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Effects of attention and stimulus probability on ERPs in a Go/Nogo task *

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Event-related potentials were measured to letters presented either to the left or the right of a fixation point that were preceded by a precue that indicated the position of the upcoming letter either correctly (valid trials) or incorrectly (invalid trials). One letter required a response (Go stimulus), while the other letter required no response (Nogo stimulus). In experiment I, Nogo letters occurred only on 25% of the trials, while in experiment II, Go and Nogo letters were equiprobable. In both experiments, Nogo stimuli elicited larger N2 components and P3s with a more anterior topography than did Go stimuli. The N2 enhancement elicited by Nogo stimuli showed a frontal maximum (most markedly when Go and Nogo stimuli were equiprobable) and was significantly influenced by precue validity. These results are discussed in terms of "Response Mismatch" and action inhibition processes.

Keywords: event-related potential, reaction time, Go/Nogo, N2, P300, inhibition.

1. Introduction

In recent studies employing Go/Nogo paradigms, systematic differences have been found between ERPs elicited by Go stimuli and ERPs to stimuli associated with the withholding of a response. Mäntysalo (1987) used a choice reaction task where frequent stimuli required a motor response while reaction had to be withheld to rare (10%) stimuli. She found that the N2 amplitude to Nogo stimuli was larger than the N2 to Go stimuli, the largest amplitude difference being visible at CZ. N2 latency was longer for Go as compared with Nogo stimuli. Pfefferbaum, Ford, Weller and Kopell (1985) reported a larger frontal N2 (peaking about 275 ms post-stimulus) to Nogo stimuli as compared with equiprobable Go stimuli at FZ. N2 latency was not

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affected by this manipulation. In a second experiment, a similar N2 modulation was observed when the required response was to count Go stimuli rather than to press a button, indicating that this effect was not confined to the withholding of overt motor responses. Using an S1–S2 Go/Nogo paradigm with equal probabilities for Go and Nogo stimuli, Kok (1986) found an enhanced negativity around 400 ms elicited by Nogo stimuli at frontal and central sites, but not at PZ. Employing a memory search paradigm, De Jong, Kok and Van Rooy (1988) found that N2 amplitude was enlarged at frontocentral sites when non-targets were presented as compared with equiprobable Go stimuli. This effect was present for attended as well as for unattended stimuli.

The Nogo P3 usually has a later peak latency as well as a more anterior topography than the P3 evoked by Go stimuli (Hillyard, Courchesne, Krausz & Kok, 1986; Picton, 1976; Pfefferbaum & Ford, 1988; Pfefferbaum, Ford, Weller & Kopell, 1985; Pfefferbaum, Ford, Wenegrat, Roth & Kopell, 1984; Simson, Vaughan & Ritter, 1977; Sutton, 1976; Tueting & but cf. Jodo & Inoue, 1990, for an effect of practice on P3 latency). These topographic differences have been attributed, at least partially, to different CNV resolution times for Go and Nogo trials (Simson et al., 1977). An enhanced P3 elicited by Nogo stimuli at central sites might be due to an earlier CNV resolution superimposed on the development of the P3. In Go trials, CNV may be maintained until the motor response has been performed. However, Pfefferbaum et al. (1985) have shown that differential effects of Go and Nogo stimuli on P3 amplitude and latency remain almost the same regardless of whether the required response consisted in overt button pressing or in covert counting, thereby demonstrating that these effects cannot be completely explained with reference to overlap with motor-related potentials. An alternative explanation is that the topographic differences between Go and Nogo P3 reflect a functional difference and that these components are produced by "distinctive brain systems" (cf. Hillyard et al., 1976).

With regard to the effects of Nogo stimuli on the N2 component, two interpretations have been offered recently. According to Mäntysalo (1987), the negativity elicited by Nogo stimuli reflects a cerebral mismatch process that is caused by a deviation from an established association between a Go stimulus and an overt response. Being deviant in this respect, Nogo stimuli elicit an early negativity ("Response Mismatch Negativity", RMMN) that is functionally similar to the auditory MMN. MMN is elicited by rare auditory stimuli differing in intensity, pitch, or other physical parameters from the standard context and is supposed to reflect an automatic cerebral mismatch process (Näätänen & Gaillard, 1983; Näätänen, 1986). According to the second interpretation, the N2 enhancement to Nogo stimuli is due to a response inhibition process. Kok (1986) suggests that the Nogo-related negativity is a reflection of a cortical action-inhibitory process ("red flag")

probably generated in prefrontal areas. However, as mentioned above, Pfefferbaum et al. (1985) have shown that withholding overt motor behavior is not a necessary prerequisite for this process to occur.

The present study has been designed to distinguish between these two interpretations. According to the "Response Mismatch" hypothesis, the N2 Nogo effect should be most prominent when Nogo stimuli are rare. Furthermore, these N2 enhancements should be elicited automatically, that is, independent of attention. The action inhibition hypothesis makes no prediction about the automaticity of the process underlying the N2 effect, nor does it predict an influence of Nogo probability on N2. In the experiments reported below, the probability of Go and Nogo stimuli and the direction of visual–spatial attention was manipulated within an S1–S2 choice reaction design. We employed a variant of the Posner paradigm (Posner, Nissen & Ogden, 1978). Subjects had to react to a specific letter and to withhold reaction to another letter. The type of reaction to be performed (left vs. right button press) was conditional on the position of the Go stimulus (left vs. right visual field). Shortly before the imperative letter appeared, a symbolic precue (an arrow pointing to the left or right side) was presented. On 75% of the trials, the cue pointed in the direction where the letter would occur (valid trials). Since the required response to Go stimuli was dependent on their position, the cue pointed to the correct response side on valid trials.

The a priori probability of Go and Nogo stimuli was varied between experiments I and II. In experiment I, Nogo trials were rare events (occurring in approximately 25% of the trials), while in experiment II, both Go and Nogo stimuli had a probability of 50%. The precue was supposed to influence the direction of spatial selective attention. Since cue validity was high, it was expected that subjects direct their attention to the visual hemifield indicated by the cue. This attentional orienting should result in faster overt responses to valid as compared with invalid Go trials. Orienting was also expected to influence ERP components. Mangun, Hansen and Hillyard (1987) have found that valid stimuli elicit enhanced occipital P1 and N1 components followed by a broad negative shift as compared with invalidly cued targets (cf. also Mangun & Hillyard, 1991; Hillyard & Hansen, 1986). If the precue is effective in directing attention, a similar influence of selective attention on the ERP is to be expected.

2. Methods

2.1. Experiment I

2.1.1. Subjects

Seven paid volunteers participated in the experiment. One of them had to be excluded because the percentage of trials including ocular artifact ex-

ceeded a fixed criterion (40% of the total number of trials). Thus six subjects (2 female), aged 25–31 years (mean age: 27.7 years), remained in the sample. All subjects were right-handed and had normal or corrected-to-normal vision.

2.1.2 Stimuli and apparatus

Each trial began with a 200 ms presentation of a centrally located arrow that pointed on a random basis either to the left or to the right side (0.5 probability each). The cue indicated the likely position of the following letter stimulus. 700 ms after cue offset, an uppercase letter (either M or W) appeared 6° to the left or right of fixation for 100 ms, subtending a visual angle of $1° \times 1°$, and designated as Go or Nogo stimulus, respectively. The interval between letter offset and onset of the next arrow was 2 s. Stimuli were presented white-on-gray on a computer display. The subject was seated in a dimly lit, electrically-shielded and sound-attenuated cabin, with response buttons under the left and right hand. The display was placed 100 cm in front of the subject's eyes and carefully positioned so that the stimuli occurred on the subject's horizontal straight-ahead line of sight.

2.1.3. Procedure

Twenty-four experimental blocks of 2.5 min duration each were run, each block consisting of 60 trials. After twelve blocks, response assignment (Go vs. Nogo) was reversed between the letters. The order of response assignments was balanced across subjects. Letters appeared with equal probability on the left and right side and were preceded either by an arrow pointing to the side where the letter appeared (valid stimuli) or by an arrow pointing to the opposite side (invalid stimuli). In 36 out of 60 trials (60%) of each block, the imperative stimulus was a valid Go letter, while the stimulus categories "Valid–Nogo", "Invalid–Go" and "Invalid–Nogo" each appeared eight times per block. Thus, on 44 out of 60 trials, a response was required, and on the same number of trials, a letter was preceded by a correct cue. This resulted in an overall cue validity and response probability of approximately 75%, respectively. Subjects were to respond with the right hand when a Go stimulus appeared on the right side of the display and with the left hand when it appeared on the left side. Responses had to be withheld when a Nogo stimulus appeared, regardless of its location. Subjects were instructed to maintain their fixation upon a central point. Because this proved to be difficult at the beginning for most subjects, three training blocks were run at the beginning of the experiment.

2.1.4. Recording and data analysis

EEG was recorded with Ag–AgCl electrodes from FZ, CZ, PZ, O1, O2, referenced to the right earlobe. To obtain the lateralized readiness potential, a bipolar recording was made from C3′–C4′ (these data are not presented in

this article). Horizontal EOG was recorded from electrodes at the outer canthi of both eyes, vertical EOG was recorded from electrodes above and beside the right eye. Electrode impedance was kept below 10 kΩ. The amplifier bandpass was 0.10–70 Hz. EEG and EOG were sampled on-line every 7 ms, and stored on disk. Reaction times were recorded for each trial. EEG and EOG were analyzed from 100 ms before the onset of the imperative stimulus until 800 ms after letter onset. Trials with eyeblinks (vertical EOG greater than ± 60 μV) or horizontal eye movements (horizontal EOG greater than ± 20 μV) and trials on which an overt response error was recorded (false button press or miss in the case of Go trials, false alarm in the case of Nogo trials) were excluded from further analysis.

EEG was averaged separately for conditions (left letter/right letter $\times$ Go/Nogo $\times$ valid/invalid) relatively to a 100 ms prestimulus baseline. For each condition, separate averages were computed for the first half (blocks 1–12) and second half (blocks 13–24) of the experiment. This procedure resulted in 16 average waveforms for each subject and electrode site. Peak and mean amplitude values were determined within the following intervals: 80–130 ms (P1), 130–180 ms (N1), 200–260 ms (P2), 250–310 ms (N2), and 300–550 ms (P3). Peak latencies were determined for the N2 and P3 time windows. Separate repeated measures analyses of variance were performed on amplitude measures for P1 and N1 (only for occipital sites), for P2, N2 and P3, as well as on N2 and P3 latencies for the following variables: electrode site (FZ, CZ, PZ, O1, O2), response assignment (Go vs. Nogo), cue validity (valid vs. invalid), letter location (left vs. right) and experimental session (first half vs. second half). When appropriate, a Greenhouse–Geisser adjustment to the degrees of freedom was performed (indicated in the result section by GG). Separate ANOVAs were performed for each electrode site. Effects of attention on the Nogo-induced N2 modulations were measured by performing repeated measures ANOVAs on the Go/Nogo difference waves for the factor cue validity. For the RT data conditions were used as factors.

2.2. Experiment II

2.2.1 Subjects

Six paid subjects (2 female), aged 24–41 years (mean age: 34.8 years), participated in the experiment. All subjects were right-handed and had normal or corrected-to-normal vision.

2.2.2. Stimuli and apparatus

Stimuli and apparatus were identical to those in experiment I, except that the probability of Nogo trials was raised to 50%, Nogo trials being thereby equiprobable to Go trials. The ratio of valid trials to invalid trials was kept constant at 44/16 (that is, on approximately 75% of the trials, a letter was

presented on the side indicated by the cue). This resulted in a total of 22 Valid–Go trials (instead of 36 validly indicated Go trials in experiment I) and 22 Valid–Nogo trials (as compared with 8 Valid–Nogo trials in experiment I).

2.2.3. Procedure, recording and data analysis
These were identical to experiment I.

3. Results

3.1. Experiment I

3.1.1. Reaction times
RTs to invalid Go letters were significantly longer than to valid letters (371 ms vs. 340 ms; $F(1,5) = 13.43$; $p < 0.015$). Overt error reaction occurred in only 5% of the trials. Response errors were significantly faster than correct reactions (298 ms vs. 356 ms; $F(1,5) = 76.45$; $p < 0.000$) and were almost exclusively false alarms. 70% of the false alarms occurred in response to valid Nogo stimuli.

3.1.2. Overall characteristics of ERPs
Upon visual inspection of the averaged curves for the different experimental conditions, three characteristics can be observed. After running parallel during the first 170 ms following stimulus presentation, the curves for valid and invalid trials diverge: on valid trials, ERPs were more negative than on invalid trials. The second characteristic bifurcation of the average curves occurs after approximately 250 ms, where the response assignment of the letter (Go vs. Nogo) becomes relevant: Nogo letters elicit a negative peak around 300 ms, while this is not the case for Go trials. Owing to the previous negative shift connected with precue validity, the absolute value of this peak is more negative for valid Nogo stimuli than for invalid Nogo letters. Thirdly, Nogo letters elicit a greater P3 than Go letters, and P3 amplitude is maximal in the case of valid Nogo stimuli.

3.1.3. Effects of response assignment (Go vs. Nogo)
N2 peak amplitude was more negative for Nogo letters than for Go stimuli ($F(1,5) = 20.33$; $p < 0.006$; see Fig. 1). This effect was significant at all sites, but maximal at FZ and at CZ (interaction response assignment × electrode site: $F(4,20) = 12.83$; $p < 0.004$,GG). Mean amplitude in the N2 range was less positive for Nogo than for Go stimuli over all locations ($F(1,5) = 11.27$; $p < 0.02$; see Fig. 2). N2 peak latency was shorter for Go trials than for Nogo trials ($F(1,5) = 26.28$; $p < 0.004$). This effect was significant for all sites,

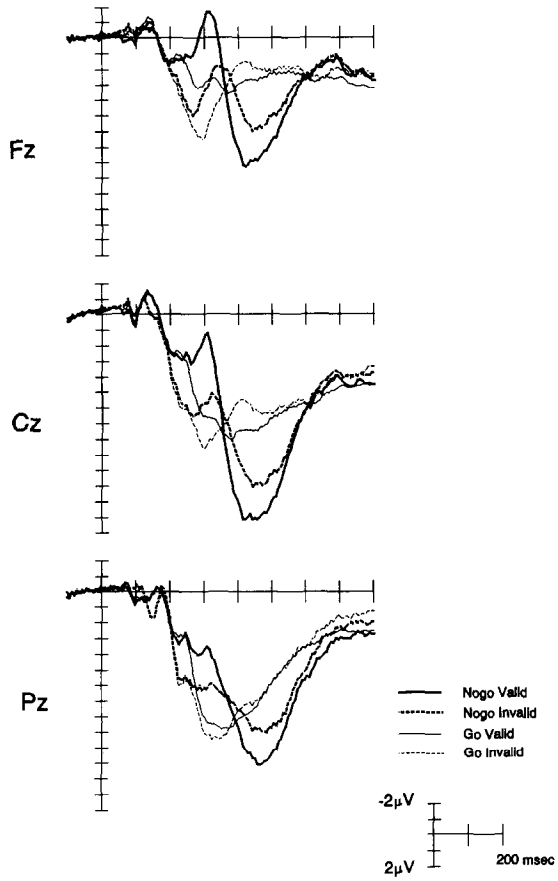

Fig. 1. Grand-average ERPs over six subjects at midline recording sites, Experiment I. Nogo stimuli are indicated by thick lines, Go trials by thin lines. Valid trials are indicated by solid lines, invalid trials by broken lines.

except for FZ, where it only approached significance. Nogo letters also elicited a greater P3 than did Go stimuli ($F(1,5) = 8.24$; $p < 0.035$), but this effect was significant only at frontal and central sites (interaction response assignment × electrode site: $F(4,20) = 5.71$; $p < 0.04$, GG). In the case of Nogo stimuli P3 amplitude tended to be greater at CZ than at PZ $F(1,5) = 6.51$; $p < 0.051$). For Go trials, no P3 amplitude difference was measured between central and parietal sites. P3 latency was also influenced by response assignment: Nogo P3s were delayed as compared with Go P3s ($F(1,5) = 33.82$; $p < 0.002$). At PZ, Go P3 latency was 366 ms, as compared with 475 ms for Nogo P3.

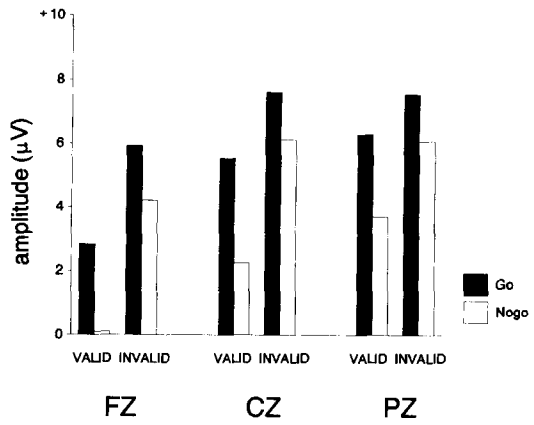

Fig. 2. Mean amplitudes (μV) in the N2 time window for valid and invalid trials (collapsed over left and right side of presentation). Experiment I.

3.1.4. Effects of cue validity

Valid letters elicited an enhanced negativity between 200 and 350 ms as compared with invalid letters, resulting in a more positive P2 for invalid letters ($F(1,5) = 33.47$; $p < 0.002$) and a more negative N2 for valid letters ($F(1,5) = 24.46$; $p < 0.004$). These effects were significant at all electrode sites, and were visible also in the mean amplitude values for the P2 and N2 range ($F(1,5) = 60.31$ and 17.29; $p < 0.001$ and < 0.009, respectively). However, cue validity had no significant effect on P1 and N1 mean amplitudes at occipital sites. For P1 mean amplitude, the effect of cue validity only

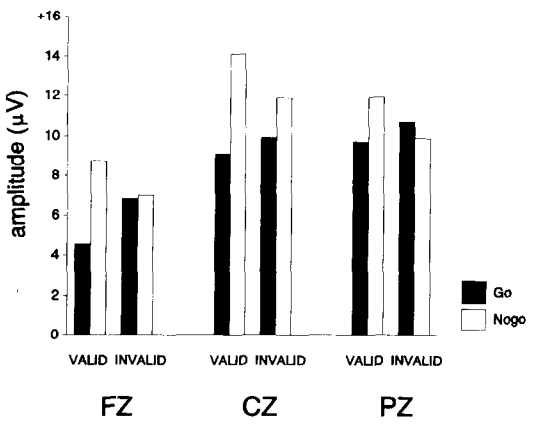

Fig. 3. P3 peak amplitude values (μV) for valid and invalid trials (collapsed over left and right side of presentation). Experiment I.

approached significance ($F(1,5) = 5.13$; $p < 0.073$), and was most pronounced at occipital sites contralateral to the visual field of the stimulus ($p < 0.057$). For N1 mean amplitude, no effect of cue validity could be found.

4.1.5. Interaction between cue validity and response assignment

To test the effects of attention on ERP modulations related to response assignment, mean amplitudes of the Go/Nogo difference waves (see Fig.4, top) in the N2 range were compared for valid and invalid trials. The difference wave's mean amplitudes were greater in the case of valid trials ($F(1,5) = 8.9$; $p < 0.031$). This effect was significant only for central and parietal electrode sites, and approached significance at FZ (interaction electrode site × validity: $F(4,20) = 5.24$; $p < 0.031$,GG). A strong interaction between cue validity and response assignment was found for P3 amplitude ($F(1,5) = 15.48$; $p < 0.011$): In the case of Nogo stimuli, P3 amplitude was greater when the letter was valid, while for Go stimuli, invalid letters elicited a larger P3 (see Fig. 3). This interaction was found to be significant at all central as well as at occipital leads.

3.2. Experiment II

3.2.1. Reaction times

RTs to valid target letters were again faster than to invalid letters (341 ms vs. 360 ms; $F(1,5) = 12.19$; $p < 0.02$). Error reactions were faster than correct reactions (308 ms vs. 351 ms; $F(1,5) = 52.39$; $p < 0.001$) and consisted almost exclusively of false alarms. Although half of the trials were Nogo trials, the rate of overt response error (6.5%) was comparable with that for experiment I.

3.2.2. Effects of response assignment (Go vs. Nogo)

Mean amplitude in the N2 range was less positive for Nogo stimuli as compared with Go stimuli ($F(1,5) = 11.40$; $p < 0.02$; see Fig. 6). Although this effect was highly significant at FZ ($F(1,5) = 20.99$; $p < 0.006$), and significant also at parieto-occipital leads, it was not significant at CZ. The effect of response assignment on N2 peak amplitude was significant only for FZ ($F(1,5) = 12.14$; $p < 0.018$) and approached significance at parietal sites ($F(1,5) = 5.20$; $p < 0.072$), but failed to reach significance over all electrodes ($F(1,5) = 5.27$; $p < 0.07$; see Fig. 5). Contrary to experiment I, N2 peak latency was not influenced by response assignment. P3 latency was massively influenced by response assignment: Nogo P3s were delayed as compared with Go P3s ($F(1,5) = 52.86$; $p < 0.001$). At PZ, Go P3 latency was 375 ms, as compared with 424 ms for Nogo P3. The difference between Go and Nogo letters was also reflected in P3 amplitude, although in a rather complex way: at frontal and central sites, Nogo stimuli elicited a larger P3 than did Go

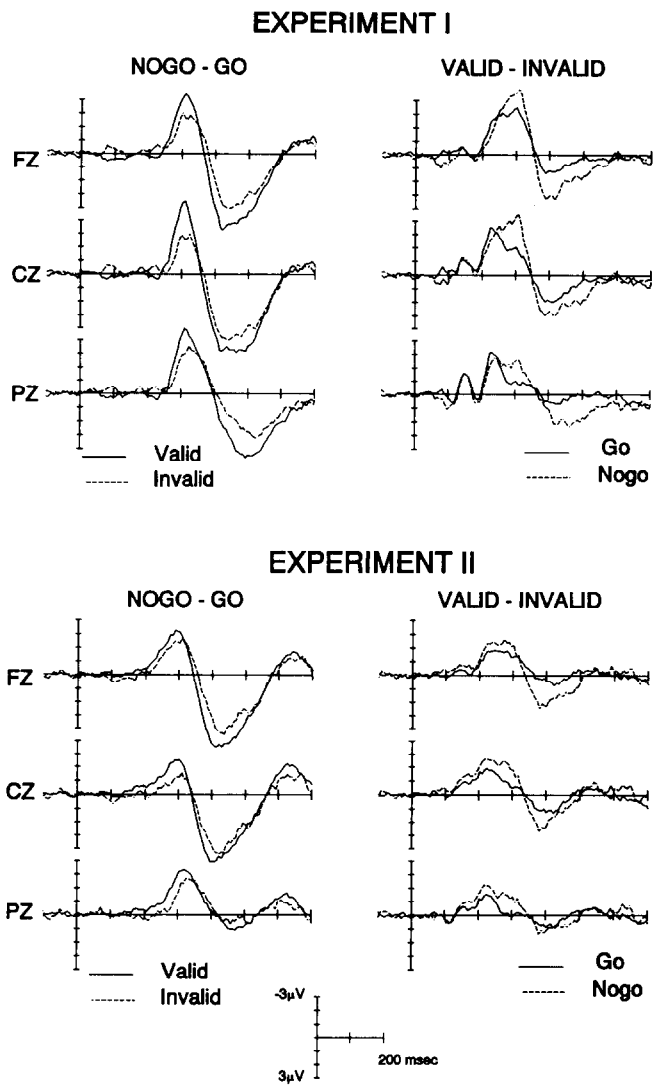

Fig. 4. Difference waveforms obtained by subtracting Go ERPs from Nogo ERPs separately for valid and invalid trials (left side) and by subtracting ERPs to invalid trials from valid trial ERPs separately for Go and Nogo trials (right side). Experiment I (top) and Experiment II (bottom).

Stimuli ($F(1,5) = 15.26$ and 27.94; $p < 0.011$ and < 0.003, respectively). At parietal sites, no P3 amplitude effect could be observed, while at occipital leads, the frontocentral effect was reversed: Go stimuli tended to elicit larger occipital P3s as compared with Nogo letters (O1: $F(1,5) = 5.15$; $p < 0.073$; O2: $F(1,5) = 6.66$; $p < 0.049$). P3 amplitude to Nogo stimuli was larger at CZ

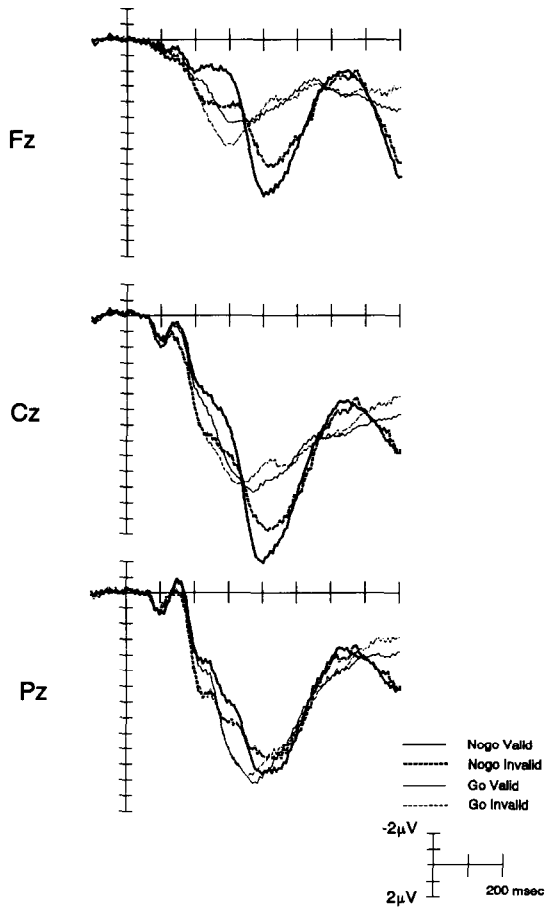

Fig. 5. Grand-average ERPs over six subjects at midline recording sites, Experiment II. Nogo stimuli are indicated by thick lines, Go trials by thin lines. Valid trials are indicated by solid lines, invalid trials by broken lines.

as compared with PZ ($F(1,5) = 47.37$; $p < 0.001$), while for Go letters, there was no difference in P3 amplitude between central and parietal sites (see Fig.7).

3.2.3. Effects of cue validity

As in experiment I, valid letters elicited a less positive P2 peak than invalid letters ($F(1,5) = 13.16$; $p < 0.015$). Contrary to experiment I, this enhanced negativity for valid letters did not extend to the N2 range at all sites: neither N2 peak amplitude ($F(1,5) = 4.09$; $p < 0.099$) nor the mean amplitude in the N2 range ($F(1,5) = 5.84$; $p < 0.06$) was significantly influ-

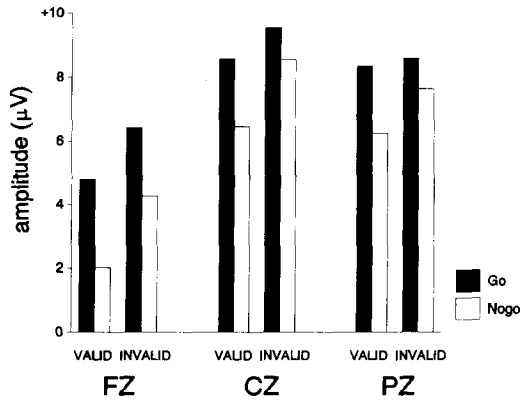

Fig. 6. Mean amplitudes (μV) in the N2 time window for valid and invalid trials (collapsed over left and right side of presentation). Experiment II.

enced by cue validity. The only significant effect of cue validity on N2 mean amplitude could be observed at FZ($F(1,5) = 12.30$; $p < 0.017$). Cue validity did not result in a significant modulation of occipital P1 mean amplitude ($F(1,5) = 3.45$; $p < 0.122$) and had no influence on N1 mean amplitude at occipital sites.

3.2.4. Interaction between cue validity and response assignment

Effects of attention on ERP modulations related to response assignment were again measured by comparing the mean amplitudes of the Go/Nogo difference waves for valid and invalid trials in the N2 range (cf. Fig. 4,

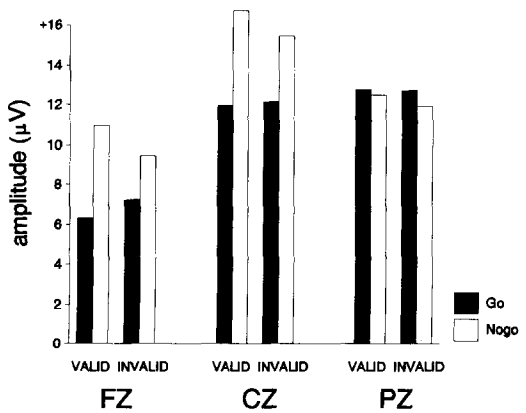

Fig. 7. P3 peak amplitude values (μV) for valid and invalid trials (collapsed over left and right side of presentation). Experiment II.

bottom). Mean difference amplitudes were greater for valid trials ($F(1,5) = 28.49$; $p < 0.003$). This effect was significant at frontal and parietal sites, and approached significance at occipital leads. For CZ, no significance test was run, because the overall difference between Go and Nogo trials failed to reach significance (see above). The strong interaction between cue validity and response assignment for P3 amplitude that was found in experiment I (greater P3 amplitude for Nogo stimuli when the stimulus was validly cued, and larger P3 to Go stimuli in the case of invalid trials) was almost missing, being significant only at FZ($F(1,5) = 7.23$; $p < 0.043$).

4. Discussion

This study was designed to investigate how ERP components are influenced by the difference between Go and Nogo stimuli and whether this influence is modulated by the direction of attention and the a priori probability of Go and Nogo letters. Attended stimuli elicited a broad negative shift that started around 180 ms. Similar amplitude modulations for a trial-by-trial cuing paradigm were reported by Mangun, Hansen and Hillyard (1987) and Mangun and Hillyard (1991). They attribute this negative shift to an enlarged P3 elicited by invalid trials that have a low probability. Two findings from the present experiment do not support this interpretation. Neither in experiment I nor in experiment II, was there a main effect of cue validity on P3 amplitude. Moreover, the observed amplitude shifts related to cue validity started well before 200 ms and did not extend beyond the P2 range (200–260 ms) in experiment II. The negative modulation observed for valid trials may therefore be interpreted as a broad processing negativity elicited by attended as compared with unattended stimuli. However, no attentional modulation was found for early ERP peaks (P1, N1) at occipital sites. This failure to reproduce the early attention effects reported by Mangun et al. may be due to the inclusion of Nogo trials. Furthermore, these effects are supposed to be largest over occipital scalp sites lateral to O1 and O2. The fact that reaction times were faster for valid than for invalid Go letters strongly supports the assumption that the symbolic arrow precue was effective in directing attention. Since the cue also indicated the correct response side on valid Go trials, part of the observed RT benefit might be due to selective motor preparation in the cue–target interval.

Nogo stimuli elicited an enhanced negative shift peaking at about 300 ms. Prior to this, a positivity is visible in experiment I that may be interpreted as a P3a (see Renault, 1983). However, the existence of a P3a is less obvious in experiment II (see Fig. 4), and it bears no obvious functional relationship to the difference between Go and Nogo responses. In accordance with prior ERP studies on Go/Nogo tasks (Kok, 1986; Mäntysalo, 1987; Pfefferbaum et

al., 1985, 1988) the negative shift elicited by Nogo trials is interpreted as an enhancement of a late N2 component.

In experiment I, Nogo letters were rare events, while in experiment II, Go and Nogo trials were equiprobable. N2 enhancements in experiment I may therefore be due to a combined effect of stimulus probability and response assignment. In experiment II, N2 amplitude effects for Nogo stimuli are directly attributable to the response assignment of the letters. As expected, the N2 amplitude enhancement elicited by Nogo stimuli was more pronounced in experiment I than in experiment II. At frontal sites, N2 amplitude enhancements for Nogo letters decreased least between experiments. This may be taken as an indication that N2 modulations directly related to response assignment have an anterior distribution, while N2 enhancements connected to stimulus probability is more broadly distributed. This interpretation is in line with the notion suggested by Kok (1986) that Nogo-related ERP modulations in the N2 range are indicative of a "red flag" signal in the pre-frontal premotor area that is related to response inhibition processes.

In contrast to the results reported by Mäntysalo (1987), the Nogo N2 was found to have a longer latency than the Go N2. However, this effect disappeared in experiment II, indicating that it is likely to be related to the difference in a priori probabilities in experiment I. N2 amplitude enhancements to Nogo stimuli remained present even when these stimuli were equiprobable to Go letters. Mäntysalo (1987) regarded the N2 modulation as a sort of "Response Mismatch" elicited when a required response deviates from a standard stimulus–response association. The results from experiment II indicate that response deviance in itself is not crucial for the N2 effect. It is more plausible to assume that the observed N2 modulations indicate that Nogo stimuli elicit different response-related processes from those elicited by Go stimuli (active response inhibition vs. response activation). To test this hypothesis more directly, the a priori probability of Go and Nogo stimuli should be varied within a single experimental design.

The "Response Mismatch" hypothesis assumes that the N2 effect for Nogo stimuli is automatic in the sense of being elicited independent of the direction of attention. In both experiments, the difference in N2 amplitude between Nogo and Go letters was greater when stimuli were valid and therefore attended. The former assumption is therefore not supported. However, the N2 effect was present also for invalid trials, which implies that the underlying process can be elicited by stimuli outside the attentional focus. This is to be expected, since invalid stimuli were designated as targets on some of the trials and thus could not be ignored. The precue may therefore not have led to a sharp focusing of spatial attention. It is an open question whether an N2 effect is still present under conditions when unattended Nogo stimuli are completely ignored.

The present study confirmed prior findings concerning topographic and

latency differences between Go and Nogo P3s. P3 latency was prolonged for Nogo stimuli. This effect remained significant when the probability of Nogo letters was increased, although the absolute latency difference between Go and Nogo P3s at central and parietal sites decreased between experiments from about 100 ms to about 50 ms. In experiment I, invalid Go letters elicited a greater P3 than valid Go letters, while for Nogo letters, the P3 was greater to valid trials (although valid and invalid Nogo trials were equiprobable). The former effect may be due to the lower a priori probability of invalid Go trials. The other effect may be explained by the hypothesis that after, a valid cue, a Go letter was expected at the position where the Nogo stimulus turned up. Systematic topographic differences were found between Go and Nogo P3s: Nogo P3 amplitude had a central maximum, while the P3 to Go stimuli had equal amplitudes at central and parietal sites. At frontal and central recording sites, Nogo letters elicited a considerably greater P3 than did Go stimuli. This pattern of results was most striking in experiment II, where the frontocentral effect of an enhanced Nogo P3 was reversed at occipital sites. These findings can be taken as evidence for the existence of two functionally different brain processes related to response activation and inhibition. However, it is still possible that differential CNV resolution times for Go and Nogo trials or motor potentials at central recording sites have contributed to the topographic and latency differences for Go and Nogo P3s (see Kok, 1988).

In summary, it was found that Nogo stimuli elicited a negative amplitude shift in the N2 range that has to be distinguished from N2 enhancements ("mismatch") elicited by rare events. The former component was elicited even when the Go and Nogo trials are equiprobable. It had a frontal maximum and was almost absent at central sites. Since it was modulated by attention, the underlying process cannot be regarded as automatic. Nogo trials elicited P3s that systematically varied in topography and latency from the P3s evoked by Go stimuli. The Nogo P3 was later, and had a frontocentral distribution, while the Go P3 had a shorter latency and was distributed more posteriorly. This pattern leads to the assumption that Go and Nogo P3s reflect different underlying processes.

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