

CLINICAL EEG and NEUROSCIENCE

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Error Detection, Correction, and Prevention in the Brain: A Brief Review of Data and Theories

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ABSTRACT

Errors during speeded response tasks are typically immediately followed by a large component in the event-related potential, the error-related negativity; various lines of research have suggested that this component is primarily generated by the anterior cingulate cortex. This error-related activity has generated a high level of interest and investigation by cognitive neuroscientists because of the importance of online action monitoring for theories of cognitive regulation. A subsequent component, the error positivity, has remained more elusive to date. In this review we will discuss some of the extensive research which has suggested that these components are related to performance monitoring, and, should performance be compromised, dynamically adjusting control processes. Furthermore, evidence from patients with mental illnesses, including schizophrenia and obsessive-compulsive disorder, suggests that such illnesses might be understood as resulting in part from disturbances in this action monitoring function.

INTRODUCTION

In this article, we aim to review some of the data and theories on how the brain reacts to errors, corrects them, and compensates for them. We are referring specifically to the kinds of errors sometimes referred to as "action slips" — that is, fast, impulsive errors, based on insufficient processing of the relevant stimuli, and which are generally immediately detected and corrected (as opposed to "mistakes," which might derive from faulty knowledge). We are furthermore interested in disturbances of this ability in mental illness.

It has long been known that such errors can be corrected with extremely fast reaction times¹; while such an error is being committed, processing of the stimulus continues, leading to a very fast tendency to correct the error.²

It has also long been known that following an error, people tend to compensate; that is, after people have made an error, there is generally a tendency to be slower and more careful.^{1,3} This is often referred to as "post-error slowing." Thus, after recognizing an error, people are able to adjust their behavior to minimize future errors. The research reviewed in this paper deals with how this ability is implemented in the brain.

In the early 1990s, a fast neural response to error commission during interference tasks was discovered, independently in two laboratories.^{4,5} This neural response consists of a large negative-going sharp deflection in the event-related potential of the scalp EEG, with an onset around the same time of the onset of the erroneous response (as determined by electromyography) and a peak around 50-150 ms following the error, and has a maximum at frontal/central electrodes. It is usually referred to as error-related negativity (ERN) or error negativity (NE). A subsequent component, peaking some 150-400 ms following the erroneous response, has received far less attention; this component is generally referred to as the error positivity (PE). These components are generally thought of as reflecting the activation of the performance monitoring mechanisms related to detecting and correcting the error, and making the necessary adjustments related to prevention of future errors.

Neural sources of error-related ERP components

Studies using source localization — a method to estimate which structures generate the EEG or MEG patterns observed on the scalp — have consistently modeled the ERN component as having a generator in the dorsal anterior cingulate cortex (ACC) (e.g.^{6,7}). Although we should be careful drawing firm conclusions from dipole models about the precise localization of the generators of ERP components, converging evidence that the ACC responds to errors has come from several sources. Studies using intracranial electrodes have also found ERN-like potentials

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in the dorsal ACC and related structures (e.g.⁸), supporting the dipole models. Furthermore, functional MRI studies have repeatedly shown activation of the ACC on error trials (e.g.^{9,10}). An impressive recent study, in which EEG and functional MRI (fMRI) were measured simultaneously, has shown a strong relationship between the ERN and error-related ACC activation.¹¹ Thus, the evidence suggests that the ERN is, at least for a large part, generated by the ACC and related structures in the brain.

Van Veen and Carter⁷ attempted dipole localization of the PE in addition to the ERN, and found differences between an early PE subcomponent (100-250 ms) and a late PE subcomponent (250-400 ms). The early subcomponent was well modeled by the same source that accounted for the ERN. The subsequent late subcomponent was explained by one source in the rostral ACC and one in the parietal cortex. Interestingly, fMRI studies of error commission have frequently suggested engagement of more rostral areas of the ACC in addition to the dorsal ACC (e.g.¹²), suggesting that this rostral activation might be temporally dissociated from the dorsal activation. A similar model was proposed by Herrmann et al.,¹³ using a different source localization algorithm (LORETA). They modeled the PE as being generated slightly more inferior in the dorsal ACC than the source of the ERN. These somewhat inconsistent results might be explained by the fact that Herrmann and colleagues only modeled the peak of the components, and did not distinguish between an early and late subcomponent of the PE; thus, their PE model might correspond to our own model of the early PE component.⁷

ERN: relation to motivational significance, response preparation, and post-error slowing

The ERN is generally thought of as related to the motivational significance of an error. It is greater the more emphasis participants are asked to put on accuracy over speed in their performance.^{5,14} Errors on trials during which participants are more motivated to perform correctly — either because they are told they will receive a higher reward for correct trials, or because they are told they are being evaluated by a researcher and compared to their peers — also elicit greater ERNs.¹⁵

The amplitude of the ERN appears to be related to the corrective response that follows the error. It has been shown that the ERN is positively related to the probability that the participant makes a corrective response,⁵ to the speed of this corrective response (that is, the faster the corrective response, the greater the ERN amplitude¹⁶) and also to the amplitude of the corrective response as indexed by the lateralized readiness potential (LRP) or response force.¹⁷ Furthermore, it also appears to have a greater amplitude when participants correct their errors, compared to when they are explicitly forbidden to do so.¹⁶

Results are more mixed regarding the relationship between the ERN and the amplitude of the initial erro-

neous response. Gehring et al.⁵ originally reported a negative relationship between the ERN amplitude and erroneous response force; other studies have found a positive relationship.^{17,18}

The ERN appears furthermore to be related to compensatory mechanisms, i.e., future error prevention. It has often been related to the degree of post-error slowing; that is, the greater the amplitude of the ERN, the slower participants tend to be on the trial following the error.^{5,11} Functional MRI studies have consistently shown that the error-related ACC activation is related to post-error slowing.^{10,19} Debener et al.,¹¹ simultaneously recording both ERPs and fMRI, observed a within-subject, trial-to-trial coupling between the ERN amplitude, error-related ACC activation, and post-error slowing. They suggested that the occasional failure to find a between-subject relationship between ERN and post-error slowing (e.g.²⁰) might be due to other between-subject factors such as skull thickness, morphological variation of the ACC, and trait factors.

One fMRI study has also found a relationship between error-related ACC activation and control-related activation of the right dorsolateral prefrontal cortex; activation in this area of the prefrontal cortex was found to be related to the amount of post-error slowing.¹⁰

Theories of the functional significance the ERN

Several theories describing the cognitive functionality of the ERN have been proposed. We will describe three of these theories; we will refer to these as the comparator theory, the conflict detection theory, and the reinforcement learning theory.

According to the first interpretations of the ERN, it reflected the workings of a process that compared a representation of the intended response to a representation of the actual response; the ERN reflects a mismatch between these two representations.^{4,18} One of the advantages of this theory is that it treats the ERN as comparable to other midline negativities that have been related to a mismatch detection process including the "Mismatch Negativity" and the N400.⁴ However, we find many instances of ACC activation during correct trials; therefore, we believe that the competing, non-comparator theories described below are more general, and can account for more data. Furthermore, this theory is built around the assumption that fast errors were nevertheless intended to be correct; we do not know of evidence supporting this assumption.

According to the conflict theory,²¹⁻²³ the ACC monitors for the presence of conflict between two incompatible information processing streams, and upon the detection of such conflicts, calls for the prefrontal cortex to pay more attention. From this point of view, the ERN reflects the conflict between the fast erroneous response and the slower corrective response.^{7,21} Thus, on error trials, conflict immediately follows the erroneous response.^{7,21} Van Veen and Carter⁷ have argued that conflict precedes the actual

response on correct high-conflict trials in interference tasks, based on the available psychophysiological evidence. Correct high-conflict trials are frequently characterized by a small, but fast activation of the incorrect response, and a slower activation of the correct response (e.g.¹⁷); thus, we have argued that during correct trials, the conflict between the initial incorrect activation and the overriding correct response takes place before the correct response. We furthermore proposed that this conflict is reflected in the frontocentral N2, as it is similar to the ERN,¹⁷ and can be modeled by an ACC-based generator comparable to that of the ERN.^{7,24} Consistently, fMRI studies have shown that the ACC activation typically observed on high-conflict trials is comparable to error-related ACC activation (e.g.^{9,10}). Thus, conflict occurs prior to the response on correct, high-conflict trials, but follows the response on errors trials.⁷

As this theory is based on rather simple computational models, the predictions of this theory are straightforward,^{21,25,26} and as it has turned out, the conflict theory can account for a large amount of both ERP and fMRI data, including the inconsistent results about the relationship between the amplitude of the ERN and the magnitude of the incorrect response.²⁶ However, this theory has trouble accounting for certain findings. Specifically, psychopharmacological studies have consistently found that, with the exception of lorazepam,²⁷ various substances alter the ERN while not affecting the N2.²⁷⁻³¹

The reinforcement learning theory presented by Holroyd and Coles³² grew out of the comparator theory. Holroyd and Coles propose that behavior is monitored by a basal ganglia-based “adaptive critic,” that determines whether events are better or worse than expected, and signals this with a phasic increase or a decrease, respectively, in dopaminergic activity in the ACC. According to this proposal, the function of the ACC is to select between different cognitive processes competing for access to the motor system. The ERN is assumed to be generated because the inhibitory influence of the dopaminergic innervation in the ACC is briefly disrupted, fine-tuning the ACC to do a more appropriate selection job on future trials.

A critical assumption that Holroyd and Coles³² make is based on findings involving an ERP component somewhat resembling the ERN (or N2), which appears to be elicited by error feedback stimuli and stimuli indicating loss or punishment. Several studies have modeled this component as having an ACC-based generator,^{33,34} and Holroyd and Coles therefore assume that this error feedback negativity is functionally equivalent to the ERN. In addition, both components are modified by reward-based learning; in a task in which participants had to learn the correct stimulus-response mapping by processing feedback stimuli, both components were observed to behave more-or-less as Holroyd and Coles’ computational model predicted.³² The

model is furthermore consistent with psychopharmacological manipulations³¹ and with single cell recordings from nonhuman primates.³⁵

However, several fMRI studies have failed to find significant ACC engagement to error feedback stimuli^{36,37} despite the fact that these studies employed a task that had previously been shown to elicit a reliable error feedback negativity during ERPs.³³ These null findings have shed some doubts on the plausibility of dipole models that have estimated the generator of the error feedback negativity to lie in the ACC. Thus, despite the fact that the error feedback-related negativity appears to behave like a reliable psychophysiological indicator of feedback valence, there does not — at this time — appear to be sufficient evidence for the assumption that this component has the same functional significance as the response-locked ERN. More research is therefore needed to substantiate this assumption.

The error positivity

Subsequent to the ERN is a component that has been referred to as error positivity (PE), the significance of which has eluded researchers so far. After discarding the hypotheses that the PE might reflect error correction, a delayed parietal P3, or an inactivation or reset of the ERN, Falkenstein et al¹⁴ proposed that this component reflects “further processing” of the error. They suggested three such possibilities: the PE could be involved in adjustment of response strategies after an error, subjective/emotional assessment of errors, or conscious error recognition.

We see problems with all three of these proposals. First, the possibility that the PE is related to post-error slowing has received some support,^{20,38} but not every study has reported this relationship, instead finding a relationship between post-error slowing and the ERN amplitude.¹¹ Second, inconsistent results have also been found with regards to the relationship between the PE and negative affect. Some research supports the notion; for instance, it has been found that the PE amplitude appears to be positively related to skin conductance.²⁰ Other data is inconsistent; while Falkenstein et al¹⁴ found that participants who cared less about their errors had smaller PEs, it has also been found that it appears to be inversely related to negative affect.³⁹ Third, there are very few data to evaluate the notion that the PE is related to conscious error detection. Interestingly, in an anti-saccade task, in which people occasionally make an error without being conscious of doing so, it has been found that unconsciously made errors elicit a smaller PE, while the ERN did not differ much between consciously and unconsciously made errors.³⁸ In that study, post-error slowing was only observed following consciously made errors. This study is consistent with the notion that the PE is related to conscious error recognition. However, this notion appears to assume that “consciousness” is a distinct cognitive structure. We find this assumption problematic; we tend to view consciousness as per-

haps a property of certain cognitive processes, but the notion that it is a separate process (cognitive or neural) is, we believe, homuncular.

Thus, the functional significance of the PE remains somewhat of a mystery. It is possible that multiple neural/cognitive processes underlie this component, which can be varied by experimental manipulations in different ways. Much more research is needed to evaluate this component and its interpretation; studies that have managed to vary this component within-subject, in a parametric fashion, such as has been done with the ERN (e.g.⁵), are still missing.

Psychopharmacological manipulations

An interesting line of research involves the effects of various psychopharmacological agents on error-related brain activity. Consistent effects on the ERN have been found for noradrenergic, dopaminergic, and GABA-ergic drugs. For instance, haloperidol, a dopamine antagonist, reduces the ERN compared to a placebo, however, it does not appear to reduce post-error slowing.³¹ Caffeine, on the other hand, increases the ERN.⁴⁰ Riba et al³⁰ found that yohimbine, an epinephrine antagonist which stimulates the locus coeruleus and norepinephrine release at its synaptic terminals, increased the ERN amplitude. While yohimbine did not affect post-error slowing, it reduced the number of errors that immediately followed an error. Alprazolam, a benzodiazepine, reduces ERN amplitude but does not affect post-error slowing.²⁹ De Bruijn et al²⁷ administered either D-amphetamine (an indirect dopamine agonist), lorazepam (a benzodiazepine), the antidepressant mirtazapine (possibly a histamine and serotonin antagonist) in an Eriksen task,⁴¹ and found that compared to a placebo, D-amphetamine increased the ERN, lorazepam decreased it, while mirtazapine had no significant effect. None of the drugs affected post-error slowing. However, analysis of another measure of post-error adjustments used by De Bruijn et al,²⁷ namely, the interference effect (incongruent-congruent in the Eriksen task), which is also reduced after errors,²⁸ suggested that post-error control adjustments were impaired following lorazepam.²⁷ Finally, it has been found that alcohol reduces both the ERN amplitude and post-error slowing compared to a placebo.²⁸

In contrast, only a few studies have investigated the effects of psychopharmacological agents on the PE. Specifically, these studies have found that the PE is increased by caffeine⁴⁰ and alprazolam,²⁹ but unaffected by yohimbine.³⁰ Thus, again, more research is needed to investigate what drives the PE.

In sum, the ERN is consistently modulated by noradrenergic and dopaminergic modulation; agonists increase its amplitude, antagonists decrease it. Furthermore, GABA agonists including alcohol and benzodiazepines reduce its amplitude. Curiously, post-error slowing is hardly affected by such drugs. Furthermore, it is unknown why caffeine

and alprazolam, which have opposite effects on the ERN, affect the PE in similar ways.

Mental illness

Disturbances in the neural and behavioral response to errors have been found in several clinical populations. This suggests that at least part of the symptomatology and the impaired executive control in various mental illnesses can be understood as arising from disturbances in action monitoring. For instance, several investigators have reported a reduction in ERN amplitude in schizophrenia patients.^{42,43} Rist and Kopp⁴² also note a reduction of the N2 in this illness (note that these authors refer to this component as NE). Consistently, fMRI studies have found decreased ACC activation to error trials and high conflict trials.⁴⁴ Moreover, people with schizophrenia tend to show reduced post-error slowing,⁴⁴ suggesting impaired performance monitoring in people with schizophrenia.

Attention Deficit Hyperactivity Disorder (ADHD), also characterized as a disorder of executive functioning, is also characterized by a reduced ERN⁴⁵ and reduced post-error slowing.⁴⁶

In contrast, an enhanced ERN has been observed in Obsessive-Compulsive Disorder,^{47,48} and in Tourette Syndrome, which has a high comorbidity with OCD.⁴⁹ Consistently, Ursu et al⁵⁰ found increased error- and conflict related activation of the ACC in people suffering from this illness. These findings thus suggest that OCD might be characterized by an overactive action monitoring system. Curiously, however, OCD patients do not appear to show excessive post-error slowing.⁴⁸

CONCLUSIONS

In sum, a large amount of evidence points to the ERN and PE as being related to the control of action. These components are typically thought of as indices of performance monitoring, and related to the control adjustments observed after people make an error. However, the exact nature of the relationship between the ERN, PE, their neural sources, and post-error slowing needs to be investigated further, since the present body of data is inconsistent in this regard. Why, for instance, is post-error slowing sometimes related to the ERN and sometimes to the PE? What is the relationship between affect and these components? And how is it that certain drugs can affect the ERN and PE amplitude but not affect post-error slowing? Given the tight link between error detection and correction on the one hand, and cognitive control on the other, we believe that understanding these issues will be of great importance in understanding cognitive control and its dynamics. Furthermore, we believe that such an understanding will not only greatly improve our understanding of normal cognitive functioning, but also our understanding of those mental illnesses in which performance monitoring and cognitive control are impaired.

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