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Event-related potentials elicited by errors during the stop-signal task. I: Macaque monkeys

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Abstract

The error-related negativity (ERN) and positivity (Pe) are components of event-related potential (ERP) waveforms recorded from humans that are thought to reflect performance monitoring. Error-related signals have also been found in single-neuron responses and local-field potentials recorded in supplementary eye field and anterior cingulate cortex of macaque monkeys. However, the homology of these neural signals across species remains controversial. Here, we show that monkeys exhibit ERN and Pe components when they commit errors during a saccadic stop-signal task. The voltage distributions and current densities of these components were similar to those found in humans performing the same task. Subsequent analyses show that neither stimulus- nor response-related artifacts accounted for the error-ERPs. This demonstration of macaque homologues of the ERN and Pe forms a keystone in the bridge linking human and nonhuman primate studies on the neural basis of performance monitoring.

Introduction

To thrive, organisms must detect when their responses fail to meet expectations through *performance monitoring*. Researchers investigating performance monitoring in humans have made inferences based on event-related potentials (ERPs) or neuroimaging methods, whereas investigators using monkeys have relied on intracranial recordings (reviewed by Paus, 2001; Ridderinkhof et al., 2004; Schall and Boucher, 2007; Taylor et al., 2007; Hikosaka and Isoda, 2010; Passingham et al., 2010). The present work addresses this fundamental question: Is the monkey executive control system a valid model of human performance monitoring?

The first electrophysiological correlate of performance monitoring discovered in humans, the error-related negativity (ERN, also known as the Ne), was independently reported by Falkenstein et al. (1990) and Gehring et al. (1993). The ERN has a frontocentral scalp distribution and peaks ~50–100 ms following incorrect manual responses (reviewed by Gehring et al., 2011). Several groups have observed the ERN during the stop-signal task (also known as the countermanding task), which is used to investigate behavioral inhibition and executive control (Endrass et al., 2005; Liotti et al., 2005; van Boxtel et al., 2005; Kramer et al., 2007; Stahl and Gibbons, 2007; Vocat et al., 2008). Although the ERN is clearly associated with error commission, a variety of hypotheses concerning its relation to

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cognitive processes have been proposed (e.g. Gehring et al., 1993; Falkenstein et al., 2000; Holroyd and Coles, 2002; Luu et al., 2003; Yeung et al., 2004; Brown and Braver, 2005). A number of these theories make specific predictions concerning the anatomical, neurophysiological, and neurochemical mechanisms of the ERN. However, these theories have proven difficult to distinguish using behavioral and imaging data from humans. Animal models of error-ERPs can provide leverage to distinguish between alternative hypotheses of performance monitoring.

However, some have proposed that macaque monkeys do not have the neural substrates necessary to generate performance monitoring ERPs similar to those observed in humans (Cole et al., 2009; 2010; but see Schall and Emeric, 2010). The argument is based on cytoarchitectural differences in medial frontal cortex between species, as well as perceived differences in the signals observed in human and monkey medial frontal cortex. The presence or absence of an ERN in monkeys would therefore shed light on an important, unresolved issue.

We recorded ERPs from monkeys while they performed the saccade stop-signal task. On trials without stop-signals (no-stop trials), monkeys made saccades to peripheral targets. These correct responses were rewarded. On trials containing stop-signals (stop trials), monkeys often made saccades to targets. These errant responses were not rewarded. Thus, saccades led to either correct responses or errors. By contrasting response aligned ERPs from these two trial types, we demonstrate the first evidence of error-ERPs in nonhuman primates.

Materials and Methods

Animal care

Data were collected from one male bonnet macaque (*Macaca radiata* ~8.5 kg) and one female rhesus macaque (*Macaca mulatta* ~7 kg). Both animals were cared for in accordance with policies set forth by the USDA and Public Health Service Policy on Humane Care and Use of Laboratory Animals and all procedures were carried out with supervision and approval from the Vanderbilt Institutional Animal Care and Use Committee.

Surgical details have been described (Godlove et al., 2011). Most critically, solid gold surface electrodes, Teflon coated stainless steel wires, and plastic connectors were constructed and implanted following the method of Woodman et al. (2007). Implanted electrode locations are provided in Table 1.

Stimuli and task

Stimulus presentation, task contingencies related to eye position, and delivery of liquid reinforcement were all under computer control in real time (TEMPO, Reflective Computing, Olympia, WA). Behavior and electrophysiological signals were recorded during the saccadic stop-signal (countermanding) task (Figure 1). Stimulus properties and task timing have been reported in detail (Godlove et al., 2011). Additional details about the behavioral training regime and task have also been described (Hanes and Schall, 1995; Hanes et al., 1998).

Trials were initiated when monkeys fixated a centrally presented square. After a variable time, the central fixation point was extinguished and a target simultaneously appeared at 10° to the left or right of fixation. On no-stop trials (Figure 1 top), no further visual stimuli were presented. Monkeys were required to make saccades to targets and hold gaze for 600 ms to obtain reward. On stop trials (Figure 1 bottom), the fixation point was re-illuminated after a variable delay providing a *stop-signal*. To obtain reward on stop trials, monkeys withheld eye movements and maintained fixation for a minimum of 1800 ms. These trials were

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designated as *canceled*. If monkeys were unable to inhibit the movement, a 1500 ms timeout was added to the normal inter-trial interval of 200 ms, no rewards were given, and the trial was termed *noncanceled*. Thus, identical responses could be either correct or errant depending on trial context.

An initial set of stop-signal delays (SSDs) from 0 to 420 ms and separated by either 40 or 60 ms steps was selected for each recording session. We then manipulated SSD using an adaptive, stair-casing algorithm, which adjusted stopping difficulty based on performance. Stop trials made up 30 to 40% of all trials in a given session with a typical session consisting of several thousand trials. Saccade initiation and termination were detected offline using a custom algorithm which first detected instantaneous velocity elevated above 30°/s and then calculated the beginning and ending of the monotonic change in eye position. We adopted the procedures of Logan and Cowan (1984) implemented by Hanes et al. (1998) to estimate stop signal reaction time (SSRT). In brief, we estimated SSRT using one method which assumes that SSRT is a constant, and another method which assumes that SSRT is a random variable. Since there is no reason to assume an advantage of either of these methods, we averaged the two estimates together to obtain final SSRT measures.

Data acquisition

Eye position was monitored using an infrared eye-tracking system (ASL, Bedford, MA). Implanted EEG surface electrodes were referenced to linked ears using ear-clip electrodes (Electro-Cap International, Eaton, OH). All electrode impedances were less than 10 kΩ. The EEG from each electrode was amplified with a high-input impedance head stage (Plexon, Dallas, TX) and bandpass filtered between 0.7 and 170 Hz.

ERP analyses

ERPs were time-locked to saccade initiation or stop-signal onset. Waveforms were baseline corrected during the interval from 150 ms to 50 ms before these events. Stop trials on which subjects responded before stop-signal presentation (37% monkey F, 49% monkey Y) were not included in error-ERPs since subjects did not have the necessary information to deduce that an error had been committed at time of response. When constructing grand averages collapsed across left and right target locations, the number of trials presented at each location was matched in a given condition by excluding random trials from one target (26% monkey F, 18% monkey Y). Trials with voltage deflections greater than ±300 μV and trials with amplifier saturation were also excluded from analysis (3% monkey F, 1% monkey Y). Single trial EEG signals were truncated 50 ms before the onset of the second, non-task-related saccade to eliminate artifacts arising from temporally smeared second-saccade activity (Godlove, 2010).

Significant ERP differences were assessed using the method of Emeric et al. (2008). This method tests for differences between error and correct ERPs using a thresholding approach similar to those often employed in single unit studies measuring activity onsets in spike-density functions. First, a difference wave was calculated by subtracting noncanceled error-ERPs from no-stop correct ERPs. Negative difference wave values indicated that error-ERPs were more negative than correct ERPs, while positive difference wave values indicated an opposite polarity effect. Difference wave values near zero indicated that error and correct ERPs did not differ. Thus, to test for significant differences between error and correct ERPs, we simply observed periods when the difference wave deviated from zero (i.e. baseline) by values larger than those expected by chance.

The intrinsic variability of the difference wave was assessed by calculating the standard deviation across time during the baseline period. This provided a measure of chance

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fluctuations between error and correct ERPs. Significant epochs were defined as periods when the difference wave deviated from baseline by >2 standard deviations for longer than 50 ms, provided it exceeded 3 standard deviations in that interval. For presentation, the grand average ERP collapsed across both monkeys was digitally filtered with a zero phase shift 35 Hz low-pass hamming window (*sd* = 6 ms). Unfiltered ERPs are presented individually for each monkey, and all statistical analyses were carried out on the unfiltered data.

Current density estimation

MRIs were acquired with a Philips Intera Achieva 3 Tesla scanner using SENSE Flex-S surface coils placed above and below the head. T1-weighted gradient-echo structural images were obtained with a 3D turbo field echo anatomical sequence (TR = 8.729 ms; 130 slices, 0.70 mm thickness). Segmentations of skin, skull, and brain were carried out in CURRY 6 (Compumedics Neuroscan, Singen, Germany). The cranial surface electrode locations were co-registered to the head model guided by stereotaxic coordinates recorded during surgery. From this 3D head model, a three-compartment Boundary Element Method (BEM) volume conductor geometry was generated.

Source estimation used ERP difference waves (noncanceled error minus no-stop correct) at time windows of ±30 ms (ERN) and ±40 ms (Pe) centered on the peak amplitude of the difference wave from electrodes showing maximal ERN and Pe. Current density was estimated using the sLORETA-Weighted Accurate Minimum Norm method (SWARM) (Wagner et al., 2007). SWARM combines the methods of diagonally weighted Minimum Norm Least Squares (MNLS) (Dale and Sereno, 1993) and sLORETA (Pascual-Marqui, 2002) to compute a current density vector field with low localization error (see Wagner et al., 2007).

Tests for behavioral adjustments

For all analyses of behavioral adjustments related to error-ERPs, data were collected from electrodes that displayed maximum error-related amplitude differences, and data were drawn from the same windows used in current density analysis. We used two methods to test for relationships between error-ERPs and post-error RT adjustments. The first method relied on single trial amplitude measures. We identified errant noncanceled trials (trial n) which were followed by no-stop trials (trial n+1). We measured the maximum negative and positive deflections during ERN and Pe windows on trial n, and then determined post-error RT adjustments defined as ΔRT (RT on trial n+1 minus RT on trial n). We measured the correlation coefficient (ρ) values for maximum ERN/Pe amplitude versus ΔRT and subjected these distributions of ρ values to 1 sample t-tests. This allowed us to determine if correlations tended to deviate from zero across the entire data set. For our second method, we first calculated median ΔRT separately for each monkey. We then constructed 2 ERPs aligned to the errant saccade on trial n based on a median split of ΔRT and tested for significant differences using 2 sample t-tests. To account for the effects of non-stationarity on RT estimates we repeated both of these analyses using the correction suggested by Nelson et al. (2010). For this correction, ΔRT was calculated as RT on trial n+1 minus RT on trial n−1. Since similar findings were obtained using both ΔRT measures, only data from the first ΔRT analyses are reported.

Tests for conflict

We also tested for relationships between the amplitude of the ERP negativity around SSRT and neural response conflict. We first normalized the raw EEG traces by z-scoring them to remove incidental inter-subject and inter-electrode amplitude differences. We then identified successfully canceled trials at each SSD. According to findings from Hanes et al. (1998) and

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Paré and Hanes (2003) canceled trials are those containing the largest magnitude of neural response conflict in the saccadic stop-signal task (see also Stuphorn et al., 2000). We identified no-stop trials from each session with RTs > SSD + SSRT. These latency-matched trials are those which were slow enough to have been successfully canceled had stop-signals been presented, providing an appropriate control for canceled trials (e.g. Hanes et al., 1998; Godlove et al., 2011). We then constructed ERPs from canceled and no-stop trials at each SSD and measured mean amplitude on canceled trials and latency matched no-stop trials in the window −50 to +100 ms around SSRT. This window corresponds to the time of conflict-related neural modulation in the supplementary eye field (SEF)(Stuphorn et al., 2000; Emeric et al., 2010). By subtracting mean no-stop voltage from mean canceled voltage we obtained measurements of the amplitude of the canceled-trial negativity. Finally, we tested to see if the amplitude of canceled-trial negativity was related to response conflict by assessing its correlation with SSD and the probability of committing an errant saccade.

Results

Behavior

Behavioral results are summarized for each monkey in Table 2. Both animals exhibited noncanceled trials with probability slightly > 50%. Because we used a stair-casing algorithm to adjust SSD, this departure suggests that both animals tended to speed up, causing a reduction in SSD. We have observed and reported this pattern of behavior before in animals performing the saccadic stop-signal task (Godlove et al., 2009, 2011). Other than a small tendency to speed responses, the data summarized in Table 2 suggest that the monkeys performed the task in a manner consistent with the race model of Logan and Cowan (1984).

Grand average error-ERPs

Figure 2 shows the saccade-aligned ERPs at electrode Fz collapsed across sessions and monkeys. In both instances, monkeys made saccades to target locations, but different contexts rendered no-stop trial responses correct and stop trial responses errant. In comparison to the correct no-stop ERPs, the errant noncanceled ERPs show a negativity beginning approximately 8 ms after the error and ending 73 ms later. This ERN reverses from 150–215 ms post response, becoming an error-related positivity (Pe). Taking into consideration known neural conduction velocity differences between human and macaque nervous systems (Woodman, 2011), the timing of these potentials show a tight correspondence to the time courses of the ERN and Pe observed in humans (Reinhart et al., 2011).

Individual monkey error-ERPs

Figures 3 and 4 show that a similar pattern of ERN and Pe components was apparent in the frontal medial electrodes of each monkey. In monkey F, the observed ERN was maximal at electrode Fz, 97 ms after the response, and the Pe was maximal at electrodes F1 and F2 at 170 ms and 176 ms after the errant saccade, respectively. Monkey Y was implanted with a denser electrode array. This monkey showed a maximal ERN deflection at electrode FCz, 64 ms after the saccade, and a maximal Pe deflection at electrode Fz, 188 ms after the saccade.

Current density model

The dense electrode array of monkey Y, enabled us to investigate the spatial distribution of error-related components in more detail. We calculated current density distributions with the SWARM algorithm using the anatomical MRI from monkey Y. This method takes into account individual skull and brain morphology. Figure 5 shows the results. The current distribution contributing to the ERN explains 84% of the variance; while that contributing to

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the Pe explains 86% of the variance. The current density maps show that the ERN has a broad frontocentral distribution while the Pe has a more focal frontal distribution. The distribution of current sources extended onto medial frontal cortex for both the ERN and the Pe (Figure 6). These results are strikingly similar those obtained from humans performing the same task (Reinhart et al., 2011).

Because the stop-signal was only presented on trials in which errors were committed, our ERN and Pe results include a contribution from visually evoked ERPs elicited by the stop-signal. We addressed the contributions of this potential confound by comparing current density distributions of canceled and noncanceled trial ERPs during the same time periods relative to the stop-signal (Figure 5C). On these trials, the monkey held gaze at central fixation for at least 1800 ms following stop-signal presentation. Therefore, the current distribution on canceled trials reveals the contribution of stimulus-related ERPs to the error-related ERPs. No significant current densities were observed over medial frontal cortex during the ERN epoch when saccades were correctly canceled. Instead, significant effects were restricted to occipital and parietal regions. These results show that the ERN and Pe we observed are not sensory artifacts associated with stop-signal presentation.

Error-ERPs, RT adjustments, and response conflict

Some studies have suggested that error-ERP magnitudes are correlated with performance adjustments such as post-error slowing (Gehring et al., 1993; Nieuwenhuis et al., 2001; Rodriguez-Fornells et al., 2002; Kerns et al., 2004; Debener et al., 2005; Holroyd et al., 2005; Klein et al., 2007; Ladouceur et al., 2007; West and Travers, 2008; Huster et al., 2011; but see Gehring and Fencsik, 2001; Hajcak et al., 2003; Nunez Castellar et al., 2010). To test for these effects and relate our ERP data to intracranial local-field potentials recorded in the SEF and anterior cingulate cortex (ACC) during this task (Emeric et al., 2008; 2010), we examined the relationship between error-related ERPs and post-error RT adjustments, as described above. Figure 7A illustrates this analysis for a sample session. Neither ERN amplitude nor Pe amplitude were significantly correlated with ΔRT in this session. Figure 7B and 7C display distributions of ρ values collapsed across all sessions. Neither of these distributions deviated significantly from zero (ERN $t_{(14)} = 0.68$, $p = 0.51$; Pe $t_{(14)} = -1.67$, $p = 0.12$).

Because raw EEG contains a great deal of variability, we also averaged the ERPs using a median split based on ΔRT and measured amplitudes during the windows centered on the peak ERN and Pe. Figure 8A displays mean ERN amplitude for the fastest and slowest ΔRT trials separately for each monkey and averaged across both monkeys. Figure 8B displays the same relationship between Pe amplitude and ΔRT. Neither monkey showed significant differences in ERN amplitude (monkey F $t_{(18)} = -0.18$, $p = 0.86$; monkey Y $t_{(8)} = -1.17$, $p = 0.28$; grand $t_{(28)} = -0.71$, $p = 0.48$) or in Pe amplitude as a function of ΔRT (monkey F $t_{(18)} = -0.06$, $p = 0.96$; monkey Y $t_{(8)} = -1.47$, $p = 0.18$; grand $t_{(28)} = -1.07$, $p = 0.29$).

An influential theory posits that the ERN is produced by neural processing of response conflict (Botvinick et al., 2001; Yeung et al., 2004). The occurrence of response conflict is not restricted to error trials only, but is hypothesized to occur with varying timing and magnitude on all trial types (Yeung et al., 2004). In the stop-signal task, subjects must choose between committing responses and canceling them. Thus, in the saccadic stop-signal context, response conflict is engendered when subjects must choose between producing saccades and maintaining fixation. Distinct neural populations are active in the superior colliculus (SC) and frontal eye field (FEF) when saccades are produced or fixation is maintained. We and others have classified these neurons as *movement cells* and *fixation cells* (Bruce and Goldberg, 1985; Munoz and Wurtz, 1993; Hanes et al., 1998), although alternate classifications have been proposed (Hafed et al., 2009). Movement and fixation cells in the

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SC (Paré and Hanes, 2003) and FEF (Hanes et al., 1998) are briefly coactive on canceled trials around the time that saccade cancelation occurs (SSRT). This coactivation is largest when saccades are successfully canceled under conditions in which they are likely to occur. Similar coactivation of movement and fixation cells has not been observed on errant noncanceled trials. In sum, the probability of successfully canceling action which varies as a function of SSD yields a reliable proxy measurement of neural response conflict on canceled trials in the saccadic stop-signal task. Using this metric, it has been shown that response conflict may be reflected in single cell and LFP signals of SEF (Stuphorn et al., 2000; Emeric et al., 2010). But this conflict signal has not been observed in the single cell and LFP responses of ACC (Ito et al., 2003; Emeric et al., 2008).

We tested for conflict-related activity in ERPs aligned to SSRT on canceled trials using the method of Stuphorn et al. (2000). The mean voltage differences between canceled and no-stop trials in the −50 ms to +100 ms time window around SSRT at each SSD are plotted in Figure 9 as a function of both SSD and the probability of failing to cancel. These voltage differences did not show significant correlations with either SSD ($\rho_{(35)} = -0.08$, $p = 0.63$) or the probability of committing errant noncanceled responses ($\rho_{(35)} = 0.27$, $p = 0.11$).

Control for saccade related artifacts

To ensure that the observed error-ERPs were not due to response-related components, we quantified saccade dynamics on no-stop and noncanceled trials. Because we time-locked our ERPs to response onset, we could rule out the confounding effects of RT differences between trial types. However, if the task-related saccade amplitude or duration differed between correct and errant saccades it could lead to differences in the electromyogram or the corneoretinal potential between trial types (Luck, 2005; Godlove et al., 2011) and these artifacts could be interpreted as performance monitoring ERPs (Godlove, 2010).

Figure 10 summarizes saccade amplitude and duration separated by monkey, target, and trial type. We carried out 3-way ANOVAs to test the hypotheses that saccade amplitude, velocity, or duration differed between monkey, target, or trial type. Saccade velocity and duration both differed significantly between monkeys. Monkey F made saccades with higher peak velocity ($F_{(1,52)} = 12.37$, $p < 0.001$), and longer duration ($F_{(1,52)} = 5.22$, $p < 0.05$) than monkey Y. This means that monkey F also tended to make slightly larger amplitude saccades although this comparison did not reach statistical significance. Saccade dynamics differed modestly between targets for both monkeys. When monkeys made saccades to the rightward target, they tended to be of larger amplitude ($F_{(1,52)} = 27.36$, $p < 0.001$), higher peak velocity ($F_{(1,52)} = 71.19$, $p < 0.001$), and shorter duration ($F_{(1,52)} = 28.66$, $p < 0.001$). This may be an artifact induced by the monocular eye tracking procedures we employed. Because we only tracked the right eye of each monkey, saccade traces to the right target reflected abduction of the tracked eye while saccade traces to the left target reflected adduction of the tracked eye. To ensure that target bias did not affect ERP component analysis, approximately equal numbers of trials were included in each ERP for each trial type (see Methods). A marginally significant effect was noted for saccade velocity between correct and errant trials. Both monkeys tended to make higher velocity saccades on error trials than correct trials ($F_{(1,52)} = 4.17$, $p = 0.05$). However, velocity effects could not explain the different ERPs observed on error and correct trials unless saccadic endpoints also differed, shifting the corneoretinal potential to a greater degree on one type of trial relative to the other, or saccade duration differed smearing temporal saccade artifacts in one condition more than another. Critically, neither saccade amplitude ($F_{(1,52)} = 1.62$, $p = 0.2$) nor saccade duration ($F_{(1,52)} = 1.17$, $p = 0.3$) differed significantly between correct and errant trials. Therefore, no difference in correct and error saccade dynamics could explain the error-ERP effects.

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Discussion

We have shown that during a saccadic stop-signal task macaques exhibit ERN and Pe components homologous to those recorded from humans. The precise timing and distributions of these error-related ERPs might initially appear to differ from those reported in humans using manual responses (reviewed by Gehring et al., 2011). However, parallel experiments with humans show that the anterior distribution of monkey error-ERPs recorded during the saccadic stop-signal task is virtually identical to that found in humans (Reinhart et al., 2011).

In agreement with our parallel experiments with human subjects (Reinhart et al., 2011), we observed no single-trial correlations between ERN or Pe amplitude and post-error RT adjustments. Reported relationships between error-ERPs and post-error RT adjustments vary across the literature. Although some report such correlations (Gehring et al., 1993; Nieuwenhuis et al., 2001; Rodriguez-Fornells et al., 2002; Kerns et al., 2004; Debener et al., 2005; Holroyd et al., 2005; Klein et al., 2007; Ladouceur et al., 2007; West and Travers, 2008; Huster et al., 2011), others report no or contradictory evidence (Gehring and Fencsik, 2001; Hajcak et al., 2003; Nunez Castellar et al., 2010). Additionally, post-error slowing is not consistently observed in the stop-signal task (Emeric et al., 2007; Nelson et al., 2010; Bissett and Logan, 2011). Single-trial EEG has variability that is approximately an order of magnitude higher than ERPs (Luck, 2005), so we also analyzed the ERP data using a median split of post-error RT. Even with reduced variability due to averaging, we did not observe consistent error-ERP fluctuations predicting post-error RT adjustments. In addition, we did not observe conflict related modulation of ERPs. These findings agree with those from our parallel study with humans performing the same task, but challenge the view that error-ERPs reflect the activity of a general conflict monitoring system (Yeung et al. 2004).

We previously reported error-related LFPs recorded in ACC and SEF during the saccadic stop-signal task (Emeric et al., 2008; 2010). It is tempting to speculate that these LFPs give rise to the error-ERPs recorded at the surface, but several observations complicate this interpretation. First, error-related LFPs and ERPs differ in their relationships to behavior. Error-related LFP amplitude recorded from SEF is correlated with post error RT adjustments. And SEF LFPs also exhibit a negative potential during periods of increased response conflict (Emeric et al., 2010). In contrast, LFPs recorded in ACC exhibit a positivity with increased response conflict (Emeric et al., 2008). We observed no such correlations in surface ERPs. These conflicting results from recording inside and outside the brain support the assumption that ERP signals arise from the summation of LFPs generated broadly throughout the brain (Luck, 2005; Nunez and Srinivasan, 2006). Thus, surface ERPs cannot be fully explained by LFPs in SEF or ACC. Second, timing differences between the ERPs and LFPs are obvious (Figure 11). The onset of the ERN and Pe on the surface precede intracranial LFP onsets. One possible explanation for these results is that subjects may show individual differences in timing of error-ERP onset, or that timing may change as subjects gain experience. Simultaneous ERP and LFP recordings must be carried out to test these explanations.

The observation of monkey error-ERPs will allow for their detailed neurophysiological characterization. Single units in ACC (Shima and Tanji, 1998; Ito et al., 2003; Amiez et al., 2005), and SEF (Stuphorn et al., 2000) are modulated when monkeys commit errors. Neurons in ACC modulate when monkeys switch responses after errors (Johnston et al., 2007; Quilodran et al., 2008). SEF neurons also show activity which may bias performance toward rewarding responses (Coe et al., 2002; Stuphorn et al., 2010), and stimulation of SEF improves saccadic stop-signal performance (Stuphorn and Schall, 2006). Despite these results, some have wondered whether intracranial recordings in monkeys are generated by

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the same error monitoring processes reflected in human ERPs (Cole et al., 2009; 2010; but see Schall and Emeric, 2010). The finding of monkey error-ERPs thus bridges a gap between human and monkey studies of executive control.

Several issues require clarification. First, the precise neuroanatomical loci of error-ERPs have not been described. Second, the neurophysiological events which give rise to the ERN and Pe remain obscure. Third, several models have been proposed to explain the relationship of error-ERPs to performance monitoring generally, but the neural plausibility of these theories remains speculative. Neurophysiological recordings with nonhuman primates can shed much needed light on these questions. For the remainder of the Discussion, we will consider each of these issues in turn.

What are the anatomical sources of error-ERPs?

Dipole source modeling efforts and fMRI results suggest a central role for the dorsal ACC (e.g. Dehaene et al., 1994; Carter et al., 1998; Holroyd et al., 1998; reviewed by Taylor et al., 2007). However, it is well known that dipole source techniques rely on under-constrained solutions to the inverse problem of ERP localization (Helmholtz, 1853; Luck, 2005). Moreover, the link between electrophysiology and haemodynamic regulation is poorly understood (Logothetis and Wandell, 2004). Evidence indicates that other areas may play an important role in producing error-ERPs (reviewed by Gehring et al., 2011). In addition to the studies with monkeys described above, researchers have implicated the supplementary motor area, and rostral ACC as potential substrates for error-ERPs (Dehaene et al., 1994; Kiehl et al., 2000; Menon et al., 2001; Luu et al., 2003). And intracranial recordings in patients show error-related activity in multiple cortical areas beyond medial frontal cortex (Brázdil et al., 2002; Halgren et al., 2002). An animal model will be a great asset in the search for definitive neural generators.

What is the physiology underlying observed ERN and Pe?

The idea that mesocortical dopaminergic (DAergic) signaling produces the ERN has received substantial interest (Holroyd and Coles, 2002). However, relatively few researchers have experimentally manipulated DAergic signaling pathways (reviewed by Jocham and Ullsperger, 2009). Because studies using the ERN have been carried out in humans, invasive neuroscientific techniques have been impractical. This is discouraging, since the DA hypothesis is rooted in classic neurophysiological studies using monkeys (Schultz et al., 1997; Schultz, 1998; see also Redgrave et al., 1999a, b). Although DA has received the most attention, several other neurotransmitters may also play a role in generating error-ERPs. These include norepinephrine (Riba et al., 2005b), serotonin (Fallgatter et al., 2004), and GABA (Johannes et al., 2001; de Bruijn et al., 2004; Riba et al., 2005a). The discovery of monkey error-ERPs will open new avenues for research on the neurochemical and neurophysiological events underlying these components. Now, tools such as selective agonist and antagonist micro-injections can be combined with surface electrode recordings to determine the contributions of various neurotransmitters and cell populations to error-ERPs.

What is the relationship between error-ERPs and performance monitoring?

The relationship between error-ERPs and performance monitoring is unclear. They were first thought to reflect neural processing of mismatch between committed and intended responses (Falkenstein et al., 1990; Gehring et al., 1993; Coles et al., 2001). This view has several drawbacks. For instance, if some area has access to a representation of the intended response, why was a different response executed (but see Murthy et al., 2007; 2009)? Other theories allow performance monitoring to proceed without *a priori* knowledge of future outcomes. Several of these have been expressed as computational models that frame precise

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hypotheses (Botvinick et al., 2001; Holroyd and Coles, 2002; Yeung et al., 2004; Brown and Braver, 2005). The most influential computational models cannot be resolved using behavioral data alone. For instance, a major obstacle in testing the conflict monitoring theory arises from an inability to measure response conflict directly (Gehring et al., 2011). Similarly, reinforcement-learning theories (e.g. Holroyd & Coles, 2002; Brown & Braver, 2005) have proven difficult to test using behavioral measures alone. However, specific assumptions of these models can be tested with neurophysiological measures in an animal model of error-ERP.

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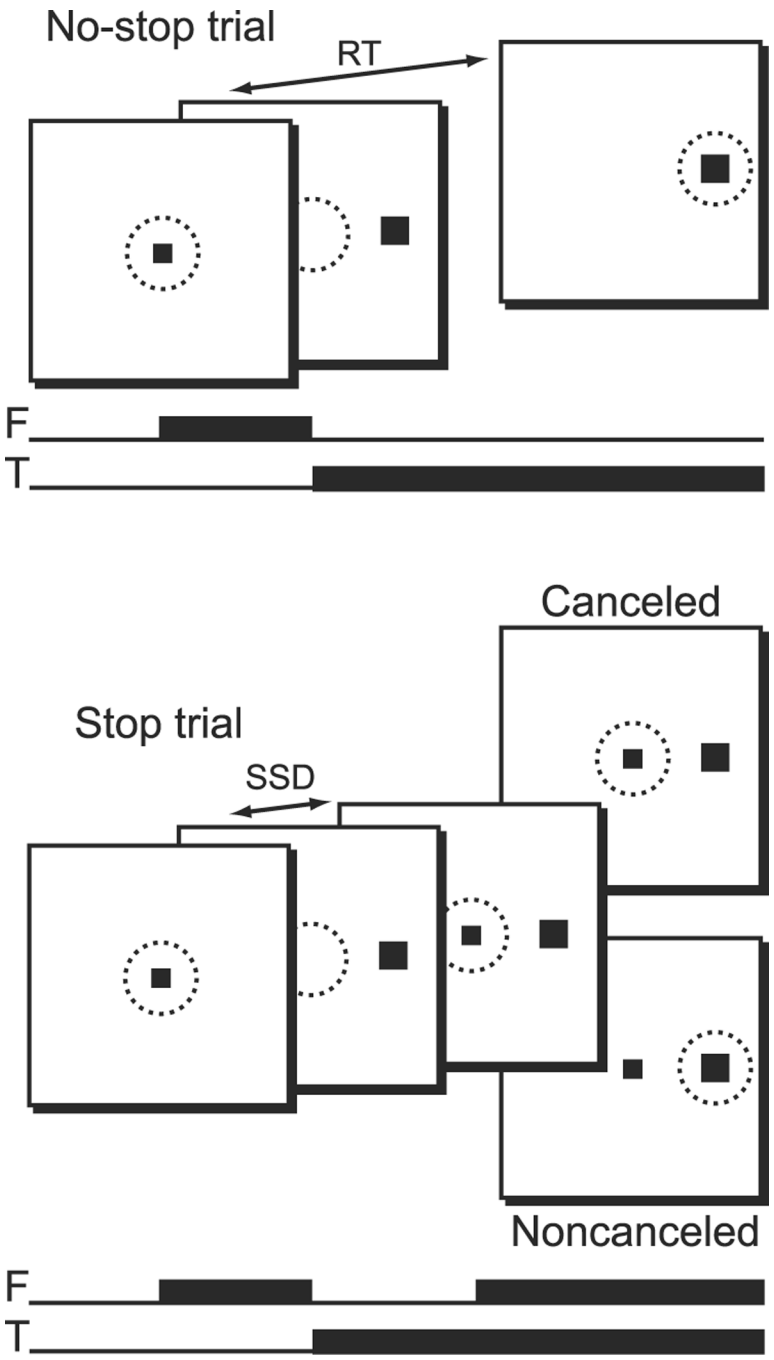

Figure 1.

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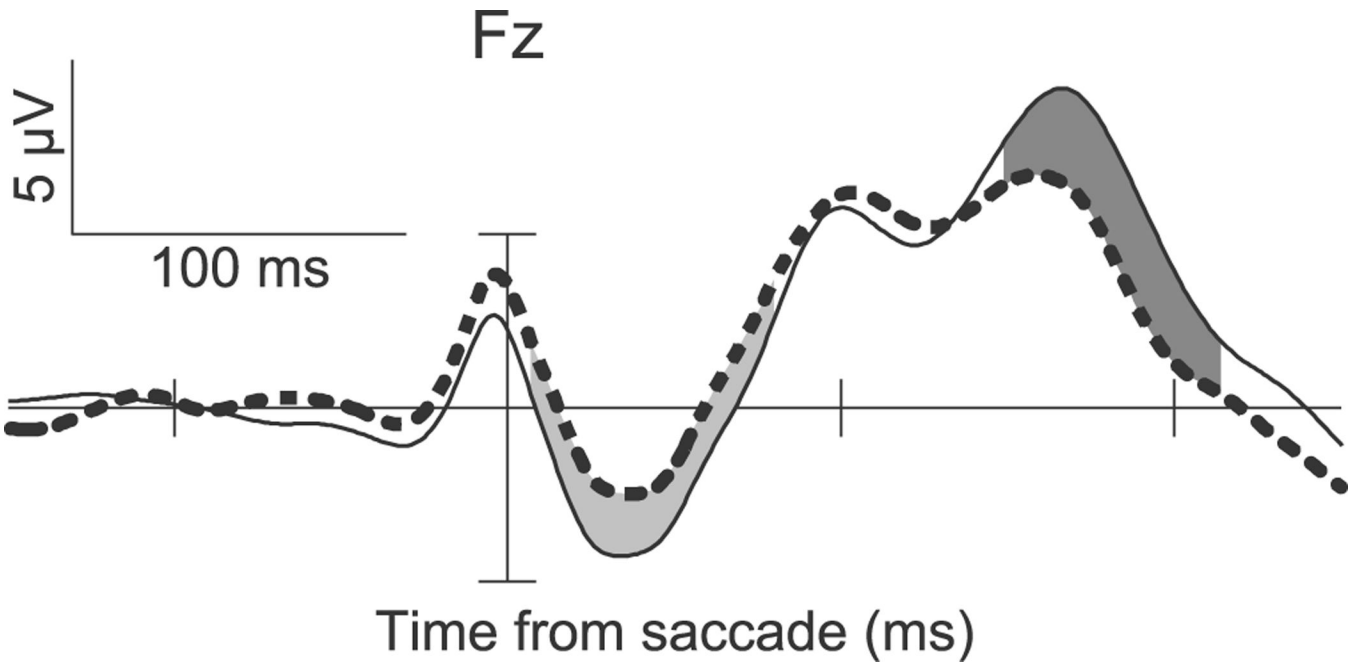

Figure 2.

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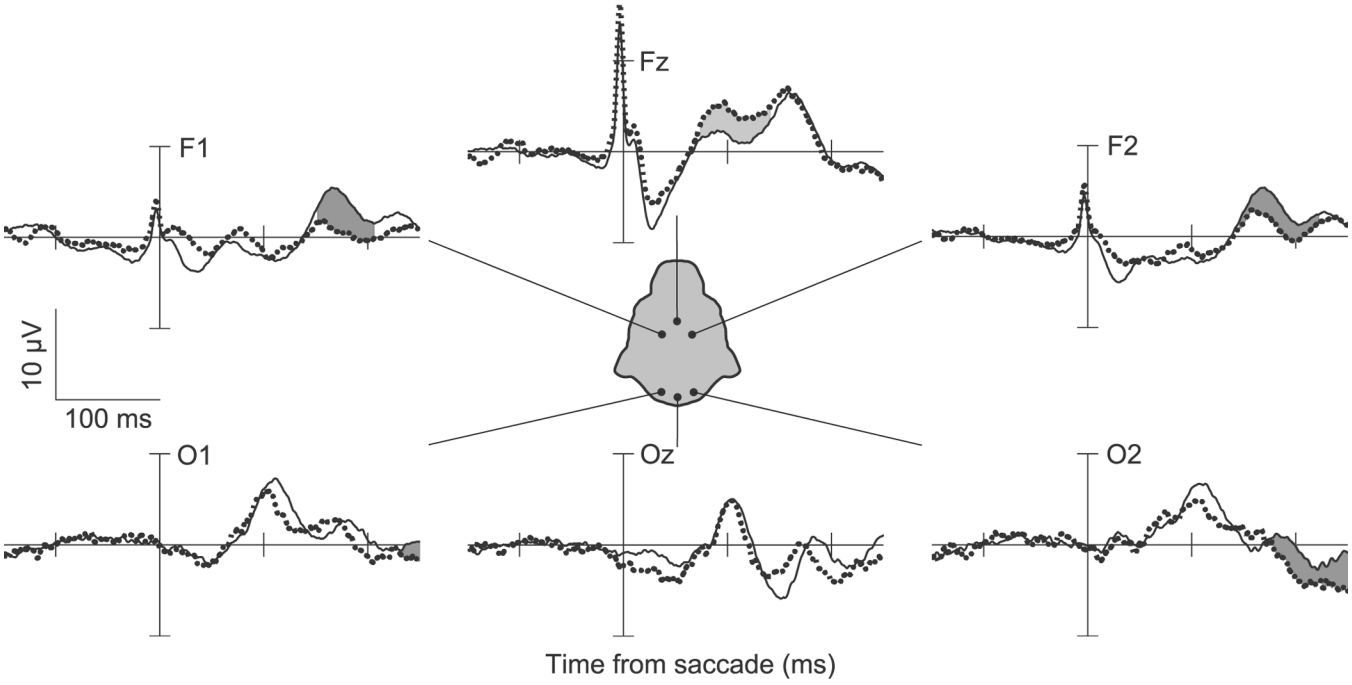

Figure 3.

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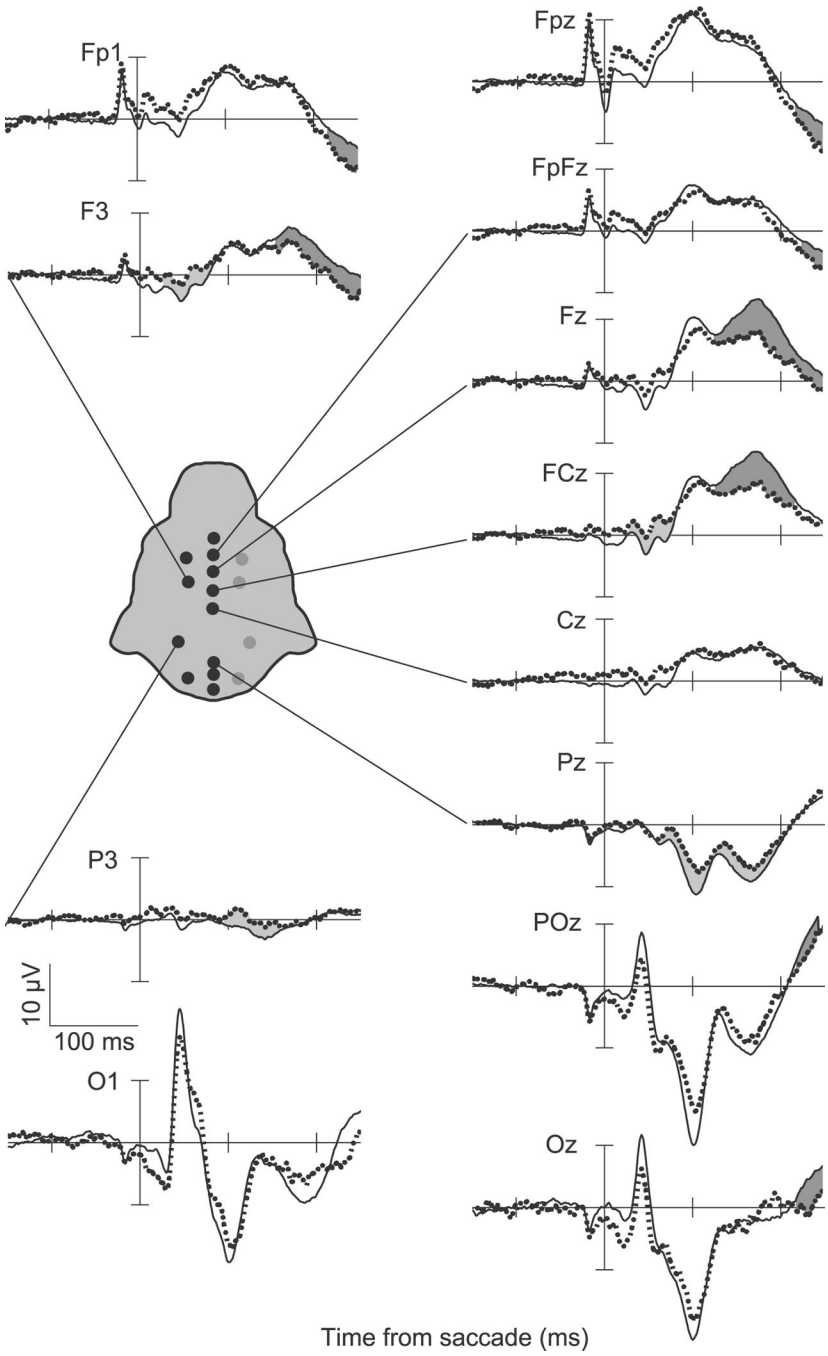

Figure 4.

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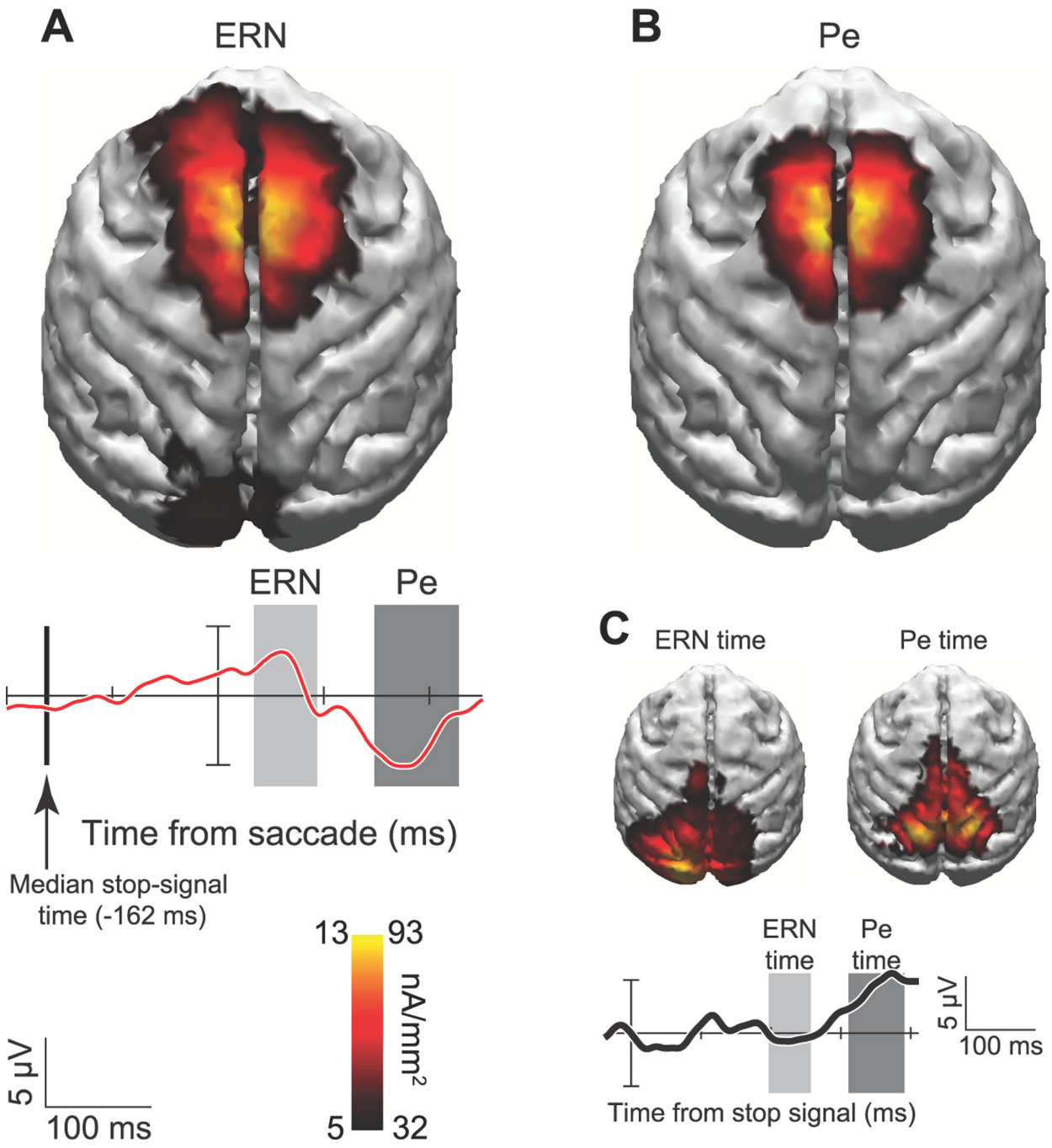

Figure 5.

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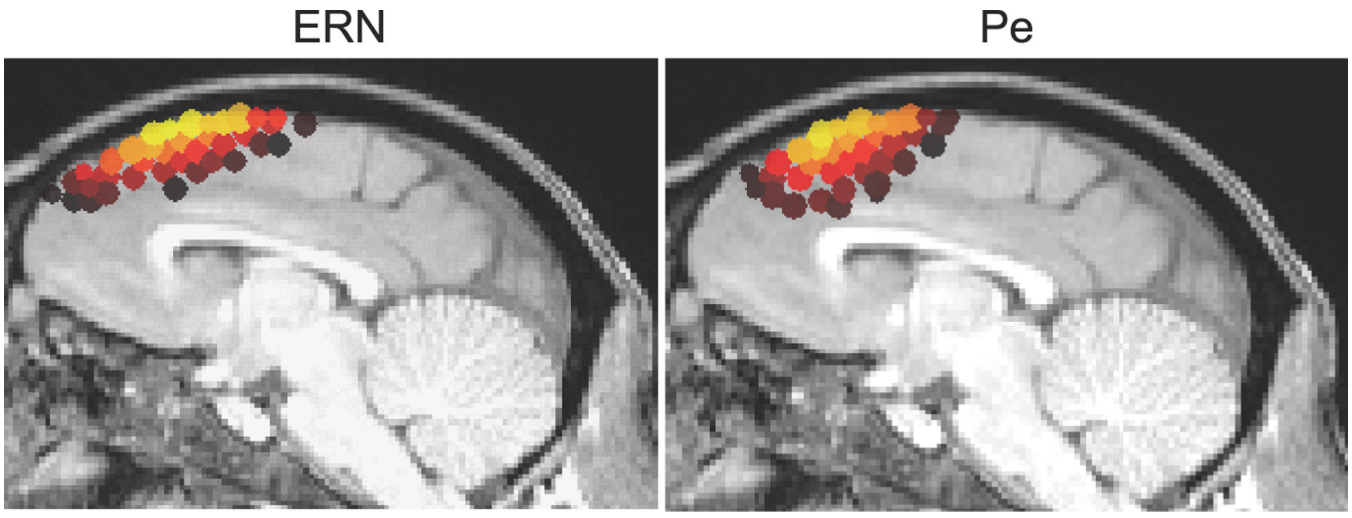

Figure 6.

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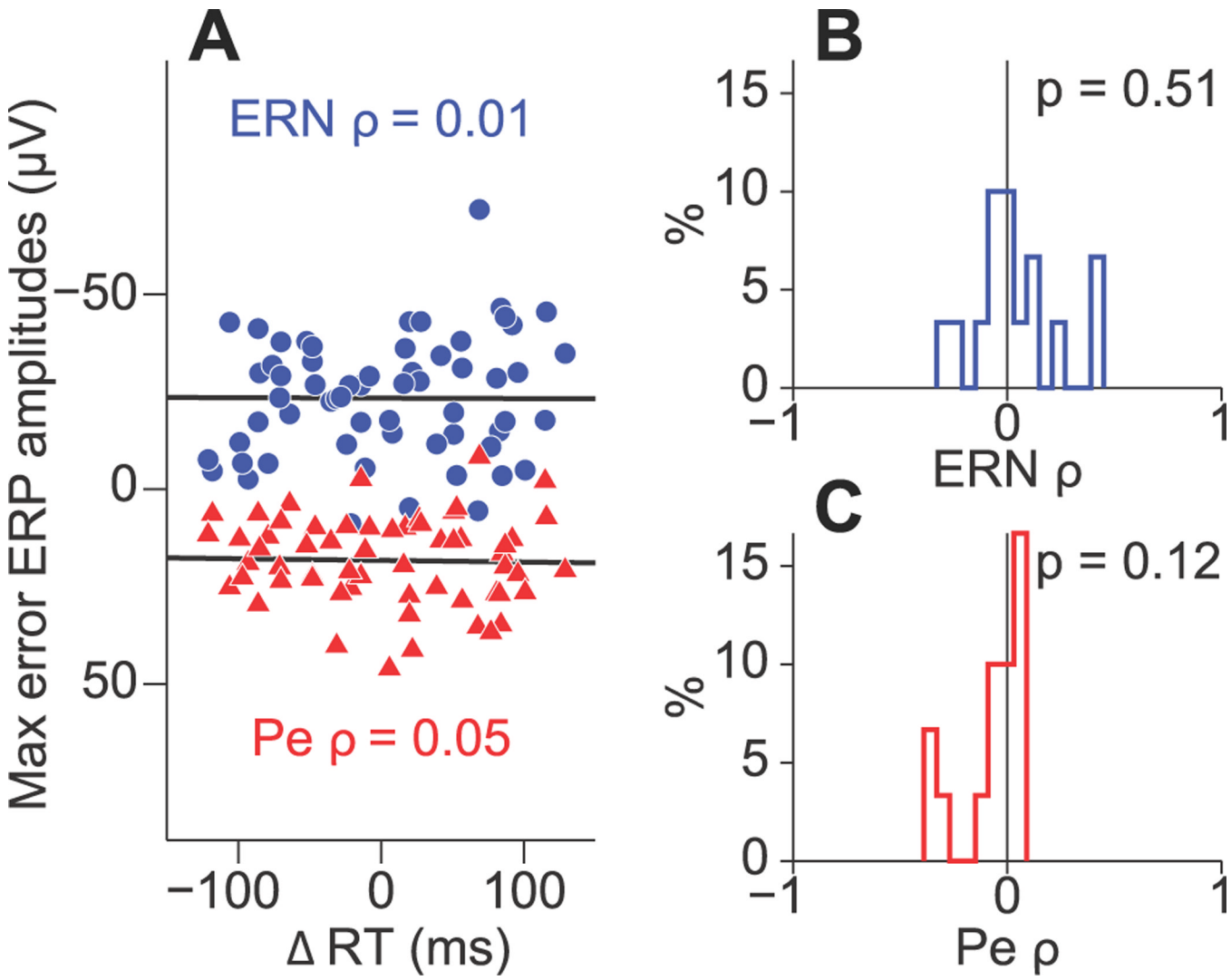

Figure 7.

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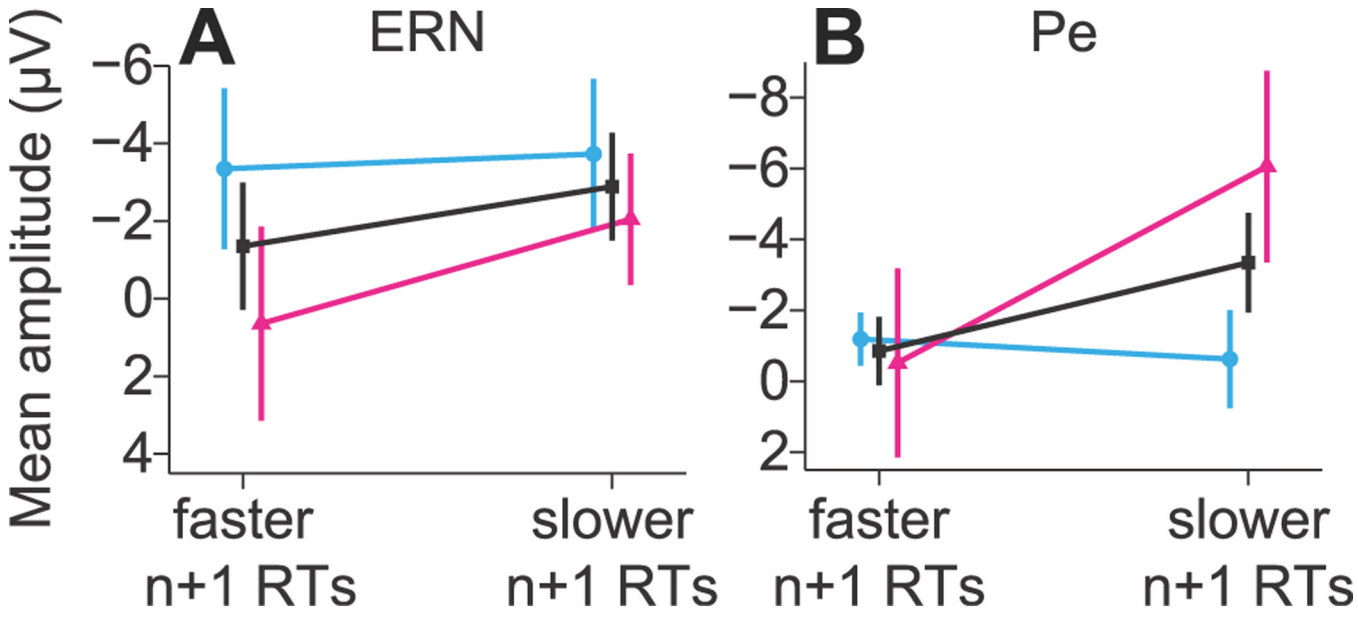

Figure 8.

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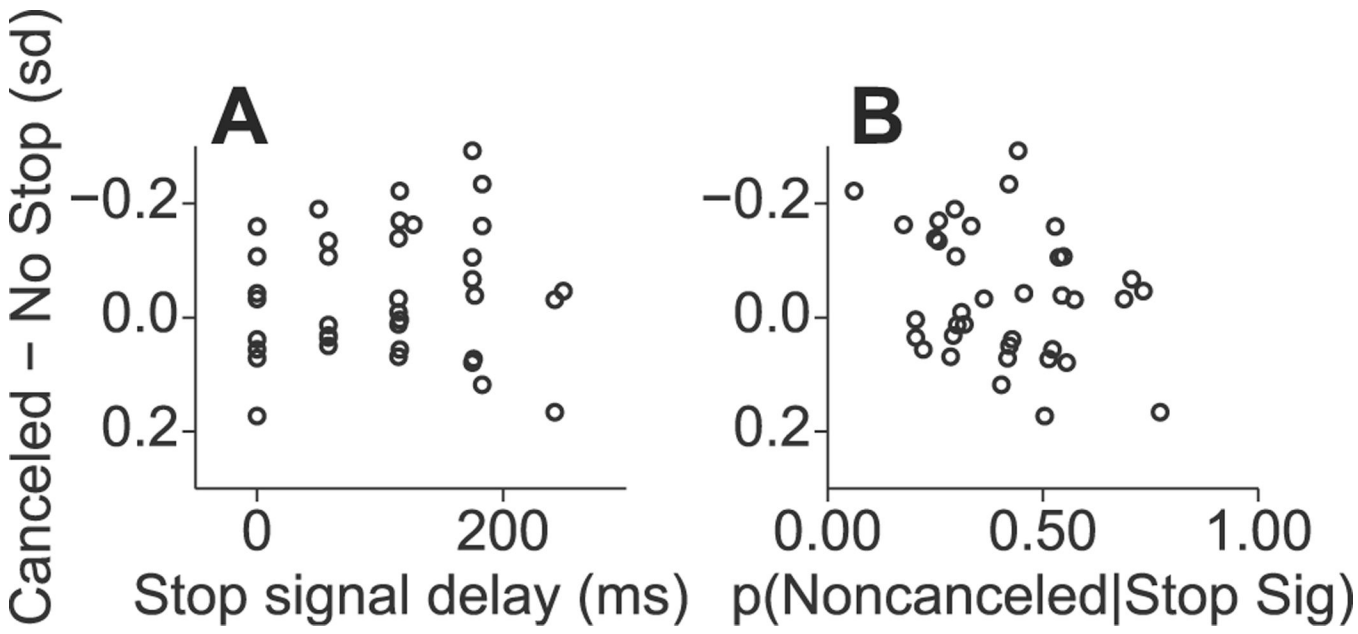

Figure 9.

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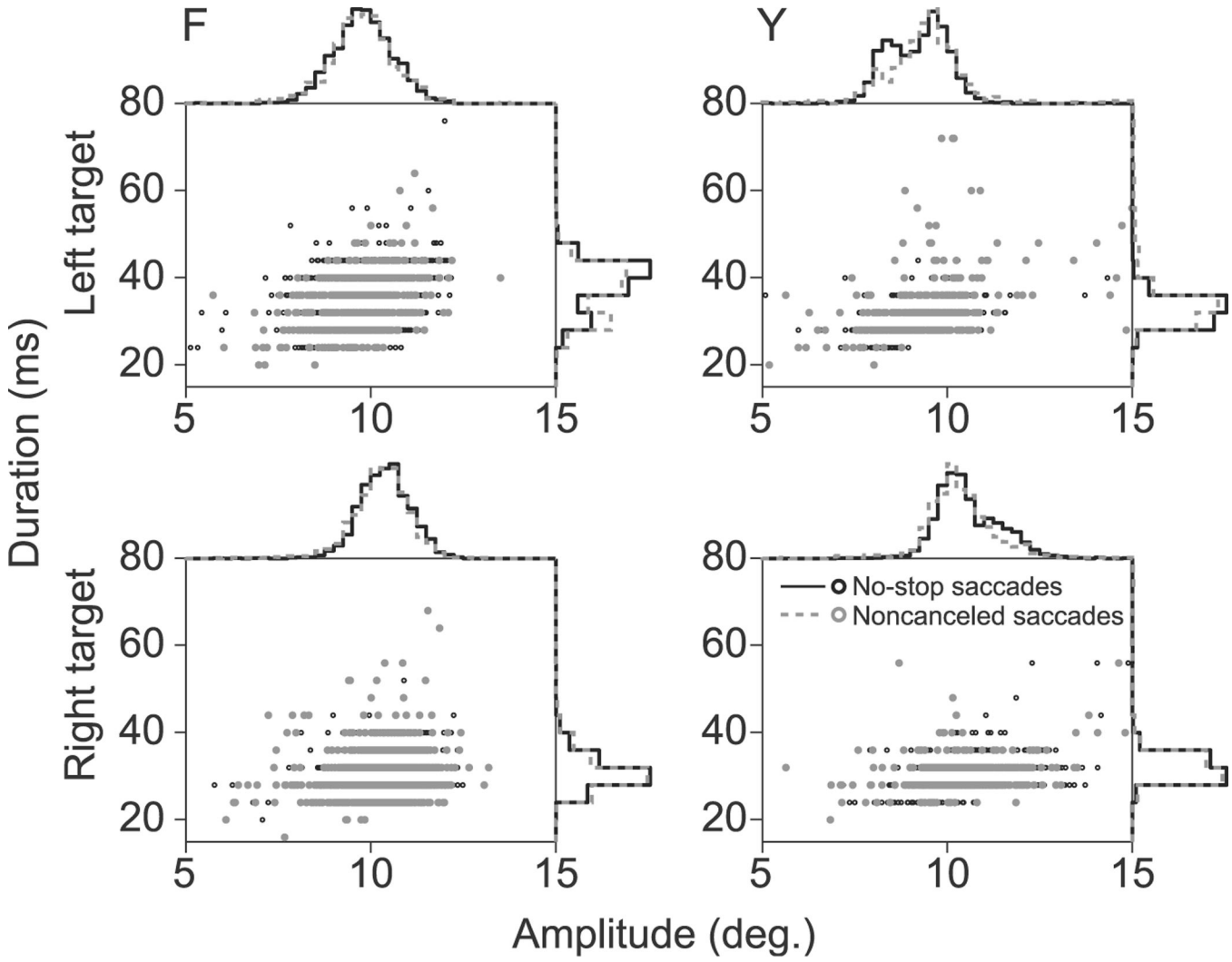

Figure 10.

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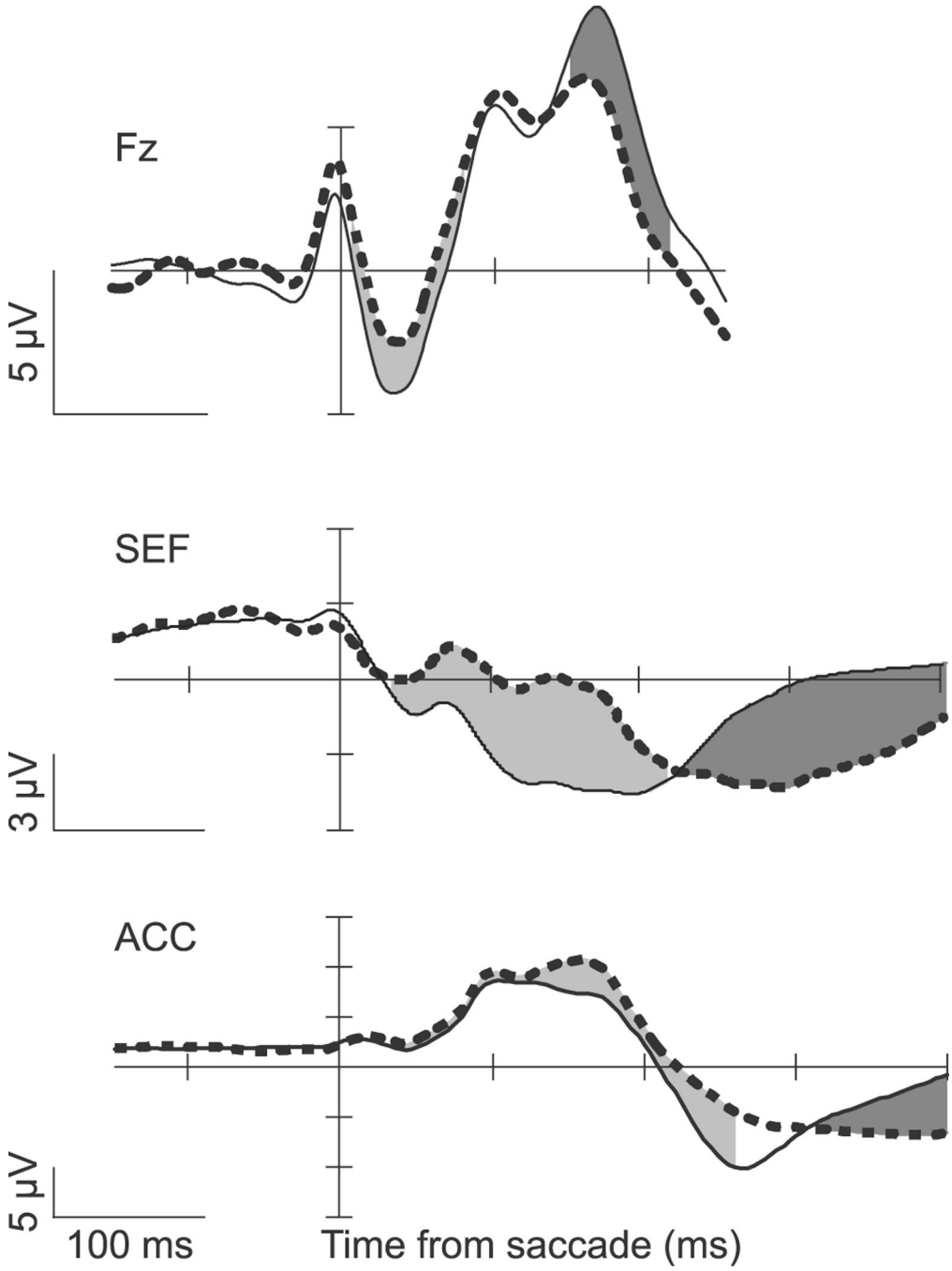

Figure 11.

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Table 1

Implanted electrode locations

	Monkey F		Monkey Y	
Electrode	AP	ML	AP	ML
Fpz	\	\	5.33	0
FpFz	\	\	4.3	0
Fp1	\	\	4.12	−1.75
Fp2	\	\	4.12	1.63
Fz	4.1	0	3.28	0
F1	2.7	−1.4	\	\
F2	2.7	1.4	\	\
F3	\	\	2.69	−1.59
F4	\	\	2.69	1.49
FCz	\	\	2.24	0
Cz	\	\	1.23	0
P3	\	\	−0.61	−2.19
P4	\	\	−0.61	2.19
Pz	\	\	−1.75	0
POz	\	\	−2.5	0
O1	−2.1	−1.4	−2.81	−1.64
O2	−2.1	1.4	−2.81	1.54
Oz	−2.5	0	−3.58	0

Stereotaxic locations of implanted electrodes in cm relative to interaural zero. Electrode names refer to homologous human electrode locations from the international 10–20 placement system. ML = medial to lateral, AP = anterior to posterior.

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Table 2

Summary statistics for stop-signal task performance

	No-stop RT	Noncanceled RT	p(Noncanceled)	SSRT
Monkey F	285 ± 77	250 ± 75	0.54	94
Monkey Y	252 ± 87	218 ± 92	0.53	71

Reaction times (± 1 standard deviation), probability of committing errant noncanceled saccades, and SSRTs for each subject collapsed across sessions