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Electrophysiological Underpinnings of Response Variability in the Go/NoGo Task

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Abstract

Response variability has been identified as a useful predictor of executive function and performance in non-clinical samples in the Go/NoGo task. The present study explores the utility of reaction time variability (RTV) and EEG measures as predictors of Go/NoGo performance outcomes and ERP component amplitudes. Forty-four young adults had EEG recorded across eyes-closed (EC) and eyes-open (EO) resting states, and during an auditory Go/NoGo task. The 18 individuals with the lowest/highest RTV were assessed for differences in behavioural outcomes. As expected, individuals with high RTV committed more Go/NoGo errors and had smaller Go P3b and NoGo P3a amplitudes, and greater Go Slow Wave positivity, reflecting inefficient decision-making and response control efforts underlying performance. When RTV and EEG measures were modelled as predictors of Go/NoGo responses, RTV and task-related changes in delta were identified as positive predictors of Go SW amplitude; while RTV and prestimulus delta amplitudes negatively predicted NoGo accuracy rates. Prestimulus delta was also found to solely predict Go mean RT and NoGo SW negativity; effects that were independent of RTV. As delta has been implicated in attention-related mechanisms, these findings suggest that inadequate attention and task engagement underpin the variability in Go/NoGo performance.

Keywords: Electroencephalography (EEG); Brain Dynamics; Attention; Cognition; Event-related potentials (ERPs); Principal Components Analysis (PCA); Response Variability

Electrophysiological Underpinnings of Go/NoGo Task Performance

1. Introduction

Laboratory tasks, such as the Go/NoGo, stop-signal and continuous performance task (CPT), are widely used to examine cognitive processes including attention, decision-making and working memory, and are utilised in clinical research to aid in the detection of deficits in these domains (Corbett and Constantine, 2006; Hester et al., 2005; Huster et al., 2013; Riccio et al., 2002; Ruchsow et al., 2008; van Boxtel et al., 2001). Behavioural measures including response accuracy, speed, and variability are used to index an individual's cognitive functioning, serving as proxies for brain dysfunction and/or impairment (Kaiser et al., 2008; Karalunas et al., 2014; Kofler et al., 2013; MacDonald et al., 2006; MacDonald et al., 2009; O'Connell et al., 2009; Schiff et al., 2014). In healthy samples, inverse relationships between reaction time variability (RTV) and accuracy rates in the Go/NoGo task have consistently been reported, reflecting the efficiency in cognitive control processes being executed (Bellgrove et al., 2004; Karamacoska et al., 2017; Karamacoska et al., 2018; Simmonds et al., 2007). Our research extends on previous fMRI efforts (Bellgrove et al., 2004; Simmonds et al., 2007) to evaluate the neural underpinnings of Go/NoGo performance using event-related potentials (ERPs), and examines the electroencephalographic (EEG) determinants of these responses.

1.1 ERP links to behaviour

A wealth of research has identified P3 component involvement in executive control processes (for reviews see Nieuwenhuis et al., 2005; Polich, 2007). Response facilitation to Go-type stimuli are marked by a parietal P3b positivity, and the magnitude of this component has been found to inversely relate to RTV (Ramchurn et al., 2014; Saville et al., 2011; Saville et al., 2012). We replicated this P3b-RTV relationship in the Go/NoGo task and also found that RTV correlated positively with the preceding P2 and post-P3 Slow Wave (SW)

component amplitudes (Karamacoska et al., 2017; Karamacoska et al., 2018). These findings highlight the evoked neural activity underlying decision-making efforts and response variability, but effects on NoGo ERPs have not been directly examined.

The withheld NoGo response has been associated with a frontocentral P3a positivity that has been directly related to NoGo accuracy rates (Fogarty et al., in press; Karamacoska et al., 2018). If individuals with high RTV have suboptimal cognitive control, then this should be reflected in their smaller NoGo P3a amplitudes. This hypothesis will be explored in the present study by analysing the Go/NoGo ERPs of individuals with low/high RTV.

1.2 EEG determinants of responses

The variability in performance has been attributed to top-down attentional (Hultsch et al., 2002; Unsworth et al., 2010) and response preparation control processes (Dankinas et al., 2015). These effects may stem from the brain's prestimulus state, as measured by EEG activity, which has been shown to affect ERP genesis (Mathewson et al., 2009; Rahn and Başar, 1993a, b). In an equiprobable Go/NoGo task, De Blasio and Barry (2013b) found that greater prestimulus delta levels increased the overall positivity of Go and NoGo ERPs, implicating this band in attentional processes (Başar et al., 2001a; Harmony, 2013; Harmony et al., 2009; Karakaş et al., 2000); while low theta levels reduced NoGo N2b and P3a amplitudes, and enhanced Go P3b positivity, linking this band to response control mechanisms (Başar et al., 2001b; Gulbinaite et al., 2014). These bands will be investigated here as potential determinants of performance outcomes.

Top-down preparations for stimulus-response processes have also been associated with prestimulus alpha (Foxe and Snyder, 2011; Klimesch et al., 2007; Mathewson et al., 2011) and beta activity (De Blasio and Barry, 2013a). Greater prestimulus alpha (8–13 Hz) has been shown to enhance N1, P2, and P3 amplitudes across a variety of paradigms (visual: Brandt et al., 1991; auditory Go/NoGo: De Blasio and Barry, 2013a; auditory dishabituation:

De Blasio et al., 2013; auditory oddball: Jasikutas and Hakarem, 1988). A direct relationship between beta increases and P1, P2, and P3 positivity was also reported (De Blasio and Barry, 2013a; De Blasio et al., 2013). Higher prestimulus alpha (10–11 Hz) levels were found for NoGo error trials *cf.* correctly withheld NoGo trials (Mazaheri et al., 2009) in a MEG study. RTV was not assessed in these studies, but given both alpha and beta relations with P2 and P3 amplitudes, fluctuations in these bands may affect response consistency and accuracy.

The above findings highlight the substantial impact of the brain's state on stimulus-evoked responses, but how the brain's state changes with task engagement should also be considered. Our prior study (Karamacoska et al., 2018) found that the shift in EEG activity, from a resting state to the task-situation, was predictive of Go/NoGo performance. Relative to eyes-open resting state activity, increases in prestimulus delta were associated with greater Go omission errors, RTV, and SW component positivity, while theta increases led to lower NoGo accuracy rates, and beta-1 changes predicted NoGo SW negativity. Earlier research investigating this task-related change identified right-temporal beta reductions that corresponded with increases in Go omissions in the CPT paradigm (Arruda et al., 2007; Arruda et al., 1999; Valentino et al., 1993). Together, these studies show that distinct task-related delta, theta, and beta changes underpin attentional processes that affect decision-making abilities and response outputs. These shifts in the EEG oscillations will also be examined here as potential neural mechanisms underlying performance outcomes.

1.3 The current study

This study aims to identify the electrophysiological underpinnings of Go/NoGo responses by analysing the ERP and EEG activity of participants with low/high RTV. After having EEG recorded across eyes-closed (EC) and eyes-open (EO) resting states, and during a Go/NoGo task, all included participants' (N = 44) behavioural data and ERP/EEG

topographies were examined. This ensured replicability with prior studies; these confirmatory analyses are reported in the Supplementary Materials.

We expected RTV to correlate with Go/NoGo accuracy rates, and those with the lowest vs. highest RTV outcomes were examined further. As RTV has been viewed as an indicator of cognitive control efforts (Bellgrove et al., 2004; Simmonds et al., 2007), this variable was included as a predictor of behavioural outcomes and the P2, P3, and SW amplitudes (previously shown to be associated with RTV) in our regression analyses. EEG measures were also included as predictors in these models, to determine how resting state and prestimulus EEG activity, and task-related change, contribute to these stimulus-response outcomes. It is hypothesised that, together with RTV, prestimulus delta, theta, alpha and beta amplitudes will predict response accuracy rates, mean RT, and P2 and P3 amplitudes. Task-related changes in delta, theta, and beta may also predict response accuracy rates and SW component amplitudes. Resting state EEG measures were also included as predictors, but based on previous findings (Karamacoska et al., 2018), impacts on behavioural measures and these ERP component amplitudes were not expected.

To our knowledge, this is the first study to comprehensively examine both ERP and EEG activity in the RTV context, and so a large number of hypotheses and tests are being conducted. Careful statistical considerations were made with all predicted correlations tested using one-way significance levels, whilst controlling for multiple comparisons where necessary. We also report findings that approach significance to encourage further research in this area.

2. Method

2.1 Participants and procedure

Forty-six right-handed university students (15 males), aged 18–27, gave written informed consent to voluntarily participate in this study. Participants had normal or corrected

vision and hearing. None reported a history of head injury, psychiatric or neurological disorders. All abstained from tobacco, alcohol, caffeine and psychoactive substances for a minimum of 12 hours before participating. After reviewing behavioural Go/NoGo performance data, two female participants were excluded for having excessively slow and variable RT outcomes that were 3 *SD* above the across-subjects mean.

After being fitted with EEG recording equipment, participants were seated in a darkened room 2.2 m from a projected display. Continuous EEG was recorded during an eye calibration task to enable offline corrections of electro-oculogram (EOG) activity. Participants were then instructed to relax for 2 minutes with EC, followed by 2 minutes with EO while fixating on a white cross in the display's centre. Two blocks of an auditory equiprobable Go/NoGo task were then performed while wearing Sony® MDR-V700 circumaural headphones. Go and NoGo stimuli were tones of 1000 and 1500 Hz, with the Go tone counterbalanced between blocks and participants. Each block consisted of 150 randomised tones that were 80 ms in duration (including 15 ms rise/fall times) at 60 dB SPL, presented at a stimulus onset asynchrony (SOA) of 1.25 s. Using a Logitech® controller, participants pressed a button with their right index finger to the Go tone of each block (following the procedures outlined by Barry and colleagues, 2013-2017). Task instructions emphasised response speed and accuracy, and to refrain from responding to the NoGo tone. Each block began with a practice involving 15 random trials. Participants were instructed to fixate on a white cross in the display's centre for the duration of the task. The University of Wollongong and Illawarra and Shoalhaven Local Health District Health and Medical Human Research Ethics Committee approved the study protocol.

2.2 Electrophysiological recording

Using Compumedics Neuroscan Acquire software (Version 4.3) on a Synamps 2 system, EEG activity from 30 electrodes (Fp1, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4,

FT8, T7, C3, Cz, C4, T8, TP7, CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, O1, Oz, and O2) and M2 were recorded. Activity from DC to 70 Hz was sampled, amplified, and digitized at 1 kHz. Electrodes were grounded by an electrode in the middle of Fp1, Fp2 and Fz, and referenced to M1. EOGs were also recorded with electrodes placed 1 cm beside the lateral canthus of each eye and above and below the left eye. For the cap and EOG, sintered Ag/AgCl electrodes were used, and impedances were kept below 5 kΩ.

2.3 Data quantification

2.3.1 Go/NoGo task data

Accuracy rates for Go and NoGo were measured as the percentage of correct responses relative to the number of stimuli presented. Misses to Go (omission errors) and button presses to NoGo (commission errors) were eliminated from further processing (< 11% of trials across participants). Following the rejection parameters used in previous studies (Ramchurn et al., 2014; van Dongen-Boomsma et al., 2010), extreme RTs to Go stimuli (≤ 150 ms or ≥ 700 ms) were rejected (< 9.3% of trials). This also reduced the variability in the ERPs for Go stimuli. Participants' RT mean and *SD* (representing RTV) for correct Go responses were calculated.

2.3.2 Pre-processing of EEG data

Recorded EEG data were EOG corrected using the revised aligned-artefact average EOG Correction Program (Croft and Barry, 2000). EOG-corrected data were then re-referenced to the mean of digitally-linked mastoids using Neuroscan Edit software (Compumedics, Version 4.5).

2.3.3 Go/NoGo ERPs

A band-pass filter from 0.1–30 Hz (zero phase shift, 24 dB/Octave) was applied to the EOG-corrected data for ERP derivation; prestimulus EEG epochs were extracted from the unfiltered data (detailed below). ERPs were epoched -100 to + 750 ms around stimulus onset

and baseline-corrected using the prestimulus period. Epochs with activity exceeding ± 100 μV, at any electrode, were excluded and the remaining epochs were confirmed by visual inspection. This resulted in an average of 69 (*SD* = 2.7) NoGo trials in each block accepted into participants' ERPs. For Go ERPs, epochs were accepted if the response was made within 1.5 SD of the participant's mean RT; resulting in a mean of 61 (*SD* = 4.7) Go epochs per participant's ERP from each block.

2.3.4 Temporal PCA decomposition of ERPs

Using Dien's ERP PCA toolkit (v. 2.23; Dien, 2010) in MATLAB, temporal PCAs were performed separately on Go and NoGo ERPs to better extract factors relevant to each stimulus (Barry et al., 2016b). All 44 participants' ERP data from each block were used (44 participants × 30 sites × 2 blocks = 2,640 cases) and the data were half-sampled to 425 time-points/variables, providing a cases/variables ratio of 6.2:1. The covariance matrix with Kaiser normalization was used for each PCA where all 425 factors were orthogonally rotated using Kayser and Tenke's (2003) Varimax4M software (available at: http://psychophysiology.cpmc.columbia.edu/software/index.html). Factors were selected in order of variance and identified as ERP components with reference to the latency and topography data reported in other similar ERP-PCA studies (Barry and De Blasio, 2013; Barry et al., 2014; Barry et al., 2016a; Barry et al., 2016b; Fogarty et al., in press).

2.3.5 Resting and prestimulus EEG quantification

Epochs of 1 s duration were extracted from the unfiltered 2 min of EC and EO resting EEG. Also, prestimulus EEG was taken from the unfiltered task data, using the 500 ms preceding stimulus onset (-500 to 0 ms) of the final Go/NoGo trials accepted for ERP formation. As separate PCAs were performed to extract Go/NoGo ERP components, prestimulus epochs were also separated as pre-Go and pre-NoGo. Differences in prestimulus EEG amplitudes were not anticipated due to this separation as the stimuli were presented

without a cue, at a fixed SOA rate, and were equally probable (see also De Blasio and Barry, 2013a, 2013b; Karamacoska et al., 2018). DC-correction was applied across epochs, and if activity exceeded ± 100 μV, at any site, the epoch was rejected. MATLAB was used to apply a 10 % Hanning window to epochs where discrete Fourier transforms (DFTs) were conducted with 1 Hz resolution. DFTs were performed on the 1,000 data points of the resting state epochs; the 500 data points of the prestimulus epochs were zero-padded to 1000 points. Corrections for having used the window, and for the prestimulus padding, were then applied. Amplitudes in the one Hz bins were summed to form amplitudes for each frequency band: delta (1–3 Hz), theta (4–7 Hz), alpha-1 (8–10 Hz), alpha-2 (11–13 Hz), beta-1 (14–20 Hz), beta-2 (21–29 Hz); these band limits were selected from prior research (Intrilligator and Polich, 1995; Karamacoska et al., 2018; Polich, 1997; Valentino et al., 1993). The mean band amplitude was calculated across epochs, at every site, for EC (with a minimum of 92 epochs) and EO (minimum of 80 epochs) resting states and pre-Go/NoGo periods. As the task was completed with eyes open, the EO resting state was selected as the baseline to measure task-related EEG change (as recommended by Barry et al., 2007). The change was calculated as pre-Go/NoGo minus EO amplitude.

2.4 Confirmatory Statistical Analyses

For replication purposes, the entire sample (N = 44) was analysed for their patterns in behavioural performance and electrophysiological activity. The outcomes of these confirmatory tests are presented in the Supplementary Materials.

2.4.1 Go/NoGo task behavioural measures

As a consistent relationship between Go/NoGo performance measures has been identified in past studies (where greater RTV was associated with higher error rates), we sought to confirm these performance patterns here. Pearson's one-tailed correlations (r) were conducted between the behavioural measures, with 42 degrees of freedom. The false-

discovery rate (FDR) control procedure (Benjamini and Yekutieli, 2001) was used to account for the multiple correlations conducted on each dependent variable.

2.4.2 ERP/EEG topographies

To efficiently assess ERP/EEG topographic amplitudes, 9 topographic regions were formed by calculating a mean across electrode groupings: Frontal-left (FL: Fp1, F3, FC3, F7, FT7), frontal-midline (FM: Fz, FCz), frontal-right (FR: Fp2, F4, FC4, F8, FT8); central-left (CL: C3, CP3, T7, TP7), central-midline (CM: Cz, CPz), central-right (CR: C4, CP4, T8, TP8); parietal/occipital-left (POL: P3, P7, O1), parietal/occipital-midline (POM: Pz, Oz), parietal/occipital-right (POR: P4, P8, O2). Separate 3 × 3 multivariate analyses of variance (MANOVAs) were conducted in SPSS on ERP component and EEG band amplitudes. The within-subjects factors consisted of the sagittal topographic plane: frontal (FL, FM, FR), central (CL, CM, CR), parietal/occipital (POL, POM, POR); and coronal plane: left (FL, CL, POL), midline (FM, CM, POM), right (FR, CR, POR). Orthogonal contrasts were planned to compare the regional amplitudes within each plane: frontal (F) versus parietal/occipital (PO), and central (C) versus the frontal and parietal/occipital mean (F/PO); left (L) versus right (R), and midline (M) versus the left and right mean (L/R). The interactions between these planes were also assessed.

The MANOVA *F*-tests all had (1, 43) degrees of freedom and, as they contained a single degree of freedom, Greenhouse-Geisser corrections were unnecessary (O'Brien and Kaiser, 1985). Bonferroni-type α adjustments were also not required as contrasts were planned and did not exceed the degrees of freedom for effect (Tabachnick and Fidell, 2013).

The MANOVAs conducted on the ERP and EEG topographies, across the whole sample, are described in Supplementary Materials. These analyses guided the selection of regions where ERP and EEG amplitudes were maximal. Where multiple sites were

identified, the mean across these regions was calculated. These regions were then utilised in the subsequent regressions below.

2.5 Between-Group Assessments

2.5.1 RTV groupings

We sorted the sample based on RTV outcomes and selected the 18 individuals at each extreme to form the Low/High RTV groups. Previous studies examining P3 activity have reported significant group differences with sample sizes of 13-16 participants (Ramchurn et al., 2014; Saville et al., 2012). The 18 individuals with the lowest RTV means (7 males; aged 19.7 ± 1.8 years) formed the Low RTV (LRTV) group, and the 18 with the highest RTV values (6 males; aged 21.1 ± 2.7 years) formed the High RTV (HRTV) group.

2.5.2 Group differences in behavioural Go/NoGo outcomes

Behavioural outcomes were compared between the two RTV groups using independent samples *t* tests for mean RT and Welch's *t* test for RTV data (as groups were found to have unequal RTV variances). Due to the skew and unequal distribution in the groups' Go and NoGo accuracy rates, one-tailed Mann-Whitney *U* tests of mean ranks were used to compare the groups.

2.5.4 Determinants of Go/NoGo performance

Stepwise multiple regressions were conducted separately for each dependent variable (Go/NoGo error rates, mean RT, and P2, P3, and SW component amplitudes[1]). The predictors of these stimulus-response outcomes included RTV group, entered as a categorical variable, and the 6 EEG bands from each state measure (overall resting state, prestimulus and task-related change). The mean amplitude from the maximal region of ERP/EEG topographies, as identified in the confirmatory tests, was utilised here. The entrance criterion

[1] These ERP components were selected as previous studies identified relationships between RTV and their amplitudes.

for predictors was set at $\alpha = .075$ so that any predictors approaching significance were included and could be investigated in future studies.

3. Results

3.1 RTV group differences in Go/NoGo task outcomes

3.1.1 Behavioural measures

Go/NoGo behavioural data for each group are presented in Table 1. Levene's test indicated unequal group variances for RTV ($F = 13.14$, $p = .001$) and so Welch's t test was used here: RTV was significantly greater in the High RTV group, $t(25) = -13.21$, $p < .001$. Although data suggest slightly longer mean RTs for the High RTV group, this difference was found to approach significance, $t(34) = -2.00$, $p = .053$. One-tailed Mann-Whitney U tests indicated that the High RTV group also had significantly lower accuracy rates for both Go, $U = 60.5$, $p < .001$, and NoGo, $U = 76.5$, $p = .003$.

Table 1
Behavioural Performance Data of the RTV Groups

	LRTV Group		HRTV Group	
	Range	M ± SD	Range	M ± SD
RTV (ms)*	55.84–76.80	67.30 ± 5.20	92.16–121.04	102.94 ± 10.20
Mean RT (ms)	281.97–472.33	373.13 ± 44.42	350.02–448.72	398.03 ± 28.44
		Mean Rank		Mean Rank
Go Accuracy (%)*	95.33–100.00	24.14	94.00–100.00	23.25
NoGo Accuracy (%)*	95.33–100.00	12.86	93.33–98.67	13.75

Note. * Indicates significant group differences with $p < .01$.

3.1.2 Go/NoGo ERPs

The grand mean Go and NoGo ERPs (at Fz, Cz and Pz) are displayed in the top panels of Figures 1 and 2, respectively. Note that there appears to be some activity in the baseline period, particularly at Pz, which likely corresponds with the resolution of the late positive complex (Barry et al., 2018). The dashed lines represent the ERP waveforms derived from the extracted PCA factors. The ERPs of each RTV group are also compared

against the grand mean data (N = 44). Obvious group differences can be seen from ~ 250 ms in the Go/NoGo ERPs.

3.1.3 Go PCA outcomes

The first eight components in the Go PCA accounted for 88.7 % of the variance. These were labelled, in latency order, as the N1-1, Processing Negativity (PN), P2, TF06 (tentatively labelled as an N2), N2c[2], P3b, the first Slow Wave (SW) component, and a second SW (SW2). TF03 (SW2) was excluded as it likely represents the non-zero activity at the end of epochs (Verleger and Möcks, 1987). Figure 1 depicts the grand mean Go ERPs in the top panel, with a good fit seen between the actual data and PCA reconstituted ERPs. The topographic headmaps and summary for each factor are presented in temporal order in the middle panel with the bottom panel representing each factor's scaled loading.

Figure 1 about here.

3.1.4 NoGo PCA outcomes

As shown in Figure 2 (middle panel), the first seven factors extracted for NoGo explained 82.1 % of the variance and were labelled as the N1-1, PN, P2/N2b, P3a, NoGo P3, N470, and SW. A good fit can be seen between the PCA-derived data and raw ERPs in the top panel of Figure 2. TF06 (N470) was excluded from analysis as it could not be recognised as a known ERP component for this paradigm and carried little variance.

Figure 2 about here.

3.1.5 ERP component topographies

[2]The N2c-P3b pairing has been postulated to mark response activation and execution processes (see the work of Barry and colleagues, 2015-2017). To determine which factor represented the N2c here, topographic amplitudes of the two N2 factors were correlated with the P3b amplitudes across all 30 sites. TF06 (N2) amplitudes did not significantly correlate with P3b amplitudes (r[28] = .014, p > .05, one-way) while TF08 significantly correlated with the P3b (r[28] = .33, p = .037, one-way). Based on these data, TF08 was labelled as the N2c and analysed further. Note: This factor's latency and topography are also consistent with Karamacoska et al. (2017). The unidentified TF06 was excluded from further investigation.

The MANOVA outcomes confirming Go/NoGo ERP component topographies, across the whole sample, are presented in Supplementary Materials sections S2.1 and S2.2. These topographies replicated previous PCA-derived ERP component data from this paradigm (Barry and De Blasio, 2013; Barry et al., 2014; Barry et al., 2016a; Barry et al., 2016b; Fogarty et al., in press; Karamacoska et al., 2018). Based on these outcomes, regions of interest (ROI) were identified as follows: Go P2 was assessed at the CM site, Go P3b was maximal across the CM, POM and POL areas, and SW positivity was centrally dominant (CL, CM, and CR); NoGo P2 amplitudes were maximal centrally in the hemispheres (CL and CR), P3a was assessed at the dominant FM and CM regions, the second NoGo P3 was maximal centrally, and the predominant negativity in the SW component was assessed at the FM region.

3.2 Confirming EEG patterns of activity

Figure 3 displays the grand mean amplitude spectra at the midline sites, and the topographic headmaps of each EEG band for the overall resting state (EC and EO mean), EC, EO, and prestimulus states (shown as the mean of pre-Go and pre-NoGo data), and the task-related change calculated from EO to the prestimulus period. RTV group differences in EEG spectral amplitude can be seen at ~ 10 Hz across resting and prestimulus states. The confirmatory topographic MANOVAs conducted for each EEG measure, across the whole sample, are presented in Supplementary Materials S3.1-S3.4. The topographic distributions were generally consistent with prior studies of resting state activity (Barry et al., 2007; Intrilligator and Polich, 1995; Karamacoska et al., 2018; Tenke et al., 2015), with delta and theta dominant over FM and CM regions, alpha-1 and alpha-2 were maximal across CM and POM regions, beta-1 was largest in the PO hemispheres, and beta-2 was dominant in the FM area; all 6 bands were found to decrease in amplitude, especially across the PO region, from the EC to EO state. Prestimulus EEG patterns indicated delta and theta were FM and CM

dominant, alpha-1/2 were largest across the CM and PO (POL, POM, POR) regions, beta-1 was maximal over FM and CM areas, and beta-2 dominated the frontal areas (FL, FM, FR); these findings are comparable to previous studies (De Blasio et al., 2013; Tenke et al., 2015; Min and Herrmann, 2007; Min and Park, 2010). These regions also demonstrated the largest task-related changes, as amplitudes increased from EO to the prestimulus state, replicating Karamacoska et al. (2018). These ROIs were utilised in the subsequent regressions.

Figure 3 about here.

3.3 Determinants of Go/NoGo performance: Regression outcomes

Stepwise multiple regressions modelled RTV grouping and the 6 EEG bands, at their ROIs (as identified in section 3.2), from each state as predictors of Go/NoGo behavioural outcomes (Go mean RT and accuracy; NoGo accuracy) and the ERP components (Go P2, P3b and SW, and NoGo P2, P3a, P3, and SW). The regressions were run using the EEG measures relative to the Go/NoGo variables being tested (e.g., task-related EEG changes and the prestimulus amplitudes calculated for Go were used when modelling Go behavioural data).

For Go responses, RTV grouping negatively predicted Go accuracy rates (β = -.47, t = -3.09, p = .004) explaining 21.9 % of the variance, and P3b amplitudes (β = -.32, t = -1.94, p = .030) accounting for 9.9 % of the variance. Go mean RT was positively predicted by prestimulus frontocentral-midline delta amplitudes (β = .35, t = 2.18, p = .018), explaining 12.3 % of the variance. Go SW positivity was found to be positively predicted by RTV grouping (β = .38, t = 2.62, p = .013) and task-related change in frontocentral-midline delta (β = .36, t = 2.51, p = .017), $F(2, 33) = 8.40$, p = .001 (VIF = 1.05), accounting for 33.7 % of the variance. There were no significant models obtained for Go and NoGo P2 amplitudes.

NoGo accuracy rates were found to be negatively predicted by RTV grouping (β = -.37, t = -2.54, p = .016) and prestimulus frontocentral-midline delta (β = -.39, t = -2.71, p =

.011), together explaining 36.8 % of the variance, $F(2, 33) = 9.61$, $p = .001$ (VIF = 1.09). P3a amplitudes were inversely predicted by RTV grouping ($\beta = -.48$, $t = -3.14$, $p = .003$), explaining 22.5 % of the variance. There was no significant model obtained for the second NoGo P3. NoGo SW negativity was predicted by prestimulus frontocentral-midline delta ($\beta = .32$, $t = 1.95$, $p = .030$), accounting for 10.1 % of the variance. Being a negative component, the amplitudes of these measures were inversely related i.e., greater delta amplitude was associated with less negativity.

4. Discussion

This study examined the electrophysiological activity underpinning the Go/NoGo responses of participants with low vs. high RTV. As reported in Supplementary Materials, analyses of all 44 participants' data replicated the behavioural and electrophysiological patterns of activity from prior studies. Importantly, the predicted relationship between RTV and response accuracy rates was confirmed, indicating that individuals with higher RTV were susceptible to committing more errors. RTV also correlated with mean RT but this measure did not differ significantly between the two groups. When RTV and EEG measures were modelled as predictors of Go/NoGo responses, RTV was found to negatively predict Go accuracy rates, and Go P3b and NoGo P3a amplitudes. RTV and task-related changes in delta were identified as positive predictors of Go SW amplitude, while RTV and prestimulus delta amplitudes negatively predicted NoGo accuracy rates. Prestimulus delta was also found to solely predict Go mean RT and NoGo SW negativity; effects that were independent of RTV.

4.1 RTV predicts Go/NoGo performance

The negative relationships reported between RTV grouping and Go/NoGo accuracy rates, Go P3b, and NoGo P3a amplitudes, supports our hypotheses that these measures are attenuated for individuals with high RTV. Notably, a positive relationship was obtained for

RTV grouping and Go SW amplitudes, consistent with Karamacoska et al. (2018). These findings highlight the involvement of these ERP components in decision-making and response control, and supports the utility of RTV as a marker of executive function. The Go ERP findings replicated prior studies, the only exception being the absent effect on P2. This is likely due to differences in task parameters as we used a fixed SOA in the task here, instead of the variable SOA in Karamacoska et al. (2017), which can affect attentional demands and P2 and P3 magnitudes (Borchard et al., 2015). The NoGo P3a effects are novel, as the NoGo ERPs have not been explored with response variability. Here, we have confirmed the hypothesis that individuals with higher RTV have inefficient cognitive control efforts, as reflected by their smaller P3a amplitudes and greater commission error rates. Together, these findings confirm the patterns of evoked neural activity linked to response consistency and accuracy.

4.2 RTV and EEG determinants of Go/NoGo responses

As the EEG was hypothesised to underlie Go/NoGo responses, this study explored resting state EEG, prestimulus activity, and the change from the EO resting state, alongside RTV as determinants of ERP amplitudes and behavioural outputs. As expected, resting state EEG did not contribute to task-based response measures. Both RTV grouping and task-related changes in delta positively predicted Go SW positivity, indicating the concurrent effect of having higher RTV and delta increases from EO to the task. This trend is consistent with Karamacoska et al. (2018), reinforcing delta's mechanistic role in attention and task-related engagement. The increase in prestimulus delta amplitude appeared to have an ongoing effect as lower NoGo accuracy rates were predicted by having greater RTV and prestimulus delta amplitude. Interestingly, Go mean RT and NoGo SW negativity were both predicted by prestimulus delta, whereby greater prestimulus amplitudes led to longer mean RTs and less NoGo SW negativity. These effects were independent of RTV, suggesting a

broader impact of prestimulus delta activity. Notably, across these regression models, delta was the only EEG measure found to predict Go/NoGo responses. Together, these findings provide evidence for delta's role in attentional lapses that contribute to less efficient and correct responding (Başar et al., 2001a; De Blasio and Barry, 2013b; Guntekin and Başar, 2016; Harmony, 2013; Knyazev, 2012). Contrary to expectations, no corresponding EEG effects were found in the Go P3b and NoGo P3a amplitudes. This may be because the P3s in the present task reflect response control processes more so than attention-related activity. Thus, delta could be impacting earlier ERP components linked to attention that were not analysed here, e.g., N1-1.

EEG effects in theta, alpha, and beta were also not found. The lack of theta-related findings suggests that participants were not implementing preparatory response strategies, perhaps due to the unwarned and random nature of the stimuli. This could be further explored in paradigms utilising cues to examine the preparatory activity between the cue and imperative stimuli. For alpha and beta, it is possible that these bands do not directly relate to response processes, but affect the preceding ERP components involved in stimulus registration and discrimination. Further research examining the N1-1, PN, and P2 can elucidate alpha and beta involvement in top-down preparations for stimulus-response processes (De Blasio and Barry, 2013a; De Blasio et al., 2013; Foxe and Snyder, 2011; Klimesch et al., 2007; Mathewson et al., 2011).

Overall, the present findings highlight the evoked neural activity involved in decision-making and cognitive control processes that were determined by RTV and delta activity. As this is the first study to comprehensively assess the ERP and EEG amplitudes of participants grouped according to their RTV outcomes, replication is required. Although this study utilised ERP and EEG derivation techniques established within the literature, there are limitations to these methods. ERP component latency jitter is a problem faced when

averaging epochs for PCA decomposition, especially for the Go P3b and SW components that covary with RT (Verleger et al., 2016). We attempted to minimise this variability by accepting epochs within a certain RT range, but we note that the jitter dampens the present amplitude findings. These outcomes could be enhanced by performing single-trial analyses of ERPs (see Pernet et al., 2011) with PCA decomposition, such as in Saville et al. (2011). The analysis of EEG data using predefined frequency bands was also limiting. Much like the temporal PCA approach to decomposing ERPs, better estimations of EEG activity using frequency-PCA have been proposed by Tenke and Kayser (2005; see also Tenke et al., 2011). This technique was also recently applied to assess the EEG during resting and task-based states (Barry and De Blasio, in press). These studies have identified multiple delta, alpha and beta frequency components that are overlooked when using predefined band limits. Thus, the present study could be enhanced by performing separate temporal- and frequency-PCAs on the groups to better capture their ERP and EEG activity, respectively. While the dichotomisation of groups allowed us to identify the potential EEG measures contributing to RTV differences, this results in the loss of data from discarded individuals. Future research can extend on the present findings by analysing the electrophysiological data of RT distributions at the within-subjects level. This could confirm the pattern of relationships identified in the present study. Importantly, these electrophysiological assessments continue to enhance our understanding of brain functioning and the neural activity underpinning behavioural processes.

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Figure Captions

Figure 1. Grand mean Go stimulus ERPs (*N* = 44) for the actual data and PCA-derived output data, at Fz, Cz and Pz, are displayed in the top panel. The topographic headmaps of the PCA factors representing Go ERP components and their factor loadings are shown, in temporal order, in the lower panels. Go ERPs for the RTV groups are provided for comparison. Greyed factors (TF06 and TF03) were excluded from analyses.

Figure 2. Grand mean NoGo stimulus ERPs (*N* = 44), at the midline sites, for the PCA input and output data are shown in the top panel. Topographic headmaps of the NoGo PCA components and their factor loadings are shown, in temporal order, in the lower panels. NoGo ERPs of the RTV groups are provided for comparison. TF06 (greyed out here) was excluded from analyses.

Figure 3. Grand mean (N = 44) frequency spectral amplitudes at Fz, Cz and Pz, for the EC, EO and pre-Go/NoGo states are displayed in the top panels with Low/High RTV group comparisons of EEG spectra. The topographic headmaps for the overall resting state (mean of EC and EO), EC, EO, prestimulus (mean of pre-Go/NoGo) and task-related change measures are shown below.

Supplementary Materials

Introduction

Analyses of all 44 participants' behavioural performance patterns and ERP/EEG topographies were conducted to confirm the replication of previous studies. Go/NoGo behavioural data were correlated to confirm the performance patterns observed in previous studies. Here, we expected significant correlations between RTV and Go/NoGo accuracy rate, but non-significant associations between these measures and mean RT (Bellgrove et al., 2004; Karamacoska et al., 2017; Karamacoska et al., 2018).

Principal Components Analyses (PCAs) were used to derive ERP component amplitudes. These were statistically assessed for their topographic distribution with the following expected for each component: N1-1 would be frontocentrally distributed; PN amplitudes would dominate the central hemispheres; a central-midline positivity would be apparent for P2; NoGo P3a and Go P3b amplitudes dominate the frontocentral and parietal regions, respectively; and SW would be frontally negative and centroparietally positive (see also the work of Barry and colleagues, 2013-2016). EEG amplitudes were also subjected to the same topographic analyses with the following predictions for their distributions: Across EC, EO and prestimulus states, delta and theta topographies were expected to be dominant in the frontocentral-midline, alpha-1 and alpha-2 activity would be largest over the parietal/occipital-midline region, with a frontocentral-midline dominance for beta-1 and beta-2 amplitudes (Barry and De Blasio, in press). The change between states is expected to show parietal reductions in the shift from EC to EO, and amplitude increases from EO to the prestimulus period (Karamacoska et al., 2018; Tenke et al., 2015; Valentino et al., 1993).

Method: ERP/EEG Topographic Analyses

For ERP components, the PCA output data were averaged across the blocks and submitted to the 3 × 3 MANOVA design described in section 2.4.2 of the study. The EEG

data were first assessed for the EC and EO resting states where the within-subjects factor of resting state (EC, EO) was included. This design allows for the overall resting state topographies to be analysed (i.e., the EC and EO mean) with a subsequent comparison between EC and EO for the reactive change between resting states (termed EC to EO reactivity). Prestimulus EEG topography was analysed using the within-subjects factor of stimulus (Pre-Go, Pre-NoGo). Task-related change was examined using the difference data calculated between EO and pre-Go/NoGo amplitudes (reported as TR-Go/TR-NoGo). Stimulus was included as a within-subjects factor in these designs to test whether the prestimulus EEG differed between the stimuli.

Results

S1 Go/NoGo Task Behavioural Outcomes

Go and NoGo accuracy rates were positively correlated (r = .44, p = .002), and RTV correlated negatively with Go response accuracy (r = -.51, p < .001) and with NoGo accuracy rates (r = -.42, p = .004). RTV also correlated positively with mean RT (r = .32, p = .037), but mean RT did not correlate with error rates (both $|r| \leq$ -.22, $p \geq$ .151).

S2 Go/NoGo ERP Component Topographies

Tables S2.1 and S2.2 refer to the outcomes of the topographic MANOVAs conducted for the PCA-derived Go and NoGo ERP component amplitudes, respectively. Only the significant F, p (< .05) and η_p^2 statistics for the main sagittal and coronal orthogonal contrasts are listed. Go/NoGo ERP component topographies can also be viewed in the middle panels of Figures 1 and 2, respectively.

S2.1 Go PCA-Derived ERP components. The Go N1-1 component was larger frontally and dominant in the midline, particularly in the frontocentral-midline region. Go PN amplitudes were more negative frontocentrally, particularly in the central-left. The Go P2 was found to be more positive in the midline, particularly centrally, but overlapped with a

frontal negativity that was greater in the right hemisphere; this contributed to a larger frontal/parietal/occipital mean (*cf.* central) and interacted to show a frontal enhancement in the hemispheres, especially on the right. Topographically, the Go N2c was more negative frontally, particularly in the midline, and centrally in the hemispheres. Go P3b positivity dominated the parietal/occipital, central and midline regions. Centrally, the positivity was greater in the midline and on the right, but the parietal/occipital enhancement was larger on the left. The bipolar Go SW component was centrally positive with a left hemispheric bias and enhanced positivity in the central-midline and left regions. More positivity was also apparent in the left hemisphere of the parietal/occipital region. Note that the negativity of this component is also reflected in the frontal-midline interaction effect (italicised in Table S2.1), however, this was relatively small compared to the positive amplitudes in the central region.

Table S2.1
PCA-Derived Go ERP Component Amplitude Topographies

	Effect	*F*	*p*	η_p^2
N1-1	F > PO	74.28	<.001	.63
	M > L/R	119.72	<.001	.74
	F > PO × M > L/R	66.95	<.001	.61
	C > F/PO × M > L/R	66.80	<.001	.61
PN	F > PO	11.31	.002	.21
	C > F/PO	17.49	<.001	.29
	C > F/PO × L > R	5.56	.023	.11
P2	*F > PO*	*4.64*	*.037*	*.10*
	C < F/PO	*5.48*	*.024*	*.11*
	L < R	*9.62*	*.003*	*.18*
	M > L/R	15.08	<.001	.26
	F > PO × L < R	*12.64*	*.001*	*.23*
	F > PO × M < L/R	*7.04*	*.011*	*.14*
	C > F/PO × M > L/R	17.05	<.001	.28
N2c	F > PO	5.62	.022	.12
	F > PO × M > L/R	4.52	.039	.10
	C > F/PO × M < L/R	7.80	.008	.15
P3b	F < PO	51.65	<.001	.55
	C > F/PO	75.14	<.001	.64
	M > L/R	16.96	<.001	.28
	F < PO × L > R	37.41	<.001	.47

	C > F/PO × L < R	4.75	.035	.10
	C > F/PO × M > L/R	39.16	<.001	.48
Slow Wave	C > F/PO	93.05	<.001	.68
	L > R	15.68	<.001	.27
	F < PO × L > R	26.50	<.001	.38
	F > PO × M > L/R	9.57	.003	.18
	C > F/PO × L > R	11.46	.002	.21
	C > F/PO × M > L/R	11.28	.002	.21

Note: The bipolar topographic nature of the P2 and SW components are listed with italics representing the effects for the negative polarity (not analysed).

S2.2 NoGo PCA-Derived ERP components. NoGo N1-1 negativity was larger over the frontal and midline regions, interacting for an enhancement in the frontocentral-midline. The NoGo PN was more negative frontocentrally, in the right hemisphere *cf*. the left, with a frontal enhancement in the midline also apparent. Centrally, there was greater negativity on the right; this was reduced in the midline. The next extracted factor appears to be the conglomerate of the P2 and N2b components that occur at a similar latency. This amalgamated component was found to be more positive frontocentrally and in the hemispheres, interacting to show a frontal enhancement in the left and right regions. P3a positivity was larger centrally and across the midline, producing an enhancement in the central-midline. The positivity in the midline was also greater frontally, and centrally on the left. The second NoGo P3 had greater positivity centrally, particularly in the hemispheres, with larger frontal amplitudes in the right and midline regions. The bipolar SW was defined by greater frontal and parietal/occipital negativity *cf.* the central region, with more negativity in the midline, particularly frontally.

Table S2.2
PCA-Derived NoGo ERP Component Amplitude Topographies

	Effect	*F*	*p*	η_p^2
N1-1	F > PO	95.13	< .001	.69
	M > L/R	125.07	<.001	.74
	F > PO × M > L/R	33.36	<.001	.44
	C > F/PO × M > L/R	62.54	<.001	.59

PN	F > PO	11.34	.002	.21
	C > F/PO	7.45	.009	.15
	L < R	28.65	<.001	.40
	F > PO × M > L/R	6.96	.012	.14
	C > F/PO × L < R	7.56	.009	.15
	C < F/PO × M < L/R	4.70	.036	.10
P2/N2b	F > PO	6.86	.012	.14
	C > F/PO	36.22	<.001	.46
	M < L/R	20.92	<.001	.33
	F > PO × M < L/R	5.15	.028	.11
P3a	C > F/PO	33.60	<.001	.44
	M > L/R	52.98	<.001	.55
	F > PO × M > L/R	24.08	<.001	.36
	C > F/PO × L > R	6.10	.018	.12
	C > F/PO × M > L/R	58.30	<.001	.58
P3	F > PO	4.75	.035	.10
	C > F/PO	81.44	<.001	.65
	F > PO × L < R	4.85	.033	.10
	F > PO × M > L/R	30.79	<.001	.42
	C > F/PO × M < L/R	14.50	<.001	.25
Slow Wave	C < F/PO	49.45	<.001	.53
	M > L/R	6.97	.011	.14
	F > PO × M > L/R	10.38	.002	.19

These Go/NoGo ERP component topographies replicate previous PCA-derived ERP component data across young adult samples (Barry and De Blasio, 2013; Barry et al., 2014; Barry et al., 2016a; Barry et al., 2016b; Fogarty et al., in press; Karamacoska et al., 2018).

S3 EEG Band Topographies

Tables S3.1-3.4 show the topographic effects obtained from the MANOVAs conducted to confirm each band's scalp distribution for the overall resting state, EC to EO reactivity, prestimulus and task-related change measures. The tables present the significant *F*, *p* (< .05) and η_p^2 statistics for the main sagittal and coronal effects, and their interactions for each band. Topographic headmaps displaying this EEG activity can also be viewed in the bottom rows of Figure 2.

S3.1 Overall resting state EEG. Delta and theta were dominant across the frontocentral regions and were maximal in the midline, interacting for a frontocentral-midline enhancement. A right hemispheric bias was apparent, however, this was smaller frontally and centrally. Alpha-1, alpha-2 and beta-1 were also dominant over the midline region, particularly centrally, and parietal/occipitally with enhancements in the hemispheres, largely on the right. Laterally, a right hemispheric bias was evident in alpha-1 to beta-1 band amplitudes; this was smaller centrally for alpha-2 and beta-1. Beta-2 showed a midline dominance that was larger frontocentrally; a slight right bias in the parietal/occipital region was also apparent, contributing to a larger F/PO mean on the right *cf.* the central-right.

Table S3.1
Overall resting state (EC/EO mean) EEG Topographies

Band	Effect	*F*	*p*	η_p^2
Delta	F > PO	29.96	<.001	.41
	C > F/PO	20.17	<.001	.32
	L < R	9.79	.003	.19
	M > L/R	799.32	<.001	.95
	F > PO × L > R	10.59	.002	.20
	F > PO × M > L/R	430.21	<.001	.91
	C > F/PO × L > R	12.68	.001	.23
	C > F/PO × M > L/R	114.07	<.001	.73
Theta	F > PO	16.80	<.001	.28
	C > F/PO	39.82	<.001	.48
	L < R	12.94	.001	.23
	M > L/R	516.73	<.001	.92
	F > PO × L > R	31.60	<.001	.42
	F > PO × M > L/R	643.90	<.001	.94
	C > F/PO × L > R	12.81	.001	.23
	C > F/PO × M > L/R	165.79	<.001	.79
Alpha-1	F < PO	35.25	<.001	.45
	L < R	21.15	<.001	.33
	M > L/R	157.52	<.001	.79
	F < PO × L < R	39.08	<.001	.48
	F < PO × M < L/R	165.91	<.001	.79
	C > F/PO × M > L/R	84.99	<.001	.66

	F < PO	50.25	<.001	.54
	L < R	30.16	<.001	.41
	M > L/R	152.93	<.001	.78
Alpha-2	F < PO × L < R	62.01	<.001	.59
	F < PO × M < L/R	68.50	<.001	.61
	C < F/PO × L < R	11.52	.001	.21
	C > F/PO × M > L/R	44.88	<.001	.51
	F < PO	8.77	.005	.17
	C > F/PO	10.18	.003	.19
	L < R	18.08	<.001	.30
	M > L/R	138.56	<.001	.76
Beta-1	F < PO × L < R	51.77	<.001	.55
	F < PO × M < L/R	208.29	<.001	.83
	C < F/PO × L < R	7.30	.010	.15
	C > F/PO × M > L/R	37.39	<.001	.47
	M > L/R	40.28	<.001	.48
	F < PO × L < R	8.82	.005	.17
Beta-2	F > PO × M > L/R	56.25	<.001	.57
	C < F/PO × L < R	4.82	.034	.10
	C > F/PO × M > L/R	10.91	.002	.20

S3.2 EC to EO EEG reactivity. Table S3.2 presents the topographic effects for the resting EC to EO differences in EEG where a broadband reduction in amplitude was found (main effect of state, EC > EO: all $F > 4.70$, $p \leq .036$, $\eta_p^2 \geq .10$). Delta reductions were greater parietal/occipitally and in the right hemisphere but less so centrally *cf.* frontal and parietal/occipital regional mean. Theta decreased in the parietal/occipital and midline regions, and most notably in the central-midline. A greater decrease was apparent in the right hemisphere, particularly in the parietal/occipital region, contributing to a larger parietal/occipital hemispheric mean reduction. Centrally, the decrease was smaller on the right. Alpha-1 reactivity showed a parietal/occipital decrease that was greater in the hemispheres. The decrease in alpha-1 reactivity was also smaller in the central region *cf.* the mean of frontal/parietal/occipital regions. Laterally, the reduction in alpha-1 amplitude was larger in the midline, most notably in the central region. Alpha-2 reactivity was

predominantly localised to the parietal/occipital region, particularly in the hemispheres and especially on the right; the reduction was smallest centrally in the midline. Beta-1 reactivity showed reductions largest in the parietal/occipital region, especially in the hemispheres with a right bias. The decrease in beta-1 was smaller centrally *cf.* the frontal/parietal/occipital mean, except in the midline where the reduction was greatest. Beta-2 reactivity similarly showed a large parietal/occipital decrease that was greater in the hemispheres. A midline reduction was also apparent.

Table S3.2
EC to EO Reactivity Changes in EEG Topography

Band	Effect	*F*	*p*	η_p^2
Delta	EC > EO × F < PO	10.27	.003	.19
	EC > EO × C < F/PO	9.97	.003	.19
	EC > EO × L < R	7.67	.008	.15
Theta	EC > EO × F < PO	44.40	<.001	.51
	EC > EO × L < R	8.21	.006	.16
	EC > EO × M > L/R	42.32	<.001	.50
	EC > EO × F < PO × L < R	15.88	<.001	.27
	EC > EO × F < PO × M < L/R	10.31	.003	.19
	EC > EO × C < F/PO × L < R	4.54	.039	.10
	EC > EO × C > F/PO × M > L/R	50.21	<.001	.54
Alpha-1	EC > EO × F < PO	49.03	<.001	.53
	EC > EO × C < F/PO	17.59	<.001	.29
	EC > EO × M > L/R	34.54	<.001	.45
	EC > EO × F < PO × M < L/R	45.82	<.001	.52
	EC > EO × C > F/PO × M > L/R	52.24	<.001	.55
Alpha-2	EC > EO × F < PO	38.00	<.001	.47
	EC > EO × C < F/PO	15.67	<.001	.27
	EC > EO × F < PO × L < R	4.27	.045	.09
	EC > EO × F < PO × M < L/R	27.81	<.001	.39
	EC > EO × C < F/PO × M < L/R	61.38	<.001	.59
Beta-1	EC > EO × F < PO	77.10	<.001	.64
	EC > EO × C < F/PO	11.00	.002	.20
	EC > EO × L < R	4.26	.045	.09
	EC > EO × M > L/R	55.71	<.001	.56
	EC > EO × F < PO × L < R	4.92	.032	.10

	EC > EO × F < PO × M < L/R	55.09	<.001	.56
	EC > EO × C > F/PO × M > L/R	33.84	<.001	.44
Beta-2	EC > EO × F < PO	63.07	<.001	.59
	EC > EO × M > L/R	31.10	<.001	.42
	EC > EO × F < PO × M < L/R	62.52	<.001	.59

These overall resting state EEG and EC to EO reactivity topographies are comparable to prior research (Barry et al., 2007; Intrilligator and Polich, 1995; Karamacoska et al., 2018; Tenke et al., 2015).

S3.3 Prestimulus EEG. The topographic MANOVA effects obtained for pre-stimulus EEG amplitudes are presented in Table S3.3. Delta and theta shared a similar topography with amplitudes being largest at the midline, frontal and central regions; interacting to show frontocentral-midline enhancements. Both delta and theta amplitudes were greater in the right hemisphere, however, the frontal enhancement was greater on the left. Alpha-1 and alpha-2 amplitudes were greater parietal/occipitally, on the right, and were largest in the midline, especially centrally. A parietal/occipital enhancement in the hemispheres was also apparent, especially on the right. Beta-1 showed greater amplitudes centrally and in the midline, interacting for an enhancement in this region as well as in the frontal-midline. A right bias was also apparent, particularly in the parietal/occipital region. Beta-2 amplitudes showed a frontal dominance that contributed to a larger F/PO mean *cf.* the central region, and interacted to show an enhancement in the hemispheres. Across all bands, there were no significant stimulus (pre-Go vs. pre-NoGo) main effects (all $F \leq 2.56$, $p \geq .117$) or interactions involving stimulus and topography (all $F \leq 3.26$, $p \geq .078$). These topographic distributions of the prestimulus EEG are largely consistent with previous studies (De Blasio et al., 2013; Tenke et al., 2015; Min and Herrmann, 2007; Min and Park, 2010).

Table S3.3
Prestimulus EEG Topography

Band	Effect

		F	*p*	η_p^2
Delta	F > PO	42.11	<.001	.49
	C > F/PO	25.98	<.001	.38
	L < R	22.91	<.001	.35
	M > L/R	596.78	<.001	.93
	F > PO × L > R	25.06	<.001	.37
	F > PO × M > L/R	236.38	<.001	.85
	C > F/PO × M > L/R	116.72	<.001	.73
Theta	F > PO	36.18	<.001	.46
	C > F/PO	42.43	<.001	.50
	L < R	9.57	.003	.18
	M > L/R	541.61	<.001	.93
	F > PO × L > R	27.00	<.001	.39
	F > PO × M > L/R	310.09	<.001	.88
	C > F/PO × M > L/R	113.89	<.001	.73
Alpha-1	F < PO	31.19	<.001	.42
	L < R	25.59	<.001	.37
	M > L/R	190.65	<.001	.82
	F < PO × L < R	34.92	<.001	.45
	F < PO × M < L/R	54.41	<.001	.56
	C > F/PO × M > L/R	82.52	<.001	.66
Alpha-2	F < PO	40.51	<.001	.49
	L < R	23.09	<.001	.35
	M > L/R	158.14	<.001	.79
	F < PO × L < R	54.97	<.001	.56
	F < PO × M < L/R	16.67	<.001	.28
	C > F/PO × M > L/R	52.98	<.001	.55
Beta-1	C > F/PO	12.02	.001	.22
	L < R	6.49	.015	.13
	M > L/R	65.93	<.001	.61
	F < PO × L < R	22.98	<.001	.35
	F > PO × M > L/R	67.82	<.001	.61
	C > F/PO × M > L/R	29.68	<.001	.41
Beta-2	F > PO	21.30	<.001	.33
	C < F/PO	4.46	.041	.09
	C < F/PO × M < L/R	12.45	.001	.22

S3.4 Task-Related changes in EEG. Table S3.4 shows the topographic MANOVA outcomes for task-related change. An increase in EEG band amplitudes from the EO state to the prestimulus period was apparent (see also bottom row of Figure 2). Task-related delta and theta changes were dominant in the midline, being enhanced frontocentrally in this region. Sagitally, theta showed greater increases frontally and centrally. Laterally, the delta increase was greater in the right hemisphere *cf.* the left region, however, this was smaller frontally in both delta and theta. Alpha-1 and alpha-2 change showed increases that were larger parietal/occipitally, particularly in the hemispheres and especially on the right. The parietal/occipital dominance of the alpha-2 increase contributed to a larger F/PO mean *cf.* the central region. Alpha-1 and alpha-2 increases were greatest in the midline, particularly centrally. A midline increase was apparent for beta-1 change, particularly in the frontocentral regions. A slight enhancement in the parietal/occipital right region was also apparent. Beta-2 amplitudes increased largely frontally and in the hemispheres, interacting for a frontal enhancement in these regions. The increase was smaller centrally, especially in the hemispheres. The task-related EEG changes calculated for Go (TR-Go) and NoGo (TR-NG) did not differ (all main effects: $F < 2.56$, $p < .117$; all interactions involving stimulus and topography: $F \leq 3.26$, $p \geq .078$). These topographic distributions of task-related EEG change are consistent with our previous study (Karamacoska et al., 2018) but cannot be compared with prior work utilising bipolar channels (Arruda et al., 1995; Valentino et al., 1993). The amplitude increase, from the resting state to the task, remains consistent with these studies.

Table S3.4
Task-Related Changes in EEG Topography

Band	Effect	*F*	*p*	η_p^2
Delta	L < R	6.41	.015	.13
	M > L/R	44.01	<.001	.51
	F > PO × L > R	6.26	.016	.13
	F > PO × M > L/R	6.54	.014	.13

	C > F/PO × M > L/R	4.12	.048	.09
Theta	F > PO	7.99	.007	.16
	C > F/PO	8.10	.007	.16
	M > L/R	242.29	<.001	.85
	F > PO × L > R	11.27	.002	.21
	F > PO × M > L/R	39.18	<.001	.48
	C > F/PO × M > L/R	43.48	<.001	.50
Alpha-1	F < PO	37.81	<.001	.47
	M > L/R	105.71	<.001	.71
	F < PO × L < R	8.67	.005	.17
	F < PO × M < L/R	14.94	<.001	.26
	C > F/PO × M > L/R	51.87	<.001	.55
Alpha-2	F < PO	32.12	<.001	.43
	C < F/PO	4.55	.039	.10
	M > L/R	121.13	<.001	.74
	F < PO × L < R	13.06	.001	.23
	F < PO × M < L/R	5.80	.02	.12
	C > F/PO × M > L/R	50.52	<.001	.54
Beta-1	M > L/R	26.33	<.001	.38
	F < PO × L < R	5.13	.029	.11
	F > PO × M > L/R	12.34	.001	.22
	C > F/PO × M > L/R	14.80	<.001	.26
Beta-2	F > PO	18.25	<.001	.30
	C < F/PO	7.40	.009	.15
	M < L/R	5.27	.027	.11
	F > PO × M < L/R	5.33	.026	.11
	C < F/PO × M < L/R	6.37	.015	.13