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# Neurophysiological markers of alert responding during goal-directed behavior: A high-density electrical mapping study

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**The ability to dynamically modulate the intensity of sustained attention (i.e., alertness) is an essential component of the human executive control system, allowing us to function purposefully in accordance with our goals. In this study we examine high-density ERP markers of alert responding during the fixed sequence sustained attention to response task ($SART_{fixed}$). This paradigm has proven to be a sensitive clinical metric in patient populations with deficits in their ability to sustain attention (e.g., attention deficit hyperactivity disorder). In this task subjects withhold a button press to an infrequent no-go target ('3') embedded within a predictable sequence of numbers ('1' to '9'). Our data reveal a complex pattern of effects across the trial sequence of the SART, with clear contributions from frontal and parietal cortices to sustained attentional performance. Over occipito-parietal regions, early visual attention processes were increased during trial 2 (i.e., trial in which the digit '2' was presented) and trial 3, giving rise to the so-called selection negativity (SN). Two prominent late components were manifest during trial 2: LP1 (550–800 ms) and LP2 (850–1150 ms) over occipito-parietal and central sites. We interpret the LP1 component on trial 2 as reflecting retrieval of the task goal and the subsequent LP2 as reflecting competition between the currently relevant go response and the subsequent no-go response. On trial 3, an enhanced "no-go N2" (250–450 ms) was seen fronto-centrally in the absence of the "no-go P3" that typically follows. Fronto-polar activity was also seen across all trials and may be indicative of subgoal processes to integrate the association between stimulus and goal. Prior to a lapse of attention (i.e., failure to inhibit a response to "3") the LP1 was significantly attenuated on the preceding trial 2 indicating a failure of anticipatory goal-directed processing. The results are discussed in terms of models of sustained attention involving frontal and parietal cortices.**


*Keywords:* Sustained attention; Go/no-go task; High-density ERPs; Lapses of attention; Early visual attention; Selection negativity; Goal-directed behavior; Delayed intentions

## Introduction

A driver approaches a crossroads, notes the red light and pulls to a stop. Harried and worn out from a fairly dismal day at the office, his eyes are fixed on the lights; his only thoughts are for home, slippers, a Manhattan and an evening in front of the TV. The lights, after what feels like an age, change back to green and he hits his accelerator, relieved to be on his way again. Unfortunately, he has somehow neglected to notice that the car in front of him has yet to pull away. This, he ultimately understands, only as his car's forward progress is all too abruptly arrested by the rear fender of the car in front. Such momentary lapses in attentional re-allocation are unfortunately all too commonplace in life. Avoiding them relies particularly on our ability to sustain attention and to flexibly redeploy our attention to additional environmental factors that might be relevant. This latter function often requires reactivation of a subgoal, outside the immediate spotlight of attention. That is, the primary goal above is to go when the light is green, but an important subgoal is to ensure that there is nothing in the way. Although the example above is of a relatively benign circumstance where a lapse in attention results in an inappropriate response to current environmental circumstances, such lapses can also have catastrophic consequences. For example, a considerable proportion of major road traffic accidents can be attributed to momentary lapses in goal maintenance—that is, distraction or lapses in sustained attentional mechanisms (see, e.g., National survey of distracted and drowsy driving attitudes and behaviors: 2002[1]). As such, it is of great interest to understand the neural mechanisms that support sustained attention and subgoal activation.

Sustained attention requires an intrinsic maintenance of the alert state in the absence of exogenous inputs (Posner and DiGirolamo,

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[1] The Gallup organization (2002). National Survey of Distracted and Drowsy Driving Attitudes and Behaviors: 2002, http://www.nhtsa.dot.gov/people/injury/drowsy_driving1/survey-distractive03/index.htm.

2000; Posner and Peterson, 1990; Sturm et al., 1999). Lapses of sustained attention occur when there is a transient reduction in the alert state that can give rise to momentary loss of endogenous control over behavior. Recent positron emission tomography (PET) studies (Sturm et al., 1999, 2004) suggest that an extended right hemisphere network is involved in sustained attention including the right anterior cingulate, the right dorsolateral prefrontal cortex, the right inferior parietal lobule with projections to the thalamus and noradrenergic brainstem targets. Sturm and colleagues propose that right hemisphere brain structures exercise top-down control, via the thalamus, on noradrenergic structures in the brainstem.

A task that has proven very effective in assessing this type of attentive responding is the sustained attention to response task (SART) (Manly et al., 1999, 2002, 2003; Robertson et al., 1997). In one version of this task, a predictable series of single digits are presented (1–9) and subjects are required to make a response to each number (go trials) with the exception of the number 3 (no-go trial). A PET study showed that this task increased activation in both the right dorsolateral prefrontal cortex and the right superior/posterior parietal cortex compared to a more challenging version of the SART in which the numbers were presented randomly (Manly et al., 2003). These findings suggest that right fronto-parietal regions are responsible for maintaining a goal-directed focus in unarousing contexts where exogenous stimuli are not present to increase alertness through novelty, demand or perceived difficulty (Robertson and Garavan, 2004).

The SART has also proven to be a sensitive clinical measure, discriminating between traumatically brain injured (TBI) patients and healthy control subjects (Dockree et al., 2004; Manly et al., 2003; McAvinue et al., 2005; Robertson et al., 1997) and between ADHD children and controls (Shallice et al., 2002). Clinical groups exhibit increased errors of commission (false presses on the 3) during this task and fail to show anticipatory slowing on the trials before the upcoming no-go trial, a general finding in subjects who are successful at this task (Dockree et al., 2004), suggesting a loss of endogenous control at strategically important points during the task. Although, the functional anatomical correlates of the SART have been investigated (Fassbender et al., 2004; Manly et al., 2003; O'Connor et al., 2004), only one study (Dockree et al., 2004) has examined the electrophysiological dynamics during the task. In the latter study, alpha (~10 Hz) desynchronization was observed in healthy controls before the no-go trial. By contrast, TBI patients failed to show this modulation. This state of desynchronization has been associated with increased attentive processing (Klimesch et al., 1998; Mulholland, 1965; Pfurtscheller and Lopes da Silva, 1999) in the transition from a relaxed to an alert state, and with anticipatory preparation of visual cortices during selective attention tasks (Foxe et al., 1998; Fu et al., 2001; Worden et al., 2000). No studies to date have characterized the broad-band ERP componentry of the SART in neurologically normal adults.

In the present study, we utilize the excellent temporal resolution of high-density electrical mapping to examine the spatiotemporal dynamics of electro-cortical activity during the fixed sequence SART (hereafter referred to as the $SART_{fixed}$). Our first aim was to examine event-related potentials (ERPs) and their topographical distributions during periods of accurate sustained attention performance described as 'successful runs' of trials 1 through 9 (i.e., when subjects successfully withhold responses to trial 3). In previous investigations of the $SART_{fixed}$, adequate characterization of errors of commission has not been possible due to the rarity of their occurrence. In this study we addressed this limitation by testing subjects over the course of a full day (with regular breaks) so that adequate numbers of errors were committed. A criticism of this approach pertains to the ecological validity of the task. Can long-term engagement with a laboratory task over ~108 min relate to a more naturalistic situation? We argue that the task has features in common with everyday scenarios that require this kind of sustained attentional effort over long periods. For example, taking a long distance trip in the car interspersed with regular breaks may require similar periods of sustained attention to critical events during largely routine behavior. The SART also correlates with everyday reported cognitive failures in patients with traumatic brain injury (Robertson et al., 1997) suggesting that the propensity for attentional lapses on the task is related to greater everyday slips of attention.

Accordingly, as a second aim, we conducted an exploratory analysis of correct withholds and commission errors as well as the trials that preceded and followed these responses. The current study, in neurologically healthy subjects, will provide an important baseline for future understanding of clinical populations and their documented sustained attention deficits on this task. We acknowledge that we would not be able to test clinical groups for long periods of time due to excessive fatigue. However, in these groups the number of errors mounts up much more rapidly, circumventing the need to test for long periods.

We outline a number of predictions regarding the ERP componentry during the critical anticipatory period before the upcoming no-go target that will serve as important markers for alert responding during the task. On trial 2, we predict that early visual attentional processes will be mobilized because of the significance of this trial as an upcoming cue for the critical target trial. The most commonly reported attentional modulation is that of the P1 and N1 components that show increased amplitudes when spatial attention is directed to the stimulus location (i.e., stimuli are validly cued) compared to when it is directed elsewhere (Mangun and Hillyard, 1991; Mangun et al., 1987). In contrast, selectively attending to relevant visual features has been shown to elicit the so-called selection negativity (SN) (Anllo-Vento and Hillyard, 1996; Harter et al., 1984; Kenemans et al., 1993; Smid et al., 1999; Molholm et al., 2004). Although the $SART_{fixed}$ is not a selective visual feature attention paradigm per se, it is reasonable to propose that similar processes of visual selection might be engaged as the relevance of trials in the sequence increase before the critical target trial. Indeed, previous work has demonstrated that increased ventral-stream visual object-recognition processes underlying SN can be elicited by relevant visual stimuli when relevance is defined on the basis of a non-spatial feature(s) (Anllo-Vento and Hillyard, 1996; Harter et al., 1984; Kenemans et al., 1993; Smid et al., 1999).

We further predict that the recollection of the task goal ("withhold response to 3") will be critical on trial 2. Research investigating the dynamics of prospective remembering (West et al., 2001; West and Krompinger, 2005) has shown that the realization of a delayed intention is associated with two ERP modulations: an N300 and a "prospective positivity". They propose that the N300 is associated with detection of prospective memory cues and is seen as a phasic negativity over occipito-parietal scalp between 300 and 500 ms. Additionally, the later prospective positivity, seen as a broadly distributed positivity (500–1000 ms) over parietal areas, may reflect neural processes

supporting the recollection of a task goal. Interestingly, the later phase of the prospective positivity (600–800 ms) is not influenced by the salience of the prospective cue suggesting that the prospective positivity is elicited under different circumstances than the more commonly observed parietal P3b—a component that is observed over parietal scalp in the context of target detection and that is augmented by target distinctiveness (Comerchero and Polich, 1998).

From trials 2 to 3, subjects must switch between a go response to a withhold response. Wylie et al. (2003) have examined task switching during a paradigm in which subjects regularly alternate between two tasks (categorizing numbers and categorizing letters) on every third trial. On the preceding trial to a switch trial, a period of sustained positivity was observed over bilateral parietal regions. This effect was interpreted in the context of a "competition" model in which either the currently relevant task set is suppressed or the subsequent trial task set is activated, or both. It is probable that competition needs to be resolved in the context of different task sets during the $SART_{fixed}$ and there will be sustained positivity that is indicative of this competition in the transition between responses on trials 2–3.

Two possibilities can be proposed regarding the sensory processing that the target (trial 3) stimulus might receive. One possibility is that since this stimulus is specifically not to be responded to, sensory processing of trial 3 might actually be suppressed, with visual attentional resources being withdrawn from the stimulus. The second, and perhaps more plausible prediction, is that trial 3 will be processed in a state of high visual attentiveness so that subsequent subgoal engagement can occur. These two possibilities lead to distinct predictions regarding the sensory-evoked activity that will be generated to trial 3. If processing of trial 3 is selectively suppressed, then the early P1–N1 complex of the VEP should be reduced in amplitude. Alternately, if trial 3 receives extra visual attentional processing, then the pattern of activity during sensory processing should parallel that seen during trial 2; that is, we should see attentional enhancement and an SN. Further, we also expect that trial 3 will result in generation of the N2–P3 complex that has frequently been observed during the presentation of no-go stimuli (Bekker et al., 2004; Kok, 1986; Pfefferbaum et al., 1985). The N2 component has been interpreted as reflecting conflict monitoring during infrequent trials whereas the P3 is more associated with response inhibition or evaluation of an erroneous response after response execution (Kok et al., 2004).

In addition to evaluating the predictions above, we performed a second exploratory analysis as a means of fully characterizing the richness of our data set and as a hypothesis generation tool for future research. We asked whether there are different ERP time courses prior to and during a correct withhold versus prior to and during a commission error.

## Methods

### *Subjects*

Fourteen (six female) right-handed neurologically normal volunteers participated. They were paid $100 for 1 day of participation. Subjects were aged between 18 and 32 years (mean = 23.86, SD ± 4.24). All subjects gave written informed consent, and the Institutional Review Board of the Nathan Kline Institute approved the procedures. All subjects reported normal or corrected-to-normal vision.

### *SART paradigm and procedure*

Digits were presented sequentially from '1' through '9'. For each block, 225 digits were presented sequentially (25 of each of the nine digits) over a period of ~4.7 min. Subjects undertook as many blocks as possible over the course of a day with regular breaks as required. Subjects were seated in a dimly lit, sound-attenuated, electrically shielded room. Subjects completed, on average, 24.4 blocks (range: 13–30 blocks). For each trial, a digit was presented for 150 ms followed by an inter-stimulus interval (ISI) that varied randomly between 1000 and 1500 ms. A variable ISI was included to prevent subjects succumbing to a speed accuracy trade-off that can occur when ISIs are regularly paced. Subjects were instructed to respond with a left mouse button press with their right forefinger upon presentation of each digit (go trials) with the exception of the 25 occasions per block when the digit 3 (target) appeared, where they were required to withhold their response.

Five randomly allocated digit sizes were presented to increase the demands for processing the numerical value and to minimize the possibility that subjects would set a search template for some perceptual feature of the target trial ('3'). Digit font sizes were 100, 120, 140, 160 and 180 in Arial text. The five allocated digit sizes subtended 1.39°, 1.66°, 1.92°, 2.18° and 2.45°, respectively, in the vertical plane, at a viewing distance of 152 cm. Digits were presented 0.25° above a central yellow fixation cross on a grey background. The task specifications were programmed and stimuli were delivered using the Presentation® software package (Version 0.75, http://www.neurobs.com).

### *Measurements*

High-density EEG recordings were acquired from 128 scalp electrodes (inter-electrode spacing ~2.4 cm) referenced to the nasion. Electrophysiological data were recorded in AC mode with a gain of 1000 and a band pass of 0.15–100 Hz and the A/D conversion rate was 500 Hz. Recording began when electrical impedance had been reduced to less than 5 kΩ at all scalp sites. Vertical eye movements were recorded with two VEOG electrodes placed above and below the left eye, while HEOG electrodes at the outer canthus of each eye recorded horizontal movements.

### *Analysis strategy*

A twofold approach was adopted to examine the data. First, we selected epochs for each trial during the SART, thus trials 1–9 were examined independently. Only correct behavioral responses were analyzed to monopolize the high data yield for this type of response, thus producing a well-characterized average waveform consisting of, on average, 611 trials after all artifact rejection. Second, each trial was analyzed as a function of response. Of particular interest were trials preceding and including a correct withhold on the no-go trial and trials preceding and including false presses (or commission errors) on the no-go trial. Because of the relatively low data yield for commission errors compared to correct withholds it was important to equate the number of single trials that contributed to the averages for these comparisons. Consequently, withholds and commission errors and the trials that preceded them

were randomly matched to ensure correct withholds were not over-represented in these comparisons. This led to an average of approximately 40 trials per condition.

*ERP morphology*

We investigated the componentry of the ERPs over occipito-parietal, central and frontal scalp following the strategy outlined in Wylie et al. (2003). Accordingly, the ERP component structure was assessed by visual inspection of overall data. First, the presence of early components P1, N1, P2, N2 and the P3-like component P450 was verified through inspection of grand average composite waveforms (i.e., collapsed across all trials 1–9 of the SART). For the P1, N1, P2 and N2 components, subject-specific maximal amplitude scalp locations and peak latencies at these locations were identified from individual composite waveforms. This accounts for any spatial variation in the expression of these components across individuals—we argue that VEP components are the summation of many simultaneously active cortical generators that will not be evoked in the same spatially distinct location for each individual (see, e.g., Foxe and Simpson, 2002; Murray et al., 2001). The latency and electrode sites for the P450 component were identified from the grand average composite waveforms and thus were fixed for all individuals. Second, later ERP components were defined by identifying periods of divergence in the grand average ERP time courses separated by trial. In particular, late positivities, LP1 and LP2, were identified as key components due to their divergence during critical trials (1, 2 and 3) compared to non-critical ("nested") trials 5 through 8.

For each of the components P1, N1, P2 and N2 we measured amplitude at the selected subject-specific occipito-parietal scalp location for that component and grand-averaged these amplitudes across individuals. The remaining later ERP components (P450 and late positivities, LP1 and LP2) had broader scalp distributions with much less or no spatial variation in topography between individuals. Thus these component amplitudes were measured at fixed electrode locations. Component amplitude measures for each electrode and each trial were derived by calculating the area under the average ERP waveform (compared to the 0-mV baseline) within a latency window centered on the peak latency of that component. The width of the latency window was chosen depending on the duration and spatial extent of each component process. Latency bins of ±5 ms were used surrounding peak P1 and N1 latencies. For the P2 and N2 a slightly larger window (±10 ms) was used as these are slower components, and for the P450 a fixed window from 400 to 500 ms was used. The interval 550–800 ms was chosen as the latency window for the LP1 component, and 850–1150 ms for LP2.

For the first five components (P1, N1, P2, N2 and P450) a one-way repeated-measures ANOVA was conducted comparing trial type. Later components (LP1 and LP2) showed variations in morphology as a function of region (over fronto-polar, central and occipito-parietal sites) and therefore Trial × Region repeated-measures ANOVAs were conducted to test this. The area measures defined above were the dependent variables for each statistical test.

While the use of broadly defined component peaks is a good means of limiting the number of statistical tests that are conducted, as mentioned above, these components often represent the activity of many simultaneously active brain generators at any given moment. As such, effects may not necessarily be coincident with a given component peak, especially in the scenario that only a subset of the brain generators responsible for producing a given component are affected by a given experimental condition. Thus, limiting the analysis to a set of discrete component peaks represents a very conservative approach to the analysis of high-density ERP data.

*ERP topography*

Scalp topographic maps were produced using the EEGLAB toolbox (Delorme and Makeig, 2004). These represent interpolated field potential distributions, derived from 128-channel measurement. First, for each of the components analyzed, a general topography is presented, averaged across all trials and referred to hereafter as the composite topography. This allowed us to illustrate the distribution of activity for each component irrespective of trial. Second, when statistically significant effects were found between trials for a particular component, difference topographies were derived by making subtractions between trials, for example, trial 2 minus trial 1. In all scalp topographic maps, amplitude is integrated over the latency window of the component in question.

*Statistical cluster analysis*

ERP componentry was also examined as a function of response (correct withhold, error of commission). We performed statistical cluster analysis on the data. Using this technique, we calculate pointwise, paired, two-tailed $t$ tests between correct withholds and commission errors for all time points across all scalp sites, and display the $P$ values as a two-dimensional statistical color-scaled map (see Molholm et al., 2002). This was done for the target trials themselves (i.e., 3) and for the 9, 1 and 2 that preceded. This approach gives an assessment of significant effects of response type across the entire epoch. A strict significance level of 0.01 is used for this stage of the analysis. We refer the reader to other advocates of this approach and a full discussion of the merits and disadvantages of statistical cluster plots (Guthrie and Buchwald, 1991; Wylie et al., 2003). In agreement with these authors we argue the use of statistical cluster plots provides a useful means of estimating the onset and offset of ERP effects and primarily serves as a useful hypothesis generation tool.

## Results

*Behavioral results*

Errors committed during the task were categorized as commission errors defined as a false press on the target trial ('3'). We adopted conservative inclusion criteria for commission errors and rejected those that were preceded by or followed by a non-response, reasoning that these non-genuine commission errors may reflect an early or late go trial response. Owing to the different number of blocks completed by subjects, percentage error scores were calculated. Mean percentage commission errors = 9.47 (SD = 5.77).

First, the patterns of RTs during periods of accurate sustained attention performance were analyzed. Consequently, errorless trial sequences of '1' through '9' were selected for analysis. A one-way repeated measures analysis of variance revealed an effect of trial

type [$F(7,84) = 3.9$, $P < 0.001$]. Repeated contrasts revealed significant lengthening of response times (RTs) from '9' to '1' ($P < 0.0001$) and a significant shortening of RTs between '1' and '2' ($P < 0.007$). It is notable that there was a trend towards lengthening RTs between '2' and '4' ($P = 0.072$) and further shortening of RTs between '4' and '5' ($P = 0.063$). No other contrasts reached near significance. The RT findings suggest that significant changes in stimulus processing occur in anticipation of the target stimulus (see Fig. 1A). Second, RT patterns were examined as a function of response. Trials '1' through '9' were categorized as belonging to successful runs (correct withhold on the target) and unsuccessful runs (false press on the target) with trials 6, 7, 8, 9, 1 and 2 considered pre-target trials and trials 4 and 5 post-target trials. To ensure that successful runs were not over-represented in this comparison, they were randomly selected to equate with the number of unsuccessful runs. A two-way repeated measures ANOVA was conducted. The factors were response run (successful vs. unsuccessful) and trial (9, 1, 2, 4, 5, 6, 7 and 8). No effect of response run ($F < 1$) or Response run × Trial interaction [$F(7,91) = 1.04$, $P = 0.41$] was observed: only a main effect of trial was reliable [$F(7,91) = 6.64$, $P = 0.0001$]. Repeated contrasts demonstrated that RT differences were as follows: trial 9 < 1 ($P = 0.001$), trial 1 > 2 ($P < 0.0001$), trial 2 < 4 ($P = 0.001$), trial 4 > 5 ($P = 0.003$). These differences are shown in Fig. 1B.

A separate analysis of mean go trial RTs (collapsed across all trials) versus mean commission error RTs confirmed that commission errors yielded significantly longer RTs than go trials, $t(13) = 3.53$, $P = 0.004$.

### *Electrophysiological results*

#### *Trial comparisons*

Fig. 2 shows a sampling of the recorded responses in midline posterior, central and frontal scalp sites to illustrate the general response morphology seen during performance of the SART. Trials 9, 1, 2, 3 and 4 show differential time courses whereas the superimposition of trials 5, 6, 7 and 8 illustrates a strikingly similar morphology in posterior and central locations. We reasoned that the period spanning trials 5–8 represented the least demanding portion of the trial sequence (i.e., the target had recently been responded to and was not due again for a number of trials) and as such, the attentional system maintains a stable set through this portion of the sequence. This is born out by the similarity of the responses to each of these 'nested' trial types. In all statistical analyses that follow, trials 5, 6, 7 and 8 were averaged to establish a "reference waveform" (hereafter referred to as REFW) to which other trials of interest could be compared.

Inspection of the group-averaged visual-evoked potentials for each trial during the SART revealed the expected series of ERP components, including the P1, N1, P2 and N2. As is characteristic, these components were maximal over occipital and occipito-parietal cortices. Of primary interest during this early sensory stage of processing were the amplitude differences as a function of trial type based on a given trial's position within the SART sequence. It was expected that sensory processing of trials from 5 through 8 would reflect the lowest attentional demand because these are the most temporally distinct from the period of critical target processing and thus would receive relatively automatic sensory processing. By contrast, as the sequence recommences after trial 9 subjects enter the critical target processing period, in which visual attentional processes may become more actively engaged.

#### *Selection negativity (SN)*

Fig. 3 shows the ERP morphology within the P1/N1 timeframe at electrode POZ. It is apparent that modulation of the P1 and N1 components is not independent but together forms a broader monophasic divergence resembling selection negativity. Accordingly, to test for an effect of sensory processing, we compared amplitude across the P1–N1 timeframe (120–160 ms) at POZ. A one-way ANOVA with trial (1, 2, 3, REFW) as the factor revealed a significant effect [$F(3,39) = 5.95$, $P = 0.002$]. Contrasts showed no reliable differences between SN amplitudes on trial 1 and REFW

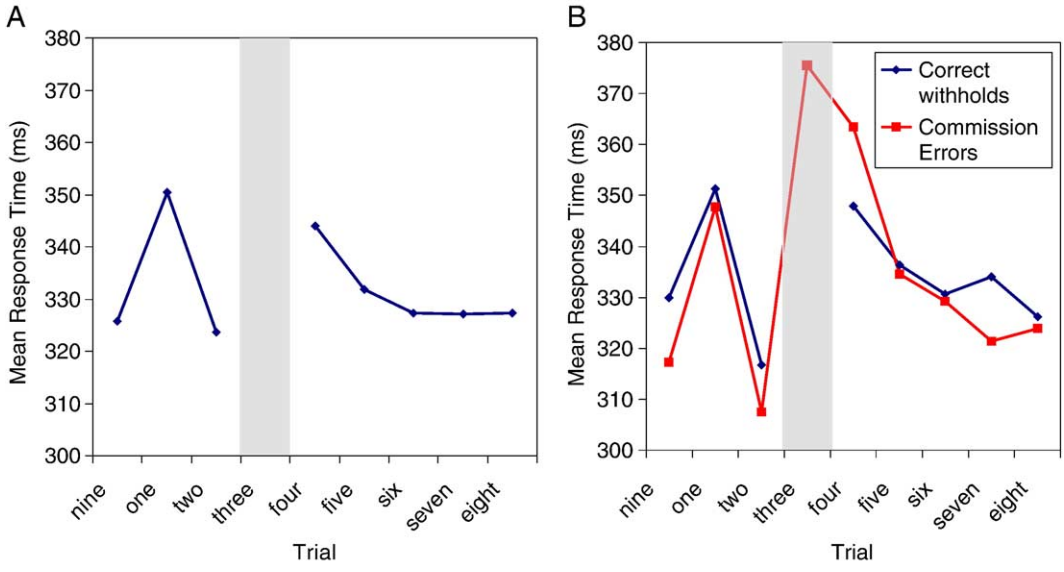


Fig. 1. (A) Response times (ms) as a function of trial during "successful runs" during the $SART_{fixed}$. The shaded area represents the withhold response. (B) Response times (ms) as a function of trial and response (correct withholds, commission errors).

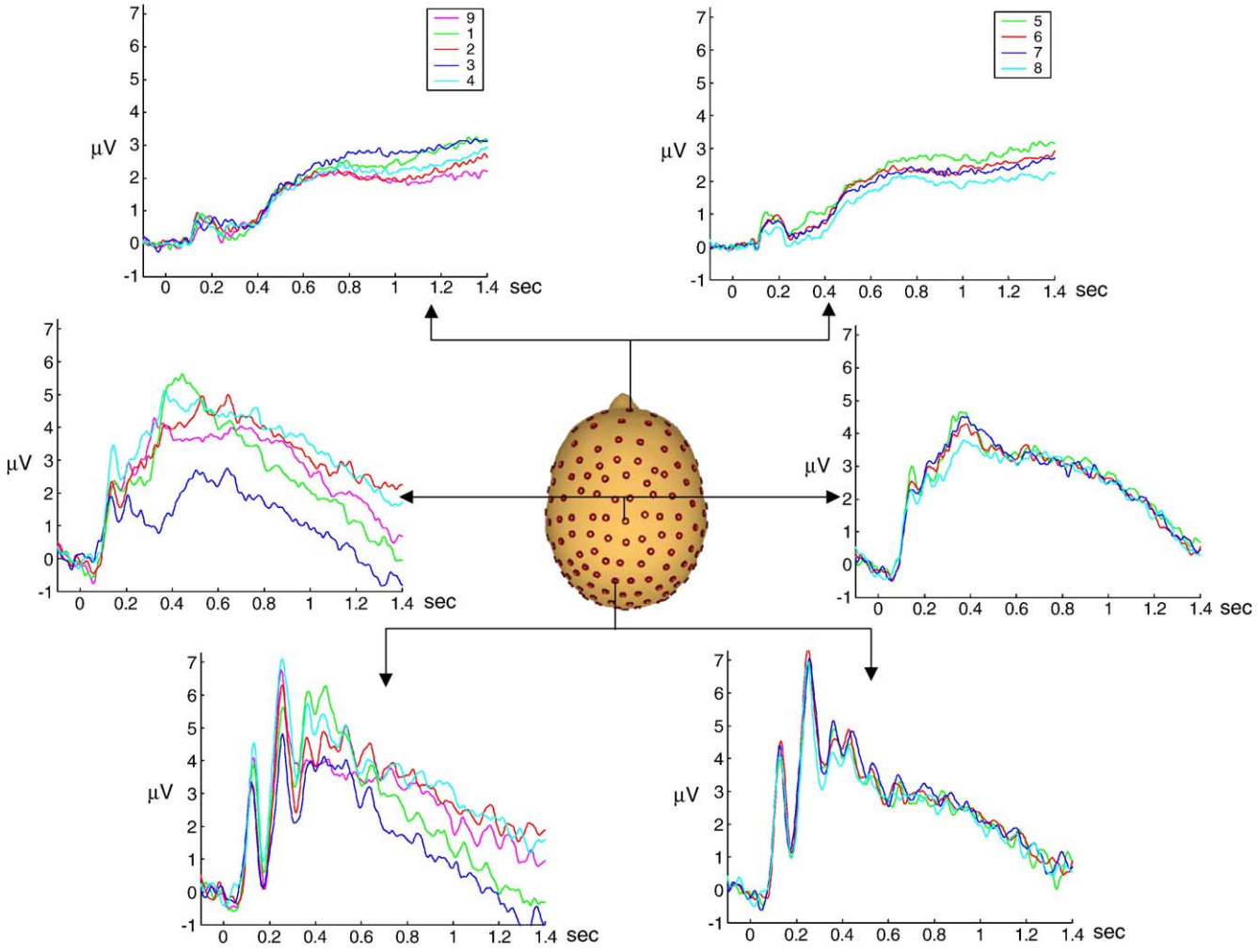


Fig. 2. Grand-averaged waveform morphologies in posterior (POZ), central (CZ) and frontal (FPZ) scalp regions. The plots on the left show ERPs for trials 9, 1, 2, 3 and 4, which represent a period of critical target processing. By contrast the plots on the right depict trials 5, 6, 7 and 8 that we hereafter describe as the reference waveform (REFW), which represent a period of relatively low attentional deployment. Notice the diverging time courses during the critical target processing period and the relatively similar superimposition of traces during the REFW trials.

[$F(1,13) = 1.74$, $P = 0.210$], which is clearly seen in Fig. 3A. However, significant differences were found between trial 2 and REFW [$F(1,13) = 9.66$, $P = 0.008$] and trial 3 and REFW [$F(1,13) = 20.88$, $P = 0.001$] with both trials 2 and 3 showing enhanced SN amplitudes compared to REFW. There was no difference between trial 2 and trial 3 in this period ($F < 1$). Difference topographies between trial 2 and REFW and between trial 3 and REFW both showed a similar occipito-parietal field pattern (see Fig. 3B). It is noteworthy that reported selection negativity effects frequently show a broad occipito-parietal distribution (see Anllo-Vento and Hillyard, 1996; O'Donnell et al., 1997), similar to that recorded here.

*P2*

A distinct P2[2] (244 ms ± 10 ms[3]) varied as a function of trial [$F(5,60) = 11.43$, $P = 0.0001$]. Amplitude for the P2 reduced from trials 9 to 1 ($P = 0.033$) and again from trials 2 to 3 ($P = 0.045$) and then increased substantially from trials 3 to 4 ($P = 0.0001$). Compared to REFW, trials 1 ($P = 0.001$), 2 ($P = 0.002$) and 3 ($P = 0.0001$) exhibited reduced P2 amplitude.

*N2*

The amplitude of the N2 (316 ms ± 10 ms) also differed as a function of trial over occipital and occipito-parietal areas [$F(5,60) = 3.49$, $P = 0.008$]. N2 amplitude increased from trials 9 to 2 ($P = 0.025$), from trials 1 to 2 ($P = 0.011$) and from trials 1 to 3 ($P = 0.002$). Trials 2 ($P = 0.005$) and 3 ($P = 0.008$) also showed increased N2 amplitudes compared to REFW. Moreover, during trial 3 a distinct "no-go N2" was apparent over central scalp that is most likely associated with absence or inhibition of the motor response. Fig. 4A shows that there is a negative deflection in amplitude (250–450 ms) relative to REFW. In order to examine this difference more closely, a scalp topography of the difference between trial 3 and REFW was computed (see Fig. 4B). The distribution of the difference has a central midline topography that is consistent with the absence or inhibition of a motor response during the no-go trial.

[2] One subject was excluded from this analysis because no measurable P2 was present in this subject's data.

[3] This refers to the average latency across the individual peaks picked.

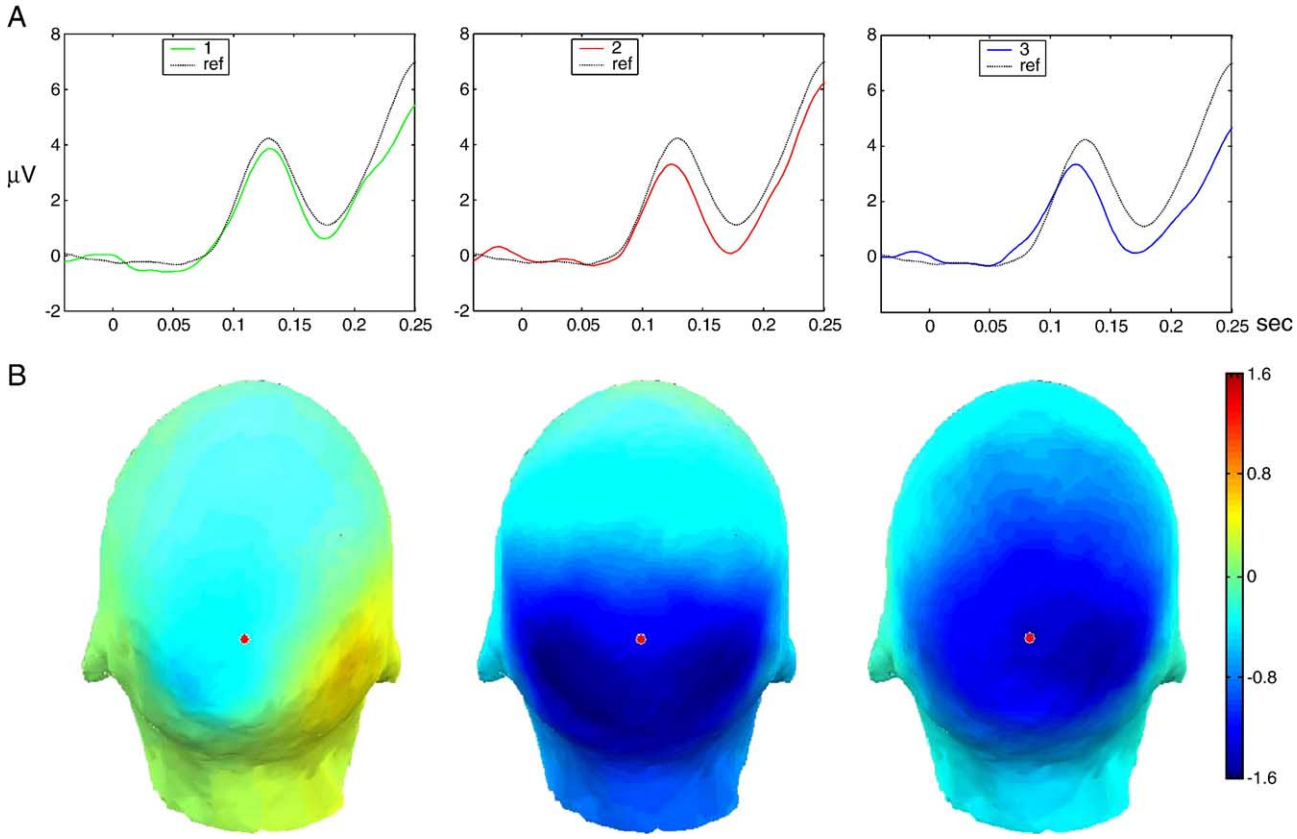


Fig. 3. (A) ERP traces to show amplitudes of the P1–N1 (120–160 ms) complex for trials 1, 2 and 3 versus REFW. Both trials 2 and 3 show relative negativity compared to REFW. (B) Scalp topographies depicting difference maps for trials 1, 2 and 3 versus REFW. In this scalp topography and all subsequent topographies, amplitude is integrated over the timeframe of the component in question, and electrode locations are pin pointed using disks. Thus, in this figure the scalp amplitude is integrated over the interval 120–160 ms. In contrast with REFW both trials 2 and 3 show similar occipito-parietal field patterns—this is absent during trial 1.

To test the relationship between the "no-go N2" amplitude and the propensity for commission errors, a Pearson's correlation coefficient was calculated. A significant inverse relationship was found between "no-go N2" amplitude and mean commission errors ($r = -0.558$, $P = 0.038$) suggesting that reduced "no-go N2" amplitude during trial 3 was associated with a greater number of commission errors across the task.

*P450*

The next positive component (400–500 ms) appeared predominately over occipito-parietal scalp. An effect of trial [$F(5,65) = 12.19$, $P = 0.0001$] was apparent. Amplitude increased from trials 9 to 1 ($P = 0.002$) and subsequently decreased from trials 1 to 2 ($P = 0.013$) and from trials 2 to 3 ($P = 0.002$). Amplitude increased again from trials 3 to 4 ($P = 0.001$). Both trial 1 ($P = 0.001$) and

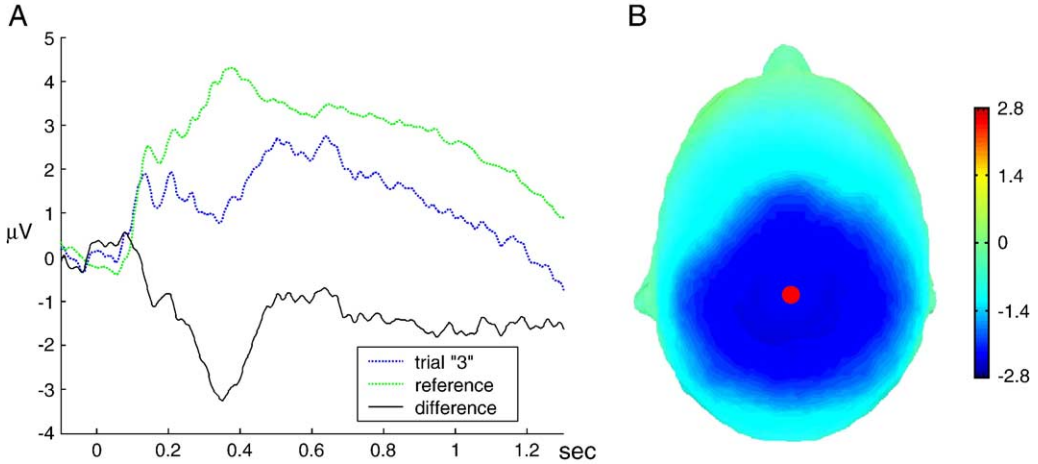


Fig. 4. (A) ERP traces showing a negative deflection ("no-go N2", 250–450 ms) in amplitude over central scalp (CZ) during trial 3 relative to REFW. (B) A difference map (trial 3 *minus* reference) illustrating the topography of the "no-go N2" over central scalp consistent with the withhold response on trial 3.

trial 4 (all $P = 0.012$) showed greater amplitudes than REFW and trial 3 was attenuated compared to REFW ($P = 0.012$). Fig. 5A shows this P450 effect.

*Scalp topography*

Fig. 5B shows the topography of composite P450 (i.e., collapsed across all trials). A bilateral parieto-occipital distribution is apparent with a large degree of spread evident over parieto-central scalp. A difference map was generated to illustrate the rise in amplitude from trials 9 to 1 (Fig. 5C). This difference has a strong midline parieto-central focus that appears to be distinct in form from the composite topography of the P450.

*Late positive 1*

Late positive 1 (LP1) component (550–800 ms) was examined over midline fronto-polar, central and occipito-parietal sites (regions of interest, ROIs) (Fig. 6A). A significant ROI × Trial interaction [$F(10,130) = 5.31$, $P = 0.0001$] was observed. Further, a significant main effect of trial [$F(5,65) = 3.32$, $P = 0.01$] was present but no main effect of ROI [$F(2,26) = 2.61$, $P = 0.093$]. Planned comparisons showed that the interaction was driven by heightened LP1 amplitude on trial 2 in occipito-parietal ($P = 0.0001$) and central sites ($P = 0.001$) compared to REFW amplitude, with no corresponding increase at fronto-polar scalp ($P = 0.72$). Additionally, amplitude markedly decreased from trials 2 to 3 at occipito-parietal ($P = 0.003$) and central sites ($P = 0.001$) followed by a subsequent increase from trials 3 to 4 at occipito-parietal ($P = 0.015$) and central sites ($P = 0.001$). No significant changes in amplitude were observed at fronto-polar sites ($P > 0.05$) for the LP1.

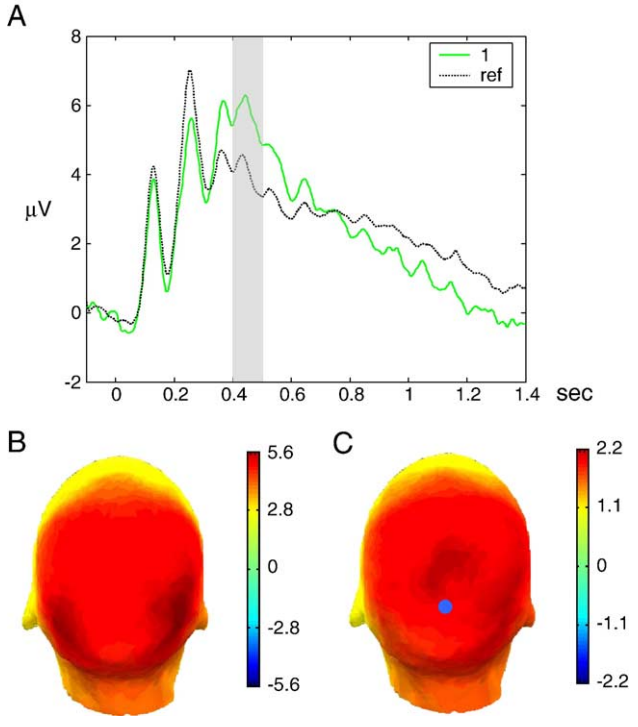


Fig. 5. (A) ERP traces depicting increased P450 (400–500 ms) amplitude (POZ) on trial 1 relative to REFW. (B) Composite scalp topography (i.e., collapsed across all trials) of the P450 effect. A bilateral occipito-parietal distribution is apparent for the P450. (C) A difference map (trial 1 *minus* reference) showing the rise in amplitude from trials 9 to 1 with a clear parieto-central focus.

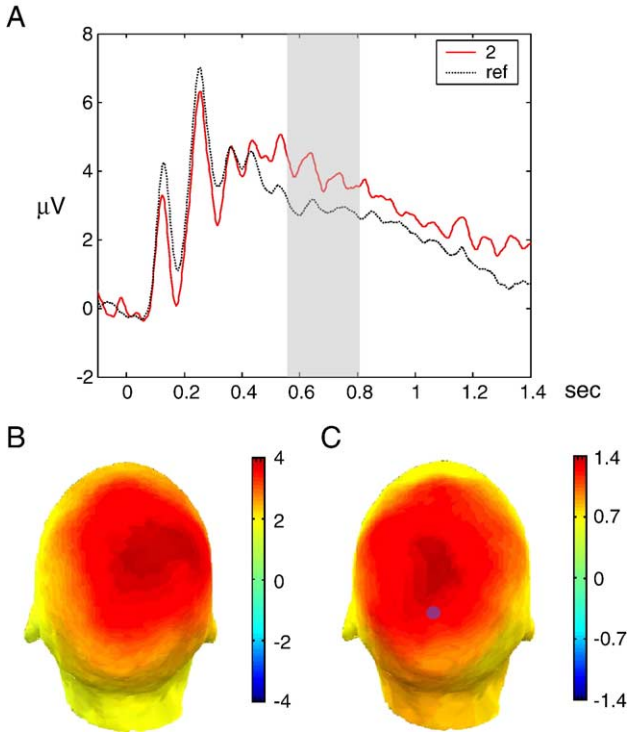


Fig. 6. (A) ERP traces showing increased LP1 (550–800 ms) amplitude (POZ) on trial 2 relative to REFW. (B) Composite scalp topography of the LP1 showing a distinct parieto-central focus. (C) A difference map (trial 2 *minus* reference) showing increased LP1 amplitude on trial 2 relative to REFW with a parieto-central focus.

*Scalp topography*

The composite LP1 waveform shows a distinct right parieto-central focus (see Fig. 6B). Topographical differences between the LP1 component on trial 2 and REFW are shown in Fig. 6C. Greater LP1 amplitude for trial 2 is distributed across parieto-central areas but the right focus seen in the composite topography is absent.

*Late positive*

Late positive 2 (LP2) component (850–1150 ms) was also examined as a function of trial and ROI (midline fronto-polar, central and occipito-parietal sites). There was no significant main effect of ROI ($F < 1$) but a significant effect of trial [$F(5,65) = 5.99$, $P = 0.0001$] and an ROI × Trial interaction [$F(10,130) = 9.88$, $P = 0.0001$] was observed. The interaction was driven by the following simple effects: at occipito-parietal and central sites LP2 amplitude increased from trials 1 to 2 (all $P < 0.02$) (see Fig. 7B), but by contrast no increase was observed over fronto-polar scalp ($P > 0.05$). Next, there were marginally significant increases in amplitude between trials 2 and 3 ($P = 0.054$) at the fronto-polar site (see Fig. 7D), and conversely a decrease in amplitude was seen from trials 2 to 3 at occipito-parietal and central locations (all $P < 0.001$). Between-ROI comparisons confirmed that over fronto-polar scalp amplitude was significantly higher than over central ($P = 0.022$) and occipito-parietal ($P = 0.017$) scalp during trial 3 (see Fig. 2 for between-region

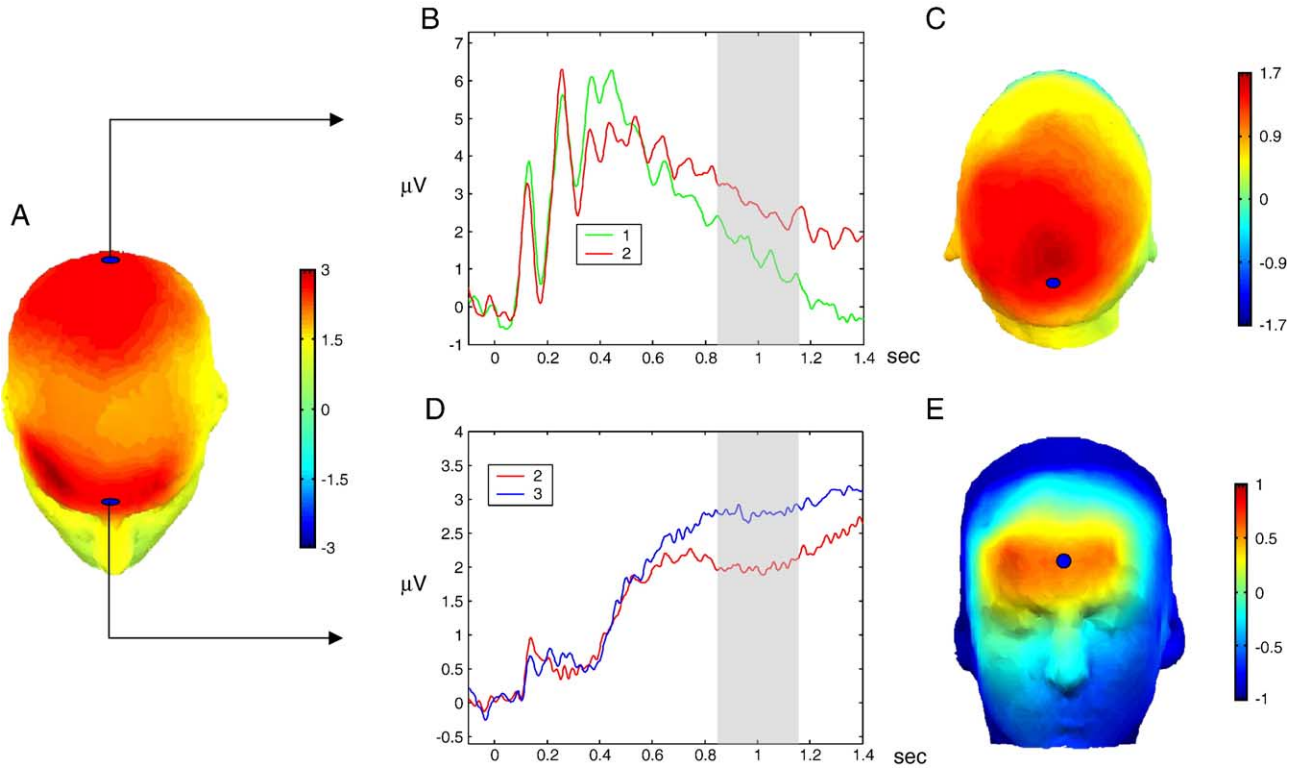


Fig. 7. (A) The topography of the composite waveform during the LP2 epoch showing a distinct focus over central scalp and during the same timeframe a bilateral fronto-polar distribution. (B) ERP trace illustrating the rise in LP2 (850–1150 ms) amplitude (POZ) between trials 1 and 2. (C) Difference map (trial 2 *minus* trial 1) showing increased amplitude at trial 2 relative to trial 1 with a parieto-central focus. (D) ERP trace illustrating the rise in LP2 (850–1150 ms) amplitude (FPZ) between trials 2 and 3. (E) Difference map (trial 3 *minus* trial 2) showing the increase in amplitude from trials 2 to 3 with a distinct fronto-polar distribution.

comparisons). Other reliable effects were driven by trial-by-trial changes in amplitudes only at occipito-parietal and/or central locations: amplitude decreased from trials 9 to 1 ($P = 0.026$; occipito-parietal site only) and increased from trials 3 to 4 (all $P < 0.0001$). Amplitude at trial 3 was attenuated compared to REFW (all $P < 0.003$), and at trial 4 amplitude increased compared to REFW (all $P < 0.03$).

*Scalp topography*

Fig. 7A shows the topography of the composite waveform during the LP2 epoch (850–1150). A distinct focus over central scalp is evident and in the same time frame there is a bilateral fronto-polar distribution. Difference maps allow us to explore differences in scalp distribution from trials 1 to 2 (Fig. 7C) and from trials 2 to 3 (Fig. 7E) where reliable differences were found. The increased amplitude at trial 2 relative to trial 1 has a parieto-central focus. By contrast, the increase in amplitude from trials 2 to 3 has a distinct fronto-polar distribution.

*Differential time courses preceding correct withholds and errors of commission*

In order to best characterize the behavior during the $SART_{fixed}$ we used statistical cluster plots to explore differential activity associated with anticipation of the no-go trial. In particular, we compared trials 9, 1, 2 and 3 as a function of response (commission error on '3' versus correct withhold on '3'). Fig. 8 depicts statistical cluster plots for trials 2 and 3 comparing activity prior to or during a correct withhold versus activity prior to or during a commission error. Further, waveform morphologies and difference topographies (Fig. 9) are presented for trials 2 and 3 as a function of response to show different time courses during this period.

*Trials 9 and 1*

No reliable effects of trial as a function of response were observed (all $P > 0.01$).

*Trial 2*

A distinct ridges of significant effects are apparent at ~600 ms in generalized scalp locations from occipital to central scalp. These indicate that amplitude is greater at trial 2 prior to a correct withhold than a commission error. The waveform plot at electrode location (POZ) depicts diverging time courses of the LP1 component that reaches maximum divergence at ~600 ms (Fig. 9A).

*Scalp topography*

A difference map was computed to examine the field patterns associated with the amplitude differences at trial 2 (550–800 ms). Fig. 9A also shows the difference in amplitude between pre-correct withholds and pre-commission errors at trial 2. The resulting topography shows a parieto-central distribution with slight spread to the right between 550 and 800 ms.

*Trial 3*

Distributed significant effects across the scalp are apparent at trial 3 with the largest effects seen between 650 and 850 ms. These

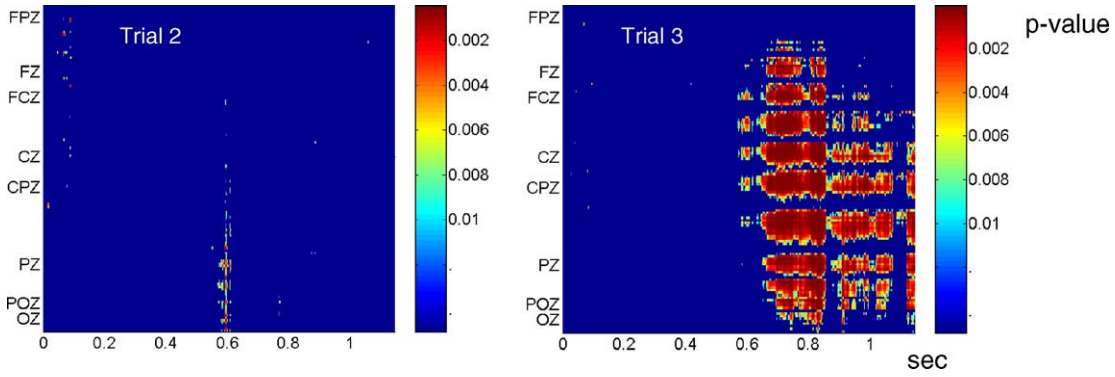


Fig. 8. Statistical cluster plots. Color values indicate the result of pointwise *t* tests comparing activity prior to or during a correct withhold versus prior to or during a commission error for trials 9, 1, 2 and 3, respectively. No reliable statistical effects are seen during trials 9 and 1. By contrast, during trial 2 a distinct ridge of statistical effects is apparent at ~600 ms in occipito-parietal regions during the expression of the LP1 component. Moreover, broadly distributed statistical effects are apparent at trial 3 with the largest effects seen between 650 and 850 ms, reflecting the increased amplitude of the error-related positivity following an error of commission.

indicate that amplitude during the commission error is enhanced relative to amplitude during a correct withhold during this epoch. The waveform plot at CZ shows the typical morphology of this error-related positivity effect in a central midline location (Fig. 9B).

Fig. 9B also shows the differences between the correct withholds and commission errors at trial 3 and exhibits differential field patterns between 650 and 850 ms displaying the error-related positivity in a parieto-central distribution. It is noteworthy that, despite the dominance of the parieto-central field pattern shown in Fig. 9B, the beginning of a distinct frontal LP2 is apparent over fronto-polar scalp showing enhanced amplitude during a correct withhold relative to an error of commission. The waveform plot (Fig. 10A) shows the LP2 differential in a midline fronto-polar site (FPZ). At this scalp location the waveform for correct withholds

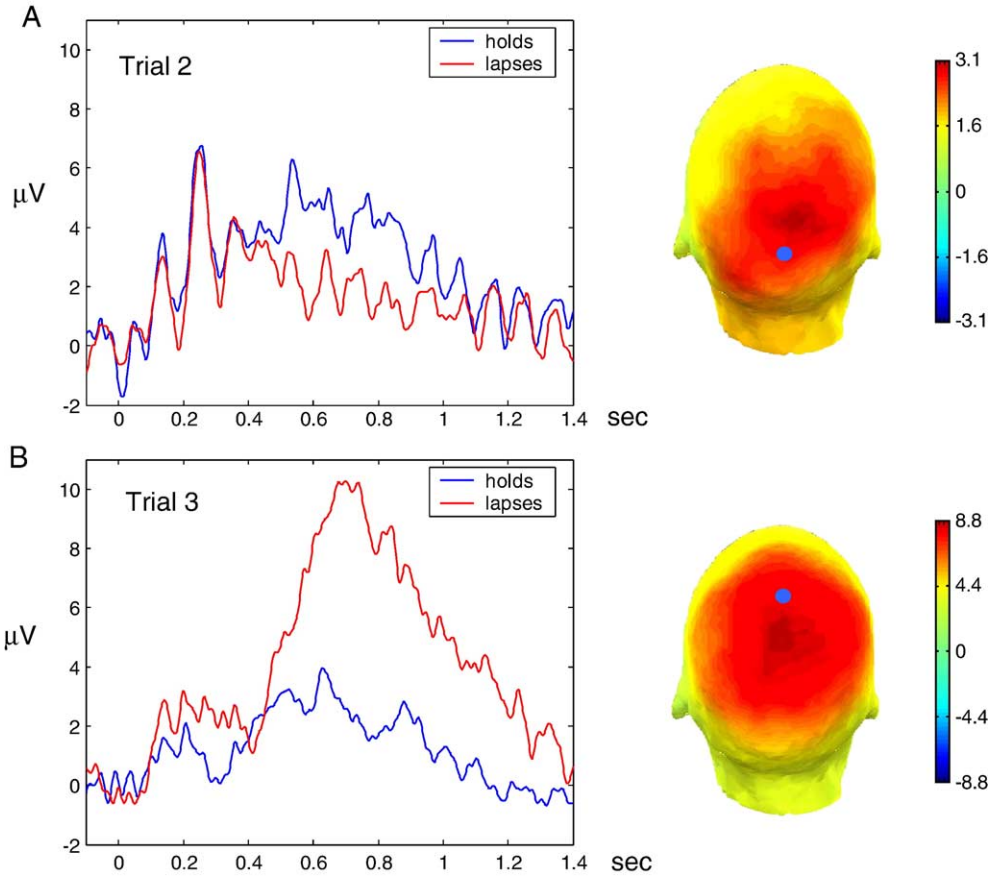


Fig. 9. (A) ERP traces (POZ) and difference topographies (pre-correct withhold *minus* pre-commission error or pre-lapses) showing diverging time courses of the LP1 component (550–800 ms) over parieto-central scalp. (B) ERP traces (CZ) and difference topographies (commission error *minus* correct withhold) showing enhanced amplitude (650–850) during a commission error relative to a correct withhold over central scalp.

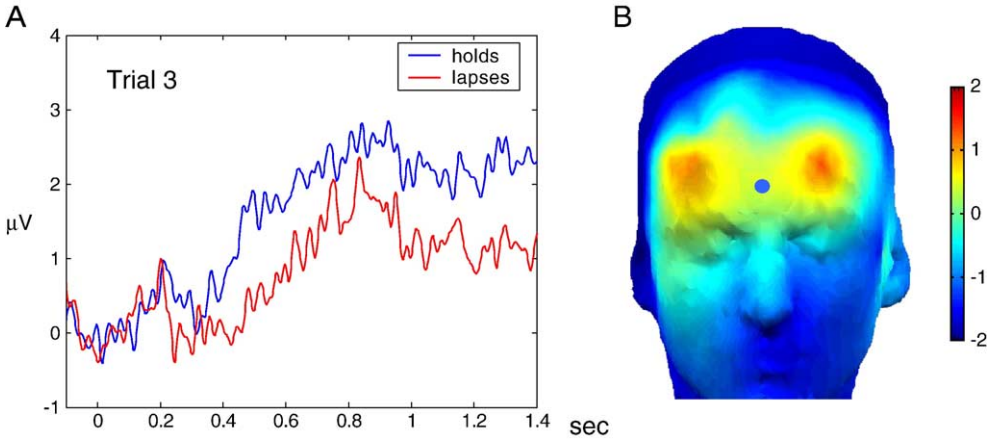


Fig. 10. (A) ERP traces (FPZ) showing the frontal LP2 component over fronto-polar scalp. During correct withholds a late sustained positive amplitude is observed. By contrast, a residual error positivity effect is seen during commission errors that is physiologically distinct in form from the sustained positivity during a correct withhold. (B) Scalp difference topographies (correct withhold *minus* commission error) show that fronto-polar field patterns are increased for correct withholds compared to commission errors.

shows a late sustained positivity. By contrast, the commission error trace shows the residual morphology of the dominant error-related (650–850 ms) effect that is physiologically distinct in form from the late sustained positivity seen for the correct withholds. To examine the distribution of this difference, scalp topography for the frontal LP2 (850–1150 ms) was computed (see Fig. 10B).

## Discussion

In this experiment we have examined response time patterns and the spatiotemporal dynamics of ERPs that characterize sustained attention performance in neurologically healthy subjects. Behaviorally, a distinct pattern of responses characterized task performance demonstrating a lengthening of RTs at the turnover of the digit sequence from trials 9 to 1 and a subsequent shortening of RTs from trials 1 to 2 prior to the upcoming target on trial 3. Electrophysiologically, we identified a number of ERP components across the scalp – over occipito-parietal, parieto-central and frontal regions – that inform our predictions regarding the anticipatory period before the upcoming no-go trial during the SART.

### Summary of ERP componentry

Distinct differences in the ERP componentry separated a critical target processing period (trials 9–3) and a non-critical 'task-driven' period with relatively lower attentional demands (trials 5–8). We used a collapsed average of the latter as a reference waveform (REFW) to which the active attentional demands during trials 9–3 could be compared. The first differences in ERP amplitude were observed between trials 9 and 1 with a reduction in the amplitude of the P2 component over occipito-parietal scalp and an increase in a P3-like component over parieto-central areas (~450 ms) on trial 1. During trial 2 an enhanced early negative potential develops over occipito-parietal regions that encompasses the P1–N1 complex. Later over occipito-parietal areas the P2 is attenuated by the negative deflection of the subsequent enhanced N2. Next during trial 2, two late sustained components were observed – LP1 (550–800 ms) and LP2 (850–1150 ms) over occipito-parietal and central sites – both were larger in amplitude compared to REFW. At trial 3, a similar early negativity was observed as during trial 2: the early P1–N1 complex, over occipito-parietal areas, was marked by a sustained negative shift compared to REFW. Between 250 and 450 ms over central scalp a pronounced "no-go N2" was observed during trial 3 that was entirely absent during all other trials. Finally, during all trials, a distinct bilateral fronto-polar distribution was observed between 850 and 1150 ms. Moreover, fronto-polar amplitude during this period increased compared to amplitude at central and occipito-parietal locations during the target trial 3. In what follows, we will discuss somewhat complex series of effects, in their turn, as they relate to the task dynamics. Table 1 provides a summary of the key ERP components that are discussed.

Table 1
Summary of key ERP components identified during the $SART_{fixed}$, their topographical distribution and the key trial comparisons where they differ in amplitude

| ERP component | Time (ms) | Scalp region[a] | Key trial comparisons and direction of effects |
|---|---|---|---|
| Selection negativity | 120–160 | Occipito-parietal | 2 < REFW<br>3 < REFW |
| Cue recognition N2 | 316 ± 10 | Occipito-parietal | 2 < 1<br>2 < REFW |
| No-go N2 | 250–450 | Central midline | 3 < REFW |
| P450 | 400–500 | Parieto-central | 1 > 9<br>1 > 2<br>1 > REFW |
| Goal activation LP1 | 550–800 | Occipito-parietal and central | 2 > REFW<br>HOLDS > LAPSES |
| Task switching LP2 | 850–1150 | Occipito parietal and central | 2 > 1<br>2 > REFW |
| Fronto-polar LP2 | 850–1150 | Fronto-polar | 3 > 2 |
| Error-related positivity | 650–850 | Central midline | LAPSES > HOLDS |

[a] Scalp regions refers to the topography of the difference for the key trial comparisons.

### Utilizing the number sequence

The data show that the turnover of the digit sequence from trials 9 to 1 results in a lengthening of RTs implying that this transition may be a critical juncture during which stimulus processing is enhanced. In support of this conjecture, our findings demonstrate that P450 amplitude increased over parieto-central regions from trial 9 to trial 1 (see Fig. 5). Our later exploratory analysis revealed that this P450 effect was present to the same magnitude prior to a correct withhold and an error of commission on trial 1. As such, it is probable that the P450, in the current study, reflects relatively automatic processing of an exceptional point in an otherwise predictable ascending sequence and is not directly related to alert responding in anticipation of the no-go trial. Recent work (Lang and Kotchoubey, 2002) has shown that modulation of the P300 can discriminate between ascending sequences of numbers that end with appropriate or inappropriate end items. Subjects were presented with four-item number sequences incrementing by one. Half of the sequences had an appropriate end item (3, 4, 5 and 6), whereas the other half ended inappropriately (3, 4, 5 and 9). Incorrectly ending sequences yielded a larger P300 waveform across all subjects. Lang and Kotchoubey conclude that incrementing number sequences builds strong expectancies with respect to subsequent numbers and violation of these expectancies is in conflict with our long-term knowledge of numerical relations (Dehaene et al., 1998). A given intensity of alertness may be necessary but not sufficient on its own to generate an enhanced P450 amplitude in the context of the digit turnover during the SART.

### Selection negativity and no-go negativity

Trials 2 and 3 were exceptional in terms of the early sensory processing they received. We proposed that early visual attentional processes would be mobilized during trial 2 because of its significance as an upcoming cue trial. Further, we suggested that trial 3 might also elicit the same visual attentional processes given its significance as the target trial. However, another possibility was that trial 3 might actually receive less processing due to a suppression of early sensory activity in preparation for the no-go response. Our findings suggest that both trials (2 and 3) actually received increased attentional deployment. On each of these trials an early negative potential shift was observed beginning during the P1–N1 complex over occipito-parietal scalp. Stimuli that are selected on the basis of non-spatial features have been shown to elicit a broad negativity over occipito-parietal scalp over the interval of 150 ms to 350 ms (Anllo-Vento and Hillyard, 1996; Anllo-Vento et al., 1998; Harter et al., 1984). Furthermore, visual N1 amplitude increases if an attentional set is established prior to stimulus delivery. This was demonstrated in a visual oddball task during which response contingencies were varied (Potts et al., 2004): subjects were instructed to respond to the targets with a keypress in two blocks and in a further two blocks they were instructed to press a key to the ignored non-targets and respond to the targets by withholding a key press. The enhanced N1 effect was specific to the instructed targets, irrespective of response contingencies, suggesting that visual attention can be allocated to task-relevant features through the establishment of an attentional set as a top-down bias prior to stimulus delivery (Foxe and Simpson, in press; Foxe et al., in press). In a similar vein, the present SN effects suggest that an important component of successful performance of the SART is the phasic redeployment of selective attentional resources during critical processing periods.

Additionally, during trial 3 over central sites, a distinct N2 component (250–450 ms) was observed and was absent in all other trials. We interpret this component as a "no-go N2" that is frequently seen as a component in visual go/no-go tasks during target processing (Eimer, 1993; Falkenstein et al., 1995; Roche et al., 2004). The N2 has been associated with the neural mechanisms underlying response inhibition and has been interpreted as a marker or 'red flag' that precedes or initiates active inhibition of a button response (Kok, 1986; Kok et al., 2004). The N2 has also been observed under other conditions where the active suppression of motor activity has not been required. For example, Pfefferbaum et al. (1985) showed that the N2 component was delayed when subjects were required to covertly suppress the silent counting of target stimuli. These authors suggested that the N2 could be elicited by target decisions instead of active motor inhibition. Interestingly, the target trial during the $SART_{fixed}$ elicited a robust N2 component but did not elicit a traditional P3 (400–600 ms) component following the N2. The P3 is more strongly associated with the physiology of response inhibition and is seen in the context of go/no-go tasks (Bruin et al., 2001) and stop-signal tasks where subjects undertake a speeded choice reaction task and occasionally receive a stop signal that instructs them to withhold their response (Kok et al., 2004). In the context of the present task the robust N2 effect is likely to be associated with the initiation of the withhold response (especially as its distribution is over motor areas and it is inversely related to the number of commission errors). However, given the ascending sequence that anticipates the target trial during the $SART_{fixed}$, active suppression of motor activity will most likely be unnecessary, explaining the absence of the traditional P3 component. It is plausible that response conflict between the go response and no-go response will be resolved on trial 2 preceding the target. This will be discussed later in the context of task switching.

### Goal recollection

We proposed that recollection of the task goal would be associated with phasic negativity over occipito-parietal scalp and positivity over parietal areas during trial 2, in keeping with previous reports (West and Krompinger, 2005; West et al., 2001). Our findings demonstrate two significant modulations occurred during trial 2: an earlier N2-like effect over occipito-parietal scalp and a later positive (LP1) modulation (550–800 ms) over occipito-parietal and central sites. Both components were enhanced on trial 2 compared to REFW. Research investigating prospective remembering suggests that a retrieval cue associated with the context of remembering together with the recollection of the action representation are necessary components of realizing a delayed intention (Mäntylä, 1993). In the current study, trial 2 will act as an imminent retrieval cue for the no-go response on trial 3. Thus, the enhanced N2 may reflect recognition of trial 2 as a retrieval cue for the no-go response task set. Interestingly, this occipito-parietal N2 was also observed on the subsequent trial 3. This modulation may also reflect recognition of the stimulus, but in this case, for the no-go response *execution*. We then may argue that the similarity of the N2 processing stage on trials 2 and 3 is indicative of generic cue recognition for subsequent goal-directed processes. It is noteworthy that although SN-like effects have been observed over

periods as long as 200 ms (e.g., Anllo-Vento and Hillyard, 1996), the ERP morphologies in these data from 100 to 400 ms exhibit two distinct negativities during this period suggesting that the selection negativity ends before the onset of the posterior N2.

The increase in the LP1 amplitude over parieto-central scalp during trial 2 is likely to be associated with the retrieval of the intended action itself from memory (see West et al., 2001) or the maintenance of the new task set prior to its execution. It is noteworthy that an fMRI investigation of the SART (Fassbender et al., 2004) reported the involvement of bilateral inferior parietal cortices during the task that were activated in association with bilateral prefrontal areas. The authors suggested that these patterns of activation were associated with an extended sustained attention network involved, in part, in the maintenance of task set. The topographic distribution of the LP1 over parieto-central areas in the current study suggests that this electro-cortical activity may also be indicative of task set maintenance during the SART.

### Task switching

We predicted that in the transition from trials 2 to 3 sustained positivity over parietal regions would be indicative of a switch from the go response to the withhold response. Between 850 and 1150 ms, amplitude markedly increased on trial 2 across occipito-parietal and central sites before the presentation of trial 3. In accordance with Wylie et al. (2003), we argue that the increase in the second late positive (LP2) amplitude reflects increased competition between two responses: either the currently relevant go response is suppressed or the subsequent withhold response is activated or an interplay between activation and inhibition is occurring. This interpretation is consistent with a competition hypothesis (see Wylie et al., 2003) that proposes that different task sets are composed of a network of "nodes" that have relative weights in a competing cognitive system. If one task set (e.g., withhold response) is selected in favor of another (e.g., go response) then the relative weights of the "nodes" that represent the selected task set – or the goal task set – are increased. Switch costs occur because the competition takes time to resolve before the cognitive system returns to a stable state. We argue that the LP2 response is indicative of this competition between the go response and withhold response.

### Fronto-polar positivity

Distinct bilateral anterior fronto-polar positive foci were highly prominent on each trial in the SART sequence. Involvement of fronto-polar cortices in the SART task is to be expected as numerous functional imaging studies have now implicated fronto-polar cortices as critical nodes in the circuit for maintaining and generating subgoals and for integrating such subgoals with the ongoing primary task (e.g., Braver and Bongiolatti, 2002; Badre and Wagner, 2004). In the present task, the main goal is to respond to each trial and the subgoal is likely reactivated with every occurrence of the stimulus as the system prepares for the withhold response. In addition to the general and sustained involvement of these structures across the sequence, it was also found that during the sustained period following a correct withhold to the "3", this fronto-polar positivity was greater in amplitude. Indeed, its activity was greater than that measured over central and occipito-parietal scalp locations during the same timeframe (850–1150 ms).

One possible explanation for this added fronto-polar activity is that it is associated with anticipation of the upcoming go response on trial 4. Indeed, very similar fronto-polar activity has been previously seen in tasks that involve switching between task conditions that occur in a predictable order (Dreher et al., 2002), consistent with the predictability of the current task. However, if this added activity on trial 3 is related to switching back to the original go response, then one would expect that a similar enhancement would be seen during the late stage of processing for trial 2 where the subject must also switch, in that case to the no-go response. However, in these data we find that during the switch between a go response and a withhold response from trials 2 to 3, occipito-parietal and central positivities (not fronto-polar positivity) are enhanced. The reason for these different field patterns may be associated with the nature of the competition between relative task sets. We speculate that switching from the more frequent go response to the withhold response may require gating of the motor response and thus more central field patterns are present over motor cortices. Alternatively, the switch from the withhold response to the go response may require re-activation of the primary goal components of the task. The requirement to refresh the primary goal of the task at the juncture between trials 3 and 4 may recruit higher order processes to integrate an association between the stimulus (trial 4) and the primary goal (go response). That is, the association between trial 4 and the go response may be refreshed at this point. Fronto-polar activity has recently been linked to integration processes in a task that required the subject to refresh the conditions for goal execution before execution of the response (Badre and Wagner, 2004). When conditions for responding were externally presented fronto-polar activation was not seen indicating that fronto-polar activation involves the evaluation of internally generated information.

### Lapses of attention: failure of goal-directed behavior

In this investigation, the high number of experimental blocks undertaken by subjects ensured that an adequate number of errors were committed over the course of testing to obtain reliable individually averaged waveforms for errors of commission and for the trials preceding these attentional lapses. The most notable difference was a clear divergence of the LP1 component, which exhibited greater amplitude on trial 2 prior to a correct withhold compared to before a commission error—the difference reached significance at ~600 ms. The distribution of this effect across parieto-central regions suggests that failed recollection of the task goal may underlie the attenuated LP1 amplitude prior to a commission error. Interestingly, the earlier N2 component did not differ as a function of response suggesting that trial 2 was 'noticed' but subsequent 'search' processes were not engaged.

The Notice + Search model of retrieval (Brandimonte et al., 1996; McDaniel et al., 2004) provides a two-stage account of intention retrieval. Initially, the target event automatically elicits feelings of knowing or acquaintance referred to as 'noticing'. Subsequently, a search of memory is initiated to identify the significance of the target. It is likely that failure of the timely activation of the task goal is related to a transient reduction in alertness that supports goal retrieval and not a specific deficit in recollecting the task goal. In the context of models of sustained attention (Posner and Peterson, 1990; Sturm et al., 1999), it is possible that a transient reduction in top-down control, via the

thalamus, on noradrenergic structures in the brainstem may account for this momentary lapse of attention.

## Conclusion

Here, we characterize the spatiotemporal dynamics of alert responding during an established task that measures one's ability to maintain a goal-directed focus during unarousing conditions. Neurophysiological results reported here inform us about several important processes that reflect alert responding and the failure of goal-directed behavior. These include the mobilization of early visual processing, cue selection, goal recollection, task switching, error evaluation and fronto-polar integration processes. The current study with healthy adults will inform hypothesis generation with respect to clinical groups with attentional deficits.

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