EEG signatures associated with stopping are sensitive to preparation

IAN GREENHOUSE\textsuperscript{a,b} AND JAN R. WESSEL\textsuperscript{b}
\textsuperscript{a}Department of Psychology, University of California, Berkeley, Berkeley, California, USA
\textsuperscript{b}Department of Psychology, University of California, San Diego, La Jolla, California, USA

Abstract
Preparing to stop may “prime” the neural mechanism for stopping and alter brain activity at the time of stopping. Much electroencephalography (EEG) research has studied the N2/P3 complex over frontocentral electrodes during outright stopping. Here, we used differential reward of the stop and go processes in a stop signal task to study the sensitivity of these EEG components to preparation. We found that (a) stopping was faster when it was rewarded; (b) the P3 amplitude was larger for successful versus failed stopping, and this difference was greater when stopping was rewarded over going; (c) the N2 component was observed only on failed stop trials; and (d) there was greater EEG coherence between frontocentral and occipitoparietal electrodes at 12 Hz during the initiation of a go response when stopping was rewarded over going. We propose that frontocentral cortical mechanisms active before and at the time of stopping are sensitive to preparation.

Descriptors: Cognition, EEG/ERP, Response inhibition, Cognitive control, Coherence

Much research has addressed how initiated responses are stopped. Recent evidence from a handful of studies suggests that preparing to stop can influence the stopping process (Chikazoe et al., 2009; Greenhouse, Oldenkamp, & Aron, 2012; Jahfari, Stinear, Claffey, Verbruggen, & Aron, 2010; Leotti & Wager, 2010; Sinopoli, Schachar, & Dennis, 2011; Verbruggen & Logan, 2009a). Behaviorally, rewarding successful stopping induces response slowing in preparation for stopping and also speeds up the stopping process (Boehler, Hopf, Stoppel, & Krebs, 2012; Leotti & Wager, 2010; Sinopoli et al., 2011). This change in the speed of stopping could result from proactive adjustments in motor excitability by reducing the amount of inhibition required to override an initiated response without directly influencing the mechanism involved in stopping. In line with this hypothesis, the stopping process itself need not be affected by preparation. The difference in the speed of stopping instead could result from changes in the perceptual processing of the stop signal stimulus or reduced excitability associated with response execution processes. Alternatively, the stopping mechanism could be “primed” to execute stopping more quickly; that is, the changes in the speed of stopping could reflect changes within the stopping network. Indeed, several fMRI studies lend support to this idea and observed that preparing to stop activates the stopping network. Here, we propose that frontocentral cortical mechanisms active before and at the time of stopping are sensitive to preparation.

The N2/P3 complex is a set of EEG event-related potential (ERP) components that has, for over 30 years, been associated with reactive inhibitory control processes during the performance of the go/no-go and stop signal tasks (de Jong, Coles, Logan, & Gratton, 1990; Dimoska & Johnstone, 2001; van Gaal, Lamme, Fahrenfort, & Ridderinkhof, 2011). The N2 component demonstrates frontocentral scalp topographies and is associated with early motor inhibition. The P3 component demonstrates frontocentral scalp topographies and is associated with later, more reactive inhibitory control processes during the performance of the go/no-go and stop signal tasks (de Jong, Coles, Logan, & Gratton, 1990; Dimoska & Johnstone, 2001; van Gaal, Lamme, Fahrenfort, & Ridderinkhof, 2011). The established finding from these studies is that both the N2 and P3 components demonstrate frontocentral scalp topographies and are enhanced during motor inhibition.

The precise differential properties of the N2 and P3 components in motor inhibition are not clear. Recent evidence suggests that these components are signatures of separate processes associated with inhibitory control, with the former reflecting control over a response plan and the latter reflecting evaluation of motor response inhibition (Gajewski & Falkenstein, 2012; Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2012). One
EEG study implemented a combined go/no-go and stop signal task and also incorporated different proportions of go versus reactive control trials (i.e., no-go and stop trials) (Enríquez-Geppert, Konrad, Pantev, & Huster, 2010). This study found that the frontal-central N2 amplitude was larger for relatively infrequent events, regardless of whether the trial was a go trial, a no-go trial, or a stop trial. In contrast, the P3 amplitude was the largest for successful stop trials, smaller for no-go trials, and smallest for go trials. This pattern was exaggerated when reactive control trials were less frequent. The implications of this functional dissociation between the N2 and P3 components are two-fold. First, these findings suggest that the N2 is a marker for relatively infrequent or unlikely events (Wessel, Danielmeier, Morton, & Ullsperger, 2012), possibly related to the conflict that arises from unexpectedly reconfiguring responses or an increased attentional demand required for such reconfiguration. Second, they suggest that the P3 component is selectively sensitive to reactive control processes. These results extended those of a previous study that also found increased P3 amplitude for infrequent relative to frequent stop trials (Ramautar et al., 2004, but see Dimoska & Johnstone, 2008). However, all these previous studies only manipulated the likelihood of a stop signal, and therefore were unable to conclude whether differences in the P3 amplitude during successful stopping were due to the novelty of the stop signal or due to the influence of preparing to stop. This is an important distinction because, in the former case, P3 amplitude differences could occur in the absence of any influence of preparation while, in the latter case, differences in P3 amplitude could index preparedness to stop. Ruling out one of these possibilities would help to elucidate the functional significance of the P3 in stopping. A more precise characterization of the functional significance of the P3 would be useful in determining whether changes in stop signal task performance represent differences in novelty detection or processes associated with motor inhibition. Therefore, in addition to characterizing the influence of preparation on reactive stopping mechanisms, the current experiment has implications for determining the functional significance of the different N2 and P3 components.

Here, we set out to test whether preparing to stop influences brain mechanisms associated with the reactive stopping of initiated responses. We did this by measuring the N2/P3 complex, which has previously been associated with reactive stopping. We developed a modified stop signal task that incorporated a points system. In one condition, stopping was emphasized over going, and, in the other condition, going was emphasized over stopping. Importantly, the probability of a stop trial was the same in both conditions. We then compared the EEG signatures of interest between the two different conditions. Thus, it was possible to test if the N2/P3 complex that occurs during successful stopping relates to the speed of stopping, and hence demonstrates sensitivity to our reward manipulation. Such a finding would suggest that preparing to stop involves preparation of a brain mechanism involved in reactive stopping.

Method

Participants

Fifteen participants (9 female, 24.7 ± 8.5 years of age, 2 left-handed) were recruited from flyers posted on the University of California, San Diego campus. All participants were screened to rule out any neuropsychological or psychiatric disorders and were not taking any neuropsychiatric medication. One subject was excluded from the analysis due to a very high noise in their EEG data.

Task

We used a modified stop signal task with two conditions. In one condition, more points were awarded for stopping than going, and, in the other condition, the point contingencies were reversed (Figure 1). The task was administered in blocks of 12 trials. A 3-s fixation screen and then one of two possible instruction screens preceded each block. The instructions were either “going = 10 points / stopping = 1 point” or “going = 1 point / stopping = 10 points.” These different point contingencies were selected to emphasize going over stopping (G > S) or stopping over going (S > G), respectively, and alternated from block to block. The starting instruction was counterbalanced across participants. Participants completed one block with each instruction as practice and a total of 1,200 trials in total during testing. This resulted in 50 blocks (600 trials) for each of the two points conditions during testing.

Each trial of the task consisted of the presentation of a left- or right-pointing white arrow stimulus presented in the center of a black computer screen for 1 s or until a response was made. On one third of trials, a red letter “X” occluded the arrow at a brief delay. This X served as a visual stop signal and remained on the screen until 1 s from target onset or until a response was made. The delay between the arrow and the stop signal (i.e., the stop signal delay, SSD) was dynamically adjusted in increments of 50 ms. Eight independent stop signal staircases were used. Two were mapped to the left arrow (starting at 150 and 200 ms) and two were mapped to the right arrow (starting at 150 and 200 ms) for each of the two instruction conditions. This resulted in 50 stop trials within each staircase, or 200 stop trials from each of the two different instruction conditions (400 stop trials in total).

All trials were followed by a 500-ms blank screen interval and then a 500-ms feedback screen, which displayed the points earned on the immediately preceding trial (i.e., 0 points, 1 point, or 10 points). A jittered intertrial interval was used (1.2 ± 0.1 s) to prevent anticipation of the target onset. Additionally, following every 6 blocks of the task, participants were presented with a feedback graph of their overall mean reaction time (RT), averaged across the two different instruction conditions. This informed participants of their overall tendency to change the speed of responding across the testing session.

Participants were seated with their hands in their laps and their little fingers facing downwards. Button boxes were suspended beneath the edge of a desk on which the stimulus computer was positioned. This positioning necessitated an upward movement of the index fingers to make a response and permitted the recording of electromyography (EMG) from the first dorsal interosseous muscles of each hand. These EMG data will not be discussed here. Participants were instructed to respond quickly and accurately to the target arrows using the index fingers of their left and right hands and to try to stop to the stop signal. Participants were told that stopping would not be possible on approximately half of the stop trials due to the SSD adjustments, but that they should try to earn as many points as possible.

The point system was constructed to selectively manipulate the preparedness for stopping. For the G > S condition, 10 points were awarded on trials in which the correct go RT fell within the fastest 25% of the cumulative go RT distribution for that condition, and 1 point was awarded for successful stopping. For the S > G
condition, 1 point was awarded on trials in which the correct go RT fell within the fastest 25% of go RTs for that condition, and 10 points were awarded for successful stopping. Otherwise, zero points were awarded for trials with slow go RTs, choice errors, or failed stopping in both conditions. For the initial G > S and S > G blocks, the 25% RT cutoff was determined using the practice RT distributions.

This point system had two particularly important features. First, awarding points only for fast go RTs and successful stopping encouraged participants to respond quickly and also to try to stop in both conditions. Second, the total number of points that could be earned was largely predetermined since points were awarded on only 25% of go trials (i.e., the fastest 100 trials within each condition) and approximately 50% of stop trials (i.e., approximately 100 successful stop trials in each condition), based upon the dynamic staircase adjustments. Therefore, one condition was not expected to result in a greater number of points earned than the other.

Behavioral Analysis

Mean go RT, total points earned, the mean SSD, the probability of successful inhibition (i.e., p(inhibit)), failed stop RT, and the stop signal reaction time (SSRT) were calculated separately for each of the two task conditions, that is, S > G and G > S. SSRT is an estimate of the duration of the stopping process. Here, we used the integration method to calculate SSRT (Verbruggen & Logan, 2009b; Verbruggen, Chambers, & Logan, 2012). In brief, the integration SSRT is calculated by determining which RT values from the go RT distribution fall at percentiles that correspond to the p(inhibit) for the three most common SSDs. Each SSD is subtracted from its corresponding go RT value. The average of these differences is the integration SSRT.

EEG Acquisition

EEG data were sampled at 512 Hz, using a 32+8 channel BioSemi ActiveTwo system (BioSemi Instrumentation), with a CMS-DRL reference. Four extra electrodes were placed as follows: one on each mastoid (digitally linked, signal average used for subsequent rereferencing of the montage), one lateral to the left eye, and one below the left eye (as electrooculograms [EOGs] to monitor eye movements.)

EEG Preprocessing and Analysis

The EEG data were preprocessed using a combination of EEGLAB (Delorme and Makeig, 2004) (http://www.sccn.ucsd.edu/eeglab) and custom MATLAB scripts in the following stages. First, the mean of each channel was removed. Second, the data were rereferenced to external electrodes placed on the left and right mastoids. Third, the data were filtered with a two-way least-squares FIR filter (low cutoff: 0.5 Hz, high cutoff: 30 Hz) using the EEGLAB “eegfilt” function. Data were then binned into trial epochs, and trials with a signal greater than three standard deviations from the mean probability distribution within each epoch were excluded from subsequent analysis (Delorme, Sejnowski, & Makeig, 2007). The remaining data were submitted to a temporal infomax independent component analysis (Bell & Sejnowski, 1995). Independent components (ICs) that corresponded to stereotypical
artifacts—eye blinks, saccades, electrode artifacts—were identified using a published technique that compares favorably with other artifact rejection techniques (Jung et al., 2000). For each subject, at least one component that corresponded to eye movements/BLINKS was found and removed. Visual inspection before and after rejection confirmed the effectiveness of this procedure. The channelwise reconstructed EEG data was then reconstructed using only the nonartifact ICs using inverse matrix multiplication.

All the remaining stop trials were time-locked to the onset of the stop signal, and these epochs were then averaged within each of the two task conditions. Thus, it was possible to make direct comparisons between the two task conditions for the ERP of interest, that is, the ERP to the stop signal.

In addition, as an exploratory analysis of the condition differences on go trials, we assessed communication between frontal and posterior brain areas that exhibited ERP responses to the stop signal and were also sensitive to our points manipulation. To this end, we computed phase-locking value (PLV; Lachaux, Rodriguez, Martinerie, & Varela, 1999) between frontocentral electrode Cz and occipital electrode Oz, separately for the G > S and S > G successful go trials. These regions of interest were selected based upon the scalp topographies of the ERP components on stop trials. To account for potential effects of volume conduction on the PLV, the continuous channel data were transformed to current source density (CSD) using a MATLAB toolbox (Kayser & Tenke, 2006a, 2006b). These data were then transformed into frequency space using a Hilbert transform for each individual frequency (from 2 to 30 Hz). Subsequently, only the phase-angle information of the resulting transform was used to compute PLV separately for correct go trials in each of the G > S and S > G conditions. To compute event-related PLV, the phase spectra were divided into 873-ms epochs starting from 500 ms before go stimulus onset. This 500-ms prestimulus interval was used as a baseline and subtracted from the 373-ms poststimulus interval. This poststimulus interval was determined based upon the mean go RT for the faster of the two task conditions (G > S). We restricted our analysis to this go RT interval because we were interested in early fronto-occipital communication that preceded motor responses and was therefore less likely to be caused by the spread of cortical motor activity. Differences between the G > S and S > G conditions were tested with samplewise t tests (p < .05, two-sided, corrected for multiple comparisons in the time domain using the false discovery rate procedure) (Benjamini, Krieger, & Yekutieli, 2006).

Results

Behavior

For stopping, the mean SSRT was significantly longer for the G > S condition (288 ± 26 ms) than the S > G condition (249 ± 46 ms), t(13) = 3.6, p < .005 (Figure 2a). This pattern of behavior confirmed that the reward manipulation was influencing subjects’ stopping behavior.

The mean SSD was significantly shorter for the G > S condition (63 ± 39 ms) than the S > G condition (291 ± 121 ms), t(13) = 7.6, p < .001 (Figure 2a). The mean p(inhibit) was also significantly smaller for the G > S condition (0.35 ± 0.14) than the p(inhibit) for the S > G condition (0.58 ± 0.05), t(13) = 5.7, p < .001, and the mean failed stop RT was significantly slower for the S > G condition (420 ± 20 ms) than the G > S condition (328 ± 9 ms), t(13) = 5.7, p < .0001 (Figure 2b). The pattern of p(inhibit) and failed stop RT also replicated the results of the previous study (Leotti & Wagner, 2010). However, the p(inhibit) was relatively low in the G > S condition, and although every subject provided at least 44 successful stop trials, the resulting SSRT estimate may be unreliable (Verbruggen & Logan, 2009b).

The mean go RT was significantly faster for the G > S condition (373 ± 36 ms) than the S > G condition (494 ± 78 ms), t(13) = 6.1, p < .001 (Figure 2b). The mean total points earned for the G > S condition was 1,205 ± 249 and for the S > G condition was 1,148 ± 66, and this was not a significant difference, t(13) = 0.92, p = .4. Combined, this pattern of go RT and points earned indicates that participants were modifying their proactive control strategies in accordance with the different point contingencies; that is, they favored stopping over going in the S > G condition and vice versa.

EEG

Three distinct ERP components were identified for successful and failed stop trials in both task conditions. These included the N2, the P3, and, interestingly, the posterior visual N1. The frontocentral N2
The N2 component peaked around 210 ms after the stop signal, centered over Cz (Figure 3). The P3 component peaked around 300 ms after the stop signal, with a frontocentral topography, centered over Cz (Figure 3). The posterior occipitoparietal N1 peaked first, approximately 190 ms after the onset of the stop signal, centered over electrode Oz (Figure 4).

For each subject, we derived the minimum (N1 and N2) and maximum (P3) peak amplitude of the ERPs at their corresponding electrode sites for successful and failed stop trials in each of the two task conditions. We ran separate analyses of variance (ANOVAs) for each of the three peak ERP amplitudes with the factors condition (S > G vs. G > S) and type of stop trial (successful vs. failed).

Notably, we did not observe a frontocentral N2 component for successful stop trials in either the S > G or G > S condition, quantified at electrode Cz. However, we did observe an N2 at electrode Cz emerging around 200 ms for failed stop trials in both reward conditions (Figure 3a). The N2 peak amplitude on failed stop trials was larger for the S > G than the G > S condition, t(13) = 3.6, p < .005.

As predicted, the P3 amplitude was larger for successful versus failed stopping and was also larger for the S > G than G > S condition, F(1,13) = 117.3, p < .0001 and F(1,13) = 8.3, p < .05, respectively (Figure 3a). Moreover, there was a significant interaction, F(1,13) = 16.3, p < .01 (Figure 3c). Follow-up t tests indicated that the P3 amplitude difference between successful and failed stop trials was larger for the S > G condition than the G > S condition, t(13) = 4.0, p < .01, two-tailed.

The posterior N1 showed a much larger amplitude for the S > G condition for both successful and failed stop trials, F(1,13) = 25.7, p < .0001 (Figure 4a and b). Interestingly, in contrast to the chronologically later frontocentral P3, there was no main effect of successful versus failed stopping and no interaction.

For our exploratory analysis of coherence between electrodes Cz and Oz on go trials, PLV was significantly greater at 12 Hz for the S > G condition than the G > S condition (p < .05, false discovery rate [FDR] corrected) (Figure 5). This difference emerged shortly following Go stimulus onset and persisted for approximately 150 ms. Importantly, this is well before the mean go RT for the G > S condition (373 ms) and the S > G condition (494 ms).
There were no other significant differences between the two task conditions at any other time point or frequency.

**Discussion**

Here, we manipulated the numbers of points that could be earned for stopping successfully versus going quickly in a stop signal task while keeping the probability of a visual stop signal constant. We did this to determine whether neural activity at the time of stopping is influenced by preparation. To our knowledge, this is the only EEG study that has used a points system to manipulate the relative value of stopping versus going to investigate signatures associated with successful stopping. Other EEG studies have manipulated only the likelihood of a stop signal and were therefore unable to disentangle the effects that result from the occurrence of an infrequent event (e.g., an oddball) from those that result from endogenous processes involved in preparing to stop (Dimoska & Johnstone, 2008; Enriquez-Geppert et al., 2010; Ramautar et al., 2004). Our experimental design ensured that the probability of a stop signal was identical between the two reward conditions. Therefore, any differences in the EEG signatures of interest between our task conditions cannot be attributed to differences in the probability of task stimuli. The effects we observed can only be attributed to the way in which subjects modified their behavior in accordance with the different task instructions.

We found that stopping was faster when it was rewarded over going and that the amplitude of a well-characterized ERP signature that emerges at the time of stopping, the frontocentral P3, was also sensitive to the reward manipulation. Specifically, the P3 amplitude was larger for successful than failed stopping, and this difference was more pronounced when stopping was rewarded over going quickly. Interestingly, we also observed that the occipitoparietal N1 component to the stop signal, commonly associated with visual attention (Luck, Woodman, & Vogel, 2000), showed increased amplitude when stopping was rewarded over going quickly. However, N1 amplitude did not differ between successful and failed stop trials. Thus, manipulating reward for stopping influenced brain activity over middle frontal cortex (indexed by the P3) that related to both stopping speed and success. This implies that preparing to stop facilitates stopping by influencing the dorsomedial frontal cortex and/or its connected regions.

---

**Figure 4.** A: The average ERP measured at electrode Oz is presented for successful and failed stop trials for the two different task conditions. B: The ERP difference wave for successful–failed stopping is presented for the two different task conditions and indicates that there were no condition differences in the N2. C: Scalp topographies for the peak amplitude of the occipitoparietal N1 are presented for successful and failed stop trials for each condition along with the difference and t-value maps. The emergence of the frontocentral N2 is also visible for failed stopping. SS = successful stop; FS = failed stop; G > S = going quickly earned 10 points and successful stopping earned 1 point; S > G = successful stopping earned 10 points and going quickly earned 1 point.
Moreover, we observed greater coherence between frontocentral and occipitoparietal electrodes on go trials during blocks in which stopping was valued over going. Such a signature may indicate communication between brain areas involved in the detection of a visual stop signal and brain areas involved in implementing outright stopping. Other studies have observed this type of prefrontal-occipital communication during adaptive behaviors, ostensibly to amplify early sensory information processing relevant to a motor task (Danielmeier, Eichele, Forstmann, Tittgemeyer, & Ullsperger, 2011; King, Korb, von Cramon, & Ullsperger, 2010; Zanto, Rubens, Thangavel, & Gazzaley, 2011).

The P3

We found that the frontocentral P3 showed greater amplitude for successful versus failed stop trials and that this difference was larger when successful stopping was rewarded over going. Notably, these changes in the P3 amplitude coincided with changes in SSRT. Therefore, the P3 may reflect a neural mechanism that is involved at the time of stopping and is also sensitive to preparation. Thus, preparing to stop could prime the dorsomedial frontal cortex for stopping.

Although the P3 peak occurred after the estimated completion of the stopping process for most subjects, the peak amplitude may result from earlier neural events. Importantly, differences in the P3 wave started to emerge around 200 ms after the stop signal, within the time frame of the stopping process. One previous study that also reported a P3 amplitude difference between successful and failed stopping noted that the difference reached significance approximately 20 ms before completion of the estimated SSRT for that study (Kok et al., 2004). Our results are in accordance with that previous finding.

It is commonly accepted that the P3 is comprised of two positive-going potentials that occur in close temporal proximity: an early P3a with a frontal distribution that peaks around 240 ms after a stimulus and a later P3b with a parietal distribution that peaks around 350 ms after a stimulus (Squires, Squires, & Hillyard, 1975). The P3 waves we observed here contain two peaks at the approximate latencies of the P3a and P3b. The P3a and P3b are believed to occur when working memory is updated with new information (Polich & Kok, 1995), and the P3a is hypothesized to reflect an attentional process that initiates the inhibition of ongoing activity (Polich, 2007). However, further evidence is required to substantiate this hypothesis. The larger difference between successful and failed stop trials that we observed for the S > G condition than the G > S condition may reflect a larger change in an underlying attention-driven process that may or may not itself be inhibitory. Such a putative mechanism could facilitate the initiation of the stop command and lead to faster stopping.

The N2

We observed a frontocentral N2 component time-locked to the stop signal for both task conditions at electrode Cz but only for failed stop trials (Figures 3a and 4c). This pattern replicates a previous study that observed changes in the frontocentral N2 for failed but not successful stopping (Kok et al., 2004) and extends the results of two previous studies that reported greater sensitivity to the stop signal for the P3 than the N2 component (Enriquez-Geppert et al., 2010; Ramaurat et al., 2004). Thus, the frontocentral N2 may be sensitive to the reward manipulation implemented here, but only when there is a failure to stop. This leaves open the possibility that the N2 reflects error processing in the stop signal task, similar to the feedback-related negativity (Milner, Braun, & Coles, 1997; Simons, 2010; Wessel et al., 2012). We note that the topography of the N2 we observed was not right lateralized as has been reported in some previous studies (e.g., Liotti et al., 2010; Schmajuk et al., 2006). The frontocentral N2 component reported here may reflect a different mechanism than that which contributes to the right frontal N2, although these signatures do sometimes co-occur (Enriquez-Geppert et al., 2010).

The N1

We also detected a large difference between the two reward conditions in the posterior occipitoparietal N1 component, centered at electrode Oz. This component is believed to reflect visual attention processes (Luck et al., 2000), and in this case likely reflects differences in attention to the visual stop signal. Interestingly, we did not observe a difference between successful and failed stop trials in
either reward condition. This pattern suggests that visual attention to the stop signal may not be related to stopping success, at least in this task. Moreover, it suggests that the differences in the P3 amplitude for successful versus failed stopping are unlikely to have resulted from changes in visual attention.

**Frontal-Occipital Phase Coherence**

In addition to the ERP measures of interest, we observed greater phase coherence between electrode Cz and Oz for the S > G condition than the G > S condition. This occurred within 150 ms of go stimulus onset on successful go trials and only at 12 Hz. Coherence in the alpha (8–12 Hz) frequency band between frontal and posterior electrodes has recently been identified as a candidate for top-down modulation of visual attention in working memory tasks (Bollinger, Rubens, Zanto, & Gazzaley, 2010; Capotosto, Babiloni, Romani, & Corbetta, 2009; Zanto et al., 2011; Zanto, Rubens, Bollinger, & Gazzaley, 2010). We observed EEG phase coherence between occipitoparietal brain areas, likely involved in the detection of the visual stop signal, and frontocentral areas implicated in outright stopping. Coherence of this kind is hypothesized to reflect neural communication (Fries, 2005). While we are unable to make claims about the direction of this communication, the difference between the S > G and G > S conditions suggests that it is sensitive to the points manipulation we used. Therefore, this early phase coherence (within 150 ms of the target stimulus) may reflect an anticipatory stopping-related strategy that is mediated by motivation.

**Neural Mechanisms**

Putative neural sources of the N2/P3 complex were recently identified in a study that utilized simultaneous EEG and fMRI (Huster et al., 2011). This study reported that the N2/P3 complex was associated with an increased blood-oxygen-level-dependent (BOLD) signal within the presupplementary motor area (pre-SMA), striatum, anterior midcingulate, and anterior insula. These brain regions largely overlap with those believed to participate in a frontobasal ganglia network for stopping outright (Aron et al., 2007). Considered together with our results, the P3 component may reflect activity changes at frontocortical cortical nodes within this network during stopping, for example, the pre-SMA and its connected regions.

**Conclusion**

We implemented a modified stop signal task to investigate the effects of differentially rewarding either stopping or going on EEG signatures associated with response inhibition. We observed that rewarding stopping over going resulted in faster SSRT and a larger frontocentral P3. The P3 component exhibited increased amplitude for successful versus failed stopping, and, critically, this effect was greater when stopping was rewarded over going. This finding suggests that the P3 reflects a control process at the time of stopping that is dynamically adjusted with changing motivational states.

**References**


(Rceived December 21, 2012; Accepted April 18, 2013)