biomechanics

Communication

Similar Postural Response Improvements Following a Single Session of Transcranial Direct Current Stimulation in Fallers and Non-Fallers with Parkinson's Disease: A Pilot Study

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Abstract

Background/Objectives: People with Parkinson's disease (PwPD) exhibit impairments in postural responses to perturbations, increasing their risk of falls. While transcranial direct current stimulation (tDCS) has been shown to enhance postural responses in PwPD, its effects considering history of falls remain unclear. Thus, we aimed to analyse the effect of tDCS on postural responses after external perturbation in PwPD with and without a history of falls. **Methods:** Twenty-two PwPD were distributed into two groups—faller (n = 12) and non-faller (n = 10)—based on their history of falls over the 12 months preceding the experiment. A 20 min anodal tDCS was applied to the primary motor cortex (M1) under two conditions (2 mA and sham), performed on two different visits (at least 2 weeks apart) with a randomised order. Seven trials with temporally unpredictable external perturbation (i.e., backward translation of the support base) were performed after tDCS. Electromyographic (i.e., medial gastrocnemius (MG) onset latency, magnitude of muscle activation of MG and tibialis anterior (TA), and MG/TA coactivation index) and centre of pressure (CoP) parameters (i.e., range of CoP, peak of CoP velocity, and recovery time) were analysed to assess postural response. A two-way ANOVA (Group × Stimulation Condition) was performed. **Results:** Both groups had shorter recovery time (determined by CoP) and MG onset latency in the active vs. sham condition. **Conclusions:** The results of our pilot study suggest that a single 20 min tDCS session (2 mA) applied over M1 enhances postural responses similarly in PwPD with and without a history of falls in the past year.

Keywords: Parkinson's disease; transcranial direct current stimulation; postural control; falls

Academic Editor: Bryan Riemann

Received: 30 November 2025
Revised: 23 January 2026
Accepted: 28 January 2026
Published: 3 February 2026

1. Introduction

Postural instability is one of the most common impairments associated with falls in people with Parkinson's disease (PwPD), evidenced mainly in situations with external

perturbation [1,2]. PwPD demonstrates excessive antagonist muscle activity and coactivation, and prolonged recovery time after perturbation [2,3]. These alterations are related to reductions in cortical excitability caused by the physiopathology of Parkinson's disease (PD) [4–6], and are responsible for impaired information processing, which results in a delayed onset of postural responses after external perturbations [2]. In addition, PwPD demonstrated greater peak, velocity, and displacement of the centre of pressure (CoP) after external perturbations when compared to their healthy age-matched counterparts [3,7,8]. Specifically, PwPD fallers present a greater range of CoP than non-fallers in response to support base perturbation [1]. Considering that postural control is a complex system involving numerous sensorimotor processes [9], it makes sense to understand some neuromuscular and biomechanical characteristics involved in postural response after perturbation in PwPD.

Although pharmacological treatment is widely used in PD, postural control is less responsive [2]. Pharmacological treatment reduces bradykinesia and rigidity, which allows PwPD to explore the environment [2]. However, PD medication does not improve key postural response components [2]. Thus, complementary therapies, such as transcranial direct current stimulation (tDCS), have been explored [10,11].

Anodal tDCS increases cortical and subcortical excitability and enhances neural network abilities, even in neurodegenerative diseases such as PD [12]. A previous study showed that tDCS improved the recovery time and onset latency of the medial gastrocnemius (MG) [10]. However, despite its promising potential [10,11], variable responses have been reported regarding postural control in PwPD. While previous studies have not identified clear predictors of tDCS response [10,11], PwPD with poorer baseline postural responses (e.g., fallers) appear to benefit more from tDCS, regardless of the clinical characteristics of the disease [13]. Besides the greater range of CoP [1], PwPD fallers demonstrated worse UPDRS motor subscale scores, more advanced disease stage, as measured by the Hoehn & Yahr scale, and also an increase in postural sway, measured by a swaymeter that shows body displacement at the waist [14,15].

In this sense, identifying factors that influence tDCS-related changes in PwPD is important and may contribute to the optimisation of treatment protocols for this population. Thus, we expand our previous findings [10,13], in the present pilot study, by investigating the effects of a single session of anodal tDCS on reactive postural responses in PwPD, considering their history of falls in the past year. We hypothesised that tDCS would improve the reactive responses in PwPD, particularly in fallers, by reducing CoP range, recovery time, and MG latency.

2. Materials and Methods

2.1. Participants

Twenty-two PwPD (Hoehn & Yahr stages 1–3) were selected for this pilot study. To be included, participants must have been diagnosed with PD by a particular neurologist following the criteria from the London Brain Bank [16]. Exclusion criteria included the presence of orthopaedic or vision impairments that could affect balance, uncontrolled diseases that could affect peripheral sensory information, cognitive decline (indicated by a score under 20 on the Mini Mental State Exam—MMSE) [17], and risk for tDCS [10]. The study was approved by the Research Ethics Committee of São Paulo State University (CAAE: 87653818.2.0000.5465), and all experimental procedures were conducted after the participants had provided written consent.

The participants were distributed into two groups considering their fall history: fallers (n = 12) and non-fallers (n = 10). The fall history was obtained from a weekly follow-up database covering the previous 12 months. Participants were classified as "fallers" if they

experienced at least one fall during this period. The present study considered "fall" to mean the unintentional movement of the body to the ground or to a lower level without the ability to correct it in time [18]. Participants were instructed to report any falls and their characteristics (i.e., the number, circumstances/reasons, and location of falls, and the state of PD medication during them).

2.2. Procedures

This is a crossover, randomised, double-blinded, and sham-controlled pilot study. All procedures occurred in the "ON" state of the PD medication. The experimental protocol was conducted over three laboratory visits. In visit 1, clinical characteristics were assessed (Table 1). In visits 2 and 3, each individual participated at the same time, with at least 2 weeks apart [19]. During these visits, tDCS and postural assessment were performed (Figure 1). Finally, after the third visit, participants reported their perception of tDCS (i.e., what type of stimulation condition was performed) on each day.

Table 1. Demographic, clinical, and cognitive data for sample characterisation. Continuous variables are presented as mean and standard deviation values. Discrete variables are presented by median and quartiles (25–75).

	Faller (*n* = 12)	**Non-Faller (*n* = 10)**	***p*-Value**
Sex (male/female)	6/6	8/2	0.204
Age (years)	69.50 ± 10.11	68.20 ± 6.92	0.734
Body mass (Kg)	67.50 (61.00–73.00)	75.00 (66.60–86.52)	0.129
Height (cm)	162.99 ± 11.14	166.89 ± 10.81	0.417
MDS-UPDRS III (0–132 pts)	31.50 (30.00–48.50)	36.50 (23.25–45.50)	0.691
MMSE (0–30 pts)	28.00 (25.25–28.00)	27.50 (25.50–28.25)	0.946
Disease time (years)	3.00 (2.25–7.00)	4.00 (3.00–7.50)	0.325
LEDD (mg/day)	521.48 ± 281.41	611.01 ± 305.43	0.483

MDS-UPDRS III = Movement Disorders Society—Unified Parkinson's disease Rating Scale (motor part); MMSE = Mini-Mental State Examination; LEDD = Levodopa Equivalent Daily Dose.

Anodal tDCS (Microestim GENIUS, NKL Electronic Products, Brusque, SC, Brazil; electrodes = 35 cm^2) was applied over the M1 [19,20] most affected by PD (i.e., C3 or C4) [19,21] before postural response assessment. The cathode was positioned over the supraorbital contralateral region [19]. All participants received both active (2 mA/20 min) and sham (2 mA/10 s) [19,20] stimulation in a randomised and counterbalanced order [10]. To analyse the possible adverse effects of tDCS, a questionnaire was administered immediately at the end of each stimulation [22–24].

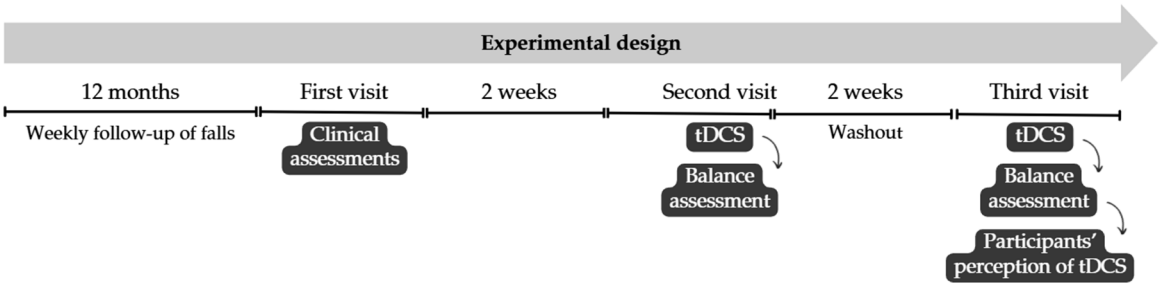


Figure 1. Experimental workflow. tDCS = transcranial direct current stimulation protocol (active and sham); Washout = period necessary to minimise possible effects of tDCS based on previous studies. Note: arrows represent the sequence of the assessments.

To assess balance, the participant stayed in a bipodal position on a force plate (AccuGait, Advanced Mechanical Technology Inc., Boston, MA, USA, 200 Hz), which was

placed on perturbation equipment. Seven trials with temporally unpredictable backward perturbations were performed (20 cm/s and 5 cm of magnitude) [1,3]. Each trial had an approximate duration of 40 s, with 30 s between them. The protocol was performed in this way to maintain a complete duration under 40 min after tDCS; the peak of the acute effect of tDCS seems to be during this period [25].

CoP was analysed during the balance assessment, including the range of displacement, the peak velocity, and the recovery time [10]. The range of displacement represents the distance between the maximum and minimum positioning of the CoP. The peak velocity represents the maximum speed of the CoP displacement. Both were analysed during the period from the onset of perturbation to 800 ms [8]. The recovery time (i.e., time to recover balance) was calculated as the interval from the onset of the perturbation to the moment the CoP displacement stabilised, which means the moment when the CoP variability (standard deviation over 1 s) after the perturbation was less than or equal to the CoP variability during the baseline period (determined as 1 s before the perturbation) [10].

To assess muscle activity, a 16-channel electromyograph (EMG – TrignoTM Wireless System, Delsys, Inc., Boston, MA, USA, 2000 Hz) was used. The EMG sensors were positioned [26] on the MG and tibialis anterior (TA) muscles of the limb most affected by PD [10]. The following EMG parameters were analysed: MG onset latency time, integral (iEMG) of MG and TA activity, and MG/TA coactivation [10,27]. Onset latency time represents the period between the perturbation and the onset of muscle activity. iEMG values (i.e., the magnitude of activation of MG and TA muscles) were calculated by the area below the curve, determined by $iEMG_{period} - iEMG_{reference}$ ($iEMG_{reference}$ was analysed from 3500 to 3450 ms before the perturbation) [28]. The coactivation index MG/TA was determined through the ratio (((2 × root mean square_antagonist)/(root mean square_agonist + root mean square_antagonist)) × 100) [28,29]. Perturbation, EMG, and CoP behaviour were synchronised. All EMG and CoP analyses were performed using algorithms developed in a Matlab^{TM} environment (Mathworks, Natick, MA, USA).

SPSS 22.0 (IBM, Armonk, NY, USA) was used for statistical analysis (significance at $p < 0.05$). Normality and homogeneity were verified by Shapiro–Wilk and Levene tests, respectively. The range of CoP, peak of CoP velocity, and iEMG of MG and TA were log10-transformed due to a non-normal distribution. Table 2 shows the non-transformed values of the outcome variables. The log10-transformed values are presented in the Supplementary Materials (Table S1). A two-way ANOVA was conducted with factors for group (fallers vs. non-fallers) and stimulation condition (2 mA vs. sham), with repeated measures for the second factor. The effect size of the ANOVA was reported in terms of Partial eta-squared (η_p^2) (small = 0.21 to 0.50, medium = 0.51 to 0.79, and large $\geq$ 0.80 effect) [30]. The adverse effects of tDCS in each group were analysed by the Wilcoxon test.

Table 2. Mean and standard deviation of the CoP and EMG parameters that did not demonstrate statistical differences.

	Faller (*n* = 12)		**Non-Faller (*n* = 10)**	
	Active	**Sham**	**Active**	**Sham**
Range of CoP (cm)	3.71 ± 1.04	3.67 ± 1.11	3.25 ± 0.84	3.25 ± 0.60
Peak of CoP vel. (cm/s)	16.75 ± 5.69	16.91 ± 5.43	14.89 ± 4.20	14.8 ± 2.55
iEMG MG (μV/ms)	13.34 ± 5.70	11.69 ± 5.73	16.54 ± 27.48	11.85 ± 10.38
iEMG TA (μV/ms)	9.03 ± 5.93	15.93 ± 15.77	15.72 ± 22.24	16.66 ± 19.45
MG/TA coactivation (%)	66.66 ± 21.04	63.61 ± 13.31	61.77 ± 23.34	70.91 ± 9.94

Vel. = velocity; iEMG = magnitude of muscle activation-integral; MG = medial gastrocnemius; TA = tibialis anterior.

3. Results

Statistical analysis indicated no significant differences between groups for the characterisation variables (Table 1).

Regarding the characteristics of the falls (Figure 2), 37.5% of individuals experienced only one fall during the follow-up period. In addition, 62.5% of individuals experienced a recurrence of falls in this period (i.e., 12.5% experienced two falls, 37.5% experienced three falls, and 12.5% experienced four falls) (Figure 2a). Approximately 78% of PwPD reported having a "Satisfactory effect" from the medication, 11.11% reported an "Unsatisfactory effect", 5.56% stated they "Did not take the medication", and 5.56% "Did not know" (Figure 2b). Regarding the circumstances or reasons for the falls, 22.22% were due to being "Unbalanced without evident cause", 16.67% due to "Slips", another 16.67% due to "Stumbles", 5.56% due to "Collisions and/or pushes", 5.56% caused by the individual's "Freezing of gait", and 33.32% occurred without classification (Figure 2c). Finally, the percentage values for the location of the falls were 50% "Indoor" and 50% "Outdoor" (Figure 2d).

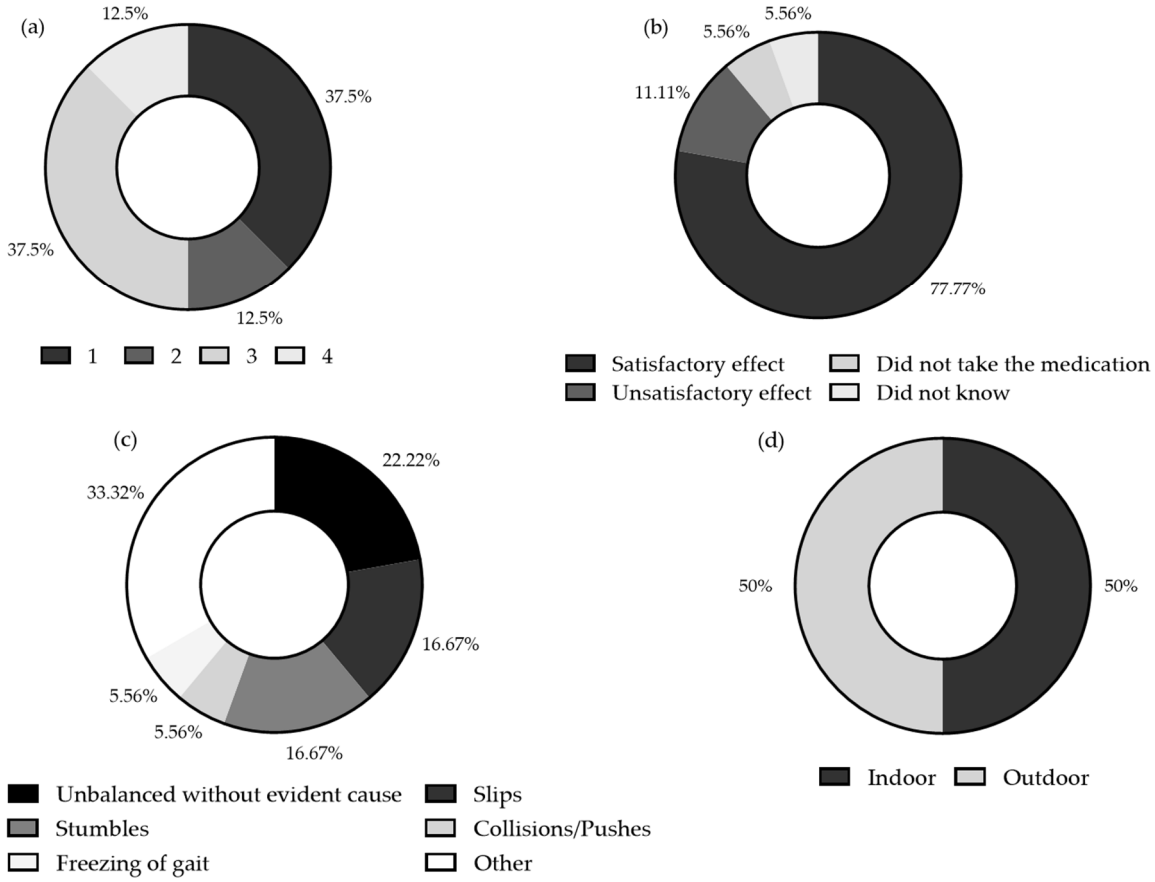
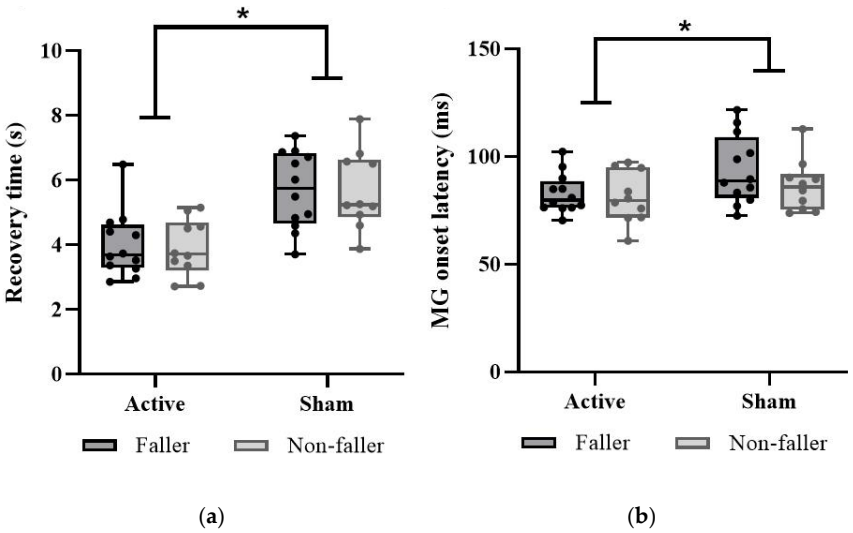


Figure 2. Self-reported number (**a**), medication state (**b**), circumstances (**c**), and location of falls (**d**). Other = falls without self-reported circumstances or with circumstances that are unclassifiable (fell out of the bed, for example).

No interaction between group*condition was evidenced by ANOVA for any parameters: recovery time ($F_{1,20}$ = 42.738; $p < 0.001$; η_p^2 = 0.032); range of CoP ($F_{1,20}$ = 0.329; p = 0.573; η_p^2 = 0.016); peak of CoP velocity ($F_{1,20}$ = 0.012; p = 0.914; η_p^2 = 0.001); MG onset latency ($F_{1,20}$ = 9.057; p = 0.007; η_p^2 = 0.052); iEMG TA ($F_{1,20}$ = 0.485; p = 0.494; η_p^2 = 0.024); iEMG GM ($F_{1,20}$ = 0.210; p = 0.652; η_p^2 = 0.010); coactivation index MG/TA ($F_{1,20}$ = 1.352; p = 0.258; η_p^2 = 0.064).

ANOVA indicated a main condition effect for the recovery time ($F_{1,20}$ = 42.738; $p < 0.001$; $\eta_p^2 = 0.861$) and MG onset latency ($F_{1,20}$ = 9.057; $p = 0.007$; $\eta_p^2 = 0.312$), exhibiting large and small effect sizes, respectively, based on partial eta-squared values. The analysis indicated that PwPD demonstrated short recovery times and MG onset latency in active vs. sham experiments ($p < 0.001$ and $p = 0.007$, respectively) (Figure 3). No statistical differences were evidenced for the other parameters (Table 2). Complete statistical analyses are presented in the Supplementary Materials (Table S2).



(**a**) (**b**)

Figure 3. Means and standard deviations of (**a**) recovery time and (**b**) MG onset latency for falling and non-falling groups in the active and sham conditions. * indicates a difference between tDCS conditions. Note: dots represent values of each individual.

The adverse effects more frequently related were tingling and itching (Table 3). However, the intensity was considered light according to the questionnaire score. Both effects were reported in both stimulation conditions. In addition, 36% of the participants were able to identify correctly the type of stimulation performed in a given session.

Table 3. Adverse effects of tDCS and perceptions of stimulation condition expressed in median and interquartile ranges (25–75).

	Faller (*n* = 12)			**Non-Faller (*n* = 10)**		
	Active	**Sham**	***p*-Value**	**Active**	**Sham**	***p*-Value**
Participant's report						
Headache	0 (0–0)	0 (0–0)	0.32	0 (0–0)	0 (0–0)	1.00
Neck pain	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Scalp pain	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Tingling	0 (0–1)	0 (0–1)	0.317	1 (0–1)	1 (0–1)	1.00
Itching	0 (0–0.75)	0 (0–1)	0.564	0 (0–0)	0 (0–0)	0.317
Burning sensation	0 (0–0)	0 (0–0)	0.564	0 (0–0)	0 (0–0)	1.00
Sleepiness	0 (0–0)	0 (0–0)	0.317	0 (0–0)	0 (0–0)	1.00
Metallic/iron taste	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Fatigue	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Trouble concentrating	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Acute mood change	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	1.00
Investigator's report						
Skin redness	0 (0–0)	0 (0–0)	0.157	0 (0–0)	0 (0–0)	0.317
Skin irritation	0 (0–0)	0 (0–0)	1.00	0 (0–0)	0 (0–0)	0.317

0 = none (I did not feel the sensation addressed); 1 = mild (I mildly felt the sensation addressed); 2 = moderate (I felt the sensation addressed); 3 = strong (I felt the sensation addressed to a considerable degree).

4. Discussion

The present pilot study aimed to investigate the effects of a single session of anodal tDCS on reactive postural responses in PwPD, considering their fall history. We hypothesised that tDCS would improve the reactive responses in PwPD, particularly in fallers, by reducing CoP range, recovery time, and MG latency. Our hypothesis was partially confirmed. As expected, a 2 mA single session of tDCS improved postural responses in PwPD after external perturbations. Improvements were evidenced by a decrease in time taken to recover a stable position and in the onset latency time (Figures 3a and 3b, respectively). Unexpectedly, our results suggested no differences between fallers and non-fallers, since our data demonstrated similar postural responses between them after tDCS. However, this similar response could be influenced by a type II error due to our small sample size. Besides this important limitation, we present some aspects that could explain, at least in part, these results.

The contribution of tDCS to reactive postural responses may be attributed to improvements in sensorimotor integration and a greater reliance on the direct postural control pathway [10]. PwPD have reduced sensory signalling in cortical and subcortical structures [31], leading to difficulties in using this information to generate/control postural responses [10]. tDCS over M1 may increase the excitability of cortical and subcortical structures, improving postural control in PwPD by decreasing muscle onset latency [10]. Our results corroborate previous studies on healthy adults, which reported a decrease in muscle onset latency after tDCS [19]. Onset latency time represents the period between the perturbation and the onset of the response, which means the time required for motor nerve conduction to the muscle, including the processing of sensory information to perform a reactive response [32,33]. Also, M1 is involved in the integration and processing of sensory information, elucidating its role in postural control [34]. Thus, the idea of improvement in the identification and processing of sensory information after tDCS is reinforced by the observed reduction in onset latency time, resulting in a faster response after external perturbation. It is important to highlight that, although studies do not demonstrate an immediate reduction in fall risk from a decrease in onset latency, some studies showed that impairments in response speed and time of muscle activation contribute to postural instability in PwPD [35,36], which may be an important indicator for the occurrence of falls. A longer time to respond after an external perturbation suggests a lower chance of recovering balance. This reinforces the importance of future studies evaluating the consequences of reductions in muscle onset activation time with more robust protocols (i.e., multiple sessions of tDCS) and the monitoring of falls after the protocol.

In addition, tDCS over M1 may have increased the involvement of the motor cortex in postural control. Previous studies demonstrated that tDCS over the motor cortex may increase the activation of subcortical structures involved in postural responses (e.g., pedunculopontine nucleus and reticular formation) [37]. The motor cortex and the subcortical structures are involved in the direct postural control pathway, which includes primarily functions in automatic control situations (which is impaired in PwPD), such as reactive postural responses [37,38]. This greater involvement of the direct postural control pathway is also supported by studies showing that tDCS over M1 decreases prefrontal cortex activity during postural response in PwPD [10].

It should be noted that the duration of tDCS effects remains unclear. A recent study demonstrated that a single session may improve motor symptoms of PD [39]. However, they did not provide follow-up information about the long-term duration of tDCS effects. Previous studies with multiple sessions have shown the potential of tDCS over different cerebral areas to improve motor symptoms, executive function, gait, and even postural

response for a longer period. The effects were assessed during the protocol and after it (1 week–3 months of follow-up) [40–43].

Regarding fall history, of the 22 participants in the study, ~54% had experienced at least one fall over the past year. These results are similar to previous studies, in which more than half of the PwPD experienced falls [44,45]. Of the individuals who fell during this period, 62.5% experienced recurrent falls (Figure 2). Similar results were observed in a systematic review, in which 70% of PwPD fallers experienced recurrent falls [46]. Furthermore, the majority (78%) of the individuals reported being under satisfactory medication effect at the time of the fall, which confirms the idea that medication does not seem to be able to improve postural instability (i.e., it does not avoid fall occurrence). In this sense, the individual feels more confident when taking the medication due to the improvement of other motor symptoms, such as reduced bradykinesia and rigidity, but the magnitude and speed of postural responses are not enhanced by the medication, which explains, at least in part, the occurrence of falls [47,48]. The main causes of falls in PD are postural instability, freezing of gait or festination, and stumbles [44,49], which was corroborated by our present study (Figure 2).

As mentioned, our data suggest similar tDCS-related changes in postural responses between fallers and non-fallers. A previous study showed that baseline characteristics seem to predict the tDCS response in individuals with chronic stroke [50]. In addition, a previous study including PwPD showed that baseline postural control seems to influence tDCS-related changes in postural responses [13], so fallers should benefit more from tDCS than non-fallers due to their more pronounced postural deficits [1,13], which was not observed. However, both groups showed similar responses in the sham condition, which does not indicate a worse postural control at baseline (i.e., even without anodal tDCS) in fallers. A possible explanation is the low number of falls in the group. We considered individuals with only one fall in the previous 12 months as "fallers". However, one fall does not indicate a recurrent faller status, as it could have been incidental rather than reflective of underlying fall-related changes. Even so, previous studies have shown that both single and multiple fallers exhibit the fear of falling. Added to postural instability, a fear of falling impairs quality of life and mobility in PwPD [51,52]. These results reinforce the importance of analysing fall history in PwPD. It should be highlighted that this study exhibits a complementary analysis of our previous study [10,13]. It is important to understand whether and how sample characteristics (e.g., fall history) influence responsiveness to tDCS. Another explanation is that the similar responses between fallers and non-fallers could be due to the small sample size, which may affect the group comparison (i.e., increasing the risk of statistical type II error). Thus, the results of our pilot study should be considered with caution, mainly regarding the comparison of fallers vs. non-fallers. Future studies with a large sample size are needed to confirm the effect of tDCS on postural control, considering fall history. The small sample size is an important limitation of our study.

Another study limitation is that the postural control assessments were performed immediately after the tDCS protocol, which could interfere with the necessary time for cortical activity modulation [53]. A previous study demonstrated that cortical excitability may not increase immediately after tDCS in older adult individuals [54]. Also, the direction of the perturbation must be considered as well. The present protocol includes only the backward direction. To represent real-world situations, future studies should consider multi-directional perturbations (i.e., forward, backward, left, and right). In addition, falls were considered as a dichotomic measure. In this sense, the effect of the recurrence of falls in PwPD remains unclear since the comparison was performed with individuals classified only as fallers or non-fallers (i.e., without considering the specific number of falls). Future studies must consider this. Finally, this is a single-tDCS-session study, which provides

only the immediate effects of the stimulation on postural control. It should be noted that the effects of a single session of tDCS may be temporally limited (i.e., a few hours after stimulation). Thus, future studies should assess the long-term effect of tDCS on postural control in PwPD fallers and non-fallers, since this remains unclear.

Besides motor benefits, it should be highlighted that the present study demonstrated that tDCS may be a safe technique of non-invasive brain stimulation, in which participants reported no or light adverse effects (Table 3) [23,24,55].

5. Conclusions

Our pilot study indicated that a 20 min session of anodal tDCS at 2 mA applied to M1 improved postural responses similarly in PwPD with and without a history of falls in the past year.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/biomechanics6010017/s1, Table S1: Statistical analysis of CoP and EMG parameters (Table 2) presented by partial eta-squared, *p*-value, and f-value; Table S2: Log-transformed values of CoP and EMG parameters expressed as mean and standard deviation.

Author Contributions: Conceptualization, R.V.e.M.R. and V.S.B.; methodology, R.V.e.M.R., G.A.G.M., D.O.-S. and V.S.B.; formal analysis, R.V.e.M.R., B.R.L., G.A.G.M., D.O.-S. and V.S.B.; data curation, R.V.e.M.R., B.R.L., G.A.G.M., M.L.T., T.M.S. and D.O.-S.; writing—original draft preparation, R.V.e.M.R.; writing—review and editing, B.R.L., G.A.G.M., M.L.T., T.M.S., D.O.-S. and V.S.B.; visualisation, R.V.e.M.R. and V.S.B.; supervision, V.S.B.; funding acquisition, R.V.e.M.R. and V.S.B. All authors have read and agreed to the published version of the manuscript.

Funding: The present study was supported by the São Paulo Research Foundation (FAPESP) [grant number #2018/07385-9, #2023/14387-6, #2023/12924-4]. FAPESP had no role in the study design, collection, analysis, or interpretation of the data, in the writing of the manuscript, or the decision to submit the paper for publication.

Institutional Review Board Statement: The study was approved by the Research Ethics Committee of São Paulo State University (CAAE: 87653818.2.0000.5465, Protocol number: 2.739.491) on 27 June 2018.

Informed Consent Statement: Written informed consent to publish this paper was obtained from the patients.

Data Availability Statement: The data presented in this study are available only on request from the corresponding author due to ethical reasons.

Acknowledgments: We gratefully thank all patients for their participation in our study. In addition, the authors thank the São Paulo Research Foundation (FAPESP).

Conflicts of Interest: The authors declare no conflicts of interest.

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