Relationship bettwen movement initiation and the termination of the previous movement in bradykinesia: a systematic review.

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Abstract—**Parkinson's disease (PD) is a progressive neurodegenerative disease related to the degeneration of dopaminergic neurons in the Nigrostriatal system. Bradykinesia is one of the symptoms of PD and consists of slowness of movement and decrease in amplitude or speed as movements are continued and has been traditionally considered the consequence of a failure of basal ganglia output to the primary motor cortex (M1). In PD, the slowness of voluntary movement has been ascribed to the difficulty to initiate movement (akinesia), but studies obtained incompatible results with the hypothesis that the compromised ability to initiate a new movement is the main cause of bradykinesia. Thus, the present work aims to investigate, through the scientific literature, whether the difficulty of initiating voluntary movements in Parkinson's disease is due to the impaired interruption of the termination of previous movement. For this, the present review was conducted in accordance with the guidelines outlined by Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement. Articles were searched in the PubMed, Web of Science, Scopus and Science Direct databases, with the descriptors "Parkinson's Disease", "Bradykinesia" and "Movement termination", published between the years 2011-2021. In total, 328 records were identified through literature search procedures. After performing the selection steps, with the aid of Rayyan - Intelligent Systematic Review, 4 articles were included for review. Literature studies report and suggest that the difficulty in initiating the volitional movement in PD bradykinesia is related to the difficulty in the previous movement termination.**

Keywords — Parkinson's Disease, Bradykinesia, Akinesia, Movement Termination.

I. INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disease related to the degeneration of dopaminergic neurons in the Nigrostriatal system [1]. It was estimated that, in 2004, there were about 4 million people with PD and, based on that, it is expected that in 2030 twice as many people will be affected by this disease [2]. Dopamine is a neurotransmitter necessary for the correct movement control [3], therefore, its reduction causes a decrease in movements, being the first perceptible sign of PD (cardinal signs and symptoms) [4][5]. In general, the movement disorders in PD have been considered to result from imbalances in the direct, indirect and hyperdirect pathways of the basal ganglia [6][7].

These cardinal motor symptoms are tremor, bradykinesia/akinesia, rigidity and postural instability, in addition, the clinical picture includes other motor and non-motor symptoms [8]. The diagnosis is principally clinical and made through subjective clinical scales. The most used scale in the clinical setting for this evaluation is the Unified Parkinson's Disease Rating Scale (UPDRS) [9].

According to the first formal diagnostic criteria for PD, bradykinesia consists of a slow onset of voluntary movement with a progressive reduction in the speed and amplitude of repetitive actions [10]. The definition was maintained by the European Federation of Neurological Societies (EFNS) criteria for Parkinson's disease diagnosis [11] and by the current Movement Disorders Society (MDS) criteria, that define bradykinesia as slowness of movement and decrement in amplitude or speed (sequence effect) as movements are continued [12][13]. The tools available for the evaluation of bradykinesia are subjective (clinical evaluation scales), as is for PD, but several studies and technologies have emerged with the aim of making the characterization of bradykinesia more quantitative [14][15].

Movement disorders, including bradykinesia, have been attributed to imbalances in the direct and indirect/hyperdirect pathways in the basal ganglia, resulting in reduced output activity of the motor cortex [4][16][17]. Moreover, altered mechanisms of sensorimotor integration may also play a role in the pathophysiology of bradykinesia [18]. In Parkinson's disease, the slowness of voluntary movement has been ascribed to the difficulty to initiate movement (akinesia) [19], however, in their study, Evarts et al. (1981) reported that both reaction time and movement time tend to be prolonged in Parkinson's disease, both of which reflect on the speed of the movement performed. However, the increase in reaction time, which is related to the speed of onset of the response, is relatively slight. Furthermore, their results are incompatible with the hypothesis that the main cause of bradykinesia is the impaired ability to initiate a new movement [21].

Some studies have shown that complex (multi-joint) movements such as reaching, grabbing, pulling are also slower in Parkinson's disease [21-24]. The execution of two different tasks performed simultaneously in Parkinson's disease patients leads to a more severe slowing in movement than that observed when each task was performed alone [25-27]. In related to the execution of sequential movements, the slowness of movement is even worse in patients with PD [28][29]. Therefore, this study aims to investigate, through the scientific literature, whether the difficulty in initiating voluntary movements in Parkinson's disease is due to the

impaired interruption of the termination of the previous movement.

II. Methodology

This systematic review was conducted in accordance with the guidelines outlined by Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) statement [30].

A. Data Sources and Search Strategy

A literature search was carried out using the academic databases MEDLINE via PubMed, Web of Science, Scopus and Science Direct. The search strategy included the descriptors "Parkinson's Disease", "Bradykinesia" and "Movement termination". In tracking the publications, the logical operator "AND" was used in order to combine the descriptors. Articles published in the last 10 years, including articles up to June 2021, were searched.

B. Inclusion and Exclusion Criteria

The studies meeting the following inclusion criteria were included in the current review: (1) participants with a clinical diagnosis of PD according to the criteria of the UK Parkinson's Disease Society Brain Bank criteria; (2) articles unrelated to the movement termination; (3) published in English or Portuguese; (4) Published between 2011-2021. Restrictions on age, disease duration and condition severity were not adopted. Overlapping or duplicate articles, thesis proceedings, conferences, book chapters, encyclopaedias, abstracts and articles that did not present results related to the objective of this work were excluded.

C. Study Selection

One reviewer carried out the literature search, according to the search strategy and all results were imported into Rayyan – Intelligent Systematic Review [31] for screening. After searching, the selection steps of the resulting articles were carried out. First, the duplicates were removed using Rayyan. Inclusion and exclusion criteria were applied to titles and abstracts, and articles were selected accordingly. Full text of the studies retained during the previous step was screened by the reviewer. The relevant aspects of the selected studies were analyzed according to the objective of this work.

III. Results

A. Selection of the Studies

In total, 328 records were identified through literature search procedures. Once duplicates (24 articles) had been removed, and titles and abstracts were screened, the full text of 6 articles was evaluated for the final inclusion. Finally, 4 articles were selected for the current review. The selection flow chart is shown in Fig. 1. Percentage data exported from Ryyan show that 98.4% of articles were excluded and 1.6% were included, after duplicates removal (Fig. 2).

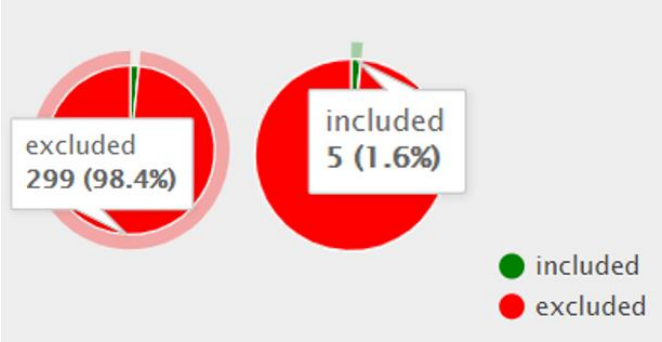

Fig. 2. Percentage of articles excluded and included after screening

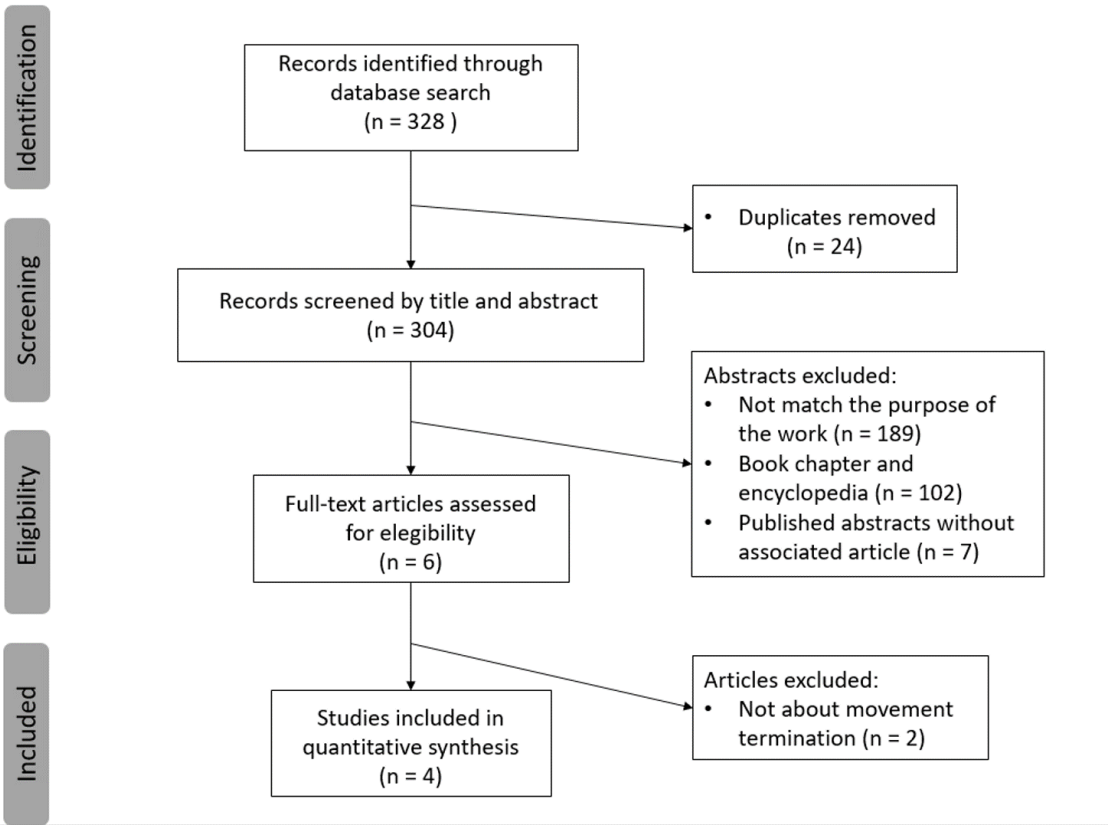

Fig. 1 – Flowchart for systematic literature search across the databases.

B. Participants

All patients in the studies had a clinical diagnosis of PD based on the criteria established by the UK Parkinson's Disease Society Brain Bank. Three of the studies subdivided PD patients based on their severity by the Hoehn-Yahr stage of bradykinesia, the classifications were moderate, severe and maximum, they also included patients without dementia (mini mental state examination scores above the cut-off of 23) [32-34]. One article included patients with bilateral implantation of deep brain stimulation (DBS) electrodes in the subthalamic nucleus (STN) [35]. Finally, in addition to a PD group, three studies provided a sample of healthy controls matched for age with patients [32-34].

C. Characteristics of the studies

The analysis of the studies allowed the identification of two categories, due to the thematic recurrence. Three of them related the difficulty in initiating volitional movement with the end of the previous movement, being the first category [32-34]. In both works the authors perform similar tasks in the methodology to assess this relationship: the gap, no-gap and overlap tasks. In the gap-task the fixation-stimulus was turned off 200 ms before the trigger on-stimulus, in the no-gap task the fixation-stimulus was tuned off, and simultaneously, the on-stimulus was turned on, and in the overlap task the trigger on-stimulus was turned on during the visual-conditioning-stimulus presentation (200 ms-overlap) [32-34]. Of the three studies, one analyzed that relationship of bradykinesia in the upper limb moviment [32], whereas the others in gait [33, 34]. The other category was related to evaluating the role of the STN at the end of the movement, it included only one article that carried out the study in PD patients with implantation of STN DBS electrode [35]. The literature review results, presented in Table 1, show the two categories and some points of the articles in each category (study, objective, study design, results and their conclusions).

IV. Discussion

This review aimed to investigate, through studies developed in the scientific literature, whether the difficulty in initiating volitional movements in Parkinson's disease, in bradykinesia, is due to an impaired interruption at the end of the previous movement. Participants of included studies satisfied the UK Parkinson's Disease Society Brain Bank criteria and had mild to maximum disease severity (H&Y 2–4), except for the study of Hsu et al. [35], who did not classify patients by disease severity on the H&Y scale.

Of the studies included in the review, three responded directly to the research question of this work [32-34]. The results of Warabi et al. [32] confirmed the view that during gap tasks, patients with PD have prolonged ocular and manual movement latencies [36]. However, this difficulty was significantly less than during the overlay task. These results have indicated that the difficulty in initiating movement during the gap task is less severe than the difficulty in initiating during the overlap task in PD, concluding that the latency prolongation is due to the difficulty in finishing the motor program of the primary posture proceeding from the cognitive visuospatial situation of conditioning stimuli [32] [37].

Following the same line of direct relationship with the research question, Warabi et al. [33] and Warabi et al. [34] studied, respectively, the relationship of the difficulty of movement initiation due to the difficulty of ending the previous movement and how the spatiotemporal parameters in bradykinesia are related to this difficulty in initiation/termination, but when it comes to gait. The dynamic gait initiation is controlled by the continuity synchronized antagonist electromyography activity, this activity involves the pause of the soleus tonic EMG activity and the burst of the EMG activity from the tibialis anterior, observed in the termination/initiation process [38] [39]. The results of Werabi et al [33] indicated that the decrease in gait speed of bradykinesia is correlated with the unsynchronized off/on latency interval. This is due to excessive long tonic EMG activity of the soleus muscle in the advanced stages of the disease.

Werabi et al [34] showed that the changes of spatiotemporal parameters in gait bradykinesia are correlated to the unsynchronized of/on-latency difference. Their results even demonstrated that unsynchronized off/on latency EMG activities in homogeneous movement result in a bradykinesia in homogeneous gait synergistic movement. Finally, the results of Hsu et al. [35] on the role of the STN in the movement termination suggest that alpha event-related de-synchronization (ERD), in addition to low- and high-beta ERD, is important for generating tonic movement termination. Differently from what is shown by Cassim et al [40] which illustrates that only beta event-related de-synchronization/synchronization (ERS) is present both for the beginning and for the termination of a tonic movement. A difference shown between ignition and termination of tonic movement, shown by the authors, is that ERD and ERS alpha, low beta and high beta behave similarly, both temporarily and spatially, in the terminating tonic movement [35]. Furthermore, a simultaneous ERD across wide frequency bands in STN, may play a pivotal role in terminating volitional movement, unlike asynchronous ERD frequency bands present in movement initiation. Thus, Hsu et al [35] suggested that the STN participates in the preparation of volitional movement termination, but through a different mechanism from that in movement initiation.

V. Conclusion

Literature studies report and suggest that the difficulty in initiating the volitional movement in PD bradykinesia is related to the difficulty in the previous movement termination and that the sub-thalamic nucleus participates in the preparation for the termination of the voluntary movement, but through a different mechanism than that of the initiation.

Acknowledgment

The present work was carried out with the support of the National Council for Scientific and Technological Development (CNPq), Coordination for the Improvement of Higher Education Personnel (CAPES – Program CAPES/DFATD-88887.159028/2017-00), and the Foundation for Research Support of the State of Minas Gerais (FAPEMIG-APQ-00942-17). A. O. Andrade is fellow of CNPq, Brazil (304818/2018-6).

Table 1 - Literature review results.

Category 1: Relationship of the difficulty in starting a movement with the end of the previous movement in PD.				
Study	Objective	Study Design	Results	Their Conclusions
Warabi et al., 2011 [32]	To determine whether the difficulty of initiating volitional movements in Parkinson's disease is primarily due to impaired termination of preceding movement/posture or to impaired initiation of new movement.	23 PD patients and 6 controls were first asked to visually fix a stationary spot and simultaneously align wrist position precisely with it. They were then asked to make quick eye and wrist movements to a test stimulus presented by LEDs. The latencies of ocular and manual movements to the test stimulus were analyzed under two conditions: gap and overlap task.	Stage II Parkinson's disease ocular and manual movement latencies in the overlap task were longer than those of elderly controls and the same patient in the gap task. The latencies of ocular and manual movements of the patient with stage IV Parkinson's disease were even higher. In contrast, during the gap task, the same patient exhibited much shorter latencies.	"The prolonged ocular and manual latencies in the overlap task in Parkinson's disease patients mainly reflect the difficulty in terminating preceding movement/anterior posture. Movement initiation was less impaired than movement completion."
Warabi et al., 2018 [33]	To examine whether the dynamic gait initiation changes observed in Parkinson's disease are due to the difficulty in terminating the previous motor program.	35 patients with PD and 9 healthy, were fixed on a visual fixation target (conditioning-stimulus), the voluntary-gait was triggered by a green (go) or yellow (no-go) visual stimulus. While the subject walked on a level floor, soleus EMG latencies, tibialis anterior, and the y-axis vector of the single-floor reaction force were examined. Three paradigms were used to distinguish between off (termination, soleus EMG pause) / on (initiation, burst of tibialis anterior) latencies: gap, no-gap, overlap.	In the gap task, the on-latency remained unchanged for all subjects. In Parkinson's disease, the visual fixation targets prolonged both the on/off latencies in the overlap-task. In all tasks, the off-latency was prolonged and the off/on latencies were unsynchronized. The off/on latency difference decreased with disease progression, and the off/on latency interval was desynchronized in the advanced stages of the disease, which changed the synergistic movement to a slow short-step-gait.	"Delayed gait initiation of synergic movement in Parkinson's disease was due to difficulty in the termination of the sensory-motor program (fixation) which is linked with the cognitive visuospatial condition. The slowness of the synergy of gait in Parkinson's disease was involved in the difficulty (long off-latency) in terminating the prior motor program which had been controlling the posture/movement."
Warabi et al., 2020 [34]	To examine how spatiotemporal parameters in gait bradykinesia link to difficulty in terminating posture and initiating gait locomotion.	41 PD patients and 15 healthy ones participated in the study, they were fixed on a visual fixation target and gait was triggered by visual or vocal stimulus-suggestion. The LED instructed subjects to quickly achieve their own comfortable walking speed on a level floor. The posterior-anterior force of the y-axis vectors of sole relating to soleus and tibialis-anterior EMGs were examined. The pause in tonic soleus EMG was defined as the off-latency of posture (termination) and the onset of a tibialis-anterior EMG-burst as the on-latency of gait.	Unsynchronized off/on-latency differences correlated with spatiotemporal parameters of dynamic-ratios, step-gains, gait-initiation, and gait speed in gait bradykinesia. A delayed and deficient initial backward body-shift of y-axis vector was linked to each difficulty in terminating posture and initiating gait, changing to random gait akinesia.	"In Parkinson's disease, the spatiotemporal parameters of gait bradykinesia in heterogeneous gait synergic movement stemmed from unsynchronized off/on-latency EMG activities in homogeneous gait movement, linking to each difficulty in terminating posture and initiating gait through an initial backward body-shift"
Category 2: The subthalamic nucleus and the movement termination.				
Study	Objective	Study Design	Results	Their Conclusions
Hsu et al., 2012 [35]	To assess the role of the subthalamic nucleus in movement termination.	14 patients with PD received recordings of local field potentials (LFPs) in the left STN on the fourth day after deep brain stimulation surgery. They performed phasic (quickly raising and releasing the right wrist) and tonic (dorsiflexing the right palm and holding the posture for 7 s before placing the palm back to the original flat position) movements. Movement onset (Mon) and movement offset (Moff) of the electromyographic activities were used as triggers to determine an 8 s (4 s before and after) LFPs epoch for time-frequency analysis. Movement-related power changes were assessed.	Pre-movement event-related de-synchronization (ERD) was found from ~ 2 s before to ~ 1 s after the Mon in both movements. The pre-movement ERD occurred earlier in the phasic movement, which persisted until ~ 0.8 s after the Moff. Post-movement event-related synchronization (ERS) immediately occurred around ~ 1 s after to the Mon in the phasic movement but not in the Mon tonic movement. The post-movement ERS was greater in the phasic than in the tonic movements and appeared ~ 0.8 s posterior to the Moff. High-beta (ERD) appeared earlier (3 s prior to Mon) than those of low-beta and alpha for the Mon phasic movement. There was no alpha ERD for the Mon tonic movement. Alpha, low-beta, and high-beta ERD all appeared about 1 s prior to the Moff tonic movement.	"The findings suggest that STN participates in the preparation of volitional movement termination but via a different mechanism from that in movement initiation. Unlike asynchronous ERD frequency bands present in movement initiation, a simultaneous ERD across wide frequency bands in STN may play a pivotal role in terminating volitional movement."

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