Available online at www.sciencedirect.com
SCIENCE DIRECT
Cognitive Brain Research 24 (2005) 317–325
COGNITIVE BRAIN RESEARCH
www.elsevier.com/locate/cogbrainres

Research report

Error processing and impulsiveness in normals: Evidence from event-related potentials

Martin Ruchsow*, Manfred Spitzer, Georg Grön, Jo Grothe, Markus Kiefer

Department of Psychiatry, University of Ulm, Leimgrubenweg 12-14, D-89075 Ulm, Germany

Accepted 7 February 2005
Available online 10 March 2005

Abstract

Electrophysiological correlates of impulsiveness were investigated in thirty-two healthy subjects using event-related potentials (ERP). Impulsiveness was determined by calculating individual reaction times (as a function of general response speed) in order to split the entire group into two subgroups with a more controlled (n = 16) and less controlled (n = 16) response style. Participants performed a Go/Nogo task while a 64 channel EEG was recorded. Artifact-free EEG segments were used to compute ERPs on correct Go trials and incorrect Nogo trials, separately. Three ERP components were of special interest: the error-related negativity (ERN)/error negativity (Ne) and the "early" error positivity (Pe) reflecting automatic error processing and the "late" error positivity (Pe) which is thought to mirror the awareness of erroneous responses. Subjects with higher impulsiveness showed smaller amplitudes than subjects with lower impulsiveness for the ERN/Ne component and the "early" Pe component. With regard to the "late" Pe groups did not differ. Hence, ERP measures appear suitable for detailed analyses of impulsiveness in healthy participants. Moreover, present results argue for the necessity of careful control of impulsiveness when including normal comparison groups in the context of clinical studies.

Theme: Neural basis of behavior
Topic: Cognition

Keywords: Impulsiveness; Event-related potential (ERP); Error processing; ERN/Ne; Pe

1. Introduction

The concept of impulsiveness has a long tradition in psychiatry [3]. Impulsiveness is a core feature of a variety of psychiatric diseases like attention-deficit/hyperactivity disorder, drug intoxication, borderline personality disorder, and antisocial personality disorder [24,31]. Pharmacological studies found close connections between impulsive personality traits and a dysfunction of the serotonergic and noradrenergic system [26]. This finding was confirmed by several positron emission tomography (PET) studies which showed hypometabolism in prefrontal cortical areas [8,27] reflecting a diminished serotonergic turnover and consecutively an impaired regulation of impulsive behavior [17].

Event-related brain potentials (ERPs) are a useful tool to investigate impulsiveness because they permit tracking the time course of fast cognitive processing on-line with a time resolution in the range of milliseconds. During the last years, special attention was paid to the error negativity (Ne; [10]) or error-related negativity (ERN; [13]), an ERP component which mirrors erroneous responding in forced choice reaction time paradigms like the Eriksen flanker task [9,19,23]. The ERN/Ne is a negative ERP deflection peaking between 100 and 150 ms after the onset of an erroneous response [25]. Larger amplitudes of the ERN/Ne were found when task instructions emphasize accuracy over speed (speed accuracy trade-off; [14]). Experimental evidence from dipole solutions of the ERN/Ne with brain electric source analysis (BESA) and from several fMRI

* Corresponding author. Fax: +49 731 50021549.
E-mail address: martin.ruchsow@medizin.uni-ulm.de (M. Ruchsow).

0926-6410/$ - see front matter © 2005 Elsevier B.V. All rights reserved.
doi:10.1016/j.cogbrainres.2005.02.003

studies (e. g. [5]) pointed to neural generators in the anterior cingulate cortex (ACC).

Originally, the ERN/Ne was considered in the context of error detection resulting from a mismatch between the representation of the correct response and the representation of the actual (false) response [10,14]. Alternative accounts view the ERN/Ne as a brain potential reflecting the response evaluation process itself rather than the outcome of this process [29]. Rather contrary to these interpretations, Cohen and coworkers interpret the ERN/Ne to be associated with the detection of response conflict [4,5].

Several studies showed that variability in the amplitude of the ERN/Ne depends on mood and personality variables. Luu, Collins, and Tucker [19] found large ERN/Ne amplitudes in college students who were high on negative affect (NA) and negative emotionality (NEM) in the beginning of an Eriksen flanker task. Moreover, a shift on response patterns was found during the experiment. By means of a post-task questionnaire parts of the subjects were reported to have been bored and dissatisfied with their performance resulting in motivational problems and disengagement from the task. When EEG data were re-analyzed for members of the high-NA and high-NEM groups with motivational problems, the amplitude of the ERN/Ne decreased. This pattern of results was strikingly different from results of participants who were low on NA and NEM. Similarly, Dikman and Allen [9] demonstrated that individuals low on socialization exhibit smaller ERN/Ne amplitudes during tasks which penalize error responses. In the same vein, Pailing and coworkers [23] found smaller ERN/Ne peak amplitudes in subjects with a tendency towards impulsive responding. Impulsivity was rated based on linear regression from correct individual reaction times on reaction times from erroneous responses. Mean RT residual scores were defined as mean difference of observed RTs (Y) minus predicted RTs ($\hat{Y}$) for error trials ($\Sigma(Y - \hat{Y})/n$). Less negative mean residual RTs were regarded as indicating a more cautious (controlled) response strategy whereas more negative residuals were interpreted to indicate a less controlled (i.e., more impulsive) response style. Furthermore, in their study, ERN/Ne latencies were positively related with percentage of errors, suggesting that individuals with shorter ERN/Ne latencies should have more opportunity to control for erroneous response tendencies [23].

Another ERP component discussed in the context of error processing is the error positivity (Pe), first described by Falkenstein and coworkers [10,11]. The Pe is a slow positive wave with centroparietal distribution which usually follows the ERN/Ne in a time window between 300 ms and 500 ms after erroneous responses. The Pe has been differentiated from the P300 by some authors [12], whereas others interpret the Pe as a P300 on the erroneous response [7]. A source localization analysis using BESA revealed that the Pe consists of two components: an "early" Pe component with probable generators in an area around the caudal ACC and a "late" Pe component with probable generators in an area around the rostral ACC. The "early" Pe has been regarded as functionally belonging to the ERN/Ne [28], whereas the "late" Pe component was associated with awareness of erroneous responses and was more pronounced for perceived than for unperceived errors [20].

In the present study, we investigated ERPs related to errors of commission (i.e., pressing a button when one is not supposed to do in a Go/Nogo task) and correct responses (i.e., pressing a button when one is supposed to do so). Errors due to delayed response ("faster" as feedback) were excluded from ERP analysis. We analyzed the relationship between amplitudes and latencies of the three error-related ERP components (ERN/Ne, "early" Pe, and "late" Pe) and two behavioral indices of response control (RT residual values and error rates). Similar to the method originally introduced by Pailing et al. [23], subjects were split into a high (henceforth: HI) and low (henceforth: LI) impulsiveness group based on individual mean RT residuals. We reasoned that individuals with high impulsiveness (more negative RT residual values) should demonstrate smaller ERN/Ne amplitudes (less negative) and smaller "early" Pe amplitudes (less positive) than individuals with a more controlled response strategy (less negative RT residual values). Besides that, we expected longer ERN/Ne latencies and "early" Pe latencies in less controlled subjects. From their finding of a positive correlation between error rates and ERN/Ne latencies, Pailing et al. [23] reasoned that subjects with faster ERN/Ne's have a more controlled response strategy as they have more opportunity to catch erroneous intentions (see also [32]). With regard to the "late" Pe component, group differences on this component should indicate differences in the awareness of errors between HI and LI subjects as has been suggested by Nieuwenhuis and coworkers [20].

2. Materials and methods

2.1. Participants

Thirty-two right-handed [21] healthy subjects (eleven males) with no history of neurological or psychiatric disorders took part in the study. After complete description of the study to the subjects, written informed consent was obtained. The study was approved by the local ethical committee and was in accordance with the Declaration of Helsinki. The entire group had a mean (SD) of 29.4 (10.9) years of age (range, 20–65) and a mean of 12.2 (1.7) years of education (range, 8–13). We calculated reaction time residuals for each subject in order to split the entire group in a high (HI) and low (LI) impulsiveness subgroup. As errors in forced choice RT tasks like the Eriksen flanker task are most likely due to slips [11] reflecting impulsive responses, we focused our analysis on RTs of error trials. Pailing et al. [23] proposed to derive impulsiveness scores from reaction times (RTs) by means of the following method. First, a

regression from correct RTs (the predictor) onto incorrect RTs (the criterion) was computed. Afterwards, mean RT residuals were calculated by subtracting predicted RTs ($\hat{Y}$) from observed RTs (Y) on error trials for each subject ($Y - \hat{Y}$). These residual values represent subjects' RTs on error trials with their general response speed (correct trial RT) statistically controlled for. Afterwards, individual mean RT residuals were averaged in order to calculate mean RT residuals ($\Sigma(Y - \hat{Y})/n$). Per definition, subjects with a more controlled response strategy showed less negative residual values whereas subjects with a more impulsive response strategy showed more negative mean RT residuals. There was a significant correlation ($r = -0.50$; $P = 0.004$) between mean RT residuals and error rates (see also Fig. 1).

The HI group consisted of sixteen subjects (five males) with a mean of 27.8 (9.2) years of age (range, 20–61) and a mean of 12.2 (1.8) years of education (range, 8–13). The LI group consisted of sixteen subjects (six male) with a mean of 31.1 (12.5) years of age (range, 20–65) and a mean of 12.1 (1.6) years of education (range, 8–13). Age and years of education were not different between groups (P values above $P > 0.400$).

2.2. Stimuli and procedure

We combined a Go/Nogo task with an Eriksen flanker paradigm. Eight different letter strings (congruent: BBBBB, DDDDD, VVVVV, and UUUUU; incongruent: BBDBB, DDBDD, UUVUU, and VVUVV) were presented on a computer screen in randomized order. Subjects had to focus on the target letter in the middle of an array and had to press a right response key upon appearance of letters B and U (Go condition) and to withhold key press upon appearance of D and V (Nogo condition). The whole experiment consisted of 5 blocks with 120 trials each (300 Go trials; 300 Nogo trials). The four incongruent letter strings BBDBB, DDBDD, UUVUU, and VVUVV were presented 120 times each, whereas the four congruent letter strings BBBBB, DDDDD, VVVVV, and UUUUU were presented 30 times, each with a presentation time of 400 ms. As it is known from previous studies that errors are more frequent for incongruent letter strings than for congruent letter strings [23], we presented incongruent letter strings more often in order to sufficiently increase error rates in our study.

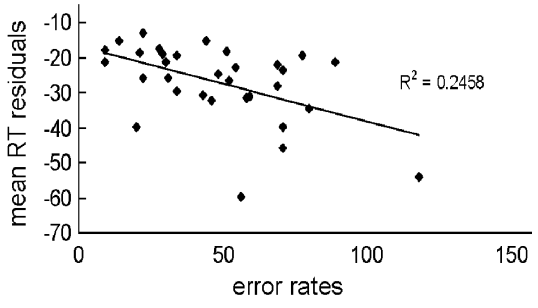

Fig. 1. Scatterplot of mean RT residuals and error rates.

Subjects got feedback according to their performance 750 ms after key press. As feedback stimuli, we used the German expressions for "correct" and "false". If subjects missed the RT deadline, the feedback "faster" was presented. Feedback stimuli were presented for another 500 ms. Intertrial interval was 2600 ms. Participants got a monetary reward, winning or losing a small amount of money each trial (five Euro-cent). However, instruction emphasized speed over accuracy. Before recording the EEG, subjects had a training period of 120–240 trials. RT deadlines were calculated individually by averaging RTs of the training periods and subtracting minus 10% afterwards. Time windows ranged between 250 and 400 ms. Participants were seated in a comfortable chair in a sound-attenuating, electrically shielded booth. The whole experiment lasted about 2.5 h, including pauses, electrode placement, and removal.

2.3. EEG recording and analysis

EEG was recorded using 64 channels mounted in an elastic cap (Easy-cap® system). Electrodes were positioned with equal distances. All electrodes were referenced to an electrode between Cz and FCz, and re-referenced to average reference off-line. Eye movements were registered by vertical and horizontal EOG. Electrode impedances were kept below 5 kΩ. The EEG was amplified by Neuroscan amplifiers (bandwidth DC-50 Hz; 50 Hz notch filter) and A/D converted with 12-bit resolution at a rate of 250 Hz and digitally low-pass filtered with 16 Hz and digitally high-pass filtered with 0.10 Hz. The EEG was baseline corrected to an interval between −200 ms and 0 ms before the onset of the key press. Ocular artifacts were corrected by using a method proposed by Gratton, Coles, and Donchin [16]. EEG segments of 750 ms were used to compute ERPs to correct Go trials and incorrect Nogo trials separately.

For statistical analysis, electrodes were selected from two different scalp regions: a central electrode group included midline electrodes FCz and Cz and the lateral pair of electrodes C1/C2. For these electrode positions, an ERN/Ne component and an "early" Pe component were expected as shown previously (e.g., [10]). A parietal electrode group included midline electrodes CPz and Pz and the lateral pair of electrodes P1/P2. For these electrode positions, a "late" Pe component was expected as shown previously (e.g., [12]).

We based our analyses both on averaged waveforms for correct and incorrect trials and difference waveforms (incorrect minus correct trials). Both methods have their advantages and disadvantages (for an extensive discussion, see [23]). For the ERN/Ne and "early" Pe component, we performed two peak analyses in a time window ranging between 0 ms and 250 ms, one for the ERN/Ne peak and one for the "early" Pe peak. As the "late" Pe component has a broad plateau-like shape, we calculated mean voltages instead of peaks in a time window between 250 and 750

ms (presentation of the feedback stimuli). Correlational analyses were performed to assess relationships between error-related ERP components (peak or mean amplitudes from difference waves), mean RT residuals, error rates, and age.

Voltages at central midline, central lateral, parietal midline, and parietal lateral electrodes were analyzed by means of separate repeated-measures analyses of variance (ANOVAs) with the between-subjects factor 'group' (HI, LI) and two within-subjects factors 'electrode position' (left–right or anterior–posterior) and 'condition' (correct, error). ANOVAs on data from difference waves included the factors 'group' and 'electrode position'. Where appropriate, differences from conditions, groups, or condition-by-group interactions were further evaluated with Fisher LSD post hoc tests (nominal level of alpha: $P < 0.05$).

3. Results

3.1. Behavioral data

Given the task, only false positive responses on Nogo trials were of interest (commission errors). Consequently, error rates were individually calculated as number of false positive reactions during Nogo trials. For HI subjects, mean number of errors was 54.9 (SD: 24.4), corresponding to an error rate of 18.3%. LI subjects demonstrated a mean number of 40.8 errors (SD: 24.4), corresponding to an error rate of 13.6%. An ANOVA on the mean number of correct and incorrect trials including the factors 'group' (HI, LI) and 'condition' (correct, error) revealed an effect of correctness ($F(1,30) = 102.66$, $P < 0.001$) but no interaction of group by correctness ($F(1,30) = 2.70$, $P = 0.111$), demonstrating no group differences in terms of error rates. Also, there were no significant group differences with regard to the number of trials where "faster" was presented as feedback whenever subjects extended the RT deadlines ($t(30) = 1.78$, $P = 0.084$).

Considering mean RT of the entire group, incorrect Nogo trials were significantly faster than correct Go trials ($t(31) = -3.40$; $P < 0.002$). Within groups, mean reaction time (RT) was 228.4 ms (SD: 26.4) for correct Go trials and 221.9 ms (SD: 31.2) for incorrect Nogo trials in HI subjects. In LI subjects, mean RT was 231.8 ms (SD: 24.7) for correct Go trials and 222.5 ms (SD: 26.5) for incorrect Nogo trials. An ANOVA on mean RT data including the factors 'group' (HI, LI) and 'condition' (correct, error) revealed an effect of correctness ($F(1,30) = 11.28$, $P < 0.001$) but no interaction of group by correctness ($F(1,30) = 0.33$, $P = 0.569$). LI subjects were significantly faster on error trials than correct trials (mean difference: 9.3 ms; $t(15) = -3.33$; $P < 0.005$). HI subjects were faster on error trials than correct trials, too (mean difference: 6.6 ms). However, this difference was not significant ($t(15) = -1.72$; $P = 0.105$), most likely due to increased standard deviations of RT data in the HI group.

3.2. Event-related potentials

To control whether the number of analyzed ERP data was not unbalanced between groups due to control of artifacts, the number of analyzable segments were calculated per each group. HI subjects had 112.8 (SD: 38.3) analyzable segments of correct Go trials and 42.1 (SD: 24.5) analyzable segments of incorrect Nogo trials. For LI subjects, 134.6 (SD: 25.3) segments of correct Go trials were analyzable and 28.9 (SD: 19.8) of incorrect Nogo trials. There were no group differences with respect to the amount of analyzable segments (all P values above $P > 0.103$).

3.2.1. Averaged waveforms for correct and incorrect trials

3.2.1.1. Voltages. Significant group differences for the ERN/Ne component (time window: 0–250 ms) were locally constrained at central electrode positions (FCz, Cz, C1, and C2). For both central midline and central lateral electrodes, we found a significant condition effect (central midline: ($F(1,30) = 131.93$, $P < 0.001$); central lateral: ($F(1,30) = 163.90$, $P < 0.001$)) and a significant interaction of group by condition (central midline: ($F(1,30) = 7.77$, $P < 0.009$); central lateral: ($F(1,30) = 7.40$, $P < 0.011$)). Post hoc tests revealed that voltages in the error condition were significantly more negative in the LI group compared to the HI group (all P values below $P < 0.046$), whereas voltages in the correct condition did not differ between groups (all P values above $P > 0.489$). At parietal electrode positions (CPz, Pz, P1, P2), there was a significant effect of condition for parietal midline ($F(1,30) = 211.41$, $P < 0.001$) and parietal lateral electrodes ($F(1,30) = 198.14$, $P < 0.001$). However, the interaction of group by condition was not significant at these electrode positions (all P values above $P > 0.100$).

With regard to the "early" Pe component (time window: 0–250 ms), we found a significant condition effect for central electrodes (central midline: ($F(1,30) = 287.54$, $P < 0.001$); central lateral: ($F(1,30) = 328.51$, $P < 0.001$)) and a significant interaction of group by condition (central midline: ($F(1,30) = 13.84$, $P < 0.001$); central lateral: ($F(1,30) = 15.44$, $P < 0.001$)). Post hoc tests showed that voltages in the error condition were significantly more positive in the LI group compared to the HI group (all P values below $P < 0.035$) whereas voltages in the correct condition did not differ between groups (all P values above $P > 0.140$). At parietal electrodes, there was a significant condition effect with voltages for error trials more positive than for correct trials (parietal midline: ($F(1,30) = 12.06$, $P < 0.01$); parietal lateral: ($F(1,30) = 12.68$, $P < 0.01$)). There was no significant interaction of group by condition at parietal electrodes (all P values above $P > 0.102$).

In the time window of the "late" Pe component (250–750 ms), we found a significant condition effect both for central (central midline: ($F(1,30) = 64.88$, $P < 0.001$); central lateral: ($F(1,30) = 77.90$, $P < 0.001$)) and parietal electrodes

(parietal midline: ($F(1,30) = 81.08$, $P < 0.001$); parietal lateral: ($F(1,30) = 69.18$, $P < 0.001$)). At all electrodes, voltages in the error condition were more positive than voltages in the correct condition. For all electrodes, we could not find any significant interaction of group by condition (all P values above $P > 0.106$). Fig. 2 shows averaged waveforms for incorrect and correct trials.

3.2.1.2. Latencies. With regard to the ERN/Ne, the "early" Pe, and the "late" Pe component, there were no group differences for latencies (all P values above $P > 0.302$).

3.2.2. Difference waves

3.2.2.1. Voltages. Results for ERN/Ne amplitudes, the "early" Pe, and the "late" Pe calculated from difference waves are summarized in Table 1.

Again, significant group differences for the ERN/Ne component were locally constrained at central electrode positions (FCz, Cz, C1, and C2) (central midline: ($F(1,30) = 7.40$, $P < 0.011$); central lateral: ($F(1,30) = 6.81$, $P < 0.014$)). At these positions, HI subjects demonstrated significantly lower amplitudes than controls. At electrode positions CPz, Pz, P1, P2, there were no significant group differences (parietal midline: ($F(1,30) = 2.87$, $P = 0.100$); parietal lateral: ($F(1,30) = 2.00$, $P = 0.168$)).

Group differences for the "early" Pe were locally constrained only at central electrode positions (FCz, Cz, C1, and C2) (central midline: ($F(1,30) = 6.68$, $P < 0.015$); central lateral: ($F(1,30) = 5.75$, $P < 0.023$)). At these positions, HI subjects demonstrated significantly lower "early" Pe amplitudes than controls. For parietal electrodes (CPz, Pz, P1, P2), there were no significant differences between groups (parietal midline: ($F(1,30) = 2.23$, $P = 0.146$); parietal lateral: ($F(1,30) = 2.59$, $P = 0.118$)).

In the time window of the "late" Pe component (250–750 ms) for all electrodes, we could not find a significant group difference (all P values above $P > 0.106$). Fig. 3 shows difference waveforms at central and parietal electrodes.

3.2.2.2. Latencies. With regard to the ERN/Ne, the "early" Pe, and the "late" Pe, there were no group differences for latencies (all P values above $P > 0.106$).

3.3. Correlational findings

As computation of averaged waveforms for correct and incorrect trials and computation of difference waves led to the same results with respect to group differences, correlations between amplitudes (peak or mean, respectively) and behavioral data (error rates), or age were computed for difference waves. As there were no significant group

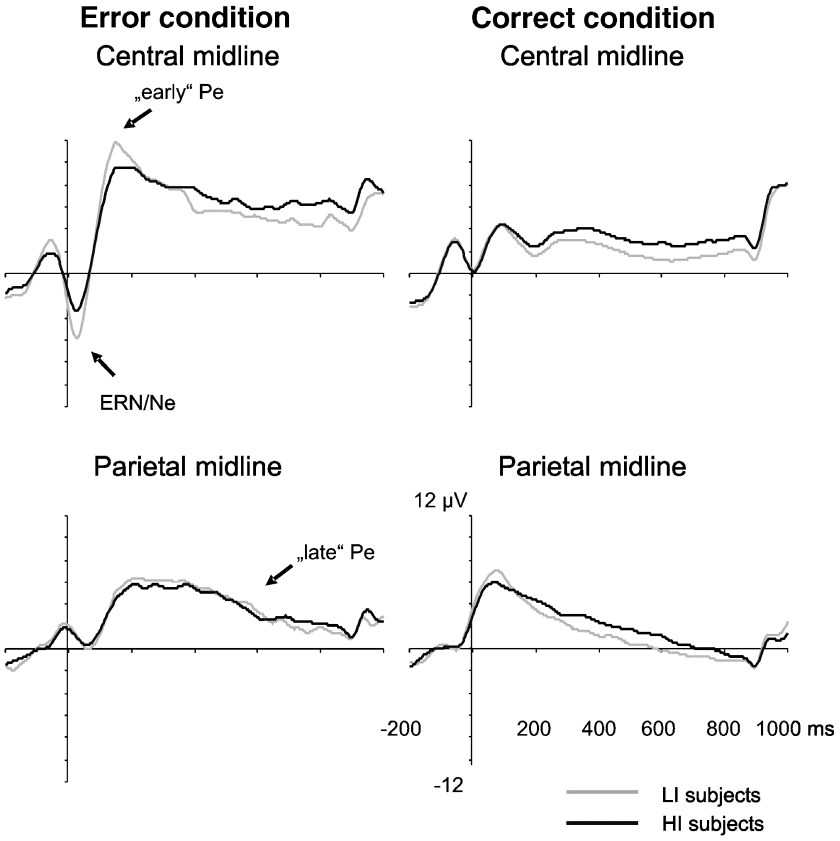

Fig. 2. ERP waveforms for high (HI) and low (LI) impulsiveness subjects at central electrode positions. Averaged waveforms ("raw" waves) for incorrect (left) and correct trials (right) in HI subjects (dark grey) and LI subjects (medium grey).

Table 1
Means and standard deviations (SD) of ERN/Ne, "early" Pe, and "late" Pe amplitudes from difference waves for subjects with high (HI) and low (LI) impulsiveness

		HI group		LI group		*P*
		Means	SD	Means	SD	
ERN/Ne	FCz	−4.278	2.506	−7.543	3.661	*
	Cz	−5.223	2.456	−7.921	3.771	*
	C1	−4.333	2.243	−6.503	2.620	*
	C2	−4.653	2.199	−7.145	3.202	*
	CPz	−5.977	2.233	−7.766	3.124	n.s.
	Pz	−5.751	2.685	−7.058	2.868	n.s.
	P1	−5.488	2.235	−6.636	2.700	n.s.
	P2	−5.552	2.491	−6.867	2.653	n.s.
"early" Pe	FCz	7.751	2.475	10.929	5.006	*
	Cz	6.398	2.496	9.873	4.498	*
	C1	6.410	2.074	8.854	4.131	*
	C2	5.704	2.258	8.461	3.541	*
	CPz	2.116	3.141	3.351	1.606	n.s.
	Pz	0.647	3.211	1.987	2.842	n.s.
	P1	0.311	2.531	1.928	2.076	n.s.
	P2	1.182	2.763	2.235	2.907	n.s.
"late" Pe	FCz	2.144	2.262	3.332	2.346	n.s.
	Cz	2.772	2.047	3.403	1.247	n.s.
	C1	2.943	1.933	3.911	2.151	n.s.
	C2	1.904	1.510	2.323	1.208	n.s.
	CPz	3.151	2.074	4.359	1.931	n.s.
	Pz	2.027	2.175	3.174	2.218	n.s.
	P1	2.435	1.759	3.323	1.917	n.s.
	P2	1.898	1.689	2.951	2.193	n.s.

Note. Amplitudes measured in μV; group differences were tested on significance by means of Fisher LSD tests (nominal level of $P < 0.05$) within analyses of variance (ANOVAs) for repeated measures. *P* values are reported in the result section; n.s.: non-significant.

differences on ERN/Ne latencies, correlations between these measures and error rates were not calculated.

3.3.1. Error rates and ERN/Ne amplitudes

To test whether ERP data were correlated with high and low impulsiveness, we calculated correlational analyses between ERN/Ne and error rates.

3.3.1.1. Entire group. For the entire group (HI and LI subjects ($n = 32$)), we found a significant positive relationship between the ERN/Ne amplitude and error rates at central electrodes C1 and C2 (C1: $r = 0.38$; $P = 0.031$; C2: $r = 0.36$; $P = 0.042$). Relationships at electrodes FCz and Cz were short of significance (FCz: $r = 0.35$; $P = 0.053$; Cz: $r = 0.35$; $P = 0.051$).

3.3.1.2. Within-group correlations. Neither for HI subjects ($n = 16$) nor LI subjects ($n = 16$) there was any significant correlation between ERN/Ne amplitudes at central electrodes and error rates (all *P* values above $P > 0.156$). Scatterplots of significant correlations are depicted in Fig. 4.

3.3.2. ERN/Ne and age

To control for age effects, we performed correlational analyses between age and ERN/Ne amplitudes from central and parietal electrodes. We could not observe any significant correlations neither with central nor with parietal electrodes (all *P* values above $P > 0.101$). This pattern of results was evident for the entire group as well as for HI and LI subgroups.

4. Discussion

In the present study, we used a Go/Nogo paradigm to investigate neurophysiological correlates of impulsiveness in healthy controls. Following a proposal by Pailing and coworkers [23], we calculated individual mean reaction time residuals as kind of scores in order to determine response control in participants. Using these RT residuals, the entire group ($n = 32$) was split into two subgroups with high ($n = 16$) and low impulsiveness ($n = 16$), respectively. Comparing performance data of HI and LI subjects revealed that groups did not differ with respect to reaction times and error rates: for "raw" waves as well as for difference waves at all central electrodes (FCz, Cz, C1, and C2), HI subjects had significantly smaller ERN/Ne amplitudes than LI subjects. Additionally, electrode positions demonstrating significant ERN/Ne effects group differences were locally confined. Besides that, we found a pronounced positivity immediately following the ERN/Ne at central electrodes which appears to be identical with the "early" Pe recently described by van Veen and Carter [28]. HI subjects had significantly smaller positive amplitudes than LI subjects both for "raw" waves and difference waves. Group differences were again locally confined to central electrodes. Finally, at central electrodes, the entire group showed significant positive correlations between ERN/Ne amplitude and error rates. Contrary to our predictions, we could not replicate Pailing et al.'s [23] finding of longer ERN/Ne latencies in less controlled subjects. One possible explanation for the discrepancy between the present finding and that of Pailing et al. [23] could be the high pressure on speed in our task which might preclude latency differences between groups. From Pailing et al. [23], it could have been expected that the LI group should have presented with shorter latencies compared to the HI group, as shorter latencies are correlated with more controlled response strategy and, consequently, lower error rates. With respect to the present task, the rather high pressure on response speed implemented could have led to a pronounced reduction of response control especially in the LI group, thus yielding latencies of equal extent in both groups.

In the present study, we could not find any group differences with regard to the "late" Pe component. The functional significance of this ERP component has been discussed controversially in the last years [12]. Nieuwenhuis and coworkers [20] found that the ("late") Pe amplitude is related to awareness of errors. As they have explicitly asked subjects to evaluate erroneous responses with respect to their associated awareness across the entire experiment, this

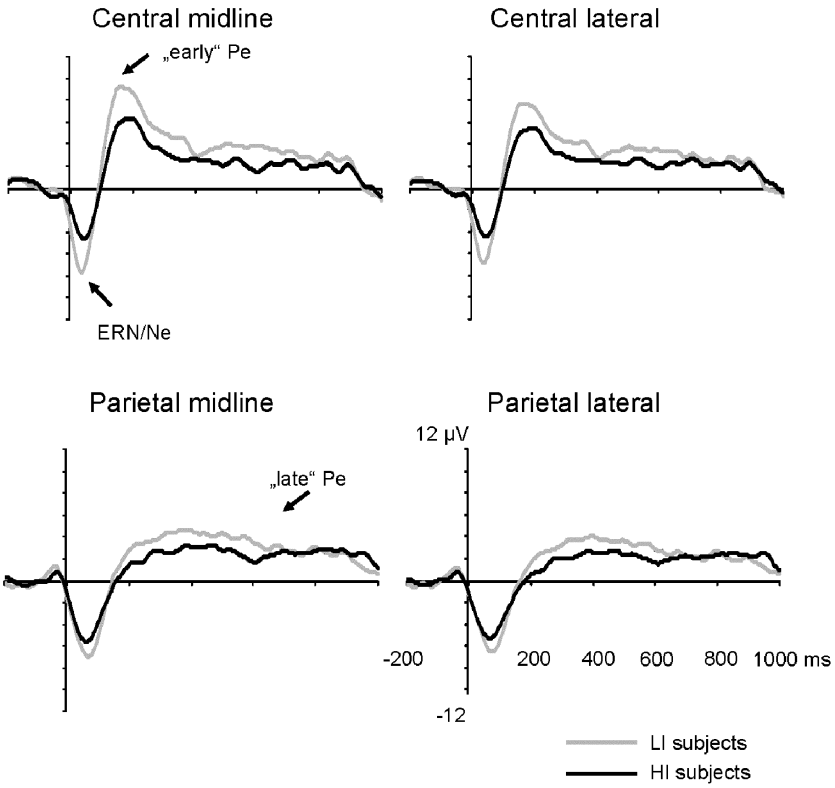

Fig. 3. ERP waveforms for high (HI) and low (LI) impulsiveness subjects at central and parietal electrode positions. Grand averaged difference waves (incorrect minus correct) in HI subjects (dark grey) and LI subjects (medium grey).

condition might have led to different processing strategies that did not match with our present investigation where we did not explicitly run tests on awareness and just relied on the "late" Pe amplitudes as an indicator of awareness.

Consistent with previous findings, all participants were generally faster on error trials (Nogo trials) than correct trials (Go trials) [9,10,13,23]. The significant RT difference when considering the entire group could not be replicated by means of within-group RT analysis in HI subjects. However, the increased variance of RT measures in HI subjects might have masked significance.

Considering further commonalities between our findings and previous ERN/Ne studies, it was demonstrated that smaller ERN/Ne amplitudes were observable in impulsive individuals [23] or in subjects with elevated measures of negative affect [19]. This is in accordance with our present results as smaller ERN/Ne amplitudes in HI subjects were found when compared to LI subjects. This is of very interest with respect to future ERN/Ne studies as it imposes to control for personality traits that may modulate ERN/Ne and Pe measures.

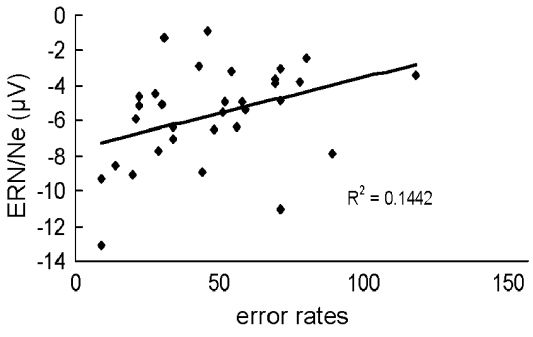

Fig. 4. Scatterplot of averaged ERN/Ne amplitudes (C1, C2) and error rates.

Most importantly, we observed a positive relationship between the ERN/Ne amplitude and error rate, confirming Pailing et al.'s findings [23]. As suggested by van Veen and Carter [28], the "early" Pe functionally belongs to the ERN/Ne. Therefore, smaller amplitudes of the ERN/Ne as well as "early" Pe appear to be a valid marker of higher impulsiveness in healthy controls. In this context, it is to remark that higher error rates can be linked with decreased ERN/Ne amplitudes. However, as error rates were rather high in both groups of LI and HI subjects, group differences on peak amplitudes should not be influenced by this factor. Moreover, the correlation between error rates and ERN/Ne amplitudes was only significant when coefficients were calculated across the entire group. Within HI subjects, there was no correlation between these variables.

In summary, our study could replicate Pailing et al.'s [23] finding of smaller ERN/Ne's in HI subjects compared to LI subjects. We could not replicate their finding of differences in ERN/Ne latencies between the two groups. In addition to Pailing et al. [23], we differentiate between "early" and "late" Pe in the present study demonstrating an effect of impulsiveness on the former but not on the latter.

The present study could demonstrate that ERP measures from response conflict paradigms are well suited to reflect personality traits in healthy subjects. An even broader aim of our study was to investigate the extent of variance in ERN/Ne and "early" Pe amplitudes in healthy subjects due to personality traits. This is of great clinical importance as clinical samples can be characterized by electrophysiological patterns of ERN/Ne (and "early" Pe) (e.g., less negative ERN/Ne amplitudes in patients with schizophrenia [1,2]; more negative ERN/Ne amplitudes in patients with obsessive-compulsive disorder [15]). Our data suggest that there is a broader range of impulsiveness even in healthy controls which might mask or pronounce between-group differences in clinical studies if not controlled for. However, in this context, it is an open question whether the concept of impulsiveness will hold in future studies in order to describe one of the factors that may contribute to variability in ERN/Ne measures. While the term certainly has face validity, other personality traits might also explain some of the variance observed here.

For example, in a recent study, Pailing and Segalowitz [22] found that the ERN/Ne amplitude correlated with neuroticism and conscientiousness, two factors of the International Personality Item Pool (IPIP, [18]). In this context, it has been discussed whether impulsiveness is a feature of conscientiousness as subjects with high scores of conscientiousness were found to be less impulsive than participants with low scores of conscientiousness, and vice versa [30]. Furthermore, patients with impulse-control disorders like borderline personality disorder are clinically characterized to show reduced inhibition of impulsive responses. However, it was also shown that these patients score high on items of neuroticism [6]. Therefore, our present findings could also be discussed in terms of personality traits like neuroticism and conscientiousness. In a similar vein, there is a broad overlap in the concepts of Negative Affect and Negative Emotionality with neuroticism in the study by Luu et al. [19]. Therefore, it remains an open question whether Negative Emotionality, neuroticism, or impulsiveness is the critical factor which results in smaller ERN/Ne and "early" Pe amplitudes.

References

[1] A.T. Bates, K.A. Kiehl, K.R. Laurens, P.F. Liddle, Error-related negativity and correct response negativity in schizophrenia, Clin. Neurophysiol. 113 (2002) 1454–1463.

[2] A.T. Bates, P.F. Liddle, K.A. Kiehl, E.T. Ngan, State dependent changes in error monitoring in schizophrenia, J. Psychiatr. Res. 38 (2004) 347–356.

[3] G.E. Berrios, M. Gili, Abulia and impulsiveness revisited: a conceptual history, Acta Psychiatr. Scand. 92 (1995) 161–167.

[4] M.M. Botvinick, T.S. Braver, D.M. Barch, C.S. Carter, J.D. Cohen, Conflict monitoring and cognitive control, Psychol. Rev. 108 (2001) 624–652.

[5] C. Carter, T. Braver, D. Barch, M. Botvinick, D. Noll, J. Cohen, Anterior cingulate cortex, error detection, and the online monitoring of performance, Science 280 (1998) 747–749.

[6] P.T. Costa, R.R. McCrae, Personality disorders and the five-factor model, J. Pers. Disord. 4 (1990) 362–371.

[7] P.L. Davies, S.J. Segalowitz, J. Dywan, P.E. Pailing, Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing, Biol. Psychol. 56 (2001) 191–206.

[8] J.M. De La Fuente, S. Goldman, E. Stanus, C. Vizuete, I. Morlan, J. Bobes, J. Mendlewicz, Brain glucose metabolism in borderline personality disorder, J. Psychiatr. Res. 31 (1997) 531–541.

[9] Z. Dikman, J. Allen, Error monitoring during reward and avoidance learning in high- and low-socialized individuals, Psychophysiology 37 (2000) 43–54.

[10] M. Falkenstein, J. Hohnsbein, J. Hoormann, L. Blanke, Effects of errors in choice reaction tasks on the ERP under focused and divided attention, in: C. Brunia, A. Gaillard, A. Kok (Eds.), Psychophysiological Brain Research, Tilburg Univ. Press, Tilburg, The Netherlands, 1990, pp. 192–195.

[11] M. Falkenstein, J. Hohnsbein, J. Hoormann, L. Blanke, Effects of crossmodal divided attention on late ERP components: II. Error processing in choice reaction tasks., Electroencephalogr. Clin. Neurophysiol. 78 (1991) 447–455.

[12] M. Falkenstein, J. Hoormann, S. Christ, J. Hohnsbein, ERP components on reaction errors and their functional significance: a tutorial, Biol. Psychol. 51 (2000) 87–107.

[13] W.J. Gehring, M.G.H. Coles, D.E. Meyer, E. Donchin, The error-related negativity: an event-related brain potential accompanying errors, Psychophysiology 27 (1990) 34.

[14] W. Gehring, B. Goss, M. Coles, D. Meyer, E. Donchin, A neural system for error detection and compensation., Psychol. Sci. 4 (1993) 385–390.

[15] W. Gehring, J. Himle, L. Nisenson, Action monitoring dysfunction in obsessive-compulsive disorder, Psychol. Sci. 11 (2000) 1–6.

[16] G. Gratton, M.G. Coles, E. Donchin, A new method for off-line removal of ocular artifact, Electroencephalogr. Clin. Neurophysiol. 55 (1983) 468–484.

[17] M. Hansenne, W. Pitchot, E. Pinto, J. Reggers, G. Scantamburlo, S. Fuchs, S. Pirard, M. Ansseau, 5-HT1A dysfunction in borderline personality disorder, Psychol. Med. 32 (2002) 935–941.

[18] International Personality Item Pool, A Scientific Collaboratory for the Development of Advanced Measures of Personality Traits and Other Individual Differences. http://ipip.ori.org/ipip/, 2001.

[19] P. Luu, P. Collins, D. Tucker, Mood, personality and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring, J. Exp. Psychol. Gen. 129 (2000) 43–60.

[20] S. Nieuwenhuis, K.R. Ridderinkhof, J. Blom, G.P. Band, A. Kok, Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task, Psychophysiology 38 (2001) 752–760.

[21] R.C. Oldfield, The assessment and analysis of handedness: the Edinburgh inventory, Neuropsychologia 9 (1971) 97–113.

[22] P.E. Pailing, S.J. Segalowitz, The error-related negativity as a state and trait measure: motivation, personality, and ERPs in response to errors, Psychophysiology 41 (2004) 84–95.

[23] P.E. Pailing, S.J. Segalowitz, J. Dywan, P.L. Davies, Error negativity and response control, Psychophysiology 39 (2002) 198–206.

[24] H. Saß, S.C. Herpertz, Psychopathic disorder, Curr. Opin. Psychiatry 7 (1994) 437–441.

[25] M.K. Scheffers, M.G. Coles, P. Bernstein, W.J. Gehring, E. Donchin, Event-related brain potentials and error-related processing: an analysis of incorrect responses to go and no-go stimuli, Psychophysiology 33 (1996) 42–53.

[26] L.J. Siever, K.L. Davis, A psychobiological perspective on the personality disorders, Am. J. Psychiatry 148 (1991) 1647–1658.

[27] P.H. Soloff, C.C. Meltzer, C. Becker, P.J. Greer, T.M. Kelly, D.

Constantine, Impulsivity and prefrontal hypometabolism in borderline personality disorder, Psychiatry Res. 123 (2003) 153–163.
[28] V. Van Veen, C.S. Carter, The timing of action-monitoring processes in the anterior cingulate cortex, J. Cogn. Neurosci. 14 (2002) 593–602.
[29] F. Vidal, T. Hasbroucq, J. Grapperon, M. Bonnet, Is the 'error negativity' specific to errors? Biol. Psychol. 51 (2000) 109–128.
[30] D. Watson, L.A. Clark, A.R. Harkness, Structures of personality and their relevance to psychopathology, J. Abnorm. Psychol. 103 (1994) 18–31.
[31] J.H. Woodcock, A neuropsychiatric approach to impulse disorders, Psychiatr. Clin. North Am. 9 (1986) 341–352.
[32] N. Yeung, J.D. Cohen, M.M. Botvinick, The neural basis of error detection: conflict monitoring and the error-related negativity, Psychol. Rev. 111 (2004) 931–959.