Performance Monitoring and Error Processing During a Lexical Decision Task in Patients With Parkinson’s Disease

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ABSTRACT

To evaluate performance monitoring and error processing during lexical decision tasks, event-related potentials (ERPs) obtained by time-locked to correct and error responses were studied in 17 Parkinson's disease (PD) patients without dementia and 15 healthy elderly participants. The amplitude of error negativity (Ne) obtained by averages time-locked to error response was significantly reduced in the PD patients, whereas there were no significant differences in the negative component for the correct response (Nc) between the two participant groups. The amplitude of the error positivity (Pe) and correct positivity (Pc) after the Ne and Nc components was also significantly reduced in the PD patients. The PD patients showed significantly slower reaction times and higher error rates. The reduced amplitude of the Ne, Pe, and Pc components in the PD patients suggested impaired performance and conflict monitoring as well as abnormal response strategy adjustments and deviant in later error monitoring processes associated with emotional, conscious evaluation of the error. (J Geriatr Psychiatry Neurol 2006;19:46-54)

Keywords: Parkinson’s disease; event-related potentials; reaction time; error negativity, error-related negativity; error positivity

Patients with Parkinson’s disease (PD) often show several cognitive deficits. These cognitive deficits have been defined variably as attributable to an internal regulation deficit, a deficit in the inhibition of unwanted responses, or an impairment in the maintenance of the internal representations that control actions,¹ and these deficits may result from dysfunction of processes controlled by the prefrontal cortex.² Linguistic disorders were generally believed to be rare in PD patients compared with patients with Alzheimer's disease. However, lexical processing and semantic memory were also impaired in nondemented PD patients.³

Error negativity (Ne)⁴ or error-related negativity (ERN)⁵ of event-related potentials (ERPs) obtained by averages time-locked to error reaction is considered to reflect performance monitoring and was initially thought to be related to error detection.⁵,⁶ A negative component like Ne/ERN can also be observed on correct trials (correct negativity [Nc] or correct-response negativity [CRN]), so that the Ne/ERN reflects either a comparison process leading secondarily to error detection or an emotional reaction.⁷ An alternative interpretation is that Ne/ERN reflects the detection of conflict but not necessarily overt errors.⁸,¹⁰ Source localization studies have found that the anterior cingulate cortex (ACC) is the likely generator of Ne/ERN.⁸,¹¹

Ne/ERN was also evaluated in patients with neurological diseases.¹²-¹⁶ In healthy participants, Ne/ERN showed a larger amplitude than Nc/CRN.⁶,¹² In patients with lateral prefrontal damage, correct-trial Nc/CRN activity was equal to error-trial Ne/ERN activity,¹² so that the lateral prefrontal cortex was thought to interact with the ACC in monitoring behavior and guiding compensatory systems.¹² Patients with damage to the medial prefrontal cortex, including the anterior cingulate region,
were aware of errors, although Ne/ERN was not produced. In nondemented PD patients, 1 study using a modified Eriksen flanker task, a Simon-type task, and a complex go/no-go task showed reduced Ne amplitude, so that the basal ganglia were thought to play an important role in error detection. However, another study using an Eriksen flanker task showed no significant differences in Ne/ERN amplitude between control participants and PD patients.

Ne/ERN and positive component (error positivity [Pe]) after the Ne/ERN were supposed to reflect the activity of two separate error-monitoring processes. In patients with Alzheimer’s disease, Pe was detectable, so that error awareness was thought to be preserved. Three hypotheses for the Pe have been proposed. First, the Pe might reflect conscious error recognition; second, it might be an adaptation of response strategy; or third, it might be subjective/emotional error processing.

Most Ne/ERN studies used a flanker task or a time estimation paradigm. Ne/ERN can be evoked during performance of other paradigms involving error processing or conflict monitoring. The Pe component varies independently of Ne/ERN and shows a high variance across tasks. The Pe component was not evaluated in the previous PD patient studies. The PD patients committed more errors than the healthy elderly participants while they performed the lexical decision task in our previous study, although psychometric tests did not suggest impaired semantic memory in these patients. To evaluate performance monitoring and error processing in PD patients and healthy elderly participants, we analyzed not only the Ne but also the Pe components of ERPs.

METHODS

Participants
Seventeen idiopathic PD patients without dementia (8 males and 9 females, mean 64.1 years) and 15 healthy age-matched control participants (7 males and 8 females, mean 63.8 years) participated in the present study. Motor symptoms in the PD patients ranged from stage I to III (I, n = 3; II, n = 9; and III, n = 5) on the Hoehn and Yahr scale. Duration of illness was 1.3 to 10 years (mean 6.1 years). Two patients had motor symptoms localized on the right side, whereas the remaining patients had motor symptoms on both sides. At the time of testing, they were treated with levodopa/dopa-decarboxylase inhibitors and dopamine agonists (dopa 7, dopa+amantadine 7, dopa+trihexyphenidyl 1, dopa+bromocriptine+amantadine 2). Experiments were performed during their “on” phase. None of the control participants or PD patients had taken any other drugs that might have affected the ERP components.

The patients and the control participants were heterogeneous in terms of socioeconomic conditions. Years of education did not differ significantly between the 2 participant groups. All the PD patients were evaluated with the Hasegawa dementia scale (the maximum score is 30 and the cutoff value for dementia is 20), and their scores were within the normal range (24-30, mean 28.3 ± 2.3). Scores on the Mini-Mental State Examination (control participants 29.1 ± 0.6; PD patients 28.6 ± 0.8) and vocabulary scores on Wechsler Adult Intelligence Scale (WAIS) subtests (control participants 11.9 ± 0.6; PD patients 12.1 ± 0.8) showed no significant differences between the 2 participant groups. There were no significant differences in depression scores between the 2 participant groups. To assess frontal lobe function, a new modified version of the Wisconsin Card Sorting Test (WCST) was used in 13 PD patients (mean 62.9 years) and 8 control participants (mean 65.9 years). In this version, there are 2 main modifications: the order of the reaction cards (eg, total of the reaction cards was changed from 128 of the original to 48 and 10 cards of the original to 6 cards per category) and the process of giving instructions. The achieved categories (CA) classification score and the perseverative errors (PE) reported by Nelson were used to evaluate the results of this test. Scores of CA (full core is 6) were 2.9 ± 1.8 in the PD patients and 5.2 ± 0.9 in the control participants, and those of PE were 5.9 ± 2.9 in the PD patients and 2.1 ± 1.1 in the control participants. The scores of CA were significantly lower in the PD patients (t = 6.889, P < .00001) and those of PE were significantly higher in the PD patients (t = 2.963, P < .008).

Experiments were performed in a dimly lit, relatively sound-proof shielded room with a room temperature of about 25°C. Each participant was seated in a chair in front of a computer monitor.

Procedure
A lexical decision task similar to the previous study was used. In each trial, S1 (a word) and S2 (a word or a nonword) were presented on the computer monitor and the participants were instructed to determine whether the S2 was a word (as the antonym or antithesis of S1) or nonword by pressing either of 2 buttons with their index finger of their preferred hand. The two response categories of word/nonword had respective probabilities of .5/.5. All the word stimuli were common Japanese nouns constituted of two “kanji” (ideogram or morphogram) characters. Each S1-S2 pair appeared only once within a block. The sequence of events for each trial was (1) a 2.3-cm horizontal bar (a warning stimulus) presented for 400 milliseconds in the center of the computer monitor; (2) S1 presented above this bar for 400 milliseconds; (3) only the bar presented again for 400 milliseconds; and (4) S2 presented above the bar until a response was made. The maximal duration of each S2 presentation was 2000 milliseconds. The intertrial intervals varied from 7.2 to 7.4 seconds. One block was composed of 30 S1-S2 pairs, and
6 blocks were presented in a random sequence for individual participants.

An electroencephalograph (EEG) was recorded using Ag/AgCl electrodes placed at 6 scalp locations (Fz, Cz, Pz, Oz, C3, C4) based on the International 10-20 System, and all electrodes were referenced to linked earlobes. The electro-oculogram (EOG) was recorded from the left outer canthus and the right supraorbital region. The electrode impedance was kept below 5 kΩ. The EEG and EOG were amplified with a bandpass filter of 0.05 to 30 Hz (3 dB down, 6 dB octave/slope), digitized (250 Hz/channel), and stored on a magnetic tape for offline analysis. Timing of switch closure for either of the two buttons was also recorded for measurement of reaction times (RTs) and response-locked averages.

The EEG epochs were time-locked to the response in each trial and averaged separately for the correct and error trials. Trials in which the EEG or EOG exceeded ±60 µV in amplitudes were automatically rejected. At least 15 error trials and correct trials immediately before the error trials obtained from the 6 runs in each participant were averaged, respectively. To obtain a sufficient number of error trials for averaged waveforms, individual stimulus types were not considered in the present study. The RTs in error trials and those in correct trials immediately before and after errors were measured. Error correction rate (percentage of correct button press immediately after errors) was also measured.

Components were identified according to their polarity, latency, and distribution over the scalp. The following search epochs were used to determine the latencies of ERP components: the negative component for error trials in the time-window of 10 to 200 milliseconds was designated as Ne, and that for correct trials was designated as Nc. Positive components following the Ne and the Nc were designated as error positivity (Pe) and correct positivity (Pc), respectively. Peak amplitudes of individual ERP components were measured with respect to a 200-millisecond preresorponse baseline, and peak latencies were defined as the points with maximal amplitudes for the respective search epochs. Peak-to-peak amplitude of the Ne component relative to the preceding positive point or takeoff point and mean amplitude of the Ne component measured in the time window of 40 to 120 milliseconds were also measured. ERP's recorded from the Oz were not included for the statistical analysis because of the contamination of EMG artifacts in some of the PD patients.

ERP components were subjected to an analysis of variance with the participant group (controls, PD), condition (correct, error), and electrode (Fz, Cz, Pz, C3, C4). The criterion for significance was less than .05. Spearman's correlation coefficient was used to relate the ERP components and the WCST scores.

**RESULTS**

The Ne amplitude was greater than the Nc in both participant groups (control participants: $F_{1,28} = 13.304$, $P = .001$; PD patients: $F_{1,32} = 5.274$, $P = .028$, Figure 1, Table 1). In the control participants, the Ne amplitude showed significant differences among the three midline electrodes sites (peak amplitude: $F_{2,42} = 4.682$, $P = .015$) and showed greatest negativity at Fz. The peak Ne amplitude and the mean Ne amplitude measured relative to the preresorpose baseline were significantly smaller in the PD patients than in the control participants (peak amplitude: $F_{1,30} = 8.089$, $P = .008$; mean amplitude: $F_{1,30} = 5.016$, $P = .033$). The peak-to-peak Ne amplitude measured relative to the preceding maximal positive point was also reduced in the PD patients ($F_{1,30} = 4.803$, $P = .036$). There were no significant differences in the Nc amplitude between the two participant groups. The latency of the Ne and the Nc components did not show significant differences between the two participant groups.

The amplitude of the Pe and the Pc components in the PD patients was significantly reduced (Pe amplitude: $F_{1,30} = 6.286$, $P = .018$; Pc amplitude: $F_{1,30} = 5.956$, $P = .0021$), whereas the latency of the Pe and the Pc components did not show significant differences. In the control participants, the Pe latency was significantly longer than the Pc latency ($F_{1,28} = 4.863$, $P = .036$), whereas differences between the Pe and Pc latency did not reach significant levels in the PD patients. In the control participants, the amplitude of the Pe and the Pc components showed significant differences among the three midline electrodes sites (Pe: $F_{2,42} = 6.993$, $P = .002$; Pc: $F_{2,42} = 4.066$, $P = .024$), showing Pz-maximal distribution. In the PD patients, the Ne, Pe, and Pc components tended to show diffuse scalp distribution. In both participant groups, none of the ERP components showed asymmetric distribution.

The Ne amplitude at Fz showed significant correlation with the scores of CA in the WCST ($R = .689$, $P < .01$) and negative correlation with the scores of PE ($R = -.692$, $P < .01$). The Pe amplitude at Pz was significantly correlated with the scores of CA ($R = .672$, $P < .01$). None of the other ERP components were correlated with the scores in the WCST. We also conducted the correlation analysis within the PD group. The Ne amplitude still showed significant correlation with the CA score ($R = .580$, $P < .01$) and negative correlation with the PE score ($R = -.661$, $P < .01$).

The error rates were significantly higher in the PD patients than in the control participants ($F_{1,30} = 24.628$, $P = .00002$) and were higher in response to nonword stimuli (Table 2). The error correction rates were lower in the PD patients than in the control participants ($F_{1,30} = 19.085$, $P = .00014$).

The PD patients showed significantly slower RT than the control participants, not only in the correct trials (correct RT, $F_{1,30} = 18.011$, $P = .00019$) but also in the error trials (error RT, $F_{1,30} = 19.379$, $P = .00015$). The error RT
was significantly faster than the correct RT in the control participants \(F_{1,28} = 13.310, P = .001\), whereas no significant differences were found in the PD patients. The correct RTs immediately after errors were significantly slower than those immediately before errors in the control participants \(F_{1,28} = 8.997, P = .006\) and in the PD patients \(F_{1,32} = 5.444, P = .026\).

**DISCUSSION**

In the PD patients, the Ne, Pe, and Pc amplitudes were significantly smaller and the RTs were significantly slower. Not only the peak Ne amplitude measured relative to the preresponse baseline but also the peak-to-peak Ne amplitude were significantly smaller in the PD patients and their error rates were higher. Amplitudes of contingent negative variation and movement-related potentials have shown to be reduced in PD patients.\(^{24,25}\) In healthy participants, amplitudes of prestimulus negative shift to targets increased as RT decreased.\(^{28}\) Visual examination of stimulus-locked averages in the present paradigm revealed that the amplitudes relevant to about 200 milliseconds preresponse baselines in 2 participant groups tended to show smaller amplitude in the PD patients.
(unpublished data). The mean amplitude 200 milliseconds before the S2 that was measured relative to 200 milliseconds before the warning stimulus was also reduced in the PD patients (unpublished data). When the prerresponse baseline is less negative, the Ne amplitude measured relative to the prerresponse baseline may show relatively greater negativity. Thus, the smaller Ne amplitude in the PD patients might not result from the methods used for amplitude measurement, although the smaller Pe and Pc amplitudes in the PD patients could be related to the differences in the prerresponse baselines between the 2 participant groups. The reduced Ne amplitude and the higher error rates in the PD patients were similar to those of a previous study in PD patients,14 whereas another study in PD patients did not show amplitude reduction in the Ne/ERN component.15 The discrepancies in the previous Ne/ERN findings in PD patients were reported to result from differences in error rates and behavioral measures (squeeze vs button press), as well as effects of dopaminergic medication.15 The PD patients in the present study were medicated and EEG averages were triggered by response, but not by electromyogram (EMG) as in the previous studies of PD patients. Holroyd and Coles27 proposed a hypothesis that the ERN is generated when a negative reinforcement learning signal is conveyed to the ACC via the mesencephalic dopamine system and that this signal is used by the ACC to modify performance of the task at hand.27 The reduced Ne/ERN amplitude in the PD patients is in line with this hypothesis of direct links between the dopamine system and the Ne/ERN. A study using functional magnetic resonance imaging (fMRI) revealed significant signal intensity reductions during a working-memory paradigm in striatum and frontal lobe sites in PD patients with cognitive impairment compared with those who were not cognitively unimpaired.28 Executive dysfunction in PD patients has been previously shown to be extremely sensitive to the effects of controlled L-dopa withdrawal.29 However, dopaminergic medication improves or impairs performance depending on the cognitive tasks and the basal level of dopamine function in the underlying corticostriatal circuitry in PD patients.30 L-dopa withdrawal in the PD patients might have caused an even smaller or normal Ne amplitude, depending on the basal level of dopamine function of individual patients. Discrepancies in the Ne/ERN findings of PD patients may have resulted from differences in the paradigms used as well as the cognitive and the basal level of dopamine functions of the PD patient groups. In patients with Tourette syndrome, overall ERN amplitude was higher than in control participants, and this finding was interpreted to be due to a hyperactive frontal-striatal-thalamic-frontal circuit.31 These findings also suggest dopaminergic involvement in action monitoring and ERN.

The Ne/ERN component is influenced by affect and motivation.32 Ne/ERN amplitudes in patients with schizophrenia were smaller than those in healthy participants, and Ne/ERN was not detected in 1 study.37 An fMRI study revealed that patients with schizophrenia were characterized by relative underactivity in the rostral ACC and the limbic system compared with healthy participants.35 These findings suggest that there may be a disturbed affective or motivational response to the commission of errors in schizophrenia.35 Enhanced Ne/ERN amplitude that correlated with symptom severity was found in patients with obsessive–compulsive disorder (OCD).38,39 In an fMRI study, the ACC in OCD patients was found to be hyperactive at rest, during symptom provocation, and after commission of errors in cognitive tasks.39 This hyperactivity in the OCD patients was thought to reflect an abnormality in conflict detection.39 Regional cerebral blood flow response to levodopa in the medial frontal gyrus and posterior cingulate cortex (PCC) has shown significant differences between advanced-stage PD patients with mood fluctuations and those without mood fluctuations.40 Mood fluctuations may arise in PD patients who have abnormal dopaminergic modulation of the caudate nucleus, ACC, or orbital frontal cortex, which innervate the PCC.40 However, the depression scores were not significantly different between the PD patients and control participants, and mood fluctuations including mania or anxiety were not clinically obvious in the PD patients. Results from positron emission tomography with 6-[18F]fluoro-l-dopa (FDopa) as the tracer showed that medication-free PD patients in the early stage had decreased FDopa uptake in the striatum and increased FDopa uptake in the cortical area including the dorsolateral prefrontal cortex and the ACC.41 The increased cortical FDopa uptake has been speculated to represent a compensatory mechanism caused by striatal dopaminergic hypofunction.41 The ACC is part of a control network involving the prefrontal cortex, basal ganglia, and limbic areas. The reduced Ne amplitude in the PD patients may result from corticostriatal dysfunction rather than the ACC dysfunction, per se.

Dipole modeling suggested that the Ne/ERN component recorded from the scalp originated from medial frontal regions, most probably the ACC.8,11,19 A study using intracranial recordings has revealed that the mesiotemporal and some prefrontal cortical sites represent integral components of the brain’s error checking system.42 Functional MRI studies revealed that the neuroanatomical areas activated during error-related processing were the ACC, presupplementary motor area (pre-SMA), lateral prefrontal cortex, inferior parietal lobe, and bilateral insular cortex.43,44 Functional MRI studies have shown that networks involving the frontomedian wall are activated during both response competition and error processing.43,45 The rostral ACC may be involved in error detection, whereas more dorsal ACC/pre-SMA may monitor for conflict.43,45

Whether the Ne/ERN is related to error detection or conflict monitoring is still under debate. There may be some similarities between prerresponse conflict in interfer-
ence tasks and underdetermined responding, that is, with uncertainty. PD patients showed reduced amplitude of N270 of ERPs time-locked to stimulus elicited by conflict condition. The N2 component in a go/no-go task is supposed to reflect conflict monitoring rather than response inhibition. In a go/no-go task, PD patients showed a significantly smaller no-go N2 amplitude and higher error rates. Because PD patients have impairment of the working memory and disrupted inhibitory processes, higher error rates in the PD patients might partly result from impairment of inhibition. Successful inhibition of prepotent responses involves conflict, and failed inhibition involves both conflict and errors. During the performance of the present paradigm, conflict also occurs. The smaller Ne amplitude in the PD patients might result from impaired conflict monitoring.

In the PD patients, the RT was significantly slower, although the vocabulary scores in the WAIS subtests and normal N400 latency of ERPs obtained by stimulus-locked averages in our previous study did not suggest impaired language information processing in the PD patients included in the present study. PD patients are able to perform tasks relatively well when external cues are available. Slow RTs in PD patients are thought to result from slowed stimulus–response linking in choice RT and not from impaired motor initiation/execution. However, another study has shown that movement-related potentials in PD patients also require extra time, and delayed onset of the movement-related potentials indicates that one or more premotor processes are also slowed in PD patients. There are conflicting findings about the RT in PD patients, depending on the tasks used or PD patient groups. The performance of the nonfrontally impaired Parkinson’s group was indistinguishable from that of control participants, whereas the frontally impaired Parkinson’s group responded significantly slower than the controls, despite unimpaired information processing and automatic functions. In the present study, the scores for Mini-Mental State Examination in the PD patients did not differ from those in the control participants. However, significantly decreased CA and increased PE in the WCST suggested frontal lobe dysfunction in the PD patients. Increased ACC activity has shown to be negatively correlated with reaction time in healthy participants. Healthy participants with short RTs have shown significantly more ACC activation and an increased error rate. In the present study, the Ne amplitude showed significant correlation with the scores of CA and negative correlation with the scores of PE, and the Pe amplitude was significantly correlated with the scores of CA. The smaller Ne amplitude and slower RTs in the PD patients may result from frontal lobe dysfunction.

Ne/ERN is associated with “slips” (participants know the right answer but fail to react correctly) rather than “mistakes.” In healthy participants, errors caused by premature responding and perceived as errors are associated with large ERNs, whereas those with uncertainty are associated with smaller ERNs. The error RTs were significantly faster than the correct RTs in the control participants, whereas differences between the correct and error RTs did not reach significant levels in the PD patients. PD patients have problems disengaging attention from a particular word in order to switch to a new word, and a number of semantic processing errors are found for PD patients. Under conditions of uncertainty, on-line monitoring of cue–stimulus relationships appears to be slower in PD patients than in elderly controls. The PD patients might be unsure whether the S2 was a word or a nonword, so that faulty knowledge might contribute response uncertainty and slower error RTs. However, the RTs in the PD patients also showed posterior slowing, so that the PD patients noticed most of the errors they committed. In an fMRI study, elderly participants showed greater ACC and pre-SMA activation, whereas slower responders showed greater activation in the parietal, lateral PFC, insular, and ACC regions during both response competition and error processing. The reduced Ne amplitude and abnormal RTs in the PD patients may be related to a different approach to dealing with stimulus–response relationships.

Ne/CRN was initially thought to result from execution of partial errors or from stimulus-related negativity contaminating response-locked ERP on correct trials. Ne/CRN was suggested to be a manifestation of the comparison process itself rather than the result of the outcome of the comparison between actual response and desired response. The Ne/CRN and Ne/ERN have been shown to reflect similar ACC activity. A study using Laplacian transformation showed that amplitude of Ne-like wave on correct responses was related to correctness of the subsequence trial, and a decrease of the amplitude of the Ne-like wave on correct responses before errors could express a lapse of the response-monitoring processes leading to a subsequent decrease of executive control and hence to errors. A study using tasks that manipulated response certainty and those designed to influence stimulus certainty has shown that error and correct responses are processed more similarly when uncertainty is present. Uncertainty is associated with attenuation of Ne/ERN and enhancement of Ne/CRN. Enhanced Ne/CRN in patients with Alzheimer’s disease was thought to reflect uncertainty toward the correct response. In schizophrenic patients, 1 study that used a go/no-go task found a smaller Ne/CRN amplitude, whereas in another study that used a picture–word matching task, schizophrenic patients had no/CRN such that the amplitude of the Ne/CRN was equal to that of Ne/ERN. Similar to the previous study, the Ne component in our PD patients was not enhanced, and these findings differed from those in patients with lateral prefrontal damage that showed enhanced Ne amplitude.

In the PD patients, the Pc amplitude was significantly reduced, whereas the Pc latency was normal. Functional
significance of Pc is much less clear. Stimulus-driven positivity (eg, P300) was thought to overlap with the response-related potential.\textsuperscript{65} Pc amplitude was correlated with P300 amplitude, and topography of the Pc was similar to that of the P300.\textsuperscript{65} The reduced Pc amplitude in the PD patients is comparable to reduction of P300 amplitudes in nonmedicated PD patients.\textsuperscript{24,64} However, Pc amplitude was normal in schizophrenic patients.\textsuperscript{66} This finding may contradict the hypothesis that the Pc is the result of stimulus-locked P300 potentials,\textsuperscript{63} because the P300 is one of the most reliably attenuated ERPs in schizophrenia.\textsuperscript{65} Because the PD patients had normal latency of the stimulus-driven positivity in the stimulus-locked averages and significantly slower RTs,\textsuperscript{20} the Pc latency in the response-locked averages should have been shorter in the PD patients. Normal Pc latency in the PD patients is also inconsistent with the hypothesis for the Pc.

PD patients had task-switching deficits resulting from depletion of attentional resources allocated to the retrieval of task-relevant information.\textsuperscript{68} Because variability of correct RTs in individual participants was significantly greater in the PD patients,\textsuperscript{69} they might have greater fluctuations of peak latency of single-trial ERPs resulting from impairment of attention. Greater trial-to-trial variability of single trials results in a reduction of averaged ERP amplitude.\textsuperscript{67} The reduced Pc amplitudes in the PD patients might result from greater trial-to-trial latency jitter.

The Pe amplitude was significantly reduced in the PD patients. Schizophrenic patients who had reduced Ne/ERN amplitudes did not show Pe abnormality.\textsuperscript{36,37} Children with attention deficit hyperactivity disorder also showed a diminished Pe, although ERN was normal.\textsuperscript{68} Pe was supposed to reflect conscious error recognition, response strategy adaptation, and later error-monitoring processes.\textsuperscript{6,16,17} The ACC also contributes to generate Pe component.\textsuperscript{19,69} A source localization study has shown different neural generators for ERN/Ne and Pe.\textsuperscript{69} The Pe generator has been shown to be localized more rostral within the ACC, and the Ne/ERN and Pe components were thought to represent different aspects of error processing.\textsuperscript{69} Because the rostral ACC is associated with affective processing, the Pe is also suggested to reflect a subjective emotional assessment process.\textsuperscript{19} The reduced Pe amplitude in the PD patients suggests abnormal response strategy adjustments and deviance in later error-monitoring processes associated with emotional, conscious evaluation of the error. The Pe component has also been shown to be significantly correlated with skin conductance response (SCR), which reflects autonomic nervous system function. Both the SCR and the Pe have been shown to be correlated with posterror slowing.\textsuperscript{70} Most of the PD patients also had signs and symptoms of mild dystautonnia, although electrophysiological autonomic tests were not performed. The reduced Pe amplitude in the PD patients could be related to autonomic nervous system dysfunction.

In summary, the smaller Ne and Pe amplitudes in the PD patients suggest impaired performance and conflict monitoring as well as abnormal response strategy adjustments and deviance in later error-monitoring processes associated with emotional, conscious evaluation of the error.

References


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