Prefrontal–cingulate interactions in action monitoring

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We found that medial frontal cortex activity associated with action monitoring (detecting errors and behavioral conflict) depended on activity in the lateral prefrontal cortex. We recorded the error-related negativity (ERN), an event-related brain potential proposed to reflect anterior cingulate action monitoring, from individuals with lateral prefrontal damage or age-matched or young control participants. In controls, error trials generated greater ERN activity than correct trials. In individuals with lateral prefrontal damage, however, correct-trial ERN activity was equal to error-trial ERN activity. Lateral prefrontal damage also affected corrective behavior. Thus the lateral prefrontal cortex seemed to interact with the anterior cingulate cortex in monitoring behavior and in guiding compensatory systems.

In demanding task situations, it is important to detect when actions are (or are likely to be) erroneous—and to correct for the problem. A number of theorists have postulated executive[1–4] or supervisory[5] systems that oversee and modulate action in this manner. Evidence from electrophysiological and neuroimaging studies suggests that the anterior cingulate cortex and the lateral prefrontal cortex (PFC) are active in situations demanding such action monitoring activity. Evidence linking the anterior cingulate cortex to action monitoring derives from studies of the error-related negativity, an event-related brain potential that occurs at the moment of an error in cognitive reaction-time tasks[6–9]. Dipole localization studies of the ERN suggest that it is generated by a medial frontal structure, most likely the anterior cingulate cortex[10,11]. Also consistent with this evidence, the ERN is enhanced in individuals with obsessive-compulsive disorder[12], which is linked to a hyperactive neural circuit involving the anterior cingulate[13]. FMRI studies examining error processing confirm the presence of anterior cingulate activation associated with errors[14,15].

Numerous studies also point to a role for lateral prefrontal regions in action monitoring. FMRI studies demonstrate that, in addition to anterior cingulate activation, activation may occur in lateral PFC on error trials[14,15]. In other situations involving demanding tasks or conflicting response tendencies, PFC activation and anterior cingulate activation often co-occur[16–19]. Co-occurrence of prefrontal and cingulate activity related to error processing is observed in single-neuron recordings from non-human primates[20]. An important question emerging from these studies, then, concerns the nature of the interaction between lateral and medial frontal regions. Do the anterior cingulate cortex and the PFC interact in these situations? If so, what is the nature of that interaction, and specifically, how do the computations performed by one structure depend on the computations performed by the other?

Investigators of anterior cingulate and PFC function make different claims about how these structures monitor for situations requiring executive control and implement the control when the need is detected. Some investigators emphasize the role of the anterior cingulate in detecting problems in the action system, speculating that other structures interacting with the anterior cingulate correct the problems. Included in these perspectives are those that assign error detection[21] or conflict detection[14,22] functions to the anterior cingulate. Other views, however, imply that structures such as the PFC evaluate the need for executive control, and that the anterior cingulate actually implements the control[23,24].

Here we examined the interaction between medial and lateral frontal regions by recording the ERN from individuals with focal lesions of the lateral PFC. A normal pattern of ERN activity in these individuals would indicate that the medial frontal regions operated independently of the lateral PFC in generating the ERN. In contrast, an absence or reduction of ERN activity in these individuals would indicate that the lateral PFC was either necessary for generation of the ERN or was itself a generator of the ERN. Other disruptions in the pattern of ERN activity would suggest that lateral PFC modulated the generation of the ERN, perhaps by supplying information or activation that was critical for medial frontal processing.

We measured the ERN and error correction behavior while participants made responses in a letter-discrimination task. Participants made a squeezing response to a pair of letters, one of which was designated as a target letter. One letter appeared in red and the other in green. One second before the letter pair, a precue (the word 'red' or 'green') indicated which letter was the target letter. The task was to respond with one hand if the target letter was 'H' and with the other hand if the letter was 'S'. On half of the trials, the irrelevant flanking letter was identical to the target letter; on the other half of the trials, the irrelevant flanking letter signaled the incorrect response (a manipulation that provoked erroneous responses).

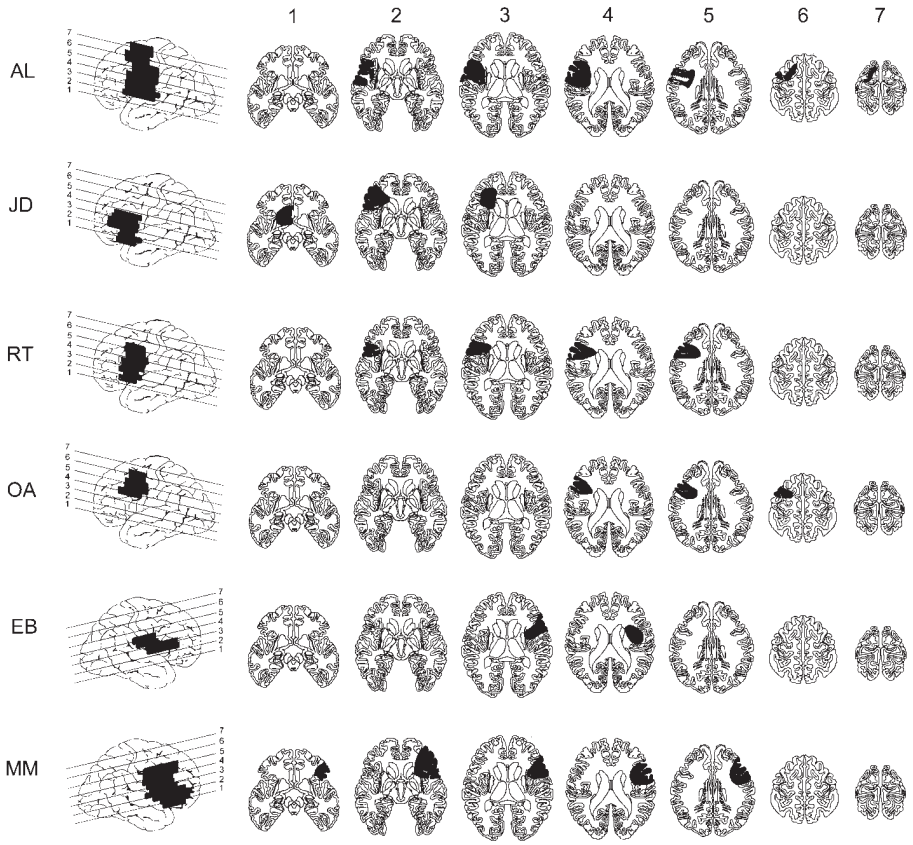

Fig. 1. Lesion reconstruction in the six individuals with unilateral damage in the lateral prefrontal cortex. The shaded areas represent the lesion. The lines on the lateral reconstructions indicate the location of the corresponding axial section.

Results

Behavioral and event-related potential data from six individuals with focal damage to the lateral PFC (mean age 69) were compared with data from 10 age-matched control participants (mean age 70) and 10 younger control participants (mean age 24). Within the PFC group, two of the individuals (one male, one female) had lesions in the right hemisphere, and the remainder had lesions in the left hemisphere (**Fig. 1**).

In overall task performance, proportion correct values were 0.90 for young controls, 0.95 for age-matched controls and 0.91 for the PFC group, a difference that was not significant ($F_{2,23}$ = 2.92, p = 0.074, mean squared error, m.s.e. = 0.009, analyzed with the arcsine transform). The mean (range) for number of errors for the young group was 46.6 (16–71), for the age-matched group, 26.6 (12–58), and for the PFC group, 41.2 (18–98). Each group differed significantly from the others in mean correct reaction time: mean reaction time was 515 ms for young controls, 761 ms for age-matched controls and 992 ms for the PFC group ($F_{2,23}$ = 38.95, $p < 0.000001$, m.s.e.= 11,322).

ERN

As reported in other studies, healthy younger and older participants showed a negative-polarity peak—the ERN—at the moment of the error response, with a reduced or no ERN on correct trials (**Fig. 2**). For the PFC group, there was an ERN peak on error trials. However, we observed a peak of equivalent size in that group's correct-trial waveform. Confirming these impressions, a significant group × accuracy interaction was obtained ($F_{2,23}$ = 6.86, $p < 0.005$, m.s.e. = 13.97). The amplitude on error trials was larger than on correct trials only in the young participants and in the age-matched controls. In the PFC group, the ERN amplitude at the Cz electrode did not differ between correct and error trials, nor did the error-trial ERN amplitude differ from that seen in age-matched controls (young/correct, 0.5 ± 0.7 μV; young/error, 4.5 ± 1.1 μV; age-matched/correct, 1.9 ± 1.2 μV; age-matched/error, 3.1 ± 1.1 μV; PFC/correct, 3.2 ± 0.9 μV; PFC/error, 2.9 ± 1.0 μV; mean ± s.e.). A test of the ERN amplitude on correct trials revealed that only the PFC group showed correct-trial ERN activity that differed significantly from zero (t_5 = 3.40, $p < 0.02$). Neither control group showed significant correct-trial ERN activity (age-matched t_9 = 1.65, $p > 0.10$; young t_9 = 0.76, $p > 0.10$). Kendall's τ rank-order correlations calculated across the six individuals with PFC damage failed to reveal significant relationships between lesion volume (computed using lesion reconstruction software) and ERN amplitude at Cz. For correct trials, τ = 0.20, $p > 0.10$, n = 6; for error trials, τ = 0.07, $p > 0.10$, n = 6.

Corrective action

To determine whether PFC damage influenced behavioral indications of error processing, we examined the proportion of errors corrected for each group. Error corrections were those error trials on which a correct response followed the error. A one-way ANOVA comparing the three groups on the proportions (arcsine transform) produced a significant main effect ($F_{2,23}$ = 5.03; p = 0.015; m.s.e. = 0.126). The percentage of errors corrected by the PFC group was smaller (mean ± s.e., 11 ± 6%) than the percentage corrected by their age-matched controls (32 ± 5%), but not significantly smaller that the proportion corrected by young controls (19 ± 5%). We also computed the proportion of correct responses that were followed by errors. In each group, fewer than 1% of correct responses were 'corrected' in this manner, and the groups did not differ ($F_{2,23}$ = 0.02, p = 0.98, m.s.e.= 0.015).

A reduction in response force on error trials is often observed in speeded response tasks, suggesting that errors are inhibited on line[21]. To examine the effects of PFC damage on this inhibition, we computed the peak force exerted on correct response trials and compared it with the peak force exerted on error trials (**Fig. 3**). Compared with both control groups, the PFC group showed less inhibition of force on error trials (group × accuracy interaction, $F_{2,23}$ = 5.30, p = 0.0128, m.s.e.= 1.03).

Another form of corrective action is post-error slowing: in speeded response tasks, individuals slow down following errors, possibly to prevent future fast-guess errors[25]. PFC damage did

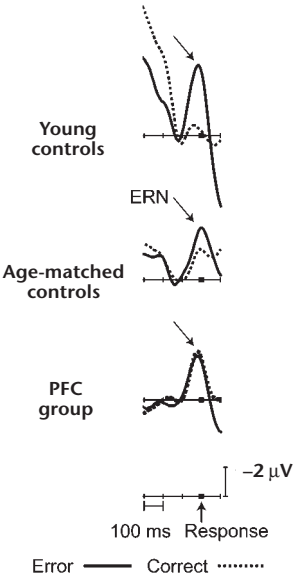

Fig. 2. Response-locked event-related potentials from the vertex (Cz) electrode. Top, data from error trials (solid lines) and correct trials (dashed lines) for the young controls. The ERN (indicated by an arrow in each plot) is evident as a peak on error trials occurring at around the moment of switch closure. Middle, corresponding ERN data from the age-matched controls. Bottom, ERN data from the group with lateral prefrontal cortex (PFC) damage. Error-trial ERN activity exceeded correct-trial activity only in the young and age-matched control groups; significant activity shown by the PFC group on correct trials was equal to that observed on error trials.

not seem to affect this post-error slowing. For all groups, correct responses on trials following error trials were slower than following correct trials ($F_{2,23} = 31.92$, $p < 0.00001$, m.s.e.= 2809; **Fig. 4**). The absolute amount of slowing was actually greater in the older controls and PFC group than in the young controls (group × accuracy interaction, $F_{2,23} = 4.06$, $p < 0.05$, m.s.e. = 2809). The proportional slowing (reaction time following error trials divided by reaction time following correct trials) did not differ between groups ($F_{2,23} = 2.09$, $p > 0.10$, m.s.e. = 0.008).

Discussion

Our data suggest that the PFC participates in action monitoring: the medial frontal generator of the ERN and corrective action were both influenced by the PFC damage. The PFC group's ERN failed to show the typical difference between error trials and correct trials. Correct trials elicited an ERN-like peak that was as large as the ERN seen on error trials. Note, however, that the amplitude of the ERN on error trials was the same in the individuals with PFC damage as it was in the age-matched controls. Thus, whereas the PFC did not itself generate the ERN, it must have participated in the circuitry that caused the usual difference between error- and correct-trial ERN activity. Moreover, the usual pattern of ERN activity must have depended upon the cooperation of the PFC in both cerebral hemispheres, because unilateral PFC lesions were sufficient to disrupt that pattern.

Some forms of corrective behavior were also disrupted in the PFC group. Compared with age-matched controls, the PFC group corrected a smaller proportion of errors and showed a smaller reduction in response force on error trials relative to correct trials. Interestingly, post-error slowing was present even in the individuals with PFC damage, consistent with the sparing of a compensatory mechanism. Note, however, that post-error slowing was an imperfect measure of corrective behavior, because problems that caused the error could persist and contribute to post-error slowing.

The PFC must have influenced the computation that caused an ERN difference between correct and error trials. If a medial frontal structure such as the anterior cingulate did give rise to the ERN, then normal anterior cingulate activity would depend on a functioning PFC. The data, however, argue against models in which the PFC detects the need for executive control and signals the anterior cingulate, which then performs the control function[23,24]. For example, a simple mapping between the amount of medial frontal ERN activity and the likelihood of an error correction is not plausible. Whereas the error correction behavior in the PFC group did distinguish between errors and correct responses, the ERN activity did not. Moreover, although the PFC group showed an unusually large ERN on correct trials, they did not 'correct' their correct responses more often than did the controls.

Simple models postulating the reverse arrangement, in which the anterior cingulate detects problems and communicates with the PFC structures that implement the executive control, are also inconsistent with our data. If the communication were one-way, with information flowing only from the anterior cingulate to the PFC, the PFC damage would not have affected the ERN, contrary to our data. Furthermore, such a model would also suggest that, if the signal represented by the ERN invariably modulated behavior, 'false' error corrections would occur.

Our data thus point toward more complex models. For example, the anterior cingulate may depend on intact PFC for information necessary to distinguish errors from correct responses. According to this view, the anterior cingulate monitors for response conflict or errors, and other systems further downstream actually implement the compensatory behavior. Several models postulate that the prefrontal cortex maintains representations that define the contextually appropriate stimulus–response mappings used for decision making[1,5,26–28]. Without such representations, the medial frontal cortex would not be able to determine what was correct and what was not (and might, by default, produce the ERN). According to this hypothesis, then, one source for the observed ERN dysfunction could be that PFC damage rendered the anterior cingulate unable to distinguish correct from incorrect responses. Alternatively, weakening the representation maintained by the PFC might have permitted multiple competing responses to become active, causing response conflict subsequently detected by the anterior cingulate. In either case, the alerting signal produced by the anterior cingulate would be less reliable. Compensatory systems may have been unable to act on such an unreliable alerting signal, or they may have made strategic adjustments to weaken the coupling between the less-reliable alerting

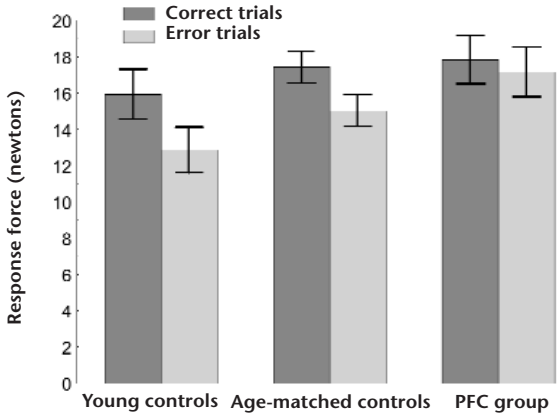

Fig. 3. Mean response force for each participant group. The reduction in force on error trials relative to correct trials was less pronounced in the PFC group than in either control group. Dark bars, mean peak force associated with correct responses. Light bars, mean peak force associated with error responses. Error bars represent ± s.e.

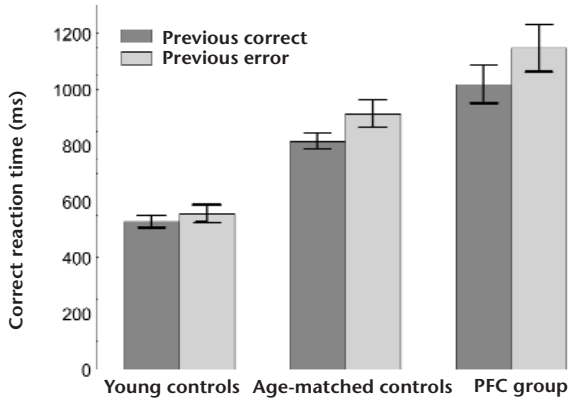

Fig. 4. Correct reaction times as a function of the response on the previous trial for each participant group. All groups showed a significant slowing on trials that followed errors relative to trials that followed correct responses. Dark bars, mean correct reaction time on trials following a correct response. Light bars, mean correct reaction time on trials following an error. Error bars represent ± s.e.

signal and the compensatory action, thus producing the observed effects of PFC damage on compensatory behavior.

An alternative model consistent with our data posits that the activity reflected in the ERN represents an affective or motivational signal[29]. The signal could serve an alerting function that mobilized affective systems, rather than immediate corrective action, perhaps via cingulate connections with the amygdala and brainstem autonomic nuclei[30]. This conception of the ERN would be consistent with the dissociation of ERN activity from compensatory behavior[21], with reports of medial frontal ERN-like activity in response to unpleasant stimuli[29] and with observed relationships between ERN activity and negative affect[31]. The PFC damage may have disinhibited a medial frontal emotional response that normally occurs only in response to errors. Of course, this model and the one outlined above may both be accurate, and the ERN could actually be a composite of several signals, with the rostral cingulate influencing affective responses and the caudal cingulate influencing compensatory motor behavior[30].

Our results suggest that a system other than the anterior cingulate or the PFC implements corrective action. One candidate system would be the basal ganglia, which could modulate the motor system to implement corrective action. Such an arrangement would be consistent with the extensive connections between the basal ganglia and both the anterior cingulate[30] and the PFC[28], and with basal ganglia hyperactivity and exaggerated compensatory behavior in obsessive-compulsive disorder[13].

Methods

The PFC lesion group consisted of six individuals (four men, two women, mean age 69) with lesions centered in the PFC as shown with computerized tomography (CT) or magnetic resonance imaging (MRI; **Fig. 1**). Participants' lesions resulted from infarction of the middle cerebral artery and occurred at least one year before the tests. Two of the individuals (one male, one female) had lesions in the right hemisphere, and the rest had lesions in the left hemisphere. Maximum lesion overlap was centered in the posterior lateral frontal cortex including portions of the middle and inferior frontal gyri and premotor cortex. A group of older adults (four males, six females, mean age 70) matched in mean age to the prefrontal group and a group of young adults (four males, six females, mean age 24) served as neurologically healthy controls. All participants were paid $10.00 per hour. The study was approved by the Institutional Review Boards of the Martinez Veterans Administration Medical Center and the University of California.

Stimuli were presented on an NEC 5FGe 21-inch color monitor. On each trial, the participant was presented with two letters, one printed in red and the other printed in green, which remained on the screen until the response. At a viewing distance of 1.5 m, each letter subtended a visual angle of approximately 1°. A precue (the word 'red' or 'green', duration 200 ms) appearing 1000 ms before the letters designated the target letter of the pair. The participant made a speeded squeezing response with one hand for the target letter 'H' and with the other for the target letter 'S'. Stimulus–response assignment was counterbalanced. Each participant completed 16 blocks of 32 trials. In half of the blocks, the precue was always 'red' or always 'green'. In the other blocks, the precue varied randomly on a trial-to-trial basis. Each target letter was accompanied by an irrelevant distractor letter that was identical ('HH' or 'SS') or different ('HS' or 'SH').

Participants responded by squeezing a device (one for each hand) consisting of a hand grip attached by a spring to a force transducer. The force transducers were 20-pound-capacity, thin-beam load cells (Omega Engineering, Stamford, Connecticut, LCL-020). Participants positioned their hands with the palm facing downward and with fingers resting on a horizontal bar attached to a vertical platform. Squeezing movement involved flexion of the fingers from a fully extended position to move the horizontal bar and platform. The transducer transformed the force applied to it into a voltage, which was digitized with the electroencephalogram (see below). A mercury switch on the platform closed when the bar moved 3 cm. The reaction time was defined as the interval between the onset of the target stimulus and the switch closure. Responses from each participant that occurred less than 50 ms after the stimulus or after the participant's mean reaction time plus 2.5 standard deviations were excluded from analysis.

The electroencephalogram (EEG) was recorded from 19 tin electrodes embedded in an elastic cap, with a ground electrode on the forehead. The reference was the left mastoid; an average mastoid reference was derived off line using right mastoid data. Eye movements were recorded using electrooculogram (EOG) electrodes. Flexor and extensor electromyogram (EMG) was recorded from 0.01 to 300 Hz. EEG and EOG were recorded from 0.01 to 30 Hz. Data were digitized at 800 Hz. EEG data were corrected for ocular artifacts[32]. EMG data were filtered off-line with a 249 point Parks-McClellan high-pass digital filter (16–20 Hz transition band) and rectified. All data were subsequently filtered with a 249-point Parks-McClellan low-pass digital filter (46–50 Hz transition band) and reduced to a 100-Hz digitization rate. ERN amplitude consisted of the mean amplitude in a 50-ms window surrounding the time of the switch closure, which corresponded to the peak of the ERN in the grand average waveforms. This measure was computed relative to a 50-ms baseline centered at the point of ERN onset in the grand average waveform (100 ms before response). Data in Figure 2 were filtered with a 59-point Parks-McClellan digital low-pass filter (10–12 Hz transition band).

We restricted the statistical analyses to 9 scalp electrodes that span the scalp region where the ERN is largest: F3, Fz, F4, C3, Cz, C4, P3, Pz and P4. Lateral electrodes for individuals with right PFC damage were switched so that F3, C3 and P3 corresponded to the side ipsilateral to the lesion. As a result, there were three levels along the anterior–posterior dimension (frontal, central or parietal) and three lateral levels (left/ipsilesional, midline, right/contralesional). ERN values for each participant were submitted to a 3 (group) × 2 (accuracy) × 3 (anterior/posterior) × 3 (lateral) mixed ANOVA with Greenhouse-Geisser corrections where appropriate. For analyses of ERN and behavioral data, main effects and interactions were evaluated with simple effects tests ($p < 0.05$) using the pooled error term.

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