Dissociable Components of Error Processing

On the Functional Significance of the Pe Vis-à-vis the ERN/Ne

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Abstract. We conducted a literature review to examine the functional significance of the error positivity (Pe), an error-related electrophysiological brain potential often observed in combination with the error negativity (Ne). The review revealed many dissociations between documented effects on the Ne and Pe, suggesting that these components reflect different aspects of error processing. We found little support for the proposed hypotheses that the Pe is associated with the affective processing of errors or with posterror behavioral adaptation. Some support was found for the hypothesis that the Pe reflects conscious recognition of an error. Finally, we discuss the notion that the Pe may reflect a P3b associated with the motivational significance of the error. We conclude that more research is needed to test predictions of the various Pe hypotheses, and that more rigorous investigation of the neural generators of the Pe may contribute to a better understanding of the neurocognitive processes involved in error monitoring.

Keywords: error processing, performance monitoring, error positivity, error negativity, error awareness

Over the last decade, the study of error monitoring has been advanced considerably by the study of two event-related brain potential (ERP) components that can be observed when people make errors: the error(-related) negativity (Ne or ERN) and the error positivity (Pe; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). The Ne is a sharp negative deflection with a frontocentral scalp distribution peaking 60–80 ms following an erroneous response. The Ne is typically followed by the Pe, a slow positive wave with a diffuse scalp distribution and maximum amplitude between 200–400 ms. Whereas the sensitivity of the Ne to various experimental factors and individual differences has been scrutinized extensively (Holroyd, Nieuwenhuis, Mars, & Coles, 2004), the Pe has not been studied with the same rigor. Likewise, whereas several theoretical and mechanistic accounts of the processes reflected in the Ne have been developed (cf. Yeung, Cohen, & Botvinick, 2004), the functional significance of the Pe remains pretty much in the dark.

We examine associations and dissociations between the reported effects on the Ne and Pe by reviewing and analyzing the available literature. Although the resulting pattern of outcomes is not entirely consistent, a picture emerges that tentatively supports a conceptualization in terms of error salience or significance, suggesting that the Pe reflects processes similar to those expressed in the P3b. We also discuss some research avenues that might lead to more rigorous experimental investigation and new hypotheses of the neurocognitive processes underlying the Pe.

Performance Monitoring Processes Reflected in the Ne and Pe

Largely overlapping brain areas in the medial frontal cortex (MFC), clustering in the rostral cingulate zone (RCZ, the posterior MFC border zone between the medial areas BA8, BA6, and BA32', with some extension into BA24'), are involved in monitoring for unfavorable outcomes, response errors, response conflict, and decision uncertainty (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). These conditions have in common that they signal an increased probability that goals may not be achieved or rewards may not be obtained. In the ERP, these processes are typically expressed in the Ne and related components, including the so-called feedback-

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ERN (Nieuwenhuis, Holroyd, Mol, & Coles, 2004) and the Ne-like component on correct conflict trials (Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003), often referred to as the CRN.

A neurobiological mechanism that captures the role of the RCZ in coding outcome- and error-related information has been proposed by Holroyd and Coles (2002). Errors in reward prediction are coded by phasic changes in activity of the mesofrontal/mesolimbic dopamine system: a phasic increase or decrease when ongoing events are suddenly better or worse (respectively) than expected (Schultz, 2002). These phasic dopamine signals are communicated to the RCZ (giving rise to the Ne), where they are used for improving task performance in accordance with basic reinforcement-learning principles. The RCZ may also be involved in the monitoring of response conflict (Botvinick, Braver, Barch, Carter, & Cohen, 2001), as may occur just prior to a correct response when a prepotent but incorrect response was prevented (e.g., Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). The detection of postresponse conflict on error trials has been proposed as a mechanistic account of internal error detection (Yeung et al., 2004).

Reconciling the conflict and reinforcement-learning theories, the RCZ may be engaged when the need for adjustments to achieve action goals becomes evident: Response conflict signals a reduced probability of obtaining a reward, whereas errors and unexpected negative feedback signal the loss of an anticipated reward (Ridderinkhof et al., 2004). Consistent with this hypothesis, Brown and Braver (2005) have proposed that areas in the medial wall, roughly corresponding to the RCZ, learn to predict the *likelihood* of imminent errors in a given context. In a meta-analysis of the human neuroimaging literature, focusing on RCZ activations in response to these types of events, Ridderinkhof et al. (2004) found the most pronounced cluster of activations in BA32'. Activation foci associated with reduced probabilities of obtaining a reward (such as response conflict) clustered slightly more dorsally than foci associated with errors and failures to obtain an anticipated reward. Together, these patterns suggest the importance of the RCZ area for a unified performance-monitoring function, the electrophysiological correlate of which is the Ne.

Thus, based on extensively documented research efforts, converging views on the functional significance of the Ne have begun to emerge. By contrast, we have only begun to scratch the surface when it comes to the Pe. The sensitivity of the Pe to various experimental factors and individual differences has not been studied systematically, and interpretational frameworks consistent with the scattered evidence are yet to be developed. As will become evident from the literature analysis presented below, studies that examine individual differences in, and/or the effects of experimental manipulations on both the Ne and the Pe often report these effects to be dissociated. Since the Ne and Pe differ also in terms of timing and scalp distribution, it appears that the Pe reflects aspects of error-related processing that are, at least in part, independent of those manifested in the Ne.

Review of Studies That Report Both Ne and Pe: Associations and Dissociations

We surveyed the ERP literature published or (to our knowledge) accepted for publication before August 2005, focusing on studies that report the effects of focal brain lesions, individual differences, pharmacological interventions, or experimental manipulations on both the Ne and the Pe. A study was selected if (1) both Ne and Pe were examined and (2) different conditions (experimental manipulations or interventions) or groups were compared.

Thirty-two published studies that met our inclusion criteria are summarized schematically in Table 1[1]. To evaluate our hypotheses, we divided these studies into three categories. First, pharmacological intervention studies were examined to determine whether the sensitivity of the Ne to dopaminergic and other agents is mimicked in the Pe. Second, we analyzed studies reporting various individual differences, including age-related, personality-related, and pathology-related differences, to explore whether systematic patterns of concordant/discordant group effects on the Ne and Pe can inform us about the nature of the processes underlying the Pe. Finally, results of various experimental manipulations on the Ne and Pe will be discussed.

Pharmacological Effects

As noted before, the MFC is densely targeted by ascending dopaminergic projections, and accordingly the Ne has con-

1 In two cases, additional unpublished data were used to allow inclusion (Pe data from Leuthold & Sommer, 1999, were augmented with unpublished Ne data from the same study, obtained through personal communication, and cited in Elton, Spaan, & Ridderinkhof, 2004; Ne data from Ridderinkhof et al., 2002, were augmented with unpublished Pe scores from the same study). Two studies using the stop-signal paradigm (Endrass, Franke, & Kathmann, 2005; Overtoom et al., 2002) met the inclusion criteria specified above, but were nonetheless excluded because the extent to which Ne and Pe components were confounded with inhibition-related ERP components could not be determined unambiguously (cf. Ramautar, Kok, & Ridderinkhof, 2004).

sistently been found to be sensitive to changes in dopaminergic neurotransmission. In contrast, the Pe does not seem to depend heavily on the dopamine system. For example, De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe (2004) showed that amphetamine, which increases dopamine release, leads to a larger Ne compared to placebo, whereas Pe amplitude was not influenced by amphetamine. Further, sedative substances that act on dopamine receptors, such as ethanol, or on GABA receptors (GABA interacts closely with dopamine and innervates the MFC), such as the benzodiazepine lorazepam, elicit a smaller Ne whereas no differences in Pe amplitude are found (De Bruijn et al., 2004; Ridderinkhof et al., 2002). Caffeine, a substance that indirectly stimulates the production and reuptake of dopamine through its effect on the dopamine precursor adenosine, elicits an increased Ne as well as an increased Pe (Tieges, Ridderinkhof, Snel, & Kok, 2004). Finally, mirtazapine, an antidepressant that primarily influences histaminergic neurotransmission (which does not innervate MFC), affects neither Ne nor Pe amplitude (De Bruijn et al., 2004).

Summary

With the exception of caffeine, all substances that directly or indirectly affect dopaminergic activity and produce (enhancing or attenuating) effects on the Ne fail to produce such effects on the Pe. Although relying on null findings carries the usual risks, the patterns appear reasonably consistent across studies and argue against the notion that the dopamine-mediated performance-monitoring processes subserved by the MFC, and expressed in the Ne, also underlie the Pe.

Individual Differences: Age, Pathology, and Personality

Age seems to differentially influence the Ne and Pe. Developmental studies have shown a smaller Ne in children compared to young adults, whereas the Pe is similar across groups, being essentially in place even in the youngest children examined (Table 1). In older compared to young adults, a reduction in amplitude is typically found not only for the Ne but also for the Pe (Table 1). The age-related differences in Ne amplitude might be related to deficiencies in the mesofrontal dopamine system in childhood and senescence (Nieuwenhuis et al., 2002). The pattern of an intact Pe in childhood but attenuated Pe amplitudes in older age bears resemblance to age-related differences seen for the P3b component of the ERP, which shows a similar pattern of change across the life span (Polich, 1997; Ridderinkhof & van der Molen, 1995).

Altered dopaminergic function could be (partly) responsible for the differential effects of some neuropsychiatric pathologies on the Ne and Pe. For example, the Ne is smaller in patients with Alzheimer's disease, Parkinson's disease, and schizophrenia than in controls, but no differences in Pe amplitude are found (Table 1). The reduced Ne amplitude in Alzheimer's disease might be related to dopaminergic deficiencies, primarily because the striatal uptake of the dopamine reuptake ligand [11C]β-CFT is decreased (Rinne, Shalberg, Ruottinen, Nagren, & Lehikoinen, 1998), whereas in Parkinson's disease this is presumably caused by dysfunction of the mesencephalic dopamine system, which, among others, has projections via the limbic circuit to the ACC (Falkenstein, Willemssen, Hohnsbein, & Hielscher, 2005). Schizophrenia is associated with relative dopaminergic hyperactivity in subcortical areas (including the striatum), but dopaminergic *hypo*activity in frontal brain areas (Weinberger, 1987), which could explain the impairment in error processing expressed in the Ne.

Bates, Liddle, Kiehl, and Ngan (2004) also report a smaller Ne during psychosis compared to nonpsychotic stages in schizophrenic patients, in the absence of effects on Pe amplitude. Dopamine release is *elevated* during psychotic phases (as opposed to periods of remission; e.g., Laruelle & Abi-Dargham, 1999), but *sensitivity* to dopamine is assumed to be altered during psychosis (e.g., Winterer & Weinberger, 2004). While deviating patterns of dopaminergic activity during psychosis relative to remission likely resemble the patterns of divergence in schizophrenic patients vs. controls, it is not yet fully understood how the error-monitoring processes reflected in the Ne are affected by acute psychosis.

In contrast, patients with obsessive-compulsive disorder (OCD) had a larger Ne compared with controls, while no effects on Pe amplitude were found (Ruchsow et al., 2005). OCD has been associated with hyperactivity of action-monitoring processes in a circuit including frontal, striatal, and thalamic regions (Maltby, Tolin, Worhunsky, O'Keefe, & Kiehl, 2005), as well as with decreased serotonin sensitivity, in particular in the frontal cortex (Aouizerate, B., Guehl, D., Cuny, E., Rougier, A., Bioulac, B., Tignol, J., & Burbaud, 2004). Serotonergic projections from the raphe nucleus exert a tonic inhibitory control over the activity of the dopaminergic system in the striatum and the cortex. The disruption of this balance by decreased serotonin sensitivity in OCD leads to elevated dopamine levels, which may in turn have caused the larger Ne in patients with OCD.

Whether cortical lesions affect the Ne and Pe seems to depend on the specific cortical areas affected by the lesion: Both the Ne and Pe are smaller or even absent in patients with frontolateral or basal ganglia lesions compared to healthy subjects, whereas both Ne and Pe are unaffected in patients with frontopolar or temporal lesions (Ullsperger & Von Cramon, 2005; Ullsperger, von

Table 1. Summary of N_E and P_E findings and P_E scoring parameters per study, divided into three groups of studies: pharmacological interventions, individual differences, and experimental manipulations.

Reference	Task	Manipulations/groups	N_E Results	P_E				
				Results	Amplitude measure	Window (in ms post-response)	Baseline (in ms pre-response)	Maximum along the midline
Pharmacological interventions								
De Bruijn et al. (2004)	Flanker	X: D-amphetamine Y: lorazepam Z: mirtazapine P: placebo	X > P Y < P Z = P	X = P Y = P Z = P	peak	200–500	100–0	X: Cz Y: Cz Z: Cz P: Cz
Ridderinkhof et al. (2002)	Flanker	X: alcohol P: placebo	X < P	X = P	peak	250–650	150–50	X: Cz Y: Cz
Tieges et al. (2004)	Switch	X: low-dose caffeine Y: high-dose caffeine P: placebo	X = Y > P	X = Y > P	peak	200–400	150–50	X: FCz Y: FCz P: FCz
Individual differences								
Alain et al. (2002)	Stroop	X: schizophrenia patients Y: controls	X < Y	X = Y	mean	300–400	400–200	X: Fz, Cz, Pz Y: Fz, Cz, Pz
Band & Kok (2000)	Mental rotation	X: young adults Y: older adults	X > Y	X > Y	mean	300–600	1500–500	X: Fz, Cz, Pz Y: absent
Bates et al. (2004)	Go/Nogo	X: schizophrenia patients Y: controls X1: during psychosis X2: in remission	X < Y X1 < X2	X = Y X1 = X2	peak	100–380	200–150	X: FCz Y: FCz
Davies et al. (2004)	Flanker	X: children Y: young adults	X < Y	X = Y	peak	?	600–400	X: Cz Y: Cz
Dywan et al. (2004)	Flanker	X: young adults Y: older adults	X > Y	X > Y	peak	150–350	600–400	X: Cz Y: FCz
	Source memory	X: young adults Y: older adults	X > Y	X > Y	peak	150–350	200–0	X: FCz Y: Cz
Falkenstein et al. (1998)	Choice Flanker	X: young adults Y: older adults	X > Y	X > Y	peak	200–500	?	X: Cz, Pz Y: Cz, Pz
Falkenstein et al. (2005)	Choice Go/Nogo	X: Parkinson's patients Y: controls	X < Y	X = Y	mean	250–550	200–0	X: Pz Y: Pz
Hajcak et al. (2004)	Stroop	X: low-NA subjects Y: high-NA subjects	X < Y	X > Y	mean	200–400	200–0	X: FCz Y: FCz
Ladouceur et al. (2004)	Flanker	X: early adolescents Y: late adolescents	X < Y	X = Y	peak	?	150–50	X: Cz Y: Cz
Mathalon et al. (2002)	Match	X: schizophrenia patients Y: controls	X < Y	X = Y	mean	200–500	100–0	X: Cz, Pz Y: Cz, Pz
Mathalon et al. (2003)	Match	X: young adults Y: older adults Z: Alzheimer's patients	X > Y > Z	X = Y = Z	mean	200–500	50–0	X: Fz Y: Fz Z: Fz
Mathewson et al. (in press)	Flanker Source memory	X: young adults Y: older adults	X > Y	X > Y	peak	150–350	Flanker: 600–400 Source memory: 200–0	X: Fz, FCz, Cz Y: Cz
Ruchsow et al. (2005)	Go/Nogo	X: obsessive-compulsive patients Y: controls	X > Y	X = Y	peak mean	early P_E: 0–250 late P_E: 250–750	200–0	X: FCz, Cz Y: FCz, Cz X: CPz, Pz Y: CPz, Pz
Santesso et al. (in press)	Flanker	X: high-scoring children Y: low-scoring children on a: neuroticism, b: extraversion, c: psychoticism, and d: lie scales	Xa = Ya Xb = Yb Xc < Yc Xd > Yd	Xa = Ya Xb = Yb Xc = Yc Xd = Yd	peak	200–500	600–400	X: Cz Y: Cz

Table 1 (continued)

Reference	Task	Manipulations/groups	N_E Results	P_E				
				Results	Amplitude measure	Window (in ms post-response)	Baseline (in ms pre-response)	Maximum along the midline
Stemmer et al. (2003)	Flanker	X: MFC lesion patients Y: controls	X ≤ Y	X ≤ Y	peak	?	?	X: Cz Y: Cz
Ullsperger et al. (2002)	Flanker	W: frontolateral lesion X: bifrontopolar lesion Y: temporal lesion patients Z: controls	W < Z X = Z Y = Z	W < Z X = Z Y = Z	mean	300–450	100–0	W: Pz X: Pz Y: Pz Z: Pz
Ullsperger & Von Cramon (2005)	Flanker	X: basal ganglia lesion Y: frontolateral lesion patients Z: controls	X < Z Y < Z	X < Z Y < Z	mean	early P_E: 120–300 late P_E: 300–500	100–0[1]	X: absent Y: absent Z: Pz
Experimental manipulations								
Dywan et al. (2004)	Flanker Source memory	W: flanker-task errors X: source-memory errors Y: many errors Z: few errors	W = X Y = Z	W > X Y < Z	peak	150–350	Flanker: 600–400 Source memory: 200–0	FCz, Cz
Ehlis et al. (2005)	Flanker	X: genuine response errors Y: erroneous error feedback	X > Y	X > Y	peak	100–400	200–100	Cz, Pz
Elton et al. (2004)	Go/Nogo	X: easy discrimination Y: difficult discrimination	X = Y	X = Y	peak	150–450	100–0[1]	Pz
Falkenstein et al. (1996)	Choice Go/Nogo	W: false alarm errors X: choice errors Y: hand errors Z: finger errors	W = X Y > Z	W > X Y = Z	peak	200–400	?	Cz, Pz
Falkenstein et al. (2000)	Go/Nogo	X: many errors Y: few errors	X = Y	X < Y	peak	200–500	electrical zero	Cz, Pz
Hajcak et al. (2003)	Choice	X: many errors Y: few errors	X < Y	X = Y	peak	N_E (0–150)–525	200–0	Cz
Herrmann et al. (2004)	Flanker	X: many errors Y: few errors	X < Y	X = Y	peak	130–450	?	Cz
Kaiser et al. (1997)	Flanker	hypnosis-sensitive subjects X: under hypnosis Y: control	X = Y	X < Y	peak	?	200–0	Cz
Leuthold & Sommer (1999)	Compatibility Simon	X: high-salient information Y: low-salient information	X = Y	X > Y	mean	500–700[2]	100–0[1]	Cz
Mathewson et al. (in press)	Flanker Source memory	X: flanker-task errors Y: source-memory errors	X = Y	X > Y	peak	150–350	Flanker: 600–400 Source memory: 200–0	X: Cz Y: Fz, FCz
Nieuwenhuis et al. (2001)	Antisaccade	X: aware errors Y: unaware errors	X = Y	X > Y	peak	200–400	100–60	Fz, Cz, Pz
Rollnik et al. (2004)	Flanker	X: no rTMS Y: lateral frontal rTMS Z: medial frontal rTMS	X = Y > Z	X = Y < Z	mean	150–350	100–150[3]	Fz, Cz
Stemmer et al. (2001)	Flanker	X: genuine errors Y: simulated errors	X > Y	X > Y	peak	?	?	Cz
Ullsperger & Szymanowski (2004)	Flanker	X: speed instruction Y: accuracy instruction	X < Y	X > Y	mean	early P_E: 200–270 late P_E: 380–500	100–0	Cz, Pz

Notes: 1 = baseline in ms prestimulus, 2 = window in ms poststimulus, 3 = baseline in ms postresponse

Cramon, & Müller, 2002). Stemmer, Segalowitz, Witzke, and Schönle (2003) examined five patients with various lesions in and near the MFC and found a smaller Ne and Pe in some patients but no differences in others.

Results of one study suggest that aspects of personality might also influence Ne and Pe, but in opposite directions. Focusing on negative affect (NA), Hajcak, McDonald, and Simons (2004) found a larger Ne in high- compared to low-NA individuals. In contrast, the Pe was smaller in high- than in low-NA subjects. Children who scored high on a lie scale showed a larger Ne but a similar Pe in comparison to low-scorers (Santesso, Segalowitz, & Schmidt, in press).

Summary

Dopamine-deviant populations compared to controls show altered Ne amplitudes but generally little or no effect on the Pe. A (double) dissociation was also found in high-NA versus low-NA subjects. Lesion studies do not show a systematic pattern of dissociations between the Ne and Pe, but insufficient data are available from patients with focal MFC lesions to allow firm conclusions.

Experimental Manipulations

Various conditions seem to affect the Pe but not the Ne. Nieuwenhuis, Ridderinkhof, Blom, Band, and Kok (2001) studied errors in an antisaccade task, which is known to incur many reflexive saccade errors, many of which are not recognized as such by subjects. Whereas the Ne was present following both recognized and unrecognized errors (as determined with trial-by-trial subjective ratings), the Pe was present exclusively on trials on which subjects were aware of their error. In addition, posterror slowing of response times was only observed following subjectively recognized errors. Consistent with the Nieuwenhuis et al. study, highly hypnotizable subjects show strongly reduced Pe amplitudes under hypnosis compared to a control condition, whereas their Ne remains unaltered (Kaiser, Barker, Haenschel, Baldeweg, & Gruzelier, 1997). Furthermore, the Pe is smaller when error-inducing information is less salient than when salience is high (Leuthold & Sommer, 1999), whereas the Ne is independent of this salience manipulation (Leuthold, personal communication, cited in Elton et al., 2004). In contrast, in a study focusing on perceptual errors, no differences in either Pe or Ne amplitude were found between easy- and hard-to-distinguish stimuli (Elton et al., 2004).

Several studies have focused on the differences in Ne and Pe between individuals who make many errors and those making few errors. The findings are inconsistent: Some between-subject comparisons suggest that more errors lead to a smaller Ne whereas the Pe is not affected by the number of errors (Hajcak, McDonald, & Simons, 2003; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004). In contrast, others indicate that the number of errors does not influence the Ne whereas more errors result in a smaller Pe (Dywan, Mathewson, & Segalowitz, 2004; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Moreover, using a within-subjects comparison, Ullsperger and Szymanowski (2004) found a smaller Ne and a slightly larger Pe in a condition promoting speed over accuracy (i.e., resulting in more errors) than in a condition promoting accuracy over speed (resulting in fewer errors).

Examination of the effects of different tasks on the Pe and Ne reveals more consistency. Genuine errors elicited both a larger Pe and a larger Ne than errors that were made intentionally (Stemmer, Witzke, & Schönle, 2001) and than correct responses that were falsely classified as errors (Ehlis, Herrmann, Bernhard, & Fallgatter, 2005). Considering genuine errors only, there is some evidence that the Pe but not the Ne is sensitive to the type of task that is used. Comparing the Pe and Ne from a Go/Nogo task with those from a choice task, Falkenstein, Hohnsbein, and Hoormann (1996) found that the Pe was larger in the first than in the second task, whereas the Ne was similar for both tasks. Furthermore, the Pe was larger in a flanker task compared to a source memory task whereas the Ne was alike in both tasks (Dywan et al., 2004; Mathewson, Dywan, & Segalowitz, in press). In contrast, a comparison of finger and hand errors revealed a smaller Ne for finger errors than for hand errors whereas the Pe was similar for these two error types (Falkenstein et al., 1996).

Summary

Several studies indicate that the amplitude of the Pe, but not the Ne, covaries with the degree of awareness of the error or the salience of the error-inducing stimulus. In as far as error salience or significance depends on speed/accuracy balance, the results are mixed: In some studies the Pe is reduced when many (compared to few) errors are made, but in other studies this effect was not replicated. Intentional errors, which presumably are not experienced as very salient (affectively or cognitively), elicit a smaller Pe than do genuine errors.

Discussion

To investigate the functional significance of the Pe beyond the scattered and often apparently contradictory ob-

servations reported in the literature, we conducted a review of the available studies, examining patterns of associations and dissociations between the reported effects on the Pe and Ne. We included ERP studies published or accepted for publication before August 2005, if they reported the effects of focal brain lesions, individual differences, pharmacological interventions, or experimental manipulations on both the Pe and the Ne. The resulting pattern of findings leaves quite a few questions to be answered and inconsistencies to be resolved, emphasizing the need for cautious interpretation. Nonetheless, several interesting patterns emerge.

The most prominent observation is that in terms of antecedent conditions (experimental manipulations, individual differences), the Pe and Ne are remarkably dissimilar; our review reveals dissociations rather than associations. Dopaminergic factors serve as an example: While the Ne is heavily influenced by dopaminergic agents and shows differences between dopamine-deviant and control groups, the Pe appears largely insensitive to such factors. These patterns of divergence further emphasize the question of what the Pe reflects.

Falkenstein (2004) listed several (not necessarily orthogonal) hypotheses: the *affective-processing hypothesis*, which suggests that the Pe reflects the emotional appraisal of the error or its consequences; the *error-awareness hypothesis*, which proposes that the Pe reflects the conscious recognition of the fact that an error was committed; and the *behavior-adaptation hypothesis*, according to which the Pe reflects a process involved in remedial performance adjustments following errors (irrespective of whether such adjustments are driven by affective or cognitive aspects of error processing, or both).

The Affective-Processing Hypothesis

Realizing that an error was made may have emotional corollaries, and the neuroaffective processes involved in appraising the erroneous event may manifest themselves at the scalp as the Pe. Evidence that led to the proposal of the affective-processing hypothesis came from a study showing that subjects who made many errors exhibited a smaller Pe ("cared less") than subjects who made fewer errors (Falkenstein et al., 2000). However, other studies did not replicate this observation (e.g., Hajcak et al., 2003; Herrmann et al., 2004), and subjects scoring high on negative affect (NA) in fact have been reported to exhibit a *smaller* Pe than low-NA subjects (Hajcak et al., 2004).

Another source of evidence that might be taken as consistent with the affective-processing hypothesis derives from dipole source modeling of the Pe scalp topography (cf. van Boxtel, van der Molen, & Jennings, 2005; Van Veen & Carter, 2002). Using this technique, these authors inferred the neural generators of the Pe to be located in the rostral portions of ACC, which have been associated with affective processing (e.g., Bush, Luu, & Posner, 2000). Another study, however, suggested that the neural generator of the Pe was situated in the caudal ACC (Herrmann et al., 2004). In general, source modeling results must be interpreted with caution because the dipole source localization problem is underdetermined (the so-called "inverse problem"). This problem may be specifically relevant for the Pe: Although the broad scalp distribution may suggest the contribution of multiple generators, it is often easy to fit such a distribution with a limited number of relatively deep dipoles. Indeed, preliminary evidence from functional magnetic resonance imaging (fMRI) appears to point at other neural generators of the Pe, as will be discussed below. In sum, although it is too early to discard the affective-processing hypothesis, direct evidence in favor of this view is lacking.

The Behavior-Adaptation Hypothesis

The behavior-adaptation hypothesis also does not receive clear-cut support. Some evidence suggests that posterror adaptation (such as posterror slowing) is contingent upon the amplitude of the Pe elicited by the erroneous response (Hajcak et al., 2003; Nieuwenhuis et al., 2001), but such contingencies are not reported in all studies. In any case, this evidence is based on between-subject correlations. A way to test the behavior-adaptation hypothesis more firmly would be to compute *within*-subjects correlations between Pe amplitude and the degree of posterror slowing or the probability of an error on the subsequent trial (cf. Gehring, Goss, Coles, Meyer, & Donchin, 1993). Such analyses have not been reported yet.

It is worth noting that the apparent relationship between the Pe and posterror slowing observed by Nieuwenhuis et al. (2001) might be mediated in part by the awareness of errors, since posterror slowing may reflect a deliberate strategy, dependent on the conscious recognition of the error. Yet, posterror slowing has also been observed for errors that were not perceived as such (Rabbitt, 2002), and has in some studies been found to be related to Ne amplitude (Gehring et al., 1993). This raises the possibility that there may be two parallel systems for instigating posterror adaptations in information processing. A rapid preconscious system in the RCZ computes and signals the likelihood of reward, thereby guiding adaptive actions (Ridderinkhof et al., 2004; Rushworth, Walton, Kennerley, & Bannerman, 2004). A slower, more deliberate error-significance evaluation system might come into play when errors are sufficiently salient. The existence of two partially redundant posterror adaptation systems might explain why patients with damage to the RCZ still exhibit adequate

posterror slowing in tasks in which errors are particularly salient, such as Stroop and Go/NoGo tasks (Fellows & Farah, 2005).

The Error-Awareness Hypothesis

As reviewed above, two studies have indicated that the amplitude of the Pe, but not the Ne, covaries with the degree of awareness of the error (Kaiser et al., 1997; Nieuwenhuis et al., 2001)[2]. Other evidence appears consistent with these results. For instance, Pe amplitude appears to be positively correlated with the salience of the error-inducing information (Leuthold & Sommer, 1999). The smaller Pe in high- compared to low-NA individuals (Hajcak et al., 2004) may also reflect an influence of error awareness; high-NA individuals have been reported to underestimate their number of errors (Luu, Collins, & Tucker, 2000). However, it is not clear whether, in general, between-group differences in Pe amplitude can be interpreted to reflect differences in error awareness. Another unaddressed question is whether the Pe is the *expression* of error awareness, or reflects the processes that *lead to* error awareness. In sum, the error-awareness hypothesis awaits further investigation.

Taken together, the affective-processing hypothesis and the posterror adaptation hypothesis receive little support in the available literature. Furthermore, although there is some empirical support for the error-awareness hypothesis, this hypothesis is somewhat underspecified and its direct relevance for some of the Pe results reviewed here is not immediately apparent.

Similarity Between the Pe and P3b

Perhaps a fruitful way of addressing the functional significance of the Pe is by considering its similarity to the P3b, a slow positive wave in the stimulus-evoked ERP, peaking at 300–500 ms following motivationally significant stimuli (Picton, 1992). As noted by others (e.g., Leuthold & Sommer, 1999), the Pe has a similar morphology and broad midline scalp distribution as the P3b. The focus of the Pe scalp distribution (modus = Cz; see Table 1) is slightly more anterior than that of the P3b, and somewhat more posterior than the scalp distribution of the frontal P3a. However, the comparison between these scalp distributions is complicated by the large variability in Pe scalp distribution across studies (see Table 1). The only study that has directly compared the scalp distributions of the P3b and Pe obtained in the same experiment found that these did not reliably differ (Leuthold & Sommer, 1999). In any case, the latency of the Pe relative to the error is similar to the latency of the P3b relative to the eliciting stimulus event. A question to be examined in future research is whether the amplitudes of the Pe and P3b correlate across individuals.

With regard to antecedent conditions, P3 components (P3a, P3b) seem to be elicited by any motivationally significant stimulus event, including task-relevant, highly deviant, and novel stimuli (cf. Nieuwenhuis, Aston-Jones, & Cohen, 2005). This suggests that the Pe may constitute a P3b associated with the motivational significance of the error (cf. Rösler, 1983). This view is consistent with the finding of a larger Pe for more salient errors (Leuthold & Sommer, 1999), and with the finding that the Pe is small or absent when the subject does not explicitly recognize the error (Kaiser et al., 1997; Nieuwenhuis et al., 2001).

According to the context-updating hypothesis (Donchin & Coles, 1988), the P3b reflects the active consolidation or revision of a mental model of the environmental context of the observer. If stimuli deliver information that mismatches with some part of the context model, the model is updated, the amplitude of the P3b being proportional to the change in the model. Thus, in the light of this theory the Pe might reflect the updating of the context model in response to the error. A prediction that may be derived from this conjecture is that the Pe should correlate with indices of learning in tasks in which subjects are required to learn on the basis of trial-and-error (cf. Holroyd & Coles, 2002).

An alternative theory holds that P3 components reflect the noradrenergic potentiation of information processing, facilitating the response to motivationally significant events (Nieuwenhuis et al., 2005). In view of this theory, the Pe might be associated with the noradrenergic facilitation ("mobilization of resources") of the correction of the error. A testable prediction of this hypothesis is that detected errors, like motivationally significant stimulus events, are associated with increased activity of the noradrenergic system. Note that the hypothesis relates the Pe to the immediate (i.e., within-trial) correction of the error, whereas the behavior-adaptation hypothesis focuses on error-related performance adjustments on subsequent trials.

The further development and specification of hypotheses regarding the functional significance of the Pe would undoubtedly be stimulated by knowledge of the neural generators of the Pe. Neuroimaging studies (especially fMRI) may provide a promising approach for examining the neural generators of the Pe. Hester, Foxe, Molholm, Shpaner, and Garavan (2005) contrasted brain activity associated with subjectively recognized and unrecognized errors, a comparison known to affect the Pe

2 A third study (using an oculomotor inhibition task; Endrass, Franke, & Kathmann, 2005) confirms this pattern, although in this study the processes underlying the Pe might to some extent be confounded with processes of response inhibition (cf. Ramautar et al., 2004).

and not the Ne (Nieuwenhuis et al., 2001). Therefore, any region showing differential fMRI activity to recognized and unrecognized errors would be an important candidate generator of the Pe. The regions showing this pattern of activity were situated in bilateral prefrontal and inferior parietal cortices. Interestingly, these are some of the main brain regions implicated in the generation of the P3b (Soltani & Knight, 2000). This reinforces the notion that the Pe and P3b may reflect similar neural and functional processes.

A Note on Scoring Methods

It is possible that some of the inconsistencies reported in the Pe literature are a result of the wide variety in methods and criteria that have been used for measuring this component (in as far as reported; see Table 1). That is, in some cases a different choice of summary measure (peak amplitude vs. mean amplitude), measurement window, or baseline might have qualitatively altered the pattern of statistical results. For example, peak amplitudes seem to be more centered around Cz whereas mean amplitudes are distributed along the entire midline. Moreover, posterior maxima are more often associated with area measures (in 8 out of 12 studies) than with peak measures (in 6 out of 22 studies). Also, more complex tasks (e.g., source memory or switch tasks) seem to elicit a more frontally localized Pe amplitude. In general, while generic cookbook instructions as to scoring methods are not available and presumably not always applicable, it is recommendable that authors at least report and justify their choices in this regard.

Conclusion

Beyond the ubiquitous observation that the Pe and Ne are differentially sensitive to antecedent conditions, the available data do not allow strong inferences to be drawn about the functional significance of the Pe. Some empirical evidence has been garnered in support of the error-awareness hypothesis, although its general applicability is still limited. Direct evidence in favor of the affective-processing hypothesis or the posterror adaptation hypothesis is largely lacking.

More promising avenues for interpreting the functional significance of the Pe came from evaluation of its similarity to the P3b, suggesting that the Pe may constitute a P3b associated with the motivational significance of the error. This view was argued to be consistent with the findings that support the error-awareness hypothesis. Several empirically testable predictions were derived from the motivational-significance hypothesis. While the Pe picture remains a bit shadowy, we anticipate that more rigorous investigation of the neural generators of the Pe may cast some light on the road ahead.

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