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GAIT VARIABILITY IN PARKINSON'S DISEASE: INFLUENCE OF WALKING SPEED AND DOPAMINERGIC TREATMENT

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Abstract

Objectives—To study the effects of levodopa and walking speed on gait variability in individuals with Parkinson's disease (PD).

Methods—Thirty-three individuals with PD were studied. Their mean age was 70.61 ± 9.23 yr. The average time since diagnosis was 9.65 ± 5.80 yr years. Gait variability was studied while "OFF" and "ON" dopaminergic medication when the subjects walked at their usual and fastest speeds.

Results—Variability of step time, double support time, stride length and stride velocity decreased significantly (p = .037; p = .037; p = .022; p = .043, respectively) after dopaminergic treatment. When subjects increased walking speed, the variability of stride length and stride velocity decreased significantly (p = .038 and p = .004, respectively) both while "OFF" and "ON" levodopa. Increasing walking speed did not change the variability of step time and double support time regardless of medication status.

Conclusions—Levodopa decreased gait variability in persons with PD. Stride length and stride velocity variability appeared to be speed dependent parameters, whereas, the variability of step time and double support time appeared to be speed independent measures. Levodopa had positive effects on gait stability in PD.

Keywords

Parkinson's disease; Gait Variability; Levodopa; Walking speed

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Introduction

Gait disturbance is one of the clinical hallmarks of Parkinson's disease (PD) and a frequent cause of disability and impairment. The PD gait pattern is characterized by reduced speed, short stride lengths, shuffling steps and, occasionally, freezing episodes. [1] Besides these visible clinical features, gait dysfunction in PD includes gait instability and arrhythmicity, as characterized by increased stride-to-stride variability, a fluctuation in the value of a gait measure from one stride to the next. [2] Increased variability reflects inconsistency in stepping patterns and reduced postural control during walking. [3] Gait disturbances and instability may predispose individuals with PD to fall.

Gait instability occurs in elderly persons, as well as in persons with neurological conditions such as in PD and Huntington's disease. [2,3] Higher values of variability indicate greater instability and have been associated with freezing of gait and falls in persons with PD. Dopaminergic treatment has been shown to decreased certain gait variability parameters, demonstrating the role dopaminergic pathways play in the impaired gait rhythmicity in PD. [4]

Although there have been previous reports on gait variability in persons with PD, it is unclear whether gait variability is speed dependent and how it is modified by levodopa. Persons with PD walk with a reduced speed, a potential confounder of the observed changes in variability. [5] Several investigators suggested that stride variability increases if gait speed is lower than an optimal value. [6,7] Conversely, others reported that walking speed and stride variability may be independent.[8,9] No significant increase in stride time variability was observed in healthy elderly subjects even though they walked significantly slower than young adults. Understanding the influences of walking speed and levodopa may further clarify mechanisms underlying gait variability in persons with PD.

The main objective of this study was to demonstrate how levodopa modulated gait variability in individuals with PD when they walk at different speeds. It was hypothesized that levodopa would influence gait variability differently when walking at different speeds.

Materials and methods

Subjects

Thirty-three individuals with idiopathic PD were recruited from movement disorder outpatient clinics in the Houston and Galveston, Texas areas. The severity of PD in all subjects was assessed to be between stage 2 and 3 on the Hoehn and Yahr scale, [10] and subjects were able to stand and walk at least 3 meters without assistance. No subject had visual or hearing deficits that would interfere with the walking test. Subjects were excluded if they had clinically significant musculoskeletal problems in their back, hips, knees or ankles that currently interfered with walking. All subjects reported no history of lower extremity fracture.

All subjects had only medication treatment for PD. None of them had had stereotactic brain surgery or deep brain stimulation. All of them were receiving dopamine treatment alone (carbidopa/levodopa or carbidopa/levodopa/entacapone) or in combination with other PD

medications. The amount of levodopa taken by the subjects ranged from 100 to 400 mg (mean = 162.90 mg). Additional medications included dopamine agonist (pramipexole, requip) for twenty subjects, monoamine oxidase B (MAO-B) inhibitor (rasagiline, selegiline) for six subjects, N-Methyl-D-aspartate (NMDA) antagonist (amantadine) for thirteen subjects, and Catechol-O-methyltransferase (COMT) inhibitor (entacapone) for five subjects.

Subjects were screened for significant cognitive impairment with the Neurobehavioral Cognitive Status Examination (Cognistat) and any subtest score in the severely impaired range resulted in study exclusion (Cognistat). [11] The Unified Parkinson Disease Rating Scale (UPDRS) [12] and the Hoehn and Yahr Staging Scale (HY) were used to assess disease severity of the subjects.

Equipment and Measures

The GAITRite system (GAITRite, CIR Systems Inc., Havertown, PA), is a 5-m, instrumented walkway containing an array of 6 sensor pads encapsulated in a roll-up carpet with an active area 61 cm wide by 366 cm long and a sampling rate of 32.3 – 38.4 Hz. While the subject walks, the system continuously scans the sensors to detect pressures, and transfers the information to the computer for calculating, recording, and storing gait characteristics.

The parameters of interest were gait variability in step time, double support time, stride length and stride velocity. Variability of each parameter (coefficient of variation [CV]) was calculated using the following formula [13]:

$$\%\,\mathrm{CV} = (\text{standard deviation} \div \text{mean}) * 100.$$

Step time is the duration from the contact of one foot to the contact of the opposite foot. Double support time is the sum of the time elapsed between the first contact of the current footfall and the last contact of the previous footfall and the time elapsed between the last contact of the current footfall and the first contact of the next footfall. Stride length is the length of two consecutive footfalls of the same extremity. Stride velocity was calculated as stride length divided by stride time.

Walking protocol

All subjects read and signed an approved consent form prior to participation. Usual speed and fastest walking speed were tested while “ON” and “OFF” dopaminergic medication on the same day. For off medication testing, the subjects were tested in the morning after abstaining from their dopaminergic medication overnight. The wash-out period was at least 12 hours in the “OFF” medication state. Subjects in the “OFF” medication state were assessed with the UPDRS Motor section III by a neurologist prior to performing the walking test. After walking on the computerized mat with the usual and fastest speeds, the subjects took their morning dose of their usual medications and waited for the medications to take effect. Once the subjects reported that they felt “ON” their dopaminergic medication, which was approximately 45 minutes to one hour after taking the medication, the same neurologist

obtained the UPDRS motor scores again. Then, the subjects followed the same walking protocol as they did in the off-medication condition.

Subjects were instructed to walk at their self-selected, usual speed and at their fastest speeds on the computerized mat. Each walk was repeated twice, and the combination of the two trials was used in data analysis. This allowed us to analyze more strides for each subject in each walking condition. All subjects wore a gait belt during the walking experiment and were closely guarded by a research assistant for safety. Verbal instruction for each walk was given before the subject started walking. No instruction was given after the subject started walking in order to prevent any influence of verbal cueing on gait performance. The verbal instruction for the self-selected speed walk was “Walk down the mat at your usual, comfortable walking speed.” The verbal instruction for the fast speed walk was “Walk down the mat as fast as you can.” Subjects were asked to start walking a few steps before entering and after leaving the walkway to allow some distance for acceleration and deceleration.

Statistical analysis

Demographic data was descriptively summarized. All analysis was performed using SPSS version 18.0. Two-way repeated-measures analysis of variance (ANOVA) was performed to demonstrate the main effects of medication status (OFF vs. ON) and walking speed (usual vs. fastest) on four gait variability parameters as well as any interaction (medication status X walking speed). The four dependent measures were step time variability, double support time variability, stride length variability, and stride velocity variability. The independent measures were medication status and walking speed. There were no between-subject factors.

Data collection was performed by the same personnel throughout the study and they were necessarily unblinded to the medication status due to the design of the study. The statistical analysis also was performed in an unblinded condition with respect to medication status. The significance level was set at $P < 0.05$.

Results

Subject characteristics

Subject characteristics are displayed in Table 1. All subjects were receiving dopaminergic treatment (carbidopa/levodopa or carbidopa/levodopa/entacapone). The amount of levodopa taken by the subjects ranged from 100 to 400 mg (mean = 162.90 mg). Dyskinesias were observed in 12 subjects while they were “ON” medication. All subjects were community-dwelling persons with PD. All subjects were able to complete the walking test without any assistive device. However, 8 subjects reported using a cane and 7 subjects reported using either a wheeled or non-wheeled walker for long distance ambulation.

Influence of Levodopa on Gait Variability

There were significant main effects of levodopa on step time variability ($F(1, 32) = 4.74$, $p = .037$), double support time variability ($F(1, 32) = 4.76$, $p = .037$), stride length variability ($F(1, 32) = 5.80$, $p = .022$), and stride velocity variability ($F(1, 32) = 4.44$, $p = .043$) (Table 2 and 3). Levodopa reduced variability in all four of these gait variability parameters.

Influence of Walking Speed on Gait Variability

There was no significant main effect of walking speed on either step time variability (F (1, 32) = 1.023, p = .319) or double support time variability (F (1, 32) = 0.05, p = .833). However, there were significant main effects of walking speed on stride length variability (F (1, 32) = 4.67, p = .038) and stride velocity variability (F (1, 32) = 9.62, p = .004) (Table 2 and 3). These findings indicated that an increase in gait speed reduced variability in stride length and stride velocity but did not affect variability in step time and double support time.

Interaction of Levodopa and Walking Speed on Gait Variability

There was no significant interaction between walking speed and levodopa on the variability of step time (p = .365), double support time (p = .989), stride length (p = .546) or stride velocity (p = .843) (Table 2 and 3). Levodopa did not significantly influence gait variability differently when walking at different speeds (usual vs. fastest). Gait speeds when walking with usual and fastest speeds during “OFF” and “ON” medications are displayed in Table 4.

Discussion

The aim of the study was to assess whether the influence of levodopa on gait variability differs depending on walking speed (usual vs. fastest) in persons with PD. Our results demonstrated several new findings. First, levodopa reduced variability in step time, double support time, stride length, and stride velocity regardless of walking speed. Second, increased walking speed reduced variability in stride length and stride velocity but not in step time and double support time. Third, levodopa did not affect gait variability differently when persons with PD walked at different gait speeds. The results indicated that stride length variability and stride velocity variability were speed dependent, whereas step time variability and double support time variability were speed independent.

Our results are in agreement with a previous study by Schaafsma et al. that the levodopa reduced stride time variability. [4] In our study, all gait variability measures including step time, double support time, stride length and stride velocity were reduced after the administration of the levodopa. Step time and stride time may have similar meanings in terms of variability, a fluctuation across strides as a person walks. Stride time is composed of one right step time plus one left step time. An increase in variability from one stride to the next, regardless of whether the unit of measure is variability in step time, variability in stride length, or variability in stride velocity, reflects an impaired ability to regulate stride-to-stride variations in gait timing in persons with PD. [14]

Almeida et al. evaluated the influence of external timing cues on gait variability and the impact of dopaminergic treatment in persons with PD. [15] In contrast to our results, they found no significant difference between medicated (“ON”) and non-medicated (“OFF”) subjects in step time and double support time variability during self-paced walking speed. Our results showed significant reduction in step time variability and double support time variability after the subjects with PD took their usual medications.

The discrepancy between the two studies could be due to a difference in study design. In the study by Almeida et al., step time variability and double support time variability of a group

of medicated subjects were compared to those of a group of non-medicated subjects. [15] Gait speed, cadence and step length were not different between the two groups during a self-paced walk. We used a repeated-measures design in this study to allow a valid assessment of levodopa effects on gait. The same subjects performed walking tests while "OFF" medication and then took their usual medications before being tested while "ON" medication. The "ON" state was assured by the subjects' self- report and a neurologist.

In addition, in the study by Almeida et al. the subjects with PD walked at different walking speeds, driven by various auditory cueing rates (60, 80, 100 steps/min). The cueing rate of 100 steps per minute was slower than the average self-paced cadence of both the "ON" (106.47 steps/min) and "OFF" (110.35 steps/min) PD groups in their study. [15] In other words, the cueing rates made the subjects walk more slowly than their usual walking speeds whereas in our study, they were asked to walk at their usual and fastest speeds.

Based on our literature search, the study by Almeida et al. is the only one that has investigated the effects of both dopaminergic therapy and walking speed on gait variability in persons with PD. Therefore, with our within-subjects design, we demonstrated, for the first time, that levodopa reduced gait variability in both temporal (step time, double support time, and stride velocity variability) and spatial gait parameters (stride length variability), whereas internal modulation to increase walking speed reduced variability in certain temporal (stride velocity) and spatial (stride length) gait parameters. These findings support the assertion by Morris that increased timing variability in persons with PD may reflect disruption of the normal internal cueing needed to string together sub-movements or a diminished capacity to perform automatic, sequential movements, [16] and which can be improved by anti-PD medications. Our results also are consistent with the view that the basal ganglia may be involved in the neural network for precise modulation of timing of repetitive movement. [17,18] The medications work directly on this neural network to alleviate these deficits, as indicated by our results on variability in the temporal parameters.

Levodopa may improve hypokinesia or bradykinesia and modulate temporal gait variability, whereas, internal modulation or attentional strategies influence only certain gait variability. Our results indicated that levodopa had positive effects on gait variability at the central control level, whereas, an increase in walking speed using an attentional strategy had positive effects on only biomechanical control. These observations have been proposed to be indicative of the role of the basal ganglia in controlling spatial characteristics such as the scaling of amplitude during gait. [19]

The results suggest that fluctuation in step time and double support time depend on some aspect of the central control system that is not merely related to walking velocity. Double support time has been considered an important indicator of abnormal balance control in healthy, older adults and those with cerebellar dysfunction, as well as those with basal ganglia disease. [2] Variability of step time reflects a disturbance of the gait patterning mechanism, whereas variability of double limb support time has been attributed to balance mechanisms. [4,20] A relationship between step time variability and falls has been previously identified. [4] These two temporal measures in variability may be predominantly determined by balance-control mechanisms, whereas stride length and stride velocity were

predominantly determined by gait-patterning mechanisms through neuromuscular/biomechanical control.

Our results are consistent with a study by Rochester and colleagues. Variability of step time and double support time were not changed in people with PD by an increase in walking speed, which was driven by rhythmic auditory cues. [13] Our results also showed that increased walking speed did not reduce variability in step time and double support time. This could suggest that gait speed alone is not the single driver of variability, which is consistent with previous reports that observed a dissociation between stride length and variability. [5,8,9]

Callisaya et al. studied the effect of gait speed on gait variability in elderly persons and reported that a faster gait speed was associated with less variability including step time and double support time. [21] From our results, a faster gait speed did not change step time and double support time in persons with PD. This might indicate that persons with PD and the elderly without PD use different controlling mechanisms to increase gait speed.

Limitations

There are some limitations of the study to be addressed. Our sample consisted of individuals with diagnosed idiopathic PD with mild to moderate severity who reported either gait or balance impairment or falls as a result of PD. Their gait patterns might be different from persons with PD who do not have gait and balance impairments or who never fall.

The design of the study necessitated that we measured gait first when the subjects were “OFF” medication and second while “ON” medication. We measured gait when they had not had levodopa for approximately 12 hours to assure the “OFF-state” testing. We could not have done the testing on the same day if we had measured them while “ON” medication first.

Fatigue might have occurred from travelling to the laboratory in the morning without the medication and having to endure impaired mobility and physical discomfort from PD. However, no subject was too fatigued to perform the walking tests. In addition, the degree of gait improvement by levodopa may vary depending on individual responses.

There were only thirty-three subjects with PD in this study. The sample size was not large enough to study the effects of PD medications other than levodopa taken by the subjects stratified by type of mechanisms (i.e. dopamine receptor agonist, MAO-B inhibitor, anticholinergic medication, COMT inhibitor). However, most PD patients usually receive a combination of PD medications to effectively control their PD symptoms, enhance quality of life and extend survival.[22] A future larger study might include other medications as a factor of interest.

Variability measures of either walking at the usual speed or while “OFF” medication might be more representative of gait variability than either walking at a faster speed or while “ON” medication because more strides were produced and examined. However, Hausdorff et al. reported that gait variability over a small number of strides is statistically similar to

variations that occur over thousands of strides because gait variability takes on a fractal organization in persons. [23] We, therefore, have confidence that our data on variability in each walking condition are valid.

Conclusions

In summary, levodopa reduced gait variability in persons with PD by decreasing variability in step time, double support time, stride length and stride velocity. This may imply that levodopa induced more normal gait patterns in persons with PD. Intentional modulation of speed reduced variability in certain spatial gait parameters including stride length and stride velocity in persons with PD, thus intentional modulation might be a useful strategy in gait rehabilitation aimed at improving stability.

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References

1. Pedersen SW, Oberg B, Larsson LE, et al. Gait analysis, isokinetic muscle strength measurement in patients with Parkinson's disease. Scand J Rehabil Med. 1997; 29(2):67–74. [PubMed: 9198255]
2. Hausdorff JM, Cudkowicz ME, Firtion R, et al. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. Mov Disord. 1998; 13(3):428–437. [PubMed: 9613733]
3. Hollman JH, Kovash FM, Kubik JJ, et al. Age-related differences in spatiotemporal markers of gait stability during dual task walking. Gait Posture. 2007; 26(1):113–119. [PubMed: 16959488]
4. Schaafsma JD, Giladi N, Balash Y, et al. Gait dynamics in Parkinson's disease: relationship to Parkinsonian features, falls and response to levodopa. J Neurol Sci. 2003; 212(1–2):47–53. [PubMed: 12809998]
5. Frenkel-Toledo S, Giladi N, Peretz C, et al. Effect of gait speed on gait rhythmicity in Parkinson's disease: variability of stride time and swing time respond differently. J Neuroeng Rehabil. 2005; 31(2):23.
6. Yamasaki M, Sasaki T, Torii M. Sex difference in the pattern of lower limb movement during treadmill walking. Eur J Appl Physiol Occup Physiol. 1991; 62(2):99–103. [PubMed: 2022210]
7. Danion F, Varraine E, Bonnard M, et al. Stride variability in human gait: the effect of stride frequency and stride length. Gait Posture. 2003; 18(1):69–77. [PubMed: 12855302]
8. Grabiner PC, Biswas ST, Grabiner MD. Age-related changes in spatial and temporal gait variables. Arch Phys Med Rehabil. 2001; 82(1):31–35. [PubMed: 11239283]
9. Hausdorff JM. Stride variability: beyond length and frequency. Gait Posture. 2004; 20(3):304. [PubMed: 15531178]
10. Hoehn M, Yahr MD. Parkinsonism: onset, progression, and mortality. Neurology. 1967; 17:427–442. [PubMed: 6067254]
11. Oehlert ME, Hass SD, Freeman MR, et al. The Neurobehavioral cognitive status examination: Accuracy of the "Screen-Metric" approach in a clinical sample. J Clin Psyc. 1997; 53:733–737.
12. Fahn, S., Elton, RL. Members of the UPDRS Development Committee. Unified Parkinson's Disease Rating Scale. In: Fahn, S.Marsden, CD.Calne, DB., Goldstein, M., editors. Recent developments in Parkinson's disease. Vol. 2. Florham Park: Macmillan Health Care Information; 1987.

13. Rochester L, Burn DJ, Woods G, et al. Does auditory rhythmical cueing improve gait in people with Parkinson's disease and cognitive impairment? A feasibility study. Mov Disord. 2009; 24(6): 839–845. [PubMed: 19199354]
14. Hollman JH, Salamon KB, Priest AW. Aged-related differences in stride-to-stride variability during dual task walking: A pilot study. J Geriatric Phys Ther. 2004; 27(3):83–87.
15. Almeida QJ, Frank JS, Roy EA, et al. Dopaminergic modulation of timing control and variability in the gait of Parkinson's disease. Mov Disord. 2007; 22(12):1735–1742. [PubMed: 17557356]
16. Morris ME, Iansek R, Matyas TA, et al. The pathogenesis of gait hypokinesia in Parkinson's disease. Brain. 1994; 117(Pt 5):1169–1181. [PubMed: 7953597]
17. Rao SM, Harrington DL, Haaland KY, et al. Distributed neural systems underlying the timing of movements. J Neurosci. 1997; 17(14):5528–5535. [PubMed: 9204934]
18. Harrington DL, Haaland KY, Knight RT. Cortical networks underlying mechanisms of time perception. J Neurosci. 1998; 18(3):1085–1095. [PubMed: 9437028]
19. Morris ME, Iansek R, Matyas TA, et al. Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. Brain. 1996; 119(Pt 2):551–568. [PubMed: 8800948]
20. Yogev G, Giladi N, Peretz C, et al. Dual tasking, gait rhythmicity, and Parkinson's disease: which aspects of gait are attention demanding? Eur J Neurosci. 2005; 22(5):1248–1256. [PubMed: 16176368]
21. Callisaya ML, Blizzard L, Schmidt MD, et al. Ageing and gait variability--a population-based study of older people. Age Ageing. 2010; 39(2):191–197. [PubMed: 20083617]
22. Rezak M. Current pharmacotherapeutic treatment options in Parkinson's disease. Dis Mon. 2007 Apr; 53(4):214–22. [PubMed: 17586328]
23. Hausdorff JM, Purdon PL, Peng CK, et al. Fractal dynamics of human gait: stability of long-range correlations in stride interval fluctuations. J Appl Physiol. 1996; 80(5):1448–1457. [PubMed: 8727526]

Table 1

Subject Characteristics

	PD (N=33)
Gender	Male 22; Female 11
Age	70.61 ± 9.23
Height (cm)	168.76 ± 11.37
Weight (kg)	75.70 ± 15.98
BMI (kg/m^2)	26.62 ± 5.30
Year of PD	9.65 ± 5.80
HY Stage (“ON”)	2.58 ± 0.42
UPDRS “OFF”	29.12 ± 11.36
UPDRS “ON”	18.39 ± 8.55

Table 2

Effects of Walking Speed (usual vs. fastest) and Levodopa Treatment (OFF vs. ON) on the gait variability.

Variability Characteristics	OFF Medication (N=33)	ON Medication (N=33)	P-Value [a] (OFF vs. ON)	P-Value [a] (Usual vs. Fast)
Usual Walking Speed				
Step Time (%)	5.38 ± 3.25	4.32 ± 1.61	.037	.319
Double Support Time (%)	7.19 ± 2.88	5.96 ± 2.36	.037	.833
Stride Length (%)	5.24 ± 4.52	4.01 ± 2.54	.022	.038
Stride Velocity (%)	5.52 ± 3.27	4.38 ± 2.00	.043	.004
Fastest Walking Speed				
Step Time (%)	4.83 ± 2.01	4.23 ± 2.14		
Double Support Time (%)	7.10 ± 4.02	5.86 ± 3.31		
Stride Length (%)	4.49 ± 4.31	2.92 ± 1.54		
Stride Velocity (%)	4.48 ± 4.12	3.43 ± 1.82		

[a]Two-way repeated measures ANOVA; Based on estimated marginal means.

Table 3

Effects of the Walking Speed and Levodopa on Gait Variability

Variable	Sum of Squares	df	Mean Square	F	Sig.
Medication					
Step time variability	22.709	1	22.709	4.738	.037
Double support time variability	50.320	1	50.320	4.762	.037
Stride length variability	64.820	1	64.820	5.803	.022
Stride velocity variability	39.546	1	39.546	4.440	.043
Error					
Step time variability	153.369	32	4.793		
Double support time variability	338.149	32	10.567		
Stride length variability	357.461	32	11.171		
Stride velocity variability	285.001	32	8.906		
Walking Speed					
Step time variability	3.421	1	3.421	1.023	.319
Double support time variability	.320	1	.320	.045	.833
Stride length variability	27.729	1	27.729	4.670	.038
Stride velocity variability	32.750	1	32.750	9.622	.004
Error					
Step time variability	106.970	32	3.343		
Double support time variability	227.649	32	7.114		
Stride length variability	189.990	32	5.937		
Stride velocity variability	108.921	32	3.404		
Walking Speed X Medication					
Step time variability	1.762	1	1.762	.844	.365
Double support time variability	.002	1	.002	.000	.989
Stride length variability	1.002	1	1.002	.372	.546
Stride velocity variability	.080	1	.080	.040	.843
Error					
Step time variability	66.816	32	2.088		
Double support time variability	299.217	32	9.351		

Variable	Sum of Squares	df	Mean Square	F	Sig.
Stride length variability	86.092	32	2.69		
Stride velocity variability	64.467	32	2.015		

Table 4

Comparison of Gait Speed of usual and fast walks during “ON” and “OFF” medication states

Gait Speed	PD-OFF(N=33)	PD-ON(N=33)	P-Value [a](Usual vs. Fast)	P-Value [a](ON vs. OFF)
Usual Speed (cm/s)	82.70 ± 26.59	98.94 ± 19.92	<.001*	<.001*
Fastest Speed (cm/s)	123.91 ± 35.62	135.76 ± 27.59		

[a]Two-way repeated-measures ANOVA.