Healthcare Innovations
How practice has changed

HERSTON HEALTH PRECINCT SYMPOSIUM 2021

DISC-0059

6 - 10 September 2021
Education Centre
RBWH

EMOTIONAL INHIBITION IN PARKINSON'S DISEASE: A HD-EEG ERP STUDY

Peter Su[1], David Hennessy[1,2], Tiffany R. Au[1], Anthony J. Angwin[3], Kartik Iyer[1], Jihyun Yang[1], David A. Copland[1,3], John D. O'Sullivan[1,4], Nadeeka N Dissanayaka[1, 2, 4]

[1]UQ Centre for Clinical Research, Faculty of Medicine, The University of Queensland, Herston, QLD, Australia | [2]School of Psychology, University of Queensland, St Lucia, QLD, Australia | School of Health and Rehabilitation Sciences, University of Queensland, St Lucia, QLD, Australia | [4]Department of Neurology, Royal Brisbane & Women's Hospital, Herston, QLD, Australia

n.dissanayaka@uq.edu.au @NadeekaDissa

BACKGROUND

Deficits across emotion, memory, attention, and executive function are common in people with Parkinson's disease (PwP). Response inhibition is an element of executive function that has been previously probed in PwP. The Go/Nogo paradigm, used in conjunction with neuroimaging, is used to probe response inhibition to perceptual stimuli [1]. It involves the presentation of target cues ("Go") to which participants are instructed to respond as quickly as possible, and distractor cues ("Nogo") to which participants are instructed not to respond. The Nogo-N2 and Nogo-P3 are deflections of amplitude in event-related potentials (ERPs), and are thought to index cognitive effort in discrepancy recognition and stimulus evaluation respectively [2,3].

AIMS: To examine ERPs elicited from an affective Go/Nogo task and assess its use as a means of tracking Parkinson's disease mediated changes in cognition during emotional processing.

METHODS: *Participants*

Twenty-two PwP and 13 healthy controls completed the study. PwP were recruited from neurology outpatient clinics, and existing databases. All participants were right handed and were fluent English speakers. Written informed consent was obtained from all participants prior to commencing the study.

METHODS: *ERP*

ERPs elicited for target word stimuli were recorded using a high density Hydrocel Geodesic Sensor Net with 128 channels. Net station was used for EEG data acquisition and analysis. Time windows chosen for the N2 and P3 ERP time windows were 150-350ms and 350-550ms respectively. Analysis was performed on fronto-central cluster. ERP amplitude and latency were examined as per previous study [1].

METHODS: *Statistical analysis*

Repeated measures ANOVA models

- **Go trials only:** Target Valence X Group
- **Nogo trials only:** Target Valence X Group
- Trial type X Group

METHODS: *Affective Go/Nogo Task*

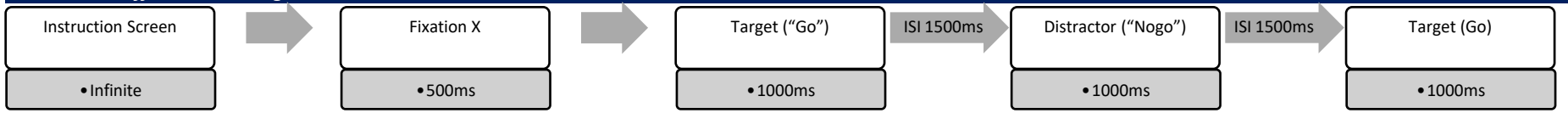

Figure 1: *Participants were tested using a visual affective Go/Nogo task. Affectively-valenced (negative, neutral and positive) words presented as target and distractor cues whilst ERPs were recorded.*

RESULTS: PwP compared to healthy older adults

Behavioural findings: *(i)* **Reaction time:** Slower across all conditions ($F_{1, 29} = 9.30$, $p = .005$); *(ii)* **Accuracy:** Less accurate in both Go and Nogo trials ($F_{1, 29} = 5.70$, $p = .024$)

ERP findings: *(i)* **Peak N2 amplitudes:** Higher across all valence conditions in both Go ($F_{1, 33} = 6.25$, $p = .018$) *(Figure 3A)* and Nogo trials ($F_{1, 33} = 5.92$, $p = .021$) *(Figure 3B).* *(ii)* **Peak P3 amplitude:** Lower across all valence conditions in both Go ($F_{1, 33} = 4.22$, $p = .048$) and Nogo trials ($F_{1, 33} = 5.38$, $p = .027$). *(iii)* **P3 latency:** Slower across all valence conditions in both Go ($F_{1, 33} = 6.0$, $p = .019$) and Nogo trials ($F_{1, 33} = 4.68$, $p = .038$). (Figure 2)

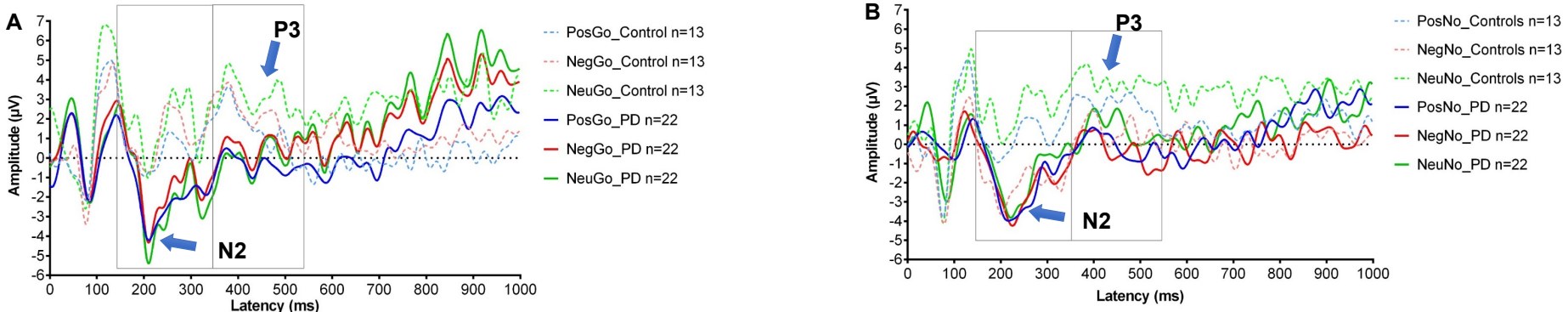

Figure 2. *ERP waveform comparison of PwP and controls. Valence in (A) Go-trials and (B) Nogo-trials*

CONCLUSION

Although emotional valence did not impact ERPs in this study cohort, differences in amplitude and latency of N2 and P3 were observed in PwP attempting the affective Go/Nogo task. These could serve as potential biomarkers for both Parkinson's disease progression and diagnosis. The results also indicate new directions for future work, such as characterising contributions of Parkinson's disease comorbid conditions like impulsivity and depression, and further development of the task for fine-grained diagnostic utility.

REFERENCES

[1] Chiu P.H et al. (2008). Neuroimage 42:988-997. ***[2]*** Kirmizi-Alsan E. et al. (2006). Brain Res 1104:114-128. ***[3]*** Bruin K et al. (2001) Clin Neurophysiol 112:1660-1671.

ACKNOWLEDGEMENTS

We thank all study participants. Dr Nadeeka Dissanayaka is supported by the Lions Medical Research Foundation Research Fellowship. This project is supported by the RBWH competitive research project grant 2014.