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RESEARCH ARTICLE

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Error processing – evidence from intracerebral ERP recordings

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Abstract Over the last decade, several authors have described an early negative (Ne) and a later positive (Pe) potential in scalp event-related potentials (ERPs) of incorrect choice reactions. The aim of the present study was to investigate the intracerebral origin and distribution of these potentials. Seven intractable epileptic patients participated in the study. A total of 231 sites in the frontal, temporal, and parietal lobes were investigated by means of depth electrodes. A standard visual oddball paradigm was performed, and electroencephalogram (EEG) epochs with correct and incorrect motor reactions were averaged independently. Prominent, mostly biphasic, ERP complexes resembling scalp Ne/Pe potentials were consistently observed in several cortical locations after incorrect trials. The most consistent findings were obtained from mesiotemporal structures; in addition to P3-like activity found after correct responses, an Ne/Pe complex was generally detected after incorrect trials. The Pe had a longer latency than the P3. Other generators of Ne/Pe-like potentials were located in different regions of the frontal lobe. The latency of the Ne was shortest in parietal, longer in temporal, and longest in frontal regions. Our findings firstly show that multiple cortical structures generate Ne and Pe. In addition to the rostral anterior cingulate cortex, the mesiotemporal and some prefrontal cortical sites seem to represent integral components of the brain's error-checking system. Secondly, the coupling of Ne and Pe to a complex suggests a common origin of Ne and Pe. Thirdly, the latency differences of the Ne across lobes suggest that the Ne is primarily elicited in posterior and temporal, and only later in frontal regions.

Keywords Error processing · Event-related potentials · Intracerebral recordings

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Introduction

Over the last decade, several authors have described an early negative and a later positive potential in scalp event-related potentials (ERPs) of incorrect choice reactions (Donchin et al. 1988; Falkenstein et al. 1990, 1991, 1996, 2000; Gehring et al. 1993, 1995; Bernstein et al. 1995; Scheffers et al. 1996; Schalk et al. 2000). The error negativity (Ne, or error-related negativity ERN), which has been more frequently studied, shows a fronto-central maximum in scalp recordings, peaking about 80–180 ms after the incorrect motor reaction. The Ne has been interpreted as a correlate of error detection, and alternatively as a correlate of response checking itself (Falkenstein et al. 2000; Vidal et al. 2000). The later positive potential (Pe), which was formerly termed "error positivity" (Falkenstein et al. 1990), shows a centro-parietal maximum, peaking about 250 ms after the incorrect reaction. Pe seems to be partially independent of Ne. It has been shown that the Pe is not a delayed parietal P3, but rather a second P3 after an error (Falkenstein et al. 1996; Leuthold and Sommer 1999), which may reflect conscious error processing (Nieuwenhuis et al. 2001), or post-error processing such as updating of error context (Leuthold and Sommer 1999).

During the last years the issue of the neural generators of Ne and Pe has been raised by several researchers. Multichannel ERP studies as well as functional magnetic resonance imaging (fMRI) studies have provided converging evidence that the main source of the Ne is the anterior cingulate cortex (Dehaene et al. 1994; Scheffers et al. 1996; Miltner et al. 1998; Carter et al. 1998; Kiehl et al. 2000; Schalk et al. 2000; Scheffers and Coles 2000). However, knowledge of other neural systems involved in error processing and in producing Ne/Pe potentials in the human brain remains substantially limited. Recent lesion and ERP studies have provided evidence that also the lateral prefrontal cortex (Gehring and Knight 2000; Kiehl et al. 2000) and the basal ganglia (Falkenstein et al. 2001b) participate in Ne generation. Most recently, an fMRI study has suggested multiple foci of error-related activity in the frontal lobe (Ullsperger and von Cramon 2001). No study has yet explored the origin of the Pe.

The aim of the present intracerebral ERP study was to identify the cerebral sites activated during correct and rare incorrect motor reactions within a performed visual oddball task.

Material and methods

Subjects

Seven patients (six males and one female) ranging in age from 17 to 36 years (average age 25.7 years), all with medically intractable epilepsies, participated in the study. A comprehensive neuropsychological examination excluded cognitive disturbances and dementia in each patient. Depth electrodes were implanted to localise the seizure origin prior to surgical treatment. Each patient received 4–9 orthogonal multicontact electrodes using the methodology of Talairach et al. (1967). A total of 231 sites in the frontal, temporal, and parietal lobes were investigated. The number of sites per patient varied from 15 to 41. Standard MicroDeep semiflexible electrodes (Dixi Medical, Besançon, France) with diameters of 0.8 mm, lengths of each contact of 2 mm, and intercontact intervals of 1.5 mm were used for invasive electroencephalogram (EEG) monitoring. Contacts at the electrodes (5–15) were always numbered from the medial to the lateral side. Their positions were indicated in relation to the axes defined by the Talairach system using the ‘x, y, z’ format where ‘x’ is lateral, millimetres to midline and positive right hemisphere, ‘y’ is anteroposterior, millimetres to the anterior commissure line and positive anterior, and ‘z’ is vertical, millimetres to the anterior commissure-posterior commissure line, positive up. The exact positions of the electrodes and their contacts in the brain were verified using post-placement magnetic resonance imaging (MRI) with electrodes in situ. The recordings from lesional anatomical structures and epileptogenic zones were not included in the analysis. All subjects had normal or corrected-to-normal vision. Informed consent was obtained from each subject prior to the experiment and the study received approval from the Ethical Committee of the Masaryk University.

Procedure

Subjects were seated comfortably in a moderately lit room with a monitor screen positioned approximately 100 cm in front of their eyes. During the examination they were requested to focus their eyes continuously on the small fixation point in the centre of the screen and to minimise blinking. Since the error potentials (Ne as well as Pe) have been shown to be particularly large in easy Go/NoGo tasks (Falkenstein et al. 1996), a standard visual oddball paradigm was performed: two types of stimuli – target and frequent – were presented in the centre of the screen in random order. The experimental stimuli consisted of clearly visible yellow uppercase letters X (target) and O (frequent) on a white background. The stimuli were displayed on a black screen, subtended at a visual angle of 3°. The duration of stimuli exposure was constant at 200 ms, and the ratio of target to frequent stimuli was 1:5 (50 targets, 250 non-targets). The interstimulus interval varied randomly between 2 and 5 s. One block of 300 stimuli was presented, with targets and standards in random order. Subjects were instructed to respond to each target stimulus as quickly as possible by pressing a microswitch button in the dominant hand, and to simultaneously count the targets silently (Brázdil et al. 1999). No specific instructions were given to the subjects regarding erroneous responses during the experiment. Although participants always struggled to complete the task correctly (i.e. to respond only to the targets), some subjects occasionally made erroneous responses to the non-targets. The principal criterion for inclusion of the subjects in this study was a sufficient number of erroneous responses (i.e. microswitch-pressing) to the non-targets. In the patients involved, this number ranged from 5 to 18 with a mean of 9.3, which is known to be sufficient for obtaining clear scalp-recorded Ne and Pe (Falkenstein et al. 1999, 2001a) as well as intracranial ERPs (Roman et al. 2000). The mean error rate was 15.76%.

EEG recording

The EEG signal was simultaneously recorded from various intracerebral structures and occasionally also from the CPz scalp electrode (situated between Cz and Pz; three subjects), using a 64-channel Brain Quick EEG system (Micromed, Freiburg, Germany). (The CPz region was used to measure simultaneously scalp Ne and Pe with one electrode). All recordings were monopolar with respect to a reference electrode on the right mastoid. All impedances were less than 5 kΩ. EEGs were amplified with a bandwidth of 0.1–100 Hz and sampled at 128 Hz. Occasional EEG artefacts were rejected manually during off-line analysis and further processing was performed with artefact-free EEG periods. For each patient, EEG data from 30–42 recording channels were analysed using ScopeWin software. Two-second periods of EEG were averaged off-line using the motor response as the trigger (response-triggered averages, RTA). For each subject, EEG epochs with correct and incorrect motor reactions were averaged separately. The main ERP components in the latency range of –100 ms to +350 ms were independently identified by visual inspection by two of the study’s authors and quantified by latency measures. Occasionally intracerebral polarity reversals or steep voltage gradients were recorded, which suggested a focal origin of the waveform. The data from the recording channel (single electrode contact) with the most prominent P3-like and Ne/Pe-like responses were chosen for further analysis.

Results

In response to correct reactions, P3-like potentials (RTA-P3s) were repeatedly recorded from various intracerebral sites. The anatomical distribution of these RTA-P3s across the investigated brain structures did not generally differ from the distribution of the classical stimulus-triggered intracerebral P3s found in our previous study (Brázdil et al. 1999). Again, hippocampus, amygdala and parahippocampal gyrus were the most frequently involved sites within the temporal lobes. The mean latency (±SD) of the RTA-P3 in the mesiotemporal recording sites was 45.4±137.48 ms (after the motor response). In the investigated frontal lobe structures (orbitofrontal, dorsolateral prefrontal, cingulate and premotor cortices), slightly shorter latencies of the RTA-P3 were found (mean 12.6±66.34 ms). In the investigated parietal lobe structures, the latency of the RTA-P3 was even –49±105.84 ms relative to the motor response of the subjects. Similar latency relationships were observed in

Table 1 Mean latencies (related to motor responses) and standard deviations of recorded potentials

Anatomical site[a]	Latency (ms)		
	RTA-P3	Ne-like potential	Pe-like potential
Amygdala (4/5)	168±108.40	122±34.99	350.2±23.98
Hippocampus (8/12)	–10.7±145.19	85.7±60.64	324.4±120.40
Parahippocampal gyrus (4/5)	35±15.78	87±29.27	338.2±21.43
Mesiotemporal region (16/22)	45.4±137.48	95.1±50.99	334.3±87.32
Lateral temporal region (11/23)	–16.4±80.04	110.2±55.66	250.5±72.64
Orbitofrontal cortex (2/4)	–52±4	108±3	176.5±1.5
Dorsolateral prefrontal cortex (5/7)	–9.4±44.47	176±76.46	320.2±133.69
Anterior cingulate gyrus (4/5)	56.8±71.46	154.7±38.79	288.3±66.63
Premotor cortex (1/3)	75±0	70±0	244±0
Frontal lobe (12/19)	12.6±66.34	148.8±63.67	279.3±107.31
Temporal lobe (27/45)	20.2±121.37	101.3±53.46	300.2±91.44
Parietal lobe (3/3)	–49±105.84	16±136.89	196.7±179.88

[a] Number of positive observations of Ne/Pe complex/ number of investigations are given in parentheses

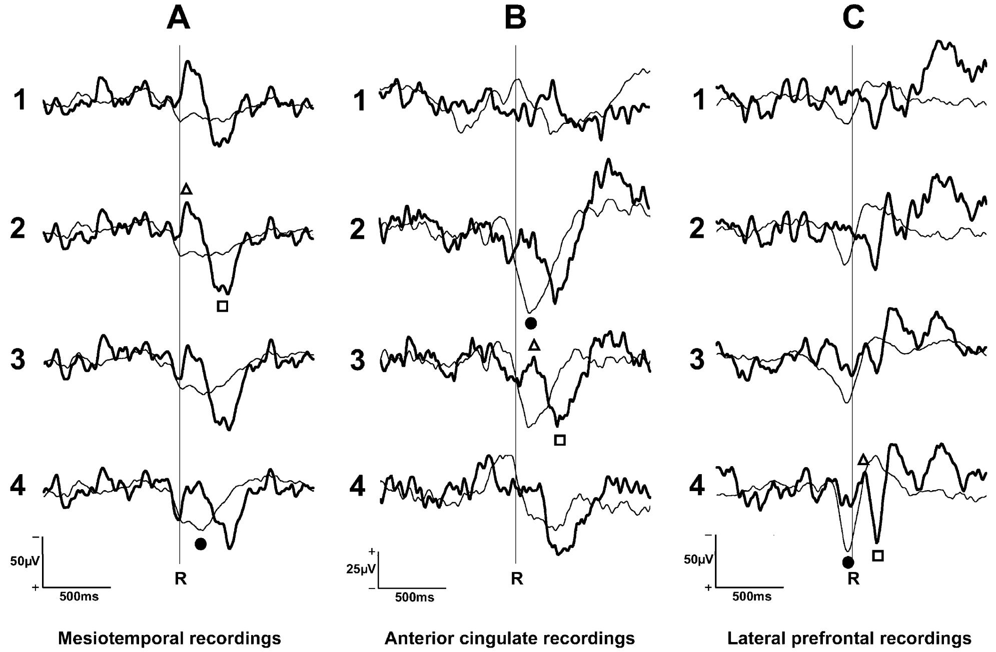

Fig. 1A–C Response-triggered averaged event-related potentials (ERPs) for correct (*thin lines*) and error (*thick lines*) trials. **A** Four adjacent electrode contacts from the mesiotemporal region (*1*,*2* parahippocampal gyrus: approximate co-ordinates in the Talairach axes of x=22 mm, y=–10 to –9 mm, z=–24 to –21 mm; *3*,*4* amygdala: x, y, z co-ordinates 23 mm, –8 to –7 mm, –19 to –16 mm). Subject L.M. with five erroneous responses; **B** Four adjacent electrode contacts from the anterior cingulate gyrus (area 32; x, y, z co-ordinates –2 to –14 mm, 3 mm, 45 mm). Subject Z.K. with six erroneous responses. **C** Four adjacent electrode contacts from the dorsolateral prefrontal cortex (area 47; x, y, z co-ordinates –19 to –31 mm, 41 mm, –10 mm). Subject M.P. with seven erroneous responses. RTA-P3s (*solid circles*), Ne-like potentials (*triangles*), and Pe-like potentials (*rectangles*) are indicated (*R* motor response)

several intracerebral studies for the stimulus-triggered P3 (Baudena et al. 1995; Halgren et al. 1995a, 1995b; Brázdil et al. 1999). The mean latency of the RTA-P3 across all recorded sites was 13.1±108.79 ms (for details see Table 1).

In the RTAs of incorrect reactions, a prominent, mostly biphasic, complex resembling scalp Ne/Pe potentials was consistently observed in several locations. The most consistent findings were obtained from bilateral mesiotemporal limbic structures (the amygdala, the hippocampus, and the parahippocampal gyrus). In these structures clear Ne/Pe wave-shapes were detected repeatedly with steep voltage gradients (Figs. 1, 2), which suggests a local source. The latencies of the Ne- and Pe-like potentials recorded in the mesiotemporal structures generally matched the latencies of the scalp-recorded Ne and Pe, if available (Fig. 2; Table 1). Other generators of Ne-like and Pe-like potentials were repeatedly recorded in the rostral anterior cingulate gyrus (area 32), the orbitofrontal cortex (area 11), and bilaterally in the dorsolateral prefrontal cortex (DLPFC; areas 9, 10, 47). Generally, the maximum of RTA-P3 and Ne/Pe complex was at the same location within a particular site. Occasionally however, the maxima of the RTA-P3 and

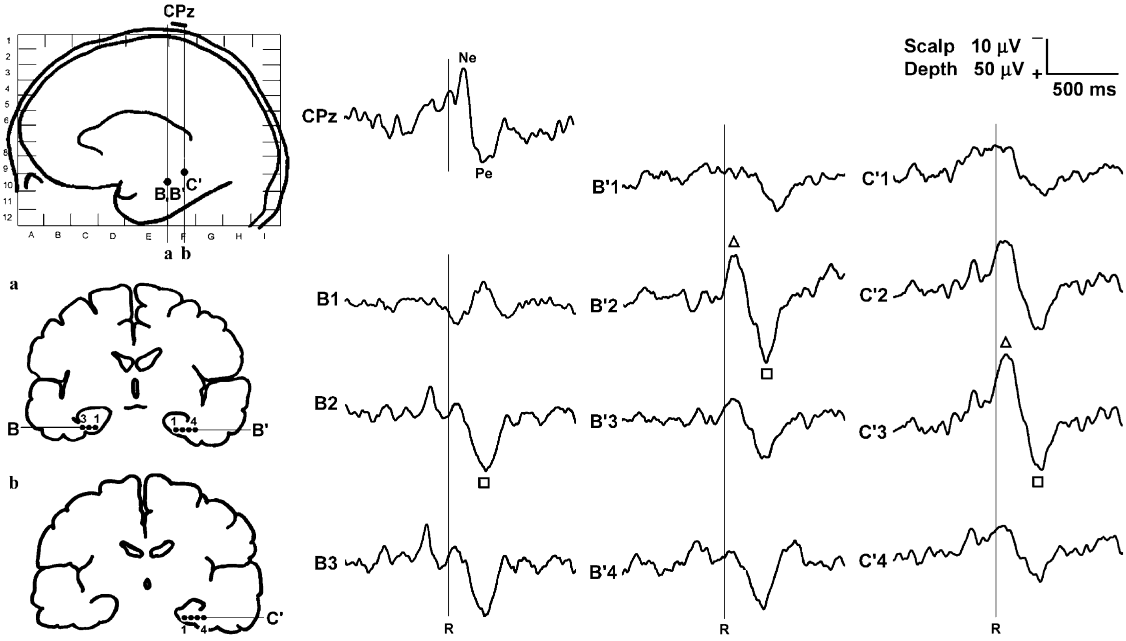

Fig. 2 Response-triggered difference waveshapes (error minus correct response-triggered averages) from simultaneous scalp (CPz) and intracerebral recordings. The latencies of error-related potentials in scalp recording (Ne, Pe) is entirely comparable with mesiotemporal findings (*B1–3* right-sided parahippocampal gyrus: Talairach's x, y, z co-ordinates 23 to 32 mm, –16 mm, –22 mm; *B'1–4* left-sided anterior parahippocampal gyrus: x, y, z co-ordinates –21 to –33 mm, –16 mm, –21 mm; *C'1–4* left-sided posterior parahippocampal gyrus: x, y, z co-ordinates –22 to –34 mm, –32 mm, –10 mm). Subject P.H with 17 erroneous responses. Ne-like potentials (*triangles*), and Pe-like potentials (*rectangles*) are indicated in the intracerebral recordings (*R* motor response)

of the Ne/Pe-complex differed slightly (with an average distance of two or three locations/channels) within the site (Fig. 1). Ne/Pe complexes were repeatedly recorded from area 40 and from the lateral temporal neocortex, but without characteristics indicating their local origin. An Ne/Pe complex was found only once in the premotor cortex. We never observed error potentials in recordings from the lingual gyrus or the sensorimotor cortex.

Although occasionally either Ne-like or Pe-like potentials predominated in the recording site, in most cases both deflections were present simultaneously. We never observed a prominent monophasic negativity without subsequent positivity after an incorrect response. Another important finding was that the Ne/Pe complex was never observed in structures in which no RTA-P3s could be recorded. In contrast, RTA-P3s were well recorded from structures in which Ne/Pe complexes were absent. The mean latency of the intracerebral Ne-like potential was 108.7±73.77 ms; the mean latency of the intracerebral Pe-like potential was 286.8±108.12 ms. The mean latencies of RTA-P3s, Ne-like and Pe-like potentials across the investigated anatomical sites and brain lobes are given in Table 1.

As anticipated from previous intracerebral ERP studies (Baudena et al. 1995; Halgren et al. 1995a, 1995b; Brázdil et al. 1999), the mean latency of the RTA-P3 was shortest in the parietal lobe, and longest in the temporal lobe (see above). The same observation was true for the Pe-like activity. In contrast, a discrepancy was observed in Ne-like activity, for which the mean latency was again minimal in the parietal lobe, but the maximum latency was obtained from the frontal lobe (Table 1).

Discussion

Our findings firstly support the hypothesis of a similar origin of Ne and Pe, as well as Ne/Pe and RTA-P3. However, this does not imply that the Pe is simply an after-effect (e.g. an inhibition) of the Ne, and hence reflects the same process (see below). Secondly, the fact that RTA-P3 as well as Ne/Pe could be recorded from different sites suggests involvement of multiple cortical structures in their genesis. Although Ne/Pe-like activities were never observed outside cortical structures in which P3-like activities were present, the opposite situation was occasionally found. This observation might suggest a lesser extent of the network that produces Ne/Pe potentials, and is thus a contradiction to the hypothesis that the Pe is merely a delayed P3. Hence, the present data further support the hypothesis that the Pe is an additional P3 (Falkenstein et al. 1996; Leuthold and Sommer 1999). However, another possible interpretation of our findings must be considered. RTAs to some few incorrect trials are extremely sensitive to any irregularities in the recording, and these RTAs could result in falsely negative findings. Six times more responses after correct reactions than after incorrect reactions were included in the averaging, and the risk of result distortion was therefore considerably lower. On the other hand, some observations of slightly diverse sites with voltage maximum of RTA-P3 and the Ne/Pe complex – mostly differing by about two or three adjacent electrode contacts (approximately 3.5–7 mm) – obviously suggest the heterogeneous anatomical origin and different functional significance of both ERP phenomena.

Unfortunately, neither the experimental paradigm used nor the characteristics of the obtained responses enabled us to assess whether Ne and Pe represent two separate phenomena or, ultimately, to study intracerebral sources of those potentials separately. Both deflections – the initial negative and the subsequent positive – were generally present in the same response. The only exception was a clear-cut finding of prominent monophasic Pe-like activity in both sides of the lateral prefrontal cortex in a single subject. The interpretation of this finding is necessarily very speculative due to its exceptional nature. Furthermore, there were no similar observations of Ne-like activity. In a recent review on error potentials, Falkenstein and colleagues argued against the possibility that the Pe "reflects the inactivation or reset of the Ne", and suggested instead that it is a further error-specific component (Falkenstein et al. 2000). Our data also show different behaviour for both components. The analysis of relationships among the mean latencies of ERPs (RTA-P3, Ne-like and Pe-like potentials) in the investigated brain lobes revealed discrepancies between Ne-like and Pe-like potentials, which suggests that the two components reflect different processes.

In the above mentioned literature, substantial error-related activation was not only found in the anterior cingulate, but also in the lateral prefrontal cortex (Kiehl et al. 2000; Gehring and Knight 2000), as well as other parts of the frontal lobe (Ullsperger and von Cramon 2001). In agreement with those findings, the present study revealed a clear-cut focal activation of lateral prefrontal cortex and anterior cingulate. More specifically, generators of Ne/Pe-like activities were found in the rostral anterior cingulate gyrus (area 32) and in the dorsolateral prefrontal cortex (areas 9, 10, 47). However, no generators were demonstrated in other parts of cingulate cortex (areas 23, 24). The latter finding suggests distinct roles of the different subdivisions of the anterior cingulate cortex in the detection of inappropriate responses. Our results show that apart from the anterior cingulate additional generators of error potentials are located in the orbitofrontal cortex and, particularly, in the mesiotemporal regions (Fig. 3). Because of the principal role of the latter in affective processing these findings can suggest the presence of such emotional aspects in error processing (Derryberry and Tucker 1992; Hariri et al. 2000; Simpson et al. 2000). Similarly, the frontal lobe and its cortico-cortical connections appear to be significant for emotional control, and for monitoring the effect of actions on the external environment (Stuss and Benson 1986). The orbitofrontal cortex is mainly involved in the modulation of impulsivity (Fuster 1995; Bechara et al. 2000). Different subdivisions of the anterior cingulate cortex may also interact with other cortical structures as a part of the circuits involved in the regulation of mental and emotional activity (Bush et al. 2000). Another important issue in the topic of error processing represents its potential lateralization. We did not find any laterality in our recordings, neither in mesiotemporal regions nor in lateral prefrontal cortical sites, or in the anterior cingulate gyrus. This is in agreement with our previous study where we also found no lateralization for the generators of the visual P3 (Brázdil et al. 1999).

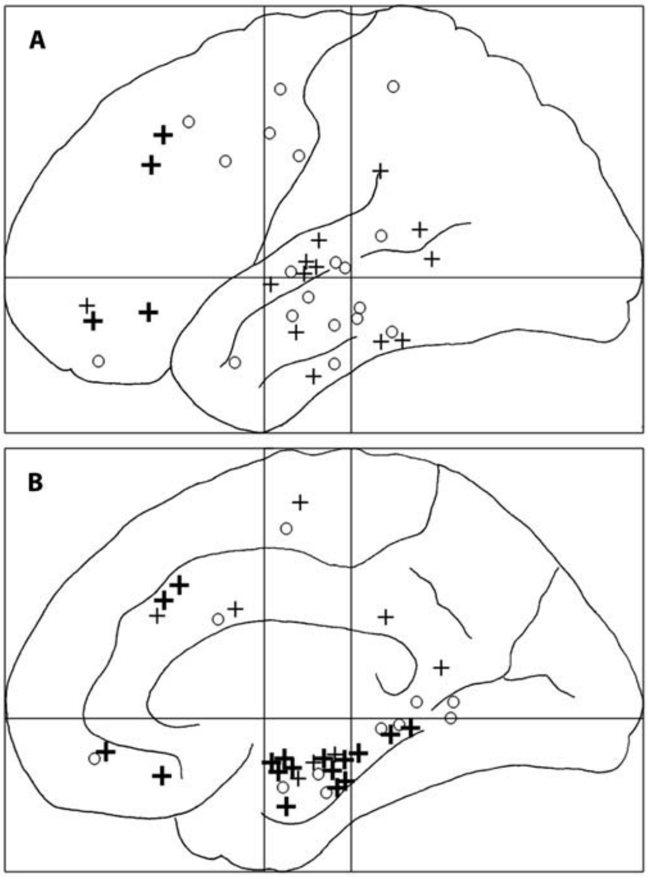

Fig. 3A,B Cortical recording sites of the Ne/Pe-like potentials. **A** lateral view of the brain. **B** medial aspect. *Thick crosses* show the sites of the local generator of the Ne/Pe-like potentials, *thin crosses* show the sites with potentials present, and *circles* indicate negative findings

Even though the present study was limited by the experimental paradigm, the results obtained clearly suggest that, in addition to the rostral anterior cingulate cortex, the mesiotemporal regions, the orbitofrontal cortex and the dorsolateral prefrontal cortex represent integral components of the brain's error checking system. Their contribution to the genesis of scalp Ne potential remains a subject of some speculation. Recent evidence suggests that the Ne, as measured at the scalp, consists of (at least) two components (Falkenstein et al. 2000; Christ and Falkenstein 2000). Gehring and Knight (2000) suggest that at least two regions, namely the lateral prefrontal cortex and the anterior cingulate cortex, seem to contribute to the generation of the Ne. In contrast, based on a recent study on the genesis of scalp P3 potential, mesiotemporal activities make only a limited contribution to the scalp recordings (Brázdil et al. 1999). Finally, a recent fMRI study clearly shows multiple generators to be active during error trials relative to non-error trials (Ullsperger and von Cramon 2001). More extensive depth ERP studies are therefore needed to verify our results and to provide a detailed investigation of other parts of the brain. Moreover, a more extended task battery is necessary to disentangle error-related activity from other related activity, such as inhibition or conflict (Carter et al. 1998; Ullsperger and von Cramon 2001).

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