

## **GAIT PARAMETERS OF INDIVIDUALS WITH PARKINSON DISEASE DECLINE DURING ONE-YEAR PERIOD**

KADRI MEDIJAINEN<sup>1</sup>, MATI PÄÄSUKU<sup>1</sup>, AET LUKMANN<sup>2</sup>, PILLE TABA<sup>3</sup>

<sup>1</sup>*Institute of Sport Sciences and Physiotherapy, University of Tartu, Estonia*

<sup>2</sup>*Department of Sports Medicine and Rehabilitation, University of Tartu, Estonia*

<sup>3</sup>*Department of Neurology and Neurosurgery, University of Tartu, Estonia*

### **ABSTRACT**

Parkinson's disease (PD) is a neurodegenerative disease, influencing mainly elderly. The key motor factor affecting the level of participation in activities of daily living is the gait function, which is known to be progressively impaired in PD. However, gait characteristics also worsen due to normal aging. The main aim of this study was to investigate whether gait parameters decline in individuals with PD in an interval of one year compared to healthy elderly. Selected gait characteristics were recorded using 3-D optoelectronic movement analysis system ELITE in 13 patients with mild-to-moderate PD and 13 age- and gender-matched controls. Hoehn and Yahr Scale and Unified Parkinson Disease Rating Scale were used for clinical assessment. It was found that PD patients walk with significantly shorter steps and stride and reduced gait speed. In one year, the stride length initiated with right foot and stride walk ratio further decrease in PD patients. On re-evaluation the percentages of stance, swing and double support phase differed significantly between groups. In second measurement, control subjects walked with reduced step width. It was concluded that gait speed and stride length decline in patients with PD in a period of one year, whereas no indication of deterioration of gait function is evident in healthy controls.

**Keywords:** *Parkinson disease, gait, deterioration of function, progression*

## INTRODUCTION

Following Alzheimer's disease, PD is the second most frequent neurodegenerative disease [16]. Generally, PD affects elderly – usual onset is between 50–70 years. Prevalence increases linearly until 80 years of age. About 2–3% of over 65-year olds are affected by this disorder [20].

Clinical features of PD result from degeneration of extrapyramidal system [18]. The pathological findings show greatly diminished neurons in substantia nigra, resulting in several neurochemical changes that cause the disease-specific features of PD: rest tremor, rigidity and bradykinesia [24]. Symptoms of PD are mainly described through motor features, but various non-motor features are also typically seen.

These impairments affect the overall motor performance and thus activities of daily living in patients with PD. Gait disturbances are one of the most disabling symptoms of PD and they contribute to person's performance in activities of daily living and through this reduce the quality of life [10]. In PD, spatiotemporal parameters of gait deteriorate significantly when the dose of the medication decreases, but also at peak dose patients walk with shorter steps and slower walking speed than control subjects [129]. Latter indicates that mobility deficits are difficult to treat with merely antiparkinsonian drugs. Therefore, referral to physiotherapy in this contingent of patients is recommended highly from very beginning of the disease. The role of physiotherapy is to teach patients with PD compensatory strategies for coping with their impairments, to improve their functional performance and avoid secondary complications due to inactivity and thereby prolonging the period of independency for the patient and preserve the quality of life for maximally long period [8].

Physiotherapists also play a role in assessing the ability of people with PD to accomplish complex tasks and assess changes in function, disability, activity, and response to therapy, as well to monitor the natural progression of the disease [2]. The analysis of annual change in spatiotemporal gait parameters could provide physiotherapists with objective data. The main aim of this study was to investigate whether gait parameters decline in individuals with mild-to-moderate PD present in an interval of one year different from healthy age- and gender-matched counterparts. Although our study included laboratory assessment of selected gait parameters, the spatiotemporal gait analysis was chosen because it is possible to perform the assessment similarly in clinical setting and in home environment.

## MATERIALS AND METHODS

### Subjects

Participants with PD were recruited from the Department of Neurology, Tartu University Hospital. All of them were living in community. Patients aged under 80, and with disease severity according to modified Hoehn and Yahr (HY) currently in stages 2.0–3.0, not diagnosed with other neurological conditions and not presenting acute medical problems and conditions affecting mobility were contacted at their scheduled neurologist appointment. Absence of severe dyskinesia and long “off”-periods was also an inclusion criterion, as well as not requiring an assistive device for indoor mobility. A convenience sample of 13 patients was enrolled and it included six female and seven male participants with idiopathic PD. Age- and gender-matched healthy control group was recruited with the help of one local family doctor confirming them to be free of any possible limiting health conditions.

The study was approved by the Ethics Committee of University of Tartu and all participants signed an informed consent declaration. General clinical and demographic characteristics of the participants are presented in Table 1.

**Table 1.** Comparison of clinical and demographic characteristics of the participants at baseline

Variable	Participants with PD (n=13)	Controlgroup (n=13)	p value
Age (years)	65.2±6.7	64.9 ±7.0	0.90
Disease duration (years)	8.7±5.5	NA	–
HY stage	2.3±0.5	NA	–
UPDRS total score	62.4±19.6	NA	–
Height (m)	1.7±0.1	1.7±0.1	0.83
Weight (kg)	71.3±16.9	73.9±13.3	0.51

Note: PD – Parkinson disease; NA – not applicable; HY – Hoehn &Yahr Scale; UPDRS – Unified Parkinson Disease Rating Scale.

### Procedures

Objective testing was conducted in the Laboratory of Kinesiology and Biomechanics of Tartu University and clinical examination of the patients occurred within few hours in the Tartu University Hospital. Both patients and controls were tested twice – at baseline and approximately 1 year later. All the participants had an adequate comprehension of instructions.

Participants with PD were medicated with ordinary anti-parkinsonian drugs and were tested in a self-diagnosed “on-phase” of medication. Current stage of PD was recorded by qualified neurologist using HY and Unified Parkinson’s Disease Rating Scale (UPDRS). The UPDRS was designed to follow the longitudinal course of the disease and has been shown to be both reliable and valid [1]. It has four parts (subscales), comprising symptoms of mentation, behaviour, and mood in part I, activities of daily living in part II, motor symptoms in part III, and complications of therapy in part IV. Each item in part I to III is quantitatively scored on a 5-point scale (from 0 to 4) [2]. The result of the UPDRS can be interpreted as higher the result of the UPDRS, the more expressed is the Parkinson’s disease of the patient. During the assessment, only patient and neurologist were in the room.

The assessment for anthropometrical parameters and was conducted in same manner for both groups. First, the anthropometrical parameters were registered (participants wearing light well-fitting clothing). Thereafter, for gait analysis 20 reflective markers were fixed on anatomical landmarks of the subject, according to the Davies protocol. Six infrared cameras and a 3-D optoelectronic movement analysis system ELITE (BTS Bioengineering, Milan, Italy) registered the displacement of 20 reflective markers fixed on anatomical landmarks of the subject. The instructions given to the subjects were following: “Walk with your normal walking speed to the end of the walkway.” Practice trial preceded data collection. For data analysis the performance of barefoot gait on 6-meter-walkway (Kistler, Switzerland) at self-selected speed was recorded and average of 3 trials was used for data analysis.

Selected gait characteristics obtained with the 3-D gait analysis were used for data analysis. The parameters (measurement units presented in parenthesis) were following: step length (mm); stride length (mm); cadence (steps per min); mean velocity of gait (m/s); step width (mm); stance and swing phase percentage of the gait cycle (%); double support phase percentage of the gait cycle (%). Step and stride length of both lower limbs are presented, for other parameters only the average of left and right side is presented.

Ducharme et al. [5] suggested that walk ratio (step length divided to cadence) could be used to discriminate between healthy and disabled individuals. In Kirtlet’s [9] overview of the temporal-spatial parameters, the walk ratio is mentioned and is calculated as stride length divided by cadence. In present study we calculated walk ratio using both methods, naming them step walk ratio and stride walk ratio, respectively (using average step and stride length).

## **Statistical Analysis**

Analysis was performed using SPSS 20.0. Descriptive analysis was performed. The Wilcoxon Signed Ranks test or Mann–Whitney U-test were used to compare groups. The effect size was calculated based on the coefficient of the product-moment correlation ( $r$ ) [26]. The coefficient of the product-moment correlation was chosen to allow comparison between parametric and non-parametric data. A level of significance  $p < 0.05$  was selected to indicate statistical significance.

## **RESULTS**

Results are summarized in Table 2. Patients with PD walked with significantly ( $p < 0.05$ ) shorter steps and strides and reduced gait speed compared to controls at both measurements. The step walk ratio and stride walk ratio and the percentage of stance, swing and double support phase was also different between groups during re-evaluation one year later.

When looking at within-group differences it can be concluded that gait parameters did not change markedly during one year at either group. In PD group, merely stride walk ratio and stride length initiated with right foot deteriorated at significant extent. Control group demonstrated reduced step width during re-evaluation.

When looking at clinical assessment results of PD presentation, a significant change was evident merely in the UPDRS active daily living score ( $p = 0.01$ ), whereas the UPDRS motor score (nearly significant,  $p = 0.06$ ) and the UPDRS total score nor HY did not indicate more severe disease presentation.

Table 2. Gait parameters at baseline and one year later along with mean change values and effect size among the PD and CG

Variable	Group	Before mean $\pm$ SD	After mean $\pm$ SD	Before vs after		Mean change	
				Effect size	p value	Lower bound CI	Upper bound CI
Dex step length (mm)	PD	555.9 $\pm$ 28.6 <sup>#</sup>	543.5 $\pm$ 27.8 <sup>#</sup>	-0.14	0.46	-12.3	26.0
	CG	638.8 $\pm$ 13.7	626.8 $\pm$ 12.6	-0.08	0.70	-12.0	21.8
Sin step length (mm)	PD	569.0 $\pm$ 27.2 <sup>#</sup>	551.6 $\pm$ 40.2 <sup>#</sup>	-0.30	0.12	-17.4	26.0
	CG	653.4 $\pm$ 20.7	650.0 $\pm$ 11.9	-0.02	0.92	-3.4	47.9
Dex stride length (mm)	PD	1126.3 $\pm$ 55.2 <sup>#</sup>	1063.4 $\pm$ 62.4 <sup>#</sup>	-0.39	0.05*	-62.9	-8.9
	CG	1295.0 $\pm$ 26.6	1297.5 $\pm$ 19.0	-0.11	0.58	2.6	67.3
Sin stride length (mm)	PD	1128.7 $\pm$ 55.0 <sup>#</sup>	1098.7 $\pm$ 75.9 <sup>#</sup>	-0.29	0.14	-30.0	52.9
	CG	1306.3 $\pm$ 39.3	1302.9 $\pm$ 21.8	-0.03	0.88	-3.5	93.0
Cadence (steps/min)	PD	108.2 $\pm$ 3.1	113.7 $\pm$ 3.1	-0.26	0.18	5.5	13.6
	CG	109.1 $\pm$ 3.5	109.5 $\pm$ 4.8	-0.03	0.89	0.5	7.5
Mean velocity of gait (m/s)	PD	1.0 $\pm$ 0.1 <sup>#</sup>	1.0 $\pm$ 0.1 <sup>#</sup>	-0.08	0.70	-0.0	0.1
	CG	1.2 $\pm$ 0.0	1.2 $\pm$ 0.0	-0.04	0.83	-0.1	0.1
Step width (mm)	PD	106.2 $\pm$ 8.7	102.3 $\pm$ 8.4	-0.12	0.56	-3.9	9.6
	CG	107.8 $\pm$ 4.7	96.3 $\pm$ 5.0	-0.38	0.05*	-11.5	-0.0

Variable	Group	Before vs after			Mean change			
		Before mean ±SD	After mean ±SD	Effect size	p value	Mean change	Lower bound CI	Upper bound CI
Stance phase (%)	PD	62.4±0.9	63.8±0.8 <sup>#</sup>	0.19	0.35	1.4	-1.4	4.1
	CG	(62.3±0.7	61.7±0.5	-0.08	0.67	-0.6	-2.6	1.6
Swing phase (%)	PD	37.6±0.9	36.3±0.8 <sup>#</sup>	-0.17	0.37	-1.3	-4.1	1.4
	CG	37.7±0.7	38.3±0.5	-0.08	0.67	0.6	-1.6	2.8
Double support phase (%)	PD	12.4±1.0	13.6±0.8 <sup>#</sup>	-0.16	0.42	1.2	-1.8	4.1
	CG	12.1±0.7	11.6±0.6	-0.08	0.70	-0.5	-3.0	1.9
Step walk ratio	PD	5.2±0.3	4.9±0.3 <sup>#</sup>	-0.42	0.03	-0.0	-0.2	0.1
	CG	6.0±0.3	6.0±0.3	-0.06	0.75	-0.1	-0.6	0.4
Stride walk ratio	PD	10.5±0.6	9.6±0.6 <sup>#</sup>	-0.47	0.02*	-0.9	-1.6	-0.2
	CG	12.2±0.7	12.2±0.6	-0.03	0.86	0	-1.1	1.1

Note: PD – group with participants with Parkinson disease; CG – control group; dex – right lower limb; sin – left lower limb; \* significant difference between baseline and re-evaluation; <sup>#</sup>significant difference between PD and CG; p≤0.05.

## DISCUSSION

Disturbances in spatiotemporal parameters of gait are well documented in patients with PD [3, 7, 12, 19, 21]. Though there are numerous data that also gait kinematics and kinetics are altered by PD [10, 25], it was not of interest to analyse these parameters in the present study. Present study aimed to illustrate the differences in decline of selected spatiotemporal parameters of gait in patients with PD and age- and gender-matched controls in an interval of one year that has not been described in the follow-up before.

In the present study, consistent with previous findings [10, 14, 25], PD patients demonstrated a gait pattern with shorter step (and stride) and smaller walking velocity at baseline compared to control subjects.

PD patients demonstrated shorter stride length (by ~13%) and reduced mean velocity than healthy controls. Other authors have reported more pronounced differences between controls and PD patients – for example Lewis et al. [10] found 24% reduction in gait velocity and 23% reduction in stride length. This is probably due more severe disease (2.8 at HY scale for the study of Lewis et al. compared to 2.6 for the present study) and longer disease duration ( $9.1 \pm 5.7$  vs  $8.2 \pm 3.9$  years). Another aspect might be related to the control group – though we were assured by the family doctor that helped with recruiting the control group that the subjects were currently healthy and free of possible limitations, they still had some health restrictions, possibly somewhat influencing the result. The step walk ratio in the present study was 6.0 mm/(step/min) – this result stays below walk ratio considered to be constant for healthy adults (6.5 mm/(step/min) [19].

However, as suggested by Ducharme et al. [5], participants with PD and healthy counterparts could be distinguished by walk ratio during re-evaluation: both step and stride walk ratio differed between groups. However, stride walk ratio was also one of the parameters that demonstrated distinct deterioration within-patient group, indicating it to be possible a slightly more sensitive parameter than step walk ratio to demonstrate decline in gait function of individuals with PD. Smaller walk ratio has been found to be associated with falling in community-dwelling elderly [15], so it seems highly favourable to use this parameter for gait assessment also in clinical setting.

In agreement with previous findings [12, 14], the cadence was similar in both measurements in PD patients and the controls. Some studies [17, 19] have suggested that patients with PD have higher cadence to compensate the reduced stride length. Nieuwboer et al. [17] reported an exponential increase of cadence with a decreasing stride length during freezing. The cadence also increased in the present study for one year, but the change was not significant.

Reduced stride length is considered the most characteristic feature of parkinsonian gait. Often it is accompanied with reduced walking speed and tendency toward longer double-support phase duration [14]. It has been suggested, that the increased percentage of double support phase in PD is directly related with levodopa concentration in organism – the lower concentration relates to the longer double support phase [17]. It is possible that because of experiencing postural instability, patients compensate this by increasing the time in which both feet are in contact with the ground [13]. Differences in double support phase in present study reached significant level by the re-evaluation.

Canning et al. [3] showed that when PD patients are able to walk at velocities comparable to healthy controls, they do not sustain this velocity over longer distances. It is possible that this is the reason for relatively unchanged gait speed in present study.

Studies have shown that the gait characteristics (speed, stride length) of PD patients can be improved significantly by the use of appropriate influences [25], for example external stimuli and didactic methods. It has been found that by simply asking PD patient to walk with longer steps, they can significantly increase the walking speed and amplitude within normal values, which can last for up to 2 hours [17]. Patients with PD typically rely on external cues for locomotion. On the walkway used in present study, there were two small dots of blue coloured stripe, and the walkway ended with a perpendicular yellow stripe. These were there for the purposes of other, routinely performed measurements in this laboratory. It is known that PD patients are able to improve their ambulation when auditory, tactile, cognitive or visual cues are given [22]. It may be that the coloured dots and stripe and the apparatus of the laboratory might have served as a visual stimulus for the patients and to improve their gait parameters at some extent.

Charlett et al. [4] showed that step width increases early in the disease, to compensate for altered balance and posture, and it narrows later in the disease. In the present study, step width of PD participants did not change, however, it decreased during one year in the control group – probably because they were more familiar and comfortable with the analysis procedure.

After one year, a significantly different stride length initiated with the right foot was observed in PD patients. This is probably explainable by higher gait variability (especially stride-to-stride variability) characteristic to PD gait according to [7].

The main limitation of our study was relatively small study sample. However, we believe interpretation of the study results has been conservative

and the conclusion we have made rely on reliable data analysis. Further, behavioural aspects such as fear of falling also contribute to gait parameters. Comorbid depression, very common in PD [11] causes slowing the walking speed [21]. Unfortunately, these aspects were not covered thoroughly in present study, as well as presence of freezing of gait etc. In addition, it may be argued that subjects should have been assessed a certain time after taking their antiparkinsonian medication. Mainly for logistical reasons, the patients were tested in self-diagnosed “on-phase” of medication. Most studies have used assessment in both off-phase and on-phase.

In the clinical characteristic’s significant differences between baseline and one year later were seen merely in UPDRS active daily living score, although total score also indicated nearly significant deterioration ( $p=0.06$ ). This supports the findings from Frysinger et al. [6], who indicated that active daily living score serves as a better marker of disease progression than other sections of the UPDRS.

In conclusion, despite some limitations, present study demonstrated the different rates of decline of gait parameters during one-year period in patients with PD and healthy controls with sufficient credibility. In accordance with previous studies, patients with mild-to-moderate PD walk with significantly shorter step and stride length and reduced mean velocity of the gait, compared with age- and gender-matched control subjects. Following one-year period, stride length initiated with right foot and stride walk ratio significantly deteriorate in patients’ group.

## ACKNOWLEDGMENTS

This study was supported by Grant 3.2.1001.11-0017 of the European Regional Development Fund and the Grant IUT2-4 of the Estonian Research Council. The authors declare that there is no conflict of interest regarding the publication of this paper.

## REFERENCES

1. Alves G. (2006) Clinical Disease Progression in Parkinson’s Disease. PhD thesis of University of Bergen.
2. Brusse. KJ, Zimdars S, Zalewski KR, Steffen TM. (2005) Testing functional performance in people with Parkinson disease. *Phys Ther*, 85: 134–141.
3. Canning CG, Ada L, Johnson JJ, McWhirter S. (2006) Walking capacity in mild to moderate Parkinson’s disease. *Arch Phys Med Rehabil*, 87: 371–375.  
<https://doi.org/10.1016/j.apmr.2005.11.021>

4. Charlett A, Weller C, Purkiss AG, Dobbs SM, Dobbs RJ. (1998) Breadth of base whilst walking: effect of ageing and parkinsonism. *Age Ageing*, 27: 49–54. <https://doi.org/10.1093/ageing/27.1.49>
5. Ducharme SW, Sands CJ, Moore CC, Aguiar EJ, Hamill J. (2018) Changes to gait speed and walk ratio with rhythmic auditory cuing. *Gait & Posture*, 66: 255–259. <https://doi.org/10.1016/j.gaitpost.2018.09.006>
6. Frysinger RC, Harrison MB, Huss D, Wooten GF, Curri LJ. (2004) UPDRS activity of daily living score as marker of Parkinson's disease progression. *Mov Disord*, 19 (Suppl 9): S268.
7. Hausdorff JM. (2009) Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. *Chaos*, 19: 026113. <https://doi.org/10.1063/1.3147408>
8. Keus SHJ, Munneke M, Graziano M, Paltamaa J, Pelosin E, Domingos J et al. (2014) European Physiotherapy Guideline for Parkinson's disease. KNGF/ParkinsonNet, the Netherlands.
9. Kirtlet C. (2006) *Clinical Gait Analysis. Theory and practice*. Elsevier Churchill Livingstone, 5–37.
10. Lewis GN, Byblow WD, Walt SE. (2000) Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues. *Brain*, 123: 2077–2090. <https://doi.org/10.1093/brain/123.10.2077>
11. Marsh L. (2013) Depression and Parkinson's Disease: Current Knowledge. *Curr Neurol Neurosch Rep*, 13: 409. <https://doi.org/10.1007/s11910-013-0409-5>
12. Morris ME, Matyas TA, Ianse R, Cummers JJ. (1996a) Temporal stability of gait in Parkinson's disease. *Phys Ther*, 76: 763–777. <https://doi.org/10.1093/ptj/76.7.763>
13. Morris ME, Ianse R, Matyas TA, Summers JJ. (1996b) Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. *Brain*, 119: 551–568. <https://doi.org/10.1093/brain/119.2.551>
14. Morris M, Ianse R, McGinley J, Matyas T, Huxham F. (2005) Three-dimensional gait biomechanics in Parkinson's Disease: evidence for centrally mediated amplitude regulation disorder. *Mov Disord*, 20: 40–50. <https://doi.org/10.1002/mds.20278>
15. Nakakubo S, Doi T, Makizako H, Tsutsumimoto K, Hotta R, Kurita S, Kim M, Suzuki T, Shimada H (2018) Association of walk ratio during normal gait speed and fall in community-dwelling elderly people. *Gait Posture*, 66: 151–154. <https://doi.org/10.1016/j.gaitpost.2018.08.030>
16. Nicita-Mauro V, Basile G, Mento A, Epifanio A, Martino G, Morgante L. (2002) Parkinson's disease, parkinsonism and aging. *Arch Gerontol Geriatr*, Suppl 35: 225–238. [https://doi.org/10.1016/S0167-4943\(02\)00138-3](https://doi.org/10.1016/S0167-4943(02)00138-3)
17. Nieuwboer A, Dom r, De Weerd W, Desloovere K, Fieuws S, Broens-Kaucsik E. (2001) Abnormalities of the spatiotemporal characteristics of gait at the onset of freezing in Parkinson's disease. *Mov Disord*, 16: 1066–1075. <https://doi.org/10.1002/mds.1206>

18. Obeso JA, Rodríguez-Oroz MC, Rodríguez M, Arbizu J, Giménez-Amaya JM. (2002) The basal ganglia and disorders of movement: pathophysiological mechanisms. *News Physiol Sci*, 17: 51–55. <https://doi.org/10.1152/nips.01363.2001>
19. Pistacci M, Gioulis M, Sanson F, De Giovannini E, Filippi G, Rosetto F, Marsala SZ. (2017) Gait analysis and clinical correlations in early Parkinson's disease. *Funct Neurol*, 32: 28–34. <https://doi.org/10.11138/FNeur/2017.32.1.028>
20. Poewe W, Seppi K, Tanner CM, Halliday GM, Brundin P, Volkman J, Schrag AE, Lang AE. (2017) Parkinson disease. *Nat Rev Dis Primers*, 23: 17013. <https://doi.org/10.1038/nrdp.2017.13>
21. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, Van Wegen E. (2004) Attending to the task: interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue, and balance. *Arch Phys Med*, 85: 1578–1585. <https://doi.org/10.1016/j.apmr.2004.01.025>
22. Rochester L, Baker K, Hetherington V, Jones D, Willems AM, Kwakkel G, Van Wegen E, Lim I, Nieuwboer A. (2010) Evidence for motor learning in Parkinson's disease: Acquisition, automaticity and retention of cued gait performance after training with external rhythmical cues. *Brain Res*, 1319: 103–111. <https://doi.org/10.1016/j.brainres.2010.01.001>
23. Rota V, Perucca L, Simone A, Tesio L. (2011) Walk ratio (step length/cadence) as a summary index of neuromotor control of gait: application to multiple sclerosis. *Int J Rehabil Res*, 34: 265–269. <https://doi.org/10.1097/MRR.0b013e328347be02>
24. Samii A, Nutt JG, Ransom BR. (2004) Parkinson's disease. *Lancet*, 363: 1783–1793. [https://doi.org/10.1016/S0140-6736\(04\)16305-8](https://doi.org/10.1016/S0140-6736(04)16305-8)
25. Sofuwa O, Nieuwboer A, Desloovere K, Willems AM, Chavret F, Jonkers I. (2005) Quantitative gait analysis in Parkinson's disease: comparison with healthy control group. *Arch Phys Med Rehabil*, 86: 1007–1013. <https://doi.org/10.1016/j.apmr.2004.08.012>
26. Tellez A, Garcia CH, Corral-Verdugo V. (2015) Effect size, confidence intervals and statistical power in psychological research. *Psychology in Russia: State of the Art*, 8: 27–47. <https://doi.org/10.11621/pir.2015.0303>

**Correspondence to:**

Kadri Medijainen  
Institute of Sport Science and Physiotherapy  
Faculty of Medicine  
University of Tartu  
Ujula 4, 51008 Tartu  
E-mail: kadri.medijainen@ut.ee  
Phone: (+372) 55 49 719