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Acta Psychologica 101 (1999) 267–291

acta psychologica

ERP components in Go/Nogo tasks and their relation to inhibition

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Abstract

In visual Go/Nogo tasks the ERP usually shows a frontal negativity after Nogo stimuli ("Nogo-N2"), which possibly reflects an inhibition process. However, the Nogo-N2 appears to be very small after auditory stimuli, which is evidence against the inhibition hypothesis. In the present study we tested this hypothesis by evaluating performance differences between subjects. Assuming that for Ss with a high false alarm rate the inhibition process is weakened and/or delayed, they should reveal a smaller and/or later Nogo-N2 than Ss with a low false alarm rate. This prediction was confirmed, which supports the inhibition hypothesis. However, the Nogo-N2 was again much smaller and had a different topography after auditory than after visual stimuli despite similar performance in both modalities. This modality asymmetry was explained by assuming that the inhibitory mechanism reflected in the Nogo-N2 is located at a pre-motor rather than at the motor level. In the second part of the study we compared the Nogo-N2 with a similar phenomenon, the error negativity (N_e), which occurs in trials with commission errors (false alarms). Earlier work suggests that the N_e is a correlate of error detection or inhibition. This raises the possibility that the N_e is a delayed Nogo-N2, i.e., the N_e may reflect a late and hence unsuccessful attempt to inhibit the response after a nontarget. However, the N_e amplitude showed no difference between performance groups and stimulus modalities, as found for the Nogo-N2. Moreover, N_e and Nogo-N2 had different scalp topographies. This suggests that different mechanisms and generators underlie the N_e and the Nogo-N2. © 1999 Elsevier Science B.V. All rights reserved.

PsycINFO classification: 2530

Keywords: Response inhibition; Event-related brain potentials; Error negativity; Nogo-N2

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PII: S0001-6918(99)00008-6

1. Introduction

1.1. Nogo-N2 and Nogo-P3

Event-related potentials (ERPs) reflect sensory, cognitive, and motor processes or mechanisms (Donchin, 1984). By using the classical Go/Nogo-task several ERP researchers claim to have found ERP correlates of a frontal inhibition mechanism. In the Go/Nogo task the subjects have to respond to one stimulus (or stimulus category, Go-stimuli) and to refrain from responding to the other stimuli (Nogo-stimuli). Most of the Go/Nogo ERP studies used visual stimuli. Pfefferbaum, Ford, Weller and Kopell (1985), using visual letter and symbol stimuli, found a P300 with maximum at Pz on Go trials, and equal P300 amplitudes at Cz and Pz for Nogo trials. Moreover, in Nogo trials a negative shift with Fz maximum was superposed on the positive-going flank of the P300. A similar shift was found when the Ss only had to count the Go stimuli. Kok (1986), using visual letter stimuli, found larger late positive components in Nogo than in Go trials at Cz. Moreover, at about 400 ms he found a negativity with maximum at Fz, which was larger on Nogo than on Go trials. Jodo and Kayama (1992), using visual LED stimuli, observed a short negative component with maximum at Fz in Nogo trials, which became larger with higher time pressure. Eimer (1993), using a visual cuing task, also found a negativity in Nogo trials with maximum at Fz, which was larger for attended (cued) than for unattended (noncued) stimuli. Nogo stimuli also elicited a larger P300 than Go stimuli at Fz and Cz. Roberts, Rau, Lutzenberger and Birbaumer (1994), using a warned Go/Nogo paradigm with visual letter stimuli, found a strong enhancement of the P3 at Fz and Cz, and (not explicitly reported) a more negative N2 at Fz for Nogo compared to Go trials. Thorpe, Fize and Marlot (1996), using picture stimuli, found a phasic negativity in Nogo trials which began already at about 150 ms after stimulus onset, though the reaction times were about 450 ms long. Kopp, Mattler, Goertz and Rist (1996b) modified an Eriksen-task (Eriksen and Eriksen, 1974) by adding Nogo trials. These authors compared ERPs in Nogo-trials that were either specifically primed (by arrows) or unspecifically primed (by rectangles). By this technique, motor-related Go/Nogo differences could be excluded. Nevertheless, a more negative N2 was observed in specifically primed trials, which require specific inhibition. In contrast, Kopp et al. could not find a fronto-central enhancement of the P3 in specifically vs unspecifically primed Nogo trials.

In sum, the studies using visual Nogo-tasks report two effects in Nogo-vs Go-ERPs: (1) a negative shift with maximum at Fz with a latency of 150–400 ms, the "Nogo-N2", and (2) (less consistently) a positive shift with maximum at Fz and Cz with a latency of 300–500 ms, the "Nogo-P3".

1.2. Interpretational problems

The Nogo-N2 possibly reflects a frontal inhibition mechanism which is active on Nogo trials. This inhibition hypothesis is e.g. suggested by Jodo and Kayama (1992), who argued that higher time pressure should increase inhibition and hence

the Nogo-N2, as well as by Kopp et al. (1996b), who argued that specific (but not unspecific) response priming requires inhibition and hence reveals a Nogo-N2. The inhibition hypothesis is further corroborated by animal studies (Sasaki & Gemba, 1986; Sasaki, Gemba & Tsujimoto, 1989). These authors trained monkeys to perform hand movements in a visual Go/Nogo task. At about 150 ms after Nogo stimuli, they found a sharp surface negative potential in the dorsal bank of the principal sulcus, a formation in the frontal cortex. The assumption that this potential is linked to inhibition was strongly supported by the second study of these authors (Sasaki et al., 1989). The source of the "Nogo"-potential, i.e., the caudal-dorsal principal sulcus, was stimulated by electrical bursts with different time lags after a Go stimulus. The stimulation caused a delay and even suppression of the motor activity normally occurring after the Go stimulus, and mainly so when the stimulation was given at about 150 ms after the Go stimulus, i.e., the time when the Nogo-potential usually appears after a Nogo stimulus. Hence it appears that the frontal cortex is the source of an inhibition potential, which may be reflected in the Nogo-N2 in humans. However, there is a fundamental problem with this hypothesis: after auditory stimuli a Nogo-N2 is hardly seen. Karlin, Martz and Mordkoff (1969), who recorded from Cz only, rather found a more negative ERP in Go than in Nogo trials in the N2 latency range. Hillyard, Courchesne, Krausz and Picton (1976) found no Go/Nogo difference for the auditory N2 at frontocentral and frontal leads. In an auditory cuing task, Schröger (1993) found a slightly more negative N2 after Nogo than after Go stimuli, if the stimuli were attended (cued). Falkenstein, Koshlykova, Kiroj, Hoormann and Hohnsbein (1995) found no significant Nogo-N2 after auditory stimuli in an audio-visual Go/Nogo task. In sum it appears, that the Nogo-N2 is either absent or very small after auditory stimuli, which is evidence against the inhibition hypothesis.

Also for the Nogo-P3 an inhibition hypothesis was suggested by several authors (e.g. Karlin et al. (1969); Roberts et al. (1994)). However, this hypothesis also faces two problems. The first is a methodological problem due to possible overlap of movement-related activity (see below). The second, more serious problem is the long latency of the Nogo-P3 with respect to the Go-response. In our earlier study (Falkenstein et al. 1995) the Nogo-P3 peaked about at or shortly after the overt response. In the study of Eimer (1993) the frontocentral P3-enhancement occurred far later than the overt response. Assuming the timing of processing of Nogo-and Go-stimuli to be roughly the same, even the onset of the Nogo-P3 seems to be too late for an inhibition mechanism. However, the "Nogo-P3" could reflect the reset or closure of a preceding inhibition process, which could itself be reflected in the Nogo-N2. In any case, the frequent presence of the Nogo-P3 deserves further study of the question whether it is linked to inhibition.

1.3. Methodological problems

The comparison of Go and Nogo trials has a methodological problem: preparatory and movement-related activity might differentially overlap Go-and Nogo ERPs and thereby influence the Go-Nogo difference. Preparatory activity (e.g. the

movement-preceding negativity; Brunia, 1998) is supposed to be present in both Go and Nogo trials, but perhaps with different time courses. Movement-related activity, which mainly consists of a short negativity before a key press (motor potential, MP) and a positive reafferent potential after the response (Kornhuber & Deecke, 1965; Shibasaki, Barrett, Haliday & Halliday, 1980), is present in Go but not in Nogo trials. Due to variance of the RT the negative MP may interfere with the subsequent positive reafferent potential and hence be attenuated in the stimulus-locked average. Nevertheless, a residual of the MP may induce a negative shift before the Go-response and hence reduce the Go-Nogo-difference. On the other hand, movement-preceding negativities may persist longer in Go than in Nogo trials, which could also reduce the Go-Nogo-difference. These influences would be similar as far as the reaction times do not largely differ across conditions to be compared, whereas in conditions with largely different RTs, a differential influence of movement-related potentials is likely, which has been shown in a simulation study by Kok (1988).

However, any influence of movement-preceding or motor potentials on the Go-Nogo-difference should be independent of stimulus modality. Hence the strongly reduced or lacking Nogo-N2 after auditory stimuli, as reported above, is evidence against a substantial movement-related influence on the Go-Nogo-difference. Also, the studies using counting (Pfefferbaum et al., 1985) or specific vs. unspecific priming (Kopp et al., 1996b) suggest that the Nogo-N2 exists (with an amplitude comparable to conventional Go/Nogo tasks) also when motor-related Go/Nogo differences are excluded.

However, motor potentials may be more critical for the Nogo-P3, since this frontocentral enhancement often coincides in time with the response in Go trials. Assuming a negative MP in Go trials, which is absent on Nogo trials, the Nogo ERP should be less negative than the Go-ERP at central electrodes shortly before and about at the time of the key press, which is in fact observed. However, the presence of a Nogo-P3 even in the absence of an overt response (e.g. Pfefferbaum et al., 1985) shows that the Nogo-P3 cannot be solely due to MPs.

In sum, the early Nogo-Go difference (Nogo-N2), is not likely to be substantially influenced by movement-related activity, while the later Nogo-Go difference (Nogo-P3) my be influenced by such activity.

1.4. The error negativity in Go/Nogo tasks

In Go/Nogo tasks two types of errors can occur: a reaction after a Nogo-stimulus is called commission error (false alarm), the failure to elicit a response after a Go-stimulus is called omission error (miss). Shortly after errors (and particularly after false alarms) a negative ERP component occurs, the error negativity (N_e) (Falkenstein, Hohnsbein, Hoormann & Blanke, 1990; Falkenstein, Hohnsbein, Hoormann & Blanke, 1991,1995; Gehring, Goss, Coles, Meyer & Donchin, 1993; Scheffers, Coles, Bernstein, Gehring & Donchin, 1996). As a result of earlier studies, the N_e is thought to be a real-time correlate of error detection or error inhibition (Falkenstein et al., 1991; Falkenstein, Hoormann & Hohnsbein, 1997; Gehring et al., 1993; Kopp, Rist & Mattler, 1996a). Assuming the inhibition hypothesis for the Nogo-N2, and

the error inhibition hypothesis for the N_e, it seems possible that the N_e is simply a delayed Nogo-N2, i.e., the N_e may reflect a late and hence unsuccessful attempt to inhibit the false alarm. Hence Nogo-N2 and N_e may reflect the same inhibition process, as assumed by Kopp et al. (1996a).

1.5. The present study

In light of the strong evidence for the inhibition hypothesis for the Nogo-N2 from visual data, this hypothesis was tested with visual and auditory stimuli. To enhance the Nogo-N2, a time pressure regimen was administered (Jodo & Kayama, 1992). A further reason for the time pressure was to induce a sufficient error rate (see below). Moreover, attention towards the modalities was manipulated, since this manipulation is also likely to enhance the Nogo-N2 (Eimer, 1993; Schröger, 1993). In our earlier study, the Ss had to attend both modalities (crossmodal divided attention, DA). However, in DA, more attention may have been allocated to the visual than to the auditory modality (Posner, Nissen & Klein, 1976; Hohnsbein, Falkenstein & Hoormann, 1998), which may have attenuated the auditory Nogo-N2 in that study. Hence, a focusing of attention on the auditory modality could enhance a possible auditory Nogo-N2. The inhibition hypothesis was tested by comparing the Nogo-N2 for Ss with a high false alarm rate (POOR) and Ss with a low false alarm rate (GOOD). Under the assumption that the inhibition process in Nogo trials is weaker in POOR Ss, they should reveal a smaller Nogo-N2 compared to GOOD Ss, if the Nogo-N2 reflects inhibition. The same group comparison was performed for the Nogo-P3 in order to assess its possible relation to inhibition. The Nogo-P3 was evaluated at Fz in order to reduce a possible MP influence (which should be maximum at Cz). In sum, the first aim of the present study was to test the inhibition hypothesis both for the Nogo-N2 and the Nogo-P3.

The second aim of the present study was to test the hypothesis that Nogo-N2 and N_e reflect the same inhibitory mechanism. To this end Nogo-N2 and N_e were measured in the same Go/Nogo-experiment and compared with respect to modality, topography, and group (GOOD/POOR) effects.

2. Methods

2.1. Subjects

Ten young subjects (six male) with a mean age of 24.1 years (range 18–33 years) were paid for participating in the study. They had normal visual acuity and no known auditory deficits. All Ss were right-handed, as verified by a German version of the Edinburgh Inventory (Oldfield, 1971).

2.2. Stimuli

The stimuli were single letter (*F* and *J*) that were presented visually and acoustically. The visual letters (0.5° high at a viewing distance of 57 cm) were presented for

200 ms just below a fixation spot in the middle of a visual display unit (VDU). For the auditory presentation, the digitized time course of the spoken letters *F* and *J* (german pronunciation) was read from the RAM of a microcomputer and presented diotically by Sennheiser HD 425 headphones. (The intensity of the *F* was 55 dB SPL, the *J* was adjusted to seem equally loud to the Ss.) The auditory presentation of a letter took 300 ms. The luminance of the visual stimuli was so adjusted that their brightness was judged to be equivalent to the loudness of the auditory stimuli. The adjusted luminance was about 50 cd/m^2, the contrast was 0.9.

2.3. Procedure

Each subject participated in four sessions. The first two were training sessions (without EEG recording) that were administered to reach a stable level of performance. The reported performance and EEG data were collected in the last two sessions. The subjects sat on an office chair in front of a desk with the VDU within a dimly lit, sound attenuated, electrically shielded room. The viewing distance of 57 cm was held constant by a chin support. The subject's hands rested on a keyboard with the left and right index fingers touching the keys *F* and *J*. Within each block the stimuli were presented one at a time with equal probability for *F* and *J*. The interval between the onset of successive stimuli was randomized with a mean of 1500 ms and a range from 1050 to 1950 ms with equal probability. Visual and auditory stimuli were interlaced at random within a stimulus block, with equal probability for each modality. Two hundred stimuli (50 *Js* and 50 *F* for each modality) were administered in each of six blocks which had an identical stimulus structure. However, the tasks assigned to the stimuli differed: In block 1 the subjects had to attend both modalities (divided attention [DA]): they had to respond to each *F*-stimulus by pressing the left-hand key, and to withhold the response to each *J* stimulus. In blocks 2 and 3 the subjects had to attend exclusively to one of the two modalities (focused attention [FA]): In block 2 they had to attend only to visual stimuli and to respond to each visual *F*-stimulus by pressing the left-hand key, and to withhold the response to all other stimuli. In block 3 they had to attend only to auditory stimuli and respond to each auditory *J*-stimulus by pressing the right-hand key, and to withhold the response to all other stimuli. In the three other blocks, the assignment of target and nontarget to the letters and the response hands was reversed: in block 4 all *J*-stimuli were targets (reaction with right-hand), in blocks 5 and 6 visual *J*-(right-hand) resp. auditory *F*-stimuli (left-hand) were targets. So *F*-targets were always responded to with the left hand, and *J*-targets with the right-hand.

All blocks were run twice in both experimental sessions. A moderate time pressure was imposed by an acoustic signal (a 300 ms, 1 kHz, 60 dB SPL tone burst), which was sounded if the subject failed to respond within 500 ms after stimulus onset. The subjects were instructed to respond to targets fast enough to avoid the feedback tone at the risk of committing errors (false alarms). (The 500 ms deadline was chosen since all subjects were able to keep it in nearly all trials after the two practice sessions). The sequence of blocks was randomized across subjects and sessions. Between successive blocks a break of 1–5 min according to subject's preference was inserted. Prior to

each block and instruction was read to the subject concerning the task and the condition. The need to respond quickly enough to avoid the feedback signal was emphasized. The subjects had to keep their eyes open and directed towards the VDU in all conditions, which was monitored by a video camera.

2.4. EEG-recording

During the two experimental sessions the EEG was recorded with Ag/AgC1 electrodes from Fz, Cz, C3, C4, Pz, and Oz against linked mastoids. The forehead was grounded. The vertical EOG was recorded from the upper and lower rim of the left eye. The recorded activity was amplified (EEG: gain = 100,000; EOG: gain = 20,000) with a 1 pole high-pass (3 dB: 0.03 Hz) and a 10 pole Butterworth low-pass as anti-aliasing filter (3 dB: 60 Hz). EEG and EOG were digitized with 200 samples per second.

2.5. Artifact rejection and averaging

EEG epochs of 950 ms length were averaged using stimulus onset as trigger. The prestimulus epoch (baseline) was 50 ms long, the post-stimulus epoch 900 ms. For Go trials also the key press was used as trigger with a pre-trigger period of 300 ms. Only trials in which the subjects kept the RT limit of 500 ms were used. Epochs with samples exceeding the preamplifier range were rejected. Small blinks were corrected by the method of Verleger, Gasser and Möcks (1982). Incorrect trials, which were nearly exclusively commission errors (false alarms), were averaged separately with stimulus and key press as triggers. To reduce residual noise the averaged ERPs were smoothed with a digital low-pass filter (cutoff at 17 Hz; Ruchkin & Glaser, 1978), and pooled across sessions. To avoid any effects due to stimulus differences, only ERPs elicited by the same physical stimuli were compared and used for Nogo-Go-difference waveshapes. ANOVAs (see below) were run with BMDP 4V (Dixon, 1990).

3. Results

3.1. Behavioral data

In all blocks the amount of misses was negligible (below 1%). However, the rate of false alarms (relative to all responses) was high due to the time pressure, namely 11.2% after visual, and 8.9% after auditory stimuli for DA (The respective figures for FA are 10.2% for visual, and 8.2% for auditory stimuli). The distribution of error rates was markedly bimodal (cf. Table 1): five subjects (termed GOOD; three men and two women) had a low false alarm rate, while it was very high in the other five subjects (termed POOR).

For the DA condition the correct reaction times on Go trials (hits) were nearly the same after visual (355 ms) and auditory (360 ms) stimuli, and for both letters and

Table 1
Individual error rates (percent false alarms of all responses) of all ten Ss (S1–S10) and means in both performance groups (GOOD: low error rate; POOR: high error rate) for both stimulus modalities; female (f), male (m), visual (vis) stimuli, auditory (aud) stimuli (means values across both attention conditions (FA, DA)

GOOD							POOR					
S	1(f)	2(f)	3(m)	4(m)	5(m)	Mean	6(f)	7(m)	8(m)	9(f)	10(m)	Mean
Vis	4.3	4.2	2.6	7.1	4.8	4.8	15.0	15.4	19.0	19.1	14.5	16.6
Aud	3.2	5.3	4.4	2.4	3.2	3.7	13.4	14.7	9.8	15.9	13.2	13.4

hands (*F*/left-hand responses: 357 ms; *J*/right-hand responses: 358 ms). The reaction times for error (false alarm) trials were shorter: the values were 293 ms for visual and 342 ms for auditory stimuli (pooled across letter), i.e. the shortening was 62 after visual and only 18 ms after auditory stimuli.

For the FA conditions the correct RTs were shorter after auditory (343 ms) than after visual stimuli (365 ms). The error RTs were 302 ms for visual and 298 ms for auditory stimuli, i.e. the shortening was large and of similar size for both modalities.

The correct RTs were subjected to ANOVA with the within factors attention (A) (DA, FA) and modality (M) (visual, auditory), and the between factor group (GOOD, POOR). Overall the RTs were not different for both performance groups ($F < 1$). However, a significant group by modality interaction ($F(1,8) = 16.50$, $p = 0.0036$) revealed that the Go-RTs were shorter for POOR Ss (342 ms) than for GOOD Ss (361 ms) after auditory stimuli, and longer for POOR Ss (368 ms) than for GOOD Ss (352 ms) after visual stimuli. Moreover, a significant attention by modality interaction ($F(1,8) = 17.35$, $p = 0.0031$) revealed that under FA the Go-RTs were shorter after auditory than after visual stimuli, while under DA there was no modality difference.

3.2. ERPs

3.2.1. Nogo-N2 and Nogo-P3

The (stimulus-triggered) ERPs of the correct trials are shown in Fig. 1. After visual stimuli (left) the early components P170, N200, and N300 are seen, which are followed by a large P3-complex. After auditory stimuli (right), the early components N140, P230, and N300 are followed by a an early frontal and a late parietal P3-subcomponent. The visual ERPs clearly show the Nogo-N2 as a frontally maximum negative displacement in Nogo trials, which began around 200 ms and peaked at about 300 ms. The Nogo-N2 decreased steadily from Fz to Oz. In contrast, after auditory stimuli, only a small difference between Nogo and Go stimuli is visible with Fz-maximum around 300 ms. No other negative displacement for Nogo vs. Go trials occurred in the interval from the stimulus to the response (about 350 ms). The Nogo-P3 is seen as a slight frontocentral enhancement of the early part of the P3-complex in Nogo vs. Go trials. In the FA condition a quite similar picture was seen.

In order to highlight the Nogo-N2 and the Nogo-P3, difference waveshapes were computed between the ERPs of Nogo and Go trials (Nogo minus Go). Fig. 2 shows the difference waves at Fz for both attention conditions and stimulus modalities. After visual stimuli a large Nogo-N2 is seen with the same amplitude (about 5 mV) and latency (about 300 ms) for both attention conditions. After auditory stimuli the Nogo-N2 is generally much smaller than after visual stimuli, but it appears to be larger and to occur earlier for FA than for DA. The "Nogo-P3" showed no modality difference, but similar attention effects as the Nogo-N2.

Fig. 3 shows the Go and Nogo-ERPs at Fz separately for both performance groups in the DA condition. The visual ERPs clearly reveal a larger Nogo-N2 for GOOD than for POOR Ss, while the preceding P170 appears rather larger for POOR Ss. The same difference of the Nogo-N2 was seen (though on a smaller level) in the

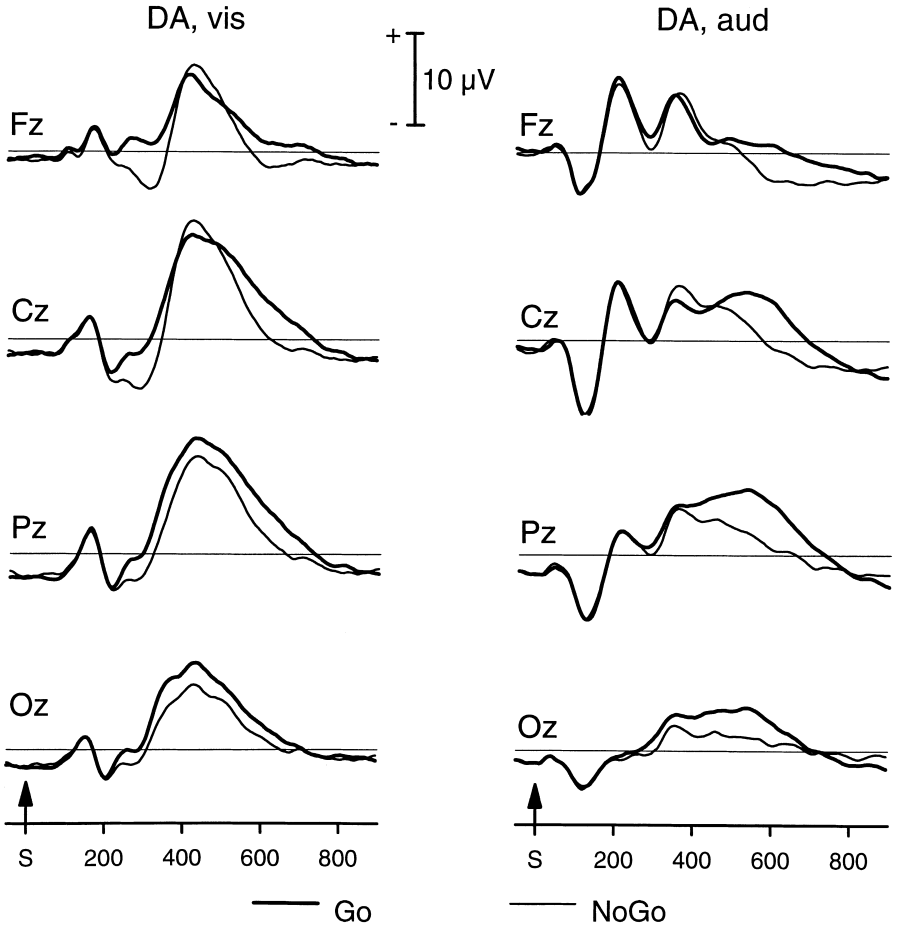

Fig. 1. Grand average ERPs of correct Go and Nogo trials after visual (left) and auditory stimuli (right) at the four midline electrodes (S = stimulus onset). The Nogo-N2 is seen as a distinct negative deflection with frontal (Fz-) maximum after visual, but is hardly seen after auditory Nogo-stimuli. The Nogo-P3 is seen as a slight fronto-central enhancement of the early P300 after Nogo stimuli. (DA: divided attention).

auditory ERPs. A frontal P3-enhancement on Nogo trials (Nogo-P3) was seen for GOOD but not for POOR Ss after visual stimuli, while after auditory stimuli a Nogo-P3 was seen only for POOR but not for GOOD Ss. The Nogo-Go difference waveshapes (Fig. 4) confirm this view: The Nogo-N2 is larger for GOOD than for POOR Ss. Moreover, the Nogo-N2 appears to begin earlier for GOOD than for POOR Ss. The Nogo-P3 shows opposite group effects in the two modalities, being larger for GOOD than for POOR Ss after visual stimuli, and larger for POOR than for GOOD Ss after auditory stimuli.

An even more pronounced group difference is seen in the FA condition (Fig. 5). The Nogo-N2 is large for GOOD and small for POOR Ss. Specifically after auditory stimuli, the Nogo-N2 has a relatively large amplitude for GOOD Ss, while it is very

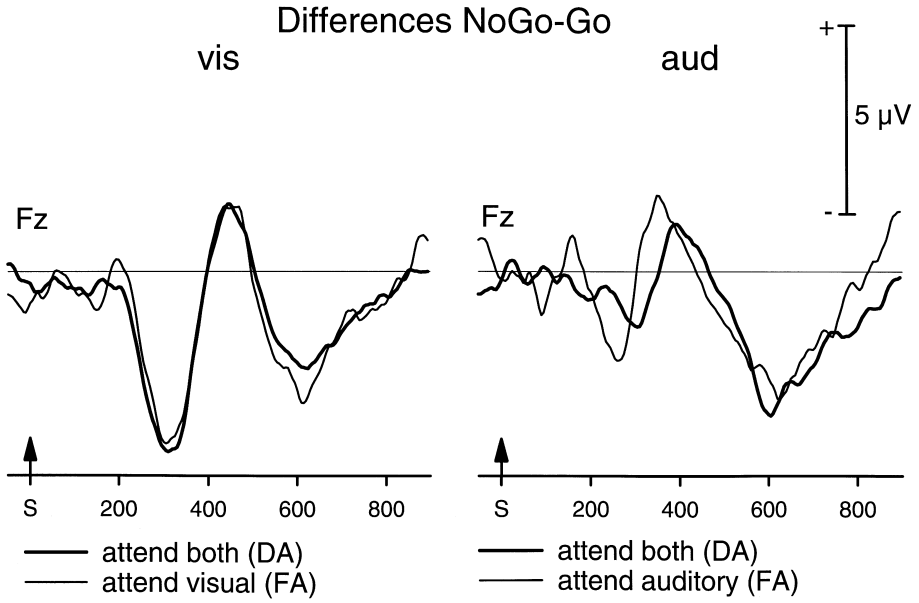

Fig. 2. Grand average difference waves of the correct trial ERPs (Nogo minus Go) at Fz for both attention conditions (DA: divided attention across both modalities: FA: focused attention on one modality) and stimulus modalities. While the focusing of attention does not influence the visual ERPs, the auditory Nogo-N2 (as well as the Nogo-P3) appears larger and earlier for FA than for DA.

small for POOR Ss. The Nogo-P3 shows no consistent amplitude difference across modalities or performance groups.

The Nogo-N2 and the Nogo-P3 were measured in the Fz-difference waves as the most negative trough between 200 and 400 ms and the most positive peak between 300 and 500 ms, respectively. The onset latency of the Nogo-N2 was measured by the segmented regression method (Schwarzenau, Falkenstein, Hoormann & Hohnsbein, in press). (In short, two straight lines were fitted to the difference waveshape between stimulus onset and the peak of the Nogo-N2, and the intersection latency of the lines was taken as onset latency.) The amplitude and latency values were subjected to a MANOVA with the factors performance group (P) (GOOD, POOR), attention (A) (DA, FA) and modality (M) (visual, auditory).

The Nogo-N2 was clearly larger after visual (6.7 μV) than after auditory stimuli (2.2 μV), as confirmed by a highly significant main effect of modality ($F(1,8) = 22.48$, $p = 0.0015$). Moreover a modality by attention interaction ($F(1,8) = 9.42$, $p = 0.0154$) revealed that after auditory stimuli the Nogo-N2 was larger for FA (2.8 μV) than for DA (1.7 μV), while there was no attention difference after visual stimuli (FA: 6.3 μV; DA: 6.9 μV). A modality by attention interaction ($F(1,8) = 7.98$, $p = 0.0024$) was also found for the latency of the Nogo-N2, showing that the auditory (but not the visual) Nogo-N2 peaked earlier for FA than for DA.

A significant group main effect ($F(1,8) = 9{,}63$, $p = 0.0146$) confirmed that the Nogo-N2 was larger for GOOD (5.8 μV) than for POOR Ss (3.1 μV). This group

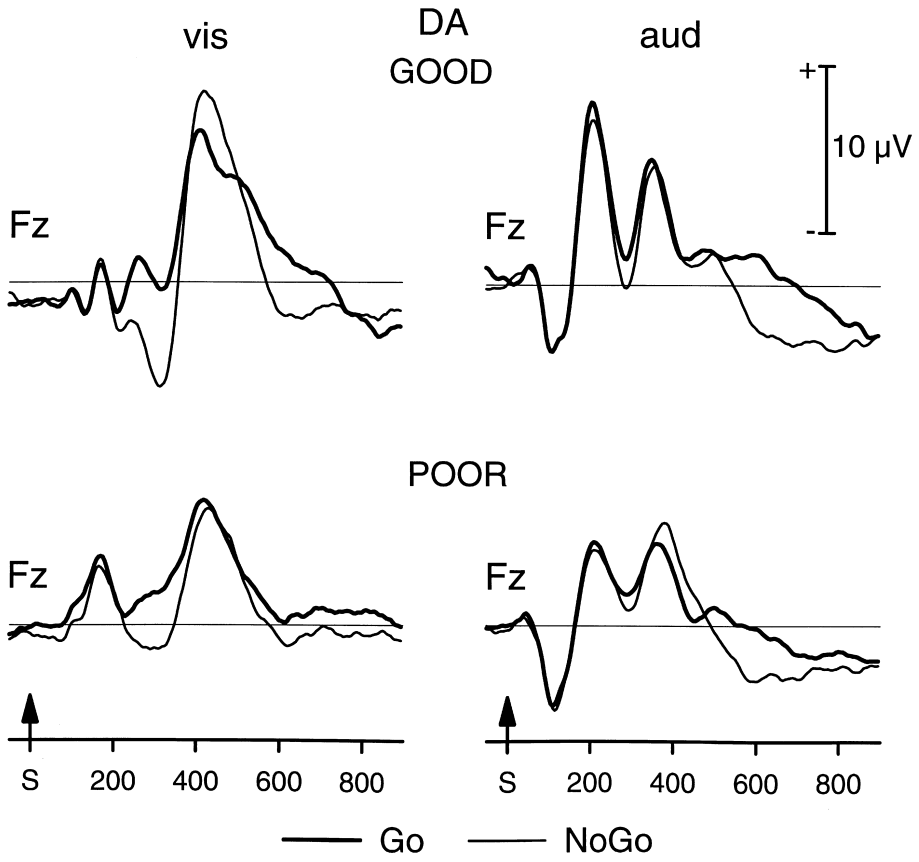

Fig. 3. Mean correct Go and Nogo ERPs separated for the performance groups (GOOD: low false alarm rate; POOR: high false alarm rate) in the divided attention (DA) condition. The Nogo-N2 is consistently larger for GOOD than for POOR Ss.

effect was not different for the modalities and the attention conditions, since there was no significant interaction of group with the other factors. The Nogo-N2 began also earlier for GOOD (198 ms) than for POOR (231 ms) Ss ($F(1,8) = 9.03$, $p = 0.0170$. There was no significant effect on the peak latency of the Nogo-N2.

The Nogo-P3 peaked later for visual (418 ms) than for auditory stimuli (380 ms) ($F(1,8) = 6.06$, $p = 0.0392$). Moreover, there was a small attention by modality interaction ($F(1,8 = 5.36$, $p = 0.0493$) showing a larger Nogo-P3 for FA (3.4 μV) than for Da (2.7 μV) after auditory stimuli, while this effect was reversed after visual stimuli. A third-order interaction ($F(1,8) = 6.88$, $p = 0.0305$) showed that this effect was only present for the GOOD Ss. No other effect was found to be significant for the Nogo-P3; in particular, there was no group main effect on the amplitude or latency of the Nogo-P3 ($F < 1$).

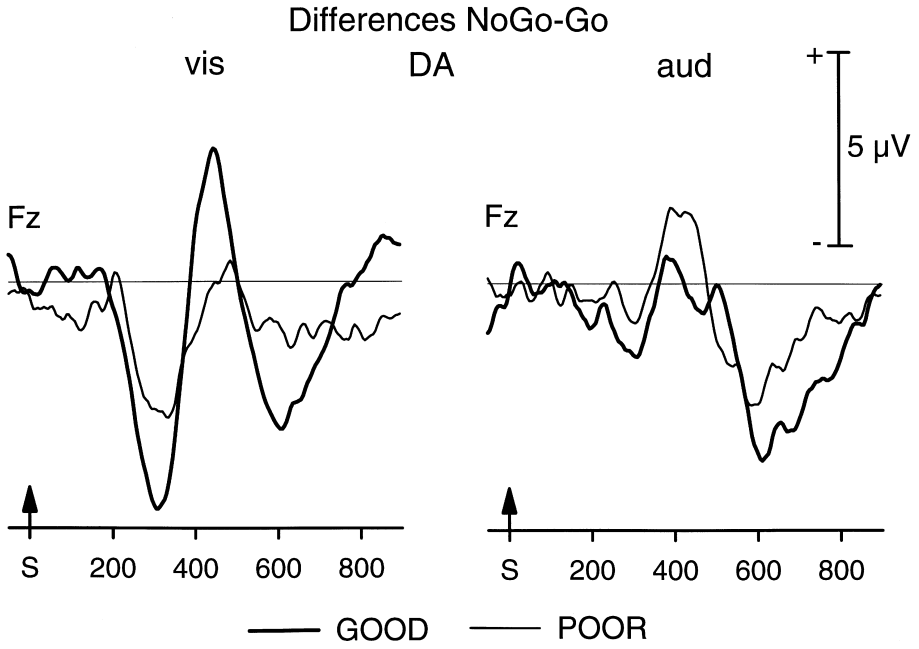

Fig. 4. Mean difference waves of the correct trial ERPs (Nogo minus Go) at Fz the performance groups. The Nogo-N2 is larger and appears to begin earlier for GOOD than for POOR Ss, while the Nogo-P3 shows no consistent effect across modalities.

3.3. Error negativity (N_e)

The N_e was evaluated only in the DA condition, where the false alarm rate was high enough to yield a sufficient amount of error trials (at least six per condition) for all Ss. From our previous work (e.g. Falkenstein et al., 1990,1991) it is known that the N_e is maximum over the central and frontal scalp, hence Pz and Oz data are not considered further.

The upper panels of Fig. 6 show the conventional stimulus-locked ERPs (stimulus triggered averages, STA) of the false alarm trials together with the Go and Nogo-ERPs (cf. Fig. 1) at Fz and Cz. After visual stimuli the N_e looks similar to the Nogo-N2, particularly at Fz. The main difference seems to be that the N_e begins and peaks later, and reaches a larger amplitude than the Nogo-N2. However, after auditory stimuli, the N_e appears at least as large as after visual stimuli, while the Nogo-N2 is small, as already reported. In order to cancel ERP activity common to false alarm and Go trials and then to compare the N_e with the Nogo-N2 we computed difference waveshapes between false alarm and Go ERPs (False alarm minus Go). The lower panels of Fig. 6 show the difference waveshapes. The Ne appears to be larger at Cz than at Fz. It is obvious that the N_e has the same shape and amplitude after visual and auditory stimuli. However, the N_e peaks later after auditory than after visual stimuli.

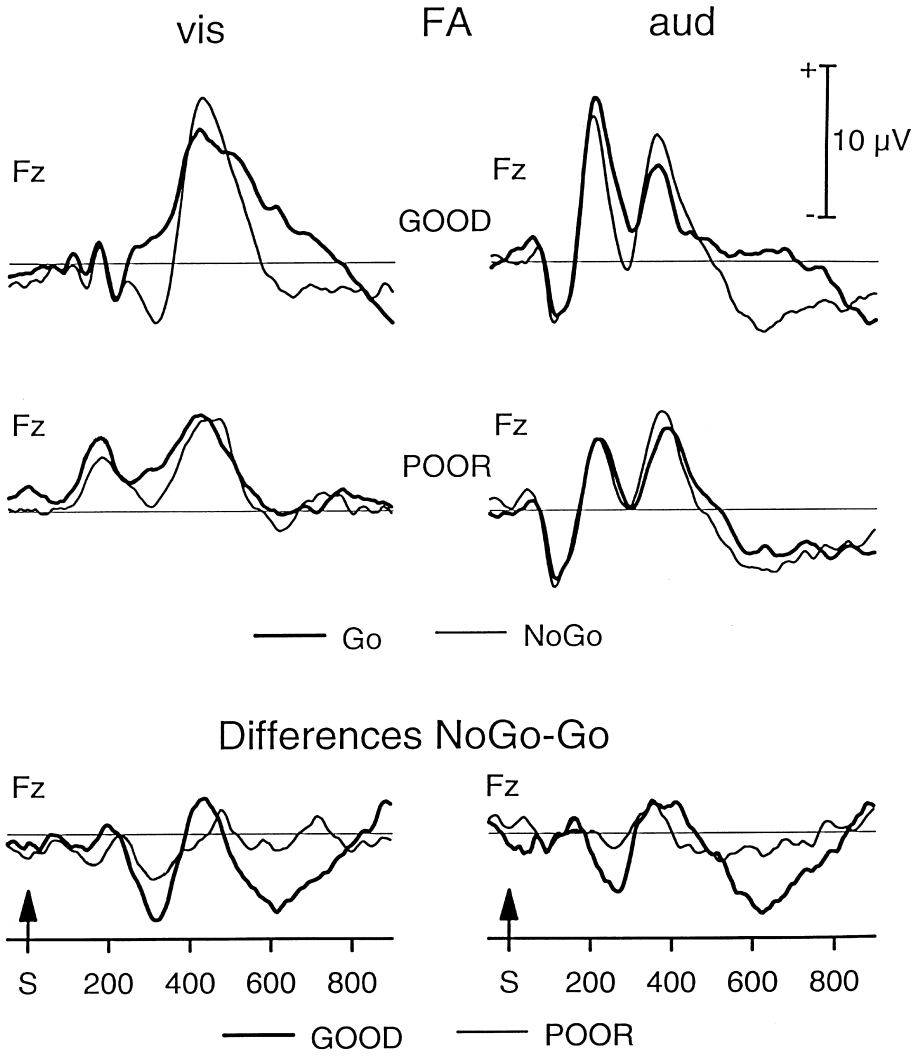

Fig. 5. Mean Go and Nogo ERPs (upper part) and difference waves (Nogo minus Go ERPs) (lower part) for the performance groups in the FA condition. Again the Nogo-N2 is larger and begins earlier for GOOD Ss. After auditory stimuli the Nogo-N2 is relatively large for GOOD Ss.

Since we have shown earlier that the N_e is time-locked more to the response than to the stimulus (Falkenstein et al., 1990,1991), we also computed response-locked averages of the False alarm and (for comparison and difference calculation) the Go-trials.

Fig. 7 shows the response-triggered averages (RTA) for error (False alarm) and Go trials as well as the difference waveshapes between them. The peak of the N_e is sharper and the peak amplitude larger in the RTAs than in the STAs. Again, no N_e

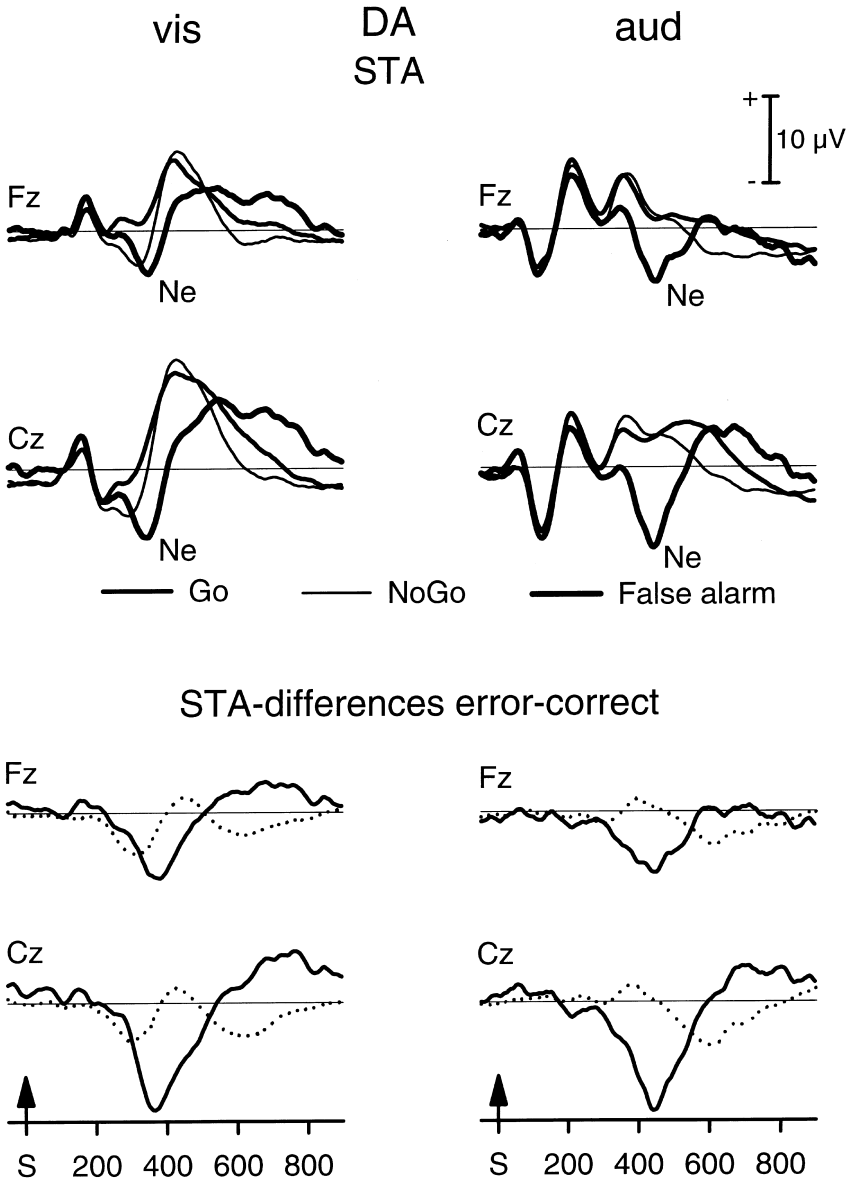

Fig. 6. Upper part: Grand average ERPs for False alarm trials (heavy lines) as well as for Go (medium lines) and Nogo trials (thin lines) (stimulus-locked averages, STA;DA condition) at Fz and Cz. The error negativity (N_e) is seen in false alarms as a prominent negative component, which peaks later than the Nogo-N2 in Nogo trials. Lower part: Difference waves error minus correct (False alarm minus Go ERPs). The N_e is highlighted as a large central negativity, which has the same amplitude for both modalities, and which peaks later after auditory than after visual stimuli. For comparison, the dashed lines show the Nogo-N2 (Nogo minus Go ERPs).

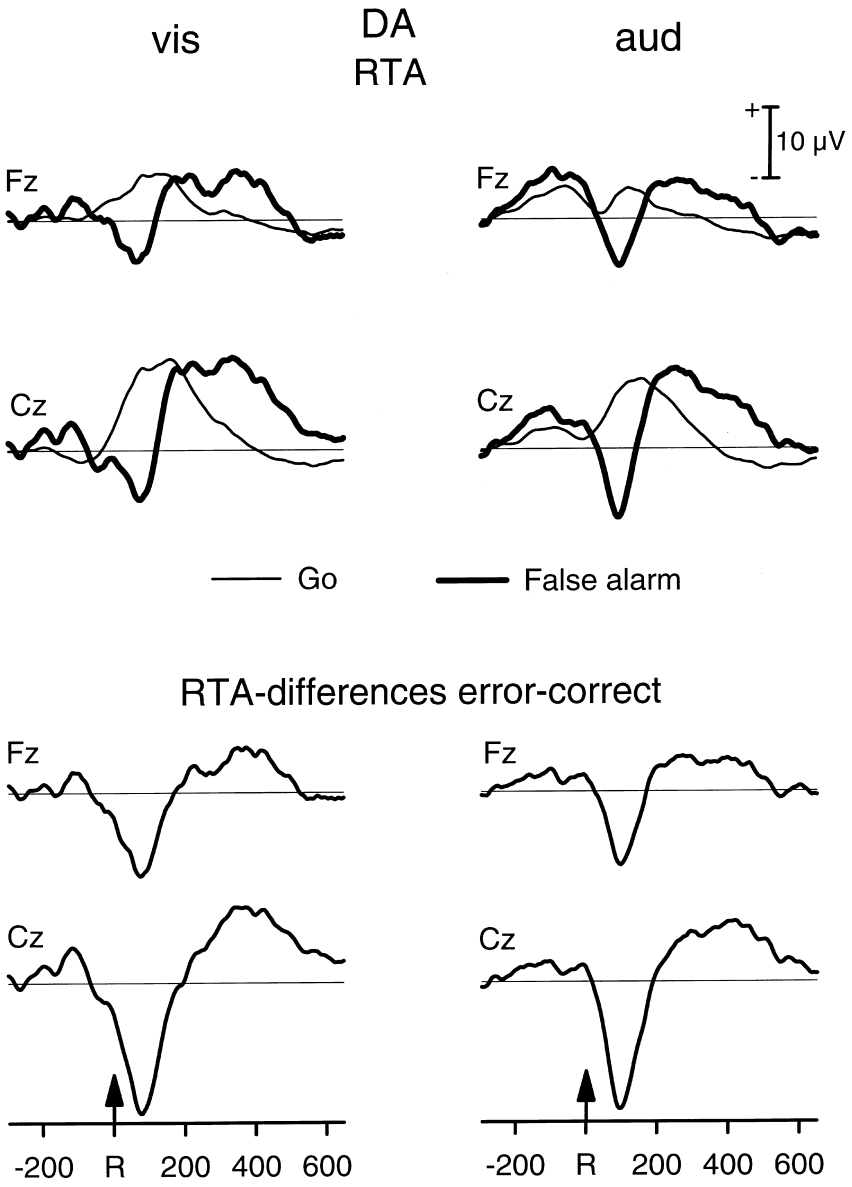

Fig. 7. Upper part: Grand average ERPs for False alarm trials (heavy lines) as well as for Go trials (thin lines) (response-locked averages, RTA; DA condition) at Fz and Cz. Lower part: Difference waves of the error minus correct RTAs (False alarm minus Go ERPs). The N_e is seen sharper in the RTAs than in the STAs of Fig. 6. Again, the N_e shows the same amplitude for both modalities and a latency delay after auditory stimuli.

amplitude difference between the modalities is seen, and the N_e appears to peak still later relative to the response for auditory than for visual stimuli.

Group differences of the N_e. For the comparison of the N_e across performance groups some methodical precaution is necessary. Since the error rate is, by definition, much smaller in GOOD than in POOR Ss, also the amount of error trials and hence EEG epochs for averaging is smaller. However, the amplitude of the averaged ERP is likely to be influenced, if the number of epochs is very different across conditions. To control for this imbalance, for each GOOD subject a companion POOR Ss was chosen by chance, and the number of error trials of the POOR subject was reduced to the respective number of GOOD subject by randomly selecting trails. This was done for both modalities. So the number of averaged sweeps was the same for both groups.

Fig. 8 presents the N_e separately for both performance groups. No obvious difference is visible with the exception of an apparently larger and sharper N_e for GOOD than for POOR Ss in the difference waveshapes. However, this apparent effect is likely due to different smearing of the N_e for the GOOD and the POOR Ss. In fact, in the RTA difference waveshapes (Fig. 9), in which latency jitter of the N_e is strongly reduced, no group difference of the N_e amplitude is visible for visual as well as for auditory stimuli. However, the N_e appears to be slightly delayed for POOR Ss.

The N_e was measured in the STA-and in the RTA-difference waveshapes relative to a baseline (the mean amplitude of the first 50 ms-epoch in the difference waveshapes). The amplitude and latency of the N_e were subjected to a MANOVA with the factors performance group (P) (GOOD, POOR) and modality (M) (visual, auditory).

In the STAs the N_e peaked much later after auditory (433 ms) than after visual (353 ms) stimuli ($F(1,8) = 28.82$, $p = 0.0007$). This effect proved to be significant also in the RTAs: the N_e peaked at about 130 ms after the incorrect response for auditory, and about 85 ms after the response for visual stimuli ($F(1,8) = 19.55$, $p = 0.0022$) While there was no group effect on N_e amplitude neither in the STAs nor in the RTA, the N_e tended to peak later for POOR than for GOOD Ss (RTA: $F(1,8) = 4.56$, $p = 0.0653$).

3.4. Comparison of Nogo-N2 and N_e

A comparison of the Fz and Cz traces of Figs. 6–9 reveals that the N_e is clearly larger for Cz than for Fz in both modalities. In contrast, Figs. 1 and 6 show that the Nogo-N2 has a frontal maximum. Fig. 10 yields an overview over the amplitudes of the Nogo-N2 and the N_e at all electrodes (with the exception of Oz, where both components had a negligible amplitude). The N_e is presented as measured in the raw False alarm ERPs (STA) as well as in the STA and RTA difference ("difs"). While the Nogo-N2 is largest at Fz, the N_e is consistently largest at Cz, regardless of the measuring mode. The figure also shows that the N_e is symmetrical to the midline, and larger at Cz than at C3 and C4. Since no lateral electrodes have been used other than C3 and C4, no statements about a possible lateral distribution of the Nogo-N2 can be made.

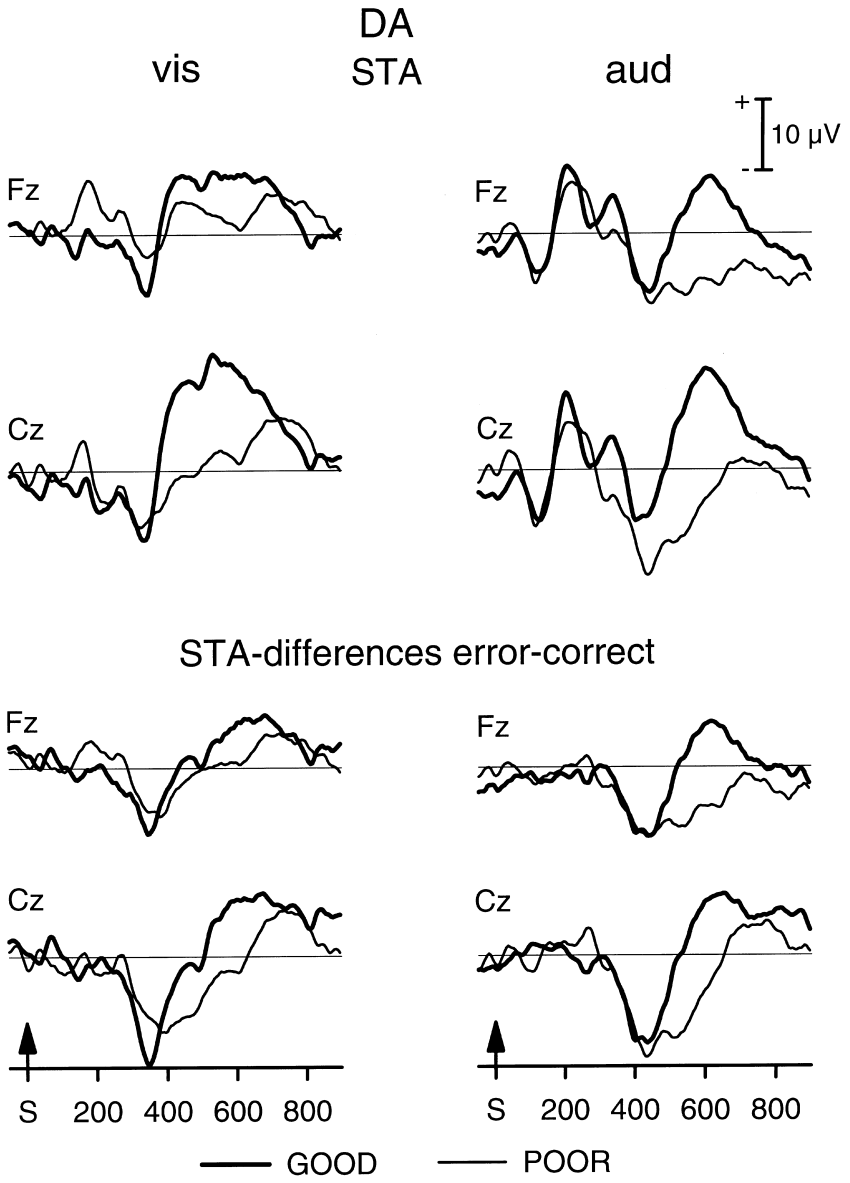

Fig. 8. Upper part: Mean stimulus-locked averages (STAs) of the False alarm trials separated for the performance groups (GOOD: low false alarm rate; POOR: high false alarm rate). The N_e shows no obvious group difference. Lower part: Difference wavers of the STAs (False alarm minus Go ERPs) for the two performance groups. After visual stimuli the N_e appears sharper and larger for GOOD Ss.

In order to statistically verify the topography differences for Nogo-N2 and N_e along the midline, as well as the different modality effects mentioned above, an ANOVA was run with the factors component (C) (Nogo-N2, N_e), M (visual,

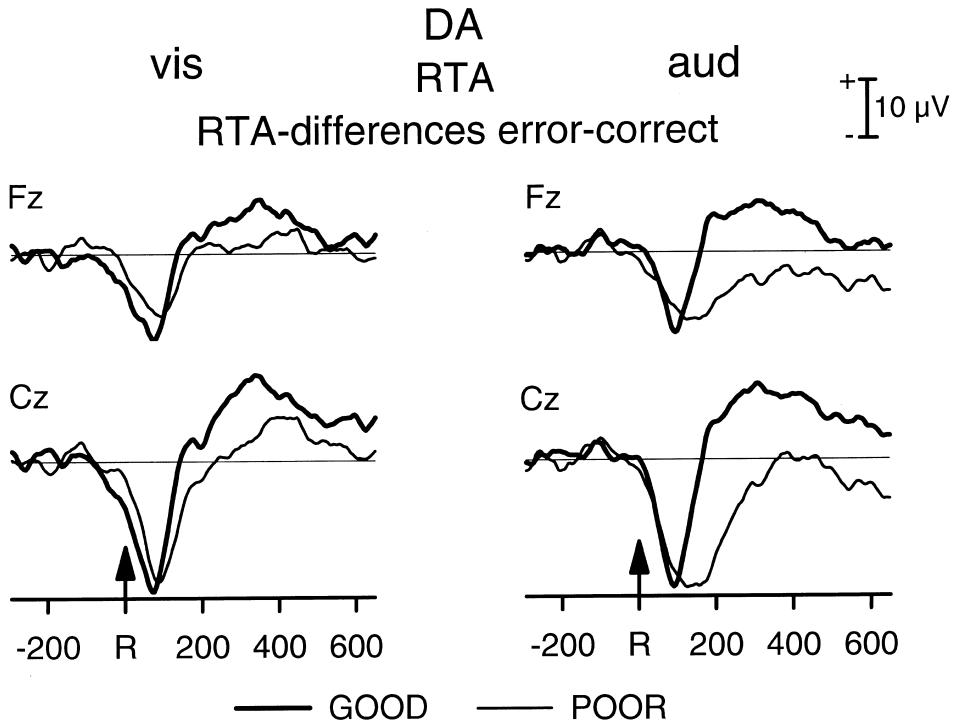

Fig. 9. Mean difference waves of the response-locked averages (RTAs) (False alarm minus Go ERPs) separated for performance groups. The amplitude of the N_e is the same for both groups. Apart from the modality difference, the N_e appears to peak slightly larger for POOR than for GOOD Ss, particularly after auditory stimuli.

auditory) and electrode (E) (Fz, Cz, Pz). The amplitudes of Nogo-N2 and N_e were parametrized by the maximum negative values in the Nogo-Go difference waves and the false alarm minus Go difference waves (difs.), respectively, i.e. for both components the same Go-ERPs were subtracted. The expected topography differences should show up in a C×E interaction. Since the N_e is larger than the Nogo-N2, the data were normalized by dividing by the mean values of Nogo-N2 and N_e (McCarthy and Wood, 1985). The degrees of freedom were reduced according to Geisser and Greenhouse (1958). The results showed, as expected, significant main effects of electrode ($F(1.71,15.43) = 20.22$, $p = 0.0001$) and modality ($F(1,9) = 14.04$, $p = 0.0046$). Moreover, there was a significant C×M interaction ($F(1,9) = 39.49$, $p = 0.0001$); the simple effects revealed a large modality effect for the Nogo-N2 ($F(1.86, 16.72) = 26.73$, $p < 0.0001$), but not for the N_e ($F < 1$). Most importantly, there was a significant C×E interaction ($F(11.56,14.00 = 33.46$, $p < 0.0001$), which verified the different topographies of Nogo-N2 and N_e. Finally, there were significant M×E and C×M×E interactions; the simple effects revealed a M×E interaction for the Nogo-N2 ($F(1.70, 15.34) = 8.20$, $p = 0.0050$), but not for the N_e ($F < 1$). This suggests a modality-specific topography of the Nogo-N2, but not for the N_e.

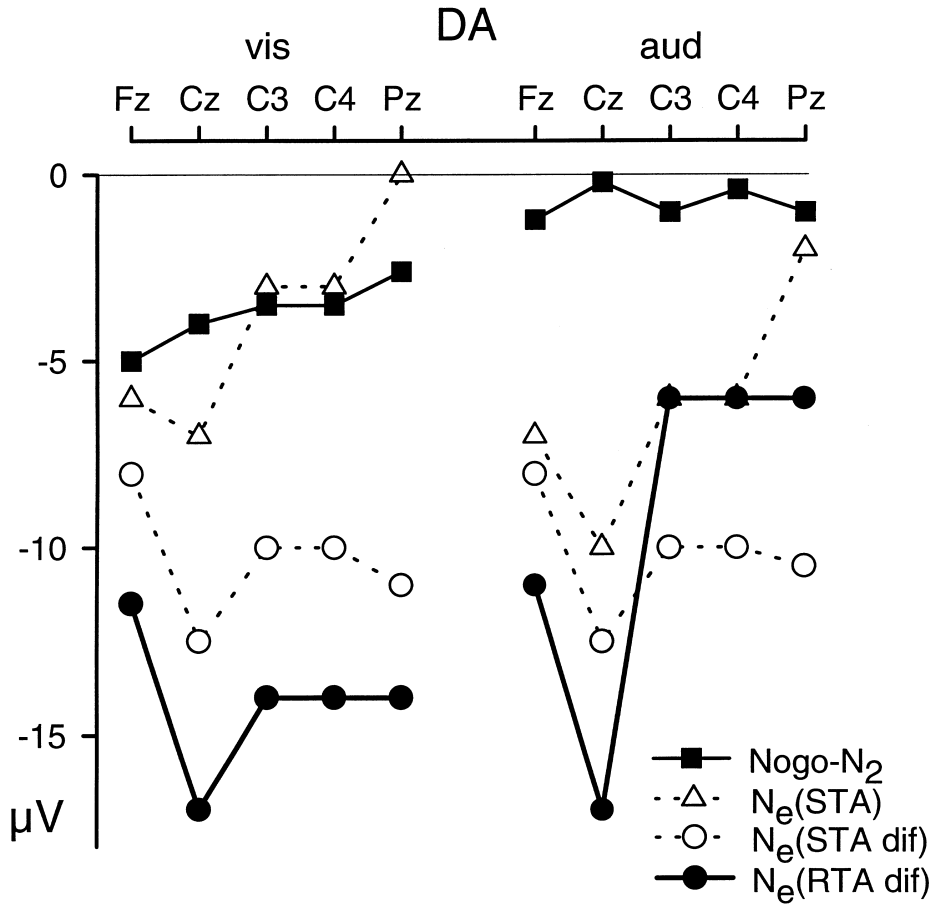

Fig. 10. Amplitudes of the Nogo-N2 as well as of the N_e at all electrodes (except for Oz). Rectangles: Nogo-N2 measured in the Nogo minus Go difference waves; Triangles: N_e measured in the False alarm STAs; Circles: N_e measured in the False alarm minus Go differences ("dif") of the STAs (open circles) and the RTAs (filled circles). Nogo-N2 and N_e have clearly different topographies.

4. Discussion

4.1. Performance

The low rate of misses is not surprising because of the very easy task. The high rate of false alarms is caused by the time pressure. The profound group difference in the false alarm rate is evident in Table 1. The reason for this difference is most probably a difference in cognitive preparation, as shown in detail elsewhere (Hohnsbein et al., 1998). The relatively small error rates after auditory stimuli suggest that inhibition is at least as efficient after auditory than after visual stimuli.

For divided attention (DA) the Go reaction times were the same for both stimulus modalities. This relative slowing of auditory RT can be attributed to the asymmetrical allocation of attention towards the visual modality, which may impair auditory processing (Hohnsbein, Falkenstein, Hoormann & Blanke, 1991). When attention was directed to the auditory modality, the RTs were shortened, which shows the effectivity of the attention manipulation for the auditory modality. The error (false alarm) RTs were shorter than the correct RTs, which suggest that in general the errors were premature responses. However, after auditory stimuli in DA, the RT shortening was very small, which may be due to the slowing of auditory processing. Indeed this relative slowing of the error RTs almost vanished when attention was focused exclusively to the auditory modality, which again shows the effectiveness of the attention manipulation for the auditory modality.

4.2. Nogo-N2, Nogo-P3 and inhibition

(a) Group effects: The main result was the clear relationship between the amplitude of the Nogo-N2 and the performance of the Ss: for Ss with few errors (GOOD) the Nogo-N2 was about twice as large as for Ss with many errors (POOR). This effect was the same for both attention conditions and stimulus modalities. Moreover the Nogo-N2 began about 30 ms earlier for GOOD than for POOR Ss. The amplitude effect is not likely due to a generally larger ERP in GOOD Ss, since the early components P170 (visual) and N120 (auditory) appeared even smaller in GOOD than in POOR Ss. The latency effect cannot be explained by overall amplitude differences. These effects on the Nogo-N2 cannot be due to group differences in RT, since these were small and in opposite direction for the modalities, while the effects on the Nogo-N2 were in the same direction for both modalities. These findings support the hypothesis that the Nogo-N2 reflects an inhibition process, which is stronger and begins earlier for GOOD than for POOR Ss, hence causing less false alarms in GOOD Ss. The timing of the Nogo-N2 makes also sense for an inhibition process, since the onset is at about 200 ms, i.e., well before the overt response in Go-trials.

For the Nogo-P3 no consistent difference was found across both performance groups, which is in contrast to the group effects of the Nogo-N2. Moreover, the Nogo-P3 also varies differently from the Nogo-N2 with respect to modality. Hence the idea that the Nogo-P3 reflects the closure of the Nogo-N2 is not supported by these data. The lack of a group difference shows that the Nogo-P3 is insensitive to performance differences. Our amplitude result agrees with Kopp et al.'s (1996b), who did not find differences of the Nogo-P3 in situations where inhibition was likely vs unlikely.

In sum, the group differences strongly support the inhibition hypothesis for the Nogo-N2, but not for the Nogo-P3.

(b) Modality and attention effects: As already reported earlier (Falkenstein et al., 1995), the Nogo-N2 was found to be much larger after visual than after auditory stimuli, when both modalities should have been attended (DA). However, the auditory Nogo-N2 was enhanced when attention was focused on the auditory

modality, while the visual Nogo-N2 was not further enhanced, when attention was focused on the visual modality. This confirms our earlier result (Hohnsbein et al., 1991) that attention is already focused on the visual modality in DA. This imbalance was reduced or even removed by focusing attention fully on the auditory stimuli. The still smaller amplitude of the Nogo-N2 after auditory stimuli under FA seems puzzling, since the false alarm rate was even lower after auditory than after visual stimuli, and the RTs were (at least in FA) shorter after auditory stimuli. This modality asymmetry of the Nogo-N2 can be explained in (at least) three different ways:

(1) Even when attention is focused on the auditory modality by instruction, there might still be an attention bias toward visual stimuli, when these can occur. Hence, the inhibition process may be weakened after auditory stimuli, too. However, this is unlikely, given the shortening of the correct RTs and the vanishing of the relative slowing of the error RTs, as well as the improvement of performance after auditory stimuli in FA. Also the comparison of the results of Eimer (1993) and Schröger (1993) with blocked modalities (i.e. attention is definitely focused on one modality), shows the Nogo-N2 to be twice as large after visual than after auditory stimuli, which is very similar to our FA results. Nevertheless, a follow-up study with fully blocked modalities could clarify this issue.

(2) A second explanation assumes that inhibition at the motor level may have different strengths for the modalities, i.e., it may be weaker after auditory stimuli, hence resulting in a small auditory Nogo-N2. This could be due to a more difficult motor inhibition after auditory stimuli, since these might activate the motor system more directly than visual stimuli. However, if motor inhibition was weaker or more difficult after auditory stimuli, this should be reflected in a higher false alarm rate after auditory than after visual stimuli, but the opposite was observed.

(3) The third explanation assumes that the inhibition mechanism reflected in the Nogo-N2 stems from modality specific generators, which project differentially to the scalp. This assumption is supported by the electrode by modality interaction for the Nogo-N2, and further corroborated by the results of Gamba and Sasaki (1990), who could demonstrate different locations of their early Nogo-potential in monkeys: after visual stimuli, the Nogo-potential emerged in the caudal part, after auditory stimuli more in the rostral part of the dorsal bank of the principal sulcus. It is well conceivable then, that the generator of the auditory Nogo-N2 in humans projects poorly on Fz. Hence it appears possible, that, with a suitable electrode positioning, a larger auditory Nogo-N2 could be measured at some other (e.g. more frontal) location. The assumption that the Nogo-N2 is modality-specific suggests that the inhibition process reflected in the Nogo-N2 works on earlier stages than on the final motor stage. Further support for this hypothesis comes from the findings of Pfefferbaum et al. (1985) and Kopp et al. (1996b), who demonstrated the Nogo-N2 in non-motor conditions, and from the results of Thorpe et al. (1996) that show a very early onset of the Nogo-N2 relative to the motor response. In sum, our data support the assumption that the Nogo-N2 reflects the activity of a modality-specific inhibition process, that works on a premotor level.

4.3. Relation of the Nogo-N2 to the error negativity (N_e)

The amplitude of the N_e was the same for both modalities and both performance groups. The absence of a modality effect on the N_e is in sharp contrast to the strong modality effect on the Nogo-N2, which was confirmed by the ANOVA. If the N_e was merely a large delayed Nogo-N2, the N_e should be also smaller after auditory stimuli, which is clearly not the case. Also, the absence of a group difference, such as seen for the Nogo-N2, shows that the N_e does not appear to be linked to the same inhibition process as the Nogo-N2. So the amplitude of the N_e is independent of stimulus modality and performance, while the Nogo-N2 depends on both. In contrast, while the latency of the N_e is clearly longer for auditory than for visual stimuli, the Nogo-N2 shows no such effect. Finally the topographies of N_e and nogo-N2 differed significantly: while the Nogo-N2 was larger at Fz than at Cz and had a modality-specific topography, the N_e was larger at Cz than at Fz and Pz, independent of modality. Hence, the present data yield converging evidence that N_e and Nogo-N2 reflect different phenomena. Accepting this, one could argue that a Nogo-N2 should be visible also in false alarm trials before the N_e. However, in false alarm trails inhibition may not occur at all; in that case no Nogo-N2 should be present. Even assuming a Nogo-N2 in false alarm trials, the leading flank of the N_e would overlap and hence cover the main part of the Nogo-N2. Nevertheless, Fig. 6 appears to show a beginning Nogo-N2 with frontal maximum at least after visual stimuli.

The differential variability of Nogo-N2 and N_e does not support the idea of Kopp et al. (1996b) that there is one common inhibition mechanism reflected in both components. However, it does not exclude that Nogo-N2 and N_e reflect anatomically and functionally different inhibition processes: while the Nogo-N2 reflects a more stimulus-specific inhibition process, the N_e might reflect a more response-specific inhibition process. Such two inhibition processes have indeed been claimed by De Jong, Wierda, Mulder and Mulder (1988). In sum, Nogo-N2 and N_e appear to reflect different mechanisms: While the Nogo-N2 is a correlate of pre-motor inhibition, the N_e either reflects some other inhibition process, or error detection, as originally proposed by us (Falkenstein et al., 1990, 1991).

5. Conclusions

The frontal Nogo-N2 was found to vary with stimulus modality and performance. From the literature and the present results we conclude that this component is a real-time correlate of a modality-specific non-motor inhibition process.

In contrast, the later positive frontal enhancement (Nogo-P3) showed no systematic relationship with performance, so its functional relation to inhibition remains doubtful. The error negativity (N_e) in false alarm trials had a similar appearance, but was found to vary completely differently from the Nogo-N2. So we conclude that the N_e is functionally different form the Nogo-N2, reflecting either error detection or activity of a second, modality-unspecific motor inhibition process.

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