Two Types of Action Error: Electrophysiological Evidence for Separable Inhibitory and Sustained Attention Neural Mechanisms Producing Error on Go/No-go Tasks

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Abstract

■ Disentangling the component processes that contribute to human executive control is a key challenge for cognitive neuroscience. Here, we employ event-related potentials to provide electrophysiological evidence that action errors during a go/no-go task can result either from sustained attention failures or from failures of response inhibition, and that these two processes are temporally and physiologically dissociable, although the behavioral error—a nonintended response—is the same. Thirteen right-handed participants performed a version of a go/no-go task in which stimuli were presented in a fixed and predictable order, thus encouraging attentional drift, and a second version in which an identical set of stimuli was presented in a random order, thus placing greater emphasis on response inhibition. Electrocortical markers associated with goal maintenance (late positivity, alpha synchronization) distinguished correct and incorrect performance in the fixed condition, whereas errors in the random condition were linked to a diminished N2–P3 inhibitory complex. In addition, the amplitude of the error-related negativity did not differ between correct and incorrect responses in the fixed condition, consistent with the view that errors in this condition do not arise from a failure to resolve response competition. Our data provide an electrophysiological dissociation of sustained attention and response inhibition. ■

INTRODUCTION

The ability to monitor and control our behavior in a goal-directed manner requires the dynamic interaction of a collection of higher cognitive abilities known as executive functions (EFs). Accumulated evidence from functional neuroimaging and lesion studies with humans, animals, and primates has provided strong evidence that executive control is not a unitary process but requires the activation of a collection of distinct but interacting brain networks (Chow & Cummings, 1999; Alexander, DeLong, & Strick, 1986). Even the most routine everyday task demands that we maintain a representation of our current goals, monitor our behavior, detect errors, and inhibit competing thoughts and actions. This complexity presents an interesting challenge to researchers seeking to target specific cognitive functions, as even the most basic behavioral task will require the coactivation of more than one EF. This issue becomes even more problematic in the study of action errors, where it is necessary to identify the precise point at which the EF system has failed. As the cognitive neurosciences endeavor to elucidate the neural processes governing executive control, there is an imperative for the application of methodologies that make it possible to differentiate its constituent components. The present study applies the high temporal resolution of event-related potentials (ERPs) to identify unique electrocortical markers for two aspects of EF, sustained attention and response inhibition.

The ability to sustain attention is a core EF that can be defined as self-sustaining a mindful, goal-directed focus in contexts whose repetitive, nonarousing qualities provide little exogenous stimulation and would otherwise lead to habituation and distraction by other stimuli (Robertson & Garavan, 2004). The performance of tasks that require sustained attention is associated with activation of a predominantly right-lateralized network and includes the dorsolateral prefrontal cortex, the anterior cingulate cortex, and the right inferior parietal lobule, with top–down modulatory projections to subcortical

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arousal structures (Sturm & Willmes, 2001). Traditionally, sustained attention deficits have been measured using vigilance paradigms, such as the continuous performance task (CPT), in which participants are asked to monitor a stream of stimuli over an extended period of time for the occurrence of a rare target stimulus to which they must make a response. These tasks are designed to mimic ''real world'' situations in which low signal probability places us at increasing risk of a critical lapse of attention (e.g., the airport security officer who fails to notice a weapon passing through the scanner). Performing such tasks is tedious and monotonous, leading to a gradual decline in target detection over time, resulting from diminished arousal. This phenomenon is known as the ''vigilance decrement'' and it, rather than the absolute number of target detection errors, has been argued to be the key index of sustained attention capacity (Parasuraman, Nestor, & Greenwood, 1989). However, the utility of this marker of attentional capacity has been questioned because a disproportionate vigilance decrement has not been reliably demonstrated in a number of patient groups with confirmed sustained attention difficulties in daily living such as attention-deficit hyperactivity disorder (ADHD) and traumatic brain injury (TBI) (Swanson et al., 2004; Manly et al., 2003).

Robertson, Manly, Andrade, Baddeley, and Yiend (1997) proposed a different operational definition of sustained attention to that used in vigilance studies, highlighting that in everyday life we are also susceptible to fluctuations in attention over much shorter periods than are measured using traditional CPTs. These slips are most likely to manifest themselves in the context of routine or mundane tasks when we are prone to mindlessly persist with automated behaviors at times when task demands have changed and increased attention to action is required (e.g., sending an e-mail without an intended attachment). Imaging studies have supported this observation by showing that the right hemispheric sustained attention network is engaged over periods of less than a minute (Paus et al., 1997) and that brief lapses of attention are preceded by momentary reductions of activity in frontal control regions (Weissman, Roberts, Visscher, & Woldorff, 2006). Yet conventional vigilance paradigms are designed to measure longer-term processes in the region of minutes to hours. Because there are large temporal gaps between target events in a typical CPT, these tasks may be insensitive to the moment-to-moment lapses that occur in the context of routine action. It is this very phenomenon that characterizes real-life slips of attention, yet developing a behavioral paradigm that can isolate these momentary fluctuations of attention has proved challenging.

With these methodological issues in mind, Robertson et al. (1997) developed a paradigm that would provide a more continuous measure of sustained attention failures in a group of patients with TBI. Robertson et al. used the Sustained Attention to Response Test (SART), a go/no-go paradigm in which a series of single digits (1–9) were presented in a random order and participants were required to press a response key to each number except the number 3. It was argued that the frequent occurrence of target stimuli would make the task more sensitive to those relatively brief lapses of attention that occur in the absence of time-on-task effects. In support of their predictions, Robertson et al. found that the SART was more sensitive to frontal lobe damage than a conventional CPT task, and that performance was well correlated with rates of everyday attentional failures as indexed by the Cognitive Failures Questionnaire (CFQ).

A notable limitation of the SART in its original form, however, was the clear emphasis it placed on response inhibition. Although performance of the task certainly demands sustained attention to action, correct performance is also highly dependent on the participant's ability to suppress the already initiated go response upon the appearance of the no-go target. Response inhibition is another core EF and has been described in terms of a horse-race model, whereby go and stop processes are stochastically independent and race to completion (Logan, 1994). Whichever process wins the race determines whether the response will be inhibited or not. Human lesion, transcranial magnetic stimulation, and imaging studies have also linked response inhibition to a right-lateralized fronto-parietal network within which the right inferior frontal cortex is thought to play a critical role (Chambers et al., 2006; Aron & Poldrack, 2005). During a go/no-go task, sustained attention will be necessary in maintaining a strong task set in the intertarget intervals and response inhibition will be required to resolve the conflicting response tendencies. Therefore, successful performance of a go/no-go task requires an ability to exert both proactive (sustained attention) and reactive (response inhibition) executive control and deficiencies in either ability can potentially lead to an error (Braver & Barch, 2006).

The combination of two different action errors in studies using the go/no-go paradigm may obscure the underlying brain–behavior relationship associated with each process. This issue is also of concern in studies of clinical groups such as ADHD where poor response inhibition is a common finding (Seidman, 2006), but where problems of sustained attention have also been reported (Johnson et al., 2007; Swanson et al., 2004). Given that there is considerable overlap in functional brain activations associated with sustained attention and response inhibition—particularly in right frontal regions (Aron & Poldrack, 2005; Sturm & Willmes, 2001; Garavan, Ross, & Stein, 1999)—there are pressing theoretical and clinical reasons to determine the underlying mechanisms of action errors on such tasks.

In order to minimize the emphasis on response inhibition, Manly et al. (2003) developed a ''fixed'' version of the SART ($SART_{fixed}$), which was identical to the earlier version, except that stimuli were now presented in a predictable, fixed sequence from 1 to 9. It was

hypothesized that the predictable target sequence would facilitate preparation of the no-go response to such an extent that the race between the go and stop process would be eliminated. Manly et al. compared the performance of patients with frontal brain injuries and a group of normal healthy controls on the $SART_{fixed}$ and the original random version, $SART_{random}$. Interestingly, it was found that although both groups made more errors on the $SART_{random}$, the fixed version actually discriminated better between the two groups. Furthermore, positron emission tomography data from the same study indicated that the $SART_{fixed}$ produced *stronger* activation of the right-hemispheric sustained attention network. The authors accounted for these findings by suggesting that decreasing task difficulty by making the appearance of targets entirely predictable rendered the task more monotonous and less exogenously alerting. Hence, the demand on endogenous frontally driven sustained attention was actually increased by making the task less challenging. This study provided further evidence that even neurologically healthy individuals are prone to periodic lapses of attention when performing a routine task for a matter of minutes.

In a follow-up to Manly et al.'s (2003) study, Dockree, Kelly, Robertson, Reilly, and Foxe (2005) and Dockree et al. (2004) used high-density electrical mapping to identify key electrocortical markers for alert responding on the $SART_{fixed}$. The most prominent marker of successful performance was a broadly distributed late positivity (termed late positive 1, LP1) over occipito-parietal and central scalp sites that was evident on all trials and gradually enhanced on trials preceding a correct inhibition. This modulation led the authors to conclude that the LP1 was likely to reflect the recruitment of the sustained attention network for the activation and maintenance of task goals. Another key marker of $SART_{fixed}$ performance, identified by Dockree et al. (2004), was a suppression of activity in the alpha band prior to the upcoming no-go stimulus. Alpha is an electroencephalogram (EEG) oscillation that varies with levels of alertness and the suppression, or desynchronization, of activity in this band has been linked to increased cognitive demand and the deployment of attention (Klimesch, 1999). Dockree et al. (2005) also noted that the N2 and P3 components on no-go trials were heavily attenuated, providing some support for the prediction that the $SART_{fixed}$ is less reliant on response inhibition. The no-go N2–P3 complex is consistently seen over fronto-central scalp sites on trials requiring response inhibition, 200–600 msec after no-go stimulus onset. Although their functions are debated, there is consensus that the N2 component probably indexes aspects of response selection or conflict detection that signal the need for response inhibition (Falkenstein, 2006; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004) while the latency and amplitude of the P3 have been shown to predict successful inhibitions in keeping with Logan's (1994) race model of inhibition (Bekker, Overtoom, et al., 2005; Bekker, Kenemans, & Verbaten, 2004; Falkenstein, Hoomann, & Hohnsbein, 1999). Hence, the findings of Dockree et al. suggest a reduced requirement for response inhibition in the $SART_{fixed}$.

In light of this evidence, the two versions of the SART provide an ideal opportunity to dissociate action errors resulting from momentary lapses of sustained attention on the one hand, and inhibitory failures on the other hand. Critically, the two versions of the task are identical in terms of their perceptual and motoric demands. That is, although both versions of the SART require participants to withhold a response to a no-go target only the $SART_{random}$ demands urgent suppression of an already initiated prepotent response. In contrast, in the absence of a response inhibition requirement, the $SART_{fixed}$ should isolate pure lapses of attention. In the present study, we employed the high temporal resolution of ERPs to provide objective physiological evidence that sustained attention failures on the $SART_{fixed}$ can be dissociated from response inhibition failures on the $SART_{random}$.

Utilizing the $SART_{random}$ as a comparison for the $SART_{fixed}$, this study addressed three principle hypotheses:

1. Correct performance on the $SART_{fixed}$ should place few demands on inhibitory processes as participants are able to anticipate targets and prepare their responses. The amplitude of the no-go N2–P3 should therefore not discriminate between correct and incorrect performances on the $SART_{fixed}$. In contrast, the $SART_{random}$ should show a strong N2–P3 effect reflecting the increased demands for response inhibition on the appearance of the unpredictable no-go targets.

2. When two competing response tendencies are activated simultaneously, as in tasks that require response inhibition, response conflict is generated (Botvinick, Cohen, & Carter, 2004). One ERP signature of response conflict is the error-related negativity (ERN), a response-locked component with a latency of 0–120 msec (Van Veen & Carter, 2002; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). If errors on the $SART_{fixed}$ do arise from disengagement of endogenous control, as opposed to inhibitory failure, then less conflict should be generated on error trials. We thus predicted an attenuated ERN on the $SART_{fixed}$ relative to the $SART_{random}$.

3. Finally, two electrophysiological makers of sustained attention will be explored: the LP1 and alpha power. We hypothesize that the level of each of these markers in the pretarget interval should distinguish between correct and incorrect responses on the $SART_{fixed}$. We predict that LP1 amplitudes should be reduced prior to an attentional lapse, whereas alpha power should be increased. We do not predict these effects for the $SART_{random}$ because errors in this condition should arise primarily from differences in processing of the no-go target.

METHODS

Participants

Thirteen normal healthy right-handed college undergraduates (8 women, mean age = 22.3 years, *SD* = 4.2) were recruited by poster advertisement within the university campus. Participants were excluded if they reported any previous history of psychosis, organic brain disorder, epilepsy, serious head injury, or learning disability. All participants gave written informed consent and all procedures were approved by the ethical review boards of St. Vincents Hospital, Fairview, and the School of Psychology, Trinity College Dublin. All participants reported normal or corrected-to-normal vision.

SART Paradigms and Procedure

All participants completed two separate testing sessions: one for the $SART_{fixed}$ and one for the $SART_{random}$. The order in which each session was completed was counterbalanced. The stimuli for the $SART_{fixed}$ were presented sequentially from ''1'' through ''9.'' For each block, 225 digits were presented, representing 25 runs of the 1 to 9 sequence. The $SART_{random}$ shared the exact same parameters as the $SART_{fixed}$, except that stimuli were presented in a random sequence. As in the $SART_{fixed}$, each digit was presented 25 times. Five randomly allocated digit sizes were presented to increase the demands for processing the numerical value and to minimize the possibility that participants would set a search template for some perceptual feature of the target (''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 approximately 150 cm. Digits were presented 0.25° above a central white fixation cross on a gray background. The task specifications were programmed and stimuli were delivered using the Presentation software package (Version 0.75; www.neurobs.com). For each trial, a digit was presented for 150 msec followed by an interstimulus interval of 1000 msec. Participants 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 (no-go target) appeared, where they were required to withhold their response. Participants were instructed to time their button presses to the offset of each stimulus. This kind of ''response-locking'' has been shown to reduce interindividual variability and eliminate speed–accuracy tradeoffs (Stuss, Murphy, Binns, & Alexander, 2003; Manly, Davison, Heutink, Galloway, & Robertson, 2000). In the present study, response locking ensured that similar response strategies were employed by participants for both conditions. Timing of task stimuli and the basic response requirements are demonstrated in Figure 1.

Participants were seated in a dimly lit, sound-attenuated, electrically shielded room. Participants completed a short practice block before each testing session to ensure they had understood the task instructions. In order to obtain sufficient single trials for an ERP analysis of errors (>20), participants undertook an average of 14.1 blocks (range 10–18) within the $SART_{fixed}$ testing session. If a participant had made over 30 errors after 10 blocks, then testing was stopped. Because participants made more errors on the $SART_{random}$, it was only necessary for participants to complete an average of 9.8 blocks (range 8–12). If a participant had made over 30 errors after 8 blocks, then testing was stopped.

ERP Acquisition and Analysis

Continuous EEG was acquired through the high impedance ActiveTwo Biosemi electrode system from 72 scalp

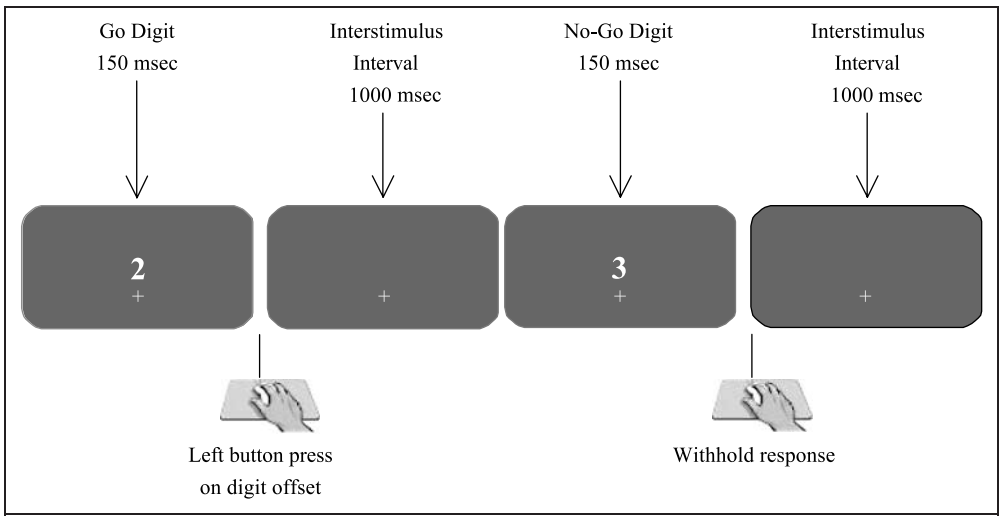

Figure 1. $SART_{fixed}$ task schematic. Demonstrates the sequence of events contained within a go trial (the digit 2) and a no-go trial (the digit 3). The $SART_{random}$ differed only in the fact that the stimuli were presented in an unpredictable sequence.

electrodes, digitized at 512 Hz with an open band-pass from DC to 150 Hz. Vertical eye movements were recorded with two electrodes placed below the left and right eyes, whereas horizontal eye movements were measured with two electrodes at the outer canthus of each eye.

Data were analyzed using BESA Version 5.1 (Brain Electric Source Analysis) software (www.besa.de). For analysis and display purposes, data were average referenced and filtered with a low-pass 0-phase shift 48-dB, 30-Hz filter and a high-pass 0-phase shift 6-dB, 0.3-Hz filter. Stimulus-locked data were segmented into epochs of 100 msec before to 1000 msec after stimulus onset and baseline-corrected relative to the interval −100 to 0 msec. Stimulus-locked data were acquired for go stimuli that were followed by a button press (correct go press), no-go targets (3) that were followed by a correct withhold, and targets that were followed by an error of commission. Response-locked data were segmented into epochs of 400 msec before to 500 msec after button press, and baseline-corrected relative to the interval −400 to −200 msec. Response-locked data were averaged separately for errors of commission and correct go presses. All electrode channels were subjected to an artifact criterion of ±100 μV to reject trials with excessive EMG or other noise transients. The single-trial EEG signals were also corrected for vertical eye movement artifacts by means of a correction procedure developed by Berg and Scherg (1994) and implemented by BESA.

ERP componentry was investigated following the same strategy outlined in Dockree et al. (2005). ERP component structure was confirmed by visual inspection of grand-average waveforms. The width of the latency window used to measure component amplitudes was based on the duration and spatial extent of each component. The early visual components, P1, N1, and P2, were analyzed in the correct go stimulus waveform. Subject-specific maximal amplitude scalp locations and peak latencies at these locations were selected for each individual. This was done to account for significant spatial and temporal variations in sensory ERPs across individuals. A late positive potential (LP1) similar to that observed by Dockree et al. was observed over right occipito-parietal scalp regions following go stimuli on the $SART_{fixed}$. There was little spatial variation in the topography of this component across participants so amplitudes were measured at fixed electrode locations. The LP1 was measured as the average positive voltage in the latency window of 650–950 msec poststimulus at P2, P4, and P6 on go stimuli for both $SART_{fixed}$ and $SART_{random}$. In order to examine modulation of this component by sustained attention, the LP1 was calculated separately for the go stimulus immediately preceding a no-go target to which the participant successfully withheld their response and preceding a no-go target to which the participant made an error of commission.

The no-go N2 and no-go P3 were analyzed on the stimulus-locked correct withhold and commission error waveforms. Because these components showed little spatial variation between individuals, a set electrode location and latency window was used for all participants, but latency windows were calculated separately for the $SART_{fixed}$ and $SART_{random}$. The no-go N2 was strongest at electrode Fz and was measured as the peak negativity between 255 and 295 msec on the $SART_{random}$ and between 235 and 275 msec on the $SART_{fixed}$. On the $SART_{random}$, the no-go P3 was strongest over FCz and was measured as the peak positivity between 339 and 379 msec. There was no equivalent component in the $SART_{fixed}$ ERP so the peak positivity at the same electrode and latency was measured.

The response-locked waveforms for commission errors in both conditions contained an early negative deflection over fronto-central sites known as the ERN and a more posterior late positivity known as the error positivity (Pe). The ERN was strongest over Fz and had a similar peak latency for both conditions. The ERN was therefore measured as the peak negativity between 20 and 100 msec postresponse. The Pe was strongest over CPz for both conditions but had an earlier peak in the $SART_{random}$ compared to the $SART_{fixed}$. Therefore, a window of 150–350 msec was chosen for the $SART_{random}$ and a window of 250–450 msec was chosen for the $SART_{fixed}$. Like the LP1, the Pe does not have a well-defined peak and was therefore measured as the average positive voltage within this latency window. Individual peak latencies for this component were also calculated.

Finally, we investigated whether errors in either condition were preceded by changes in the alpha band. The average power spectrum over the entire recording period was calculated for each participant using the discrete Fourier transform. The location on the scalp of maximum alpha power was selected for each individual, identified by inspection of topographic maps. Each participant's tonic alpha power (μV^2) was calculated as the power in a 4-Hz range centered on the individual alpha frequency (IAF) at the selected electrode site. The IAF is defined as the frequency at which the maximum peak is located within the alpha range in the power spectrum. Next, we calculated mean alpha power over a 1-sec epoch centered on the onset of the go stimulus immediately preceding a no-go target. Separate averages were calculated for stimuli preceding a correct withhold and an error of commission.

Participants made an average of 44 commission errors (*SD* = 28, range 20–92) during the $SART_{fixed}$ testing session and an average of 65 commission errors (*SD* = 48, range 20–194) during the $SART_{random}$ testing session. Because participants made more errors of commission on the $SART_{random}$, it was important to equate the number of single trials that contributed to the averages for these comparisons. This was achieved by randomly excluding individual trials until the number of trials entered into the separate averages was equivalent.

RESULTS

Behavioral Differences

Because the number of blocks completed in the fixed and random conditions was not equal, individual performance data were calculated over the first 8 blocks of testing (all participants completed at least 8 blocks in each condition). Behavioral measures were calculated as an average score per block. Individual $SART_{fixed}$ and $SART_{random}$ scores for errors of commission, errors of omission (failure to press following a go stimulus), mean reaction time on go stimuli (GoRT), mean GoRT variability, and mean reaction time on errors of commission are summarized in Table 1.

Participants made significantly more errors of commission on the $SART_{random}$ but made more errors of omission on the $SART_{fixed}$. Split-half analysis indicated that our participants' commission error rates in both conditions were stable across the entire testing session (Guttman split-half coefficients, $SART_{fixed}$ = .87; $SART_{random}$ = .89), meaning that it is unlikely that differential time-on-task effects might have operated in the $SART_{fixed}$ relative to the $SART_{random}$. There were no significant differences in terms of GoRT or GoRT variability, confirming that the response-locking instruction imposed equivalent response strategies across the two conditions. Nevertheless, the average reaction time on a commission error was significantly faster on the $SART_{random}$ than on the $SART_{fixed}$ [$t(12) = 2.78, p < .05$]. Further, paired-samples t tests indicated that although $SART_{random}$ commission error RTs were significantly faster than the average GoRT in that condition [$t(12) = 6.5$, $p < .001$], commission error RTs on the $SART_{fixed}$ were not significantly different from average GoRT [$t(12) = -0.9, p = .3$].

ERP Findings

Early Stimulus Processing

In order to compare the extent of early visual attention processes across task duration, the amplitude of the P1, N1, and P2 components were averaged across all correct go trials and compared across conditions (see Figure 2). Paired-samples t tests revealed significantly increased processing on the $SART_{random}$ at the latency of the P1 [$t(12) = 2.61, p < .05$] and N1 [$t(12) = -2.9, p < .05$], but not the P2 [$t(12) = 1.16, p = .264$].

Goal Maintenance

Dockree et al. (2005) identified a late positive component (LP1) that was enhanced immediately prior to a no-go target on the $SART_{fixed}$ and that was associated with levels of goal activation. The same component was evident in the present data for the $SART_{fixed}$ over right occipito-parietal sites (see Figure 3). When comparing go stimuli immediately preceding a correct withhold to a no-go target and go stimuli immediately preceding an error of commission, a clear divergence was evident from 650–1000 msec. An attenuated LP1 component was also evident in the go stimulus waveform on the $SART_{random}$, but its amplitude did not appear to vary as a function of the subsequent response to the no-go stimuli. A repeated measures analysis of variance (ANOVA) with two levels of condition (fixed vs. random), two levels of response (preceding a withhold vs. preceding an error of commission), and three levels of electrode (P2, P4, and P6) revealed a significant main effect of response [$F(12) = 4.5, p < .05$] and a significant Condition × Response interaction [$F(12) = 10.5, p < .01$]. The main effect of condition did not reach significance [$F(12) = 1.2, p = .2$]. Post hoc t tests revealed that the interaction was driven by a larger LP1 amplitude prior to a correct withhold, relative to errors of commission, on the $SART_{fixed}$ [P2, $p < .05$; P4, $p < .05$; P6, $p < .05$], and the absence of any such differences on the $SART_{random}$ [P2, $p = .8$; P4, $p = 0.9$; P6, $p = .2$].

In addition to mean alpha power, alpha levels were calculated for a 1-sec epoch around go stimuli that immediately preceded no-go targets on both the $SART_{random}$ and $SART_{fixed}$ (see Table 2). A repeated measures ANOVA with two levels of condition and three levels of response (mean alpha, alpha pre-withhold, alpha pre-error) indicated a close to significant main effect of re-

Table 1. Comparison of Behavioral Performance Measures on the $SART_{fixed}$ and $SART_{random}$

	Fixed	*Random*		
	Mean per Block (SD)	*Mean per Block (SD)*	*t(12)*	*p*
Errors of commission	3.5 (2.5)	7.5 (5.3)	−3.25	.007**
Errors of omission	1.7 (1.5)	0.02 (0.05)	3.35	.01**
Mean GoRT	408.6 (85.1)	415.5 (97.9)	−0.4	.7
Mean GoRT variability	129.5 (68.9)	113.9 (49.9)	−1.7	.1
Mean error RT	429.4 (114)	353.3 (86.4)	2.78	.02*

*$p < .01$.

**$p < .05$.

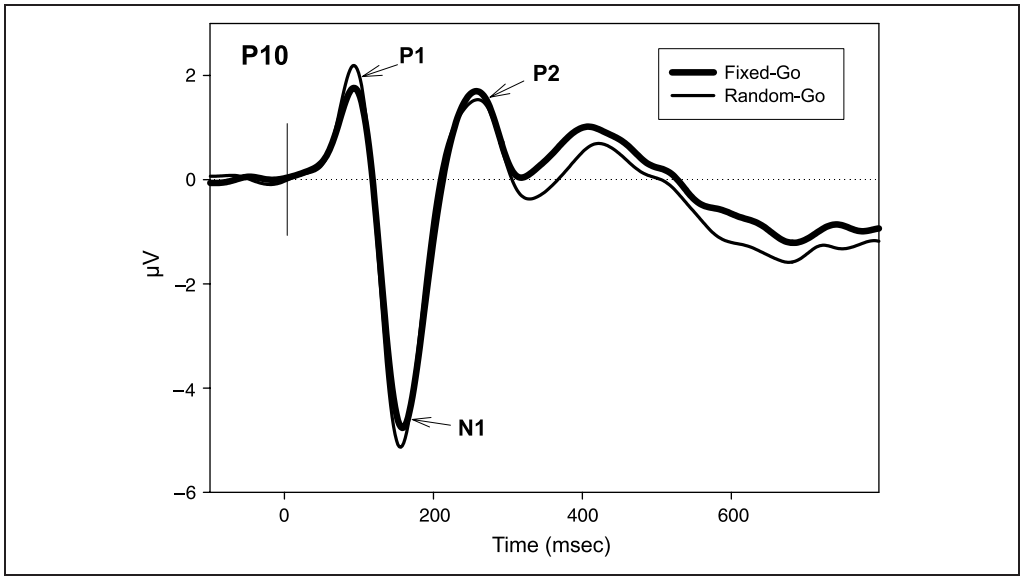

Figure 2. Early stimulus processing on the $SART_{fixed}$ and $SART_{random}$. Displays grand-average waveforms at electrode P10 averaged separately for correct go trials and time-locked to stimulus onset (time point 0). Here we see that the early visual components P1 and N1 differ in amplitude on the $SART_{random}$ relative to the $SART_{fixed}$, reflecting the increased processing requirements associated with the unpredictable stimulus sequence of the $SART_{random}$.

sponse [$F(12) = 3.4$, $p = .053$] and a significant Condition × Response interaction [$F(12) = 5.4$, $p < .05$], but no main effect of task [$F(12) = 1.2$, $p = .3$]. Post hoc analysis indicated that this interaction was driven by modulation of alpha as a function of response type during the $SART_{fixed}$. Alpha power was significantly increased prior to an error relative to the mean alpha power ($p < .05$) and relative to the period preceding a correct withhold on the $SART_{fixed}$ ($p < .05$). Relative to the mean, alpha power was decreased prior to a correct

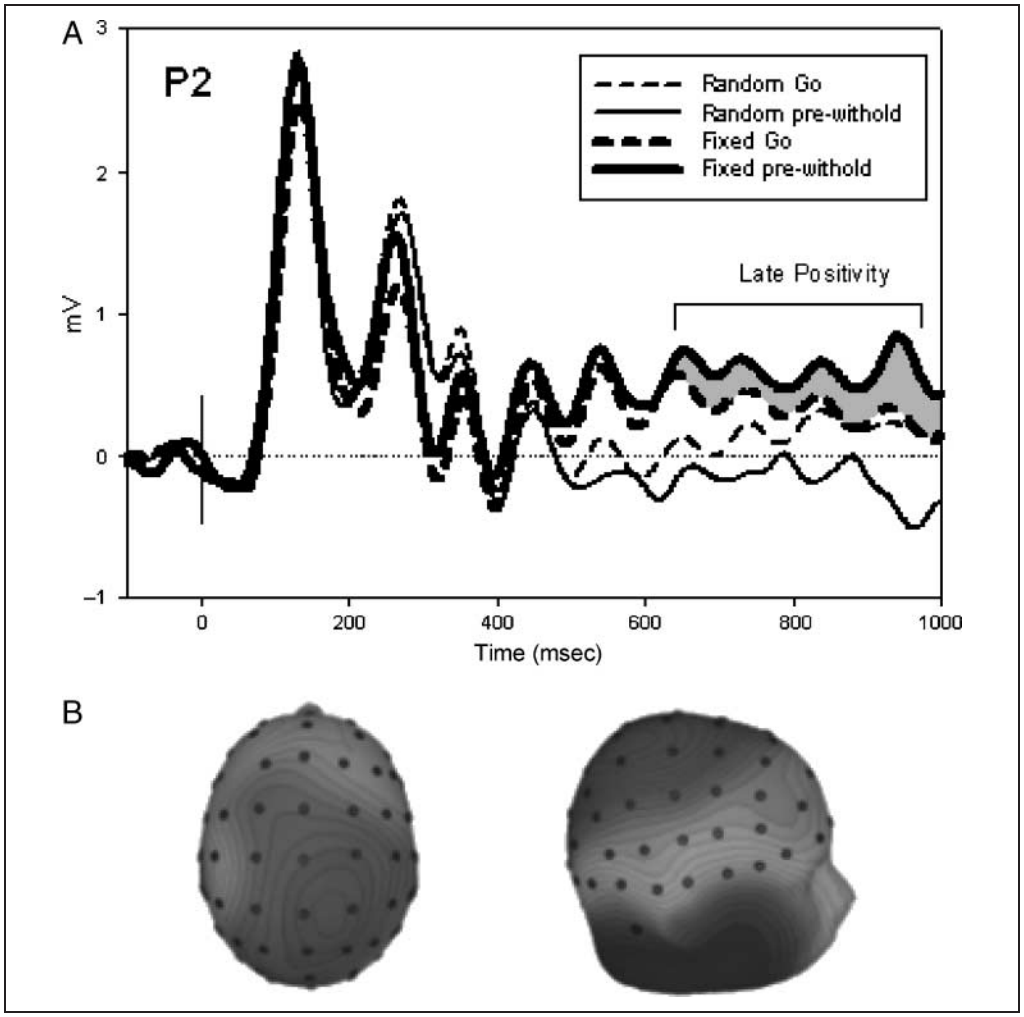

Figure 3. Electrophysiological correlates of goal maintenance on $SART_{fixed}$ and $SART_{random}$. (A) Displays grand-average, stimulus-locked (time point 0) waveform at P2 averaged separately for the go trial immediately preceding a correct withhold and the go trial immediately preceding an error of commission. For the $SART_{fixed}$, the late positive component (LP1) occurs between 650 and 1000 msec after stimulus onset and is smaller prior to an error of commission reflecting the disengagement of goal activation processes. This relationship was not observed on the $SART_{random}$. The distribution of this component across the scalp is displayed in B.

Table 2. Comparison of Alpha Power Measures on the $SART_{fixed}$ and $SART_{random}$

	Fixed	*Random*
	mV^2 (SD)	*mV^2 (SD)*
Mean alpha power	31.2 (34)	29.3 (33.7)
Alpha power pre-withhold	28.1 (32.2)	37.8 (48.6)
Alpha power pre-error	39.4 (44.3)	38.5 (49.2)

withhold, but this effect did not reach significance ($p = .2$). These relationships were not observed for the $SART_{random}$ (alpha pre-error vs. mean alpha, $p = .07$; alpha pre-error vs. pre-withhold, $p = .6$; alpha pre-withhold vs. mean alpha, $p = .8$).

Processing the No-go Target

Stimulus-locked no-go target waveforms for $SART_{fixed}$ and $SART_{random}$ were averaged separately for correct withholds and errors of commission (see Figure 4). Inspection of the no-go target waveforms preceding correct withholds on the $SART_{random}$ revealed a classic N2–P3 complex over fronto-central regions. These two components appeared to be attenuated on errors of commission. On the $SART_{fixed}$, in contrast, a no-go N2 component was observed, but the no-go P3 was absent. Furthermore, the N2 did not appear to distinguish between correct and incorrect performances on no-go trials. To test this observation, separate within-subjects ANOVAs were conducted for the N2 and P3 with two levels of condition (fixed vs. random SART) and two levels of response (correct withhold vs. commission error).

For the no-go N2 there was no main effect of condition [$F(1, 12) = 1.5$, $p = .22$] and no main effect of response [$F(1, 12) = 3.34$, $p = .09$], but there was a significant Condition × Response interaction [$F(1, 12) = 12.03$, $p < .01$]. Post hoc t tests indicated that the interaction was driven by the no-go N2 effect on the $SART_{random}$, which showed a significantly larger amplitude on correct withholds relative to errors of commission ($p < .01$). This effect was absent on the $SART_{fixed}$ ($p = .7$). For the no-go P3, there was a main effect of condition [$F(1, 12) = 14.97$, $p < .01$] and of response [$F(1, 12) = 7.55$, $p < .05$], and a significant Condition × Response interaction [$F(1, 12) = 9.85$, $p < .01$]. On the $SART_{random}$, there were significantly larger no-go P3s on correct withholds relative to errors of commission ($p < .01$); this effect was absent on the $SART_{fixed}$ ($p = .6$).

Error Processing

Commission errors in both conditions elicited an ERN with maximal amplitude over Fz. A within-subjects ANOVA with two levels of condition ($SART_{fixed}$ vs. $SART_{random}$) and two levels of response (go press vs. no-go error of commission) indicated that there was no main effect of condition [$F(12) = 3.8$, $p = .8$], but there was a significant effect of response [$F(12) = 5.2$, $p < .05$] and a significant Condition × Response interaction [$F(12) = 4.9$, $p < .05$]. Post hoc t tests confirmed that the ERN elicited by performance of the $SART_{random}$ was significantly larger following errors than following a correct go response ($p < .05$), suggesting greater response conflict on no-go trials. In contrast, there was no amplitude difference at this latency when comparing correct go responses to no-go error responses on $SART_{fixed}$ ($p = .7$). To ensure that these results were not confounded by potential task-related differences in motor preparation, the analysis was

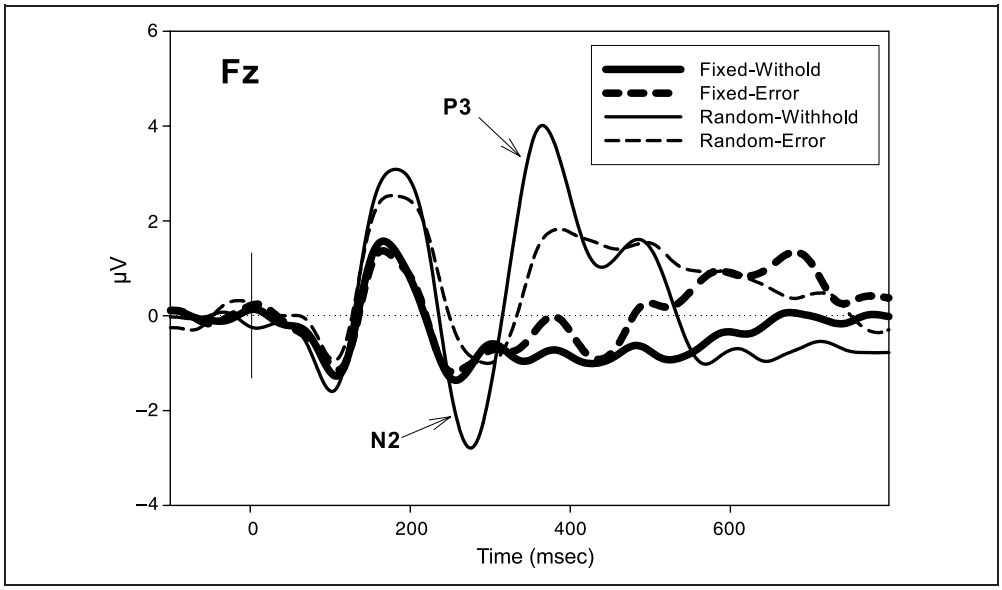

Figure 4. Electrophysiological markers of response inhibition on the $SART_{fixed}$ and $SART_{random}$. Displays grand-average waveforms at Fz averaged separately for correct withholds and commission errors on 3 and time-locked to stimulus onset (time point 0). On the $SART_{random}$, we see that the strong no-go N2–P3 complex on correct withholds is attenuated when participants make an error. On the $SART_{fixed}$, the no-go N2–P3 is virtually absent and does not differentiate between correct and incorrect responses.

repeated using a −100 to 0 preresponse baseline. The Condition × Response interaction was confirmed [$F(12) = 8.9$, $p < .05$]. There were no significant differences in the amplitude of the Pe which was maximal over CPz [$t(12) = -1.67$, $p = .119$], but there was a significant difference in its latency [mean peak latency $SART_{random} = 297.2$, $SD = 95.4$; mean peak latency $SART_{fixed} = 410.4$, $SD = 98.2$; $t(12) = -4.03$, $p < .01$]. Pe amplitude differences remained nonsignificant following reanalysis with the −100 to 0 baseline [$t(12) = -1.7$, $p = .11$]. Error-related ERP components for $SART_{fixed}$ and $SART_{random}$ are illustrated in Figure 5.

DISCUSSION

Our results provide electrophysiological evidence for separable inhibitory and sustained attention neural mechanisms producing errors on go/no-go tasks. Despite the fact that the two task conditions shared identical perceptual and motor demands, the manipulation of target predictability produced two qualitatively different types of error that were associated with distinct electrocortical markers.

As expected, participants made significantly more errors of commission on the $SART_{random}$, which is compatible with the increased demands on inhibitory control caused by the unpredictable target sequence. Asking participants to time their responses to the offset of each stimulus ensured that there were no overall differences in the response strategies employed for the two conditions. The only RT difference observed was on commission errors themselves. On the $SART_{random}$, RTs for commission errors were significantly faster than the average GoRT in that condition suggesting that the erroneous response had been executed before the inhibition process was completed. This is a common finding with go/no-go tasks and, indeed, is a key prediction of race models of response inhibition (Logan, 1994). Conversely, RTs for errors of commission on the $SART_{fixed}$ did not differ from the average GoRT consistent with the view that sustained attention errors occur when participants mindlessly persist with the default go-response mode. Although participants made very few errors of omission in general, significantly more errors of this kind were made on the $SART_{fixed}$. Errors of omission occur when a participant fails to make the requisite response following a go stimulus and are therefore likely to reflect instances where the participant has drifted off task. The increased prevalence of these errors on the $SART_{fixed}$ suggests that the monotony of the task engendered a greater number of momentary drifts of attention than the $SART_{random}$.

The ERP data provide clear evidence that the fixed and random versions of the SART emphasize different neural processes. The P1, N1, and P2 are early visual attention potentials evoked in the occipital cortex that reflect the initial extraction of information from sensory analysis of the stimulus (Luck, Woodman, & Vogel, 2000). The present data indicate that both the P1 and N1 are enhanced on go trials for the $SART_{random}$ relative to the $SART_{fixed}$. According to biased-competition models of attention, frontal control regions bias sensory regions to favor the processing of behaviorally relevant stimuli, resulting in enhanced perceptual representations of those stimuli (e.g., Hopfinger, Buonocore, & Mangun, 2000). Because go stimuli on the $SART_{fixed}$ are entirely predictable and require the same default response, their behavioral relevance is limited. In contrast, because the $SART_{random}$ is unpredictable and every stimulus requires

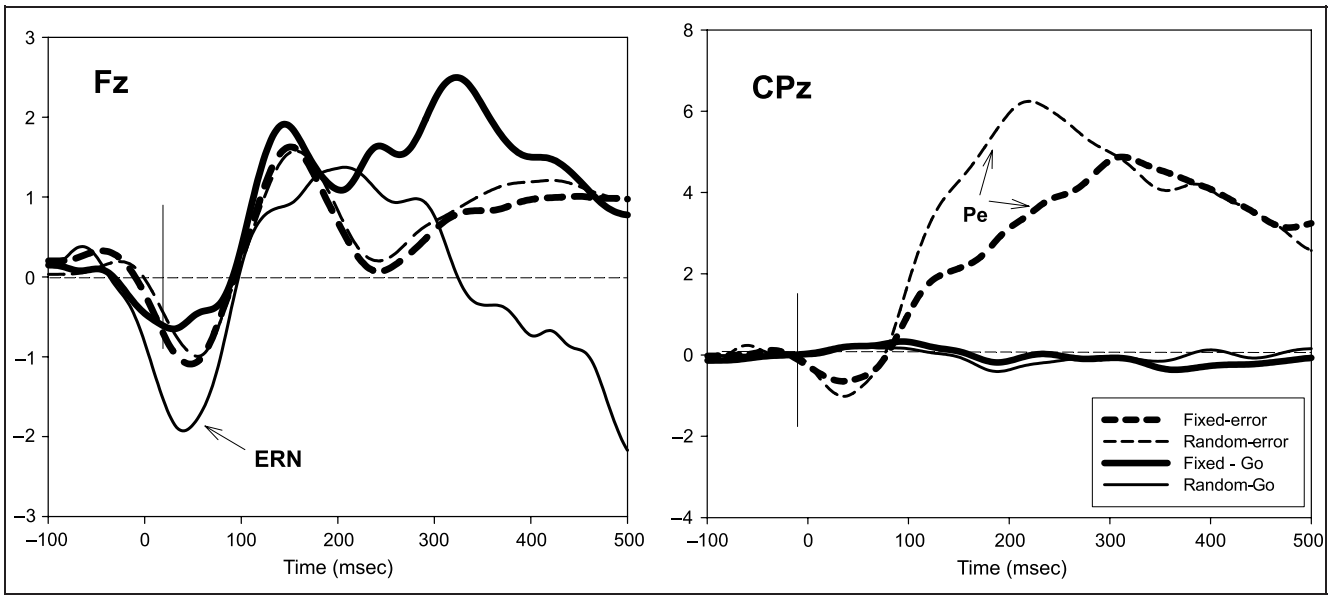

Figure 5. Electrophysiological markers of error processing on $SART_{fixed}$ and $SART_{random}$. Displays grand-average waveforms at Fz and CPz averaged separately for commission errors and correct go responses and time-locked to button-press response (time point 0). At Fz we see a clear ERN for $SART_{random}$ errors but not for $SART_{fixed}$ errors. At CPz we see that errors in both conditions elicited Pe amplitudes of similar magnitude.

identification, enhancement of early stimulus processing would facilitate the speedy detection of a potential no-go stimulus.

The most prominent ERP marker of sustained attention was seen during the later aspects of go stimulus processing. Dockree et al. (2005) previously noted a broadly distributed late positivity over right occipito-parietal areas visible on all go trials of the $SART_{fixed}$. This activity was interpreted as a reflection of goal maintenance and predicted performance on subsequent no-go trials. In the present study, we compared LP1 amplitudes preceding a correct withhold and preceding an error of commission on both the $SART_{fixed}$ and $SART_{random}$. Differences were only apparent on the $SART_{fixed}$, where there was a clear attenuation prior to a lapse. This finding is consistent with the initial hypothesis that reduced levels of sustained attention during the inter-target interval on the $SART_{fixed}$ should weaken the representation of task goals leading to error. Our analysis of alpha power revealed a very similar relationship. We found that levels of alpha only predicted accuracy on the $SART_{fixed}$ such that, relative to mean alpha levels, there was significant synchronization prior to an error, whereas there was a nonsignificant desynchronization prior to a correct withhold. Alpha synchronization has been associated with a resting state of the brain where mental activity is minimal, whereas alpha desynchronization has been linked to the mobilization of attentional resources in response to changing task demands (Klimesch, 1999). Hence, changes in the LP1 and alpha power provide useful electrophysiological precursors of errors on the $SART_{fixed}$ that are evident prior to target onset.

The analysis of ERPs on no-go trials also provides clear evidence that correct withholds and errors of commission on the $SART_{fixed}$ and $SART_{random}$ arise from qualitatively different cognitive mechanisms. No-go trials on the $SART_{random}$ were characterized by a strong N2–P3 complex, which was attenuated when participants made an error. On the $SART_{fixed}$, in contrast, only the no-go N2 component was evident and it did not distinguish between correct responses and errors. In keeping with the present data on the $SART_{random}$, most studies report that the no-go N2 is larger for successful than unsuccessful inhibitions (Falkenstein, 2006). Because a similar component is also observed on trials where there is no inhibitory requirement but where response conflict or uncertainty is high, it has been argued that the no-go N2 does not index response inhibition per se, but rather, reflects related performance monitoring processes (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Van Veen & Carter, 2002). For example, detection of response conflict, reflected in the no-go N2, may provide a ''red flag'' that precedes or initiates response inhibition (Kok et al., 2004). In contrast to the no-go N2, there is a closer link between the no-go P3 and response inhibition in ERP research (e.g., Bekker, Kenemans, Hoeksma, Talsma, & Verbaten, 2005). In the present study, the no-go P3 was clearly modulated by no-go trial performance on the $SART_{random}$, but was absent irrespective of performance on the $SART_{fixed}$. These data indicate that the two ERP components most closely associated with response inhibition do not distinguish correct and incorrect no-go responses on the $SART_{fixed}$.

The response-locked ERP waveforms revealed a distinctive ERN effect when comparing errors of commission to correct go presses on the $SART_{random}$. In contrast, although an attenuated negativity was visible at the same latency of the response-locked waveforms elicited by the $SART_{fixed}$, it did not distinguish between correct and incorrect responses. Like the no-go N2, the ERN is thought to reflect performance monitoring processes such as detection of response conflict or uncertainty (Van Veen & Carter, 2002; Falkenstein et al., 2000). It is therefore revealing that on the $SART_{fixed}$, even when participants responded incorrectly to no-go stimuli, there was no enhancement of the ERN as commonly reported in the error processing literature. Coupled with the finding that RTs did not differ for go and no-go trials, these data are again consistent with the original proposal that errors on the $SART_{fixed}$ occur due to a temporary deactivation of the primary task goal (withhold on 3), leading to persistence with the routine response mode (Manly et al., 2003). In contrast, commission errors on the $SART_{random}$ elicited a clear ERN effect and were associated with faster RTs, suggesting that errors in this condition are more closely related to a failure to cope with two active and competing response contingencies at the moment of no-go stimulus onset. The second error-related component that was evident was a late low-frequency positive component frequently noted in error processing literature and known as the error positivity or Pe (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). The precise function of the Pe is poorly understood, but studies have shown that this component is only apparent when a participant is aware that they have committed an error (O'Connell et al., 2007; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). The absence of any amplitude differences points to equivalent levels of conscious error processing in both conditions but the longer latency of the Pe in the $SART_{fixed}$ condition suggests slower recognition of the error event in keeping with the notion of a temporary drift of attention.

The present study provides direct evidence that electrophysiological processes relating to response inhibition and sustained attention are temporally and physiologically dissociable. The $SART_{random}$ was distinguishable from the $SART_{fixed}$ in terms of its distinct no-go and error-related componentry, indicating a greater requirement for urgent response inhibition and conflict monitoring in this condition. In contrast, activity relating to continuous goal maintenance (LP1 and alpha desynchronization) appeared to be the distinguishing

feature of the $SART_{fixed}$. Errors on the $SART_{random}$ appeared to arise primarily from inefficiencies in reactive cognitive control processes (response inhibition) but errors on the $SART_{fixed}$ were characterized by reduced activation of proactive control processes (sustained attention).

Although it is possible to rule out inhibitory failures as contributing to attentional lapses, sustained attention is inevitably likely to be a prerequisite for response inhibition. Future studies should explore new analysis strategies that allow the differentiation of qualitatively different types of action error within the same task. For example, in a recent study, we reported behavioral evidence to suggest that pure lapses of sustained attention during an inhibitory go/no-go task could be isolated by assessing levels of error awareness (Shalgi, O'Connell, Deouell, & Robertson, 2007). Alternatively, it may be that in neurologically healthy populations the impact of sustained attention on response inhibition is most evident in the form of individual differences. For example, Roche, Garavan, Foxe, and O'Mara (2005) found that participants with higher ratings of everyday absentmindedness had larger N2 and P3 components for successful inhibitions, suggestive of a greater reliance on reactive inhibitory control.

Although only limited neuroanatomical inferences can be drawn based on the current dataset, our findings demonstrate that seemingly identical action errors can result from distinct EF processes that are temporally and physiologically dissociable. This electrophysiological dissociation may prove useful both for characterizing impairment in clinical groups such as TBI and ADHD, and in experimental investigations of the neurochemical and/or molecular genetic correlates of sustained attention and response inhibition.

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