

Neurophysiological Changes Underlying Inhibitory Control in Mild Age-Related Hearing Loss

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Abstract— Emerging evidence suggests behavioral alterations in inhibitory control in older adults with mild Age-Related Hearing Loss (ARHL). Whether there are underlying alterations in the neurophysiological mechanisms linked to these behavioral changes remains unexplored. The current study examined Event-Related Potentials (ERPs) and Event-Related Spectral Perturbations (ERSPs) linked to two Go/NoGo tasks (Single-Car/Object-Animal) in 17 older adults with unaided mild ARHL and 25 normal hearing controls. Group differences in N2 and P3 (ERPs) latency and amplitude and theta and alpha (ERSPs) power were examined in addition to their association with speech-in-noise recognition. Findings revealed differences in ERPs and ERSPs for the NoGo versus Go trials in the two groups. The mild ARHL group showed longer NoGo N2 latency relative to Go N2 latency on the Single-Car task, but similar findings were not observed within the control group. The control group showed higher P3 amplitude and greater alpha desynchronization for NoGo versus Go trials on the Object-Animal task, but this differentiation was lacking in the hearing loss group. These findings suggest alterations in neurophysiological mechanisms underlying inhibitory control in unaided mild ARHL. Additionally, poorer recognition of speech-in-noise in the hearing loss group was related to higher P3 amplitude for Go trials on the Object-Animal task, with a similar trend observed for NoGo trials, suggesting that those with worse central hearing exert greater neural effort on inhibitory control tasks. The study findings add to the literature on the impact of ARHL on cognition and its association to changes in complex listening functions.

Keywords—age-related hearing loss; inhibitory control; event-related potentials; event-related spectral perturbations; Go/NoGo tasks.

I. INTRODUCTION

Inhibitory control is a cognitive control process, which allows us to suppress irrelevant information/responses in order to attend to relevant information [1]. It is often used in common listening situations, such as understanding Speech in Noise (SiN). For instance, in busy restaurants, inhibitory control allows one to suppress background noise and focus on their relevant conversation. Age-Related Hearing Loss (ARHL), a globally prevalent condition, affects various listening functions, including understanding SiN [2][3].

Growing evidence shows behavioral alterations in inhibitory control in older adults with ARHL relative to age- and education-matched Normal Hearing (NH) controls [4][5],

even in those with mild severity of hearing loss on common inhibitory control tasks such as Stroop and Go/NoGo [4]. Theoretical postulations linking hearing loss and cognition have long suggested underlying neural changes in individuals with ARHL [6]. It is plausible that neurophysiological changes underlie the overt inhibitory control changes in older adults with mild ARHL; however, this has not been examined. Event-Related Electroencephalography (EEG), which taps into real-time neural processing linked to cognitive processes, would be useful in this context. Both Event-Related Potentials (ERPs) and Event-Related Spectral Perturbations (ERSPs)/event-related oscillations measures, which are derived from EEG, might offer valuable insights. ERPs capture temporal aspects of the EEG signal, whereas ERSPs delineate the spectral and temporal aspects. On inhibitory control tasks, ERP studies have typically examined N2 (negative deflection at ~200 ms) and P3 (positive deflection at ~300 ms) components, while ERSP studies have examined theta (4-7 Hz) and alpha (8-12 Hz) power [7][8].

This study primarily examined differences in N2 and P3 amplitude and latency and alpha and theta power corresponding to two Go/NoGo tasks between older adults with unaided mild ARHL relative to NH controls. Our secondary aim was to examine associations between SiN recognition and ERPs/ERSPs corresponding to Go/NoGo tasks. Findings from this study will establish whether neurocognitive alterations in inhibitory control occur in mild ARHL. This is critical given that ARHL has been considered one of only 12 modifiable risk factors for dementia [9]. Markers that can assist in the identification of neurocognitive alterations will be instrumental in early detection and timely intervention for these individuals.

The rest of this paper is described as follows. Section II describes the methods. Section III describes the main results and discussion. Section IV concludes the article. The acknowledgment closes the article.

II. METHODS

A. Participants

Participants included 17 older adults with unaided mild ARHL and 25 NH controls with comparable age and education. Those with a history of neurological and psychological disorders, and other known etiologies of hearing loss were excluded.

B. Tasks and Procedure

All participants completed a comprehensive audiological examination, including the Quick Speech-in-Noise (QuickSIN) test to examine SiN recognition. Inhibitory control was examined using two in-house developed Go/NoGo tasks, Single-Car, and Object-Animal tasks [10]. The simpler Single-Car task was a basic categorization task, where participants were shown line drawings of a car (160 trials) and a dog (40 trials) and were required to push a button to the stimuli of cars (Go trials) but withhold button push to stimuli of dogs (NoGo trials). For the more complex Object-Animal task, a superordinate categorization task, participants saw multiple exemplars of objects (160 trials) and animals (40 trials) and were required to push a button to stimuli of objects but withhold to stimuli of animals. Reaction time and accuracy were obtained.

C. EEG Data Collection and Processing

EEG was collected while participants performed the two Go/NoGo tasks using a 64-electrode Neuroscan QuikCap. Collected data were pre-processed offline with noisy data and poorly functioning electrodes removed. Subsequently data were epoched from -500 to 0 ms. For ERP analyses, baseline correction was done from -500 to 0 ms, and ERP averages were created separately for trial type (Go/NoGo) and task (Single-Car/Object-Animal). Guided by previous research and visual inspection, N2 component between 150-300 ms was extracted across an average of frontal (F1, Fz, F2) and frontocentral (FC1, FCz, FC2) electrodes [7]. P3 was extracted between 250-600 ms at an average of frontocentral (Fc1, Fcz, Fc2), central (C1, Cz, C2), and centroparietal (CP1, CPz, CP2) [7]. Latency and mean amplitudes were used as measures. For ERSP analyses, EEGLAB toolbox with *newtimef.m* function [11] was used and baseline correction was conducted using a gain model [12]. Theta and alpha power were obtained across five electrode clusters: frontal (F1, Fz, F2), frontocentral (FC1, FCz, FC2), central (C1, Cz, C2), centroparietal (CP1, CPz, CP2), and parietal (P1, Pz, P2).

D. Statistical Analyses

All data were analyzed using IBM SPSS Statistics (Version 26). General Linear Models (GLMs) for analyses, with group (ARHL/NH) as a between-subject factor and trial type (Go/NoGo) as a within-subject factor. Alpha was fixed at 0.05, and in the case of significant group-by-trial interactions, post hoc comparisons were carried out. Bonferroni corrections were used to correct for multiple comparisons. Given the small sample size, separate analyses were conducted for the simpler Single-Car task and the complex Object-Animal task.

III. MAIN RESULTS AND DISCUSSION

Behavioral data showed evidence of changes in inhibitory control in individuals with mild ARHL. This was observed on post-hoc comparisons, with lower accuracy on NoGo versus

Go trials within the ARHL group on the Single-Car task ($p < 0.001$), although similar differences were not observed within the NH group ($p > 0.05$). These findings suggest that the mild ARHL group experienced challenges in withholding a prepotent response. Furthermore, this was noted during perceptual processing, since the findings were observed on the basic categorization task, Single-Car, which mainly consisted of perceptual stimuli.

EEG findings also revealed differential processing of NoGo versus Go trials between the two groups. These findings were observed on post-hoc comparisons. Particularly, longer N2 latency was noted on the NoGo versus Go trials within the ARHL group for the Single-Car task ($p = 0.006$), but similar patterns were not seen within the NH group ($p > 0.05$). This finding suggests individuals with mild ARHL had prolonged neural processing times early on (150-300 ms) for the NoGo (inhibition trials) versus Go trials, but this differential processing was not seen within NH controls. However, the control group showed differential neural processing at later time points. Higher P3 amplitude was noted for NoGo versus Go trials within the control group for the Object-Animal task ($p = 0.033$) between 250-600 ms. The control group also showed more negative alpha power for the NoGo versus Go trials on both tasks ($p < 0.001$) between 300-650 ms, but the same pattern was not observed within the ARHL group. Given that P3 ERP and alpha band have been linked to neural effort [13], and that the reaction time for both tasks was within 450 ms, it seems that the NH group devotes more neural resources/effort to evaluate the stimuli of the inhibition trials at later time points, likely after making a response, but this is not done by the ARHL group. On analyses for our secondary aim, we found a positive relationship between QuickSIN score and P3 amplitude on Go trials on the Object-Animal task ($p = 0.021$) in the mild ARHL group. A similar trend was observed with NoGo trials ($p = 0.062$). These findings suggest that individuals with ARHL who had poorer SiN recognition scores used greater neural effort/resources for performing an inhibitory control task that involved superordinate categorization.

IV. CONCLUSION AND FUTURE WORK

Our study shows that neural processing related to inhibitory control in those with mild ARHL is different from normal hearing controls. This differentiation was evident in visual tasks, suggesting modality-independent changes in inhibitory control in those with untreated and mildest degree of ARHL, which constitutes the largest percentage of older adults with this condition [13]. Furthermore, these inhibitory control changes are related to complex listening functions such as SiN recognition. In summary, our work significantly advances the knowledge of neural changes underlying cognitive alterations in older adults with ARHL. However, the current work has some limitations. While our groups were not significantly different in age, they were unequal in number. Larger sample sizes with equal groups are needed to validate the findings. Additionally, future work examining visual inhibitory control is needed to examine the replicability of the current findings.

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