Article

Kinematic Relations during Double Support Phase in Parkinsonian Gait

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1. Introduction

Parkinson’s disease (PD) is the second most common neurodegenerative disorder, severely affecting the elderly population [1,2]. Pathology of the disease consists of loss of dopaminergic neurons in the brainstem and their projections to basal ganglia deterioration between excitatory and inhibitory neural oscillators [3] that leads to inappropriate extent and timing of movement sequences [4,5]. In addition to the cardinal motor symptoms (rest tremor, muscle rigidity, and bradykinesia), fatigue [1,6], postural instability [1,7], problems in turning [1,8], standing up, or movement initiation [1,9], and/or freezing of gait [1,10]...
are associated with the disease. All these symptoms disrupt the gait cycle [1,6–12]. The ratio of single support time (SST) and double support time (DST) is consistent in a healthy population (4:1) [13,14]; in PD the DST is more lengthy [4,15–20]. The PD gait is shuffling and slow, and the stride is shorter [7,18,20,21], although it sustains its natural width [18]. Contrary to a vastly accepted concept of decreased joint range in limbs based on some published results [17,21], a meta-analysis found no significant decrease in range of motion (ROM) in any joint except the hip joint [18].

A very notable disruption of gait in PD lies in the ankle and foot—the gait malfunction is exhibited as a flatter foot strike, different force distribution between heel and toe, and different timing of motion in the ankle [22,23]. Importantly, the ankle function does not respond well on medication [3,19,24,25].

The side asymmetry of gait parameters often occurs as the PD progresses [1,26], however, it is rarely statistically captured [15,27,28]. Although many particular PD gait aspects have been addressed independently, there are only a few integral kinematic studies [15,23]. The aim of this study is to find out how tightly velocity, SST:DST ratio, and angular parameters in the ankle and shoulder are interrelated and how they communally contribute to the characteristic walking of PD patients during the double support phase (DSP), i.e., initial contact (I) and opposite toe-off (T).

2. Materials and Methods

2.1. Participants

The study was conducted on 11 PD patients, tested on medication (5 females, 6 males, 63.76 ± 10.33 years; Hoehn and Yahr score 2.81 ± 0.68; disease duration 11.36 ± 7.33 years; 171.6 ± 7.6 cm). The control sample (CS) consisted of 11 age-matched volunteers (5 females, 6 males, 66.1 years ± 9.2; 170.5 ± 13 cm).

The PD patients were recruited during their neurological examination by a cooperating neurologist of the 1st Department of Neurology, St. Anne’s University Hospital, Brno. The discrimination for recruitment was PD with L-dopa treatment and a Hoehn and Yahr score of 2–4. The exclusion criteria were brain surgery any time in the past and clinically substantial gait disability.

The CS comprised volunteers recruited from the research volunteers pool of Faculty of Sports Studies, also without substantial gait disability.

The research was approved by the ethics committee of the Faculty of Sports Studies of Masaryk University.

2.2. Recording Method

The participants’ gait was recorded using SIMI Motion optical tracking system (Mo-Cap); the recorded subjects were provided with 15 retro-reflective markers (placed on glabella, left and right acromiale, left and right elbow, left and right wrist, left and right trochanterion, left and right knee, left and right lateral ankle, and left and right toe), which were continuously tracked by an infrared imaging camera network.

The applied protocol incorporated eight infrared imaging cameras covering a cuboid of 8 m × 4 m × 3 m. The optical tracking accuracy of measuring was estimated as 0.25 cm.

The participants were asked to walk barefoot at their comfortable speed. The participants set off at least two steps before they entered the recorded area and they were asked to walk past the area to exclude the walk termination. The recorded area was spacious enough to contain two complete gait cycles. All participants were recorded five times.

2.3. Analysed Gait Parameters

The gait parameters under the research scope (Table 1) were of three modalities: spatial, temporal, and angular parameters (the hip angle: knee-hip-shoulder; the knee angle: hip-knee-ankle; the ankle angle: knee-ankle-tiptoe; the shoulder angle: hip-shoulder-arm; the trunk lean: hip-shoulder-vertical axis). The centre of gravity (CoG) was calculated by the SIMI motion software (using 13-point Gubitz model).
The parameters were analysed at the two points of the gait cycle, initiation (I) defined by heel strike, and termination (T) defined by toe-off. The limb analysed (both upper and lower limb) is seen as same-sided (S) if it shares the side (right or left) with the initiating or terminating lower limb, or opposite-sided (O), if it counts for the opposite side (see Figure 1).

Table 1. Analysed spatial, temporal, and angular data of Parkinson’s disease (PD) and control sample (CS) groups. The spatial variables are in cm, the indexes are dimensionless. The temporal variables are in s, the indexes are in %, the cadence is in n/s. The angular variables are in °. IqR stands for interquartile range.

| Spatial variables | PD (n = 165) | CS (n = 165) | Mann-Whitney U (p) | Cohen d |
|-------------------|-------------|-------------|---------------------|---------|
| Variables         | Median      | IqR (25–75) | Median              | IqR (25–75) |          |         |
| Step length       | 0.448       | 0.139       | 0.576               | 0.083     | 0.0000   | 1.6      |
| Step length index | 0.265       | 0.089       | 0.346               | 0.035     | 0.0000   | 1.9      |
| Step width        | 0.199       | 0.085       | 0.159               | 0.051     | 0.0000   | −0.8     |
| Step width index  | 0.115       | 0.036       | 0.092               | 0.029     | 0.0000   | −0.8     |
| Step-time         | 0.540       | 0.110       | 0.521               | 0.080     | 0.0076   | −0.7     |
| DST               | 1.070       | 0.190       | 1.040               | 0.090     | 0.0023   | −0.7     |
| SST               | 0.165       | 0.080       | 0.130               | 0.040     | 0.0000   | −1.1     |
| DST %             | 0.380       | 0.070       | 0.400               | 0.030     | 0.0078   | 0.0       |
| SST %             | 15.311      | 4.600       | 12.500              | 2.679     | 0.0000   | −1.2     |
| Walking cadence   | 33.905      | 5.270       | 37.383              | 2.597     | 0.0000   | 1.2       |
| n/s               | 1.869       | 0.333       | 1.923               | 0.165     | 0.0237   | 0.6       |
| I CoG v (medio-lateral) | 0.082     | 0.073       | 0.049               | 0.047     | 0.0000   | −0.8     |
| I CoG v (anterio-posterior) | 0.908      | 0.505       | 0.785               | 0.561     | 0.0252   | −0.2      |
| T CoG v (vertical) | 0.100      | 0.087       | 0.058               | 0.066     | 0.0000   | −0.8     |
| T CoG v (abs)     | 0.921       | 0.315       | 0.795               | 0.561     | 0.0139   | −0.3      |
| T CoG v (medio-lateral) | 0.095     | 0.069       | 0.084               | 0.080     | 0.4310   | −0.1      |
| T CoG v (anterio-posterior) | 0.935      | 0.479       | 1.253               | 0.174     | 0.0000   | 1.2       |
| IS hip α          | 155.087     | 8.536       | 152.981             | 4.841     | 0.0013   | −0.3      |
| IO hip α          | 169.160     | 7.345       | 166.818             | 7.299     | 0.0000   | −0.7      |
| TS hip α          | 166.861     | 6.969       | 167.810             | 4.911     | 0.1754   | 0.2       |
| TO hip α          | 0.095       | 0.089       | 0.108               | 0.048     | 0.4144   | 0.1       |
| IS knee α         | 169.251     | 16.130      | 168.687             | 6.730     | 0.8527   | 0.3       |
| IO knee α         | 157.394     | 11.205      | 160.322             | 8.840     | 0.1041   | 0.2       |
| TS knee α         | 126.587     | 10.893      | 130.260             | 8.383     | 0.0031   | 0.3       |
| TO knee α         | 136.027     | 11.571      | 137.821             | 5.700     | 0.1428   | 0.2       |
| IS ankle α        | 96.468      | 15.750      | 97.358              | 5.792     | 0.4608   | 0.0       |
| IO ankle α        | 95.763      | 10.878      | 89.039              | 4.261     | 0.0000   | −1.1      |
| TS ankle α        | 105.093     | 17.782      | 107.625             | 5.864     | 0.0857   | 0.3       |
| TO ankle α        | 102.454     | 7.633       | 100.561             | 4.542     | 0.0037   | −0.3      |
| IS shoulder α     | 15.185      | 6.049       | 24.480              | 7.132     | 0.0000   | 2.2       |
| IO shoulder α     | 16.178      | 10.133      | 21.180              | 11.552    | 0.0000   | 0.4       |
| TS shoulder α     | 18.042      | 10.145      | 18.908              | 7.762     | 0.9094   | 0.0       |
| TO shoulder α     | 12.366      | 6.688       | 22.202              | 4.445     | 0.0000   | 2.1       |
| I Trunk lean      | 1.827       | 2.278       | 4.050               | 3.359     | 0.0000   | −0.7      |
| Trunk rotation    | 4.374       | 5.428       | 4.357               | 4.532     | 0.5723   | −0.1      |
| T Trunk lean      | 2.166       | 1.585       | 3.550               | 3.498     | 0.0001   | 0.5       |
| T Trunk rotation  | 3.819       | 5.268       | 3.402               | 3.571     | 0.0939   | −0.3      |
was used, the median of the variables was first computed for each participant followed by statistical evaluation of the difference between groups using the Mann–Whitney U test, Cohen’s d for effect size, and for target parameters the Spearman correlation was computed.

The confidence interval for all statistics was set at 95%.

2.4. Statistical Evaluation

The spatial parameters (step length and width) were expressed as a percentage of the body height. The values of SST and DST were also approached in form of percentage of a whole gait cycle duration (as SST% and DST%). The Kolmogorov–Smirnov normality test was used, the median of the variables was first computed for each participant followed by statistical evaluation of the difference between groups using the Mann–Whitney U test, Cohen’s d for effect size, and for target parameters the Spearman correlation was computed. All statistically significant differences of all parameters had a big effect size according to Cohen’s d (except for IO shoulder).

3. Results

Neither substantial sex differences nor side asymmetries were captured in the record (for further results see Sitek et al. (2020) [27]); therefore, the strides were merged and analysed together. We were thus counting the statistics with 165 single strides for each group. Analysed data of both groups are presented in Table 1, raw data are available in [29]. The parameters were analysed at the two points of the gait cycle, initiation (I) defined by heel strike, and termination (T) defined by toe-off. The limb analysed (both upper and lower limb) is seen as same-sided (S) if it shares the side (right or left) with the initiating lower limb, or opposite-sided (O), if it counts for the opposite side (see Figure 1).

Figure 1. Limb labels in initiation (I) and termination (T) points of the gait cycle. The circles label the leg defining I (left) and T (right). IS marks a leg that defines initiation phase and the same-side arm. IO marks the limbs of opposite side during I. TS marks a leg that defines termination phase and the same-side arm. TO marks the limbs of opposite side during T.

During I, the forward CoG velocity (Figure 2) was significantly higher in PD patients compared to CS (0.908 m·s⁻¹ vs. 0.785 m·s⁻¹, p < 0.05); however, during T, the CoG forward velocity of PD was lower (0.935 m·s⁻¹ vs. 1.253 m·s⁻¹, p < 0.05). In mediolateral axis, the PD generated higher speed in both I (significant) and T (not significant) compared to CS (respectively, 0.082 m·s⁻¹ vs. 0.049 m·s⁻¹, p < 0.05 and 0.095 m·s⁻¹ vs. 0.084 m·s⁻¹, p > 0.05).

The SST was significantly shorter in PD compared to CS (0.38 s vs. 0.4 s, p < 0.05), as well as the SST% (33.905 vs. 37.383, p < 0.05). The DST was extended significantly in PD (0.165 s vs. 0.13 s, p < 0.05), which was reflected significantly in DST% as well (15.311 vs. 12.5, p < 0.05).

During I, the PD angles in both IS (same-sided) and IO (opposite) hip were significantly more obtuse compared to CS (respectively, 155.087° vs. 152.981°, p < 0.05 and 169.160° vs. 166.818°, p < 0.05). Subsequently, during T, the difference in TS hip angle...
became insignificant, nevertheless, it remained significantly more obtuse in PD in TO hip (160.282° vs. 156.292°, p < 0.05).

Figure 2. Centre of gravity (CoG) velocity development in PD and CS during step initiation (I) and termination (T). Parameters with a statistically significant difference between PD and CS are marked with an asterisk (*).

In the knee joint, there was no significant difference between PD and CS during I phase. During the T phase, however, the PD TS knee adopted a significantly sharper angle compared to the CS knee (126.587° vs. 130.260°, p < 0.05). The TO knee angle showed no difference between the groups.

During I, the angle in IS ankle was very similar in PD and CS. However, in IO, the ankle of the PD patients posed significantly more obtuse angle compared to CS (95.763° vs. 89.039°, p < 0.05). Subsequently, during T, the difference in TS ankle angle became insignificant; nevertheless, it was significantly more obtuse in PD in TO ankle (102.454° vs. 100.561°, p < 0.05).

During I, the PD patients had significantly decreased shoulder angle both on the IS side and on the IO side compared to CS (12.866° vs. 22.2°, p < 0.05 and 16.18° vs. 21.18°, p < 0.05). During T, the PD patients had significantly decreased shoulder angle in TO arm compared to CS (12.866° vs. 22.2°, p < 0.05); in the TS shoulder angle, the PD reach was still decreased, but not statistically significantly. Angle parameters are presented in Figure 3.

Figure 3. Hip (a), knee (b), ankle (c), and shoulder (d) joint α in PD and CS during double support phase (DSP). Leg during gait cycle turns from IS into TO and from IO into TS. Arms fluently change from IS into TO (rear arm) and from IO into TS (front arm). Parameters with a statistically significant difference between PD and CS are marked with an asterisk (*).
The PD group showed significantly less pronounced trunk lean during both I and T phases (respectively, 1.827° vs. 4.050°, \( p < 0.05 \) and 2.166° vs. 3.550° \( p < 0.05 \)). However, the trunk rotation difference was insignificant throughout the DSP.

The parameters most significantly differing between PD and CS were further explored for communal correlations in PD and CS gait models to find out the characteristic relationships between the parameters in both groups (Table 2).

### Table 2. Selected Spearman correlations in PD and CS.

| Variable I            | Variable II          | \( \rho \) (PD) | \( \rho \) (CS) |
|-----------------------|----------------------|----------------|----------------|
| Step width index      | I CoG v (medio-lateral) | 0.6702        | 0.0376         |
| T CoG v (antero-posterior) | I CoG v (antero-posterior) | 0.8957        | 0.4052         |
| TS shoulder \( \alpha \) | TO ankle \( \alpha \) | -0.4718       | 0.4632         |
| TS shoulder \( \alpha \) | IO shoulder \( \alpha \) | 0.3660        | 0.9143         |
| IO shoulder \( \alpha \) | IS shoulder \( \alpha \) | -0.2488       | 0.3795         |
| DST %                 | I CoG v (antero-posterior) | -0.5469       | -0.0337        |
| SST %                 | I CoG v (antero-posterior) | 0.5062        | 0.3120         |
| Walking cadence N/s   | I Trunk lean          | -0.3178       | 0.3595         |
| Step length index     | I CoG v (antero-posterior) | 0.7559        | 0.1081         |

In PD, there was a positive correlation trend found between step width index and CoP velocity in the mediolateral axis (\( \rho = 0.6702 \)). In CS, in contrast, any such correlation was missing (\( \rho = 0.0376 \)). Moreover, there was a substantial correlation found in PD between forward velocity during I and T (\( \rho = 0.8957 \)), whereas in CS the correlation was notably weaker (\( \rho = 0.4052 \)).

During T, in the PD, the shoulder and ankle angle correlated negatively (\( \rho = -0.4718 \)), but in CS they correlated positively (\( \rho = 0.4632 \)). In CS, there was also found a strong correlation in shoulder angle between the I and T phases (\( \rho = 0.9143 \)); however, in PD, the same arm relation was less interconnected (\( \rho = 0.366 \)).

In PD, there was found a weak correlation between DST% and SST% and I CoG v (anterior-posterior), i.e., forward velocity, (\( \rho = -0.5469 \) and 0.5062, respectively). In CS, nevertheless, the same-parameters correlation was none (for DST%: \( \rho = -0.0337 \)) or very weak (for SST%: \( \rho = 0.3120 \)).

In PD, the walking cadence weakly negatively correlated with trunk lean during I, whereas in CS there was a positive weak correlation (\( \rho = -0.3178 \) vs. 0.3595).

Finally, in PD step length index positively correlated with forward velocity during I; in the CS any correlation was missing (\( \rho = 0.7559 \) vs. 0.1081).

### 4. Discussion

It has been shown that the gait of PD patients differs from the gait of healthy controls in many motor aspects [4,15–20,22,23]. Our MoCap-based research supports the findings and provides greater insight into the topic by unveiling relations that have not been fully considered hitherto.

It is often reported that the PD patients attain lower velocity compared to the healthy controls [20,23,30]. Higher gait velocity is associated with higher physical fitness [31] or higher life quality [32]. However, as our results surprisingly indicate, the PD patients are not slower throughout the whole gait cycle. During I, the PD CoG velocity was nearly 20% higher compared to CS. We explain this higher speed as a consequence of the preceding—less balanced—SSP resulting in a less controlled and therefore faster incursion into the I.

Although the PD patients were faster during I phase compared to the CS, their speed was constant, unlike the speed of the CS whose speed was dynamically corresponding to the actual needs of the particular gait phase (i.e., slower during I and faster during...
T, as seen in the Figure 2). Thus, the PD patients were nearly 20% faster at heel strike (0.921 m·s\(^{-2}\) compared to 0.795 m·s\(^{-2}\)); however, their velocity at heel off was about 28% slower compared to the CS (0.945 m·s\(^{-2}\) compared to 1.265 m·s\(^{-2}\)). In such fashion, the CS were periodically adjusting the gait velocity whereas the PD patients sustained rigid or careful speed that was chiselled also in a tight correlation between speed in the I and T phases, which was much fainter in the CS (Table 2).

The possible reasons for the careful gait of PD patients have often been disputed [14,33,34]. Throughout the gait cycle, the CoG forward velocity for overcoming SSP is generated mostly by the pushing foot during T (along with the ongoing body mass inertia) [17]. This push-off is stronger in the CS, probably owing to stronger plantar flexors and better confidence in movement conduction [14,34]. What is more, it is likely that where the CS can boldly predict, release, and utilise the force necessary for unrestrained smooth gait, the PD patients intentionally push-off less forcibly for fear of impeded balance control in the subsequent less stable SSP [34]. The lack of gait speed adaptability can represent an important root of many gait-related problems and could be thus considered by physiotherapists and neurologists.

The extended DST is viewed as a pattern of disrupted gait control (in PD, it extends when in OFF state [17] and shortens when 3D cueing is provided [4]). The extension in the PD patients often breaks physiological ratio of SST:DST (4:1) [4,15–19]; according to a meta-analysis [20], it changes to 3.93:1.08. In our sample, the SST:DST ratio in PD was even more shifted towards DST (3.45:1.55). The PD DST was nearly 27% longer compared to the CS.

Is the DST extension an adaptation or rather a consequence? There is no clear answer among the authors yet. One hypothesis is that the extension of DST is a non-intentional adaptation to fear of falling, providing extra time for regaining balance [14,30,35]. The consequential hypotheses speak of willingly decreased step length and lower velocity for greater gait stability [14,20,31] or of muscle atrophy and/or the PD-inherent disbalance contributing on the gait [14]. Even though our study is in accordance with the factual gait changes, it cannot bring any clear evidence to favour either hypothesis.

The step width in the elderly can indicate fear and predict risk of falls [30]. Although the meta-analysis of Zanardi et al. (2021) [18] informed about low evidence of wider step occurring in PD, in our sample, we detected a significantly broader step of PD patients compared to the CS (similar results were reported by Gazibara et al. 2015 [11]). With the CoG velocity in mediolateral direction correlated the broader step in PD—the velocity was also significantly higher in PD, especially during I. Those faster mediolateral shifts may result from higher forefoot pressure in PD during toe-off (T) reported by Kimmeskamp and Hennig (2001) [34]—caused by faster body vault forward rather than by active push-off. The same study noted a significant shift of centre of pressure medially in PD that may be, along with higher forward and mediolateral velocity, the origin of less balanced (and possibly less controlled) heel strike of PD patients.

In the lower extremities, the difference between PD and CS was most pronounced in the hip joint—the angle was more obtuse in PD, signalling limited ROM of the whole lower extremity. In the knee, the compactness of PD hip joint probably caused the knee to adopt a sharper angle at TO (however insignificantly). Finally, the more obtuse angle in PD ankle was observed (especially in the rear ankle) in connection with lower forward velocity. Similar results of reduced ROM in plantar flexion (19.8%) were captured by Sofuwa et al. [20].

The question is what is the cause and the consequence of these angular changes. In other words, are ankle angles the compensating consequence of the angles at the higher joints (causing an overall lower range of limb movement) or an outcome of weaker or more rigid ankle dorsif and plantar flexor muscles [23] that is barely affected by medication [17,19,23]? In any case, at least one cause-effect relation seems tangible. We assume that the negative correlation in shoulder and ankle angle in PD during T (which is positive in CS) is caused by a disrupted Parkinsonian arm synchronisation with the pace of landing and take-off of the foot.
The most striking example of compact range motion was seen in the PD shoulder joint. Especially, the PD rear arms were over 40% more compact (less extended) in TO and over 35% in IS compared to the CS. The PD shoulder angle was remarkable also due to a fact that there was only a small correlation in angle during I and T (compared to CS), suggesting arm in PD changes position in a quicker and more haphazard manner than in the healthy population.

Interestingly, the PD patients had more upright posture (i.e., less pronounced trunk lean) during the gait compared to the controls and did not conform to the stereotypic bent posture of the PD patient [1,6]. In our opinion, the cause of the limited ROM of the trunk may be axial rigidity, so the patient holds a universal upright position, which gradually develops into a characteristic pathological anteflexion. The second cause, according to our assumption, may be the fear of falling. A forward trunk lean shifts the body’s centre of gravity in this direction, which thus moves at a higher velocity in the anterior-posterior axis, which makes the initiation phase more challenging in terms of balance.

5. Conclusions

The gait of PD patients is slower than that of healthy controls, characteristically longer in DSP and shorter in SSP. Their step is shorter and wider, the push-off feeble and slower. This Parkinsonian gait of our sample was also manifested in a straight back and decreased ROM in joints, especially in the hip, ankle, and shoulder.

Notwithstanding those stereotypes, our study shed light on the disrupted adaptability of the PD gait. To the best of our knowledge, no other study thus far reported PD patients attaining higher gait velocity compared to controls (in any gait cycle segment). The faster PD patients during I could be explained by uncontrolled incursion into the DSP. During T, the PD patients were already slower, as a result of either weaker plantar flexors or careful push-off. Regardless the significant difference in speed during I and T, the speed of PD patients remained constant throughout the gait cycle, whereas the CS speed was continuously changing to reflect needs of the actual gait phase.

The variables specific for PD gait are usually correlated with speed; however, the speed-related correlations proved too weak to shortcut the research by applying a single speed-based motor test, such as timed-up-and-go test [36].

The studied relationships show us the essential links of the Parkinsonian gait but they do not answer which is the cause and which is the consequence in the individual chain of motion. Once it is known which component of motor sequence is more closely associated with the disease and which represents rather an adaptation mechanism, the former one could be used as a more accurate physiotherapy target and what is more, it might even become a biomarker of the disease itself if proven specific enough. Nevertheless, more research of causal relationships still needs to be done.

In conclusion, the surprising detection of higher speed of PD patients during one segment of their gait may draw attention towards the segmentation of gait generally, as there may lay many similar hitherto overlooked details in the current gait-related paradigms.

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