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# ON THE INDEPENDENCE OF THE CNV AND THE P300 COMPONENTS OF THE HUMAN AVERAGED EVOKED POTENTIAL[1]

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The form of the average cortical potential is determined both by the physical characteristics of the eliciting stimulus and by the nature of the information processing activities invoked by that stimulus. In particular, much evidence has shown that P300, a component with a positive polarity, and a modal latency of about 300 msec, is considerably enhanced in the average evoked potential (AEP) when the eliciting stimulus invokes information processing activities (Donchin and Cohen 1967; Donchin *et al.* 1973). The evidence is strong that P300 represents endogenous cortical processes (Sutton *et al.* 1967; Klinke *et al.* 1968; Weinberg *et al.* 1970).

It has been suggested by several investigators (Karlin 1970; Näätänen 1970) that the amplitude of P300 is determined by generalized preparatory activity preceding the eliciting stimulus rather than by processes invoked by the stimulus. Such accounts for P300 stress that in most experimental circumstances in which P300 is enhanced by the eliciting stimulus it is preceded by a contingent negative variation (CNV). This event-related potential (ERP) has been shown to precede forewarned events requiring a response by the subject (Walter *et al.* 1964). It has been proposed therefore that P300 is essentially a reflection of the CNV preceding the eliciting stimulus. This argument has taken several different forms, ranging from the suggestion that the resolution of the CNV and P300 are possibly coincident in time (Donchin and Smith 1970) to suggestions that the P300 component is essentially a "reactive change" to the development of the CNV (Karlin 1970).

It has, however, been demonstrated that the CNV and P300 can be dissociated. Thus, for example, it has been shown that the amplitude of P300 can vary though preceded by CNVs of equal amplitude (Lombroso 1969; Donald and Goff 1971; Donchin *et al.* 1972; Tueting and Sutton 1973). It has also been shown that P300 can be elicited in experiments in which differential anticipatory activity by the subject preceding the eliciting stimulus was precluded (Donchin and Cohen 1967; Eason *et al.* 1969; Tueting *et al.* 1970; Harter and Salmon 1972; Friedman *et al.* 1973; Hillyard *et al.* 1973). Yet the issue persists and several recent reports were interpreted as supporting the notion that the P300 component is the poststimulus concomitant of the processes which prior to the presentation of the stimulus result in the development of the CNV (Hartley 1970; see Regan 1972; Wilkinson and Lee 1972; Wilkinson and Spence 1973).

The investigation reported here emerged from a discussion of the relationship between the CNV and P300 conducted as part of the deliberations of the Third International Congress on Event Related Slow Potentials of the Brain that was held in Bristol, England, in the summer of 1973 (McCallum and Knott, in press). We refer the reader to the proceedings of that conference for a very detailed airing of the theoretical issues and the various points of view reflected at the con-

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ference (Donchin, in press). The consensus of the discussants has been that, in part, the difficulty in resolving the issue derives from the lack of experiments which attempted to manipulate simultaneously P300 and the CNV. A design for such an experiment was developed in which variables known to affect either P300 or the CNV would be manipulated. We report here the results of a factorial experiment the intent of which was to determine the degree to which the CNV and P300 do interact in determining the final waveform of the cortical evoked potential.

## METHOD

### *Experimental design*

An experimental session consisted of series of trials (runs). On each trial the subject was presented with either a high (1200 c/sec) or a low (800 c/sec) tone. There were "Reaction Time" runs in which the subject had to press one of two switches, as fast as he could, and there were "Predict" runs in which the subject had to guess which of the two tones would be presented on the following trial.

Within any run some of the tones were preceded, at an interval of 1000 msec, by a warning flash. On other trials the tones were presented without warning. The subject was always informed as to whether or not there would be a warning stimulus on any given trial. The letter A was displayed in a tachistoscope when warning stimuli were included in the trials, the letter B was displayed when no warning stimuli were used. Runs also differed in the sequence with which the tones were presented. In some runs the tones were presented in a regular, alternating sequence, that is, high tones always followed low tones and *vice versa*. On other runs the tones were presented in an irregular, random, sequence where the probability that a high or a low tone would be presented on any trial was equal to 0.5 and was independent of the outcome of the previous trial.

There were thus three independent variables, with two values per variable—the subject's task (reaction time *vs.* predict), the sequence of tones (random *vs.* alternate), and the presence or absence of the warning stimulus (warned *vs.* unwarned). As each variable was completely crossed with the other two variables there were 8 different types of runs, for the 8 possible combinations of the 3 variables.

### *Subjects*

Twelve undergraduates (8 male) were paid for participating in the experiment.

### *Electrodes and recording*

Thirteen Beckman biopotential electrodes (No. 6509), filled with Beckman electrode paste, were affixed to the subject's scalp with collodion. Electrode impedance, measured with a Grass E-Z-M impedance meter, did not exceed 10 kΩ. Electrodes were placed at $C_z$, $F_{p_2}$, $C_3$, $C_4$, $P_3$, $P_4$ and $O_1$ and referred to a linked-mastoids electrode. The subject was grounded by a mid-forehead electrode. The electrooculogram (EOG) was recorded between a supraorbital and a canthal electrode. The EEG was amplified with Brush amplifiers (No. 13-4218-00), set to a band pass of 0.01–30 c/sec (6 dB/octave roll-off). Data were recorded, at 1 7/8 ips, on a Hewlett-Packard 3955 FM tape recorder. Analog-to-digital conversion and averaging were performed on an IBM 1800 computer.

### *Stimuli*

The subject was seated in a reclining lounge chair in a Faraday cage located in a darkened room. He looked into an Iconix 3-field look-into exposure box (No. 6134) with cold cathode-fluorescence lamps which illuminated black on white letters, the letter "A" or the letter "B", subtending 55 min horizontally and 1 deg 26 min vertically. Field luminance was 0.74 mL. One of the two letters was always illuminated.

The third field of the tachistoscope contained the warning stimulus which consisted of a transilluminated annulus (inner diameter 39 min, outer diameter 41 min). The duration of the warning stimulus was 100 msec, its luminance was 8.68 mL.

The high, 1200 c/sec tone was generated by a Hewlett-Packard push button oscillator (No. 314A). The low, 800 c/sec tone was generated by a Wavetek function generator (No. 114). The 50 msec tone burst was gated through Iconix audio gates and presented to the subject binaur-

ally through Grason-Stadler earphones (No. TDH39-10Z), at an SPL of 65 dB re. 0.0002 $\mu$ bar.

*Procedures*

Each subject was studied in a single 105 min session. Trials were presented in blocks (subjects rested between blocks). Each block included 32 trials free of EOG activity. The subject fixated the letter ("A" or "B") which informed him of the regime he was operating under (*i.e.*, whether or not a warning stimulus would be presented). The experiment was controlled by a LAB 8/E system (Digital Equipment Corporation) through an Iconix logic system (system No. 136). The presentation of warned trials in a series was random with the constraint that a group of warned or unwarned trials was at least 2 and at most 14 trials long. The transition was always at the onset of the intertrial interval. Seven channels of EEG were digitized, averaged and displayed on-line. For each trial the computer squared and summed all the values on the EOG channel. If this sum of squares exceeded a preselected criterion value the trial was rejected. The criterion value, determined in preliminary work, faithfully led to the rejection of trials contaminated by eye movement artifacts.

During the reaction time series the subject held a two-button response box in his lap. He pressed one of the two buttons with either the right or the left middle finger. A force of 450 g was required to trip each of the two microswitches. In reaction time trials the subject had to respond within a prespecified interval (which for different subjects ranged between 350 and 500 msec); the tone was repeated at the end of that prespecified interval whenever the subject's reaction time exceeded it. Trials on which the tone was repeated were not used for averaging. A random interval ranging between 4.5 and 6.5 sec elapsed between trials. In the predict series the experimenter indicated to the subject when to make his prediction. The experimenter then entered the subject's prediction into the computer and started the trial.

The sequence of reaction time and predict sessions was counterbalanced across subjects, as was the relationship between tone frequency and the responding hand.

For the first 4 subjects the interval between the warned and the imperative stimulus was 800 msec long; they will be treated separately. This report is primarily concerned with the last 8 subjects, with whom a foreperiod of 1000 msec was used.

## RESULTS

The primary data analyzed in this report consist of the cortical evoked potentials elicited from seven electrode locations in each of 8 subjects in the 8 different experimental conditions. Each of the evoked potentials presents one combination of the three independent variables. In any run a subject could perform one of two tasks, trials could include, or not, a warning stimulus and the sequence of trial outcomes could be predictable or unpredictable. In Fig. 1 we summarize the experimental comparisons that need to be made in this experiment. Each panel of the figure presents the 8 AEPs, recorded at the vertex electrode, and averaged over all 8 subjects. These "grand averages" are presented only as an aid in showing waveform differences. An impression of the range of inter-subject variability can be gained from Fig. 1, *D* where the AEPs from each of the 8 subjects for one condition are shown. Measurements were made on each individual subject's waveform.

In the first three panels of Fig. 1 we have superimposed the 8 AEPs to emphasize the effects of each of the three independent variables. In panel *A* we show the effects of outcome predictability (random *vs.* alternate), in panel *B* we study the relation between the task (reaction time *vs.* predict) and the AEPs and in panel *C* we evaluate the effects of the presence or the absence of the warning (warn *vs.* unwarned) stimulus. It is clear that a large CNV is elicited on trials in which a warning stimulus preceded the imperative stimulus (see panel *C*). Equally clear is the enhancement of the P300 component during the predict series when the tones are presented in a random sequence. Other relationships are also suggested by the figure. The CNV appears larger during the performance of a reaction time task; furthermore, larger CNVs are elicited when the tones are presented in a random sequence and the effect of stimulus uncertainty

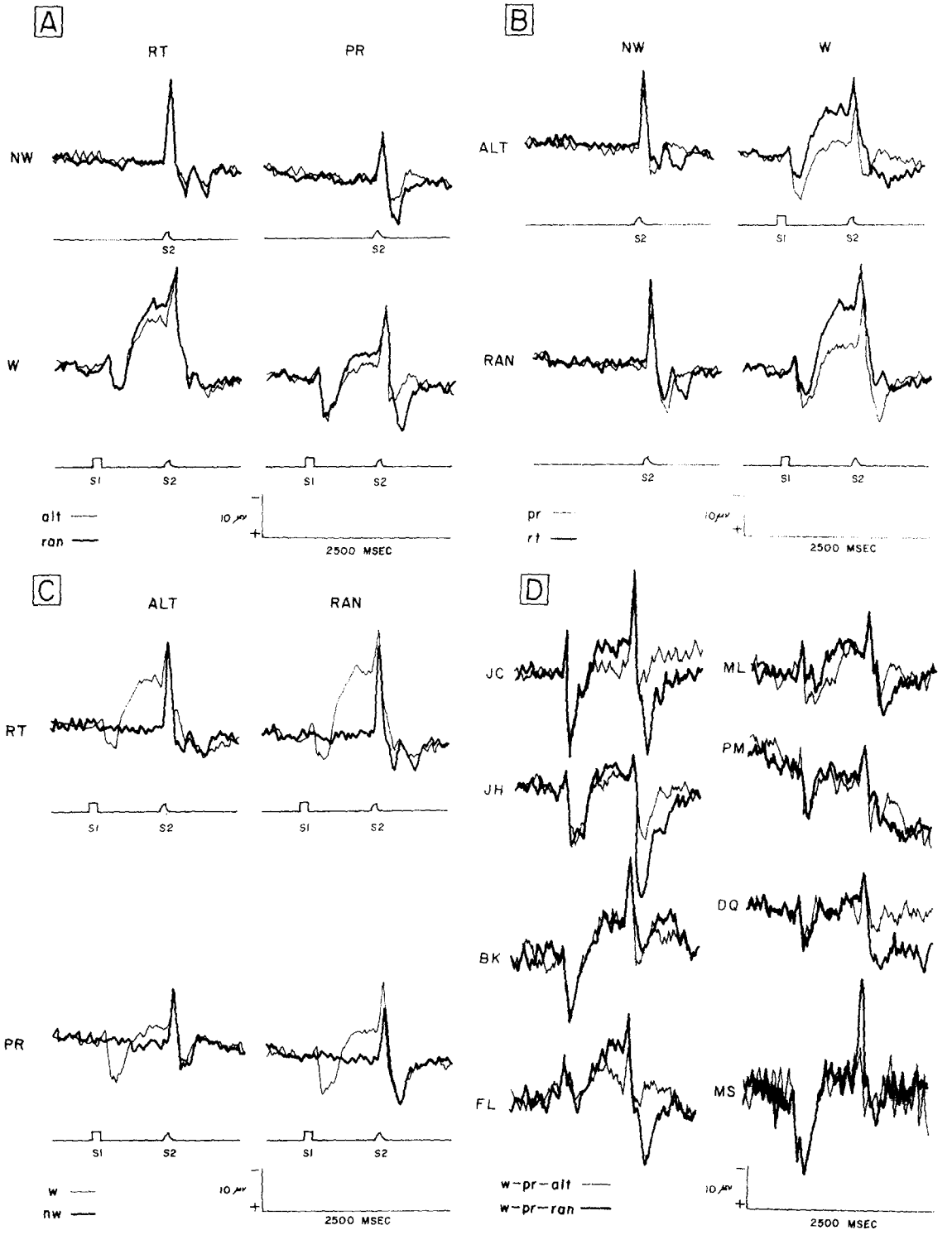


Fig. 1. A comparison of cortical evoked potentials at the vertex electrode averaged over all subjects for each of the 8 experimental conditions. Each of the first three panels presents a direct comparison of one of the three independent variables at each of the 4 combinations of the other two independent variables as follows. In *A* we superimpose data obtained with a random and alternating presentation of S2, at *B* we superimpose data for the predict and the reaction time conditions and at *C* we superimpose averages obtained in the presence or absence of a warning stimulus. (In this, and in the following figures and tables: RT, reaction time; PR, predict; W, warning stimulus present; NW, warning stimulus absence; ALT, high and low tones alternate; RAN, high and low tones vary randomly from trial to trial.) In panel *D* we plot the evoked responses obtained from each of the 8 subjects for the W–PR–ALT and the W–PR–RAN conditions.

on P300 amplitude is clearly reduced during a reaction time task (panel *B*).

Most of these observations are neither novel nor suprising. In fact, we replicate here a substantial body of experimental literature. The figure does demonstrate that each of our three independent variables exercises the anticipated effect on either the P300 or the CNV. Our purpose in this study was to determine the degree to which these three variables interact in determining the amplitude or waveform of the P300 and the CNV. A study of Fig. 1 reveals that the assessment of such interactions by visual inspection of the data would not be possible. It is particularly important, in the present case, to define measures of P300 and the CNV that would not be *a priori* interactive. The difficulties of visual inspection increase if we consider the need to study the distribution of the potentials across the scalp.

Our analysis was based on the following measurements (see Fig. 2): For each AEP 8 levels were defined: (a) the arithmetic mean of the activity preceding the presentation of S1 by 500 msec (BASE); (b) the peak of the N100 component of the S1 evoked potential (N1S1); (c) the peak positivity within 500 msec after S1 (PS1); (d) the maximum negative amplitude over the portion of the interstimulus interval preceding S2 by 300 msec (CMAX); (e) the average of the activity over that interval (CAVG); (f) the peak of the N100 component for the evoked potential associated with the second stimulus (N1S2); (g) the peak of the P300 component for this second AEP (P3S2)—the range was 250–350 msec post S2; (h) the average level of the evoked potential over the final 200 msec of tracing (*i.e.*, 800 msec after S2) (POST). Using these 8 levels we obtained for all subjects, for all electrode locations and for all experimental conditions the 16 measurements indicated by the arrows in Fig. 2.

These peak-to-peak and base-to-peak measurements are often obtained in evoked potential studies. Usually, a few are used in any one study. Two problems arise, first these various measures are intercorrelated to varying extents. Secondly, there are no rational rules for preferring one of these measures over the others. A possible solution to this problem is to develop a set of composite measures from these raw measures. Each composite will be some weighted function

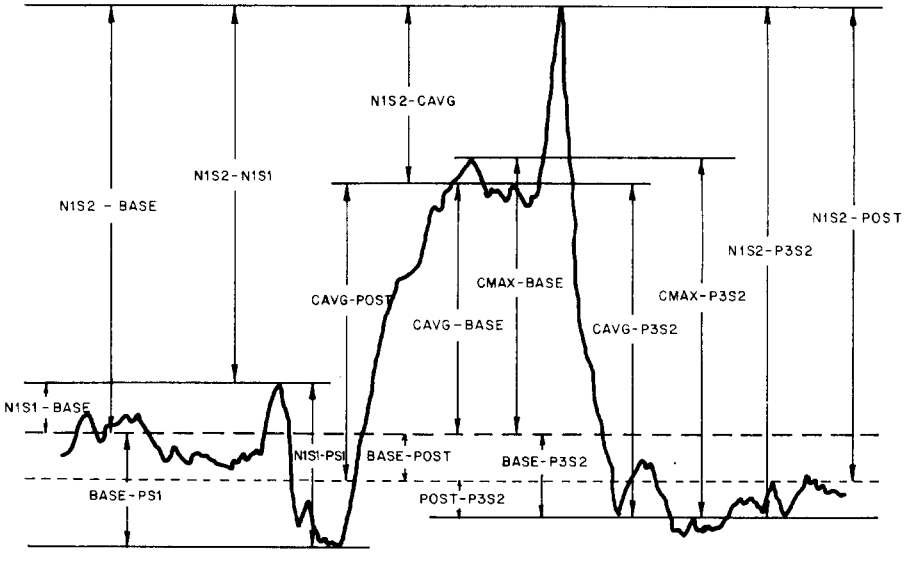


Fig. 2. This figure illustrates the 16 measures obtained from each evoked potential for each of the experimental conditions for each of the subjects. For each record, 8 levels indicated by the horizontal lines were defined and the 16 measures indicated by the arrows were taken. The levels and the amplitude measurements were obtained by a FORTRAN program operating on the digitized data. The figure illustrates the nature of the results obtained by this program.

of the original measures, and the weights would be selected in a manner that would make each composite measure orthogonal to the others. Such a set of measurements is, of course, provided by Principal Component Analysis (Tatsuoka 1971). It should be noted that the components derived in this manner are not "arbitrary". They are determined according to rigorously defined procedures. For an explanation of the use of Principal Component Analysis with evoked potentials see John *et al.* (1964, 1973), Donchin (1966, 1969), Suter (1970) and Chapman (1973).

*Principal component analysis*

We obtained the principal components of a matrix consisting of the 16 raw scores for each of the 8 subjects for each of the 8 experimental conditions (using the SOUPAC statistical package, Dickman 1972). Six principal components accounted for 92% of the variance of this matrix. A Varimax rotation was then performed on the 6 principal components. Loadings for each of the 16 raw variables are shown in Table I. Clearly the first factor is heavily loaded on the variables which involve the P300 component elicited by the tones. The second factor is clearly associated with the CNV, the third with the N1S2 while the fourth factor represents measures associated with the baseline and the S1 evoked potential, the fifth and sixth factors are not as easily identified with EP components, they account, however, for a small percent of the variance. It is, perhaps, necessary to emphasize that the orthogonality of the CNV and P300 factors is not an artifact of the manner in which the original raw measurements were taken. There was no *a priori* reason for the P300 measures and the CNV measures to segregate themselves as they did.

We have thus obtained composite measures of the AEP which are related to the CNV and to P300. These measures being by definition, and derivation, orthogonal can now be subjected, separately, to an analysis of variance so that we could assess for each of these measures, independently and separately, its relation to the three independent variables. Table II presents the results of this analysis. The values analyzed in this case are the so called "factor scores" associated with each of the factors for each of the 64 evoked potentials obtained from 8 subjects for 8 experimental conditions. Both the presence and absence of a warning stimulus and the nature of the task have significant effects on the CNV factor. On the other hand, the only independent

TABLE I

Factor loadings on each of 6 factors for each of 16 peak-to-base evoked potential measures.

| Waveform measure | Factor | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| N1S1–BASE | −0.07 | 0.32 | 0.12 | 0.13 | 0.13 | 0.92 |
| BASE–PS1 | 0.08 | −0.02 | −0.03 | 0.09 | −0.99 | 0.11 |
| N1S1–PS1 | −0.10 | 0.17 | 0.08 | −0.01 | 0.91 | 0.35 |
| CMAX–BASE | −0.05 | 0.96 | 0.18 | −0.02 | 0.09 | 0.17 |
| CAVG–BASE | −0.07 | 0.95 | 0.17 | −0.03 | 0.10 | 0.21 |
| N1S2–BASE | −0.01 | 0.47 | 0.84 | 0.11 | 0.07 | 0.25 |
| N1S2–N1S1 | 0.02 | 0.39 | 0.91 | 0.07 | 0.03 | −0.13 |
| N1S2–CAVG | 0.06 | −0.39 | 0.90 | 0.18 | 0.00 | 0.10 |
| P3S2–BASE | 0.88 | 0.40 | 0.15 | 0.19 | −0.03 | 0.10 |
| P3S2–CAVG | 0.90 | −0.35 | 0.02 | 0.21 | −0.10 | −0.07 |
| P3S2–CMAX | 0.89 | −0.38 | 0.01 | 0.20 | −0.10 | −0.04 |
| P3S2–N1S2 | 0.78 | −0.06 | −0.60 | 0.07 | −0.09 | −0.13 |
| POST–BASE | 0.15 | 0.29 | 0.21 | 0.90 | −0.03 | 0.21 |
| CAVG–POST | −0.19 | 0.59 | −0.02 | −0.77 | 0.11 | 0.02 |
| N1S2–POST | −0.13 | 0.26 | 0.72 | −0.61 | 0.11 | 0.10 |
| P3S2–POST | 0.83 | 0.19 | −0.01 | −0.53 | −0.01 | −0.06 |

TABLE II

Analysis of variance table for factor scores of "CNV" and "P300" factors for the 16 measures factor analysis (vertex data only).

| Source | *df* | Mean square | *F* ratio | Probability |
|---|---|---|---|---|
| *Factor 1 ("P300")* | | | | |
| Warning (NW *vs.* W) | 1/7 | 0.060 | 0.178 | 0.686 |
| Task (RT *vs.* PR) | 1/7 | 0.079 | 0.039 | 0.848 |
| Sequence (ALT *vs.* RAN) | 1/7 | 10.137 | 14.753 | 0.006 |
| Warning × Task | 1/7 | 0.003 | 0.023 | 0.883 |
| Warning × Sequence | 1/7 | 0.157 | 0.659 | 0.444 |
| Task × Sequence | 1/7 | 1.132 | 1.380 | 0.278 |
| Warning × Task × Sequence | 1/7 | 0.244 | 1.336 | 0.286 |
| *Factor 2 ("CNV")* | | | | |
| Warning | 1/7 | 24.776 | 60.651 | 0.000 |
| Task | 1/7 | 5.072 | 7.818 | 0.027 |
| Sequence | 1/7 | 0.615 | 5.796 | 0.047 |
| Warning × Task | 1/7 | 7.413 | 23.520 | 0.002 |
| Warning × Sequence | 1/7 | 0.511 | 6.771 | 0.035 |
| Task × Sequence | 1/7 | 0.105 | 0.427 | 0.534 |
| Warning × Task × Sequence | 1/7 | 0.077 | 0.364 | 0.565 |

variable that has a significant effect on the P300 factor is the predictability of the outcome stimuli. In other words the antecedent presence of a warning stimulus has no significant effect on the amplitude of the P300 factor.

The results described above relate to the recordings at the vertex electrode ($C_z$) only. We obtained in a similar manner the principal components of the raw measures associated with all electrode locations, for all subjects, for all experimental conditions. The results are strikingly similar to the results obtained with the vertex data alone. In fact the magnitude of the relationships between the raw variables and the axes, and the segregation of raw variables among the factors is identical. Table III presents an analysis of variance of the data obtained from all electrode locations using the factor scores for each of the variables derived from the vertex data. Note that, for the P300 factor, in addition to the predictability of the stimulus, significant effects can be observed for the electrode position as well as for the task. The CNV factor on the other hand is affected by the three main independent variables but not at all by electrode position. The reader will note that several of the third order interactions are significant. We do not have the space here to discuss these interactions, though it is worth noting that the interaction patterns of the P300 and CNV factors are quite different.

*Factor analysis on evoked response waveforms*

The above analysis was based on the, somewhat arbitrarily chosen, 16 measures of each AEP record. To check the validity of the factors discussed above we performed a factor-analysis on the actual evoked potential waveforms in the manner suggested by Donchin (1966) (see also John *et al.* 1973). All the AEPs were condensed to arrays of 50 points (50 msec per point). The principal components of the entire 50 by 64 matrix were obtained, and a Varimax rotation performed. In Fig. 3 we show the evoked potential waveform averaged over all 64 ERPs and the loadings on the 5 rotated principal axes. In Table IV we indicate the percentage of the variance accounted for by each factor as well as the independent variables which produced significant effects ($P < 0.01$). Again, distinct principal components appear to be associated with the P300 and the CNV. The P300 component is affected solely by the tone predictability. Inasmuch as these analyses tend to confirm the

TABLE III

Analysis of variance table for factor scores of "CNV" and "P300" factors for the 16 measures factor analysis (all electrode locations).

| Source | *df* | Mean square | *F* ratio | Probability |
|---|---|---|---|---|
| *Factor 1 ("P300")* | | | | |
| Warning (NW *vs.* W) | 1/7 | 0.482 | 0.406 | 0.545 |
| Task (RT *vs.* PR) | 1/7 | 10.251 | 1.152 | 0.319 |
| Sequence (ALT *vs.* RAN) | 1/7 | 71.493 | 18.926 | 0.003 |
| Electrode ($C_z$, $C_3$, $C_4$, $P_3$, $P_4$, $O_1$, $F_{p2}$) | 6/42 | 6.612 | 12.069 | 0.000 |
| Warning × Task | 1/7 | 0.986 | 0.734 | 0.420 |
| Warning × Sequence | 1/7 | 4.931 | 2.880 | 0.134 |
| Warning × Electrode | 6/42 | 0.587 | 8.937 | 0.000 |
| Task × Sequence | 1/7 | 2.602 | 0.823 | 0.395 |
| Task × Electrode | 6/42 | 0.282 | 0.862 | 0.531 |
| Sequence × Electrode | 6/42 | 0.341 | 3.121 | 0.013 |
| Warning × Task × Sequence | 1/7 | 0.453 | 0.614 | 0.459 |
| Warning × Task × Electrode | 6/42 | 0.167 | 5.853 | 0.000 |
| Warning × Sequence × Electrode | 6/42 | 0.216 | 5.658 | 0.000 |
| Task × Sequence × Electrode | 6/42 | 0.135 | 1.132 | 0.361 |
| Warning × Task × Sequence × Electrode | 6/42 | 0.091 | 1.818 | 0.119 |
| *Factor 2 ("CNV")* | | | | |
| Warning | 1/7 | 115.631 | 46.636 | 0.000 |
| Task | 1/7 | 17.411 | 4.969 | 0.061 |
| Sequence | 1/7 | 3.208 | 8.090 | 0.025 |
| Electrode | 6/42 | 0.392 | 1.067 | 0.397 |
| Warning × Task | 1/7 | 35.579 | 15.853 | 0.005 |
| Warning × Sequence | 1/7 | 4.878 | 4.438 | 0.073 |
| Warning × Electrode | 6/42 | 2.852 | 18.776 | 0.000 |
| Task × Sequence | 1/7 | 0.188 | 0.164 | 0.698 |
| Task × Electrode | 6/42 | 0.560 | 1.682 | 0.149 |
| Sequence × Electrode | 6/42 | 0.067 | 0.443 | 0.846 |
| Warning × Task × Sequence | 1/7 | 0.399 | 0.324 | 0.587 |
| Warning × Task × Electrode | 6/42 | 0.821 | 7.948 | 0.000 |
| Warning × Sequence × Electrode | 6/42 | 0.113 | 1.455 | 0.217 |
| Task × Sequence × Electrode | 6/42 | 0.062 | 0.374 | 0.891 |
| Warning × Task × Sequence × Electrode | 6/42 | 0.086 | 1.221 | 0.315 |

TABLE IV

Results of ANOVAS (at $P \leqslant 0.05$) of factor scores based on analysis of entire waveforms.

| Factor | Percent variance | Independent variables producing significant effect |
|---|---|---|
| 1 | 32.5 | Warning (NW *vs.* W), Task (RT *vs.* PR) |
| 2 | 27.4 | None |
| 3 | 13.4 | Warning (NW *vs.* W) |
| 4 | 12.1 | None |
| 5 | 9.5 | Sequence (ALT *vs.* RAN) |

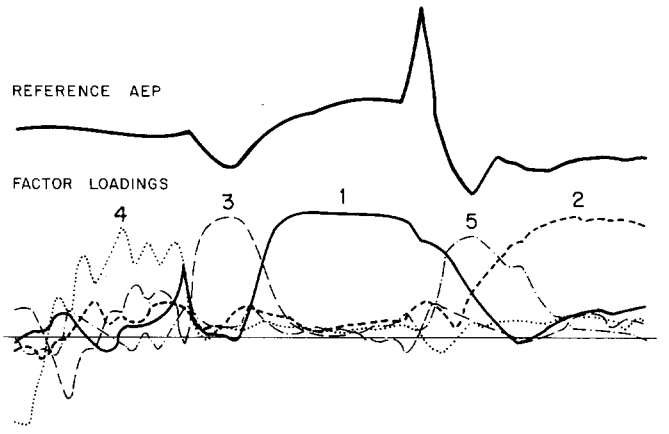


Fig. 3. Factor loadings for each of the 5 factors extracted from a factor analysis of the evoked potential. The "average waveform" was obtained by averaging over all experimental conditions and all subjects and serves primarily to identify specific time regions over the time base. It will be noted that factor 1 is associated with the CNV, factor 2 with the post-S2 baseline, factor 3 with the S1 evoked potential, factor 4 with a pre-S1 baseline and factor 5 with the P300 of the S2 evoked potential.

analyses reported above we shall not discuss them in detail.

*Discriminant analysis on the raw measures*

The results of the above analyses were further corroborated by analyzing the same data using still a different approach. The data could be dichotomized in 3 different ways; ERPs could be pooled according to stimulus predictability, subject task and warning stimulus presence. We used stepwise discriminant analysis to determine which combination of the 16 raw variables best discriminates between the groups created by each of these dichotomies. Each of these comparisons was analyzed by the stepwise discriminant analysis program (Dixon 1970; Donchin and Herning 1975), using two groups of 28 "observations" per case, 16 variables per observation. When the groups are segregated by the predictability of the trial's outcome (*i.e.*, by the random *vs.* alternate variable) the stepwise discriminant analysis program selects only one variable, (the N1–P3 amplitude for the S2 evoked potential). Interestingly, no additional measure of either P300 or the CNV can improve the discrimination between the random and alternate evoked potentials. When the data are segregated according to the presence or absence of the warning stimulus, the discrimination is formed on the basis of four measures: three reflecting a contribution by the S1 AEP, and one representing the CNV. None of the P300 related measures is utilized in discriminating between the warned and and unwarned AEPs. It appears then that when we evaluate the between-group variances associated with our different independent variables (discriminant analysis) the results corroborate the analyses which were based on the within-group variance (factor analysis). Either way we find that the presence or absence of a warning stimulus, and by implication the presence or absence of the CNV prior to the eliciting stimulus, has virtually no effect on the P300 component elicited by that stimulus.

The above discussion focused only on the degree to which statistically significant differences appear among the potentials associated with different experimental variables. We have said nothing about the direction of the observed significant differences. In Fig. 4 we plot the vertex factor scores for the CNV and P300 factors. We find that during the random condition we elicit a larger P300 than during the alternate condition. Furthermore, we find that there is a tendency for P300 effects to be larger during the predict than during the reaction time conditions. The CNV on the other hand seems rather unrelated to the predictability of the tones. A large CNV is elicited in the presence of a warning stimulus, no CNV in its absence.

Of interest is the relationship between the experimental variables and the scalp distribution of the CNV and of P300. In Fig. 5 we plot the CNV and P300 factor scores, computed for

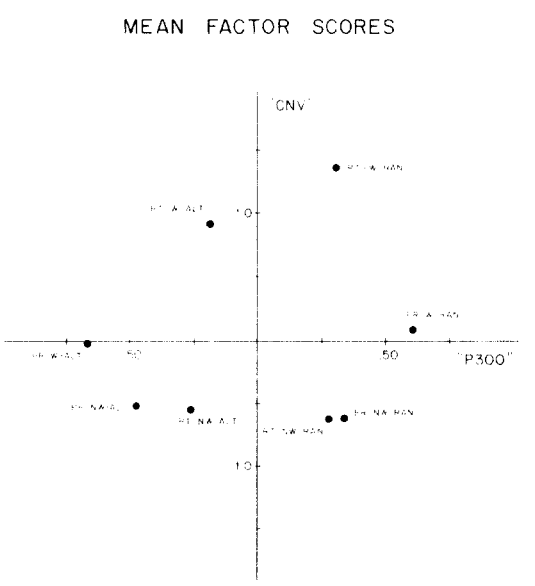


Fig. 4. The factor scores for the CNV and P300 factors for the vertex averaged evoked potential are plotted. Each point is identified by the values of the three major independent variables.

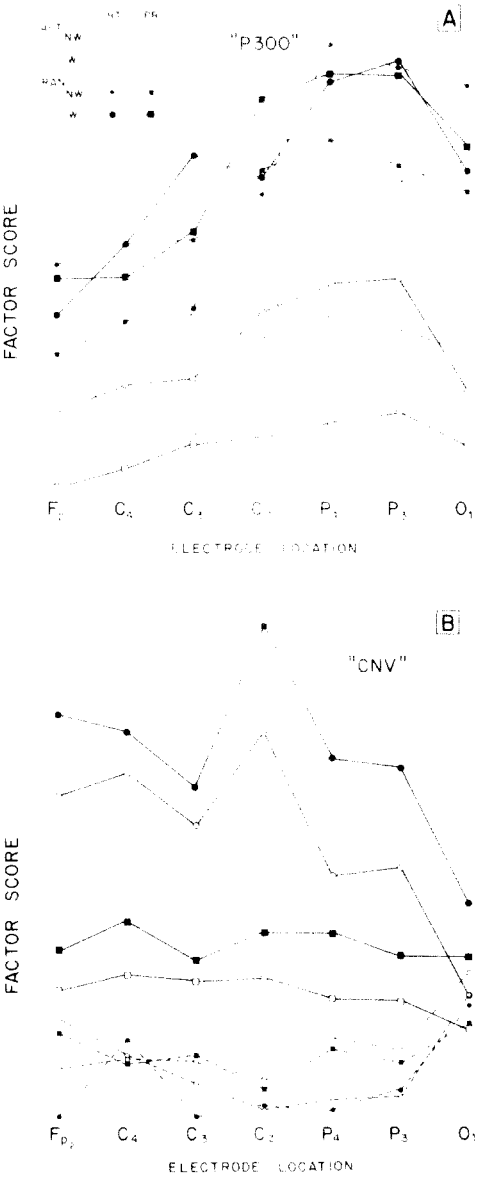


Fig. 5. Factor scores for the P300 and CNV factors for each of the 8 experimental conditions plotted as a function of electrode location.

grand averages over all subjects, for each of the 7 electrode locations. Each of the lines in the figure thus represents the scalp distribution of either the CNV or of P300 for one of our 8 experimental conditions. It is evident that the two factors are distributed quite differently across the head. The eight P300 distributions are quite similar in shape, though they are quite different in amplitude. For all 8 conditions, P300 has the largest amplitude at the parietal electrodes (as reported previously by Vaughan and Ritter 1970).

While the distribution of P300 suggests that our experimental manipulation modulated the amplitude of a uniform component there are clear indications that it is the distribution as well as the amplitude of the CNV that is modulated by the same manipulations. As expected, when no warning stimulus is used, the amplitude of the CNV is uniformly small across the scalp. Presenting the warning stimulus during the predict conditions elicits a uniform increase in the negativity during the foreperiod for the different electrodes. The introduction of a response requirement, coupled with the demand for high speed responding, yields a CNV distribution which is *not* uniform across the electrodes, the vertex clearly yielding the largest amplitudes. These differences in the distributions of the CNV and the P300 component lend further support to the suggestion that the two are quite independent.

*Data collected with the 800 msec foreperiod*

Four subjects were tested using a foreperiod of 800 msec. The 8 evoked potentials averaged

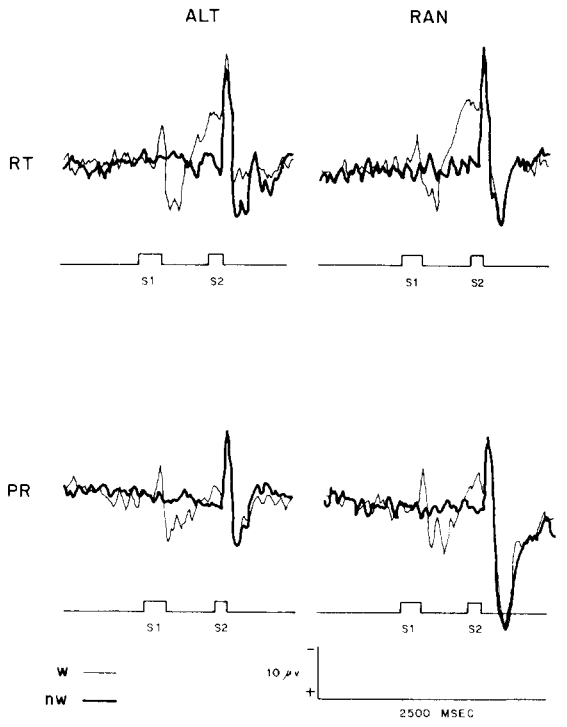


Fig. 6. Average evoked potential (vertex), averaged over subjects for each of the 8 experimental conditions obtained from the 4 subjects in which the S1–S2 interval was 800 msec. Note similarity of basic data patterns to those shown in Fig. 1.

over these 4 subjects are shown in Fig. 6. These data are consistent with the data obtained with the 1000 msec foreperiod, but for the expected greater steepness of the CNV slope. In all other respects the data from these subjects are much like those obtained from the other 8 subjects. We have subjected the data of these 4 subjects to similar analyses and obtained the same results.

## DISCUSSION

We report a factorial study of the effect of different experimental variables on the CNV and the P300 component. The data are consistent with the hypothesis that the P300 component is an endogenous cortical component invoked by the information processing requirements of the subject's task rather than by pre-stimulus, diffuse, generalized preparatory activity. These data confirm several previous studies but a direct study of joint and simultaneous interactions of P300 and the CNV has not been reported.

It might be noted that our data also confirm the report that the general effect of unpredictability on the amplitude of the P300 component is diminished when a requirement for a speedy response is introduced into the subject's task. As reported previously by Donchin *et al.* (1973), the difference at the vertex between evoked potential elicited by predictable and unpredictable stimuli is smaller during the reaction time than during the predict task (see also Tueting and Sutton 1973).

With respect to the relationship between prestimulus preparation and poststimulus P300 there remains one possibility to which our experiment has not addressed itself. It is possible to argue that a CNV is present during the unwarned condition, but that we cannot observe it because it is continuously "resident" during long intervals (Wilkinson, in press; also Donchin, in press). This resident CNV is presumably generated when the subject must maintain his preparation over a long period. If there is such a resident CNV then the S2 in the unwarned condition may be presented on the background of functional negativity equivalent to that observed in the typical CNV experiment. In such a view the warned *vs.* unwarned distinction does not truly distinguish between CNV presence or absence.

Presumably one could detect the presence or absence of such a resident CNV by recording the EEG using DC amplifiers and searching for generalized shifts in the spectrum of the EEG as we move from the warned to the unwarned conditions and back. However, because of its presumed long time constant and imprecise time locking a resident CNV would be technically difficult to record and average. At any rate we have not done this and in this sense we have perhaps not fully resolved the CNV/P300 issue. Yet, we feel that until the presence of a resident CNV is demonstrated our data are relevant to the issue on hand.

There remains one puzzle. Our data suggest that the P300 component is independent of the CNV. The amplitude of P300 was determined neither by the amplitude of the preceding CNV nor, indeed, by the presence or absence of a

preceding warning stimulus. It is the case, however, that a positive-going component ordinarily terminates the CNV and it seems our data need to be interpreted as indicating that this component and the P300 are independently elicited. If so, why do they not summate to produce a far larger positivity after the imperative stimulus when it is preceded by CNV than when it is not? If two independent generators act to produce a positive-going potential some 300 msec following the stimulus, one would have expected their effects to summate and to produce a larger positivity at the appropriate time region. This is obviously not the case, as our data show the antecedent CNV has no effect on the positivity following the stimulus. Where then is the downsweep of the CNV? Is there a ceiling effect? Do the two generators somehow cancel each other? Or are we perhaps, after all, observing an interaction between the CNV and P300 reflected by the absence of a difference rather than by its presence? These questions remain as topics for further research.

SUMMARY

We report an experiment designed to assess the interactions between the CNV and the P300 components of human event-related potential. Eight subjects were each presented with series of experimental trials on all of which either a 1200 c/sec or an 800 c/sec tone was presented. There were three independent variables: (a) The presence or absence of a warning flash 1000 msec prior to the tone. (b) The task assigned to the subject—that is subjects were either to make a discriminative response to the tone or, on half the series, to predict prior to the trial which of the two tones would be presented. (c) The predictability of the tone frequency. On half the series high and low tones alternated from trial to trial. On the other series, tones were chosen randomly on each trial.

The data show that the amplitude of the P300 component is not affected by the presence or absence of a warning stimulus. Furthermore, the distributions of P300 and the CNV over the scalp are quite different. These conclusions are supported by a principal component and a discriminant analysis of the data.

We conclude that the CNV and the P300 reflect the activity of functionally distinct cortical mechanisms.

RESUME

SUR L'INDEPENDANCE DE LA VCN ET DES COMPOSANTES P300 DU POTENTIEL EVOQUE HUMAIN MOYEN

Les auteurs rapportent une expérimentation destinée à mesurer les interactions entre la VCN et les composantes P300 du potentiel lié à un évènement chez l'homme. 8 sujets ont subi des séries de séquences expérimentales au cours desquelles un ton soit de 1200 c/sec, soit de 800 c/sec leur est présenté. Il y avait trois variables indépendantes. (a) La présence ou l'absence d'un éclair d'avertissement, 1000 msec avant le ton. (b) La tâche assignée au sujet, c'est-à-dire que les sujets devaient soit faire une réponse discriminative du ton, ou pour la moitié des séries prédire avant la séquence expérimentale lequel des deux tons serait présenté. (c) La prédictibilité de la fréquence du ton. Dans la moitié des séries expérimentales les tons hauts et bas alternaient d'une séquence à l'autre. Dans l'autre moitié, les tons étaient choisis au hasard pour chaque séquence.

Les données montrent que l'amplitude de la composante P300 n'est pas affectée par la présence ou l'absence d'un stimulus d'avertissement. De plus la distribution de l'onde P300 et de la VCN sur le scalp est tout à fait différente. Ces conclusions sont sous-tendues par une composante principale et par une analyse discriminante des données. Les auteurs concluent que la VCN et l'onde P300 reflètent l'activité de mécanismes corticaux fonctionnellement distincts.

The authors would like to thank the other members of the Bristol CNV/P300 task force: Merlin Donald, Steven Hillyard, Demetrios Papakostostopoulos, Michael Posner and Samuel Sutton, whose help was crucial in developing this experimental paradigm. Readers interested in the values of the 16 measures, and ANOVAs based on these raw measures can write to E. Donchin.

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