Neuroscience 555 (2024) 178–183

Contents lists available at ScienceDirect

Neuroscience

journal homepage: www.elsevier.com/locate/nsc

Research Article

Galvanic vestibular stimulation modulates EEG markers of voluntary movement in Parkinson's disease

Shelley J. Duncan [a,b,*], Kamyla Marques [b], Jade Fawkes [b], Laura J. Smith [b,c], David T. Wilkinson [b]

[a] Department of Sport and Health, Solent University, Southampton SO14 OYN, UK
[b] School of Psychology, University of Kent, Canterbury, UK
[c] Wolfson Institute of Population Health, Queen Mary University of London, UK

ARTICLE INFO

Keywords:
Galvanic vestibular stimulation
Electrophysiological changes in electro-cortical activity
Mechanisms of action linked to voluntary movement
Idiopathic Parkinson's disease

ABSTRACT

We recently showed that vestibular stimulation can produce a long-lasting alleviation of motor features in Parkinson's disease. Here we investigated whether components of the motor related cortical response that are commonly compromised in Parkinson's – the Bereitschaftspotential and mu-rhythm event-related desynchronization – are modulated by concurrent, low frequency galvanic vestibular stimulation (GVS) during repetitive limb movement amongst 17 individuals with idiopathic Parkinson's disease. Relative to sham, GVS was favourably associated with higher amplitudes during the late and movement phases of the Bereitschaftspotential and with a more pronounced decrease in spectral power within the mu-rhythm range during finger-tapping. These data increase understanding of how GVS interacts with the preparation and execution of voluntary movement and give added impetus to explore its therapeutic effects on Parkinsonian motor features.

Introduction

The vestibular system helps control autonomic motor reflexes, as well as higher-order functions serving volitional movement and cognition (Smith and Zheng, 2013). Growing evidence indicates that this higher-order influence can be therapeutically harnessed via artificial stimulation of the vestibular periphery by thermal or galvanic current. One especially promising line of research indicates that vestibular stimulation can improve motor (and non-motor) outcomes in individuals who live with Parkinson's disease (PD). In a recent randomized, controlled, double-blinded trial, Wilkinson and colleagues (2019) showed that 8 weeks of daily caloric stimulation was associated with clinically important differences in the Movement Disorder Society Unified Parkinson's Disease Rating Scale (MDS-UPDRS) Part II (motor aspects of experiences of daily living) and Part III (motor exam), with statistically significant differences also observed in the Modified Schwab & England Activities of Daily Living Scale, the 10 m Self-Paced Walk and the Timed-Up-and-Go task. Active treatment was also associated with reductions in the MDS-UPDRS Part IV (motor complications), driven primarily by reduced dyskinesia. Other studies that have applied galvanic as opposed to caloric stimulation have reported improvement in balance (Samoudi et al., 2015), finger tapping, and gait control.

The physiological bases of these improved motor outcomes for people with PD during vestibular stimulation are gradually becoming clearer. Positron emission tomography and functional magnetic resonance imaging studies conducted in normative samples have uncovered widespread peri-sylvian excitation associated with vestibular stimulation (Della-Justina et al., 2015; Stephan et al., 2005), with studies conducted in people with PD showing increased cortical and cerebellar interactivity (Cai et al., 2018; Liu et al., 2021). Using Electroencephalography (EEG), Lee et al. (2019) also showed that the abnormal cortical coupling of theta, alpha and gamma frequency bands between motor cortex (M1), supplementary motor areas (SMA) and premotor areas is reduced in Parkinson's when sinusoidal GVS is applied. In a later study, the same group showed that beta-band activity in people with PD becomes more closely associated with motor vigour during sinusoidal GVS, as measured by the time taken to reach maximum force on a squeeze bulb task.

The above physiological findings have been taken to support the idea that vestibular stimulation, particularly when sinusoidal, exerts a powerful, potentially entraining, effect on the Parkinsonian brain (Kim et al., 2013; Smith, 2018). To further investigate this effect within the

* Corresponding author at: Department of Sport and Health, Solent University, Southampton SO14 OYN, UK.
E-mail address: shelley.duncan@solent.ac.uk (S.J. Duncan).

https://doi.org/10.1016/j.neuroscience.2024.07.048
Received 23 February 2024; Accepted 26 July 2024
Available online 27 July 2024

motor domain, the present study explored whether GVS modulates several prominent, well-characterised EEG signatures that are associated with voluntary movement control and are impaired in PD: the early/late Bereitschaftspotential (BP) and mu-rhythm event-related spectral power.

The BP is a component of the broadly defined movement-related cortical potential (MRCP) and represents activation across the motor, premotor and supplementary motor areas, as well as the somatosensory areas (Dick et al., 1989). The early phase of the BP is associated with voluntary movement preparation and is most evident over the vertex, while the late phase is associated with both preparation and execution and evident across areas M1, SMA and adjacent areas. The BP is typically investigated using simple, self-paced and self-initiated movements such as finger extensions or ankle dorsiflexion (Jahanshahi and Hallett, 2003). Dick et al. (1989) first reported that the early BP elicited by finger extensions is significantly diminished in PD compared to age-matched controls which has led to the view that the BP is a useful and relatively accessible marker of the movement initiation difficulties observed in PD. The motor potential (MP) follows the late BP and is associated with increasing cortical excitability particularly within the pyramidal tract neurons in the primary motor cortex and is taken to index activity occurring immediately before and during the onset of movement (Lucci et al., 2014).

In addition to these amplitude reductions in the BP, it is well-established that the normal suppression of mu-rhythm activity (8–12 Hz) that occurs across the sensory-motor cortex prior to voluntary movement (i.e. desynchronisation) is delayed in PD (Defebvre et al., 1998). The effect seems to reflect a deficit in sensorimotor integration more than motor preparation per se (Devos and Defebvre, 2006), and correlates with clinical motor features that include bradykinesia (Devos et al., 2004). Importantly, mu-rhythm event-related desynchronisation (ERD) is partly reversible with levodopa or deep brain stimulation (Devos and Defebvre, 2006), which raises the question as to whether GVS can exert the same effect.

To address these questions, here we describe a study that measured the effects of a single session of sub-sensory, sinusoidal GVS on the BP and mu-related spectral power in a group of individuals with mild to moderate idiopathic PD while they performed self-initiated finger and foot tapping tasks. We reasoned that if GVS could be shown to increase BP amplitudes and/or promote mu-rhythm ERD during finger and/or foot movements then subsequent studies could seek to identify clinical correlates.

Methods

Participants

Seventeen volunteers diagnosed with idiopathic PD were recruited from Parkinson's UK local branches, other community-based local organizations and from a database of former participants who consented to being re-contacted. All PD participants fulfilled the eligibility criteria with none having co-morbid neurological conditions, skin abrasions behind the ears, implanted electronic devices (e.g., deep brain stimulation, pacemakers, etc.), or in receipt of dopamine or apomorphine infusion therapy. All PD participants provided documented diagnostic evidence from their neurologist of idiopathic PD according to the UK PD society brain bank clinical diagnostic criteria. Participants remained on their stable anti-parkinsonian medication regime for the duration of the study. Other demographic and clinical characteristics of the sample are shown in Table 1 below. All procedures were conducted in accordance with the Declaration of Helsinki (https://www.wma.net) and were approved by the University of Kent's School of Psychology Research Ethics Committee. All procedures were carried out with the adequate understanding and written consent of the participants.

Table 1
Demographic and clinical characteristics of the sample.

Mean demographic scores (SD)	
Age	65 (7)
Gender	6 female, 11 male
Years since diagnosis	5.9 (4)
Hoehn Yahr	2.3 (1.0)
Mean baseline assessment scores (SD)	
MDS-UPDRS	
Part I	11.1 (5.4)
Part II	13.1 (5.5)
Part III	31.2 (11.7)
Part IV	5.6 (4.0)
MoCA	26.4 (2.6)
MiniBEST	21.8 (3.4)
HADS anxiety	5.5 (4.1)
HADS depression	4.6 (3.3)

Abbreviations: MDS-UPDRS = Movement Disorder Society Unified Parkinson's Disease Rating Scale; MoCA = Montreal Cognitive Assessment; MiniBest = Balance Evaluation Systems Test; HADS = Hospital Anxiety and Depression Scale.

Procedure

The study was conducted over three sessions on three separate days to help mitigate fatigue, and to ensure PD medication remained efficacious throughout testing. Each session was conducted 30–60 min following medication intake and lasted approximately two hours.

The initial participant session included administration of a neuropsychological test battery including the MDS-UPDRS, Montreal Cognitive Assessment, Hospital Anxiety and Depression scale, and the Mini-BEST. The two stimulation sessions occurred 1 week later, were separated by a period of 48 h and comprised either the finger or foot tapping task which included a block of both sham and active stimulation (see Fig. 1). The order of task and type of stimulation was counter-balanced across participants. The affected side was utilized for the motor tasks as determined by the scores on the motor examination of the MDS-UPDRS and the participant's self-report of their worst side. 12 participants presented with more right-sided impairment, and 5 presented with more left-sided motor impairment.

Galvanic vestibular stimulation (GVS)

A sinusoidal signal with an amplitude of 0.25–0.35 mA and frequency of 0.01 Hz was administered from a *Neuroconn DC Stimulator* via a pair of rubber, self-adhesive, disposable electrodes (5.1 cm × 10.2 cm; ComfortEase, Empi Inc.), with the cathode and anode electrodes positioned over the right and left mastoid, respectively. Our previous work has shown that the scalp-artifact induced by this waveform can be successfully removed via blind source separation without compromising measurement of the underlying MRCP (Duncan et al., 2022). We also note that very low stimulation frequencies can ameliorate motor features of PD (Wilkinson et al., 2016; Wilkinson et al., 2019), and that amplitudes within the 0.2–0.3 mA range elicit subtle oculomotor torsion and body-roll-tilt indicative of central vestibular activation yet are rarely perceived by participants so appropriate for blinding (Duncan et al., 2022; Wilkinson et al., 2012). Sham stimulation was administered (in separate blocks) in which the experimental choreography was identical to the active stimulation with the exceptions that the device was switched off and participants falsely informed that they were receiving stimulation.

Electromyography (EMG) recording and data analysis

EMG was employed to detect the onset of voluntary muscle activation. For full methodological details please see our previous paper, Duncan et al. (2022).

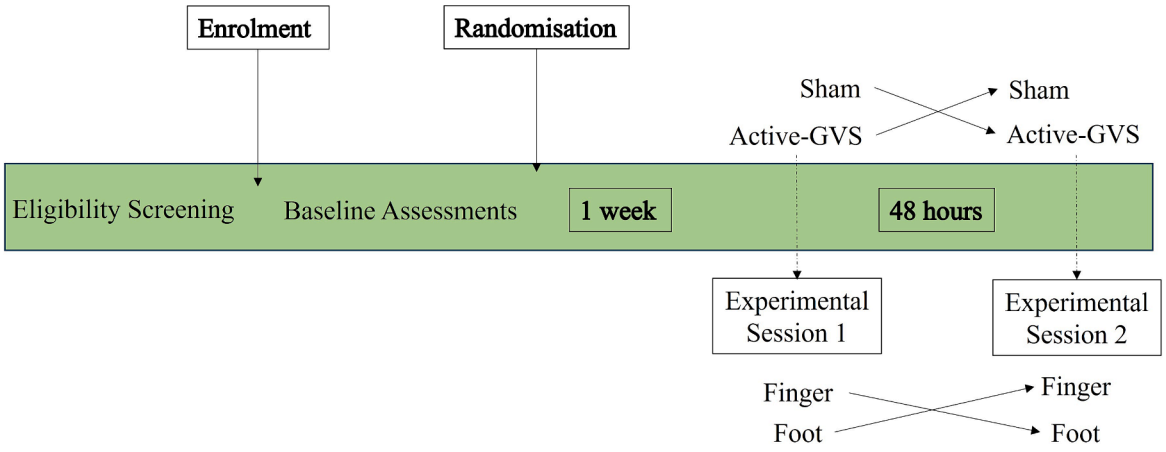

Fig. 1. Illustration of the participant protocol.

Electroencephalography (EEG) recording and data analysis

EEG was recorded using an eegoTMsports 64 (ANT Neuro, Enschede, Netherlands) amplifier (see Duncan et al., 2022 for full description of EEG methods). Trials for which there was not an EMG trace to accurately determine movement onset (i.e. lack of an abrupt and clear deviation from the steady state EMG) led to loss of 43 trials for finger-taps (10 GVS, 33 sham). Grand averages were calculated using 99 % ($n = 2311$) and 98 % ($n = 2393$) of trials for the GVS and Sham conditions, respectively. For foot movement data, 15 trials were lost (11 GVS, 4 Sham). Grand averages were calculated using 99 % ($n = 2430$) and 99 % ($n = 2389$) of trials for the GVS and Sham conditions, respectively.

MRCP waveforms were identified using a collapsed localizer average for the active and sham GVS conditions. Electrode sites over the bilateral and central motor cortex (C3, Cz, C4, CP1, CP2) were selected based on the largest voltage deflections identified in the grand collapsed averaged data and topographical maps. The BP component was divided into the subcomponents of the early (−1500 ms to −500 ms) and late (−500 ms to 0 ms) BP and the MP (100 ms to 250 ms) was measured after the onset of muscle activation.

Exploratory analysis of mu-rhythm ERD involved the transformation of the segmented EEG data into time–frequency continuous data with continuous wavelet transformation (CWT). A total epoch length of −2500 ms to 750 ms was employed to capture movement preparation and onset. A baseline correction using data from −2000 ms to −1500 ms prior to movement onset was conducted. Due to the larger epoch required for this type of analysis compared to our main event-related potential (ERP) analysis, for finger tapping 28 % ($n = 657$) of GVS trials and 34 % ($n = 818$) of Sham trials were lost. Spectral power was calculated using 72 % ($n = 1654$) and 66 % ($n = 1608$) of trials for the GVS and Sham conditions, respectively. For foot tapping, 27 % ($n = 656$) of GVS trials and 28 % ($n = 677$) of Sham trials were lost. Spectral power was calculated using 73 % ($n = 1774$) and 72 % ($n = 1712$) of trials for the GVS and Sham conditions, respectively. The 8 to 12 Hz (mu-rhythm) frequency band data was calculated for spectral power to analyse patterns of ERD. This phenomenon was calculated using 40 logarithmically spaced frequency bins using a number of wavelet cycles including 3, 5 and 7, with 5 cycles providing the best temporal and frequency resolution. The time–frequency decomposition was performed using the value sum of induced power over the bilateral and central motor cortex (C3, Cz, C4), (As reported in Pfurtscheller et al., 1996).

Mean amplitudes (ERP analysis) and spectral power (time–frequency analysis) were computed for each condition across the respective time windows. Measurements of the MRCP across central and parieto-central sites were interrogated using a 2 (Stimulation: sham vs. active GVS) x 5 (Electrode: C3 vs. Cz vs. C4 vs. CP1 vs. CP2) within-subjects repeated measures ANOVA. Measurements of time–frequency were interrogated using a 2 (Stimulation: sham vs. active GVS) x 3 (Electrode: C3 vs. Cz vs. C4) within-subjects repeated measures ANOVA. Violations of sphericity were adjusted with the Greenhouse Geisser Epsilon corrected, and significant main effects and interactions were interrogated using the Tukey test. ANOVA effect sizes were computed using the partial eta squared with magnitudes of $\eta_p^2 = 0.01$, $\eta_p^2 = 0.06$ and $\eta_p^2 = 0.14$ considered small, medium, and large, respectively (Miles and Shevlin, 2021). See Supplementary material for full summary of statistical results.

Testing procedure

The experiment was conducted in a quiet, temperature-controlled laboratory, where participants were seated upright in a comfortable chair. Once all equipment (EMG, EEG and GVS) was set up, participants were given verbal instructions for the finger and foot tapping tasks. In both the initial 5 min practice and data collection phases, participants were instructed to perform voluntary extensions of the index finger or foot, at their own pace, but with an interval between movements of 2–5 s (see Duncan et al., 2022).

EEG data were recorded across 3 blocks of 50 trials for each condition (active and Sham), with a 30 s interval between each block to attenuate the risk of fatigue. Experimenters silently counted movements as participants executed them and indicated when it was time to stop. Only movements that commenced from complete muscle relaxation (steady-state EMG) were considered acceptable.

Results

Finger

Early BP

No statistical effects reached significance (all F ratios < 0.6).

Late BP

The main effects of Stimulation and Electrode were reliable ($F = 6.51$; $p = 0.02$; $\eta_p^2 = 0.29$, $F = 6.28$; $p < 0.01$; $\eta_p^2 = 0.28$), but there was no interaction between Stimulation and Electrode ($F = 1.28$; $p = 0.29$; $\eta_p^2 = 0.07$). The main effect of Stimulation revealed greater mean amplitude in the active compared to the sham condition ($M = -.80$ μV, $SE = .16$ and $M = -.49$ μV, $SD = .16$ respectively) (see Figs. 2 and 3). Post-hoc testing indicated that the main effect of Electrode was driven by a difference between electrode Cz and all other electrodes.

MP

The main effects of Stimulation and Electrode were reliable ($F = 5.01$; $p = 0.04$; $\eta_p^2 = 0.24$, $F = 11.62$; $p < 0.01$; $\eta_p^2 = 0.42$), but there was no interaction between Stimulation and Electrode ($F = 1.47$; $p = 0.23$; $\eta_p^2 = 0.08$). The main effect of Stimulation revealed greater mean amplitude in the active compared to the sham condition ($M = -1.54$ μV,

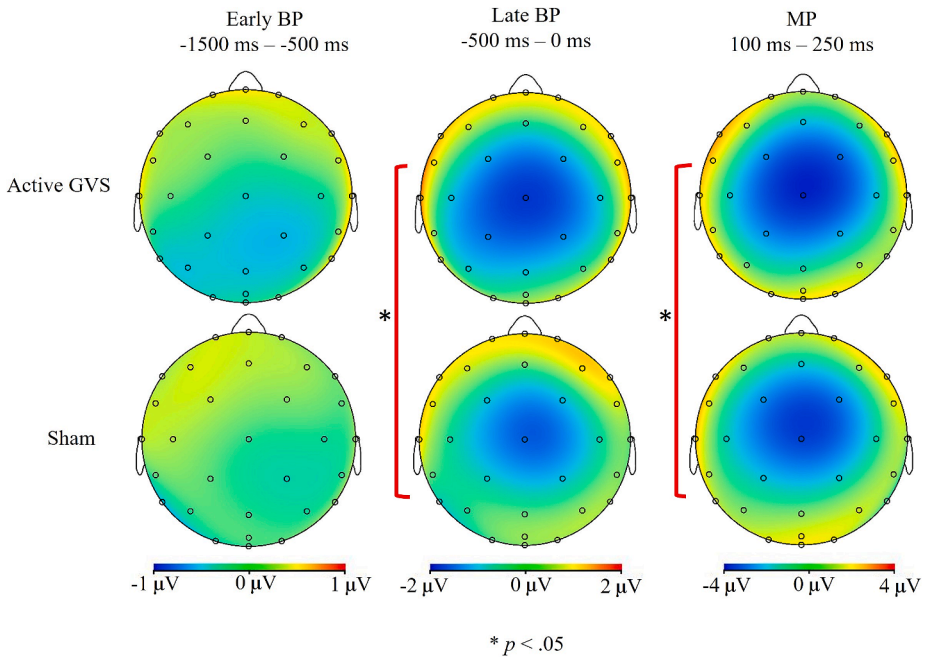

Fig. 2. Topographical distribution of finger MRCPs.

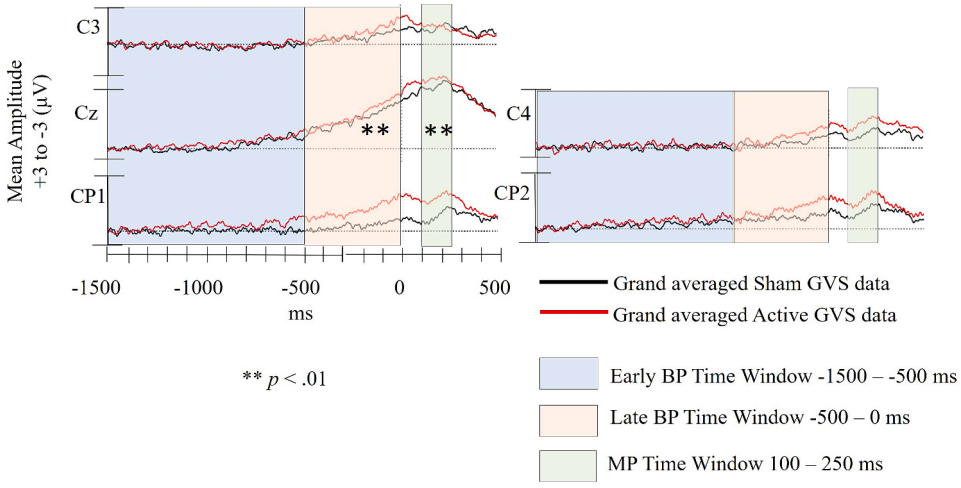

Fig. 3. MRCPs finger wavelets.

SE = .30 and *M* = −1.10 µV, *SD*=.29 respectively) (see Figs. 2 and 3). Post-hoc testing indicated that the main effect of Electrode was driven by a difference between electrode Cz and all other electrodes.

Mu-Rhythm ERD (Early BP)

There was a reliable effect of Stimulation ($F = 6.26$; $p = 0.02$; $\eta_p^2 = 0.28$), which was moderated by an interaction with Electrode ($F = 4.76$; $p = 0.02$; $\eta_p^2 = 0.23$), however, there was no effect of electrode ($F = 0.54$; $p = 0.59$; $\eta_p^2 = 0.03$). Post-hoc testing of the interaction between stimulation and electrode showed significant Mu-Rhythm ERD in electrode C4 (see Fig. 4). The main effect of Stimulation revealed a significant GVS-related decrease in spectral power within the period of the early BP.

Foot

Early BP

No statistical effects reached significance (all *F* ratios < 0.6).

Late BP

The main effect of Electrode reached statistical significance for the late BP ($F = 5.16$; $p = 0.01$; $\eta_p^2 = 0.24$). Post-hoc testing indicated that this main effect was driven by a difference between electrodes Cz and C4. No other statistical effects reached significance (all *F* ratios < 0.6).

MP

The main effect of Electrode reached statistical significance for the MP ($F = 12.34$; $p < 0.01$; $\eta_p^2 = 0.44$). Post-hoc testing indicated that this main effect was driven by a difference between electrode Cz and all other electrodes. No other statistical effects reached significance (all *F* ratios < 0.6).

Mu-Rhythm ERD (Late BP)

Analysis of mu-rhythm ERD within the period of the late BP yielded a significant interaction between Stimulation and Electrode ($F = 4.51$; $p = 0.04$; $\eta_p^2 = 0.22$), but this did not survive correction for multiple comparisons during post hoc analysis. Neither the main effect of Stimulation

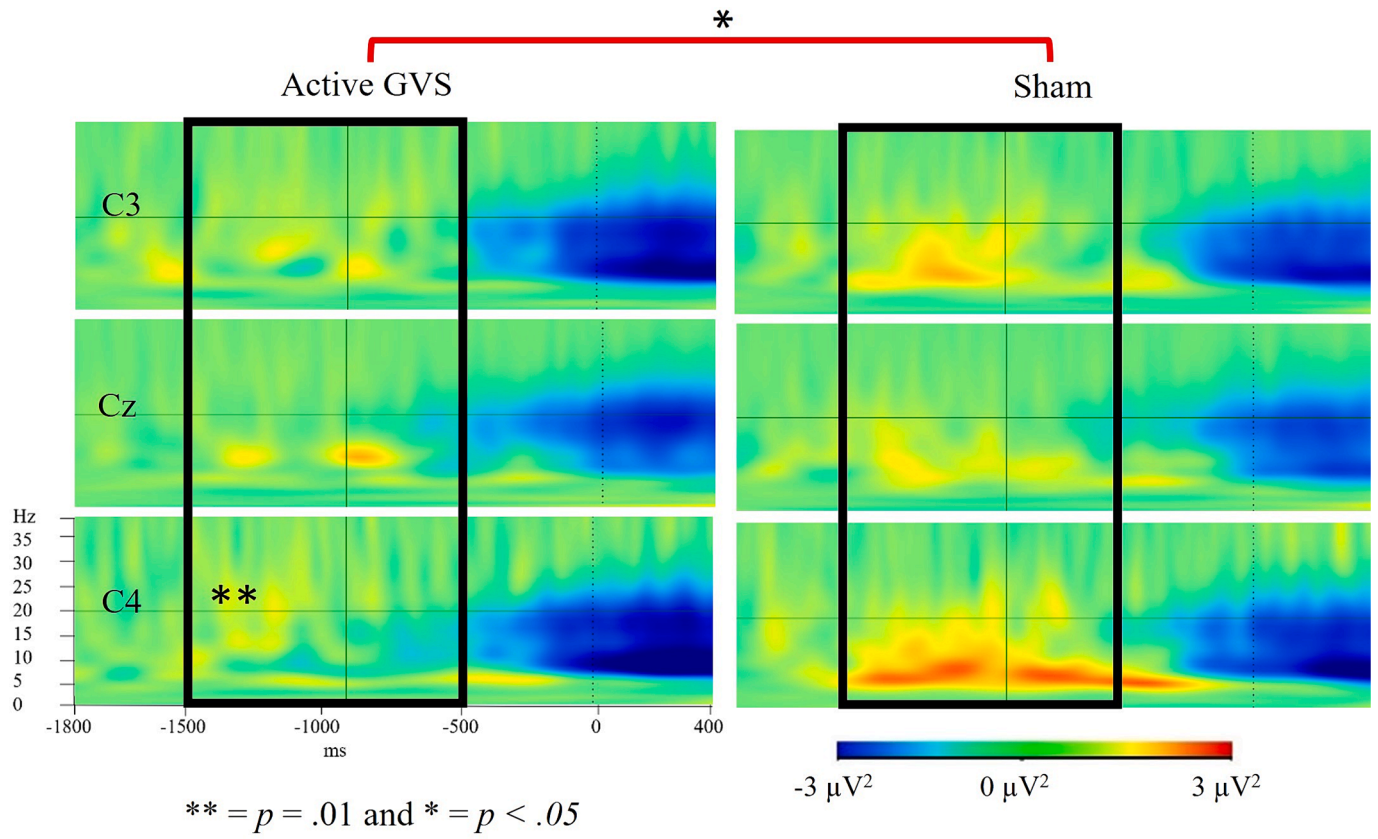

Fig. 4. Time-frequency spectral power for finger movement, highlighting GVS-related mu-rhythm event-related desynchronization within the time-period of the early BP.

nor Electrode ($F = 1.20$; $p = 0.29$; $\eta_p^2 = 0.07$ and $F = .2.10$; $p = 0.16$; $\eta_p^2 = 0.12$ respectively) were reliable.

Discussion

Previous EEG studies have shown that GVS can reduce abnormal cross-frequency coupling and improve beta-band responsivity in people with PD (Lee et al., 2019). The aim of the present study was to identify additional mechanisms of action that may help explain the beneficial effects of vestibular stimulation on motor features of PD. To this end, during finger-tapping we observed a GVS-related enhancement in mean amplitude of the late BP and the MP over bilateral somatosensory cortex. We also observed an enhanced GVS-related mu-rhythm ERD within the period of the early BP over right pre/post central gyrus (i.e. electrode C4). This localisation to electrode C4 most likely reflects compensatory processes in ipsilateral motor cortex given that the majority of participants used their right hand to respond which was also the most commonly affected (Wu et al., 2015). In support of this interpretation, an additional analysis in which C3 and C4 responses were re-categorised as either contralateral or ipsilateral to the affected limb produced the same statistical outcomes (see Supplementary material).

The observed increase in mean amplitude of the late BP and MP is suggestive of greater cortical drive and is associated the reorganisation/amplification of temporal dynamics during both the preparation and execution of voluntary movement (Dick et al., 1989). Coupled with the observed reduction of mu-rhythm within the period of the early BP (which resembles the attenuation of abnormal magnetoencephalographic-electroencephalographic activity reported by Salenius et al. (2002) after administration of L-Dopa) the current findings suggest a move away from neuronal idling towards network coherence during GVS. This 'functional facilitation' gives reason to speculate whether non-native frequencies discharged by GVS can, potentially via cross-frequency coupling, drive large-scale neural entrainment across sensory-motor networks and help mitigate dysfunctional brain rhythms. In this context, GVS (and other forms of sensory neuro-modulation) may be an especially useful stimulation modality for PD compared to other neuro-modulatory approaches, such as transcranial direct/alternating current and transcranial magnetic stimulation, given its bottom-up nature (which enables endogenous, diffuse activation of the central pathways via peripheral stimulation of the ascending vestibular pathways) and less dependence on a priori knowledge about which part of scalp to stimulate and waveform amplitude/frequency to discharge.

The absence of further electrophysiological effects in the foot data is difficult to explain. One possibility is that the somatotopic mapping of the foot to the mesial surface of the pre- and post-central gyrus makes EEG activation generally harder to capture compared to the hand which typically maps to the lateral surface and occupies a larger surface area (Brunia and Van Den Bosch, 1984; Pfurtscheller et al., 1997). In line with this anatomical distribution, Pfurtscheller et al. (1997) found that while mu-related ERD for hand movement was found in nearly all their participants, it was generally harder to find for foot movements and only evident in a subset of participants. With respect to PD, recent research found that the MRCP elicited by lower limb extremity movement shared considerable similarity between people with and without PD (Karimi et al., 2021) which, in the present study, may help explain the lack of variability between the active and sham conditions.

Future research will now need to determine whether the favourable modulation observed here is accompanied by a reduction in clinical motor features, most notably those associated with voluntary limb movement. It will also be important to recruit an age-matched control group to determine whether the brain responses that are magnified by GVS are specific to PD or more universal in appearance. In addition, future research would benefit from including a larger sample size to

enable analysis of the 3-way interactions between dominant hand, affected side, and GVS and, more widely, to assess the generality of our results across disease and demographic sub-types. For now, we wish to highlight the discovery of three novel biological markers of potential effect that, together with the excellent safety profile of GVS, the ready availability of off-the-shelf stimulation devices, and a regulatory pathway that is cheaper and quicker than that required for pharmaceutical interventions, strengthen the case for further translational development and direct comparison with other modes of potentially efficacious neuro-stimulation.

Author contribution statement

S.D, K.M, J.F, L.S & D.W. were involved in the conceptualisation of the study design. K.M, & J.F. participated in the data collection. S.D, & DW wrote the main manuscript text. S.D prepared all figures and analysed the results. S.D, L.S, & D.W. participated in the manuscript review.

CRediT authorship contribution statement

Shelley J. Duncan: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Methodology, Investigation, Formal analysis, Conceptualization. **Kamyla Marques:** Project administration, Investigation, Data curation. **Jade Fawkes:** Project administration, Investigation, Data curation. **Laura J. Smith:** Writing – review & editing, Supervision, Project administration. **David T. Wilkinson:** Writing – review & editing, Validation, Supervision, Resources, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neuroscience.2024.07.048.

References

Brunia, C.H.M., Van Den Bosch, W.E.J., 1984. Movement-related slow potentials I. A contrast between finger and foot movements in right-handed subjects. Elsevier BV.. Doi:10.1016/0013-4694(84)90087-7.

Cai, Lee, Ba, Garg, Kim, Liu, et al. 2018. *Galvanic vestibular stimulation (GVS) augments deficient pedunculopontine nucleus (PPN) connectivity in mild Parkinson's disease: fMRI effects of different stimuli* Front. Neurosci. Media SA. doi:10.3389/fnins.2018.00101.

Defebvre, L., Bourriez, J.L., Derambure, P., Duhamel, A., Guieu, J.D., Destee, A., 1998. Influence of chronic administration of L-DOPA on event-related desynchronization of mu rhythm preceding voluntary movement in Parkinson's disease. Electroencephalogr Clin Neurophysiol. https://doi.org/10.1016/s0924-980x(97)00085-4.

Della-Justina, H.M., Gamba, H.R., Lukasova, K., Nucci-da-Silva, M.P., Winkler, M., Amaro Jr, E., 2015. Interaction of brain areas of visual and vestibular simultaneous activity with fMRI. Exp. Brain Res. 233 (1), 237–252. https://doi.org/10.1007/s00221-014-4107-6.

Devos, Defebvre. 2006. *Effect of deep brain stimulation and l-DOPA on electrocortical rhythms related to movement in Parkinson's disease.* Prog. Brain Res. doi:10.1016/s0079-6123(06)59022-3.

Devos, D., Labyt, E., Derambure, P., Bourriez, J. L., Cassim, F., Reyns, N., et al. 2004. Subthalamic nucleus stimulation modulates motor cortex oscillatory activity in Parkinson's disease. *Brain (London, England: 1878), 127*(2), 408-419. doi:10.1093/brain/awh053.

Dick, J. P. R., Rothwell, J. C., Day, B. L., Cantello, R., Buruma, O., Gioux, M., et al. 1989. The bereitschaftspotential is abnormal in Parkinson's disease. *Brain (London, England: 1878), 112*(1), 233-244. doi:10.1093/brain/112.1.233.

Duncan, Kamyla, Ferguson, Wilkinson. 2022. Extraction of the GVS electrical artifact from EEG recordings of the motor related cortical potential. *J. Neurosci. Methods, 368*, 109459. doi:10.1016/j.jneumeth.2021.109459.

Jahanshahi, M., Hallett, M., 2003. The Bereitschaftspotential: Movement-Related Cortical Potentials. Hogrefe & Huber Publishers. Doi:10.1027/0269-8803.17.3.135.

Karimi, F., Niu, J., Gouweleeuw, K., Almeida, Q., Jiang, N., 2021. Movement-related EEG Signatures Associated with Freezing of Gait in Parkinson's Disease: An Integrative Analysis. Oxford University Press (OUP). doi:10.1093/braincomms/fcab277.

Kim, Yogendrakumar, Chiang, Ty, Wang, McKeown. 2013. Noisy galvanic vestibular stimulation modulates the amplitude of EEG synchrony patterns. *PLoS ONE, 8*(7), e69055. doi:10.1371/journal.pone.0069055.

Lee, Liu, Wang, Mckeown. 2019. *Abnormal phase coupling in Parkinson's disease and normalization effects of subthreshold vestibular stimulation.* Front. Neurosci. Media SA. doi:10.3389/fnhum.2019.00118.

Liu, Bi, Li, Lee, Cai, Mi, et al. 2021. Galvanic vestibular stimulation improves subnetwork interactions in Parkinson's disease. *J. Healthc. Eng., 2021*, 6632394-11. doi:10.1155/2021/6632394.

Lucci, G., Berchicci, M., Spinelli, D., Di Russo, F., 2014. The motor preparation of directionally incompatible movements. NeuroImage (Orlando, Fla.) 91, 33–42. https://doi.org/10.1016/j.neuroimage.2014.01.013.

Miles, J., Shevlin, M., 2021. Applying Regression and Correlation: A Guide for Students and Researchers. Sage.

Pfurtscheller, G., Stancák, A., Neuper, C.H. 1996. *Event-related synchronization (ERS) in the alpha band — an electrophysiological correlate of cortical idling: A review.* Electroencephalogr. Clin. Neurophysiol., Elsevier BV. doi:10.1016/s0167-8760(96)00066-9.

Pfurtscheller, G., Neuper, C., Andrew, C. 1997. *Foot and hand area mu rhythms.* Int. J. Psychophysiol., Elsevier Science.

Salenius, S., Avikainen, S., Kaakkola, S., Hari, R., & Brown, P. (2002). Defective cortical drive to muscle in parkinson's disease and its improvement with levodopa. Brain *(London, England : 1878), 125*(Pt 3), 491–500. doi:10.1093/brain/awf042.

Samoudi, G., Jivegård, M., Mulavara, A.P., Bergquist, F., 2015. Effects of stochastic vestibular galvanic stimulation and LDOPA on balance and motor symptoms in patients with Parkinson's disease. Brain Stimul. 8 (3), 474–480. https://doi.org/10.1016/j.brs.2014.11.019.

Smith, Zheng. 2013. From ear to uncertainty: Vestibular contributions to cognitive function. *Front. Integr. Neurosci., 7*, 84. doi:10.3389/fnint.2013.00084.

Smith, P.F., 2018. Vestibular functions and Parkinson's disease. Front. Neurol. 9 https://doi.org/10.3389/fneur.2018.01085.

Stephan, T., Deutschländer, A., Nolte, A., Schneider, E., Wiesmann, M., Brandt, T., Dieterich, M., 2005. Functional MRI of galvanic vestibular stimulation with alternating currents at different frequencies. NeuroImage (Orlando, Fla.) 26 (3), 721–732. https://doi.org/10.1016/j.neuroimage.2005.02.049.

Wilkinson, D., Ferguson, H.J., Worley, A., 2012. Galvanic vestibular stimulation modulates the electrophysiological response during face processing. Vis. Neurosci. 29 (4–5), 255–262. https://doi.org/10.1017/S0952523812000235.

Wilkinson, D., Podlewska, A., Sakel, M., 2016. A durable gain in motor and non-motor symptoms of parkinson's disease following repeated caloric vestibular stimulation: A single-case study. NeuroRehabilitation (Reading, Mass.) 38 (2), 179–182. https://doi.org/10.3233/NRE-161308.

Wilkinson, D., Podlewska, A., Banducci, S.E., Pellat-Higgins, T., Slade, M., Bodani, M., Ade, K.K., 2019. Caloric vestibular stimulation for the management of motor and non-motor symptoms in Parkinson's disease. Parkinsonism Relat. Disord. 65, 261–266. https://doi.org/10.1016/j.parkreldis.2019.05.031.

Wu, T., Hou, Y., Hallett, M., Zhang, J., Chan, P., 2015. Lateralization of brain activity pattern during unilateral movement in Parkinson's disease. Hum. Brain Mapp. 36 (5), 1878–1891. https://doi.org/10.1002/hbm.22743.