Error-Related Brain Activity: Ripples in the ANS

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ABSTRACT

A two-component event-related brain potential (ERP) consisting of error-related negativity (ERN/Ne) and positivity (Pe) has been associated with response monitoring and error-detection. The neural generator of the ERN has been localized to the anterior cingulate cortex (ACC)—a frontal structure implicated in both cognitive and affective processing, as well as autonomic nervous system (ANS) modulation. It is likely that the neural generator of the Pe is also somewhere in the ACC. The current study sought to examine the relationships among the ERN, the Pe, two autonomic measures, and behavior.

Electroencephalogram (EEG), heart rate (HR), and skin conductance (SC) were recorded while subjects performed a two-choice reaction time task. In addition to the characteristic ERN-Pe complex, errors were associated with larger SCRs and greater HR deceleration. The ERN correlated with the number of errors, but was unrelated to ANS activity and compensatory behavior. Pe, on the other hand, was correlated significantly with ANS activity, and like SCR and HR deceleration, was also significantly correlated with compensatory post-error slowing.

INTRODUCTION

Error-Related Brain Activity and the Anterior Cingulate Cortex (ACC):

- The error-related negativity (ERN) is an event-related brain potential (ERP) associated with response monitoring and error detection (Gehring, Goss, Coles, Meyer, & Donchin, 1993).
- ERN observed as a sharp negative deflection which generally occurs 50-150 ms following an erroneous response and is maximal over frontocentral recording sites (Fz-Cz) (Luu, Collins, & Tucker, 2000). The ERN has been localized to the ventral 'affective' subdivision of the ACC (Kiehl et al., 2000).
- ERN thought to reflect the activity of an generic response monitoring/error-processing system, active across stimulus and response modalities (Holroyd et al., 1998; Bernstein et al., 1995).
- Motivational and affective factors have been found to influence ERN magnitude, but ERN does not consistently relate to compensatory post-error measures.
- As such, the precise functional role of the ERN in response monitoring is unknown.
- The error positivity (Pe) is a positive component which immediately follows the ERN and peaks between 200-400 ms after an erroneous response.
- The Pe is thought to reflect response monitoring processes that occur after error-detection, and has been related to the subjective awareness of having made a mistake (Neiwenhuis et al., 2001).

The ACC and Autonomic Nervous System (ANS) Changes

- FMRI and PET studies have found that ANS activity is preceded by activity in the ventral ACC (Critchley et al., 2000).
- Ventral ACC activation, therefore, appears to be related both to visceral arousal and response monitoring processes.
- This relationship has been observed in heart rate slowing following negative feedback during the Wisconsin Cart Sorting Test (Somsen et al., 2000).
- It stands to reason, the, that ANS measures might be sensitive to internal processes associated with response monitoring, and that this activity might be related to the ERN-Pe complex—especially if this complex serves as an affective signal.
- The current study sought to evaluate the relationship between ERPs, ANS activity, and post-error behavioral measures.

METHOD

Participants

- 22 Undergraduate students at the University of Delaware (10 female, 12 male).
- Subjects were paid $15.00 for their participation.

Task

- Subjects were shown three large arrows that either pointed up, to the left or to the right— arrows were presented in red or green font against a black background.
- Subjects were instructed to press the right or left CTRL key in response to the color of the arrows and to ignore the direction of the arrows.
- 12 blocks of 48 trials presented to the subject (576 total trials). Each block initiated by subject, optional breaks between trial blocks.
- Stimulus duration = 200 ms.; interstimulus interval = 5300 - 5700 ms.

Psychophysiological Recording, Data Reduction and Analysis

All Biosignals

- Digitized using VPM software (Cook 1999).
- Data collection began 1500 ms prior to stimulus presentation and continued for an additional 3500 ms.
- Evaluated for two sets of data. 1) Error trials vs. *all* correct trials; 2) Error trials vs. *reaction-time matched* correct trials.

HR

- HR obtained by attaching a Grass Photoelectric Transducer Model PPS to the subject's left ear lobe.
- Photocell output was fed into a Grass Model 7P1 Low Level DC Preamplifier and Model 7D Driver Amplifier (Bandpass = 1.6 - 3.0 Hz) and then into a series of Coulbourn logic modules that did threshold detection. Interbeat intervals (IBI) computed to nearest ms by VPM.
- IBIs converted to HR for each 0.25 s offline as recommended by Graham (1978).
- HR waveforms generated by deviating quarter-second averages during a 3.0 s post-response epoch from the quarter-second average immediately preceding stimulus onset. Twelve (3 s) quarter-second averages, along with the onset point, constituted the heart-rate data that were then submitted to statistical analysis.

SCR

- Skin conductance responses were recorded using a Coulbourn Model S21-22 constant voltage (.5V) skin conductance coupler.
- Med Associates Standard (0.5 cm^2) Ag/AgCl electrodes were placed on the thenar and hypothenar eminence of the palm with Johnson & Johnson KY Jelly as the electrolyte.
- The epoch was digitized at 50 cps and quantified by identifying activity that began with an onset latency greater than 0.5 s post response and measuring the difference between the identified onset point and the maximum SC value present in the 3.0 s post-response window.

EEG

- EEG recordings taken from Fz, Pz, A1, A2 referenced to Cz using an ECI electrocap.
- EEG and vertical EOG recorded using a Grass Model 7D polygraph with Grass Model 7P1F preamplifiers (bandpass=0.05-35 Hz).
- EEG sampled at 200 Hz and corrected for EOG artifacts offline (Gratton et al., 1983; Miller et al., 1998).
- EEG data re-referenced to the average activity of the mastoid electrodes.
- Trials were rejected for excessive artifact, or if the reaction time fell out of a 200 -800 ms window.
- Response-locked ERPs extracted from EEG and averaged across trial type (correct and error trials).
- ERN defined as the most negative point occurring in a window from 0 to 150 ms post-response.
- Pe defined as the most positive point occurring from after the ERN to 525 ms post-response.

RESULTS

ERN—All trials

- Response-locked average waveforms are presented in Figure 1.
- Figure 1 shows a sharp negative deflection at frontal recording sites that peaked approximately 55 ms post-response associated with the commission of errors.
- The Pe was observed as a positive deflection that peaked approximately 300 ms after the commission of a mistake.
- A 2 (Trial Type) X 3 (Electrode Site) repeated measures analysis of variance (ANOVA) with Greenhouse-Geisser adjusted p-values confirmed that:
  - ERN was greater when subjects made mistakes ($F1, 20)=24.4$, $p<.001$).
  - ERN had a frontocentral scalp distribution ($F(2,40)=5.28$, $p<.01$).
  - Pe was greater when subjects made mistakes ($F(1,20)=45.90$, $p<.001$).
  - Pe was greatest at centroparietal sites ($F(2,40)=45.90$, $p<.001$).

SCR—All trials

- Response-locked data is presented in Figure 2 (right).
- Repeated measures ANOVA confirmed that only error trials were associated with a substantial SCR ($F(1,19)=18.93$, $p<.001$).

HR—All trials

- Response-locked data is presented in Figure 2 (left).
- Repeated measures ANOVA confirmed the impression that HR slowing was associated with errors ($F(1,17)=9.76$, $p<.01$).

Biosignals—RT matched trials

- No new information was obtained when errors were compared to a subset of RT-matched correct trials.

Performance Measures

- Mean reaction times were faster for error trials (434.6 ms), relative to correct trials (464.6 ms; $F(1,20)=15.74$, $p<.001$).
- Error trials were associated with slower RTs on subsequent trials (433.9 ms vs. 490 ms; $F(1,20)=36.9$, $p<.001$).
- Because it appeared that some regression was accounting for post-error slowing, trials that followed RT-matched correct trials were also evaluated.
- Trials that followed RT-matched correct trials were somewhat slower (433.9 ms vs. 441.4 ms), but this small difference was not significant ($F(1,20)=1.11$, $p>.30$).

Relationship Among Measures

- Bivariate correlations were computed between physiological responses and performance measures associated with errors; results are presented in Table 1.
- ERN correlated with number of errors, but not with Pe, HR deceleration, or SCR. Specifically, more errors were associated with smaller ERNs.
- Significant bivariate correlations were found among the HR, SCR and Pe measures.
- Both SCR and Pe were directly related to the RT slowdown effect.
- HR deceleration was also related to the RT slowdown effect, but the correlation did not reach statistical significance.

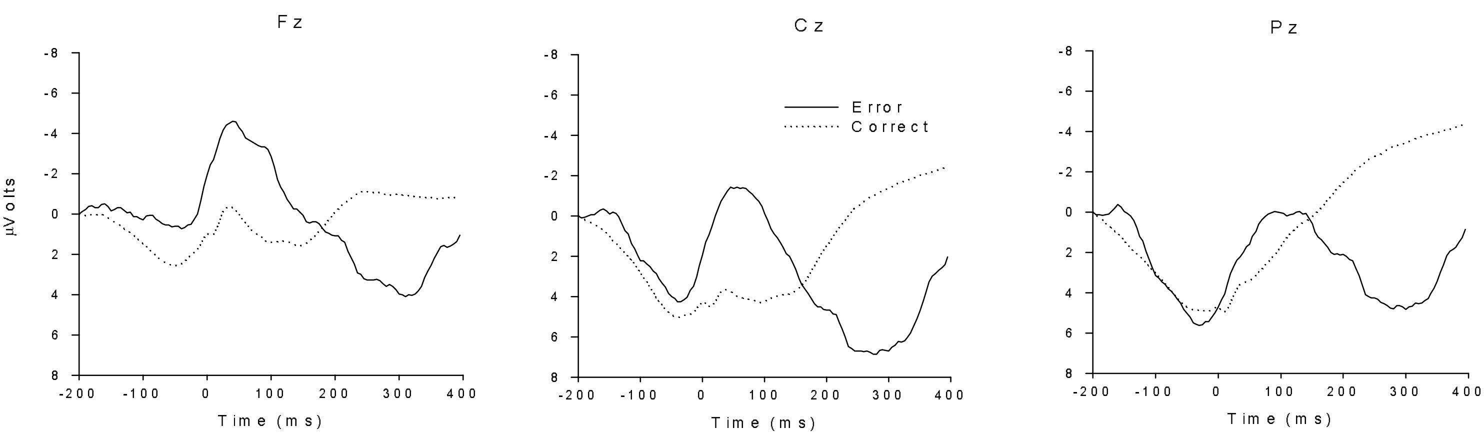

Figure 1. Averaged ERP waveforms from Fz, Cz and Pz for all error and all correct trials.

	ERN (Fz)	Pe (Fz)	HR Decel	SCR	# Errors	Slow-down
ERN (Fz)		.240	-.317	-201	.605**	-.209
Pe (Fz)			.154	.419^{+}	.025	.442*
HR Decel				.628**	-.356	.436^{+}
SCR					-.280	.760**
# Errors						-.373

$^{+}$ p<.10 * p<.05 ** p<.01

Table 1. Correlation matrix of primary error -related measures

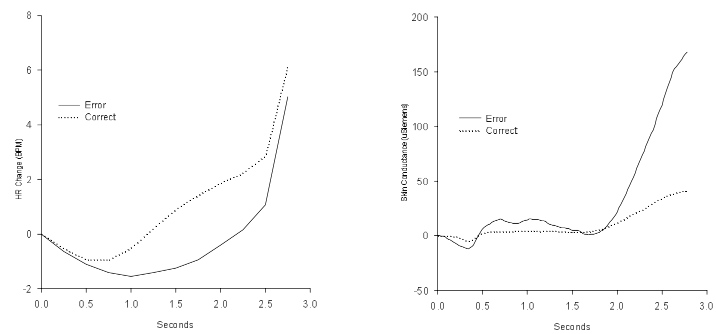

Figure 2. Averaged heart -rate (left) and skin-conductance response (right) waveforms for all error and all correct trials.

DISCUSSION

ERN

- Consistent with previous studies, a frontocentrally maximal negative deflection in the ERP was associated with the commission of errors.
- This negative component was followed by a centroparietal positivity, together comprising the typical ERN-Pe complex.
- Although the precise nature of the ERN is unknown, it has been related to conflict processing and/or error-detection processes.
- Both theories predict that ERN should be related to post-error behavioral measures, such as RT slowing (Gehring & Fencsik, 2001).
- However, consistent with the ERN literature, the present study did not find a relationship between ERN magnitude and post-error behavioral measures, suggesting that the ERN may not be related to compensatory processes following mistakes .
- We did find an inverse relationship between ERN magnitude and number of errors—consistent with an interpretation of the ERN as a measure of response control, perhaps sensitive to impulsive responding.
- Although this interpretation is attractive, this relationship may reflect simple habituation. The error rate in the present study was too low to track ERN amplitude across the testing session.

PE

- Pe magnitude was significantly related to post-error slowing. Specifically, error trials with larger Pe were associated with more post-error slowing.
- This is consistent with a Nieuwenhuis et al. (2001) study, in which unperceived errors were associated with both reduced Pe and a lack of post-error slowing.

ANS MEASURES

- Our original hypothesis that ANS activity might reflect the response monitoring process was confirmed.
- In particular, significant cardiac deceleration and SC activity was associated with errors.
- This is consistent with a Somsen et al. (2000) study in which negative feedback prompted heart rate slowing, and extends those results to include error-related bradycardia related to internal processes involved in response monitoring.
- Error-related SCR was significantly correlated with both error-related HR deceleration and with Pe magnitude.
- Although the ERN and Pe share a close temporal relationship, the Pe may be functionally more related to error-related ANS components.
- The relationship between Pe and error-related ANS measures is particularly evident when post-error compensatory slowing is examined:Pe, HR deceleration, and SCR were all more closely related to RT slowing than the ERN.

ERPS, ANS & BEHAVIOR

- Error processing typically focuses only on errors and on subsequent trials—reflecting error-detection and error-compensation, respectively.
- The current study lends support to the view that the ERN is a neural signal that is sensitive to early error-detection processes.
- Like Nieuwenhuis et al. (2001), we suggest that the Pe is a neural signal related to subsequent processes such as error awareness and compensation.
- The present data suggest, going beyond Nieuwenhuis et al. (2001), that error awareness involves an additional critical component: visceral experience. In short, when subjects make mistakes, they not only 'know' it, but they 'feel' it as well.
- Our data also suggest that this error-related ANS activity may be related to error awareness and to error compensation as well.
- In fact, the best correlate of post-error slowing was with error-related SCR.
- This is consistent with Damasio's (1996) argument that emotion plays a crucial role in what appear to be rational processes.
- However, in traditional ERN-type procedures, compensatory behavior following an error takes place within a few hundred milliseconds, and therefore, can not be dependent on the perception of a fully developed ANS response.
- It is more likely, therefore, that ANS activity is not a precursor to compensatory behavior, but rather reflects the fact that compensatory processes have been put into place.
- Perhaps, as Damasio suggests, rational processes may use this somatic information through an 'as-if' signal. That is, brain processes that generate the information leading to ANS output might supply, in parallel, a signal that is used to initiate compensatory processes.
- The Pe may function as this kind of dual-purpose signal serving to trigger error-related ANS activity and to signal more frontal areas that behavioral compensation is required.

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