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## Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing

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### Abstract

We compared individual differences in the ERP associated with incorrect responses in a discrimination task with other ERP components associated with attentional control and stimulus discrimination (N2, P3, CNV). Trials with errors that are detected by the subject normally produce a negativity ( $N_E$ ) immediately following the response followed by a positivity ( $P_E$ ). The morphology of the  $N_E$  and the  $P_E$  is similar to that of the standard N2–P3 complex on correct discrimination trials. Our findings suggest that the  $P_E$  is a P3 response to the internal detection of errors. The  $N_E$ , however, appears to be distinct from the N2. Finally, even though both the contingent negative variation (CNV) and the  $N_E$  are associated with prefrontal cortex and the allocation of attention to response accuracy, the  $N_E$  and CNV did not relate to one another. © 2001 Elsevier Science B.V. All rights reserved.

*Keywords:* Error negativity;  $N_E$ ; ERN; Error detection; ERPs; CNV; N2; P3

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## 1. Introduction

The error-negativity ( $N_E$ , or error-related negativity, ERN) is a recently documented component of the event-related potential (ERP) and has been associated with acknowledged incorrect responses that occur in target discrimination tasks (e.g. Falkenstein et al., 1991; Gehring et al., 1993; Dehaene et al., 1994; Scheffers et al., 1996). The error waveform is time-locked to the behavioral response, as opposed to stimulus onset, and consists of a negative deflection ( $N_E$ ) followed by a positive deflection ( $P_E$ ). The  $N_E$  has engendered much interest because examining the parameters within which it occurs may enrich our understanding of the processes involved in the monitoring and evaluation of response tendencies (Gehring et al., 1993; Luu et al., 2000). However, both the  $N_E$  and the  $P_E$  share functional and morphological similarities with other ERP components. The purpose of this paper is to compare individual differences in the  $N_E$ – $P_E$  with other ERP components associated with attentional control and stimulus discrimination, specifically the contingent negative variation (CNV) and the N2–P3 complex.

### 1.1. Error-negativity ( $N_E$ )

The  $N_E$  is time-locked to the execution of an incorrect response, is absent for trials on which the correct response is made when the subject is certain of the correctness of that response (Coles et al., 2001), and does not seem to be dependent on the type of error made. Scheffers et al. (1996), using a simple Go/NoGo task, found that both errors of choice (incorrect responses on go-trials) and errors of action (uninhibited responses on NoGo trials) were associated with an  $N_E$  of similar morphology, latency, and scalp distribution. Tasks used to elicit the  $N_E$  are usually not difficult (e.g. the flanker task of Eriksen and Eriksen, 1974; the letter discrimination task of Falkenstein et al., 1991) so that errors are usually caused by quick or impulsive responding rather than by the inability of the individual to discriminate the stimuli or choose the correct response.

On tasks in which individuals are unable to determine correct or incorrect responses on their own, an  $N_E$  occurs only when error-feedback is provided. Miltner et al. (1997), presented feedback 600 ms following behavioral response and found that an  $N_E$  was produced only when feedback indicated that an incorrect response had been made. These data indicate that the elicitation of the  $N_E$  is not dependent on whether the detection of the error is internally driven or signalled by external cues, typically as long as there is awareness that an error has occurred (Miltner et al., 1997). In addition to its error detection role,  $N_E$  has been associated with the magnitude of individuals' response to their own error, as well as with error correction and compensation mechanisms (Gehring et al., 1993). In fact, we have found the  $N_E$  to be related to individual differences in the impulsivity of response style on the task (Pailing et al., 1999). However, there is still some question as to whether the  $N_E$  is more directly related to the processes involved in the generation of an error signal or to the processes following it, such as emotional or remedial reactions (Bernstein et al., 1995; Stemmer et al., 2000, for evidence that the  $N_E$  follows error detection rather than being generated simultaneously with it).

It is, nonetheless, consistently observed that the  $N_E$  occurs when a mismatch results from the comparison between that which is anticipated and that which actually occurs. This observation led Falkenstein et al. (1991) to suggest that the  $N_E$  might resemble other similar negative potentials, such as the mismatch negativity (MMN; Näätänen, 1992) which reflects an automatic mismatched auditory stimulus. However, Bernstein et al. (1995) found that the  $N_E$  was influenced by response conditions and participants' response strategies, which highlight the endogenous nature of the  $N_E$  and distinguish it from the mismatch negativity, which is typically dependent on the physical characteristics of the stimuli (Coles and Rugg, 1995). It might also resemble the N400 which reflects an endogenous mismatch in the semantic (Byrne et al., 1995) or perceptual domain (Bobes et al., 2000), although this has not been examined directly.

### 1.2. Comparing $N_E$ with CNV

The first comparison we wanted to make was between the  $N_E$  and CNV. The CNV is elicited by providing the individual with a warning stimulus followed at some fixed interval such as 2000 ms by a second 'imperative' stimulus (Walter et al., 1964). In Go–NoGo versions of this task, the participant is cued as to whether or not the second stimulus requires a response. In those cases in which a response is required, a large negative potential is observed in the interval between the warning and the imperative stimuli. Behavioral measures associated with frontal lobe processing have been shown to correlate with the initial portion of the CNV (Segalowitz et al., 1992; Dywan and Segalowitz, 1996) and the CNVs of patients with unilateral prefrontal lesions are reduced (Rosahl and Knight, 1995). As well, presenting stimuli in conjunction with a high amplitude spontaneous CNV increases the likelihood of a correct behavioral response (Stamm, 1987). Similarly, the size of the CNV has been shown to correlate with performance accuracy (e.g. Stamm, 1987; Hohnsbein et al., 1998). The more negative the deflection preceding the target stimulus, the less likely the subject is to make an incorrect response. Thus, the CNV is associated with response anticipation and the  $N_E$  is associated with response monitoring, and thus both are sensitive to the production of accurate responses. In other words, the CNV and  $N_E$  are endogenous components that jointly bracket behavioral responses on tasks which require attending to imperative stimuli.

Error monitoring has been associated with the anterior cingulate, the dorsolateral prefrontal cortex, and the left premotor cortex (Carter et al., 1998). Both anterior cingulate and dorsolateral prefrontal cortex have also been associated with the control of attention (Mesulam, 1981; Posner and Petersen, 1990; Corbetta et al., 1991; Chow and Cummings, 1999; Mesulam, 1999). Physiologically, the  $N_E$  and CNV are both associated with these brain structures in the prefrontal cortex. Whereas there is some controversy concerning the specific generator site of the CNV, both magnetoencephalogram (MEG) (Basile et al., 1997; Tarkka and Basile, 1998) and intracellular recordings (Fuster, 1987) support the view that it is associated with the dorsolateral and medial prefrontal cortex. Topographical EEG mapping has shown that the response begins prefrontally and spreads posteriorly

where it reaches maximum scalp potential at the vertex (Yamamoto et al., 1986). There are however, lesion data suggesting that the CNV is associated with the cingulate gyrus and other subcortical structures (Rosahl and Knight, 1995). There is less controversy surrounding the generator site of the  $N_E$ , which has been associated with the anterior cingulate cortex (Dehaene et al., 1994; Miltner et al., 1997; Dikman and Allen, 2000; Stemmer et al., 2000).

Given this similarity in cognitive functions and anatomical source associated with error monitoring and attentional control, our goal was to examine the  $N_E$  and CNV as they occur in the same individuals during the performance of attention-demanding tasks. We hypothesized that if the amplitude of both CNV and  $N_E$  could serve as an index of attentional capacity or attentional allocation to response accuracy, then these seemingly separate components of the ERP should be related to one another.

### 1.3. Comparing $N_E$ – $P_E$ with the N2–P3 component complex

The second focus of this study is based on the observation that the  $N_E$  and the  $P_E$  have morphological features similar to other well-researched ERP components, the N2 and P3. In fact, the morphology and topography is so similar that there has been some discussion as to whether they might represent the same phenomenon (Miltner, et al., 1997; Falkenstein et al., 1999; Leuthold and Sommer, 1999). The N2 is a negative deflection that occurs at about 200 ms following stimulus onset and is maximal over the centro-frontal scalp region (Rugg et al., 1988). It is associated with stimulus discrimination (Ritter et al., 1979) and categorization (Rugg, et al., 1988). Whereas the inhibitory N2 has been compared with and shown to be independent of the  $N_E$  (Falkenstein et al., 1999), the target (non-inhibitory) N2 has not been similarly compared.

Overwhelming interest in the significance in the  $N_E$  has overshadowed the investigation of the  $P_E$  deflection. Nonetheless, the topography and polarity of the  $P_E$  has led some investigators to view it as a late P3 (e.g. Miltner et al., 1997). The P3 is a positive deflection with a latency from 300 ms following a simple stimulus to as much as 800 ms in response to complex tasks. It is characterized by a parietally maximal scalp distribution (Coles, Smid, et al., 1995) and can be elicited by several paradigms but especially when a rare target event occurs in the context of more frequent non-target stimuli (Coles and Rugg, 1995; Coles, Smid, et al., 1995). The cognitive correlates of P3 include orientation, attention, stimulus evaluation and memory, but there is a traditional controversy regarding these functional components (Ritter et al., 1979; Picton et al., 1984; Donchin and Coles, 1988; Hoffman, 1990; Coles, Smid, et al., 1995).

The  $N_E$ – $P_E$  has both overall morphological and functional similarities to the N2–P3 complex. In this context, we wished to examine whether the  $N_E$ – $P_E$  might be an N2–P3 response to the internal error-detection event. The N2–P3 component complex itself is a response to a salient stimulus (e.g. Donchin and Coles, 1988; Polich, 1993) and the detection of an incipient erroneous response could be such a salient event.

In effect, we wished to learn more about the  $N_E$  and  $P_E$  by examining them in the context of other more traditional components of the ERP responses. We accomplish this by measuring CNVs in a Go/NoGo task, and  $N_E/P_E$  in an Eriksen task and comparing these components using a correlational design.

## 2. Method

### 2.1. Participants

$N_E$  and CNV data were gathered from 14 Brock University undergraduates (13 women, one man). All were well-functioning and in good standing in their studies with no known medical conditions involving the central nervous system.

### 2.2. Procedure

The  $N_E$  was elicited by a flanker task (Eriksen and Eriksen, 1974), a visual discrimination paradigm in which half the participants pressed a computer key with their left hand when the middle letter of a five-letter array was 'H' and with their right hand when the middle letter was an 'S' (and vice-versa for the other half). Faster responses are typically recorded when the letters flanking the center are congruent, i.e. SSSS or HHHH (80 trials each), than when they are incongruent, i.e. SSHS or HSHH (160 trials each). Each array remained on the screen for 250 ms with a stimulus onset asynchrony of 1000 ms. The stimuli subtended 1.4° vertically and between 5.7 and 7.2° horizontally (H is wider than S).

The CNV was elicited in a visual Go/NoGo paradigm. Stimuli consisted of the numbers 1, 2 (warning), and 3 (imperative stimulus) which appeared one at a time on the computer screen for 250 ms with an SOA of 2000 ms and an intertrial interval of 3–7 s. The number stimuli subtended 0.35–0.95° horizontally and 1.4° vertically (1.5 cm seen from approximately 60 cm). Participants were instructed to press a key immediately on presentation of the number 3 only when the preceding number 2 appeared in green (Go trial). Participants were not to respond to the number 3, if the number 2 appeared in red (NoGo trials). Go and NoGo trials were presented in random order, each with 0.5 probability. Although a three stimulus CNV paradigm was used, only the CNV between the second and third stimuli will be reported here.

EEG was recorded from 20 electrodes of the 10–20 system, but the statistical analyses were based on frontal ( $F_z$ ), central ( $C_z$ ) and parietal ( $P_z$ ) midline sites. The topographical maps were based on a montage of all 20 electrodes on 11 of the participants. Three participants had only midline, ear and eye electrodes and could not be included in the topographical mapping. Data were sampled at a rate of 256 points per s with a bandpass of 0.02–30 Hz and signals were amplified with a hardware gain of 10 000 and a software gain of 4. Recordings were re-referenced off-line to an average of the two ears, the right mastoid served as ground, and trials with deviations greater than  $\pm 100 \mu\text{V}$  on any of the 20 EEG channels or the bipolar (outer canthus-supraorbital ridge) EOG channel were eliminated.

### 3. Results

#### 3.1. Correct and incorrect responses: $N_E$ and $P_E$

The mean error rate was 8.4% (range, 3–17%) of the 480 trials. An average of 31.3 errors were made on the 320 incongruent trials and an average of 9.0 errors were made on the 160 congruent trials. The accuracy rate was better for the trials with congruent flankers than for those with incongruent trials ( $t(13) = 3.53$ ,  $P < 0.005$ ). In addition, responses were faster for correct congruent flanker trials ( $M = 433$  ms) than for the correct trials with incongruent flankers ( $M = 463$  ms),  $t(13) = 10.28$ ,  $P < 0.0001$ , suggesting the incongruent flankers led to response interference. A negative deflection, maximal at  $C_z$ , followed incorrect but not correct responses (see Fig. 1). The  $N_E$  and  $P_E$  reported in this paper will be computed from the ERPs to incongruent error trials time-locked to response unless otherwise stated. These waveforms were essentially identical to those which included congruent error trials. The negative deflection ( $N_E$ ) relative to a  $-600$  to  $-400$  ms prerespone baseline ( $M = -10.0$   $\mu\text{V}$  at  $C_z$ ) peaked at 66.6 ms on average

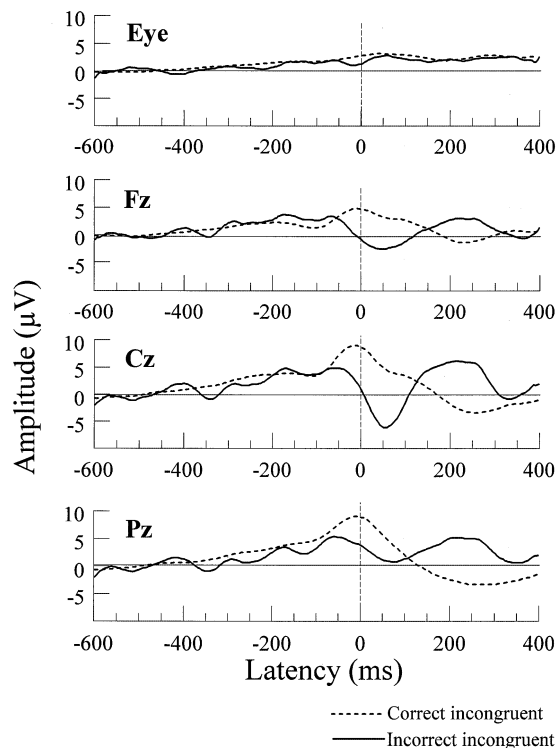


Fig. 1. Averaged ERP waveforms for correct and incorrect trials time-locked to the response ( $n = 14$ ). The group average peak amplitudes are attenuated compared with single scores due to inter-subject latency jitter. The hash marks represent the time of the response.

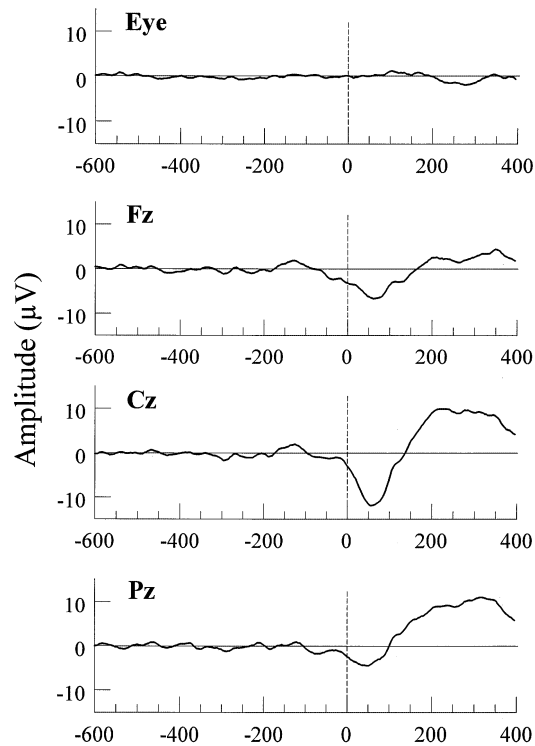


Fig. 2. The difference waveform time-locked to response (incorrect minus correct responses). The hash marks represent the time of the response.

following the response for incorrect trials and was followed by a positive peak,  $P_E$ , ( $M = 11.7 \mu\text{V}$  at  $C_z$ ) occurring about 250 ms following the response. In contrast, correct trials produced a positive peak about the time of the response followed by a negative drift peaking about 200 ms later. The dramatic distinction between correct and incorrect trial ERPs is illustrated in the difference waveform but is also seen in the original ERPs (see Fig. 1 and Fig. 2).

### 3.2. Contingent negative variation (CNV)

The CNV elicited by the Go trials was maximal at  $C_z$  ( $M = -10.34 \mu\text{V}$ , see Fig. 3). The CNV was scored time-locked to the imperative stimulus with a baseline of  $-4200$  to  $-4000$  prior to the imperative stimulus (i.e. the 200 ms before the first stimulus). As expected, the negative deflection was greater for the Go relative to the NoGo trials,  $F(1, 13) = 11.70$ ,  $P = 0.005$ , with an increased deflection leading up to the imperative stimuli (when we divide the 1400 ms CNV period into four equal epochs, there is a systematic increase in the negative deflection across epochs,  $F(3, 11) = 10.21$ ,  $P = 0.002$ ). In order to derive a more pure reflection of response expectation, we used the CNV from the Go trials with the CNV score from the NoGo trials partialled out by regression, producing a residualized CNV score.

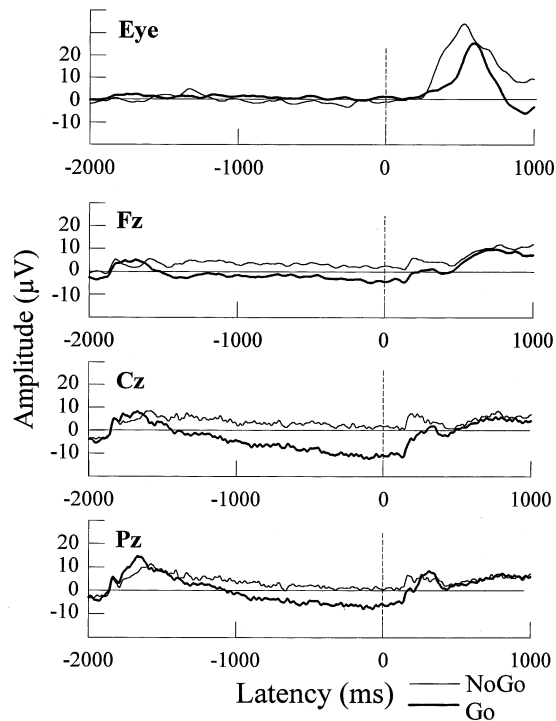


Fig. 3. Averaged waveforms for CNV. The imperative stimulus onset is indicated as zero ms. The warning stimulus onset is at  $-2000$  ms. Go trials are indicated by the thick line and NoGo trials are indicated by the thin line.

### 3.3. Relationship between $N_E$ and CNV

Correlation analyses were used to relate the amplitude of the  $N_E$  to the residualized CNV Go trials. The  $N_E$  measured at  $F_Z$  and  $C_Z$  did not correlate significantly with the CNV measured at any of the three sites. However, the  $N_E$  measured at  $P_Z$  did correlate with the CNV at  $C_Z$  and at  $P_Z$  (see Table 1). However, since the

Table 1  
Correlation of  $N_E$  amplitudes with each epoch of CNV amplitude

CNV	$N_E$		
	$F_Z$	$C_Z$	$P_Z$
$F_Z$	-0.21	0.25	0.24
$C_Z$	0.12	0.53	0.65*
$P_Z$	0.37	0.52	0.68**

The  $N_E$  was elicited during the incorrect incongruent trials time-locked to response and scored with a baseline correction of  $-600$  to  $-400$ . \*  $P < 0.05$ ; \*\*  $P < 0.01$ .



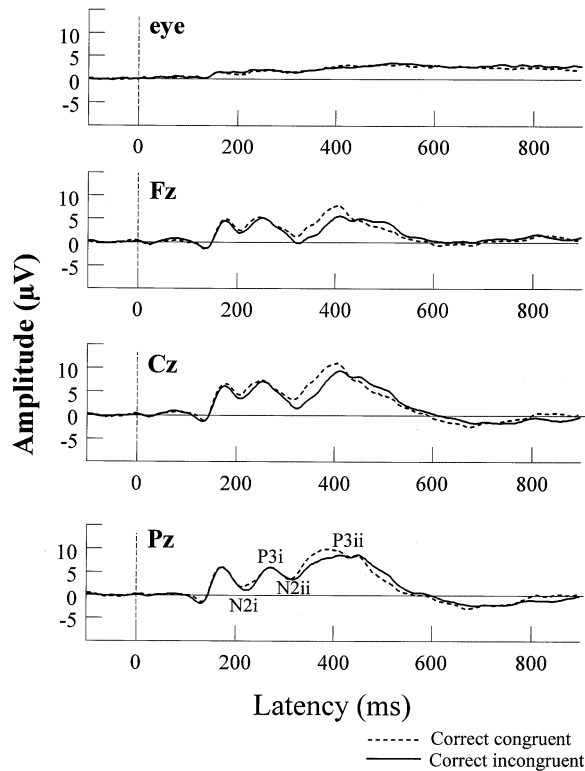


Fig. 4. ERP waveforms to correct responses time-locked to the stimulus onset. The hash marks represent the time of the stimulus onset.

degree of negative deflection of the  $N_E$  with respect to the early baseline can be influenced by the size of the abortive P3 preceding it (see Fig. 1), we recalculated these correlations using linear regression with the amplitude at  $-200$  to  $0$  ms (with respect to the  $-600$  to  $-400$  ms baseline) partialled out. The correlations remained significant (for CNV at  $C_Z$ :  $t = 2.46$ ,  $P = 0.032$ ; for CNV at  $P_Z$ ,  $t = 3.41$ ,  $P = 0.006$ ). However, the  $N_E$  is not normally measured at  $P_Z$  and indeed was only  $+0.6$   $\mu\text{V}$  (with respect to the early baseline). Thus, this correlation at  $P_Z$  does not represent strong support for a relationship between the CNV and the  $N_E$ , although this finding remains intriguing.

### 3.4. ERPs to correct responses

To examine the  $N_E$  in the context of other ERP components, the EEG waveforms elicited by trials correctly responded to in the flanker paradigm were time-locked to stimulus onset and scored using a 100 ms prestimulus baseline. This

Table 2

Correlation of  $N_E$  amplitude with ERP components measured at frontal ( $F_Z$ ), central ( $C_Z$ ) and posterior ( $P_Z$ ) midline scalp electrode sites

Site	ERP components from correct trials			
	Congruent trials		Incongruent trials	
	N2i	N2ii	N2i	N2ii
$F_Z$	−0.08	0.20	0.19	0.28
$C_Z$	0.27	0.46	0.19	0.48
$P_Z$	0.28	0.55*	0.48	0.50

ERP components were elicited by correct congruent trials on the visual flanker task and time-locked to stimulus onset. The  $N_E$  was elicited during the incorrect incongruent trials time-locked to response and scored with a baseline of −600 to −400 ms. The significance of the relationship between N2ii (congruent trials) and  $N_E$  at  $P_Z$  disappears when the P3 is partialled out (see text). \* $P < 0.05$  level.

produced six scorable deflections: N1, P2, N2i, P3i, N2ii, and P3ii (see Fig. 4)<sup>1</sup>, as has been found before with complex stimuli (e.g. Segalowitz et al., 1997). Evidently, the flanker paradigm used to elicit the  $N_E$  in this study does not produce the classic N2–P3 complex that is normally obtained in less complex oddball paradigms. Consequently, we compare the  $N_E$  to the N2i and N2ii and the  $P_E$  to the P3i and P3ii, respectively. The  $N_E$  was not significantly correlated with the N2 components at any of the fronto-central sites (see Table 2). The one significant correlation between the  $N_E$  at  $P_Z$  and the N2ii at  $P_Z$  appears to be an artifact of the P3 leading up to the  $N_E$  since it disappears when the P3 is partialled out. The  $P_E$  amplitude correlated with the P3 components elicited by correct trials at  $C_Z$  and  $P_Z$  (see Table 3).

Table 3

Correlation of the  $P_E$  with the amplitudes of ERP components at frontal ( $F_Z$ ), central ( $C_Z$ ), and posterior ( $P_Z$ ) midline electrode sites

Site	ERP components from correct trials			
	Congruent trials		Incongruent trials	
	P3i	P3ii	P3i	P3ii
$F_Z$	0.33	0.46	0.31	0.24
$C_Z$	0.66*	0.75*	0.68**	0.77**
$P_Z$	0.46	0.73**	0.72**	0.70**

ERP components were elicited during correct congruent trials of the visual flanker task and were time-locked to stimulus onset. The  $P_E$  component was elicited during the incorrect incongruent trials time-locked to response and scored with a baseline correction of −600 to −400 ms. \* $P < 0.05$ ; \*\* $P < 0.01$ .

<sup>1</sup> We have not used letters a and b to indicate the first and second peaks of the N2 and P3 because these terms have been used to reflect specific components in different contexts.

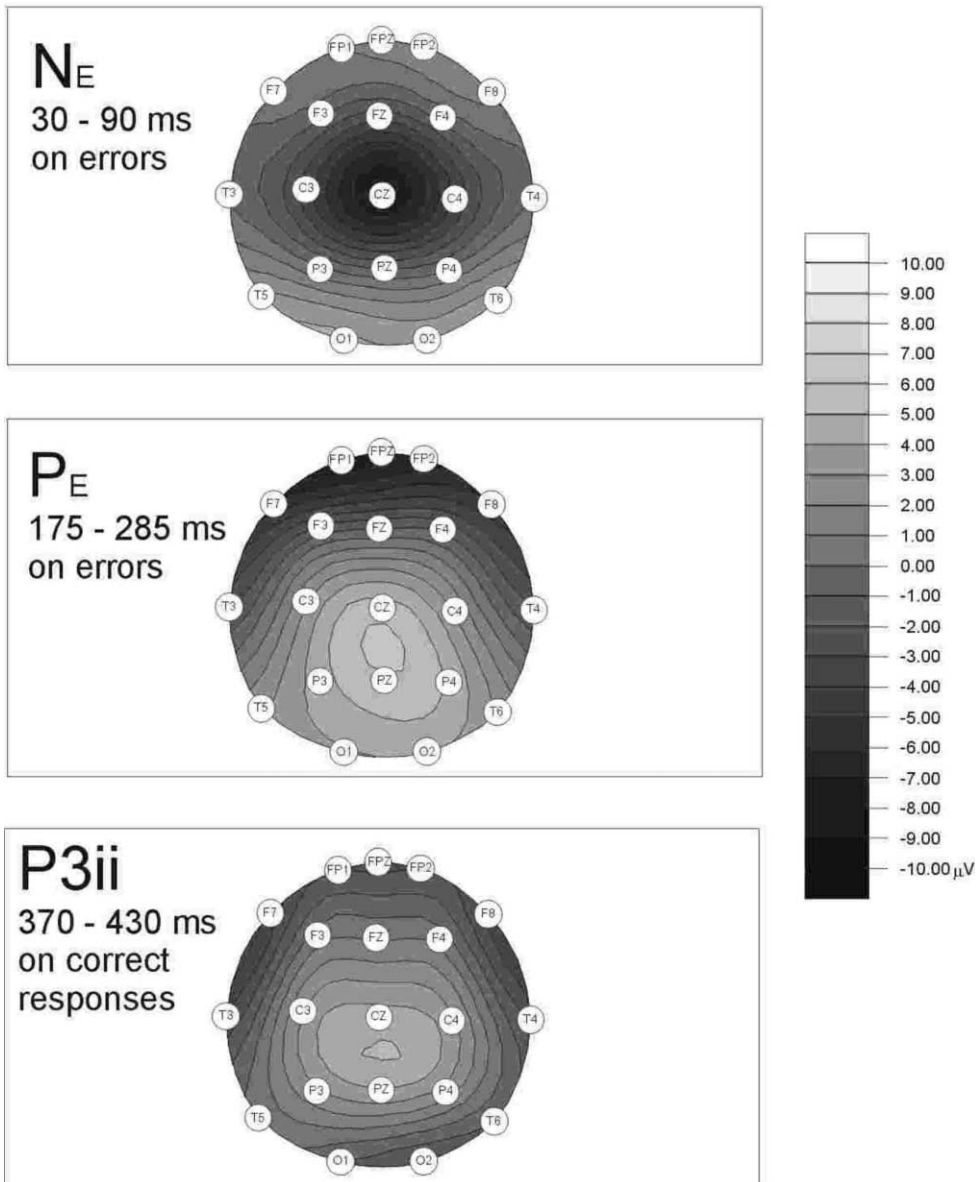


Fig. 5. Topographical maps ( $n = 11$ ) of the  $N_E$ ,  $P_E$  and the  $P3ii$  from the correct trials time-locked to stimulus onset.

### 3.5. Topographical maps

Topographical maps of the  $N_E$ , the  $P_E$ , and the P3ii of the correct trials are displayed in Fig. 5 (based on the algorithms of Junghofer et al., 1997). These maps represent the data from the 11 of the 14 subjects for whom we had a full montage. As can be seen in these maps, the  $N_E$  has a strong negativity in the centromedial frontal scalp region replicating the results of Luu et al. (2000). The  $P_E$  based on incorrect trials time-locked to response and the P3ii based on correct trials time-locked to the stimulus both display a strong positivity over the centro-parietal regions of the scalp verifying that these two components are similar (see Fig. 5).

## 4. Discussion

The detection of errors in one's own performance has been associated functionally with self-monitoring, the resolution of response conflicts, and emotional reactivity, functions associated with activation of the prefrontal cortex and especially the anterior cingulate. The ERP components associated with detected errors are a negativity immediately following the response and a later positivity, both assumed to arise in response to the person detecting an error in progress. In this study, we tested hypotheses that the error-related ERP is functionally related to other well-studied ERP components. The morphology of these ERP components — the  $N_E$  and  $P_E$  — resembles the classic N2 and P3 to salient events. The functional characteristics of error-monitoring relates to sustained attention associated with frontal activation, and manifested in the CNV waveform.

In the present experiment, the  $N_E$  did not relate straightforwardly to the CNV waveform although the CNV has been associated with attention and monitoring functions linked to prefrontal cortex (e.g. Segalowitz et al., 1992; Rosahl and Knight, 1995; Dywan and Segalowitz, 1996; Basile et al., 1997) as has the  $N_E$  (e.g. Gehring et al., 1990; Luu et al., 2000). This indicates that although both the CNV and  $N_E$  may involve prefrontal brain structures, they represent different aspects of frontally mediated neural activity. The only linkage we found required measuring the  $N_E$  at the  $P_Z$  site, where this component is weak at best, and where it is not normally measured. Therefore, there is some doubt that this posterior error-negativity really reflects the  $N_E$  component associated with the anterior cingulate cortex. We can speculate along with Falkenstein et al. (1991) that it reflects perhaps in part some negativity akin to the classic N400, a posterior component associated with mismatch decisions (Kutas and Hillyard, 1980). Thus, we conclude that the  $N_E$  and CNV reflect different cognitive and physiological processes (see also Stemmer et al., 2000 for a similar dissociation in patient data).

The lack of relation in our data between the N2 components of the stimulus-locked correct trials and the  $N_E$  confirm that the  $N_E$  is not a traditional N2 in response to the internal error-detection event, but represents a distinct neurophysiological process. These findings are similar to those reported by Falkenstein et al. (1999) with respect to the inhibitory N2. Whereas there may be a classic N2

generated to the internal event of the error detection, the  $N_E$  is not it and represents a different source of variance.

However, we did find a strong correlation between the P3 components of the stimulus-locked ERPs to correct trials and the  $P_E$ , supporting the notion that this positivity is a P3 response to the internal error-detection. These data suggest that individuals are consistent in the P3 amplitude they produce, whether it is to a salient external stimulus or to an internal error detection. Falkenstein et al. (1991) and Leuthold and Sommer (1999) compared the correct and incorrect trial ERPs, both time-locked to stimulus, and concluded that there are two P3 components in the error trials. We suggest that the  $P_E$  is functionally related to error detection and the salient stimulus is the error and therefore, the  $P_E$  emerges most clearly when the averaged EEG is time-locked to response. Thus, when making the comparison of the  $P_E$  to the P3 in correct trials, the similarities are very strong when the  $P_E$  is time-locked to the response and the P3 to correct trials is time-locked to the stimulus. The first P3 in the error trials is presumably in response to the stimulus just as in the correct trials. Thus, the two late positive deflections seen in the incorrect trials time-locked to stimulus onset are produced by separate sources, the first being to the original external stimulus (and cut short by the  $N_E$ ) and the second being to the internally generated error detection produced some short time after the ‘point of no return’, once the subject realizes that an error is being initiated.

#### 4.1. Conclusions

The purpose of this study was to examine the relationship between the  $N_E$  and the CNV, and between the  $N_E$ – $P_E$  associated with error trials and standard N2–P3 ERP components. We found that the frontocentral  $N_E$  did not relate to the CNV and suggest that the attentional and monitoring functions associated with these two ERP components represent distinct neural processes. The morphology of the negative-then-positive error-trials waveform is very similar to the morphology of the N2–P3 component complex that is prominent in the cognitive processing associated with salient events. This similarity tempts one to suggest that the  $N_E$ – $P_E$  waveform and the N2–P3 waveform are equivalent. In our study, we demonstrate that the  $P_E$  is very similar to the P3, but our data do not support the premise that the  $N_E$  is similar to the N2. These findings suggest that the  $P_E$  is most likely a P3 response to the error detection, which is an internally generated stimulus. In contrast, these current findings support the contention that the negative deflection following an error represents separate neural processes from those observed in the standard N2.

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