First publ. in: Journal of Psychophysiology 6 (1992), pp. 175-184

Probing the functional brain state during P300-evocation

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Keywords: P300, oddball, probe stimuli, event-related potentials, slow cortical potentials, cortical excitability

ABSTRACT Slow cortical potentials may represent the excitability of cortical neuronal networks. We have suggested that surface-negative potentials, such as the CNV, indicate increased cortical excitability while positive-going waves, such as P300, are produced when excitability is lowered transiently (Rockstroh, Elbert, Canavan, Lutzenberger, & Birbaumer, 1989). If true, the processing of "probe" stimuli presented during surface-positive waves, i.e., by brain tissue during phases of lesser excitability, should be inhibited, leading to slower responses and reduced evoked potential amplitudes. This hypothesis was examined by presenting a total of 900 acoustic stimuli comprising 70% standard stimuli (1200 Hz, 55 dB) and 30% target stimuli (700 Hz). Twenty subjects were instructed that their major task was to silently count the targets. On 46% of the trials, clicks were presented as probe stimuli in addition to the standard or target stimulus; a probe could follow the onset of the stimulus at delays of 260, 290, 320, 350, 380 or 410 msec. A fast button press was required to every probe. Half of the sample exhibited a clear "oddball-P300"; all subjects who failed to produce a P300 differentiation between targets and standards showed a frontally negative Slow Wave which was larger to targets than to standards. N1/P2 amplitudes to probes were smaller whenever the probes were added to a target stimulus, in particular when target stimuli had elicited a P300. In subjects with oddball P300, motoric responses to probes were delayed at times when a target-evoked positive shift was present. Subjects who produced no oddball P300 showed no such slowing of reaction time. Results support the hypothesis that widespread positive waves indicate disfacilitation of cortical excitation.

Introduction

Attempts to model the flow of information processing and response preparation have recommended searching for objective correlates of the various processing stages. Endogenous components of the event-related potential (ERP) have been discussed as possible indicators of hypothetical constructs. Success in applying such approaches depends upon linking the psychological processes with differences in the state of the underlying neurophysiological substrate. Otherwise it would be impossible to determine whether a component is tied to specific steps in the information processing or whether it varies nonspecifically with fluctuations in activation. We have pursued such an approach for slow cortical potentials (SCPs) (summarized in Birbaumer, Elbert, Canavan and Rockstroh, 1990; Rockstroh, Elbert, Canavan, Lutzenberger, Birbaumer, 1989). In this model, surface negative shifts, such as the CNV, are assumed to reveal enhanced excitability of cortical neuronal networks enabling a preparatory state or "potentiality" for cerebral processing in the underlying neuronal networks. In contrast, slow positive shifts would indicate reduced excitability or "disfacilitation" in the cortical neuronal networks. Somewhat paradoxically, a reduced neuronal excitability is to be expected during memory storage (Elbert, 1992; Elbert & Rockstroh, 1987; see also Rösler, 1977) considering Hebb's (1949) conceptualization of cell assemblies and functional networks: Activity should reverberate only in the cell assemblies actively involved in the specific information. The development and stabilization of these distinct synaptic connections require a large portion of cells irrelevant for the incoming concept to be shut off. This should be seen in a reduced depolarization or even inhibition in vast networks. The surface positivity corresponding to these inhibited networks would then dominate over the relatively

smaller spots of negativity caused by the reverberating excitation. We may hypothesize that positive waves such as the P300 result from such a disfacilitation of widespread neural activity. The generally observed centro-parietal maximum of the P300 would then be a consequence of the superposition of the distributed sources. The present experiment was set up to examine the disfacilitation hypothesis. This hypothesis is not at variance with the "context-updating-model" of Donchin and Coles (1988), which relates the P300-wave to the modification of memory traces. It is Hebb's rule which embodies disfacilitation during the psychological process ascribed to the P300 by Donchin & Coles' model.

The correspondence of surface-negative SCPs and increased cortical excitability, on the other hand, may be deduced from their relationship to response facilitation (Birbaumer et al., 1990; Rockstroh et al., 1989): (1) Working on behavioral tasks contingent upon spontaneous negative or positive DC-shifts, Stamm (1984) and Bauer (1984) observed an area-specific relationship between negativity and response efficiency (in terms of response speed and error rates). (2) Biofeedback induced negativities over the contralateral motor cortex speeded up reaction time (Rockstroh, Elbert, Lutzenberger & Birbaumer, 1982, 1990). (3) N100 amplitudes were enhanced when the eliciting stimuli were separated from the preceding stimuli by no more than 100 msec (Loveless & Hari,1989), assuming that the N100-eliciting stimulus was presented to a network which was still activated. (4) On the other hand, drugs which lower cortical excitability (carbamazepine and α benzodiazepine) also lower slow surface negativity (Rockstroh, Elbert, Lutzenberger, & Altenmüller, 1991).

The association of P300-like positivities with cortical disfacilitation is by no means inconsistent with previous theorizing: The fidelity of the updating process of an organism's context model (Donchin & Coles, 1988) should in fact be higher if more of the unrelated networks are shut off. Such a transient "shut down" might well relate to Verleger's (1988) concept of closure in a processing epoch (Elbert & Birbaumer, 1988).

Another approach to test for the momentary neural excitability is to apply test stimuli during the course of the brain waves, such as during the upward and downward slope of a P300. If the stimulus input reaches the corte during a P300, which presumably reflects state of widespread disfacilitation, it should b processed less efficiently and, hence, suc stimuli should evoke smaller or delayed re sponses, with the one possible exception bein the case when the input directly enters the re verberating circuit. Woodward, Brown, Mars and Dawson (1991) presented clicks at variou intervals during an acoustic oddball task o 50% of the trials. Motor responses were re quired to clicks. Reaction times were signif cantly slower to clicks delivered 300 to 37 msec following target stimuli than reactio times to clicks with other time lags and t those following standard stimuli. The perio with maximal reaction time slowing corre sponded to the period of maximal P300 in th oddball task.

The present study used the probe techniqu to evaluate the neurophysiological state repre sented by the P300. Two measures were mon tored: reaction time to probes and the probe evoked vertexpotential (N1/P2). Acousti probe stimuli were delivered at different point in time after target and standard stimuli in a acoustic oddball paradigm. It was hypothe sized that reaction time would be longer an N1/P2-amplitude smaller when probes fol lowed target stimuli in the latency range of th P300. The acoustic N1/P2 amplitude is know to have at least two major sources, in the su pratemporal plane, and another, more wide spread one. If the generation of the P300 i indeed widespread (as suggested by Lutzen berger, Elbert, & Rockstroh, 1987), it shoul interfere with both sources equally well. Ac cording to recent evidence from magnetoen cephalographic and MRI studies, activity re sponsible for the surface-recorded P300 com ponent may be initiated in deep structures, bu then spreads over other cortical areas and i sustained in areas near the auditory corte (Rogers, Baumann, Papanicolaou, Bourbon Alagarsamy, & Eisenberg, 1991).

Methods

Subjects

Twenty healthy, right-handed student volun teers (10 male, 10 female, mean age 23 years

received course credit for participating in the experiment that lasted about 1 1/2 hours.

Design and Procedure

Subjects listened to a total of 900 acoustic stimuli, each one being 55 dB SPL (A) and 50 msec in duration. Stimuli were delivered with a constant interstimulus interval of 2.3 sec. 700 Hz tones served as standards in 70% of the trials, while "rare" tones of 1200 Hz in 30% of the trials had to be silently counted as targets. With a probability of .46 a click (white noise of 5 msec duration) was added as probe stimulus to the oddball stimuli. Probe stimuli could follow the onset of a standard or a target stimulus at delays of 260, 290, 320, 350, 380 or 410 msec. Only one probe stimulus could occur on a given trial; on the average, 54% of all trials remained without probe. Whether or not a click was presented was varied randomly across trials, as was its delay. Subjects were asked to press a button as fast as possible to every click in addition to silently counting the targets.

During the experiment the subject sat in a comfortable reclining chair in a sound-attenuated, electrically shielded and dimly lit chamber and held the response button in the preferred hand. After the preparation phase for the physiological recordings, the subjects received a written task instruction and were asked to keep their eyes open during the whole experiment and to fixate a convenient point in front of them in order to avoid eye or head movements as well as blinking. At irregular intervals during the experiment, the experimenter asked for the number of targets counted so far in order to stress the demands of the oddball task.

Apparatus and physiological recordings

An ASYST (A Scientific System) program, running on an AT-386 computer equipped with a DT 2821 board (DMA), controlled the timing of the experimental stimuli and storage of the reaction times and electrophysiological responses. Synchronized via a serial line, the acoustical stimuli were generated by means of an ATARI ST-computer and presented via a loudspeaker. The electrophysiological data were amplified by a Beckman type 511 dynograph.

The EEG was recorded along the midsaggital line from frontal (Fz), central (Cz) and parietal (Pz) leads with a time constant of 10 sec, and the high-frequency cutoff was set at 30 Hz. A reference electrode was affixed to the right earlobe. Nonpolarizable silver-silverchloride electrodes (ZACK) were used for the EEG recordings, and Grass EC2 electrolyte served as the conducting agent. The skin under the electrodes was prepared by cleansing with alcohol and by rubbing with an abrasive paste (OMNIPREP).

The vertical EOG was recorded via Beckman silver-silverchloride electrodes centered about 1 cm above and below the left eye. Beckman electrode jelly served as the electrolyte. Again, the skin was prepared using alcohol and abrasive paste. All data were digitized at a rate of 200 Hz and were stored for off-line analyses. The response latency was stored to the nearest msec via the digital input to the interface board.

Data Reduction and Analysis

Trials with an EOG shift exceeding 150 μV or an EEG shift exceeding 100 μV were rejected from further analysis. Trials in which response latencies exceeded 1000 msec or were less than 100 msec were also excluded, as were trials with erroneous button presses to the standard or target stimuli. Data from one subject were excluded completely since less than 50% of the trials met the inclusion criteria. For the remaining 19 Ss a mean of 85% of the trials was analyzed. The EEG was averaged for 100 msec for a prestimulus baseline and 1400 msec following each stimulus. The following scores were determined and averaged separately for the two types of stimuli (standard and target) and the six probe delay conditions:

1. P300-amplitude and latency were determined for the maximum positive deflection within the latency range of 250 to 400 msec in the parietal recording. Scores were averaged only across trials without probe stimuli, separately for target and standard stimuli.
2. The Slow Wave was determined as the maximum negative deflection following the P300 within the latency range of 400msec until the end of the recording epoch in the frontal recording, averaged across trials without probe stimuli as described for the P300.
3. The Vertexpotential (N1/P2) to probe stimuli was determined as the peak-to-peak

amplitude from the maximum negative deflection during the 80 to 200 msec after click onset to the maximum positive deflection following the negative deflection up to 300 msec.

4. Reaction times (RT) to probe stimuli were determined as the median per subject separately for every delay and for probes following a target and those following a standard.

The effects of the experimental conditions on RT and N1/P2 amplitudes were evaluated by analyses of variance (ANOVA) with the factors oddball (target versus standard stimulus), delay (six different delay periods), and – for N1/P2 – electrode (frontal, central and parietal leads). All reported p-values were obtained after adjustment of the degrees of freedom with the Greenhouse-Geisser-Epsilon. Means ± Standard Errors are presented.

Results

Event-Related Potentials Evoked by the Oddball Task

The experimental protocols revealed that the subjects counted an average of 97.1% of the targets. Although they were asked not to press the button to oddball stimuli, such erroneous motor responses occurred after 0.4 ± 0.1% of the standard stimuli and after 2.9 ± 0.6% of the targets ($F(1,18) = 14.4$, $p < .01$).

Parietal P300 was larger in response to target (5.6 ± 0.5 μV) than to standard (3.7 ± 0.3 μV) stimuli: ODDBALL × ELECTRODE: $F(2,36) = 14.0$, $p < .001$, ODDBALL: $F(1,18) = 4.8$, $p < .05$, ELECTRODE: $F(2,36) = 32.0$, $p < .001$). Mean P300-latencies were similar (n.s.) for target (374.7 ± 7.6 msec) and for standard stimuli (379.2 ± 4.1 msec). While the total averages indicated an unexpectedly small P300, closer inspection of the individual ERPs to standard and target stimuli without probes suggested that this may have been due to individual differences under the present conditions: Data analysis disclosed that only eleven of the nineteen subjects exhibited an "oddball P300", i.e., a pronounced parietal positive deflection in the respective latency range which was more than 1.5 μV larger for targets than for standards. All eight subjects with smaller or no oddball P300 showed an "oddball Slow Wave", i.e., a pronounced fronto-centrally negative Slow Wave (SW) that was substantially larger in response to targets than to standards. Figure 1 illustrates the ERPs averaged across these subgroups of subjects exhibiting an oddball P300 (left) and subjects without oddball P300 (right). Across subjects, these scores were negatively correlated ($r = -.50$, $p < .05$; see Fig. 2a).

According to the hypothesis, an attenuation of reaction time and N1/P2 amplitude should be pronounced only in subjects exhibiting an oddball P300, while we had no such predictions for subjects producing negative shifts in response to targets. This was taken into account by introducing a group factor in the subsequent analyses: One group consisted of eleven subjects in whose difference in P300 amplitudes (target – standard) exceeded 1.5 μV (group oddball P300), the other group consisted of the remaining eight subjects who revealed no P300 effect (see Fig. 2b for the ERP difference curves at Cz averaged separately for the two groups). As can be seen from Figure 2a, setting 1.5 μV as the limit for a difference of P300 in target versus standard condition may have approximated the noise level as there were subjects who showed the opposite differentiation of 1.5 μV. Interestingly, all subjects with a lack of oddball P300 exhibited an oddball SW. In six subjects both effects were present.

Event-Related Potential Evoked by Probe Stimuli

Across all subjects the vertex potential (N1/P2 elicited by probe stimuli showed difference between electrodes ($F(2,34) = 35.3$, $p < .001$ with a maximum of 12.5 ± 4.3 μV at Cz. N1/P2 amplitude was smaller to probes following target stimuli than to probes following standard stimuli (ODDBALL, $F(1,17) = 17.5$, $p < .001$). This effect was most prominent at Cz leading to the interaction ODDBALL × ELECTRODE ($F(2,34) = 4.0$, $p < .05$). N1/P2 amplitudes to probes were smaller following target than following standard stimuli for all delays except 410 msec; at this delay the amplitude difference for standard- and target-associated probes was reversed (ODDBALL × DELAY $F(5,85) = 9.4$, $p < .001$; DELAY, $F(5,85) = 51.4$, $p < .001$). Again, the effects were most pronounced at Cz, revealing interactions of ODDBALL × DELAY × ELECTRODE

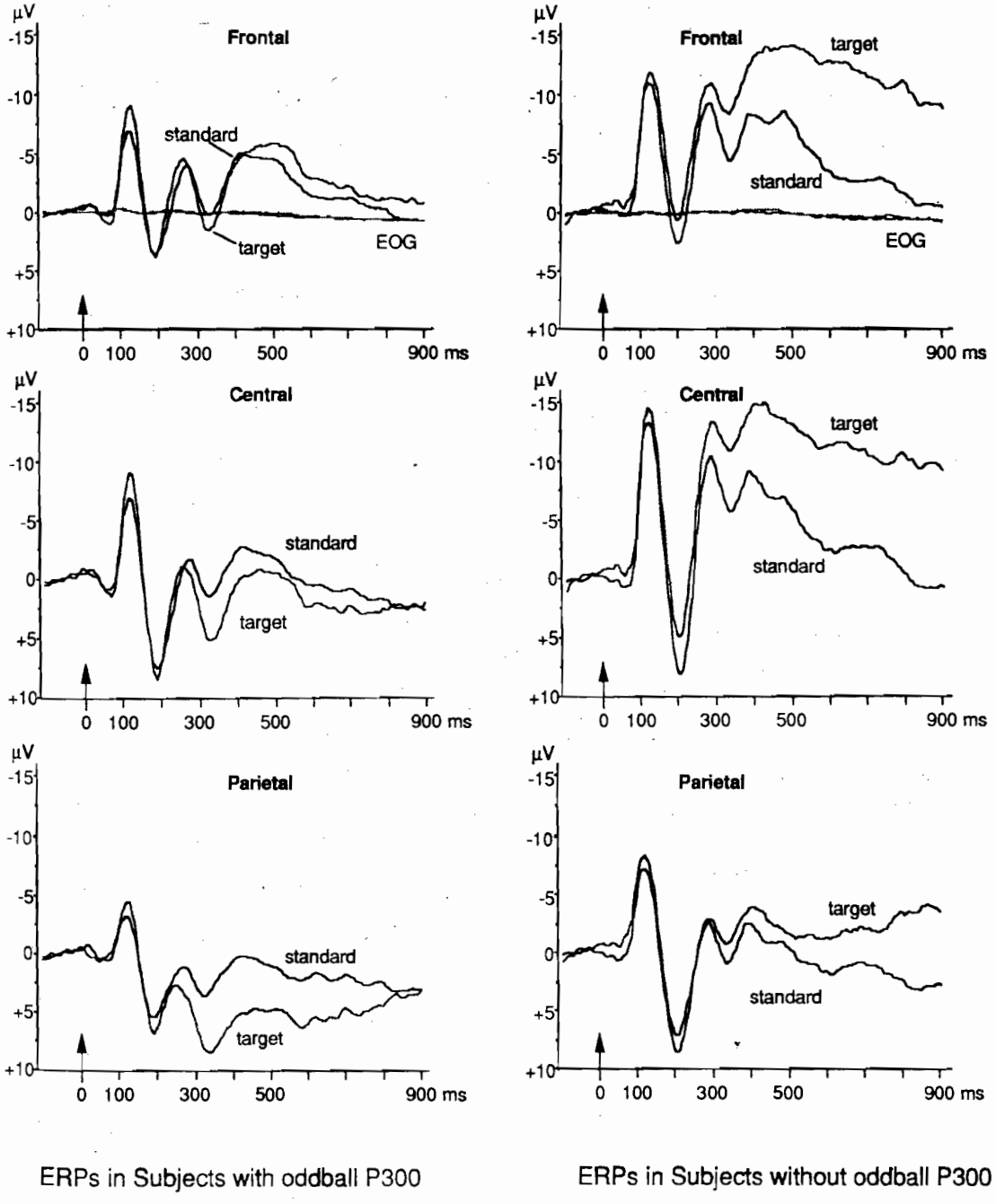

Figure 1 Event-related responses (ERPs in μV, negativity up) to standard and target stimuli in trials without additional probes. ERPs are averaged for the recording sites (frontal, central, parietal) separately for subjects "with oddball P300" (left column, N=11) and subjects "without oddball P300" (right column, N=8). EOG: across-subjects averages of vertical eye movements for standard and target stimuli.

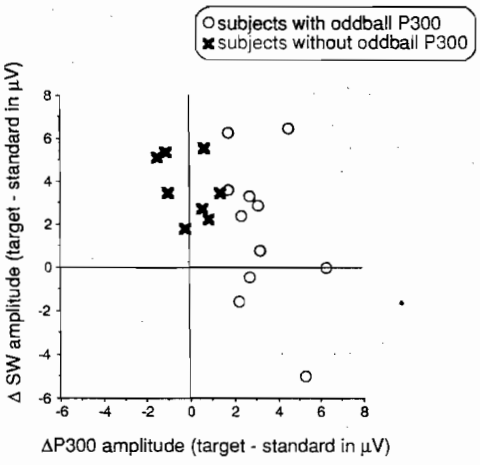

Figure 2a Relationship between P300 and negative Slow Wave amplitudes. Amplitudes are computed as difference between parietal P300 or frontal SW amplitude, respectively, under standard and target conditions (trials without additional probe stimuli). Each symbol represents a subject: circles and crosses indicate the group assignment.

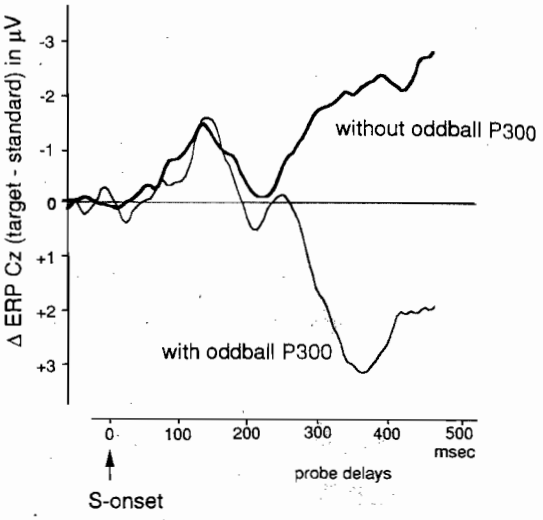

Figure 2b Difference in precentral event related potentials in response to target and standard stimuli averaged across subjects separately for the two groups (thick line: subjects without oddball P300, thin line: subjects with oddball P300).

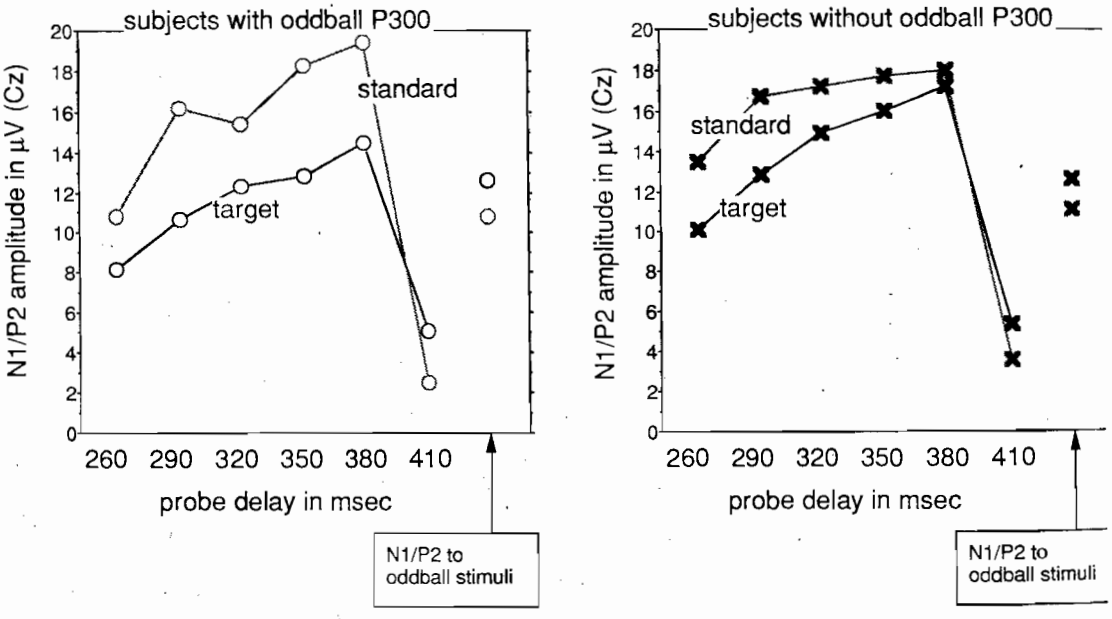

Figure 3 Peak-to-peak amplitudes of the N1/P2 vertexpotential averaged across subjects with (left) and without (right) oddball P300 separately for standard and target trials. The abscissa indicates the six different probe delays. The utmost right symbols represent the N1/P2 vertexpotential to standard and target stimuli in trials without probes.

($F(10,170) = 2.3$, $p < .05$) and DELAY × ELECTRODE ($F(10,170) = 16.8$, $p < .001$). Finally, there was a trend for an interaction GROUP × ODDBALL × DELAY ($F(5,85) = 2.0$, $p < .1$) indicating that in subjects exhibiting an oddball P300 (Fig. 3, left panel) the N1/P2-amplitude attenuation was more pronounced for probes at 290–380 msec delays (i. e., during the developing P300) than in subjects without oddball P300 (Fig. 3, right panel).

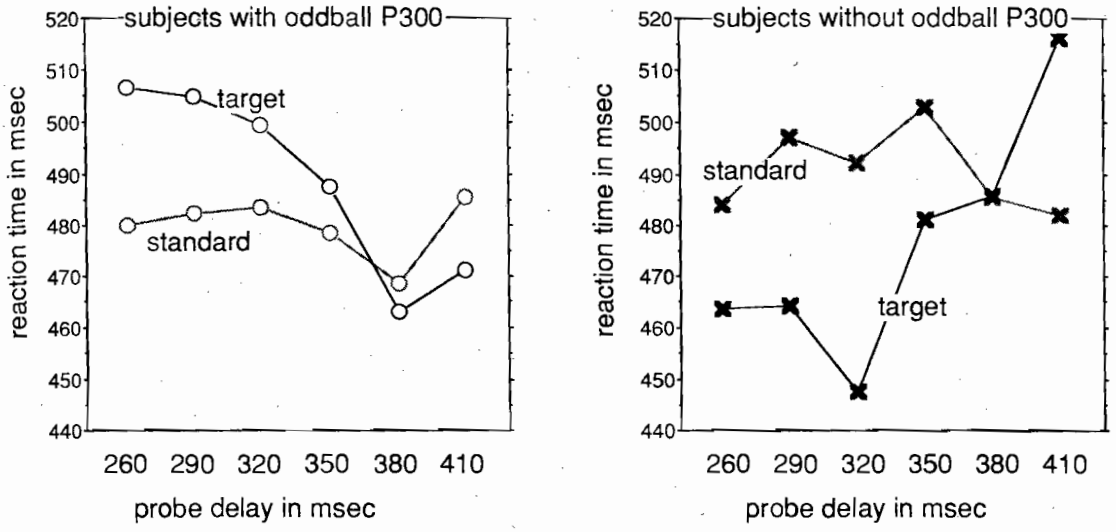

Figure 4 Reaction time averaged across subjects with (left) and without (right) oddball P300 separately for standard and target trials. The abscissa indicates the six different probe delays, the ordinate the median RT in msec.

Motoric Responses to Probe Stimuli

The reaction times (RTs) to probes are illustrated in Figure 4. The GROUP × DELAY × ODDBALL interaction reached significance ($F(5,85) = 2.4$, $p < .05$): Subjects with oddball P300 were slower when probes followed targets than when probes were added to standards for delays up to 350 msec (Fig. 4, left panel); subjects without P300 effect, on the other hand, showed a reversed pattern with faster responses to probes following targets, again up to 350 msec (Fig. 4, right panel). These group-specific tendencies are also documented by a *post hoc* comparison of the average RT for 290 and 320 msec delays relative to 410 msec delay (RT score: 290+320/2 – 410: $F(1,17)= 8.9$, $p < .01$). Independent of whether the probes followed target or standard stimuli, overall RTs (mean 485 msec) did not differ significantly. There was also no main effect of the probe delay.

Discussion

As in other reports on averaged ERPs in the standard oddball paradigm, we also found a parietally predominant P300 that was larger in response to targets than to standards, while small positivities and relatively large negative Slow Waves at the frontal and central recordings were contrary to expectations. However, these total averages seem to be due to interindividual variability, as approximately half of the sample showed a pronounced negative Slow Wave that was larger under target than under standard conditions. The present design differed from the standard oddball paradigm by introducing probe stimuli, thus creating a dual task situation. We can speculate that these conditions produced a modulation of the overall ERPs observed in the present study. In a dual task setting Horst, Ruchkin and Munson (1987) reported that P300 was smaller when elicited by the secondary task but enhanced in amplitude when elicited by the primary task. Israel, Chesney, Wickens and Donchin (1980) found decreased P300 amplitudes when a visual tracking task had to be performed in parallel to the acoustic oddball task. It is tempting to speculate that increased workload or a withdrawal of processing resources from the "primary" oddball task resulted in the ERPs observed here. While Israel et al. (1980) rejected the idea of P300 amplitude being sensitive to increased workload after raising the degree of difficulty in the secondary task, the authors discussed several possibilities of resource-requiring resource allocation modes under dual task conditions including, for instance, the "operation of a resource allocation policy" itself (p. 271). Horst et al. (1987) attributed the amplitude differences between single and dual task conditions to the overlap of positive- and negative-going ERPs and emphasized that the changes in the pattern of P300 and Slow Wave

amplitudes may be due, not to the reduced positive deflections, but rather to the addition of negative shifts with increasing workload. Negative Slow Waves have been observed to follow or overlap with P300 in various mental tasks and have been interpreted to "reflect further processing invoked by increasing task demands, beyond the processing that underlies P3b" (Ruchkin, Johnson, Mahaffey & Sutton, 1988, p.339). Although there seems to be some controversy regarding the nature, categorization and scalp distribution of Slow Waves (see for example Rösler & Heil, 1991; Ruchkin & Johnson, 1991), we may speculate that the dual task requirements in our experiment increased task demands and thereby led to the amplified Slow Wave development. This, in turn, may have increased individual differences in P300 or Slow Wave predominance. Since parietal P300 and frontally negative Slow Waves overlap in time, one might speculate that it is the balance of the two underlying processes, which becomes manifest in the "net" predominance of either P300 or Slow Wave and which reflects the preponderance of one or the other mental activity. Is the oddball P300 particularly predominant in those subjects who perform the oddball task with higher priority than the probe detection, while subjects developing a negative Slow Wave to targets pay more attention to the probes or are more distracted by the dual task situation? While retrospective reports from the subjects regarding their subjective task priorities (i. e., whether they counted the target first and then responded to the probe or vice versa) revealed no consistent differences between subjects with and subjects without oddball P300, only further experiments can answer this question by systematically varying the emphasis put on either task.

The dual-task effect of the present conditions does not interfere with testing the disfacilitation hypothesis of P300. We examined the possibility of whether P300 can be viewed as an indicator of widespread (distributed) cortical disfacilitation by presenting probe stimuli at different times within an oddball-task. We predicted that behavioral responses would be delayed and evoked potentials attenuated to these probes when they were processed during P300 generation. The probe-evoked N1/P2 amplitudes substantiate this hypothesis: Amplitudes were smaller when probes were associated with targets, particularly in those subjects producing large P300s. This supports our hypothesis of the disfacilitating nature of the P300. The effect was most pronounced in the range of 320 to 380 msec, but reversed for the 410 msec delay. This can be taken as further evidence for the postulated relationship of cortical excitation and stimulus processing, since the positive deflection often turns into a frontally negative shift during the time period when the N1/P2 is elicited by the 410-msec probes (i. e., around 550–600 msec).

Woodward et al. (1991) reported a U-shaped function of reaction times with slower responses to probes following targets. Their results are in line with the results of the present study in which the reaction times of subjects without oddball P300 were accelerated to probes following targets up to 350 msec. This finding is consistent with the hypothesis that a modulation of the reaction times can be predicted on the basis of the relative negativity of ERP components. This is even more compelling, if one considers that reaction times are a composite from a sequence of processing consisting of a number of stages: If we subtract ca. 120 msec, for the afferent and efferent transmission times, from the average reaction time (of 400–500 msec in the present study), about 300 msec remain during which the probe event is held in a stage of cortical processing, and it is during just this interval that the brain state, as measured by slow potentials, comes into play: If, for instance, a probe is presented with a delay of 290 or 320 msec after the ERP-eliciting stimulus, it will be processed during the entire P300 interval and will therefore be maximally delayed. If, on the other hand, the probe is presented 380 msec after a target, P300 positivity is still present at the stimulus onset, but much of the processing will occur beyond the positive brain potential. (It would therefore be interesting to present probe stimuli at delays between 100 and 200 msec as well, in future studies.) For the subject, the only way to avoid interference with processing the probe would be to delay processing the target stimulus. This may have been the case in subjects who did not develop an oddball P300. However, the covariation between the behavioral and the cortical measures argue against simple dual task effects. Interestingly, these suggestions received support in the results of Nash and colleagues (Nash & Pineda, 1984; Nash & Williams, 1982) who combined probe stimuli (as secondary

tasks) with visual or acoustic primary tasks of different complexity in order to induce different processing demands. Primary tasks that induced larger P300s were associated with reduced secondary task signal detection sensitivity (Nash & Pineda, 1984). The finding of a larger N100 to probe stimuli under conditions of high processing demands would argue against a simple dual task effect. Nash and colleagues interpreted their results to support a neural inhibition model of the P300.

Taken together, the present results suggest that the nature of event-related potentials such as P300 and negative Slow Wave can be examined by presenting probe stimuli, and they support the hypothesis of a disfacilitatory nature of positive slow potential shifts. Further studies will certainly be required to support this hypothesis, for instance, by presenting probes at shorter latencies relative to the oddball stimuli and/or prior to the oddball stimuli in order to "probe" both negative and positive potential shifts. Furthermore, it remains to be evaluated whether the interference by secondary tasks is a general effect or whether it is prominent only when both tasks are in the same modality, which would indicate an area-specific interference (as suggested, for instance, by Rösler & Heil, 1991).

Acknowledgements

Research was supported by the Deutsche Forschungsgemeinschaft (Ro 805).

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