, which displays the mean and standard deviation of the position of the centre of force at ten time points during stance for the two groups, shows that the curves of patients were flatter. Patients started their roll-off more towards the midfoot as indicated by the larger intercepts of 0.33 for both feet (SD = 0.13) compared with 0.24 and 0.25 (SD = 0.07 and 0.08) in controls. They also ended their movement over the feet less far forward at 0.7 and 0.73 (SD = 0.12) in comparison with controls at 0.78 (SD = 0.03). Also, a lower variability was observed for controls and this dispersion decreased at the end of stance. From Table II, it can be learned that group, sex and speed had a significant effect on the centre of force displacement. In the case of speed, for instance, this meant that individuals with a higher speed had on average a more forward position of the centre of force. However, looking at the interaction of speed with time, addressing its effect on the evolution of the centre of force over time showed that there was no significant effect of velocity ($p = 0.14$), meaning that the centre of force's actual pathway was not determined by this factor. The results for sex pointed towards a significantly different pattern for males in contrast with females in both groups. The male progression of the centre of force was characterised by a larger excursion and slope (95% CI of the mean difference between slopes = [0.08; 0.2]). Taking these findings into account, the group × time interaction remained highly significant in the right (95% CI = [−0.045; −0.017], $p = 0.0001$) as well as in the left foot (95% CI = [−0.042; −0.013], $p = 0.0006$).

Table II. *Random effects ANOVA of the progression of the centre of force taking the covariates sex and speed into account, T- and probability values (main factor is time × group)*

	Left foot		Right foot	
	T	*p*	T	*p*
Speed	2.06	0.04	2.44	0.01
Sex	4.97	0.0001	2.55	0.01
Group	3.39	0.001	4.03	0.0002
Time × speed	−1.29	NS	−1.46	NS
Time × sex	−4.77	0.0001	−3.1	0.002
Time × group	−3.67	0.0006	−4.20	0.0001

DISCUSSION

The purpose of this study was to establish whether abnormal foot loading in PD represented the slowness of gait normally observed in this condition or indicated a distinct feature of basal ganglia dysfunction. Patients from the more advanced stages of PD were compared with members of a normal control group, who, despite living in residence, were in 67% of cases enjoying unlimited mobility and whose gait speed fell within the approximate ranges of normative data for elderly subjects (33).

Gait characteristics of the patients reiterated what was reported previously: patients walked with 66% of the mean speed and 70% of the mean stride length of control subjects (3, 25, 26); stride times did not differ significantly, reflecting a normal cadence; stance and double support phases were significantly prolonged and swing phase reduced in patients (3, 26–28). Whereas previous studies established asymmetry of the temporal parameters of gait (28), this study revealed that patients with PD exhibited unequal weight-bearing as displayed by the differences of the total forces and the symmetry ratios. Nevertheless, this asymmetry did not reveal itself in the roll-off pattern as such. Only small and insignificant differences between the progression of the left and right centre of force were found between groups. Asymmetry is not a surprising result in a disease where hemidominance of one side may frequently occur. The data of this study did not allow further analysis of such a possible relationship.

When interpreting the force distribution data, a distinction was made between the three important phases of roll-off in relation to gait function: (1) initial contact at the heel and subsequent acceptance of body weight, the so-called "first rocker"; (2) midstance, or "second rocker", in which full plantar contact is reached and weight is transferred from heel to forefoot; and (3) push-

off at the end of stance phase, in which the body is propelled forward, also known as the "third rocker" (11). Results applying to the first phase showed that mean peak force and impulse at the heel were meaningfully reduced compared with normal controls (except for peak force at the left side). These results are in concurrence with earlier reports of a less pronounced heel strike observed in PD (13, 17, 28). Forssberg et al. (10) attributed the loss of heel strike in patients during the "off"-state of the medication cycle to a regression towards a more immature plantigrade walking pattern. Although Dopamine-replacement therapy evoked a return of normal foot function, this study illustrated remnants of such a primitive gait during the "on"-phase. The relative time at which peak forces were generated also showed a trend towards a delayed heel strike. The most significant alterations of foot loading, however, pertained to the second phase of roll-off. During this stage, forces applied at the midfoot of healthy subjects were found in other studies to be low, except for subjects with a collapse of the longitudinal arch such as pes planus (1). In this study, increased impulse and peak forces were demonstrated at the midfoot zones in patients, which were generally more than twice that of normal controls. Decreased heel and higher force levels at the midfoot reflected a more equal spread of force as patients were unable to produce a normal "heel to toe" roll-over. This diminished roll-over can hardly be explained by the fact that the patients from this study were collectively suffering from pes planus, as no clinical evidence of this was apparent. A more plausible explanation may be, that in analogy to stride length, patients with PD find it more difficult to maintain normal amplitudes of movement during repetitive automatic movement tasks such as gait (24). Secondly, it can be argued that patients are inclined to load this area of the feet more during midstance to compensate for possible loss of postural control frequently accompanying the disease (19). Looking at the results for the end of stance phase, a pattern of diminished mean peak forces was found at the forefeet in patients. Mean impulses at the forefeet were also smaller but this finding did not reach significance. This mild discrepancy may be explained by the fact that peak forces are a measure of dynamic action of the foot, whereas impulse indicates the general load on a specific area, as it includes duration of support. It is likely that the duration of forefoot loading was increased in patients, as the onset of peak forces occurred earlier at the forefoot and toes, which is in accordance with Pedersen et al. (28), who found the time between the heel and the ball landing on the floor reduced in patients with PD. Therefore, prolonged stance on the forefoot may have inflated patients' impulse values. Decreased forces acting at the forefoot may be ascribed to a reduced push-off, which is consistent with the finding of a decline of gastrocnemicus activation found in patients with PD compared with controls (7). The preceding gait events of a less dynamic heel strike followed by a flat roll-off may in itself impede the subsequent generation of push-off, as will the kinematic abnormalities of movements such as lack of extension amplitude in hip and knee during gait (18, 27). The functional repercussions of a reduced push-off are that the leg is not adequately accelerated into swing, thus worsening stride length and gait speed. Thus, in addition to the deficient central regulation of stride length in PD, peripheral factors such as flat-footed gait may further inhibit an already compromised stride length, creating a vicious circle of impairment.

The tendency of patients' centre of force to be further forwards at the beginning of stance and progress less far forwards confirmed the abnormal heel strike and on-going roll-off. The centre of forces' longitudinal pathways were also affected by sex. Despite this confounding influence, differences between groups were clearly significant for both feet. This study was limited to analysis of the centre of force projected onto the longitudinal axis of the foot. Future work should also include analysis of lateral sway, as this will clarify the role of postural correction in increasing midfoot load during gait.

CONCLUSION

This study revealed that patients in the later stages of PD showed an altered roll-off pattern of the foot. Force distribution became more evenly spread over the total foot. In addition, patients presented reduced forward excursions of the centre of force. These findings were gained over and above the influence of speed and sex on foot loading. Hence, flat-footed gait seems to be an expression of the disease in its own right rather than a result of reduced walking velocity. Inadequate heel strike, roll-over and push-off may hamper the foot's rocker functions, limiting the generation of normal step length and gait speed. Therefore, foot placement is an important feature of PD disability which needs to be addressed in rehabilitation research as well as in clinical practice.

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