Corticobulbar Excitability in Parkinson's Disease: Evidence from TMS-Evoked Potentials of Submental Muscles

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Abstract

Background. The corticobulbar (CB) tract links the motor cortex with orofacial muscles and is closely associated with motivational arousal and reward-related processing. In Parkinson's disease (PD), where dopaminergic dysfunction is a core feature, alterations in corticobulbar excitability may provide insights into both motor and motivational deficits. **Objective.** This study investigated whether motor evoked potentials (MEPs) recorded from the submental muscle (SbM) and extensor carpi radialis (ECR) differentiate PD patients from matched healthy controls, and whether SbM excitability is modulated by acute dopaminergic stimulation. **Methods.** Twenty-two participants (10 PD patients, 12 healthy controls) were tested using transcranial magnetic stimulation (TMS). Patients underwent stimulation in both OFF (≥12 h levodopa withdrawal) and ON (1 h after levodopa) conditions. MEP amplitude, onset latency, and elicitation rate were extracted. Linear and generalized linear mixed models tested between- and within-group effects. **Results.** At baseline, patients showed a higher proportion of elicited MEPs in ECR compared to controls ($p = 0.045$), but no differences for SbM. Within patients, levodopa intake was associated with longer SbM onset latency ($p = 0.012$), reduced SbM elicitation rate ($p = 0.034$), and a robust increase in ECR amplitude ($p < 0.001$). **Conclusions.** These findings suggest a dissociation between control (ECR) and target (SbM) excitability, SbM results differently sensitive to dopaminergic state, supporting its potential linkage to broader dopaminergic network, comprising corticobulbar and motivational function that are disfunctional in PD.

Keywords: corticobulbar tract; submental EMG; Parkinson's disease; transcranial magnetic stimulation; motor evoked potentials; motivational arousal.

Introduction

The dopaminergic system plays a pivotal role in regulating motivation, reward, and motor control (Berridge, 2004; Salamone & Correa, 2012; Baik, 2013). Parkinson's disease (PD), characterized by degeneration of nigrostriatal dopaminergic neurons, is associated not only with motor deficits but also with motivational and affective disturbances (Lees et al., 2009; Speranza et al., 2021). Identifying neurophysiological markers that capture both motor and motivational dimensions of PD has the potential to improve clinical assessment and intervention strategies.

The corticobulbar (CB) tract, projecting from the primary motor cortex to orofacial and submental muscles, is implicated in speech, swallowing, and hedonic behaviors (Love & Webb, 1992; Ertekin, 2011). Recent studies suggest that corticobulbar excitability is modulated by motivationally relevant states, including craving, appetite, and disgust (Vicario et al., 2014; Vicario et al., 2020; Vicario et al., 2022). Specifically, motor evoked potentials (MEPs) recorded from the submental muscles (SbM) may provide an index of dopaminergic and motivational modulation (Sato et al., 2021).

In the present study, we tested whether SbM excitability differentiates PD patients from healthy controls, and whether SbM responses are altered by acute levodopa administration. We compared SbM recordings with a control muscle, the extensor carpi radialis (ECR), to distinguish corticobulbar-specific from more general motor effects.

Methods

Participants

A total of 27 participants were initially recruited: 13 patients with idiopathic Parkinson's disease (PD; 8 male, aged 50–69 years) and 14 healthy controls (7 male, age-matched). Patients were referred to by local neurology clinics. Four participants were not included in the statistical analyses: one PD patient (p003) withdrew during the first TMS session due to discomfort, one PD patient (p004) was recruited but did not attend any session, one PD patient (p013) completed data collection but was not included in the preliminary analyses, and one control participant (s001) was excluded due to absent MEPs responses. The final analyzed sample therefore comprised 10 PD patients (8 male, 2 female) and 13 healthy controls. All participants provided written informed consent. Exclusion criteria included atypical parkinsonism, severe motor complications, cognitive impairment, epilepsy, or contraindications to TMS (Wassermann, 1998; Rossi et al., 2021). A comprehensive overview of participants' demographic, clinical, and questionnaire scores data is reported in **Table 1** below.

Group	**N**	**M/F**	**UPDRS** (range score)	**Severity distribution** (only PD)	**DASS-Tot** (M ± SD)	**I-DAS** (M ± SD)	**MCQ-tot** (M ± SD)
PD patients	11	9/2	46.18 ± 21.5 (11–77)	3 Mild 5 Moderate 3 Severe	12.18 ± 11.2	46.09 ± 9.4	0.042 ± 0.069
Healthy controls	14	8/6	n.a.	n.a.	20.00 ± 13.9	53.62 ± 6.6	0.077 ± 0.093

Table 1. Demographic, clinical, and psychometric characteristics of participants.

UPDRS scores reflect Parkinson's disease severity, with values <32 = mild, 32–58 = moderate, and >58 = severe (Goetz et al., 2008). For the DASS-21, higher scores indicate greater negative affect (Bottesi et al., 2015). The I-DAS provides a dimensional measure of apathy, with higher scores reflects more apathy (Santangelo et al., 2017). The MCQ index represents scoring followed by Kaplan et al. (2016). Automated algorithms estimate the delay discounting parameter k from choices between smaller-sooner and larger-later rewards. Higher k values indicate steeper discounting (greater impulsivity), while lower k values indicate preference for delayed rewards (greater self-control). Subscale scores can be computed for short, medium, and long delays, as well as a global index (geometric mean).

Experimental Procedure

As depicted below in **Figure 1**, participants were seated comfortably with EMG electrodes placed over SbM and ECR (pre-gelled Ag/AgCl electrodes, 1 cm inter-electrode spacing with the ground electrode placed on the neck/clavicle bone to minimize artifacts from both muscles. TMS was delivered using a circular coil (Magstim Super Rapid) positioned tangentially over the scalp, approximately 2–4 cm lateral and slightly anterior to Cz, over the hemisphere contralateral to the recording side (Muellbacher

et al., 1994; Urban et al., 1998). Stimulation intensity was set to 100% of the maximum stimulator output, in line with established corticobulbar protocols. To facilitate MEP elicitation, participants were instructed to maintain a slight voluntary contraction of the tongue or floor-of-mouth muscles (Muellbacher et al., 1994). Each session consisted of 25 single pulses (7 s inter-stimulus interval). PD patients were tested in two conditions: OFF (after ≥12 h withdrawal from levodopa) and ON (1 h after intake). Controls were tested once (baseline, T0).

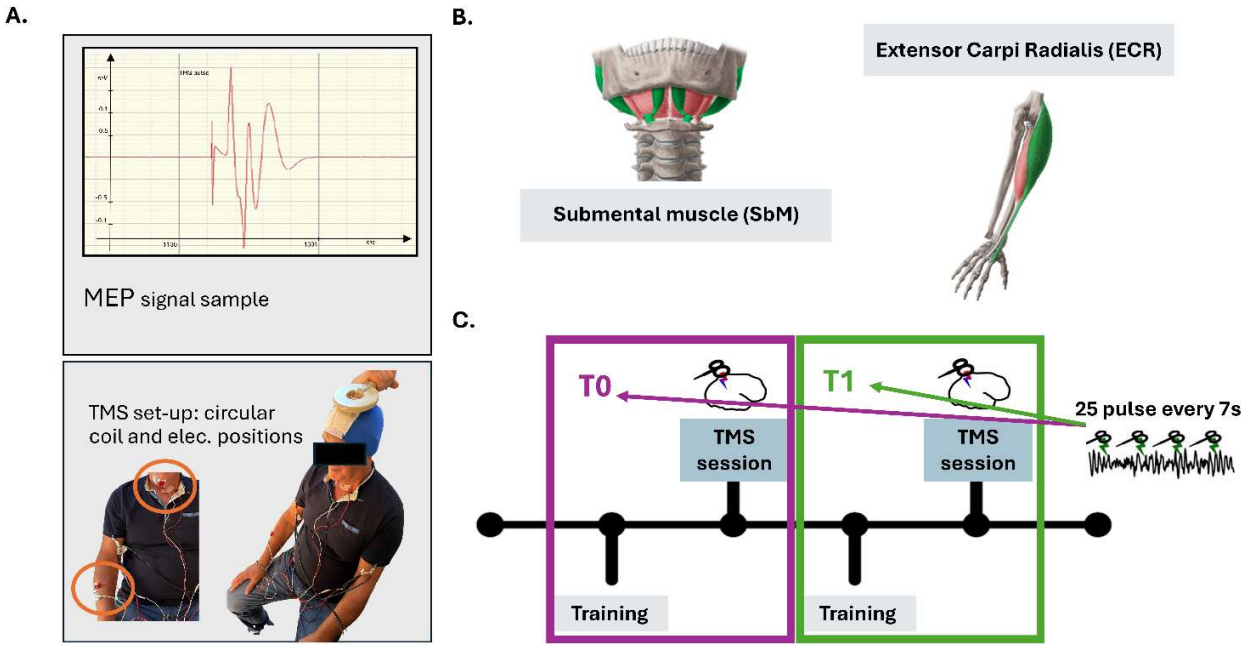

Figure 1. Experimental set-up and timeline.
A. Example set-up for recording motor evoked potentials (MEPs) from the submental muscle (SbM) following transcranial magnetic stimulation (TMS) over primary motor cortex (M1). Signals were acquired with BIOPAC AcqKnowledge 5.0. **B.** Visual representation of the target muscles: SbM and extensor carpi radialis (ECR). **C.** Timeline of experimental sessions. Each TMS session is preceded by a brief training phase to identify the optimal coil "hotspot" to elicit MEPs from both muscles simultaneously. Colored squares mark assessments before (T0, violet) and after levodopa (T1, green). Controls were tested only at T0; patients were tested at T0 and again 1 hour after levodopa at T1. Each TMS session comprised 25 pulses. Adapted from Ferraioli et al., 2025.

Data Processing and Analyses

MEPs were analyzed offline. Raw EMG (SBM and ECR), sampled at 10 kHz, was band-pass filtered 30–1000 Hz with zero-phase (forward–backward) filtering, plus a narrow notch at 50 Hz (and, when needed, at 100 Hz) to remove power-line noise. Filtering was applied to the full recordings prior to epoching around the TMS artifact for baseline subtraction and MEP feature detection. MEPs indices were extracted by customized MATLAB code, and the extracted measurements were MEP amplitude defined as peak-to-peak voltage; the Onset latency as the time from stimulus to the first deflection exceeding 2 SD above baseline; the Elicitation rate (%MEP) calculated as the proportion of trials with detectable responses. Data was analyzed in Rstudio using linear mixed-effects models (LMM) and generalized linear mixed models (GLMM), with factors Group (Patients vs Controls), Session (T0, T1), and Muscle (SbM, ECR). Specifically, LMMs were applied to continuous outcomes (amplitude,

latency). Fixed factors included Group (PD vs Controls), Session (T0 OFF vs T1 ON), and Muscle (SbM vs ECR), along with their interactions. A random intercept for participants accounted for individual variability. GLMMs with a binomial distribution and logit link function were used for the elicitation rate (%MEP), modeling the probability of obtaining a valid MEP as a function of the same fixed and random factors.

Results

Between Group Comparisons

At baseline (T0), patients showed significantly higher %MEP in ECR compared with controls ($p = 0.045$). No group differences were found for SbM in %MEP, onset latency, or amplitude (all $p > 0.09$). These results suggest altered excitability in a control muscle, while corticobulbar responses were not yet differentiated (**Figure 2**).

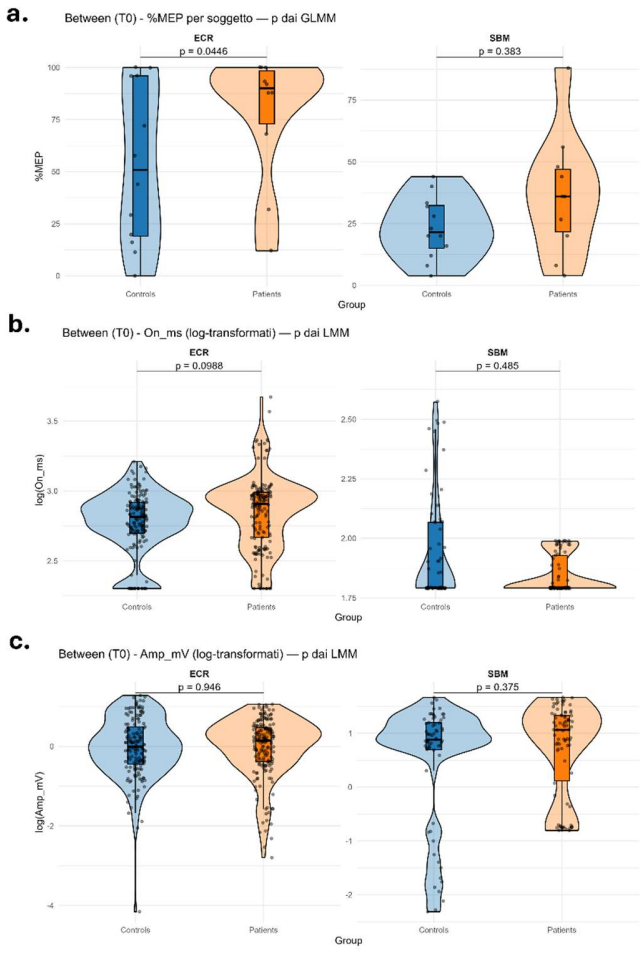

Figure 2. Between group comparisons at baseline (T0).
Violin plots show %MEP elicitation (a), onset latency (b), and amplitude (c) for controls (blue) and PD patients (orange), separately for ECR (left) and SbM (right). *p*-values are derived from mixed-effects models: GLMM (binomial, logit link) for %MEP, and LMM for latency and amplitude, testing between-group differences while accounting for participant-level variability.

Within-Patient Comparisons (OFF vs ON)

Levodopa administration induced specific changes in corticobulbar excitability. SbM onset latency was significantly increased in the ON condition compared to OFF ($p = 0.012$), while SbM %MEP decreased ($p = 0.034$). Conversely, ECR amplitude was markedly increased ($p < 0.001$), consistent with the general facilitatory effects of levodopa. SbM amplitude showed no significant change ($p = 0.953$) (**Figure 3**).

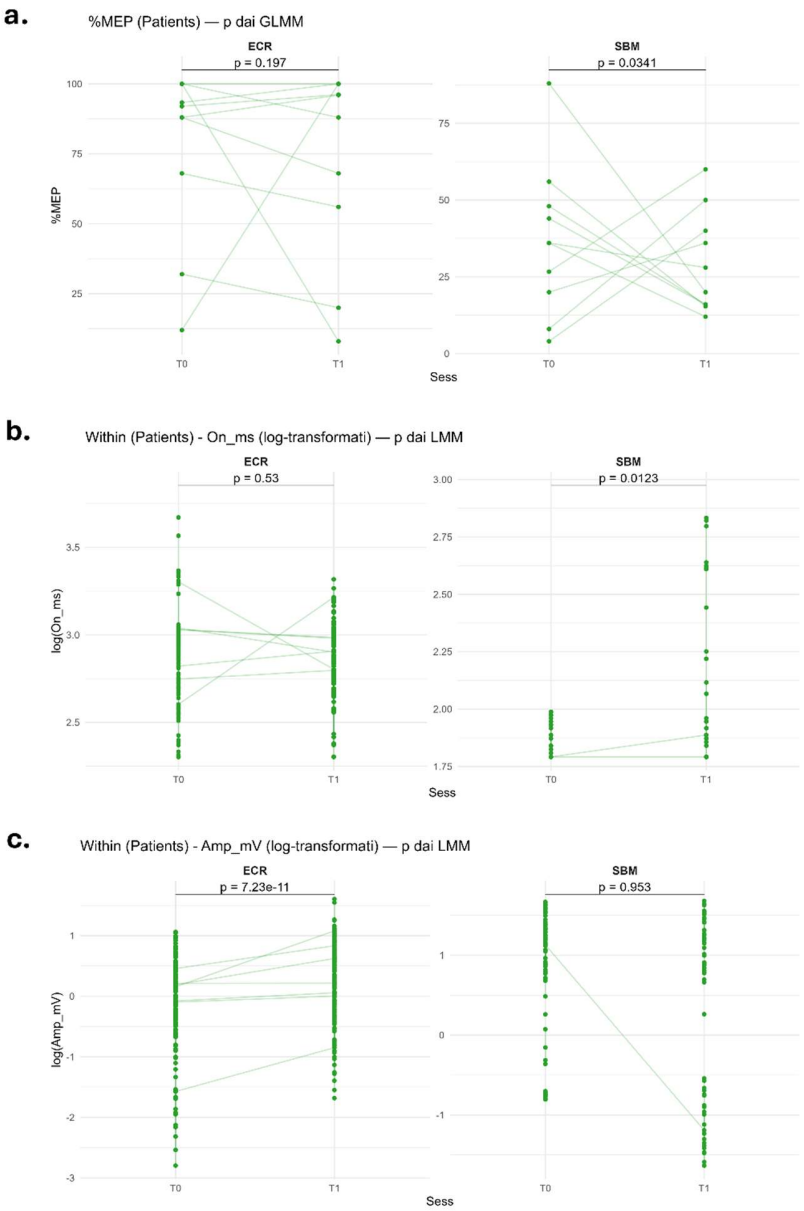

Figure 3. Within-patient comparisons (OFF vs ON levodopa).
Line plots show %MEP elicitation (a), onset latency (b), and amplitude (c) in PD patients across sessions T0 (OFF) and T1 (ON), for ECR (left) and SbM (right). *p*-values are derived from mixed-effects models: GLMM

(binomial) for %MEP, and LMM for latency and amplitude, evaluating within-patient contrasts across levodopa states.

Discussion

These preliminary explorations provide evidence that corticobulbar excitability, indexed by SbM MEPs, is sensitive to dopaminergic modulation in PD. While between-group comparisons did not reveal strong differences in SbM excitability, within-patient analyses showed clear effects of levodopa, particularly in onset latency and elicitation rate. These results complement prior studies showing that SbM activity is modulated by hedonic and motivational contexts (Sato et al., 2021; Ferraioli & Vicario, 2024) and support its role as a candidate biomarker of dopaminergic function.

This evidence provides novel insight into how dopaminergic modulation can differentially affect corticobulbar versus corticospinal excitability in PD. While ECR amplitude responded in a predictable facilitatory manner to levodopa (mirroring classic cortical excitability studies in PD; i.e., Jastrzębowska et al., 2019), the SbM muscle exhibited more nuanced modulation in latency and elicitation rate, suggestive of dopaminergic influence on corticobulbar pathways specifically. The absence of baseline SbM differences between PD and controls may reflect compensatory mechanisms or ceiling/floor effects in the excitability of bulbar circuits under chronic dopaminergic deficits, in line with findings that corticobulbar excitability can be relatively preserved in early disease stages (Michou et al., 2014).

Limitations. Our sample size, though adequate for preliminary within-subject contrasts, limits generalizability and power for more subtle between-group effects. Using only a circular coil at full stimulator output, without neuronavigation, may reduce spatial specificity and increase spread of stimulation. Surface EMG from SbM is vulnerable to crosstalk from adjacent muscles and variable electrode positioning. Additionally, we did not integrate swallowing function or dysphagia measures, despite evidence linking dopaminergic deficits to swallowing impairment in PD (Polychronis et al., 2019). Finally, we used a binary OFF/ON levodopa contrast; dose–response or temporal dynamics (e.g., at multiple post-dose intervals) were not assessed.

Future directions. The distinct patterns observed for SbM and ECR emphasize the need for region-specific analyses. Larger studies should stratify PD cohorts by disease severity, apathy, or presence of bulbar symptoms. Incorporating behavioral and clinical correlates (e.g. swallowing assessments, apathy scales) will help validate SbM MEPs as functional biomarkers. Longitudinal tracking could establish whether SbM excitability predicts motor or non-motor progression. Advanced TMS protocols (e.g. neuronavigated stimulation, paired-pulse or connectivity measures) may improve localization and allow disambiguation of cortical versus subcortical contributions. If SbM MEPs prove reliable, they could contribute to personalized monitoring of dopaminergic therapy or interventions targeting orofacial/motivational systems in PD.

Conclusions

Corticobulbar excitability, measured via SbM MEPs, is modulated by levodopa in PD patients. These findings highlight SbM activity as a potential proxy on dopaminergic state and motivational arousal, contributing to the development of neurophysiological tools for assessing PD-related motivational and motor dysfunction.

Author contributions

FF: Methodology, Writing – original draft, Writing – review & editing, Data curation, Formal analysis, Project administration. FT: Conceptualization, Funding acquisition, Supervision, Writing – review & editing. CK: Data collection. AL: Conceptualization, Funding acquisition, Writing – review & editing. VR: Data collection, Patient recruitment. CT: Data collection, Patient recruitment. SC: Conceptualization, Funding acquisition. SM: Project administration, Supervision, Validation, Writing – review & editing. CMP: Data curation, Writing – review & editing. AG: Supervision, Writing – review & editing. AF: Conceptualization, Writing – review & editing. FE: Supervision, Writing – review & editing. LT: Supervision, Writing – review & editing. CV: Conceptualization, Funding acquisition, Methodology, Supervision, Validation, Writing – original draft, Writing – review & editing.

Funding

Work supported and funded by: #NEXTGENERATIONEU (NGEU); the Ministry of University and Research (MUR); the National Recovery and Resilience Plan (NRRP); project MNESYS (PE0000006, to NT) – A Multiscale integrated approach to the study of the nervous system in health and disease (DN. 1553 11.10.2022).

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