

# Lapses in a Prefrontal–Extrastriate Preparatory Attention Network Predict Mistakes

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## Abstract

■ Mistakes are common to all forms of behavior but there is disagreement about what causes errors. We recorded electrophysiological and behavioral measures in a letter discrimination task to examine whether deficits in preparatory attention predicted subsequent response errors. Error trials were character-

ized by decreased frontal–central preparatory attention event-related potentials (ERPs) prior to stimulus presentation and decreased extrastriate sensory ERPs during visual processing. These findings indicate that transient lapses in a prefrontal–extrastriate preparatory attention network can lead to response errors. ■

## INTRODUCTION

The frontal lobes support flexible behavior by allowing a diversity of context-dependent behaviors in response to the same stimulus. Mesulam (1998) proposes that the prefrontal cortex (PFCx) enables behavioral responses to become contingent rather than obligatory. To accomplish this, the PFCx must be able to elaborate the stimulus–response associations by linking them to contextual information. The PFCx does so via the interaction of several mechanisms. Inhibitory mechanisms enable the suppression of prepotent responses, diminish interference from irrelevant information, and focus neural resources on task-relevant information. The PFCx also supports mechanisms that hold and manipulate information on-line without requiring the stimulus to be physically present (Curtis & D’Esposito, 2003). The PFCx also mediates the ability to map the significance of events and allocate attention to both internal and external events (Barcelo, Suwazono, & Knight, 2000). These PFCx-dependent mechanisms can support top-down control by creating stimulus–response associations, flexibly updating these associations when the context changes, and overriding prepotent responses to stimuli. Top-down control assures that response selection is biased in a direction consistent with behavioral goals (Boettiger & D’Esposito, 2005; Miller & Cohen, 2001; Asaad, Rainer, & Miller, 1998, 2000; Fuster, 2000; Mesulam, 1998).

Preparatory attention is a cognitive process critical for top-down control. Extensive physiological data exist on the underlying neural substrates of preparatory attention. Single cell recording studies in monkeys report baseline increases in neural firing in both the PFCx and

the parietal cortex when monkeys anticipate an event (Desimone, 1996). Furthermore, cortical slow potentials have been recorded from rhesus monkeys using transcortical electrodes while they wait for an imperative stimulus (Donchin, Otto, Gerbrandt, & Pribram, 1971). Similarly, functional magnetic imaging (fMRI) studies have documented sustained activity in the dorsal frontal–intraparietal network during the prestimulus anticipatory period (Hopfinger, Buonocore, & Mangun, 2000). In electroencephalogram (EEG), cortical slow potentials, which index cortical excitability, are evident while human subjects prepare for an event (Hillyard, 1973).

The Contingent Negative Variation (CNV) is an event-related slow wave that indexes preparatory attention (Dours, Karrer, & Rosenfeld, 1987; Tecce, 1972) and is characterized by a negative DC shift occurring between a warning and imperative stimulus (Walter, Cooper, Aldridge, McCallum, & Winter, 1964). The early phase of the CNV is associated with processes used to evaluate the warning stimulus, whereas the later phase of the CNV indexes preparation for the analysis and motor response to the imperative stimulus (Hillyard, 1969, 1973).

Single cell recordings (Fuster, 1990) and EEG studies (Rockstroh, Muller, Wagner, Cohen, & Elbert, 1993) confirm that the CNV reflects increases in PFCx excitability. EEG source localization algorithms and lesion studies suggest that the CNV is generated in part by the PFCx (Rosahl & Knight, 1995; Low, 1986). The surface DC negativity of the CNV is the result of depolarized apical dendrites of cortical pyramidal cells by thalamic afferents (Birbaumer, Elbert, Canavan, & Rockstroh, 1990). Research on the “behaviorally contingent activity” in animals, considered the equivalent of the CNV in humans, proposes that the CNV is mediated by cortical–basal ganglia–thalamo-cortical circuits (Ikeda et al.,

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1997). Moreover, slow potentials such as the CNV are dependent on the cholinergic system stemming from the nucleus basalis (Pirch, Corbus, Rigdon, & Lyness, 1986). Cholinergic neurons in the nucleus basalis in monkeys fire to conditioned stimuli (Rolls, 1982), and destruction of these neurons results in learning and memory deficits (Friedman, Lerer, & Kuster, 1983). These cortical neurons show a slow muscarinic depolarization leading to facilitation of slow excitatory postsynaptic potentials secondary to increases in acetylcholine (Marczynski, 1978).

Birbaumer et al. (1990) have proposed that slow waves such as the CNV are important in setting cortical excitability thresholds. The state in which neurons are excited, but below firing threshold, is termed “cerebral potentiality.” Birbaumer et al. suggest that when networks are prepared, their response to incoming stimuli is more efficient. Consistent with this notion, data show that activity in the extrastriate cortex may be modulated by such mechanisms (Yago, Duarte, Wong, Barcelo, & Knight, 2004; Barcelo et al., 2000; Hillyard, Vogel, & Luck, 1998; Näätänen, 1988; Harter, Aine, & Schroeder, 1982).

The effects of attention on the extrastriate cortex during visual tasks have traditionally been observed as changes in amplitude of obligatory potentials such as the P1 and N1 (Yago et al., 2004; Reynolds, Pasternak, & Desimone, 2000; Heinze et al., 1994). The P1 is a positive event-related potential (ERP) with a peak amplitude between 80 and 140 msec, and the N1 is a negative ERP with a peak amplitude between 150 and 200 msec. These potentials have been localized, using current source density measures and dipole modeling, to the ventral–lateral occipital scalp and the occipital–parietal cortex, respectively. Pre-perceptual processes such as spatial attention, rather than processes associated with analyzing stimulus features, are thought to underlie amplitude changes in these obligatory potentials (Hillyard, Mangun, Woldorff, & Luck, 1995; Mangun, 1995). Amplitude decreases in the P1 reflect suppression of unattended stimuli, whereas amplitude increases in the N1 reflect enhancement of attended stimuli (Luck & Hillyard, 1995; Luck et al., 1994).

However, local gain mechanisms, such as those proposed to mediate changes in amplitude due to spatial attention, do not account for changes in amplitude when discrimination of stimulus features is required (Hillyard et al., 1998). PFCx-mediated slow negative potentials overlap these obligatory potentials (Ritter, Simson, & Vaughan, 1983, 1988). Various names have been given to these slow waves such as the NA wave (Ritter et al., 1983), N1 effect or discrimination N1 (Nd) (Hillyard, Hink, Schwent, & Picton, 1973), processing negativity (PN) (Näätänen, Gaillard, & Mantysalo, 1978), and selection negativity (SN) (Harter et al., 1982). Therefore, sensory processing in the extrastriate cortex is influenced by both local suppression and enhance-

ment processes, as well as by PFCx-mediated sustained slow wave activity.

One might imagine that such a rich control mechanism would prevent errors. However, behavioral mistakes seem to be inevitable in virtually all real world and experimental situations. Why then do we make mistakes? Several possibilities have been proposed in the literature, including unresolved stimulus–response conflict, lapses in action monitoring, and hasty selection of the response. Furthermore, behavioral studies suggest that inherent fluctuations in executive control processes can contribute to response errors (West, 1999). These studies propose that lapses in executive control disrupt goal-directed behavior. The aim of this study was to characterize physiological deficit/s underlying temporary lapses in executive control that might result in behavioral errors. More specifically, we tested the idea that lapses in preparatory attention predict response errors. To investigate this hypothesis, we used EEG to examine changes in the CNV during a visual letter discrimination task.

## METHODS

### Subjects

Nineteen adult subjects (11 women and 8 men; mean age = 24 years) participated in the study. None reported any history of neurological or psychiatric problems. All were right-handed and had normal or corrected-to-normal vision. Each subject gave informed written consent prior to being tested and was paid for their participation. The experimental procedures were approved by and conducted in compliance with the Committee for the Protection of Human Subjects for the University of California, Berkeley.

### Task

Subjects sat in a sound-attenuated booth 4 feet in front of a PC-controlled 21-in. computer monitor. The response box was placed in the center of a wooden table built between the arms of the chair (above the subject's lap). A flanker letter discrimination task was used. Subjects were instructed to respond as quickly and accurately as possible. They were also told not to dwell on a mistake but rather keep pace with the experiment so that they could get ready for the next trial. The task required subjects to map the letter H to a right button press with their right index finger and the letter S to a left button press with their left index finger. An instruction screen was presented at time zero for 200 msec that cued the subject to the color of the letter that needed to be attended. Two color dimensions, red and green, were used in the task. The instruction screen was followed by a centrally located fixation cross that appeared for a randomly selected time length between 400

and 800 msec. A stimulus slide followed the fixation slide and was composed of the centrally presented fixation in addition to one letter on each side of it. The stimuli letters were always either an “S” or an “H” and could randomly appear on either side of the fixation mark. The letters were each colored red or green and the target letter was the letter painted in the color identified by the instruction slide. The subject had up to 1000 msec to successfully press the button corresponding to the target letter. A randomly selected time length between 400 and 800 msec was given after the response and before the onset of the next trial. Subjects completed five blocks of 96 trials each with rest periods in between the blocks (Figure 1).

### Recording Procedures

EEG activity was recorded from 63 tin electrodes according to the modified international 10–20 system in an Electro-Cap. The channels were referenced to linked electrodes placed over the left and right mastoids. External electrodes above and below the right eye monitored vertical eye movements, whereas external electrodes over the lateral aspect of the left and right eyes monitored horizontal eye movements. All channels were amplified (20K) and band-pass filtered (0.1 to 80 Hz; SA Instrumentation, San Diego, CA). The data were sampled and digitized at 256 Hz (Neuroscan 4.1, Sterling, VA and Keithley DAS-1802HC Metrabyte AD card). Post-processing of the data was done with MANSCAN (SAM Technology, San Francisco, CA).

### Data Analysis

The experiment was composed of five blocks of 96 trials each. Subjects made a mean of 60 ( $SD = 31$ ) errors in the experiment. EEG epochs containing eye blinks, saccades, movement, excess muscle activity, and amplifier-

related artifacts were removed before averaging. On average, we removed 31.7% ( $SD = 21.3\%$ ) of trials due to such artifacts. The stimulus-locked potentials were averaged from 0.2 sec before to 0.8 sec after the stimulus. The response-locked potentials were averaged from 0.2 sec before to 0.4 sec after the response. The averaged ERPs were band-pass filtered between 0.5 and 20 Hz. The first 0.05 sec of each averaged potential served as baseline (mean removed from  $-0.2$  to  $-0.15$  sec before the stimulus). We examined four different trial types: (1) correct trials neither preceding nor following an error (correct); (2) correct trials preceding an error (before); (3) correct trials following an error (after); and (4) incorrect trials (error).

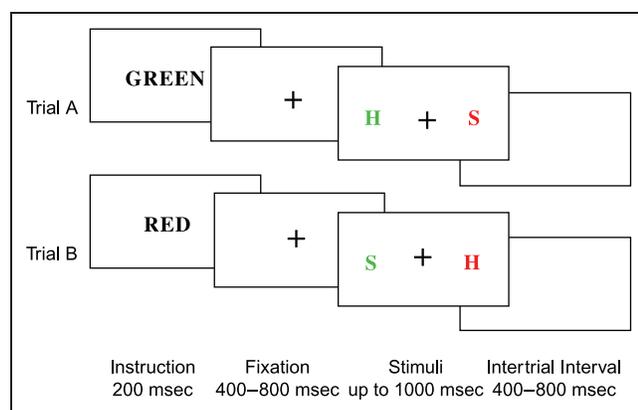
The statistical analysis of the ERP components was done on the mean ERP amplitudes. The mean amplitudes were calculated for each individual subject using the same electrode location and time window across all subjects unless otherwise specified in the Results section. The electrode locations and time windows are specified in the ERP Results section. Because the onset time and width of the reorientation potentials fluctuated between subjects, we identified a unique time window around the peak for each individual subject. Correlations were calculated using difference waves (correct minus error).

We used an initial Omnibus test to restrict comparisons when we did not have a prior hypothesis. We followed significant effects and interactions of these general tests with two-tailed paired-samples  $t$  tests. Planned comparisons were conducted when appropriate. All  $p$  statistics were reported using the Huynh–Feldt correction.

### Slow Wave

The CNV is a superposition of slow waves representing both motor preparation and stimulus anticipation. When activity related to motor preparation, termed the readiness potential (RP), is removed from the CNV, either via mathematical or experimental manipulations, the remaining waveform, named the stimulus preceding negativity (SPN), can be observed (Brunia, 1988; Damen & Brunia, 1987). As we did not segregate activity related to motor preparation, we used the term “CNV” to identify the slow wave during the preparatory attention prestimulus ( $-0.1$  to  $0$  sec) and poststimulus ( $0$  to  $+0.1$  sec) time epoch in both frontal and extrastriate electrode sites. We used the term “sustained negativity” to refer to the slow wave during the visual processing epoch ( $0.1$  to  $0.3$  sec).

We analyzed the slow wave data using a longer prestimulus and poststimulus window ( $-1$  to  $1.5$  sec), as is employed in many CNV studies. However, as a result of using the longer prestimulus window, we lost twice as many trials due to artifacts, compared to the shorter prestimulus window ( $-0.2$  to  $0.8$  sec) reported here. The reduced number of trials created noisier single-subject averages, resulting in reduced statistical



**Figure 1.** Flanker letter discrimination task (right-hand response to H). Schematic of stimuli and task parameters. In Trial A the correct answer is H (high conflict) and in Trial B the correct answer is also H (low conflict).

power. The window length we employed is conventionally used when observing stimulus-locked events and incorporates the time epoch in which we observed a difference between correct and error trials in the longer analysis window.

We used a 0.1-Hz low-frequency cutoff (9 Hz low pass and short  $-0.2$  to  $0.8$  sec analysis window) and observed a decreased fronto-central CNV for error trials. However, we were not able to observe reliable effects in lateral extrastriate sites (PO7 and PO8) with the 0.1-Hz cutoff. We assessed several low-pass filter settings and found that a 0.5-Hz low-frequency cutoff was able to reliably extract both fronto-central and extrastriate slow wave effects. It is important to note that using a frequency cutoff on the order of 0.5 Hz is not unusual (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988).

## RESULTS

### Accuracy and Reaction Time

The mean accuracy for the group was 87% ( $SD = 9\%$ ). The mean reaction time (RT) for correct trials was 590 msec ( $SD = 98$  msec), error trials 619 msec ( $SD = 121$  msec), before error trials 603 msec ( $SD = 112$  msec), and after error trials 641 msec ( $SD = 105$  msec). An initial Omnibus test revealed a difference in RTs [ $F(3,54) = 10.56, p < .001$ ]. Planned comparisons demonstrated that correct trials were faster than error trials [ $t(18) = -2.43, p = .026$ ] and that after error trials were slower than correct trials [ $t(18) = 9.30, p < .001$ ]. Although after error trials were slower than error trials, this effect was not statistically significant [ $t(18) = 1.84,$

$p = .082$ ]. Before error trials showed an increased RT compared with correct trials, but this effect was only marginally significant [ $t(18) = 2.02, p = .059$ ].

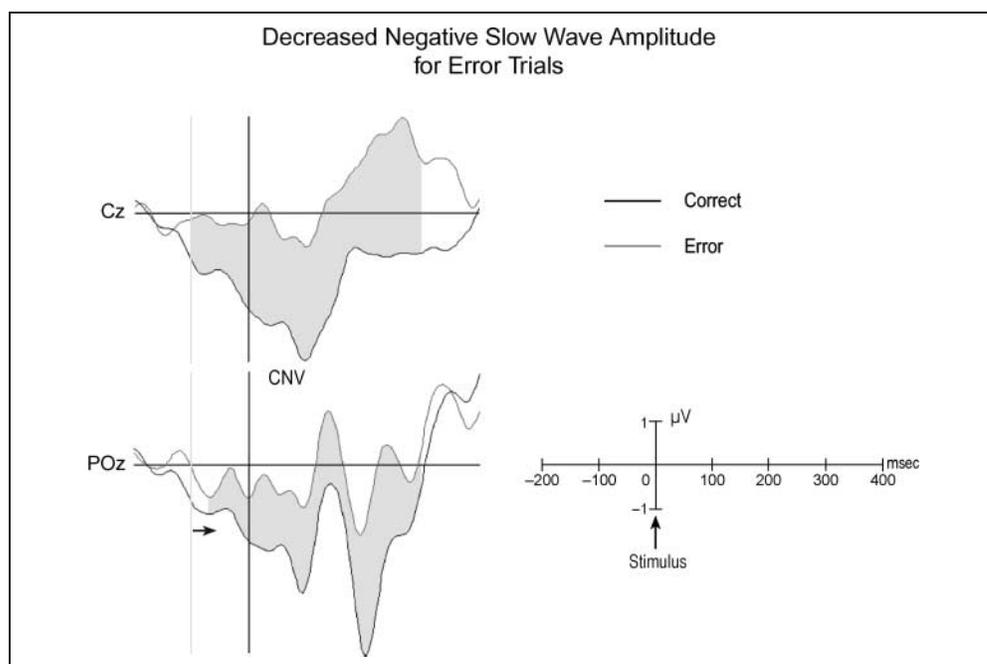
### Stimulus-locked Potentials

#### Preparatory Attention

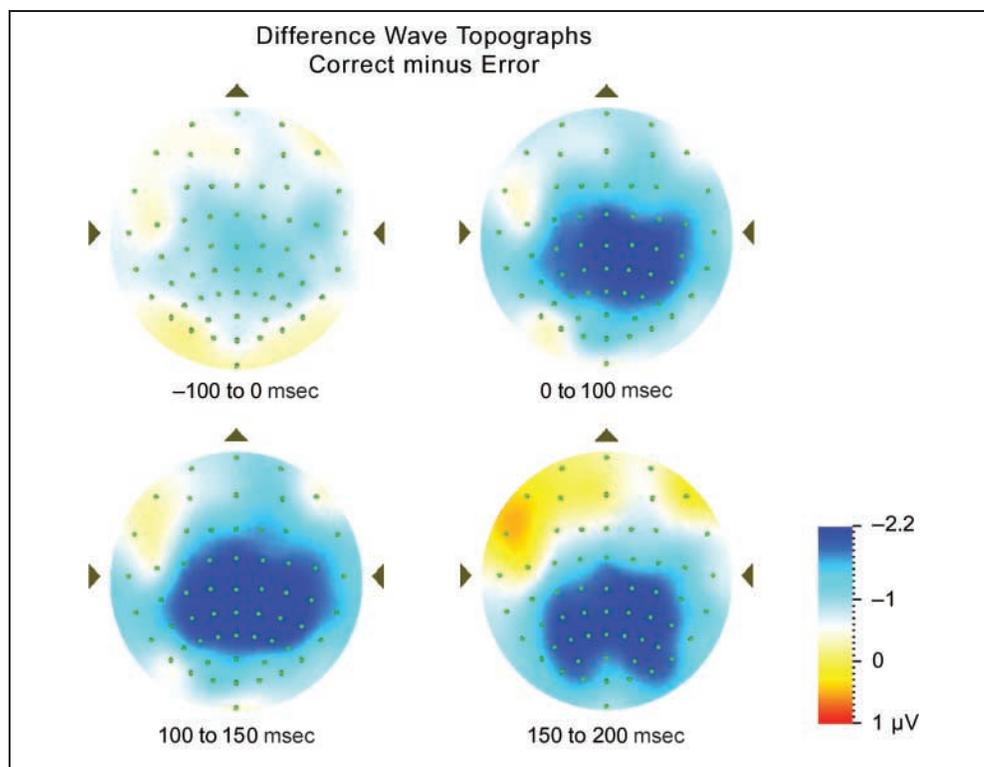
The stimulus-locked CNV was used to address our main hypothesis that errors are preceded by a decrease in preparatory attention. Error trials were compared with correct trials (condition  $-0.1$  to  $0$  sec prior to stimulus presentation in 14 electrodes: Fp1, Fp2, Fpz, F3, F4, Fz, FCz, C3, C4, Cz, CPz, PO7, PO8, POz. Electrode (14) by condition (2) analysis revealed an effect of electrode [ $F(13,208) = 6.41, p = .001$ ] and condition [ $F(1,16) = 5.52, p = .032$ ], with no interaction between these two factors ( $p = .517$ ). We followed this general test with planned comparisons and found that error trials generated a significantly decreased CNV during the pre-stimulus anticipatory processing time at frontal-central electrodes [FCz, Cz, CPz, POz:  $t(18) > -3.37, p < .013$ ] (Figures 2 and 3).

The decrease in CNV amplitude for error trials appeared to persist for the ensuing 100 msec ( $0$  to  $0.1$  sec), so we conducted the same analysis during this epoch. We found an effect of electrode [ $F(13,195) = 7.09, p < .001$ ] and condition [ $F(1,15) = 17.46, p = .001$ ], with no interaction between these two factors ( $p = .092$ ). Planned comparisons confirmed the continued reduction of negative amplitude for error trials compared to correct trials during this epoch at frontal-central sites [FCz, Cz, CPz, POz:  $t(18) > -4.10, p < .004$ ] (Figures 2 and 3).

**Figure 2.** Preparatory attention and visual processing ERPs: Correct vs. Error. ERPs are stimulus-locked. The top graph shows activity at electrode Cz and the bottom graph shows activity at electrode POz. The solid waveform represents correct trials and the dotted waveform represents error trials. Correct trials are characterized by a large negative potential over frontal, central, and posterior sites between  $-100$  and  $300$  msec that is reduced on error trials. At electrode POz, the arrow from the gray vertical line to the gray shaded area shows that error trials are statistically reduced compared to correct trials at central sites 30 msec before this effect is evident at extrastriate electrodes.



**Figure 3.** Difference wave topographical maps: Correct minus Error. The topographical maps depict the difference in activity between stimulus-locked correct and error trials. Topographs reveal that the large negativity begins at frontal-central sites approximately 100 msec before the stimulus is presented. The activity then spreads to more anterior, lateral, and posterior sites after stimulus presentation. Negative activity is maximal in lateral extrastriate visual processing sites between 150 and 200 msec.



### Visual Processing

To address whether the frontal-central CNV influenced visual processing in the extrastriate cortex, we evaluated correct and incorrect trials between 100 and 400 msec in consecutive 100-msec epochs. During the first epoch, 100 to 200 msec, an effect of electrode [ $F(13,208) = 9.62, p < .001$ ] and condition [ $F(1,16) = 5.90, p = .027$ ], with no interaction between these two factors ( $p = .290$ ), was revealed. In planned comparisons, we found that correct trials had larger negative amplitude than error trials at extrastriate sites [PO7, PO8, POz:  $t(18) > -3.55, p < .037$ ] (Figures 2 and 3). The next 100 msec in visual processing, 200 to 300 msec following the stimulus presentation, also showed an effect of electrode [ $F(13,208) = 9.62, p < .001$ ] and condition [ $F(1,16) = 8.08, p = .012$ ], with no interaction between these two factors ( $p = .133$ ). A difference between correct and error trials was present at extrastriate electrodes [PO7, PO8, POz:  $t(18) > -3.32, p < .035$ ] (Figures 2 and 3). The final visual processing epoch we analyzed, 300 to 400 msec, only showed an effect of electrode [ $F(13,208) = 3.24, p = .042$ ], but no effect of condition ( $p = .154$ ) or an interaction between these two factors ( $p = .651$ ).

### Attention Reorientation

We investigated the idea that attention is reoriented following a mistake by comparing correct after error

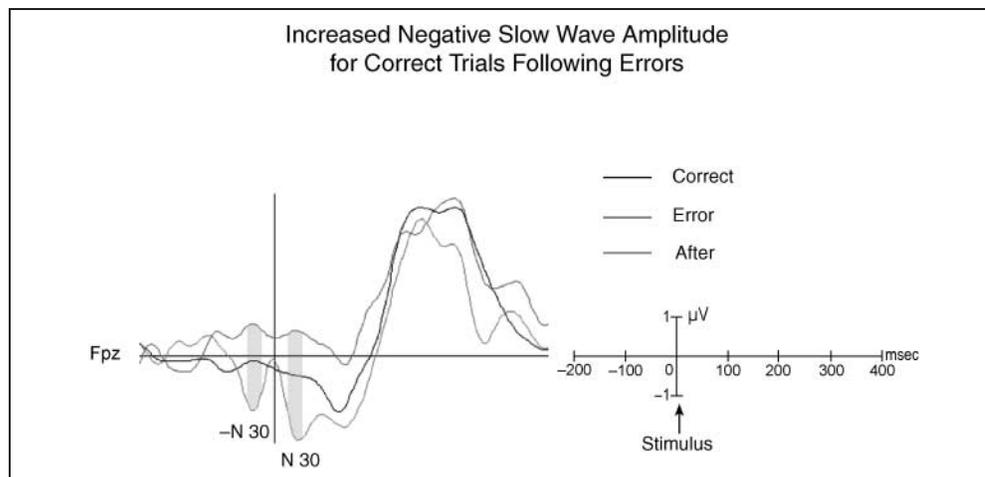
trials to both correct trials and error trials. After error trials showed two enhanced negative potentials most evident at Fpz when compared to correct and error trials. The first enhanced potential was observed *before* the stimulus presentation (frontal -N30), whereas the second enhanced potential was observed *after* the stimulus presentation (frontal N30).

The frontal -N30 peak was observed between -153 and -3 msec, and the average width of the peaks was 31 msec. After error trials were more negative in amplitude than both correct trials [Fpz:  $t(18) = -2.30, p = .033$ ] and error trials [Fpz:  $t(18) = -2.21, p = .041$ ]. The frontal N30 peak was observed between 15 and 140 msec, and the average width of the peaks was 23 msec. After error trials were more negative in amplitude than both correct trials [Fpz:  $t(18) = -4.03, p = .001$ ] and error trials [Fpz:  $t(18) = -5.47, p < .001$ ] (Figure 4).

### Correlations

The difference wave (correct minus error) at electrode FCz during the late CNV time epoch (0 and 0.1 sec) correlated with the difference wave at electrodes POz, PO7, and PO8 during the late CNV time epoch (0 and 0.1 sec) [FCz with POz: Pearson correlation = .690,  $p = .001, n = 19$ ; FCz with PO7: Pearson correlation = .580,  $p = .009, n = 19$ ; and FCz with PO8: Pearson correlation = .556,  $p = .013, n = 19$ ]. The difference wave at electrode FCz during the late CNV time epoch (0 and

**Figure 4.** Attention reorientation ERPs: Correct vs. Error vs. After Error. ERPs are stimulus-locked. The graph shows activity at electrode Fpz. The solid waveform represents correct trials, the dotted waveform represents error trials, and the dashed waveform represent after trials, correct trials following incorrect trials. Time intervals that are shaded show statistically significant differences between after error and correct trials as well as between after error and error trials. After error trials generate enhanced negative potentials compared to both correct and error trials that peak at  $-30$  msec prior to and  $30$  msec following stimulus presentation.



0.1 sec) correlated with the difference wave at electrodes POz and PO8 during the P1 time interval (0.11 to 0.14 sec) [FCz with POz: Pearson correlation = .485,  $p = .035$ ,  $n = 19$  and FCz with PO8: Pearson correlation = .564,  $p = .012$ ,  $N = 19$ ]. The difference wave at electrode FCz during the late CNV time epoch (0 and 0.1 sec) also correlated with the difference wave at electrodes POz and PO7 during the N1 time interval (0.165 to 0.195 sec) [FCz with POz: Pearson correlation = .604,  $p = .006$ ,  $n = 19$  and FCz with PO7: Pearson correlation = .456,  $p = .05$ ,  $n = 19$ ]. Similar to FCz, electrodes C3, C4, and Cz showed an almost identical pattern of significant correlations with POz, PO7, and PO8.

RTs for correct trials following an error were significantly increased compared to RTs for correct trials. This increase in RT is interpreted as representing additional processing associated with attention reorientation. We conducted a correlation analysis between these RTs and the N30. We found that the RT for the correct trials following an error correlated with the amplitude difference, between correct trials following an error and error trials, during the N30 time interval [Pearson correlation =  $-.50$ ,  $p = .03$ ,  $n = 19$ ]. This finding provides support for the significance of the attention reorientation ERP effects.

#### Onset of Correct versus Error Effect

In order to determine whether the frontal or extrastriate cortex came on-line first during the 100 msec prior to the stimulus delivery, we broke this time interval into ten 10-msec epochs. We conducted corrected paired  $t$  tests between correct and error trials at each of the 14 electrode locations (Fp1, Fp2, Fpz, F3, F4, Fz, FCz, C3, C4, Cz, CPz, PO7, PO8, POz) for each of the ten 10-msec

epochs. Electrode Cz showed an effect of condition as early as 90 msec before the stimulus presentation [Cz:  $t(18) = -2.44$ ,  $p = .025$ ]. Electrode POz showed the effect 30 msec following the activity in electrode Cz [POz:  $t(18) = -2.15$ ,  $p = .045$ ] (Figure 2).

#### Conflict

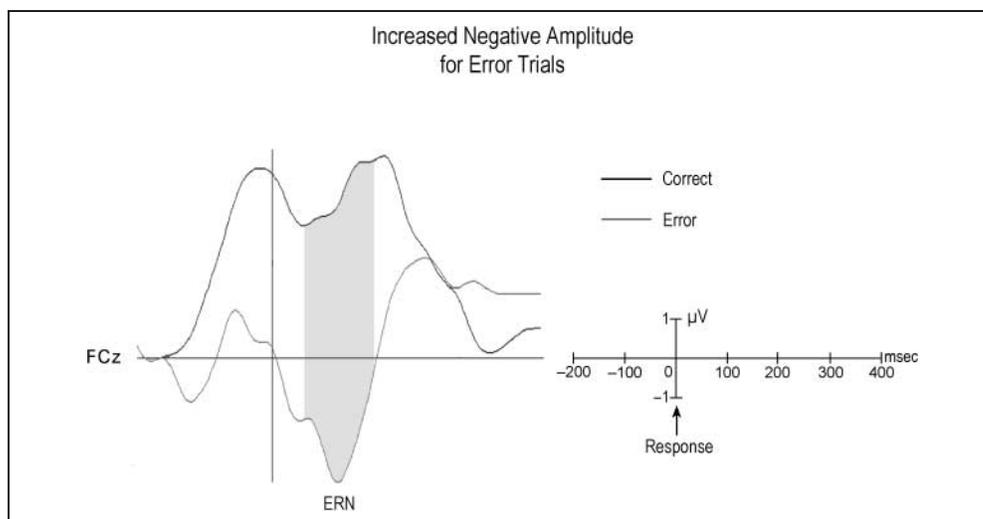
To examine whether conflict influenced preparatory potentials, we analyzed high versus low conflict for both correct and error trials. The analysis was conducted on 14 electrodes (Fp1, Fp2, Fpz, F3, F4, Fz, FCz, C3, C4, Cz, CPz, PO7, PO8, POz) during the prestimulus ( $-0.1$  to 0 sec) and then poststimulus (0 to 0.1 sec) epochs. During the prestimulus epoch, correct trials showed an effect of electrode [ $F(13,208) = 10.29$ ,  $p < .001$ ] but no effect of condition ( $p = .517$ ), and error trials showed no effect of either factor ( $p < .221$ ). Similarly, for the poststimulus time epoch, we found an effect of electrode [ $F(13,208) = 11.08$ ,  $p < .001$ ] but no effect of condition ( $p = .227$ ) for correct trials and no effect of either factor for error trials ( $p < .150$ ).

#### Response-locked Potentials

##### Error Processing and Monitoring

An initial Omnibus test showed statistical differences between the response-locked potentials for the four trial types at FCz between the time epoch 50 and 150 msec following the response [FCz:  $F(3,54) = 10.01$ ,  $p = .001$ ]. Follow-up planned comparisons revealed that error trials generated a classic error-related negativity (ERN) that was not evident in correct trials [FCz:  $t(18) = 3.898$ ,  $p = .001$ ] (Figure 5).

**Figure 5.** Action monitoring ERPs: Correct vs. Error. ERPs are response-locked. Graph shows activity at electrode FCz. The solid waveform represents correct trials and the dotted waveform represents error trials. Error trials are characterized by a negative peak at frontal–central sites between 50 and 150 msec following the response that is absent for correct trials.



We examined the hypothesis that errors are the result of a temporary monitoring dysfunction manifest in the trial preceding the error by comparing the response-locked potentials for correct trials to before error trials. There was no statistical difference between these two conditions between 50 and 150 msec following the response (ERN/error preceding positivity [EPP] latency range) [FCz:  $t(18) = -1.230, p = .235$ ].

## DISCUSSION

In this study, we evaluated whether lapses in preparatory attention prior to stimulus delivery predicted response performance in a visual letter discrimination task. We used EEG to observe changes in slow potentials during preparatory attention. We found that errors were linked to an amplitude decrease in the fronto-central CNV prior to stimulus delivery accompanied by a decreased sustained negative shift in extrastriate sites to the imperative stimulus. CNV activity at fronto-central electrodes correlated with the sustained negativity observed in extrastriate electrode sites. Further, ERP and behavioral data for correct trials following an error suggest attention is reoriented following a mistake. These findings provide evidence that transient lapses in a frontal–extrastriate preparatory attention network predict response errors in some conditions.

Our task required subjects to press, with the right or left index finger, one of two buttons. The letter S was mapped onto one button and the letter H onto the other button. The letters were colored either red or green and the cue preceding the stimulus (the word RED or GREEN) instructed which of the two letters was considered the target. Sometimes the target letter appeared on the same side as the button press (low conflict) and sometimes the target letter appeared on the opposite side of the button press (high conflict).

The aim of our study was to further characterize the neural bases of mistakes. Studies using behavioral data propose that errors are the result of temporary lapses in executive control processes. Preparatory attention is an executive control process that exerts control early in the sensory processing stream. The CNV is an index of preparatory attention (Douros et al., 1987; Tecce, 1972). Executive control processes have been localized to a dorsal fronto-parietal network (Corbetta & Shulman, 2002; Miller & Cohen, 2001; Fuster, 2000; Mesulam, 1998). The topographical distribution of the CNV is consistent with this dorsal fronto-parietal network. Traditionally, this network is engaged by cues that carry predictive information and which create an attentional set. The attentional set is described as selective sustained delay period activity. The delay period activity guides behavior in a goal-directed manner; it helps associate sensory information to motor plans, directs spatial attention, enhances task-relevant features, and holds information on-line. Increased baseline firing observed when a monkey anticipates a stimulus is similar to the increased BOLD response in humans during stimulus anticipation. Furthermore, in a recent simultaneous EEG/fMRI study, the trial by trial CNV amplitude correlated with BOLD activity increases in the anterior cingulate, the supplemental motor area, and the thalamus, all areas known to contribute to the CNV network (Nagai et al., 2004). Therefore, we reasoned that if lapses in executive control resulted in response errors, then these would be evident in the CNV. More specifically, we hypothesized that errors would be accompanied by decreased CNV amplitude.

In accord with our hypothesis, we found that correct trials generated a large negative slow wave potential (CNV) in fronto-central sites that was substantially reduced on error trials. This activity preceded the stimulus by about 100 msec and was maintained for approximately 100 msec following the stimulus. Initially, the difference

in CNV activity between correct and error trials was focused in frontal–central scalp sites; however, within 30 msec of generation, the effect was evident in the lateral extrastriate cortex. The time delay between frontal and extrastriate sites is consistent with the conduction velocity of cortico-cortical association fibers such as the superior and inferior longitudinal fasciculi (Matsumoto et al., 2004). In this view, cortico-cortical connections may provide preparatory input to task-relevant areas.

In order to address whether visual processing was influenced by PFCx preparatory activity, we evaluated the slow potential at extrastriate electrode sites. We found that error trials were also less negative than correct trials at extrastriate locations. Furthermore, we conducted an amplitude correlation analysis to explore the dynamics between the PFCx and the extrastriate cortex. The data showed that the CNV activity in frontal–central sites correlated with the CNV activity in extrastriate sites. The CNV activity at fronto-central sites also correlated with early stimulus processing activity in the extrastriate cortex.

Together our data indicate that decreased fronto-central slow wave activity reduces sensory processing in the extrastriate cortex, resulting in incorrect behavioral responses. This conclusion is consistent with evidence from single cell recordings in macaques (Desimone, 1996), electrophysiological lesion studies in patients (Yago et al., 2004; Barcelo et al., 2000), and blood flow data in humans (Corbetta & Shulman, 2002), which conclude that the PFCx exerts control of visual attention by modulating the excitability of extrastriate neurons along a goal-directed path, thereby biasing responses. Further support for this type of “top-down” regulation of the extrastriate cortex comes from Fuster (1990), who showed that extrastriate neurons show less selective delay activity when the PFCx is deactivated. In addition, lesions in the human lateral PFCx produce reduced P1 amplitudes for both attended and nonattended stimuli and simultaneously abolish the SN potential leading to performance errors in the field contralateral to the PFCx lesion (Yago et al., 2004). PFCx-mediated slow potentials are linked to a neural mechanism for one aspect of top-down preparatory control: the generation and proliferation of selective delay activity (the context-dependent representation of the S–R association) that primes relevant cortical areas in anticipation of a predicted sensory event.

Although our study offers strong support for the view that errors, in some instances, are due to lapses in preparatory attention, other explanations have been proposed. A recent study of the action monitoring system revealed the presence of a response-locked potential known as the error preceding positivity (EPP). This potential was only evident in correct trials preceding an error. Because no EPP was found in other correct trial types and the EPP was followed by errors, this led researchers to conclude that the EPP was the result of temporary dysfunction in

the action monitoring system. These researchers further concluded that errors were due to inadequate selection of actions due to a disruption in the ability to effectively monitor the previous action. In our study, we found no difference in RTs or response-locked potentials between correct trials preceding the error (EPP) and all other correct trials. Variations in paradigms resulted in different RT latencies that may have contributed to the different results. Although our data did not replicate the EPP effect, it is important to note that we were able to replicate the well-established ERN. The ERN is a response-locked action monitoring potential that is thought to represent activity related to the evaluation of the response strategy (Bartholow et al., 2005). The robust ERN to error trials and absence of the EPP indicates that the response monitoring system was not compromised in our subjects.

Behavioral studies and computational models have used stimulus and/or response conflict to account for response accuracy. In general, conflict can be described as a measure of uncertainty regarding a course of action. Any event that causes ambiguity in a task increases conflict and the likelihood of making an incorrect decision. Cognitive tasks manipulate conflict by making the target stimuli or response hard to identify as a result of degrading target stimuli, adding distracting stimuli, or matching irrelevant and relevant stimuli and/or response features. These studies propose that errors are the result of unresolved ambiguity. They suggest that the system does not have enough time or resources to fully process all of the stimulus or response features that would otherwise differentiate the target from irrelevant stimuli. Alternatively, they reason that “noise” in the system does not allow differentiation between two representations that are similarly active or that this “noise” engages the selection mechanism early. From this perspective, errors are contingent on task properties related to conflict and not necessarily on subject-based properties such as lapses of preparatory attention.

Although our study was not specifically designed to address this issue, we tested the idea that conflict might modulate preparatory attention potentials. We found no statistical differences between high and low conflict preparatory potentials for either correct or error trials. This finding provides support for the idea that task-related properties such as conflict do not affect early prestimulus preparatory processes. Instead, these seem to influence later downstream sensory processes such as stimulus or response discrimination (Donkers & van Boxtel, 2004). This may be the reason why excess conflict does not always result in an incorrect response. Presumably, we are able to employ strategies that resolve or minimize task-related ambiguity in order to respond more effectively. In this framework, task-related properties increase the probability of committing errors (increase the need for resources) but do not necessarily result in response errors.

Another proposal is that errors are the result of hasty responses. These studies find that error trials have significantly reduced RTs compared to correct trials. These decreased RTs are usually interpreted as representing early response selection before complete stimulus processing (i.e., hasty motor reactions) (Schouten & Bekker, 1967). In contrast, our behavioral analysis revealed that error trials in our task had increased RTs compared to correct trials. RTs are known to depend on the amount of stimulus–response conflict. The letter discrimination task we used intentionally increased conflict by adding an incongruent condition in which the stimulus was presented in one hemifield while the correct response was associated with the contralateral side. Consequently, our task has an increased level of conflict compared to most discrimination tasks. One possibility is that RTs in our task might have been prolonged in order to resolve the increased conflict. However, as we noted previously, conflict does not seem to influence preparatory potentials because we found no statistical difference between the amplitude of high and low conflict trials. Conflict simply seems to increase the need for resources but does not necessarily determine response accuracy. What does determine response performance in our study is the successful employment of attentional resources. Furthermore, short latency incorrect responses have also been related to disrupted slow potentials. Gratton et al. (1988) found that the variability of the RP, a slow wave that is related specifically to motor preparation, predicted RTs and response accuracy in a letter discrimination task. Thus, it appears that our proposal that preparatory attention slow potentials determine response performance extends to both short and long RT responses.

The current findings reveal that errors are not necessarily the result of action monitoring dysfunction, failure in conflict resolution, or reflexive responses. Alternatively, our data support the premise that errors can be the result of temporary disruption of preparatory attention processes. In this framework, preparatory attention functions via PFCx-mediated slow potentials that modulate the extrastriate cortex in a top-down manner. These slow potentials prime task-relevant cortical areas and influence information processing by setting activation thresholds in anticipation of a predicted event. Exciting the apical dendrites of relevant cortical pyramidal cells near activation thresholds allows these cortical neurons to respond to the anticipated event more efficiently. This predictive activity is then compared to the sensory activity generated by the stimulus in order to either confirm or reject the prediction. In this manner, decreased PFCx slow negative potentials lead to deficient sensory processing, which compromises the key influence of sensory information on response selection leading to errors.

As a final point, we predicted that if a lapse in preparatory attention was found, then we might see evidence of attention reorientation following the mistake.

An inferior frontal–temporal–parietal attention network is known to orient attention to relevant stimuli that are outside the current focus (Corbetta & Shulman, 2002). When unexpected stimulus features and low-frequency stimuli are presented, the network becomes active. This attention network is referred to as a “circuit breaker” because it is also responsible for terminating the current task set. This inferior frontal network alerts the dorsal network when a behaviorally relevant, but unexpected, stimulus is detected and then if appropriate terminates the current attention set.

To determine whether attention was reoriented following a mistake, we compared correct, incorrect, and correct trials following an error at the frontal polar region. Our analysis revealed increased RTs as well as increased potentials (–N30 and N30) for correct trials following an incorrect response. The increased RT following an incorrect response is thought to be due to additional processing associated with reorienting attention. In line with this interpretation is the fact that we found that RTs for correct trial following a mistake were correlated with the amplitude difference between the N30 for after error trials and error trials. In addition, the topographic distribution of our potentials suggests the involvement of inferior frontal areas such as those involved in the “circuit breaker” network. This inferior frontal network may help reorient attention by detecting a salient and unexpected event, in our case, the unexpected/salient event is the occurrence of a task-relevant stimulus as we assume that the subject’s attention has been captured by processing associated with the previous response, the error, or other non-task-related information. The temporal pattern of the reorientation potentials further reinforces this idea. The –N30 reaches its peak before stimulus presentation and then returns to baseline when the stimulus is presented. Following the return to baseline, possibly a resetting of the attentional state, a new potential, the N30, is formed. Therefore, we interpret these increased frontal polar potentials as evidence of attention reorientation following a mistake. Caution is warranted in neuroanatomical interpretation of these fronto-polar effects because, unlike the CNV, there is no information on the neural sources of these reorientation potentials other than their currently reported scalp topography.

Although we have identified temporary lapses in preparatory attention as the basis of response errors in our task, we do not know what causes these lapses. Examination of the cholinergic system, due to its critical role in the production and modulation of slow potentials, is a logical place to begin addressing this question. This system is involved in both tonic and transient processes, which independently interact with the preparatory attention network (Sarter, Bruno, & Givens, 2003). Some of these processes are in turn dependent on the dopaminergic system (Atzori, Kanold, Pineda, & Flores-Hernandez, 2003). The dopaminergic system is

also critical due to its role in reward prediction. Furthermore, acetylcholine acts differentially on the superficial and deeper layers of the cortex, which are associated with cortico-cortical and thalamo-cortical networks, respectively, and thus, can affect how much intrinsic versus extrinsic information influences behavior (Hasselmo & Bower, 1993). The CNV is also associated with thalamic and basal ganglia relays. These nuclei are able to control which “information specific processing gates” are opened and which remain closed (Kimura, Minamimoto, Matsumoto, & Hori, 2004; Kimura et al., 2003). Therefore, the involvement of the cholinergic system in slow potentials provides a possible mechanism for the dynamic interaction between top-down and bottom-up processes. Usually, these systems interact in a synergistic fashion, but one might speculate that disrupted integration of these systems may negatively affect network properties and cause transient dysfunction in behavior. Studies using cholinesterase inhibitors combined with ERP and behavioral measures might help to clarify these speculations.

In conclusion, the current data reveal why errors are committed in certain discrimination tasks. This study supports the idea that we make mistakes due to transient lapses in preparatory attention and that attention is reoriented immediately following a response error. Our data support a model in which the PFCx primes task-relevant cortical areas via slow preparatory attention potentials, in anticipation of an event, in order to increase the effectiveness of information processing. When this system is disturbed, sensory information processing and behavior are impaired.

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