University of Wollongong

Research Online

Faculty of Social Sciences - Papers (Archive)

Faculty of Arts, Social Sciences & Humanities

2013

Prestimulus alpha and beta determinants of ERP responses in the Go/NoGo task

Frances M. De Blasio

University of Wollongong, francesd@uow.edu.au

Robert Barry

University of Wollongong, rbarry@uow.edu.au

Follow this and additional works at: <https://ro.uow.edu.au/sspapers>

Part of the [Education Commons](#), and the [Social and Behavioral Sciences Commons](#)

Research Online is the open access institutional repository for the University of Wollongong. For further information contact the UOW Library: research-pubs@uow.edu.au

Prestimulus alpha and beta determinants of ERP responses in the Go/NoGo task

Abstract

The nature of the relationships between the level of immediately-prestimulus EEG activity and auditory ERP components remains unclear. Particularly, both inverse and direct relationships have been reported for the alpha band. Here we aim to clarify the pattern of prestimulus EEG contributions in alpha (8-13 Hz), and investigate those in beta (14-24 Hz), for five ERP components (P1, N1, P2, N2, P3) in an auditory equiprobable Go/NoGo paradigm. Separate FFTs were applied to the prestimulus Cz data of each accepted trial. The alpha and beta bands were independently assessed. The mean prestimulus spectral band amplitude was computed and used to sort the trials at nine central sites, and the upper and lower sorted trial thirds were averaged to form ERPs for Go and NoGo responses. Prestimulus EEG level effects (High vs. Low) were examined in each component's latency and amplitude, and Go reaction time was also assessed. Prestimulus alpha directly modulated the amplitude of the positive components (P1, P2, P3), while prestimulus beta directly modulated the positivity of the exogenous component amplitudes (P1, N1, P2); each amplitude effect occurred independently of the Go/NoGo stimulus conditions. Prestimulus beta also inversely modulated Go N1 latency; no reaction time effects were found for either band. The pattern of findings is intriguing and the various modulations are discussed in relation to attention and arousal. Together, these results confirm the importance of the EEG brain state immediately prestimulus, and indicate the considerable influence that these states have on event-related response processing.

Keywords

erp, alpha, responses, prestimulus, go, determinants, nogo, task, beta

Disciplines

Education | Social and Behavioral Sciences

Publication Details

De Blasio, F. M. & Barry, R. J. (2013). Prestimulus alpha and beta determinants of ERP responses in the Go/NoGo task. *International Journal of Psychophysiology*, 89 (1), 9-17.

Prestimulus alpha and beta determinants of ERP responses in the Go/NoGo task.

Frances M. De Blasio* and Robert J. Barry

Centre for Psychophysics, Psychophysiology, and Psychopharmacology; Brain & Behaviour
Research Institute; and School of Psychology, University of Wollongong,
Wollongong NSW 2522, Australia

*Corresponding author

Email: francesd@uow.edu.au

Phone: +61 2 4221 5547

Fax: +61 2 4221 4421

Address: School of Psychology, University of Wollongong, Wollongong NSW 2522, Australia

Abstract

The nature of the relationships between the level of immediately-prestimulus EEG activity and auditory ERP components remains unclear. Particularly, both inverse and direct relationships have been reported for the alpha band. Here we aim to clarify the pattern of prestimulus EEG contributions in alpha (8-13 Hz), and investigate those in beta (14-24 Hz), for five ERP components (P1, N1, P2, N2, P3) in an auditory equiprobable Go/NoGo paradigm. Separate FFTs were applied to the prestimulus Cz data of each accepted trial. The alpha and beta bands were independently assessed. The mean prestimulus spectral band amplitude was computed and used to sort the trials at nine central sites, and the upper and lower sorted trial thirds were averaged to form ERPs for Go and NoGo responses. Prestimulus EEG level effects (High vs. Low) were examined in each component's latency and amplitude, and Go reaction time was also assessed. Prestimulus alpha directly modulated the amplitude of the positive components (P1, P2, P3), while prestimulus beta directly modulated the positivity of the exogenous component amplitudes (P1, N1, P2); each amplitude effect occurred independently of the Go/NoGo stimulus conditions. Prestimulus beta also inversely modulated Go N1 latency; no reaction time effects were found for either band. The pattern of findings is intriguing and the various modulations are discussed in relation to attention and arousal. Together, these results confirm the importance of the EEG brain state immediately prestimulus, and indicate the considerable influence that these states have on event-related response processing.

Keywords: EEG/ERP dynamics; ERP genesis; Alpha; Beta; Auditory Go/NoGo; Attention; Arousal.

1. Introduction

The relationship between electroencephalography (EEG), a measure of brain state at any given time, and the event-related potential (ERP) indexing event-related sensory and cognitive processing, is of fundamental importance. The traditional models of ERP genesis differ in their account of this EEG–ERP relationship. The evoked model posits that the ERP is an evoked response occurring independently of, and adding to, the ongoing EEG activity, whereas the phase-reset model identifies stimulus-induced phase shifts in the ongoing EEG as producing the ERP (Barry, 2009; Fell et al., 2004; Jervis et al., 1983; Klimesch et al., 2007; Min et al., 2007; Sauseng et al., 2007). The difficulty in assessing the individual contributions of these proposed mechanisms is substantial (see Sauseng et al., 2007). There is evidence now suggesting that both evoked and phase-reset mechanisms are involved in ERP genesis (Min et al., 2007), and that the contributions from each mechanism differ by EEG band, ERP component, and stimulus-specific task requirements (Barry, 2009; Fell et al., 2004). Moreover, a third mechanism has recently emerged with evidence from magnetoencephalographic investigations; asymmetric modulations of ongoing oscillations, particularly those in the alpha band, are proposed to generate slow event-related potentials (Mazaheri and Jensen, 2008; van Dijk et al., 2010). Our primary concern here is not in assessing the models of ERP genesis, nor their mechanisms. As previous studies from our lab have investigated *phase* effects in this paradigm (in adults: Barry et al., 2010; and children: Barry and De Blasio, 2012), we focus here on mapping the empirically-testable relationships between the spectral *amplitude* of the ongoing EEG and the amplitude and latency of the ERP components.

The proposed mechanisms of ERP genesis are based on poststimulus EEG–ERP relationships, and we are not the first to reason an implied contribution from the prestimulus EEG (Min et al., 2007), our interest here. We consider that the within-task immediately-prestimulus EEG activity should provide an optimal picture of EEG–ERP relationships given the dynamic and fluctuating nature of brain states (c.f. ‘background’ EEG–ERP relationships; Intriligator and

Polich, 1994, 1995; Polich, 1997). In contrast to visual research, comparatively few investigations of the immediately-prestimulus EEG–ERP relationships have been conducted in the auditory domain. Moreover, while alpha is the most investigated of the EEG bands in this respect, the findings to date have been both conflicting and restricted to the midline sites (typically Cz and/or Pz). Inverse relationships have been reported between prestimulus alpha and N1 amplitude (Başar and Stampfer, 1985), N1-P2 peak-to-peak amplitude (Rahn and Başar, 1993), and P3 amplitude (Price, 1997). In contradiction, direct relationships also have been reported between prestimulus alpha and N1-P2 and N2-P3 amplitudes (Barry et al., 2000), and between prestimulus alpha and P3 amplitude (Jasiukaitis and Hakerem, 1988). The inconsistency in findings partly could be due to the variation in paradigms (single stimulus vs. oddball), stimulus probabilities (oddball vs. equiprobable), stimulus timing (varying vs. fixed, and long vs. short inter-stimulus-interval [ISI]), and task requirements (passive vs. count vs. button press). However, both Rahn and Başar (1993) and Price (1997) utilised paradigms in which the presentation of stimuli was contingent on the level of ongoing alpha activity (low vs. high), and hence much of the variation can be attributed to biofeedback-type confounds that may have been inadvertently introduced (Barry et al., 2000). Despite these discrepancies, there appears to have been no further auditory investigations of the prestimulus EEG *amplitude* – ERP relationships in alpha since 2000, and it appears that beta has not yet been assessed in this regard.

Following our earlier investigation of the prestimulus delta (1-3 Hz) and theta (4-7 Hz) band contributions in an equiprobable auditory Go/NoGo paradigm (De Blasio and Barry, in press), the present study examines the prestimulus alpha (8-13 Hz) and beta (14-24 Hz) band activity in this context. We aim to clarify and extend our understanding of the nature and strength of the prestimulus EEG–ERP relationships across the traditional EEG bands, examining five individual ERP components (P1, N1, P2, N2, and P3). The equiprobable paradigm facilitates the assessment of both Go and NoGo responses while minimising stimulus probability (Intriligator and Polich, 1994), and inhibition-related confounds (Lavric et al., 2004). The spectral amplitude

of the prestimulus EEG is examined at the vertex, and ERPs are derived at the vertex and eight surrounding scalp sites. Within-subject amplitude and latency effects of two levels of prestimulus EEG (High vs. Low) are assessed separately for each EEG band, ERP component, and stimulus condition. Performance effects are also examined; for each band, mean Go reaction times (RTs) across the High/Low prestimulus EEG level trials are compared.

Being the first investigation to assess the prestimulus alpha level effects in P1, it was uncertain what relationships, if any, might be found in this regard. Our use of an equiprobable paradigm is compatible with Barry et al. (2000) and so we expected to find significant and direct (i.e., proportional) relationships between prestimulus alpha level and the magnitude of the N1, P2, N2, and P3 component amplitudes. Moreover, no relationships were predicted between prestimulus alpha level and the latencies for each of these components (Barry et al., 2000). We were unable to find reports of prestimulus alpha–RT effects in the pertinent literature, and thus our assessment here was exploratory. Our examination of the impact of immediately-prestimulus beta effects was completely exploratory, although recent investigations of RT suggest that an inverse relationship might be found (Gola et al., 2012; Kamiński et al., 2012).

2. Method

2.1. Participants

Twenty (11 female, 9 male; 18 right-, 2 left-handed) healthy young adults aged 17-30 years ($M = 20.5$, $SD = 3.1$) were recruited from the University of Wollongong undergraduate Psychology research pool and participated to receive course credit. Each claimed normal hearing and a minimum of four hours caffeine abstinence prior to arrival. None reported recent psychoactive drug use or a history of seizures, severe head trauma, or psychiatric illness.

2.2. Physiological Recording

Continuous EEG was recorded from 19 sites using a cap with tin electrodes, in accordance with the international 10-20 placement system (Jasper, 1958). Electro-oculograms (EOGs) were recorded from electrodes placed beyond the outer canthus of each eye (horizontal), and above and

below the left eye (vertical). All electrode impedances were below 5 K Ω , and care was taken to match ear impedances. EEG was referenced to physically-linked ears and recorded with a gain of 20 000. EOG was recorded with a gain of 5 000. Data between 0.03 and 35 Hz were sampled at 512 Hz and recorded using a 16-bit A/D system (AMLAB II) for off-line analysis.

2.3. Task and Procedure

Two blocks of an equiprobable auditory Go/NoGo paradigm were presented binaurally via circumaural headphones. Each block consisted of 150 tone stimuli; 75 each of 1000 and 1500 Hz tones at 60 dB SPL, in randomised order. The tones were 50 ms in duration with 5 ms rise/fall time and a stimulus onset asynchrony (SOA) of 1,100 ms. The ‘target’ (Go) tone frequency was randomly counterbalanced across participants and required a button-press response from the dominant hand.

Participants read an information sheet, gave written informed consent, and completed a brief screening questionnaire. The physiological recording equipment was then fitted and the participants sat facing a computer monitor (CRT) located approximately 1 m ahead of them within an air-conditioned, sound-attenuated booth. To minimise eye artefact during data collection, participants were asked to fixate on a small cross appearing at the centre of the monitor. They were also encouraged to respond quickly and accurately to their designated Go tone. This procedure was approved by the University of Wollongong/Illawarra and South East Sydney Area Health Service Human Research Ethics Committee.

2.4. EEG Post-Processing and ERP Quantification

Following format conversion, Neuroscan software (Compumedics, Version 4.3) was used to epoch the data offline. Trials containing muscular or other artefact were identified via visual inspection and excluded from all further processing. MATLAB® (The Mathworks, R14SP3) and EEGLAB (Version 6.01b; Delorme and Makeig, 2004) were then utilised for the quantification of the remaining data. Each participant’s electrophysiological data were quantified separately for each frequency band (alpha and beta), and stimulus condition (Go and NoGo).

First, the Cz data stream was selected, and 1 s epochs were derived (from -500 ms). Response accuracy was assessed, and those trials found to have Go omissions ($RT > 500$ ms), or NoGo responses (commission errors), were identified and excluded from further processing steps. For the accepted trials, prestimulus epochs were derived and baselined across their 500 ms duration. The prestimulus epochs were doubled in length by reflecting the data within the time domain (i.e., 1:n, n:1) to improve the spectral resolution ($\Delta f = 1$ Hz), and overcome the Gibbs phenomenon (Pan, 2001). For each of the resultant epochs, now equivalent to 1 s data sets, a FFT was applied to transform the reflected data to the frequency domain, and the spectral band amplitude was computed as the sum of the FFT magnitude data across the corresponding frequency bins (alpha: 8-13 Hz; beta: 14-24 Hz). The spectral band amplitudes, representing the level of prestimulus EEG band activity at Cz, were used to rank the trials in ascending order and the sorting index was recorded.

The continuous raw EEG data were retrieved and the remaining procedure was applied to the data streams from each of the nine inner electrode sites (F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4). For the accepted trials (as identified in the Cz data), wide-band epochs (± 500 ms) were derived and baselined from -100 ms to onset, and the EEG band-specific sorting index was applied. The upper and lower thirds of the epochs, now sorted according to the ascending level of prestimulus Cz activity for the specified band, were separately averaged to produce ERPs for High and Low prestimulus activity levels in that frequency band. These ERPs were exported to Neuroscan for peak detection. The peak amplitudes of the components were identified within set latency ranges which were applied across Go/NoGo conditions, High/Low prestimulus EEG levels, nine electrode sites, and participants (P1: 25-140 ms; N1: 70-190 ms; P2: 100-270 ms; N2: 140-320 ms; and P3: 225-390 ms), as indicated in Figure 2 between the upper and lower panels. An automated function located the peaks/troughs within these periods, and an experienced ERP researcher visually inspected the selections and manually adjusted each as necessary. Note that the broad latency windows reflect the inter- and intra-subject variance in peak latencies.

2.5. Statistical Analysis

For each band, prestimulus EEG activity at the vertex was examined with a repeated-measures multivariate analysis of variance (MANOVA). The within-subject factors of Stimulus (Go vs. NoGo) and Level (High vs. Low prestimulus FFT band amplitude) were used to assess the appropriate separation of High/Low prestimulus EEG trials.

Latency and amplitude effects of each band were examined for five ERP components (P1, N1, P2, N2, and P3) via separate MANOVAs. The within-subject factors of Stimulus (Go vs. NoGo) and Level (High vs. Low prestimulus FFT band amplitude) were assessed, as were the Sagittal (Frontal, Central, Parietal) and Lateral (Left, Midline, Right) topographic dimensions. Planned topographic contrasts assessed regional effects within the 3 x 3 electrode array for each analysis. Sagittally, the frontal (F) and parietal (P) regions were compared, as were the central (C) and fronto-parietal regional mean (F/P). Lateral contrasts included hemispheric comparisons of the left (L) versus right (R) hemisphere and the midline (M) versus the hemispheric mean (L/R). As each contrast was planned and there were fewer contrasts than degrees of freedom for effect, Bonferroni type α adjustments were unnecessary (Tabachnick and Fidell, 2007).

Go RT was examined for prestimulus EEG effects via an F test with Level (High vs. Low prestimulus FFT band amplitude) as the single within-subject factor. Each analysis listed above was applied to the alpha and beta band datasets separately. All F tests are reported with (1,19) degrees of freedom. Note that near-significant ($.05 \leq \alpha \leq .10$) findings are reported in order to encourage further investigation, but only those effects reaching significance ($\alpha < .05$) are discussed.

3. Results

3.1. High vs. Low Prestimulus Alpha

The High/Low prestimulus alpha level EEG/ERP epochs were each derived from between 27 and 49 trials ($M = 38.4$, $SD = 5.7$). There was no difference in the number of

accepted trials in the Go ($M = 38.1$, $SD = 5.7$) and NoGo ($M = 38.7$, $SD = 5.8$) stimulus conditions ($F = 0.38$, $p = .547$, $\eta_p^2 = .02$).

3.1.1. Prestimulus EEG level.

The mean spectral amplitudes, derived from the FFT-decomposed prestimulus vertex epochs, are displayed in the upper panel of Figure 1 for the High/Low prestimulus alpha trials. There was no significant variation in spectral amplitudes between the Go and NoGo stimulus conditions ($F = 0.04$, $p = .834$, $\eta_p^2 = .00$), and no Level $\times$ Stimulus interaction ($F = 0.00$, $p = .951$, $\eta_p^2 = .00$). Level produced a main effect across the alpha band frequencies (High > Low: $F = 100.53$, $p < .001$, $\eta_p^2 = .84$); as seen in the upper panel of Figure 1, significantly increased spectral amplitudes were found for High compared to Low prestimulus alpha trials. This pattern of findings confirms the appropriate selection of High/Low prestimulus alpha trials.

Figure 1 about here.

3.1.2. Grand mean ERPs – Go/NoGo effects.

The grand mean ERP analysis across High/Low prestimulus alpha levels is not the focus of the present investigation, so only a brief summary of the results will be presented here. Refer to the Supplementary Material section S.1.1 for a complete report of the corresponding statistics.

The grand mean ERPs for accepted alpha trials were consistent with the typical response profiles for both Go and NoGo stimulus conditions. The ERP latencies failed to show Go/NoGo effects in P1 or N1. Go P2 latencies were significantly increased, Go N2 latencies were somewhat increased, and Go P3 latencies were significantly increased, compared to their corresponding NoGo responses. P1 amplitude showed no significant effect of Stimulus, while the frontal N1 was larger for Go than NoGo responses. P2 amplitudes were centro-parietal and were greater for Go than NoGo, particularly in the parietal region and the right hemisphere. N2 appeared relatively positive in the context of its surrounding peaks, and showed a strong frontal topography that was somewhat larger for NoGo than Go responses. Across conditions P3 was centro-parietal, yet it showed the parietal Go P3 and vertex NoGo P3 subcomponent separation consistent with prior

reports regarding this paradigm (Barry and Rushby, 2006).

3.1.3. ERP effects of prestimulus alpha levels.

The midline Go and NoGo ERPs, and the Go–NoGo difference waveforms, are shown in the upper panel of Figure 2 for High and Low prestimulus alpha levels. The broad latency ranges used to manually identify the peak amplitude for each component are indicated below this panel (Cz column). Across the Go/NoGo responses, P1 can be seen to peak at approximately 50 ms, followed by a dominant fronto-central N1 at approximately 100 ms. A prominent P3 can be seen to peak between 200 and 400 ms; Go P3 shows a centro-parietal distribution, while NoGo P3 responses were fronto-central. Note that although the P2 and N2 components are not evident in Figure 2, they were discernable at most sites in the individual participants' ERPs.

Figure 2 about here.

The mean ERP latency differences for High–Low prestimulus alpha levels are displayed in Table 1 (left) for the Go and NoGo responses. Prestimulus alpha level had no main effect on ERP latencies across the five components assessed (all $F \leq 1.74$, $p \geq .203$, $\eta_p^2 \leq .08$). High alpha was associated with increased N2 latencies in response to Go compared to NoGo stimuli, although this failed to reach significance (High > Low $\times$ Go > NoGo: $F = 3.70$, $p = .070$, $\eta_p^2 = .16$). None of the remaining components showed evidence of Level $\times$ Stimulus interactions (all $F \leq 2.47$, $p \geq .133$, $\eta_p^2 \leq .11$).

Table 1 about here.

The topographic distributions of the High–Low ERP component amplitude difference at nine analysed sites are displayed in the upper panel of Figure 3 for the mean across Go/NoGo stimuli (top row), and the separate conditions (bottom rows). For the enclosed headmaps, a solid black border denotes a main effect of prestimulus alpha Level, while a light grey border signifies a Level $\times$ Topography interaction.

Figure 3 about here.

P1 showed no main effect of Level ($F = 0.53$, $p = .476$, $\eta_p^2 = .03$). As seen in Figure 3

(Mean P1 headmap, upper panel), some parietal increase was apparent for High compared to Low prestimulus alpha (High > Low $\times$ F < P: $F = 3.10$, $p = .094$, $\eta_p^2 = .14$), and this reached significance in the right hemisphere (High > Low $\times$ L < R $\times$ F < P: $F = 8.93$, $p = .008$, $\eta_p^2 = .32$). No Level $\times$ Stimulus or Level $\times$ Stimulus $\times$ Topography interactions were found in P1 (all $F \leq 1.03$, $p \geq .322$, $\eta_p^2 \leq .05$). Although there appeared to be some High/Low and High/Low $\times$ Go/NoGo difference in N1 amplitudes across the midline (see Figure 2, upper panel), none of these approached significance (see N1 headmaps, Figure 3, upper panel); there was no main effect of Level ($F = 0.05$, $p = .828$, $\eta_p^2 = .00$), nor interactions involving Level, or Level and Stimulus (all $F \leq 2.67$, $p \geq .118$, $\eta_p^2 \leq .12$). P2 amplitudes were somewhat increased for High compared to Low prestimulus alpha (High > Low: $F = 3.14$, $p = .092$, $\eta_p^2 = .14$), and were significantly so in the right-central region (High > Low $\times$ L < R $\times$ C > F/P: $F = 5.46$, $p = .031$, $\eta_p^2 = .22$); see Figure 3 (Mean P2 headmap, upper panel). Failing to reach significance, Go P2 was somewhat increased for High compared to Low prestimulus alpha (High > Low $\times$ Go > NoGo: $F = 3.68$, $p = .070$, $\eta_p^2 = .16$), and more so in the midline (High > Low $\times$ Go > NoGo $\times$ M > L/R: $F = 3.58$, $p = .074$, $\eta_p^2 = .16$), while NoGo P2 appeared relatively unchanged between the alpha levels; see Go–NoGo difference waveforms (Figure 2, upper panel), and compare Go vs. NoGo P2 headmaps (Figure 3, upper panel).

In N2, there was neither a main effect of Level ($F = 0.68$, $p = .420$, $\eta_p^2 = .03$), nor higher-order interactions involving Level (all $F \leq 2.64$, $p \geq .121$, $\eta_p^2 \leq .12$). As evident in Figures 2 (Go and NoGo waveforms, upper panel) and 3 (Mean P3 headmap, upper panel), Level produced a main effect in P3, with amplitudes enhanced for High compared to Low prestimulus alpha (High > Low: $F = 6.08$, $p = .023$, $\eta_p^2 = .24$). Despite the suggested appearance of High/low $\times$ Go/NoGo interactions (ERPs: see Go–NoGo waveforms, Figure 2, upper panel; component distributions: compare Go vs. NoGo headmaps, Figure 3, upper panel), there were no significant higher-order interactions in P3 involving either Level, or Level and Stimulus (all $F \leq 0.46$, $p \geq .506$, $\eta_p^2 \leq .02$).

Go response performance showed no effect of prestimulus alpha ($F = 1.74, p = .203, \eta_p^2 = .08$), with comparable RTs found for the High ($M = 293.4, SD = 42.4$ ms) and Low ($M = 297.6, SD = 37.8$ ms) levels.

3.2. High vs. Low Prestimulus Beta

As with the prestimulus alpha separation, the number of trials contributing to the High/Low prestimulus beta level EEG/ERP epochs was comparable across stimulus conditions ($F = 0.38, p = .547, \eta_p^2 = .02$).

3.2.1. Prestimulus EEG level.

The mean spectral amplitudes at the vertex are displayed in the lower panel of Figure 1 for the High/Low prestimulus beta trials. The spectral amplitudes were comparable between the Go and NoGo stimulus conditions both across ($F = 0.48, p = .495, \eta_p^2 = .02$), and between ($F = 0.26, p = .617, \eta_p^2 = .01$), the High/Low prestimulus beta levels. High compared to Low prestimulus beta trials had significantly increased spectral amplitudes across the beta band frequencies (High > Low: $F = 162.97, p < .001, \eta_p^2 = .90$). Overall, this pattern validates the appropriate selection of High/Low prestimulus beta trials.

Increased prestimulus spectral amplitudes in some of the alpha band frequencies were also noted for the High vs. Low prestimulus beta trials (see lower panel Figure 1). Additional analyses revealed some covariation between the mean prestimulus alpha and beta band spectral amplitudes across all trials (Go: $r = .21$; NoGo: $r = .23$), although this did not approach significance (Go: $p = .386$; NoGo: $p = .328$).

3.2.2. Grand mean ERPs – Go/NoGo effects.

Again, only a brief summary of the grand mean ERP effects across accepted beta trials (i.e., across High/Low prestimulus levels) are presented here, with the details and supporting statistics reported in the Supplementary Material section S.1.2. Like the grand mean effects in alpha, those in beta exhibited common response profiles. For each component, the ERP latencies

failed to differ significantly between Go and NoGo responses, although P3 latencies were somewhat larger for Go than NoGo. Go P1 amplitudes were increased in the right-parietal region compared to NoGo P1. N1 was fronto-central and the frontal N1 was enhanced for Go relative to NoGo responses. P2 was centro-parietal and Go P2 showed regional amplitude enhancements in the right hemisphere, and in the parietal and central-right areas relative to NoGo P2. N2 was frontal despite its relatively positive appearance and the frontal (c.f. parietal) amplitude enhancement was greater for Go than NoGo. P3 showed a parietal topography across stimulus conditions, and again showed the typical Go/NoGo topographical separation (Barry and Rushby, 2006): Go P3 was maximal parietally, and NoGo P3 was maximal at the vertex.

3.2.3. ERP effects of prestimulus beta levels.

The separate Go (top row) and NoGo (middle row) ERPs, and the Go–NoGo difference waveforms (bottom row) are presented in Figure 2 (lower panel) for the High and Low prestimulus beta levels at each of the midline sites. The corresponding mean ERP latency differences (across participants and assessed sites) for High–Low prestimulus beta are reported in Table 1 (right). ERP latencies showed no main effect of prestimulus beta in any of the five components (all $F \leq 1.44$, $p \geq .246$, $\eta_p^2 \leq .07$). Four of the components (P1, P2, N2, and P3) also showed no Level $\times$ Stimulus interactions (all $F \leq 0.99$, $p \geq .331$, $\eta_p^2 \leq .05$). High prestimulus beta was associated with reduced Go N1 latency, and increased NoGo N1 latency (High $>$ Low $\times$ Go $<$ NoGo: $F = 4.40$, $p = .050$, $\eta_p^2 = .19$); this can be seen in both the latency differences for N1 reported in Table 1 (left panel), and in Figure 2 (bottom row, lower panel), particularly at Fz and Cz.

The ERP component amplitude differences for High–Low prestimulus beta levels are presented in the lower panel of Figure 3 for the mean across Go/NoGo stimuli (top row), and separate Go and NoGo responses (bottom rows). Coded borders are again used to identify significant Level effects: a light grey border signifies a Level $\times$ Topography interaction, a dashed black border indicates the concurrence of a main effect of Level and Level $\times$ Topography

interactions, and a dark grey border denotes a Level $\times$ Stimulus $\times$ Topography interaction.

Level produced a main effect in P1, with High prestimulus beta associated with increased amplitudes (High $>$ Low: $F = 4.59$, $p = .045$, $\eta_p^2 = .19$); this is evident in both the ERPs (Figure 2, lower panel) and component distributions (Figure 3, lower panel). As evident in Figure 3 (Mean P1 headmap, lower panel), this enhancement was larger in the left hemisphere (High $>$ Low $\times$ L $>$ R: $F = 4.94$, $p = .039$, $\eta_p^2 = .21$), and was greatest in the midline (High $>$ Low $\times$ M $>$ L/R: $F = 7.67$, $p = .012$, $\eta_p^2 = .29$). Go P1 was increased in the left-frontal region for High compared to Low prestimulus beta (High $>$ Low $\times$ Go $>$ NoGo $\times$ L $>$ R $\times$ F $>$ P: $F = 4.48$, $p = .048$, $\eta_p^2 = .19$); see the Go–NoGo ERP difference waveforms (Figure 2, lower panel) and the Go P1 headmap (Figure 3, lower panel). No main effect of Level was found in N1 ($F = 1.64$, $p = .215$, $\eta_p^2 = .08$), although the central amplitude enhancement was smaller for High compared to Low prestimulus beta (High $<$ Low $\times$ C $>$ F/P: $F = 7.09$, $p = .015$, $\eta_p^2 = .27$); see Figure 3 (Mean N1 headmap, lower panel). There was some indication of High/Low $\times$ Go/NoGo interactions in N1 (ERPs: see Go–NoGo difference waveform, Figure 2, lower panel; component distributions: compare Go vs. NoGo headmaps, Figure 3, lower panel); however, no higher-order interactions involving either Level, or Level and Stimulus, approached significance (all $F \leq 2.88$, $p \geq .106$, $\eta_p^2 \leq .13$). P2 also showed no main effect of Level ($F = 0.05$, $p = .820$, $\eta_p^2 = .00$). As seen in Figure 3 (Mean P2 headmap, lower panel), High prestimulus beta produced some hemispheric (c.f. midline) increase in P2 amplitude (High $<$ Low $\times$ M $>$ L/R: $F = 3.50$, $p = .077$, $\eta_p^2 = .16$), and this reached significance at central sites (High $<$ Low $\times$ M $>$ L/R $\times$ C $>$ F/P: $F = 5.83$, $p = .026$, $\eta_p^2 = .23$). Despite the suggested appearance of High/Low $\times$ Go/NoGo interactions in P2 (ERPs: see Go–NoGo difference waveform, Figure 2, lower panel; component distributions: see Go vs. NoGo headmaps, Figure 3, lower panel), interactions involving both Level and Stimulus (with/without Topography) failed to approach significance (all $F \leq 2.56$, $p \geq .126$, $\eta_p^2 \leq .12$).

There was no main effect of Level ($F = 0.01$, $p = .912$, $\eta_p^2 = .00$), nor Level $\times$ Topography

interactions in N2 (all $F \leq 2.78$, $p \geq .112$, $\eta_p^2 \leq .13$). As seen in Figure 3 (Go N2 headmap, lower panel), Go N2 was somewhat reduced in the right-frontal region for High compared to Low prestimulus beta, although this failed to reach significance (High < Low $\times$ Go > NoGo $\times$ L < R $\times$ F > P: $F = 3.80$, $p = .066$, $\eta_p^2 = .17$). P3 showed no main effect of Level ($F = 1.55$, $p = .228$, $\eta_p^2 = .08$), and there were no interactions involving either Level, or Level $\times$ Stimulus (all $F \leq 2.92$, $p \geq .104$, $\eta_p^2 \leq .13$).

Prestimulus beta produced no effects in mean RT, with similar performance seen for the High ($M = 296.9$, $SD = 44.2$ ms) and Low ($M = 294.4$, $SD = 39.4$ ms) levels assessed ($F = 1.01$, $p = .329$, $\eta_p^2 = .05$).

4. Discussion

The relationships between EEG activity present at the vertex immediately prestimulus and the resulting ERP components at nine sites were assessed here separately for the alpha and beta bands. The value of this investigation thus relies upon the appropriate separation of the trials based on the sum of the spectral amplitudes of the narrow 1 Hz frequencies contributing to each prestimulus EEG band assessed. Figure 1 clearly validates the separation of the High/Low prestimulus trials for both the alpha (upper panel), and beta (lower panel) bands; the prestimulus activity in the corresponding band frequencies differed significantly between the High/Low band separations, and did so in the appropriate direction (i.e., High > Low). Moreover, there was minimal and non-significant shared variance in prestimulus spectral alpha and beta band amplitude separations. For each 1 Hz frequency, it can be seen that the prestimulus activity did not significantly differ between Go/NoGo stimuli, including those frequencies involved in the High/Low band separations. Level effects in this study can therefore be attributed to the manipulation of prestimulus EEG activity in the corresponding band, and are not caused by the interaction of the prestimulus activity in both bands, nor frequency differences between Go and NoGo conditions.

4.1. High vs. low prestimulus alpha

ERP latency showed no significant effect of prestimulus alpha level across the five components assessed. This finding is consistent with the earlier work of Barry et al. (2000), and confirms that the level of prestimulus alpha activity is not a determining factor for component latencies in this paradigm.

Prestimulus alpha level was found to be a significant determinant of the ERP amplitudes for each of the positive components assessed (P1, P2, and P3). In each instance the nature of the resulting amplitude modulations was direct, that is, High prestimulus alpha produced amplitude increases. This finding in P1 is novel within the literature, while the direct prestimulus alpha–P2, and prestimulus alpha–P3 amplitude relationships are each compatible with the findings of Barry et al. (2000). Importantly, Barry et al. (2000) noted that the N1-P2 and N2-P3 peak-to-peak amplitude effects each appeared to be attributable to effects occurring solely in the positive components, and our pattern of results confirms this. Our findings in P3 are also in agreement with those of Jasiukaitis and Hakerem (1988), indicating the robust nature of this relationship given the differences in paradigms (equiprobable Go/NoGo with fixed short SOA vs. paired stimulus Oddball with varying long ISI), and trial sorting sites (Cz vs. Pz) between the investigations.

The direct prestimulus alpha–positive component amplitude relationships were each independent of stimulus condition. This was the first investigation to assess all five components concurrently, but also independently, and has uncovered an intriguing pattern of findings. When considered together, it is tempting to postulate that these positive component modulations are the result of some common factor, such as a general state effect. However, it is difficult to conceive of a single process that would produce an effect restricted to the positive components, and to do so across such a wide latency range (~50 – 320 ms).

EEG alpha is a measure of resting-state arousal (Barry et al., 2007), and arousal has been linked to amplitude modulations in both sensory and cognitive ERP components (Crowley and Colrain, 2004; Fruhstorfer and Bergström, 1969; Näätänen and Picton, 1987). Functionally, alpha

oscillations are considered to inhibit task-irrelevant processing regions (Foxe and Snyder, 2011; Klimesch, 2012; Weisz et al., 2011), whereby effective disengagement serves to gate and direct the flow of information to the task-relevant regions for optimal processing (Jensen and Mazaheri, 2010). Alpha oscillations are known to respond to various cognitive tasks (i.e., perceptual, working and long term memory, and attention), and also show preparatory modulations (Foxe and Snyder, 2011; Jensen and Mazaheri, 2010; Weisz et al., 2011). This latter finding has prompted the hypothesis that alpha sub-serves higher level cognitive processes and is argued to be mediated by, or closely associated with attention – particularly selective (Foxe and Snyder, 2011; Weisz et al., 2011), or anticipatory and temporal (Klimesch, 2012). Attentional suppression of alpha activity in the associated sensory processing areas has also been reported in the absence of alpha increases in unattended processing areas, suggesting that alpha activity can serve to index attentional bias (Thut et al., 2006). Extending on these findings, Mathewson and colleagues (2009; 2011) proposed the ‘pulsed-inhibition’ account, whereby alpha oscillations modulate cortical excitability about a sensory detection threshold, resulting in alternating phases of inhibition (suppressed processing) and excitation (enhanced processing). In this account, alpha acts as a mechanism (c.f. correlate) by which attentional control and inhibitory influences are expressed (Mathewson et al., 2009, 2011). Accordingly, Mathewson et al. (2009; 2011) have found that sensory processing outcomes are determined by the amplitude of the ongoing alpha activity (large vs. small), but also by the phase of the oscillations when attention is reduced (i.e., large alpha amplitude only). These interpretations are primarily drawn from visual research; although there is some indication that alpha’s inhibitory role is universal across modalities (Weisz et al., 2011). There is some evidence that arousal-related alpha is functionally distinct from attention-related alpha, and that they each have a different neural basis; thalamus vs. cortico-cortical network (particularly frontal and parietal) and possible thalamo-cortical influence, respectively (Foxe and Snyder, 2011). We assessed prestimulus alpha at the vertex only, preventing us from attributing the ERP effects found here to either arousal or selective/anticipatory

attention. The alpha oscillation/attention literature also provides a secondary interpretation. It is plausible that the prestimulus alpha activity modulated (a) the subsequent cognitive processes themselves, as reflected in the associated ERP components, or (b) the lower-level processes (i.e., the sensory representation of the stimulus) which sub-serve the higher cognitive processes. The latter possibility cannot be evaluated here so we continue by assessing the former.

The direct amplitude modulations occurred in the right-parietal region in P1, in the right-central region in P2, and across sites in P3. These topographical differences suggest different underlying cortical processes that may be influenced by some common mechanism; most likely attention or arousal, given their close association with alpha oscillations. The focal nature of the direct modulation in P1 supports a processing-related effect. P1 reflects perceptual processing (Näätänen and Picton, 1987) and resource allocation (Kok, 1997), and each of these is known to be sensitive to attentional effects (Kok, 1997; Näätänen and Picton, 1987). The topography of the P1 modulations also overlaps some attentional processing areas (Cabeza and Nyberg, 2000; Coull, 1998), providing further support. Of the different types of attention known to modulate the ERP components, temporal orienting has been inversely related to P1 amplitude at frontal sites (Rimmele et al., 2011). Our use of a fixed SOA could have facilitated anticipatory effects which might account for both the High prestimulus alpha level (i.e., preparatory inhibition), and the parietal increase (i.e., frontal reduction) in P1.

The direct prestimulus alpha–P2 amplitude modulation suggests increased poststimulus activation of processing-related resources; activity in the central-right region is typically associated with episodic memory retrieval, particularly context memory, which stores temporal and other information (Cabeza and Nyberg, 2000). This component is known to vary inversely with both attention and arousal (Crowley and Colrain, 2004), hence it is plausible that the prestimulus alpha–P2 relationship is attributable to attentional effects in the form of temporal expectancy. Rimmele et al. (2011) did not assess the P2 component in their investigation of temporal orienting effects, and so this is worthy of further study.

Prestimulus alpha directly modulated P3 amplitudes in the absence of topographic and/or stimulus condition interactions, which could be taken to suggest a global arousal effect (Barry et al., 2007). However, P3 amplitudes are typically considered to represent stimulus event categorisation, and this process is conceptualised as being directly modulated by both attention and working memory (Kok, 1997, 2001). Moreover, Rimmele et al. (2011) reported an increase in P3 amplitude for temporal expectancy conditions; together this suggests support for a link (in this modality) between prestimulus alpha and temporal attention effects broadly consistent with the alpha oscillation literature.

4.2. High vs. low prestimulus beta

Prestimulus beta produced an inverse modulation in Go N1 latency. The amplitudes of the exogenous components (P1, N1, and P2) were each significantly modulated by the level of prestimulus beta activity. The amplitude effects were most pronounced in P1, where the nature of the modulation was direct. This was found across sites and conditions, but was particularly prominent in the left hemisphere, in the midline sites, and also in the left-frontal region for Go P1. Across stimulus conditions, prestimulus beta inversely modulated N1 amplitudes at central sites, and directly modulated P2 amplitudes in the central hemispheric regions.

Each of our prestimulus beta effects are novel, and when considered together, the reduction in Go N1 latency and increased positivity in the amplitudes of the exogenous components could be taken to indicate the effects of a single mechanism or perceptual process. One possible mechanism, accounting for the restriction of effects to the exogenous components, is the view of *poststimulus* beta as a correlate of a higher order scanning mechanism, which is present from stimulation and continues until the structure of the stimulus is resolved (Giannitrapani, 1971). It is unclear, however, what effect prestimulus beta activity would have on this mechanism, and how the effects would be expressed in the exogenous components. Compared with alpha, the functional role of beta oscillations is less studied, although there is evidence that it reflects

functional inhibition in motor-cortical regions (Jensen and Mazaheri, 2010). Again we turn to ERP-based interpretations to infer the influence of prestimulus beta.

The sensitivity of the exogenous components to the effects of attention (Crowley and Colrain, 2004; Kok, 1997; Näätänen and Picton, 1987) and arousal (Crowley and Colrain, 2004; Fruhstorfer and Bergström, 1969; Näätänen and Picton, 1987) has been long reported. Furthermore, recent investigations have found empirical evidence suggesting that prestimulus beta activity is directly associated with anticipatory attention processes and arousal (Gola et al., 2012; Kamiński et al., 2012). It follows then that the prestimulus beta effects found here in each of the exogenous components are attributable to attentional orienting and alertness via a direct relationship.

The direct modulation in P1 amplitude was found across sites and stimulus conditions, suggesting that High prestimulus beta produced a global increase in cortical arousal; but also showed topographic specificity, suggesting that High beta further increased the poststimulus activation in processing-related regions (Barry et al., 2007). These findings are broadly consistent with the amplitude gains noted in the early perceptual components when there are increases in attention and/or arousal (Kok, 1997; Näätänen and Picton, 1987). However, the stimulus-specific modulation, apparent in Go P1 amplitude (direct), and Go N1 latency (inverse), were each unexpected during the sensory processing stage. An intuitive account of these stimulus effects is that the equiprobable nature of the task increased the perceived likelihood of the occurrence of a Go-NoGo, rather than a Go-Go sequence, reducing the level of attentiveness following each Go stimulus event. The left-frontal Go P1 topography offers some support for this interpretation as it partially overlaps a region implicated in attentional processes, including stimulus-response compatibility and divided attention (Cabeza and Nyberg, 2000).

Prestimulus beta inversely modulated N1 component amplitudes regionally, suggesting a processing-specific effect such as attention. Given that the attentional networks are largely fronto-parietal (Cabeza and Nyberg, 2000), the topographical shift in N1 amplitude (i.e.,

becoming less central) could be considered to reflect the deactivation of non-attentional processing areas, resulting in more efficient processing (i.e., via improved resource allocation). This interpretation is consistent with the sensory processing gain predicted with increased attention (Kok, 1997; Näätänen and Picton, 1987), and indicates a direct link between prestimulus beta activity and attention, and also between prestimulus beta and efficient sensory processing.

The direct prestimulus beta–P2 amplitude modulations are also focal (central hemispheric regions). Activations within these areas are largely associated with attentional processes, particularly stimulus-response compatibility and orientation (Cabeza and Nyberg, 2000). Together this suggests an association between High prestimulus beta and more efficient activation of attentional resources (i.e., High beta $\rightarrow$ increased P2 $\leftrightarrow$ increased attentional processing). This interpretation contradicts the reported inverse relationship between P2 amplitude and general attention effects (Crowley and Colrain, 2004), although given the range of attentional types, it is possible that these may produce differential effects in P2.

Interestingly, our prestimulus beta amplitude findings in P1 and N1 are in direct opposition to the temporal expectancy effects reported by Rimmele et al. (2011). This suggests that one or more differing attentional process(es) underlie our effects in beta. Also, although RT performance effects have been associated with beta band activity (Gola et al., 2012; Kamiński et al., 2012), we found no evidence of prestimulus beta–RT effects. This difference in findings is not surprising given the differences in task modality (auditory vs. visual; Gola et al., 2012), and quantification grouping criteria (prestimulus EEG beta vs. RT performance; Kamiński et al., 2012).

4.3. Summary and Conclusion

Here we found prestimulus alpha to be a direct determinant of the positive component amplitudes (P1, P2, and P3). High prestimulus alpha was associated with the processing of perceptual and event categorisation processes, and the effects in this band were attributed to

temporal expectancies arising from the use of a fixed SOA. Prestimulus beta was identified as a determinant of the exogenous components, inversely modulating Go N1 latency and directly modulating component amplitudes (P1, N1, and P2). High prestimulus activity in the beta band was significantly implicated in more efficient perceptual processing, and the modulations of these components were interpreted primarily in terms of attention (P1, N1, and P2), but also arousal (P1) effects.

Overall we found distinct patterns of prestimulus EEG contributions across the traditional bands, emphasising the importance and differential influence of the brain state immediately prestimulus on the resulting ERP component measures. This significant influence of the ongoing EEG suggests support for the phase-reset model of ERP genesis, and also the proposed asymmetric amplitude modulation mechanism. Our findings, particularly the prestimulus beta effects, are broadly consistent with Barry's (2009) findings suggesting that phase-reset mechanisms contribute to the earlier exogenous components, while evoked activity is more likely to modulate the later endogenous components. However, it must be noted that the methodology used here precludes us from commenting on possible contributions from the evoked model.

Many of the findings here are novel and warrant replication. Importantly, the interpretation of effects in both the alpha and beta bands in relation to attentional processes should be further explored. Considering that there are several forms of attention (i.e., sustained, selective, divided, attentional orientation, temporal, and stimulus-response compatibility), and interactions between attention and arousal (Coull, 1998), it would be useful for future investigations to include paradigm manipulations of attentional type and cortical arousal in order to attempt to discern the contributions from each. It would also be useful if future research assessed the prestimulus EEG activity at multiple sites, particularly in regard to alpha; this could be used to explore, and possibly separate, arousal-related and attention-related alpha oscillation effects. Also regarding alpha, future investigations should consider the phase of the oscillations together with the amplitude, and assess their joint effects on the subsequent ERP outcomes in the context of the pulsed-inhibition

theory.

This study has extended and clarified our understanding of the empirical relationships between immediately-prestimulus EEG activity, in the alpha and beta bands, and the ERP components. An improved understanding of these relationships across the traditional EEG bands is of fundamental importance and may eventually provide a means of understanding normative and/or deficient EEG function in clinical and developmental populations.

References

- Barry, R.J., 2009. Evoked activity and EEG phase resetting in the genesis of auditory Go/NoGo ERPs. *Biol. Psychol.* 80, 292-299.
- Barry, R.J., Clarke, A.R., Johnstone, S.J., Magee, C.A., Rushby, J.A., 2007. EEG differences between eyes-closed and eyes-open resting conditions. *Clin. Neurophysiol.* 118, 2765-2773.
- Barry, R.J., De Blasio, F.M., 2012. EEG-ERP phase dynamics of children in the auditory Go/NoGo task. *Int. J. Psychophysiol.* 86, 251-261.
- Barry, R.J., De Blasio, F., Rushby, J.A., Clarke, A.R., 2010. Brain dynamics in the auditory Go/NoGo task as a function of EEG frequency. *Int. J. Psychophysiol.* 78, 115-128.
- Barry, R.J., Kirkaikul, S., Hodder, D., 2000. EEG alpha activity and the ERP to target stimuli in an auditory oddball paradigm. *Int. J. Psychophysiol.* 39, 39-50.
- Barry, R.J., Rushby, J.A., 2006. An orienting reflex perspective on anteriorisation of the P3 of the event-related potential. *Exp. Brain Res.* 173, 539–545.
- Başar, E., Stampfer, H. G., 1985. Important associations among EEG-dynamics, event-related potentials, short-term memory and learning. *Int. J. Neurosci.* 26, 161-180.
- Cabeza, R., Nyberg, L., 2000. Imaging cognition II: An empirical review of 275 PET and fMRI studies. *J. Cogn. Neurosci.* 12, 1-47.
- Coull, J.T., 1998. Neural correlates of attention and arousal: Insights from electrophysiology, functional neuroimaging and psychopharmacology. *Prog. Neurobiol.* 55, 343-361.
- Crowley, K.E., Colrain, I.M., 2004. A review of the evidence for P2 being an independent component process: Age, sleep and modality. *Clin. Neurophysiol.* 115, 732-744.
- De Blasio, F.M., Barry, R.J. in press. Prestimulus delta and theta determinants of ERP responses in the Go/NoGo task. *Int. J. Psychophysiol.*
- Delorme, A., Makeig, S., 2004. EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J. Neurosci. Methods* 134, 9-21.

- Fell, J., Dietl, T., Grunwald, T., Kurthen, M., Klaver, P., Trautner, P., Schaller, C., Elger, C.E., Fernández, G. 2004. Neural bases of cognitive ERPs: more than phase reset. *J. Cogn. Neurosci.* 16, 1595-1604.
- Foxe, J.J., Snyder, A.C., 2011. The role of alpha-band brain oscillations as a sensory suppression mechanism during selective attention. *Front. Psychol.* 2, 154.
- Fruhstorfer, H., Bergström, R.M., 1969. Human vigilance and auditory evoked responses. *Electroenceph. Clin. Neurophysiol.* 27, 346-355.
- Giannitrapani, D., 1971. Scanning mechanisms and the EEG. *Electroenceph. Clin. Neurophysiol.* 30, 139-146.
- Gola, M., Kamiński, J., Brzezicka, A., Wróbel, A., 2012. Beta band oscillations as a correlate of alertness – Changes in aging. *Int. J. Psychophysiol.* 85, 62-67.
- Intriligator, J., Polich, J., 1994. On the relationship between background EEG and the P300 event-related potential. *Biol. Psychol.* 37, 207-218.
- Intriligator, J., Polich, J., 1995. On the relationship between EEG and ERP variability. *Int. J. Psychophysiol.* 20, 59-74.
- Kamiński, J., Brzezicka, A., Gola, M., Wróbel, A., 2012. Beta band oscillations engagement in human alertness process. *Int. J. Psychophysiol.* 85, 125-128.
- Klimesch, W., 2012. Alpha-band oscillations, attention, and controlled access to stored information. *Trends Cogn. Sci.* 16, 606-617.
- Jasiukaitis, P., Hakerem, G. 1988. The effect of prestimulus alpha activity on the P300. *Psychophysiology* 25, 157-165.
- Jasper, H.H., 1958. The ten-twenty electrode system of the International Federation. *Electroenceph. Clin. Neurophysiol.* 10, 371-375.
- Jensen, O., Mazaheri, A., 2010. Shaping functional architecture by oscillatory alpha activity: Gating by inhibition. *Front. Hum. Neurosci.* 4, 186.

- Jervis, B.W., Nichols, M.J., Johnson, T.E., Allen, E., Hudson, N.R., 1983. A fundamental investigation of the composition of auditory evoked potentials. *IEEE Trans. Biomed. Eng.* 30, 43-50.
- Klimesch, W., Sauseng, P., Hanslmayr, S., Gruber, W., Freunberger, R., 2007. Event-related phase reorganization may explain evoked neural dynamics. *Neurosci. Biobehav. Rev.* 31, 1003-1016.
- Kok, A., 1997. Event-related-potential (ERP) reflections of mental resources: A review and synthesis. *Biol. Psychol.* 45, 19-56.
- Kok, A., 2001. On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology* 38, 557-577.
- Lavric, A., Pizzagalli, D.A., Forstmeier, S., 2004. When 'go' and 'nogo' are equally frequent: ERP components and cortical tomography. *Eur. J. Neurosci.* 20, 2483-2488.
- Mathewson, K.E., Gratton, G., Fabiani, M., Beck, D.M., Ro, T., 2009. To see or not to see: Pre-stimulus α phase predicts visual awareness. *J. Neurosci.* 29, 2725-2732.
- Mathewson, K.E., Lleras, A., Beck, D.M., Fabiani, M., Ro, T., Gratton, G., 2011. Pulsed out of awareness: EEG alpha oscillations represent a pulsed inhibition of ongoing cortical processing. *Front. Psychol.* 2, 99.
- Mazaheri, A., Jensen, O., 2008. Asymmetric amplitude modulations of brain oscillations can generate slow evoked responses. *J. Neurosci.* 28, 7781-7787.
- Min, B.-K., Busch, N.A., Debener, S., Kranczioch, C., Hanslmayr, S., Engel, A.K., Herrmann, C.S., 2007. The best of both worlds: Phase-reset of human EEG alpha activity and additive power contribute to ERP generation. *Int. J. Psychophysiol.* 65, 58-68.
- Näätänen, R., Picton, T., 1987. The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology* 24, 375-425.

- Pan, C., 2001. Gibbs phenomenon removal and digital filtering directly through the Fast Fourier Transform. *IEEE Trans. Signal Process.* 49, 444-448.
- Polich, J., 1997. On the relationship between EEG and P300: Individual differences, aging, and ultradian rhythms. *Int. J. Psychophysiol.* 26, 299-317.
- Price, G.W. 1997. The effect of pre-stimulus alpha activity on the auditory P300 paradigm: A prospective study. *Brain Topogr.* 9, 169-176.
- Rahn, E., Başar, E., 1993. Prestimulus EEG-activity strongly influences the auditory evoked vertex response: A new method for selective averaging. *Int. J. Neurosci.* 69, 207-220.
- Rimmele, J., Jolsvai, H., Sussman, E., 2011. Auditory target detection is affected by implicit temporal and spatial expectations. *J. Cogn. Neurosci.* 23, 1136-1147.
- Sauseng, P., Klimesch, W., Gruber, W.R., Hanslmayr, S., Freunberger, R., Doppelmayr, M., 2007. Are event-related potential components generated by phase resetting of brain oscillations? *Neuroscience* 146, 1435-1444.
- Tabachnick, B.G., Fidell, L.S., 2007. Using multivariate statistics. Boston: Pearson Education, Inc.
- van Dijk, H., van der Werf, J., Mazaheri, A., Medendorp, W.P., Jensen, O., 2010. Modulations in oscillatory activity with amplitude asymmetry can produce cognitively relevant event-related responses. *PNAS* 107, 900-905.
- Thut, G., Nietzel, A., Brandt, S.A., Pascual-Leone, A., 2006. α -band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection. *J. Neurosci.* 26, 9494-9502.
- Weisz, N., Hartmann, T., Müller, N., Lorenz, I., Obleser, J., 2011. Alpha rhythms in audition: Cognitive and clinical perspectives. *Front. Psychol.* 2, 73.

Table 1.

Mean (standard deviation) of the High–Low prestimulus EEG level difference in ERP latencies (ms) across participants and nine assessed sites.

	α		β	
	Go	NoGo	Go	NoGo
P1	2.2 (15.7)	3.4 (13.4)	0.0 (16.9)	0.7 (13.8)
N1	-2.0 (16.2)	0.3 (20.3)	-8.4 (15.7)	2.9 (21.6) *
P2	5.6 (37.2)	-9.6 (33.8)	2.2 (38.9)	-0.3 (31.9)
N2	11.2 (37.7)	-11.0 (39.7)	2.9 (46.9)	0.3 (31.7)
P3	8.1 (31.3)	-5.9 (36.1)	9.0 (35.7)	-1.3 (32.7)

*High/Low $\times$ Go/NoGo interaction; $p < .05$.

Figure Legends

Figure 1. Mean prestimulus EEG spectra of the High/Low prestimulus alpha trials (upper panel), and the High/Low prestimulus beta trials (lower panel). All spectra are derived from the vertex with 1 Hz resolution, and both Go and NoGo trials are represented. Significant variations in mean spectra between the High/Low prestimulus levels are indicated by a bar above the constituent band frequencies (alpha: 8-13 Hz; beta: 14-24 Hz). *** $p < .001$.

Figure 2. Waveforms of the High (black) and Low (grey) prestimulus EEG separations in alpha (upper panel), and beta (lower panel) are shown for the individual Go (top row) and NoGo (middle row) responses, and the Go–NoGo difference waveform (bottom row) for the midline sites. The plot between the upper and lower panels displays the broad latency windows used in the manual identification of the peak component amplitudes (ERP labels and latency windows plotted in grey); these were uniformly applied across the Go/NoGo conditions, High/Low prestimulus levels, and nine electrode sites for each subject.

Figure 3. ERP component topographies for the High–Low prestimulus alpha difference (upper panel) and High–Low prestimulus beta difference (lower panel) at nine sites (F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4; indicated in the top left headmap of each panel). The High–Low prestimulus difference in the component mean across Go/NoGo conditions is shown (top) above the separate Go and NoGo distributions (bottom). Borders identify those components with a significant ($p < .05$) main effect of Level (solid black), Level $\times$ Topography interaction (light grey), co-occurring Level main effect and Level $\times$ Topography interaction (dashed black), or Level $\times$ Stimulus $\times$ Topography interaction (grey). Amplitude scales for the increase/decrease in component amplitude are shown below the corresponding headmaps.

Figure 1.

Figure 2.

Figure 3.

S.1. Grand mean ERPs – Go/NoGo effects

S.1.1. Across High/Low Prestimulus Alpha.

The grand mean accepted alpha trial ERPs, across High/Low prestimulus alpha levels, are presented for the midline sites in the upper panel of Figure S1. The separate Go and NoGo responses are shown (top row) in addition to the Go–NoGo difference waveforms (bottom row), and the broad latency ranges used in the manual identification of the component peaks are also indicated (grey latency windows appear below the α panel). P1 can be seen to peak at approximately 50 ms, and is followed by a strong fronto-central N1 at approximately 100 ms. Although P2 and N2 are not clearly visible in Figure S1, they were apparent in the participant level ERPs. A prominent P3 is seen at each of the midline sites and shows a fronto-central topography for NoGo, and a parietal topography for Go responses. A rising negativity was also apparent at centro-parietal sites across the -500 ms prestimulus period (not shown here); this was interpreted as the recovery of the P3 component rather than a CNV, due to its non-frontal, P3-like distribution.

Table S1 displays the mean and standard deviation latencies (across participants and sites) of the grand mean accepted alpha trial ERPs. P1 latencies showed no significant variation between Go and NoGo responses ($F = 0.62, p = .440, \eta_p^2 = .03$). Likewise, N1 latencies were comparable across stimulus conditions ($F = 0.72, p = .405, \eta_p^2 = .04$). Go P2 latencies were significantly increased compared to NoGo P2 responses (Go > NoGo: $F = 4.48, p = .048, \eta_p^2 = .19$). N2 latencies were also somewhat increased for Go than NoGo responses, although this failed to reach significance (Go > NoGo: $F = 4.11, p = .057, \eta_p^2 = .18$). P3 latencies were significantly greater for Go than NoGo responses (Go > NoGo: $F = 6.49, p = .002, \eta_p^2 = .25$).

The topographies of the grand mean component amplitudes for the accepted alpha trials are presented in the upper panel of Figure S2; the Go and NoGo distributions are

individually displayed in the top rows, and the Go–NoGo response difference is shown below these. Stimulus effects reaching significance are indicated for the corresponding headmaps via colour coded borders: green identifies a Stimulus $\times$ Topography interaction, while purple signifies the co-occurrence of a main effect and one or more Stimulus $\times$ Topography interactions.

Topographically, P1 amplitudes were somewhat larger in the midline ($M > L/R$: $F = 3.82$, $p = .066$, $\eta_p^2 = .17$), and although there was no main effect of Stimulus ($F = 0.35$, $p = .561$, $\eta_p^2 = .02$), the midline dominance was somewhat reduced for Go compared to NoGo responses ($Go < NoGo \times M > L/R$: $F = 3.10$, $p = .095$, $\eta_p^2 = .14$). N1 was maximal in the frontal ($F > P$: $F = 42.76$, $p < .001$, $\eta_p^2 = .69$), and central ($C > F/P$: $F = 4.70$, $p = .043$, $\eta_p^2 = .20$) regions. The frontal N1 topography was greater in response to Go than NoGo stimuli ($Go > NoGo \times F > P$: $F = 8.06$, $p = .011$, $\eta_p^2 = .30$); this can be seen in the upper panel of Figures S1 (waveforms) and S2 (topographical headmaps). P2 was larger in the parietal ($F < P$: $F = 24.18$, $p < .001$, $\eta_p^2 = .56$), central ($C > F/P$: $F = 23.96$, $p < .001$, $\eta_p^2 = .56$), and midline ($M > L/R$: $F = 27.59$, $p < .001$, $\eta_p^2 = .59$) regions. P2 was maximal in the midline-parietal region ($M > L/R \times F < P$: $F = 7.23$, $p = .015$, $\eta_p^2 = .28$), and at the vertex ($M > L/R \times C > F/P$: $F = 5.49$, $p = .030$, $\eta_p^2 = .22$). Stimulus produced a main effect in P2, with greater amplitudes found in response to Go than NoGo stimuli ($Go > NoGo$: $F = 4.53$, $p = .047$, $\eta_p^2 = .19$). As illustrated in the Go–NoGo P2 difference headmap (Figure S2), this effect was significantly larger in the parietal region ($Go > NoGo \times F < P$: $F = 6.58$, $p = .019$, $\eta_p^2 = .26$), in the right hemisphere ($Go > NoGo \times L < R$: $F = 5.78$, $p = .027$, $\eta_p^2 = .23$), and was somewhat increased in the right-central region ($Go > NoGo \times L < R \times C > F/P$: $F = 3.07$, $p = .096$, $\eta_p^2 = .14$).

Given the nature of its surrounding peaks, N2 amplitudes appeared relatively positive (refer upper panel, Figures S1 and S2). N2 showed a strong frontal topography ($F > P$: $F =$

36.49, $p < .001$, $\eta_p^2 = .66$, and $C < F/P$: $F = 46.99$, $p < .001$, $\eta_p^2 = .71$) that was enhanced in the hemispheres ($M < L/R \times F > P$: $F = 22.69$, $p < .001$, $\eta_p^2 = .54$, and $M < L/R \times C < F/P$: $F = 4.57$, $p = .046$, $\eta_p^2 = .19$). N2 amplitudes were greater in the right than left hemisphere ($L < R$: $F = 6.30$, $p = .021$, $\eta_p^2 = .25$), and were reduced in the midline ($M < L/R$: $F = 11.66$, $p = .003$, $\eta_p^2 = .38$). As seen in Figure S2, NoGo N2 responses were somewhat larger (i.e., more negative) than Go N2 across sites ($Go < NoGo$: $F = 3.03$, $p = .098$, $\eta_p^2 = .14$). It can also be seen that the vertex reduction was greater for NoGo N2 ($Go < NoGo \times M < L/R \times C < F/P$: $F = 4.60$, $p = .045$, $\eta_p^2 = .19$), and the frontal enhancement smaller ($Go > NoGo \times F > P$: $F = 12.76$, $p = .002$, $\eta_p^2 = .40$). The Go/NoGo effects in N2 appear to have been influenced by the dominant P3 response (note the similarities between the N2 and P3 distributions in the upper panel of Figure S2). P3 was centro-parietal ($C > F/P$: $F = 74.71$, $p < .001$, $\eta_p^2 = .80$, and $F < P$: $F = 30.60$, $p < .001$, $\eta_p^2 = .62$), and enhanced in the midline ($M > L/R$: $F = 60.51$, $p < .001$, $\eta_p^2 = .76$). In the left hemisphere the parietal enhancement was larger ($L > R \times F < P$: $F = 6.11$, $p = .023$, $\eta_p^2 = .24$), and in the midline, the parietal and central enhancements were both greater ($M > L/R \times F < P$: $F = 31.48$, $p < .001$, $\eta_p^2 = .62$, and $M > L/R \times C > F/P$: $F = 13.64$, $p = .002$, $\eta_p^2 = .42$, respectively). Go P3 was larger in the left hemisphere ($Go > NoGo \times L > R$: $F = 4.86$, $p = .040$, $\eta_p^2 = .20$), and in the parietal region ($Go > NoGo \times F < P$: $F = 60.73$, $p < .001$, $\eta_p^2 = .76$); see the Go–NoGo difference headmap, Figure S2. The parietal Go P3 enhancement was increased in the left hemisphere and in the midline ($Go > NoGo \times L > R \times F < P$: $F = 11.37$, $p = .003$, $\eta_p^2 = .37$, and $Go > NoGo \times M > L/R \times F < P$: $F = 9.44$, $p = .006$, $\eta_p^2 = .33$, respectively). NoGo P3 was larger in the midline ($Go < NoGo \times M > L/R$: $F = 7.01$, $p = .016$, $\eta_p^2 = .27$), and maximal at the vertex ($Go < NoGo \times M > L/R \times C > F/P$: $F = 28.56$, $p < .001$, $\eta_p^2 = .60$). This topographical separation of the parietally dominant Go P3, and anteriorisation of the NoGo P3, is clearly evident in both the ERPs (Figure S1) and component topographies (Figure S2); consistent with prior reports regarding

this paradigm (Barry & Rushby, 2006).

S.1.2. Across High/Low Prestimulus Beta.

The grand mean Go and NoGo midline response, and the Go–NoGo difference waveforms of the accepted beta trial ERPs are presented in the lower panel of Figure S1. Three of the five components are again clearly evident: P1 at ~50 ms; N1 at ~100 ms; and P3 between 200 and 400 ms. The P2 and N2 components, although not distinguishable in Figure S1, were identifiable in the ERPs for each participant. A rising prestimulus negativity was also present at centro-parietal sites (not shown in Figure S1); as in the grand mean alpha ERPs, this was interpreted as the P3 component recovery (c.f. CNV).

The mean Go and NoGo latencies (across participants and sites) are presented in Table S1 (right) for each component. The latencies of the P1, N1, P2, and N2 components showed no main effect of Stimulus (all $F \leq 2.27$, $p \geq .148$, $\eta_p^2 \leq .11$). Go P3 latencies were somewhat increased in comparison to NoGo P3, although this failed to reach significance (Go > NoGo: $F = 3.96$, $p = .061$, $\eta_p^2 = .17$).

The grand mean amplitude topographies are illustrated for each component in the lower panel of Figure S2; the separate Go and NoGo responses are shown above the Go–NoGo difference. A green border is again used to indicate significant Stimulus $\times$ Topography interactions. Comparison of the grand mean accepted alpha and grand mean accepted beta data (ERPs: upper vs. lower panels of Figure S1; component topographies: upper vs. lower panels of Figure S2) indicates substantial similarity, but not identity. This is appropriate given that the grand averages for each dataset are derived from different trials selected from the same sample. Due to this similarity, only those statistics unique to the beta data, or those differing in their level of significance (significant vs. near significant), are reported below.

In P1, Stimulus ($F = 1.33, p = .263, \eta_p^2 = .07$) and Topographical (all $F \leq 1.36, p \geq .258, \eta_p^2 \leq .07$) effects each failed to reach significance, although P1 amplitudes were increased in the right-parietal region for Go relative to NoGo responses ($\text{Go} > \text{NoGo} \times \text{L} < \text{R} \times \text{F} < \text{P}$: $F = 12.70, p = .002, \eta_p^2 = .40$); see Go–NoGo P1 headmap, Figure S2. As seen in Figure S2, N1 amplitudes were fronto-central, and the frontal N1 increase was greater for Go than NoGo responses. Go N1 was somewhat larger than NoGo N1 across sites ($\text{Go} > \text{NoGo}$: $F = 3.61, p = .073, \eta_p^2 = .16$), and somewhat more so at the vertex ($\text{Go} > \text{NoGo} \times \text{M} > \text{L/R} \times \text{C} > \text{F/P}$: $F = 3.31, p = .085, \eta_p^2 = .15$), although neither of these additional effects reached significance. P2 was larger in the centro-parietal and midline regions, greater in the midline-parietal region, and was largest at the vertex. Go P2 was larger than NoGo P2 in the parietal region, and in the right hemisphere, particularly the right-central region ($\text{Go} > \text{NoGo} \times \text{R} > \text{L} \times \text{C} > \text{F/P}$: $F = 10.03, p = .005, \eta_p^2 = .35$); refer to the Go–NoGo P2 headmap, Figure S2. However, there was no indication of a main effect of Stimulus in P2 ($F = 1.23, p = .281, \eta_p^2 = .06$).

Again, N2 showed a strong frontal topography despite its relatively positive appearance. N2 was reduced in the midline, particularly in the midline-parietal region, and somewhat so at the vertex ($\text{M} < \text{R/L} \times \text{C} < \text{F/P}$: $F = 3.72, p = .069, \eta_p^2 = .16$), and was enhanced in the right hemisphere. Here there was no evidence of a main effect of Stimulus ($F = 0.29, p = .594, \eta_p^2 = .02$). It can be seen in Figure S2 that the frontal enhancement was greater for Go than NoGo N2, and more so in the hemispheres ($\text{Go} > \text{NoGo} \times \text{M} < \text{L/R} \times \text{F} > \text{P}$: $F = 6.12, p = .023, \eta_p^2 = .24$). The central reduction was somewhat greater for NoGo compared to Go N2 responses ($\text{Go} < \text{NoGo} \times \text{C} < \text{F/P}$: $F = 3.44, p = .079, \eta_p^2 = .15$), as was the vertex reduction ($\text{Go} < \text{NoGo} \times \text{M} < \text{L/R} \times \text{C} < \text{F/P}$: $F = 3.69, p = .070, \eta_p^2 = .16$). P3 amplitudes were maximal in the parietal region, somewhat more so in the left hemisphere ($\text{L} > \text{R} \times \text{F} < \text{P}$: $F = 4.31, p = .052, \eta_p^2 = .18$), in the midline, and in the midline-parietal region.

P3 was also greater in the central region, and at the vertex. The topographical Go/NoGo P3 separation was again seen (ERPs: Figure S1; component topographies: Figure S2). The Go–NoGo P3 difference headmap (Figure S2) clearly indicates that Go P3 amplitudes were larger in the left hemisphere, and were greater parietally, in both the left- and midline-parietal regions. NoGo P3 was somewhat increased in the midline, and was greatest at the vertex. NoGo P3 was also increased in the central region here (Go < NoGo $\times$ C > F/P: $F = 6.91$, $p = .017$, $\eta_p^2 = .27$), further supporting the anteriorisation of the NoGo P3.

Table S1.

Mean (standard deviation) of the grand mean (across High/Low prestimulus EEG level) ERP latencies (ms) across participants and nine assessed sites.

	α		β	
	Go	NoGo	Go	NoGo
P1	55.4 (12.0)	57.1 (12.8)	52.2 (12.2)	55.2 (11.9)
N1	103.4 (16.9)	101.0 (18.6)	102.0 (15.9)	105.1 (20.5)
P2	180.8 (31.6)	165.7 (35.4) *	184.6 (30.2)	178.2 (33.2)
N2	219.4 (39.4)	201.9 (39.3)	230.4 (39.5)	216.4 (42.5)
P3	311.5 (35.2)	293.2 (36.1) *	317.2 (35.2)	295.9 (35.2)

*Go/NoGo contrast; $p < .05$.

Fig. S1. Waveforms of the grand mean accepted alpha (upper panel), and grand mean accepted beta (lower panel) ERPs at the midline sites. The separate Go and NoGo responses are shown above the Go-NoGo difference. The Go and NoGo peak amplitudes of each component were manually identified within the labelled broad latency windows displayed between the panels (Cz column; windows applied across participants and electrode sites).

Fig. S2. Component topographies of the grand mean accepted alpha (upper panel) and beta (lower panel) trials at nine sites (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4; indicated in the top left headmaps). Separate Go and NoGo distributions are shown above the Go–NoGo difference. Colour-coded borders denote a significant ($p < .05$) Stimulus × Topography interaction (green), or the joint occurrence of a main effect of Stimulus and Stimulus × Topography interaction (purple).