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"Probing" the nature of the CNV

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Summary A widespread depolarization in the dendritic trees of cortical pyramidal neurons generates surface-negative potentials. In turn, such potentials may indicate facilitatory processes while positive-going waves may result from a lowering in cortical excitability. Accordingly, we may expect the processing of "probe" stimuli presented during surface-positive waves, i.e., during phases of lesser excitability, to be inhibited and probes presented during surface-negative waves to be facilitated. This hypothesis was tested by presenting acoustic probe stimuli at various points in time during a forewarned reaction time task. The warning stimulus (WS) elicited a late positive complex followed by a negative slow potential shift (CNV). In 75% of the total of 120 trials a probe could be presented 1.5 sec prior to the WS interval, 0.5, 1, 1.5 or 2 sec after the onset of the 3 sec visual WS, and 3 sec following the imperative signal (WS offset, requiring a fast button press response), while 25% of the trials were without any probe. Only one probe occurred during a trial. The EEG was recorded along the midsagittal line; responses to the probes were evaluated by reaction time (RT) and probe-evoked potentials. RT to probes presented late in the anticipatory interval were speeded up and probe-evoked potentials were enhanced during this interval, in parallel to the development of the slow potential and the CNV in particular. Results suggest that probe stimuli presented during the development of the CNV were processed more intensely, thereby supporting the hypothesis that slow cortical potentials indicate the timing of excitability in cortical neuronal networks. Such a tuning mechanism may serve as a basis for attentional regulation.

Key words: Slow cortical potentials; Contingent negative variation; Vertex potential; N100; Probes; Dual task; Attention

Attempts to uncover the relationship between the event-related potential (ERP) and behavioural measures have been concentrated on their covariate observations in various paradigms. Using this approach, a correspondence of surface-negative slow cortical potentials (SCPs) such as the contingent negative variation (CNV) and response facilitation has been inferred from the different experimental results (for summary see McCallum 1988; Rockstroh et al. 1989; Birbaumer et al. 1990; Rösler 1991). (1) Within the 2-stimulus reaction time paradigm a relationship between CNV and response speed has been reported, but only for certain conditions; (2) when behavioural tasks were presented contingent upon spontaneous negative or positive DC shifts, Stamm (1984) and Bauer (1984) observed an area-specific relationship between spontaneous negative waves and response efficiency (in terms of response speed and error rates); (3) biofeedback-induced negativities over the contralateral motor cortex speed up reaction time (Rockstroh et al. 1982). Response facilitation is not limited to motor responses: Rösler and colleagues (Rösler et al. 1986; Rösler and Heil 1991) and Brunia (summary, 1988) for example demonstrated that stimulus-preceding negativities which develop prior to feedback and cue stimuli vary in amplitude with the informative value of the stimuli if, "the subject waits for a stimulus which is expected to provide task-relevant information" (Rösler 1991, p. 126). Motor-related and stimulus-related negativities were distinguished on the basis of their topographical distribution, more widespread, extending to parietal and frontal sites, and a right hemispheric predominance of the stimulus-preceding negativity compared to the "motor" late CNV (Rösler 1991).

Furthermore, the facilitating nature of negativity may hold not only for slow negative waves: Loveless and Hari (1989), for example, found N100 amplitudes to be enhanced if the eliciting stimuli were separated from the preceding stimuli by not more than 100 msec. The authors concluded that the N100-eliciting stimulus was presented to a still activated network. The question remains as to what extent ERP components represent merely activation processes which become specific

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only by their temporal-spatial distribution, and to what extent, if at all, the presumed ERP subunits can be linked to very specific transformations of neurally coded information. Only if we can link ERPs to the dynamics of neural mass action can we hope to understand their significance for cognitive or behavioural processes. We have previously suggested a model which might account for certain features observable in extended neuronal networks (Elbert and Rockstroh 1987; Rockstroh et al. 1989; Elbert 1993): information may be coded by an increase or decrease in spontaneous firing rates of neurones, it may be coded through distinct spatial patterns of activation, but certainly not through the simultaneous activation of all neuronal elements at once. Therefore, excitability, as represented by the depolarization of the dendritic trees, must be regulated such that excitation is elevated in areas pertaining to the ongoing process but depressed in competitive areas. This mechanism would impose limits on the dynamic patterns of neural mass action (Elbert 1993). Measures of this mass activity would be the excitation and also the excitability in a certain cortical area. As depolarization in the apical dendritic trees of pyramidal cells results in a surface-negative potential, surface-negative shifts on the scalp, such as the CNV, are hypothesized to reveal enhanced cortical excitability enabling a preparatory state or "potentiality" for cerebral processing in the underlying networks (Rockstroh et al. 1989; Rösler 1990, 1991). In contrast, slow positive shifts may result from a "disfacilitation" in cortical neuronal networks.

We have used the probe technique to test the suggested relationship between polarity of slow cortical potentials and facilitation to investigate further the functional state of the cortex during the development of an ERP: probe stimuli during surface negative potentials would be presented to already excited cell assemblies and, hence, ignition of the corresponding cell assemblies would be faster and more widespread; on the other hand, if positive ERP components such as the P300 indicate reduced excitability in cortical neuronal networks, the processing of probe stimuli presented during the development of a P300 should be inhibited. Facilitation and inhibition of the processing of probe stimuli can be evaluated by measuring the evoked potential to the probe stimuli or the reaction time if a motor response to every probe stimulus is required. In a previous study (Rockstroh et al. 1992) we used the "probe" technique to investigate the functional brain state during P300 evocation. Within an acoustic oddball paradigm probe stimuli were presented at different delays following standard or target stimuli. In subjects who developed an "oddball P300" the amplitude of the vertex potential (N1/P2) was attenuated and motor responses to the probe stimuli were delayed when probes followed target stimuli in the range of P300 development as compared to trials in which probes followed standard stimuli. These results support the hypothesis that widespread positive waves indicate reduced excitability of cortical neuronal networks. In the present study, the probe technique was used to study the functional brain state during surface negativity. According to the hypotheses outlined above it was hypothesized that motor responses to probes would be faster and that the evoked potential amplitudes would be larger when probes were presented during the anticipatory interval, i.e., during a CNV, relative to probes presented before or after the anticipatory interval. Using a similar probing technique, some evidence in favour of this hypothesis has been found in two previous studies by Wagner et al. (1993): when task-irrelevant auditory probes were presented half way between a visual warning stimulus and a visual imperative stimulus occurring 3 sec later, the vertex potential (N1/P2) evoked by these probes was larger compared to probe-evoked potentials to tones presented briefly before the warning stimulus. In another related study (Wagner 1991), auditory probes were presented early or late during a 3 sec interstimulus interval. Up to 3 more probes occurred during the interval between trials. More than the probes during the early phase of the interstimulus interval, the probes during the later phase of the ISI were found to evoke larger N1/P2 relative to probes presented during the ITI. The design of the present study should allow for a more detailed analysis of the time course of probe-evoked responses in relation to the time course of slow potential changes.

Methods

Subjects

Thirty healthy right-handed student volunteers (15 males, 15 females, mean age 25 years) were paid for their participation in the experiment that lasted for about 1 h. Right-handedness was verified by a modified version of the Edinburgh handedness questionnaire. It was assured that subjects were not under medication and had not suffered from any central nervous system abnormality.

Design and procedure

Within a reaction time paradigm a scattered light of 3 sec duration was presented as warning signal (WS; a 60 W bulb not observable by the subject indirectly illuminated the wall in front). Subjects were asked to press a black button with the left hand as fast as possible after the offset of the WS. Clicks served as acoustic probe stimuli; they could occur during the baseline (1.5 sec prior to WS onset), during the anticipatory interval (0.5 sec, 1.0 sec, 1.5 sec and 2.0 sec

following WS onset) and 3.0 sec following WS offset. No more than one probe was presented during a trial. Probe delays were chosen to occur at various times to track the development of the slow potential. Probe stimuli presented 3.0 sec following WS offset and 1.5 sec preceding WS onset served as control conditions as they were expected to be presented during periods without systematic slow potential shifts. Subjects were asked to press a red button, which they held in the right hand, as fast as possible to each click. Clicks were presented via earphones.

A total of 120 trials were presented. 25% (30 trials) were without a probe. In 25% of the trials a probe could occur either at 0.5 or 1.0 sec following WS onset (15 trials each). In 25% a probe could occur either at 1.5 or 2.0 sec delay from WS onset (15 trials each). In 25% a probe could occur 3.0 sec following WS offset or 1.5 sec prior to WS onset. The respective types of trial followed each other in random order. The interval between successive lights varied randomly between 6 and 10 sec.

After the preparation for recording the subjects received written instructions. In addition to a description of the tasks to be performed, the instructions informed the subjects that they were to adopt a relaxed position and to fixate their eyes on a spot on the wall in front of them in order to avoid possible head and eye movements. The experimental series of 120 trials was preceded by 10 practice runs; during 5 of these only the light was presented in order to familiarize the subject with the warned reaction time paradigm. The other 5 trials also comprised probe stimuli for the subject to practice the dual task. Practice trials were supervised by the experimenter.

Apparatus and physiological recordings

An ASYST programme (a scientific programming system for the control of experiments and data acquisition), running on an AT 386 computer equipped with a DT 2821 board (DMA) controlled timing of the experimental stimuli and the storage of reaction times and electrophysiological responses. The acoustic probe stimulus was a 400 Hz tone presented for 20 msec at 80 dB APL (rise and fall times 10 msec) binaurally via earphones. Coulbourn Instrument modules controlled the AC light bulb and the tone bursts. Response buttons were ordinary microswitches.

The EEG was recorded along the mid-sagittal line from frontal (Fz), central (Cz) and parietal (Pz) leads with a time constant of 30 sec, high-frequency cut-off 30 Hz. The reference electrode was affixed to the right earlobe. Non-polarizable silver-silver chloride electrodes (ZAK) were used for EEG recording, Grass EC2 electrolyte served as the conducting agent. The skin under the electrodes was prepared by cleansing with alcohol and removing the outer layers of the skin by rubbing with abrasive paste (Omniprep). The vertical EOG was recorded via Beckman silver-silver chloride electrodes, centred 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. EEG and EOG were amplified using a Beckman R504 polygraph. All data were digitized at a rate of 100 Hz and were stored for off-line analysis. Response latency was stored to the nearest millisecond via the digital input to the interface board.

Data reduction and analysis

Trials with an EOG or an EEG shift exceeding 150 μV were rejected from further analysis. For EOG artefact correction the impact of blinks obtained from calibration intervals on the EEG records were estimated by regression analyses; subsequently ocular activity was removed from the averaged EEG traces using the weights determined in the calibration intervals (Elbert et al. 1985). Data of 5 subjects were excluded from further analysis because less than 50% of the trials met the criteria. In the remaining sample, artefact rejection procedures resulted in the rejection of 11% of the 120 trials (106.7). From the remaining trials, EOG scores were determined in the same way as the ERP scores (see below).

The course of the slow potential shift during the WS interval was described by the mean negative shifts during the intervals 0.5–1.5 sec, 1.5–2.5 sec and 2.5–3.0 sec following WS onset. Responses to probe stimuli were determined by the evoked potential components N1 and P3 and reaction time (RT). It was assumed that this evoked potential would be "riding" on top of the CNV. In order to extract the evoked potential components from this composite ERP point-by-point difference curves between trials without probes (as a "template") and each of the probe conditions (with probes during the baseline, 0.5 sec following WS onset, etc.) were calculated in a first step. From these 6 difference curves the maximum negative deflection and the maximum positive deflection between 80 and 400 msec relative to a 100 msec interval prior to the probe were determined as N1 and P3, respectively. Amplitude and latency scores were determined for the 3 electrode locations, Fz, Cz and Pz. The median reaction time (RT) in every probe delay condition was determined as score for the motor response to probes.

Differences between conditions and recording sites were evaluated by means of analyses of variance with the within-subject factors Electrode (comparing Fz, Cz and Pz) and Condition (comparing trials without probes and trials with probes presented at the 6 different time points). All reported P values were obtained after adjustment of the degrees of freedom with the Greenhouse-Geisser-Epsilon. Means $\pm$ standard errors are presented.

Results

Motor and slow potential responses in the forewarned reaction time (primary) task

Subjects, on average, pressed the button to WS offset after 332 msec. In essence, these RTs were not sensitive to probe presentation with two exceptions: reaction time was faster by 25 msec when probes were presented 1 sec following WS onset compared to trials without probes (302 ± 11 versus 327 ± 13 msec; t (24) $= 2.6$, $P < 0.05$), but delayed by 22 msec when probes were presented during the intertrial interval, i.e., 3 sec

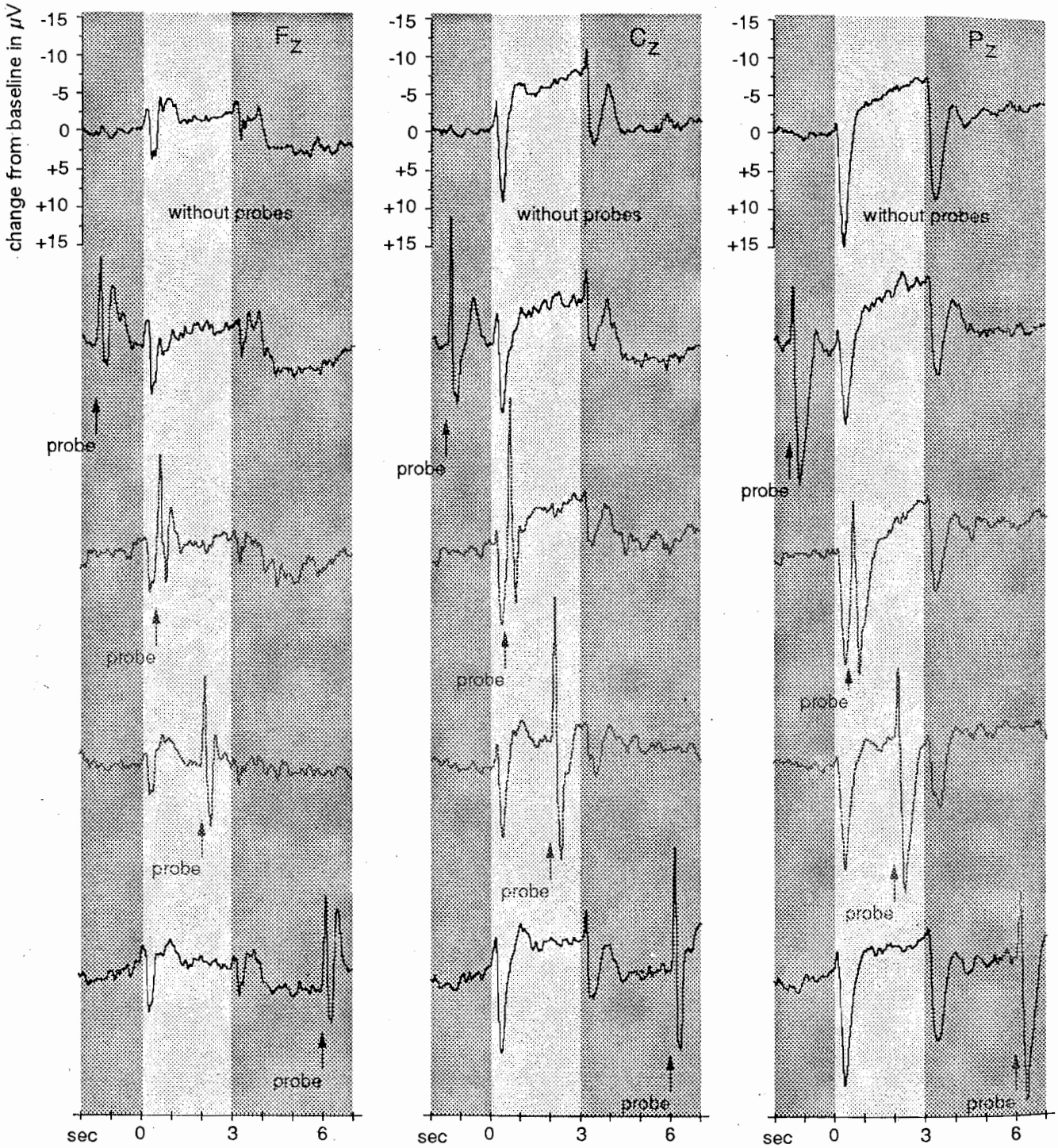

Fig. 1. Across-subject averages of CNV during trials without probes (top row) and during trials with probes presented during baseline (2nd row) 0.5 sec after WS onset (3rd row), 2 sec after WS onset (4th row) and during the intertrial interval (bottom row). The white areas mark the WS interval (3 sec duration). Left column: frontal, middle column: central, right column: parietal records.

after WS offset (349 ± 11 msec; t (24) = 2.6, $P < 0.05$). Obviously, the presentation of probes prior to the imperative event, even prior to the WS, speeds the response to the subsequently occurring primary task. The differences gave rise to a main effect of Condition (F (6, 144) = 4.7, $P < 0.01$).

Fig. 1 (top) illustrates the slow cortical potentials during the 3 sec WS interval on trials without probes. An evoked potential with a pronounced positive component to the onset of the visual WS was followed by a CNV with its distinct topographical distribution. The peak shortly after WS onset is evident only over the frontal and central areas; the following steady increase in CNV amplitude up to the termination of the visual WS and the motor response can be seen at the central and parietal locations. While negativity develops during the first 1.5 sec primarily over frontal and central areas (Electrode: F (2, 48) = 20.5, $P < 0.01$), a centro-parietal predominance is indicated for the second interval (1.5–2.5 sec: F (2, 48) = 43.7, $P < 0.01$) and the terminal CNV (tCNV) (2.5–3.0 sec: F (2, 48) = 50.9, $P < 0.01$).

Fig. 1 also illustrates the ERPs during the WS interval in trials with probes added. Up to the time of presentation of a particular probe stimulus, ERP wave shapes were similar for the different conditions. However, the presentation of a probe markedly influenced the subsequent course of the slow potential. If probes were presented early in the WS interval, negativity reached the same amplitude towards the end of the WS (tCNV) as in trials without probes.

Motor responses and evoked potentials to probe stimuli (secondary task)

Overall, responses to the acoustic probes were slower than responses to the offset of the visual WS (mean 363 msec); for the mean RTs to WS offset and the mean RTs to probes averaged across all conditions the ANOVA provided a main effect of F (1, 24) = 4.96, $P < 0.05$). Within-trial differences between RTs to WS offset and RTs to probes reached significance when probes were presented during the baseline (t (24) = 2.5, $P < 0.05$) and early in the WS interval (t (24) = 3.4, $P < 0.01$ for probes presented 0.5 and 1 sec after WS onset; F (5, 120) = 5.1, $P < 0.01$ for the interaction; for the main effects WS vs. probe: F(1, 24) = 4.9, $P < 0.05$, and Condition: F (5, 120) = 4.2, $P < 0.01$).

RTs to probes were significantly faster when probes were presented early (1 sec) and late (2 sec) during the WS interval compared to the other conditions (F (5, 120) = 3.4, $P < 0.05$, see Fig. 2). Post hoc comparisons of RTs to probes during baseline and probes during the WS interval provided significant t values for the probe delays at 1 sec (t (24) = 2.6, $P < 0.05$) and 2 sec (t = 2.8, $P < 0.01$).

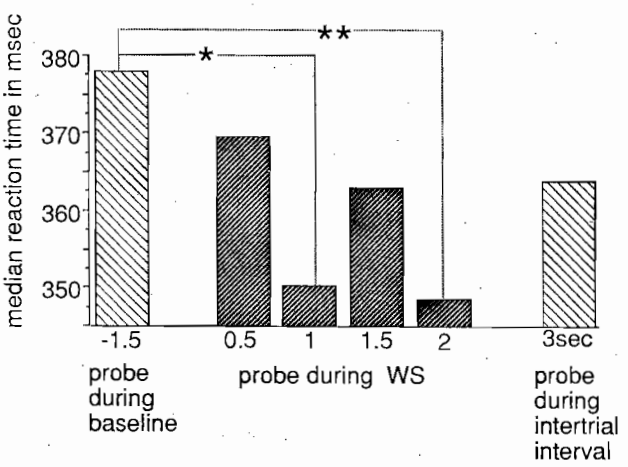

Fig. 2. Reaction time to probes, averaged across subjects (ordinate: median RTs in msec) separately for the different probe delays (abscissa). Significant differences between RTs to probes during the baseline and probes during the WS interval are marked by dotted lines; * $P < 0.05$; ** $P < 0.01$.

The *evoked potential* to probe stimuli showed a negative deflection (N1) with a mean latency of 153 ± 2.6 msec at Fz, 146 ± 2.0 msec at Cz and 136 ± 1.5 msec at Pz, a mean amplitude of 10.1 ± 0.5 μV at Fz, 15.4 ± 0.6 μV at Cz and 7.5 ± 0.4 μV at Pz (the fronto-central maximum is documented by a main effect Electrode F (2, 48) = 49.1, $P < 0.01$ for amplitude, and F (2, 48) = 25.1, $P < 0.01$ for latency), which was followed by a positive deflection and a frontally negative slow wave. The P3-like positive wave exhibited a parietal maximum of 18.1 ± 0.7 μV at 356 msec latency, while it showed smaller and earlier peak amplitudes at Cz (13.1 ± 0.7, 325 msec) and Fz (7.5 ± 0.5 μV, 320 msec). Main effects Electrode for P3 amplitude (F (2, 48) = 46.2, $P < 0.01$) and latency (F (2, 48) = 16.9, $P < 0.01$) confirm the parietal maximum of this positive deflection. Inspection of the N1 and the P3 amplitudes revealed an increase of both, primarily late in the WS interval, i.e., both were larger to probes 2 sec after WS onset than to probes 1.5 sec prior to WS onset (main effects of Condition for the N1 amplitude: F (5, 120) = 3.1, $P < 0.05$, and the P3 amplitude: F (5, 120) = 4.5, $P < 0.01$; see Table I for post hoc comparisons). The parallel development in N1 and P3 amplitudes justifies the examination of the peak-to-peak probe-EP, which will be henceforth called N1/P3. Such a measure is of interest as it is not affected by the slower potential changes.

The N1/P3 (with central maximum, F (2, 48) = 65.5, $P < 0.01$) was larger when probes were presented during the WS interval compared to when a probe was presented during the baseline interval of the forewarned reaction time trial (see Figs. 1 and 3). Unexpectedly, the N1/P3 to probes after the WS interval was also significantly larger than the N1/P3 to probes

TABLE I

Statistical evaluation of differences between EPs evoked by a probe stimulus presented during baseline, with the EPs evoked at different latencies during and after the WS interval. *P* values for separate post hoc comparisons by means of Newman-Keuls test are presented for N1-P3 peak-to-peak amplitude, and N1 and P3 baseline-to-peak amplitudes, and for the three recording sites (Fz, Cz, Pz).

Location	Difference in amplitude between probes during baseline and probes at				
	0.5 sec	1 sec	1.5 sec	2 sec	3 sec post
Frontal					
N1/P3	0.02	0.06	0.01	0.001	0.02
N1	n.s.	n.s.	n.s.	0.09	0.01
P3	n.s.	n.s.	n.s.	n.s.	n.s.
Central					
N1/P3	n.s.	0.10	0.05	0.001	n.s.
N1	0.07	n.s.	n.s.	0.05	0.06
P3	n.s.	0.10	0.10	0.01	n.s.
Parietal					
N1/P3	0.08	0.07	0.04	0.01	0.08
N1	0.16	n.s.	n.s.	n.s.	0.18
P3	n.s.	n.s.	n.s.	0.06	n.s.

n.s.: *P* values above 0.15.

prior to the WS interval (Condition, $F(5, 120) = 6.6$, $P < 0.01$). Table I summarizes the multiple post hoc comparisons of these effects for the differences in N1, P3 and N1/P3 amplitudes as illustrated in Fig. 3. At the central location the N1/P3 to probes prior to WS offset (2 sec delay) was significantly larger in amplitude than the N1/P3 to the probes during the intertrial interval (3 sec after WS offset; $t\ (24) = 3.1$, $P < 0.01$).

As can be inferred from Fig. 1, the N1/P3 overlapped with the subsequent development of a slow wave with clear-cut negative peak over frontal, but positivity over parietal, areas. While pronounced for probes during the ITI (see Fig. 1, e.g., lower left trace), this component was largely suppressed for probes during the WS interval (see Fig. 1, e.g., middle traces in the left column).

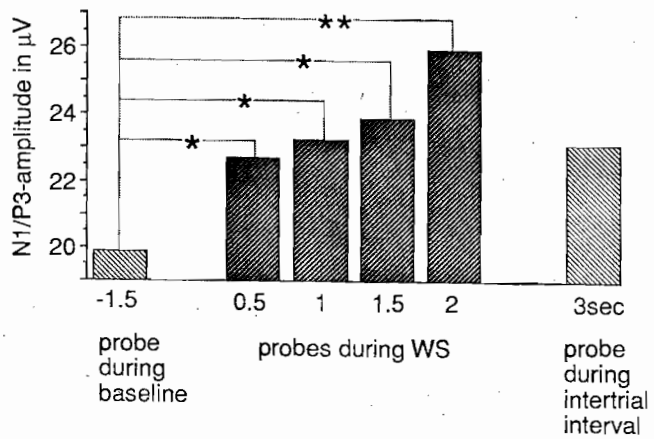

Fig. 3. Evoked potentials to probes, averaged across subjects (ordinate: peak-to-peak N1/P3 amplitude in µV) separately for the different probe delays (abscissa). Significant differences between N1/P3 to probes during the baseline and probes during the WS interval are marked by dotted lines; * $P < 0.05$; ** $P < 0.01$.

Discussion

Both dependent measures, probe RT and probe-evoked potentials, indicate a faster and a more aroused response in parallel with the development of the CNV.

Applying the probe paradigm bears a number of traps. As in quantum mechanics, the process of measurement significantly interacts with the state to be evaluated: testing the excitability of cortical structures with a secondary — probe — stimulus will significantly alter the time course of the slow brain potential that is associated with cortical excitability. Whenever we want to investigate the behavioural implications of probe presentation a dual task situation will emerge. It is therefore not astonishing that probe presentation modulated the RT to WS offset (as indicated by the Condition effect); a probe early in the WS interval may have facilitated the concentration on the primary task, while probes presented near WS offset slightly impaired this concentration. Furthermore, the comparably small amplitudes of the terminal CNV can be discussed as the consequence of dual task requirements. Attempts to specify "primary" and "secondary" tasks are to some extent arbitrary. For the present study we could argue that the faster responses to a visual stimulus with the left hand, compared to the slower right-handed responses to the click, assign the former as the primary and the latter as the secondary task. However, responses to the visual stimuli were also more frequent and the responses were forewarned.

Another problem relates to the responses observed during the post-stimulus interval. Both, the RT and the EP differed significantly between the pre-trial baseline and the post-trial presentation. As can be inferred from Figs. 2 and 3, the modulation of responses evoked during the CNV interval seems to persist during the post-stimulus interval, albeit the modifications are smaller in amplitude. We think, however, that probe-evoked responses during the post-stimulus intervals are confounded by their fixed time relation to the primary task. Only a higher number of incidences at which probe stimuli would occur during the post-stimulus interval might reveal more about the nature of the processes which dominate the intervals after completion of a primary task.

One may ask whether the modulation of probe-evoked potentials during the course of the CNV depends on the task relevance of the probes and thus may be limited to dual task paradigms like the one employed in this study. The study by Wagner (1991) directly addressed this issue by asking one group of subjects to react to the auditory probe stimuli while

other subjects were instructed to ignore the stimuli and to focus exclusively on the visual forewarned reaction time task. The observed increase in N1/P2 amplitude of the probe-EP, especially during the late phase of the interstimulus interval, as compared to the probe-EP amplitude observed during the intertrial interval, was exactly the same in both groups. Apparently the increased excitability during the CNV enhances the processing of all stimuli, whether relevant by instruction or not.

In the present study, both dependent measures, probe RT and probe-evoked potentials, became altered in parallel to the development of the CNV, indicating faster or more efficient response. These results support our hypothesis that the CNV represents increased cortical excitability, thereby facilitating the processing of stimuli presented to a more easily excitable network (Elbert and Rockstroh 1987; Elbert 1993). This description of the significance of SCPs is substantiated by the results of complementary studies (Woodward et al. 1991; Rockstroh et al. 1992) in which probes were presented during the development of a P300. Probe-evoked potentials came out smaller in amplitude and motor responses became slower when subjects had to process them in the presence of the widespread positivity. Furthermore, those subjects who did not develop an "oddball P300" did not develop such an attenuating effect (Rockstroh et al. 1992). Although the coincidence is correlational in nature, we consider the parallel development of the probe measures with the slow brain potentials across time a strong clue unravelling the nature of neural mass action. The previously and presently observed — quite simplistic — relationships point to the possibility that *slow* endogenous event-related brain potentials are just global signs of attention and arousal rather than reflections of specific steps in the flow of information processing. Given the various efforts to nail down consistent relationships between ERP components and specific psychological constructs, the limited success supports our view of a rather limited specificity of ERP. We should not expect that the plastic and variable neural networks with their non-linear dynamics would specifically respond in the same way over tens or even hundreds of trials.

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