intensity at the animal's head was adjusted to an average power density of 1 mW/cm² as determined before irradiation by a broadband radiation monitor placed at the position occupied by the animal's head. Ambient chamber temperature was 23° ± 2°C. Doses of the drug without radiation were given throughout the radiation exposure series to assure that behavioral effects of the drug alone had not changed. A complete dose-effect function was redetermined after the microwave series.

Before the effects of radiation on performance were studied, the animals were adapted over several weeks to the sleeve holder by being placed in it daily for 30 minutes before the session until baseline performance with and without the constraint was the same. An animal accustomed to the sleeve holder showed the same behavioral changes on the FI schedule when given a drug dose whether or not it had been constrained in the holder for 30 minutes before the session.

The effects of chlordiazepoxide on the FI performance were modified in the presence of low-intensity microwave radiation. Changes in the rate of responding (total number of responses in a session divided by the duration of the session) on the FI reinforcement schedule as a function of drug dose are shown for both radiation and nonradiation conditions for the four subjects in Fig. 2. The drug-alone response rates are based on preradiation and postradiation sessions as well as drug-alone sessions during the radiation series. Increasing doses of chlordiazepoxide, in the absence of radiation, produced increases in FI response rates up to a maximum; further increases in dose decreased response rate. These changes in behavior on the FI schedule as a function of chlordiazepoxide are in accord with previous results (*4*). When the same doses of chlordiazepoxide were combined with 1 mW/cm² of microwave radiation, greater behavioral effects were obtained. The general shape of the dose-effect functions remained relatively constant under the microwave condition; however, the magnitude of the effect was enhanced. Behavior always returned to baseline values by the session after the drug-alone or drug-radiation session.

A number of cumulative-response records of drug and drug-radiation sessions for one animal are shown in Fig. 1. The cumulative records G through J show the rate-increasing effects obtained with increasing doses of chlordiazepoxide, as seen by the increasing number of responses within each FI 1 interval. Comparison of records A (radiation alone) and F shows that performance on the FI schedule following 30 minutes of exposure to microwaves alone differed little from baseline performance. [This may also be seen in Fig. 2 by comparing the baseline response rate (B) with microwave response rate (M).] Records B through E in Fig. 1 show the enhanced rate-increasing effects obtained with chlordiazepoxide combined with radiation. A comparison of the records for each dose alone and the same dose with radiation shows in what manner the microwave radiation increased the response rate.

The dose-response function for chlordiazepoxide was modified under low-intensity microwave radiation exposure in that the magnitude of the behavioral effect produced by the drug was potentiated in the presence of microwaves. This finding is in accord with studies measuring a variety of biological effects that indicate an interaction between drugs and microwaves at higher intensities or under continuous-wave conditions (*5*). The present results demonstrate that brief exposure to low-intensity pulsed microwave radiation can act synergistically with another agent in affecting the behavior of an organism, although the mechanism of interaction is not clear (*6*).

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6. In animals similar in weight and size to those used in the present study, we have monitored the rectal temperature during a 30-minute exposure at 5 mW/cm² with a special temperature probe, sensitive to changes of 0.2°C, constructed from a birefringent crystal insensitive to microwave radiation. The frequency, pulse duration, and pulse repetition rate were the same as in the present study. No temperature rise was observed. This result does not prove, but makes unlikely, that any measurable core heating occurred during 30-minute exposure to 1 mW/cm².
7. Supported by Naval Medical Research and Development Command, Work Unit No. ZF51.524.015.0042. The opinions and assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large. The experiments reported herein were conducted according to the principles set forth in *Guide for the Care and Use of Laboratory Animals* (Institute of Laboratory Resources, National Research Council, DHEW Publication No. NIH 74-23, 1977). We thank Hoffman-LaRoche, Inc., for the donation of chlordiazepoxide (Librium).

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A Brain Event Related to the Making of a Sensory Discrimination

Abstract. *Event-related potentials associated with detected targets in a vigilance task were analyzed in two ways: (i) by sorting the potentials in terms of sequential reaction time bins of 50 milliseconds and (ii) by examining the single trial waveforms. A negative component (N2) covaried in latency with reaction time. These results support the hypothesis that N2 reflects a decision process which controls behavioral responses in sensory discrimination tasks.*

The timing of stimulus evaluation related to discrimination between two or more stimuli is a central issue in human event-related potential (ERP) research and in cognitive psychology. At some time after task-related stimuli are processed, certain brain events related to a decision process controlling appropriate behavioral responses must occur. A long-latency, positive component of

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ERP's (referred to in the literature as P3, P300, and LPC) has been the focus of investigation for the reflection in scalp recordings of such decision processes. An essential characteristic of brain events reflecting such decision processes is that under appropriate experimental conditions they are correlated in time with behavioral responses. In a recent report, Kutas *et al.* reviewed the positive and negative experimental results pertinent to whether P3 latency is correlated with reaction time; they presented new evidence based on data from single trials, which supports the covariation in time between the timing of P3 and behavioral responses (*1*). For cognitive psychology, the identification of ERP's that reflect discrimination processes is relevant to theories concerning stages of information processing pivotal to selective attention. Hillyard and Picton (*2*), for example, have advanced the position that ERP's support the hypotheses of cognitive theorists, such as Broadbent and Triesman (*3*), that selective attention is composed of at least two or more stages of selection. Selective attention to a subset of stimuli (stimulus set) is hypothesized to be reflected in an enhancement of a negative ERP with a peak latency around 100 msec (Nl), and discrimination between stimuli within the attended subset (response set) hypothesized to be reflected by P3.

In an earlier experiment (*4*), we found significant correlations between P3 peak latency and reaction time (RT) in data on single trials. Since in some subjects the onset of P3 occurred on the average only a few milliseconds before the estimated initiation of motor responses in the motor cortex, we suggested that an earlier ERP with a latency of approximately 200 msec (N2) was associated with discrimination, but nevertheless concluded that the onset of P3 occurred early enough to be causally related to RT. Subsequently, it was realized that unless the distribution of delays between P3 onset and motor activities on single trials was surprisingly small, or very skewed, that a substantial proportion of the subjects' responses were initiated in the motor cortex before the onset of P3 (*5*). In that P3 appears to occur too late to account for discriminative motor responses, we embarked on a series of studies to elucidate the nature of N2.

In the experiment just discussed, we had been unable to observe N2 reliably. However, Klinke *et al.* (*6*) have shown that N2 can be clearly observed when it is elicited by randomly omitted stimuli embedded in a train of stimuli. Under such a condition is N2 most clearly observed because omitted stimuli do not elicit P2, which has a latency of approximately 200 msec and therefore often obscures N2. Accordingly, we used the missing stimulus paradigm in two sensory modalities to determine the topographic distribution of N2 across the scalp (*7*). We found that the scalp distribution of N2 was modality specific. The maximum amplitude for N2 associated with omitted flashes was in the preoccipital region, whereas the maximum amplitude of N2 associated with omitted tones was in the vicinity of the vertex. The scalp distribution of P3 was not specific to the modality with parietal maxima for both the omitted flashes and the omitted tones. A follow-up study (*8*) showed that when the ERP's to the frequent stimuli in a vigilance experiment were subtracted from the ERP's to the infrequent (target) stimuli, the resultant ERP waveform resembled that elicited by the randomly omitted stimuli of the previous investigation (*7*). Again, N2 was modality specific, with maxima at the preoccipital and vertex regions for target flashes and tones, respectively, as was obtained for omitted stimuli. The P3 component had comparable scalp distributions with parietal maxima in all conditions of the two experiments.

Taken together with the results of Klinke *et al.*, these studies established that (i) N2 is an endogenous ERP, as is P3 (*9*); (ii) N2 is associated with infrequent targets, as is P3, whether the targets are omitted stimuli or physically present stimuli, and (iii) N2 and P3 derive from different intracranial sources, which, in turn, implies they reflect different functional activities.

Since N2 has a shorter latency than P3, we concluded that N2 reflects detection of targets and that P3 reflects some other functional activity because it originates from distinctly different brain sites.

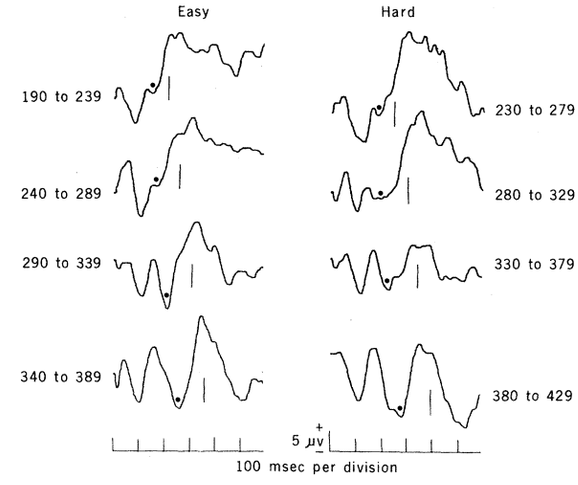

Fig. 1. Averaged ERP's recorded at the vertex for detected targets associated with sequential RT bins of 50 msec for subject J.K. in the easy and hard conditions. The number of trials included in each averaged waveform, from top to bottom, are 11, 19, 12, and 8 for the easy condition, and 7, 14, 17, and 15 for the hard condition. The vertical line beneath each waveform designates the median RT associated with that average. Large dots indicate N2. Traces begin with stimulus onset.

If the hypothesis for N2 is correct, its latency should vary as a function of the difficulty of the sensory discrimination and covary with the timing of RT within and across conditions. Furthermore, the inability to observe N2 in the averaged ERP's of the earlier experiment, in which the latency of P3 was correlated with RT (*4*), might be circumvented by averaging the ERP's according to selected RT bandwidths. Longer RT's should be associated with longer latencies of N2, and the P2 component, which has a constant latency of about 200 msec, should be less likely to obscure N2. We now present evidence that supports these hypotheses about N2.

The data of our earlier experiment (*4*), in which the latency of P3 was correlated with RT, were reanalyzed in two ways: (i) by averaging subsets of ERP's based on trials with RT's within selected ranges and (ii) by determining the latency of N2 on the single-trial data. The details of the experimental procedure are contained in the original report (*4*).

Four adult subjects participated in a vigilance task in which 60-dB, 50-msec tones were delivered through headphones at the rate of one every 2.5 seconds. Embedded within the train of stimuli were random pitch changes which occurred on the average of one in every ten tones. The subject's task was to detect the random pitch changes (the targets) and, upon doing so, to respond as quickly as possible by lifting the right index finger. There were two conditions, one in which the difference in pitch between the targets and nontargets was hard to discriminate, and another where the discrimination was easy. In both conditions the targets were 1000 Hz. In the easy condition, the nontargets were 2000 Hz; in the hard condition, the nontargets were either 1020 or 1030 Hz, the former used for the more experienced subjects.

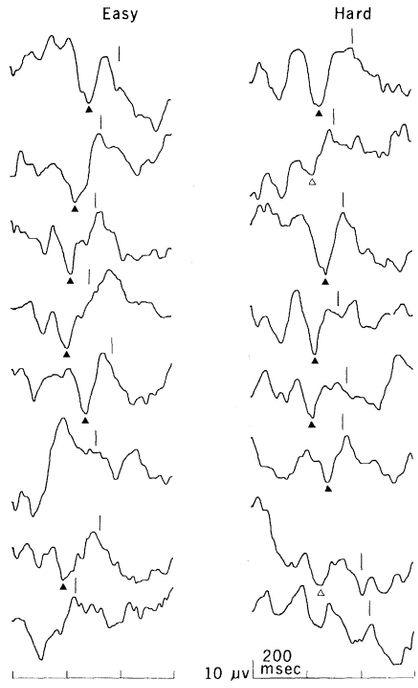

Fig. 2. Single-trial waveforms for subject J.K. for eight consecutive target detections taken from one run of the easy and one of the hard condition. Filled triangle, composite N2 latency of two scorers. Open triangle, N2 latency assigned by only one scorer. The absence of a triangle indicates neither scorer assigned a latency value for N2. The vertical lines indicate a response pulse. Traces begin with stimulus onset. Positive is up.

The ERP's were recorded from Cz, Pz, and Oz, with an electrode positioned above the left eye to monitor ocular potentials; all recordings were referred to the nose. Each run continued until 20 targets had been delivered. Three subjects had three runs of each condition, and one subject (S.V.) had four runs of each condition.

In the first analysis, the electroencephalogram (EEG) associated with the detected targets was averaged according to sequential RT bins of 50 msec. Averaging was calculated separately for each subject in both conditions. As RT increased, the latency of N2 and P3 lengthened in approximately commensurate amounts (Fig. 1). Furthermore, as N2 increased in latency, both the amplitude of P2 (about 150 msec in latency for this subject) and of N2 progressively grew in amplitude (*10*), suggesting that N2 and P2 overlapped in time for the ERP's associated with the trials on which the subject had shorter RT's, thereby canceling electrically at the scalp. Accordingly, gradual increases in N2 peak latency were associated with greater differences in the peak latencies of N2 and P2, resulting in increasingly less cancellation of the two components at the scalp (*11*).

A second analysis was conducted on the single-trial data in order to estimate the product-moment correlations between N2 and RT based on trial-to-trial covariations and to compare the results of this analysis with the single-trial analysis of P3 latency and RT of the earlier experiment (*4*). The raw EEG associated with each target was examined independently by two scorers in order to determine whether N2 could be observed and, if so, its peak latency at Cz estimated to the nearest 5 msec. Each scorer had access to all four leads, thereby taking the topography of N2 into account as well as contamination from ocular potentials. The RT data were withheld while the judgments were made of the presence and latency of N2 on the single-trial data, and we decided to restrict latency estimates between a range from 125 to 450 msec. Scoring was done blind with respect to conditions (easy versus hard). Only those trials in which the estimates of the two scorers were within 10 msec of each other were accepted for statistical analysis (*12*). As would be expected from Fig. 1, N2 was observed and scored less often for trials with short RT than for those with long RT, presumably because N2 was obscured by P2 when N2 had a shorter latency (*13*). Figure 2 presents single-trial waveforms of one subject from the easy and hard conditions.

Figure 3 presents (i) the mean peak latency for those trials which met the criteria for the four subjects in each condition and (ii) the best estimates of P3 and RT. The means of P3 were taken from latency measurements at Pz (where P3 was observed more frequently because it was largest in amplitude at that recording site) which met the criteria in the earlier experiment. Mean RT was based on all trials of a given condition (*14*). The increase in N2 mean latency from the easy to the hard condition was generally comparable to the increase in the means of P3 and RT. Whereas the mean differences between P3 and RT in the easy and hard conditions were statistically significant in the earlier experiment for all subjects, significant differences between the means of N2 in the easy and hard conditions were found for only three of the four subjects, presumably because of the smaller number of trials on which N2 could be observed (*15*).

Product-moment correlations were calculated with the single trial values for N2 and RT on the trials accepted for statistical analysis. Separate correlations were obtained for easy and hard conditions for each subject. With the exception of one nonsignificant correlation, the other seven significant correlations

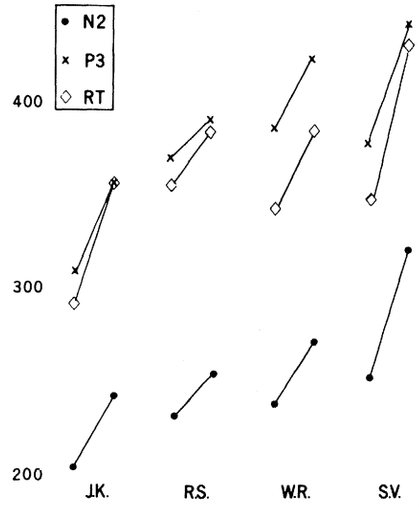

Fig. 3. Mean latencies of N2, P3, and RT for each subject in the easy (first point) and hard (second point) conditions.

ranged from .61 to .89 (*16*). In six out of eight instances, the correlation between N2 and RT at Cz was greater than that between P3 and RT at Pz (for a given subject and condition) found in the earlier paper (*4, 17*). These comparisons were not based on identical trials.

In order to estimate the covariation in latency between N2 and P3, correlations were calculated on those trials accepted for statistical analysis on which N2 latency was determined at Cz and P3 latency was determined at Pz in the previous paper (*4*). Although the *N*'s were small, the correlation coefficients ranged from .36 to .93, six of eight being statistically significant (*18, 19*).

The results of this report converge with the considerations of the relative timing of N2, P3, and RT, and of the distinctly different topographic distributions of N2 and P3; we conclude that N2 reflects a decision process related to sensory discrimination of attended stimuli. The correlation between the latency of N2 and RT supports our hypothesis (*7*) that the brain events underlying N2 are responsible for, and initiate in parallel, the neural activity related to relevant motor responses and the processes reflected by P3 (*20*). Such a hypothesis accounts for the correlations reported between the latency of P3 and RT (*1, 4*) and receives support from the correlations between the latencies of N2 and P3 obtained in this experiment (*18*). There need not be significant correlations between N2 or P3 and RT in all studies, however, as the correlation depends on the degree and kind of motor preparation in a given task, the subject's relative emphasis on speed or accuracy of response (*1*), whether subjects can ignore unex-

pected, irrelevant stimulus changes (*21*), and so forth. In this regard, P3 can be considered to index the relative timing of stimulus evaluation between experimental conditions (*1*, *21*), independent of RT. But our results suggest that P3 can be used to assess the temporal occurrence of stimulus evaluation because it is related in time to N2.

The data and theoretical implications indicate that many of the hypotheses concerning the functional significance of the brain activity P3 reflects (*22*), such as target selection (*2*), are more appropriately regarded with respect to N2 (*5*). More recent hypotheses (*23*) conceptualize P3 as reflecting brain activities concerned with future events, since P3 often occurs too late to be involved in the behavioral responses related to the eliciting stimulus. Understandably, P3 has received more attention from investigators than N2; it is larger in amplitude and therefore more readily observed and measured, whereas N2 is not only smaller but is also often obscured by P2 (Fig. 1). Whereas P3 can index the relative timing of stimulus evaluation between conditions, however, N2 can more directly measure the absolute timing of certain decision processes.

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8. ———, *ibid.* **42**, 528 (1977).
9. Endogenous ERP's are potentials that are more related to psychological states of the subject, in contrast to exogenous ERP's which are more related to stimulus parameters. The ERP components elicited by randomly omitted stimuli constitute the clearest examples of endogenous potentials.
10. H. Bostock and M. J. Jarvis, *Electroencephalogr. Clin. Neurophysiol.* **29**, 137 (1970).
11. The P2 latency was essentially constant, similar to the results of (*4*), in contrast to increases in latency of both P2 and N2 with longer RT reported by Bostock and Jarvis (*10*) and R. Parasuraman and D. R. Davies [*Percept. Psychophys.* **17**, 465 (1975)]. On the other hand, E. Donchin and D. B. Lindsley [*Electroencephalogr. Clin. Neurophysiol.* **20**, 217 (1966)], L. Morrell and F. Morrell [*ibid.* **20**, 567 (1966)], R. T. Wilkinson and H. C. Morlock [*ibid.* **23**, 50 (1967)], and D. Friedman, H. G. Vaughan, Jr., and L. Erlenmeyer-Kimling [*ibid.*, in press] found that P2 remains constant in latency despite changes in RT.
12. Of the trials accepted for statistical analysis, only one had an N2 latency greater than 400 msec and fewer than 10 percent had a latency between 300 and 400 msec.
13. For example, when combined across subjects and conditions, N2 was scored on 14 of 81 trials with RT's between 180 and 295 msec, whereas N2 was scored on 136 of 260 trials with RT's between 300 and 415 msec.
14. The P3 and RT means were taken from table 2 of (*4*).
15. For each subject in the two conditions, the mean and standard deviation of N2, N, and P values for two-tailed t-tests, respectively, were: J.K. (easy) 207 ± 45, 20; (hard) 245 ± 37, 12, $P < .05$. W.R. (easy) 239 ± 50, 20; (hard) 271 ± 51, 15, $P < .05$. R.S. (easy) 234 ± 20, 34; (hard) 254 ± 39, 32, $P < .10$. S.V. (easy) 252 ± 47, 22; (hard) 319 ± 77, 14, $P < .001$.
16. The correlation coefficients and their two-tailed P values were: J.K. (easy) .88, $P < .001$; (hard) .61, $P < .05$. W.R. (easy) .89, $P < .001$; (hard) .74, $P < .001$. R.S. (easy) .30, $P > .05$; (hard) .64, $P < .001$. S.V. (easy) .74, $P < .001$; (hard) .85, $P < .001$.
17. See table 3 of (*4*) for the correlations between P3 latency and RT on single trials.
18. For each subject and condition, the correlation coefficient, N, and two-tailed P values, respectively, were: J.K. (easy) .69, 14, $P < .01$; (hard) .53, 7, $P > .05$. W.R. (easy) .63, 18, $P < .01$; (hard) .93, 9, $P < .01$. R.S. (easy) .36, 24, $P > .05$; (hard) .63, 27, $P < .01$. S.V. (easy) .52, 18, $P < .05$; (hard) .83, 12, $P < .01$.
19. There are several reasons for believing that the N2 studied in this report is not a negative wave associated with motor potentials (MP's) that precede motor responses [L. Gilden, H. G. Vaughan, Jr., L. D. Costa, *Electroencephalogr. Clin. Neurophysiol.* **20**, 433 (1966)]. (i) In our previous experiment (*4*), a control condition in which subjects performed the finger movement in a self-paced manner revealed no observable MP's; (ii) in the same experiment the EEG was averaged with respect to the pulse generated by each subject in each condition by the finger lift, performed in response to targets, which revealed no observable premovement MP's (the lack of observable MP's was presumably related to the use of a small finger movement deliberately chosen to minimize MP's); (iii) negative-going MP's preceding large, vigorous hand movements are usually small in amplitude at the vertex, generally about 2 to 4 μV, whereas the N2 for the longer RT's in Fig. 1 are much larger.
20. See C. C. Duncan-Johnson and E. Donchin [*Psychophysiology* **14**, 456 (1977)] for a compatible view.
21. N. K. Squires, E. Donchin, K. C. Squires, S. Grossberg, *J. Exp. Psychol.: Hum Percept. Perform.* **3**, 299 (1977).
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24. Supported by PHS grants HD 10804, MH 06723, and core grant HD 01799.

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Smooth Pursuit Eye Movements: Is Perceived Motion Necessary?

Abstract. *It has recently been shown that perceived motion, in the absence of any appropriate retinal motion, is a sufficient stimulus to generate smooth pursuit eye motions. This raises the question of whether perceived motion is necessary for pursuit. In three experiments we obtained a negative answer to this question: retinal motion always governed pursuit.*

When a moving object enters the visual field, it is frequently followed by the eyes with a slow smooth motion distinctly different from the rapid ballistic eye motions known as saccades, which serve to change the eyes' point of fixation. Rashbass (*1*) presented evidence supporting the traditional viewpoint that the stimulus for these slow eye motions is the movement of an image over the retina. Recently, however, investigators have begun to argue that it is not retinal motion but rather the perceived motion of a stimulus that is a condition for smooth pursuit (*2*). There is now evidence that the eye can engage in smooth pursuit when there is perceived motion of a target in the absence of any appropriate retinal motion. It has not thus far been demonstrated, however, that perceived motion is actually necessary for pursuit. Two relevant questions to be answered are (i) whether pursuit eye motions can be elicited when there is retinal motion but no perceived motion and (ii) whether the smooth pursuit response of the eye will be to the retinal or perceived motion of a target in a situation in which there is a conflict between the two. Answers to these questions should reveal the relative importance of perceived and retinal motion in driving the pursuit system.

We now report the results of three experiments which address these questions. In one of these the pursuit behavior of the eye was examined for a set of stimulus velocities which ranged from well below to well above our subjects' detection thresholds. In the second, the addition of a stationary frame of reference could be used to render visible the slowest of these velocities, and a moving frame of reference could be used to induce the perception of motion in a stimulus which was in fact stationary. In the third experiment, eye movements were examined under conditions in which the phenomenon of induced motion was used to create a conflict between the direction of a target's retinal motion and the perceived direction of its motion.

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