pose this activated state as an animal model of mania (21). Rubidium clearly potentiated the model excitement, and this effect was consistent with that of known antidepressants; lithium had the opposite effect. These results suggest that rubidium merits clinical evaluation as an antidepressant agent in man.

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## **Evoked Potential Correlates of Auditory Signal Detection**

Abstract. A long-latency component of the averaged evoked potential recorded from the human scalp varied in close relationship with subjects' perceptual reports in an auditory signal detection task. Detected signals evoked potentials several times larger than did undetected signals, falsely reported signals, or correctly reported nonsignals. The threshold signal intensity at which detection performance exceeded chance levels was identical with concurrently obtained electrophysiological measures of threshold.

The properties of averaged evoked potentials (AEP's) recorded from the human scalp represent important information about the cerebral processes that underlie perception. Reliable changes in certain components of the AEP are associated with numerous types of perceptual and psychophysical judgments (1) and with the detection, discrimination, and interpretation of sensory input (2-5). One vital test of the validity of the AEP as a measure of perceptual processes in man is its relationship to the detection of stimuli near the threshold of sensation. Although many investigators have reported that AEP indices of signal detectability are nearly as sensitive as the observer's perceptual reports (6), two recent studies reported wide discrepancies between psychophysical and AEP measures of absolute sensory threshold (7, 8). As a result one group was led to conclude that "evoked activity . . . may play no essential or important role in determining perceptual reactions" (8).

Such conclusions were challenged by Donchin and Sutton (9), who enumerated serious methodological problems with the design and interpretation of AEP studies of threshold in general. First, in order to obtain a meaningful correlation between electrophysiological and perceptual responses, they must be obtained concurrently during the actual psychophysical task, since the AEP is highly sensitive to changes in the behavioral or attentive state of the observer (10). This is particularly true in the case of the long-latency, positive "P300" component of the AEP (11), which is reportedly enhanced during the detection of faint signals in any modality (3, 5, 12, 13). Second, prior studies of threshold were criticized (9) for failing to take into account the effect of observers' response biases and the type of psychophysical procedures used for threshold determinations. Psychophysical reports of threshold signals are codetermined by two factors: the observer's decision criterion of how intense a sensation must be in order to be reported as a signal, and the sensitivity of his perceptual system in distinguishing signals from background noise. If variations in the criterion factor are uncontrolled, artificial dissociations between perceptual and AEP measures of threshold may result (14). In the study reported here, we demonstrate that, when these critical methodological necessities are observed, the P300 component of the AEP does in fact bear close correspondence to behavioral measures of the perception of nearthreshold stimuli.

We used a signal detection procedure in which the observer's task was to decide on each trial whether or not an acoustic signal at threshold level (presented on a probabilistic basis) had been added to the continuous background noise. The AEP's were computer-averaged separately for each of the four possible combinations of signal conditions and observer's responses: detected signals (hits), failures to detect signals (misses), incorrect reports of signal presence (false alarms), and correct reports of signal absence (correct rejections). Separate computer averaging permitted an assessment of whether perceptually distinctive events were associated with different AEP wave forms. Furthermore, this method enabled us to evaluate directly the relation between the AEP and a measure of perceptual sensitivity (d') that is independent of possible variations in criterion and is closely tied to the extrac-

Table 1. Area measure of P300 magnitude (microvolts  $\times$  milliseconds) on different types of trials in auditory detection task when cue light was present in the signal interwhen val. Each tabular value is averaged over eight to ten blocks of trials and across the three observers. The probability values given in the footnotes indicate the statistical significance of difference in P300 magnitude from hit trials (two-tailed t-tests); F.A., false alarms; C.R., correct rejections.

Type of trial	Performance level: correct detections (%)		
	50-69	7089	90 +
Hit	470	639	703
Miss	269*	427*	
F.A.	250*	313†	
C.R.	281*	308†	300†

\* P < .005. P < .001. tion and utilization of sensory information (15).

During testing, the observers sat in a well-lighted, soundproof chamber and wore a headset of earphones. They kept their gaze fixated upon a small neon bulb. Each trial began with a 200-msec flash of this bulb (warning light), followed after 500 msec by a signal (1000hz tone pip of 50-msec duration) presented with a probability of .50. One second after the signal offset, another light directed the observer to report whether or not he thought a signal had been presented by pressing one of two buttons. The trial terminated with a feedback light that indicated the correctness of his report; feedback was given in order to stabilize detection performance more rapidly. Intertrial intervals varied between 2 and 4 seconds at random. The signal was kept at a constant intensity (against a continuous white noise of 60-db sound pressure level) throughout a block of 100 trials (16). On successive blocks of trials, signal intensities were varied in random order and ranged (in increments of 2 db) from those producing chance performance (50 percent correct) to signals that were 100 percent detectable. The appropriate signal levels for each of the three observers (college students) were determined on the basis of extensive training sessions prior to the recording of AEP's.

Scalp electrical activity was recorded from vertex referred to right mastoid with Ag-AgCl electrodes (17) and was amplified with a Grass P16 amplifier having a band-pass flat between 0 and 200 hz. An additional electrode pair monitored horizontal and vertical eye movements to control for electrocular artifacts in the vertex recording (18).

Fig. 1. Computer-averaged scalp activity before and after the signal observation interval on each of the four types of trials: hits, misses, false a'arms (F.A.), and correct rejections (C.R.). Records are for three different observers. These overall AEP's were taken from six to seven blocks of trials on which the percentage of correct responses (hits plus correct rejections) was within  $\pm 4$  percent of the indicated mean value. A small range of signal intensities is included within each overall AEP. The number before each trace indicates how many trials were included in the AEP. The downward deflection that follows the signal on hit trials is the P300 wave, which peaks between 300 and 350 msec after the signal. Polarity convention: vertex positivity downward.

Each trial was divided into two epochs for computer averaging of electrical activity; the first began 50 msec before the warning flash and lasted until the onset of the signal interval, and the second started with the onset of the signal interval and lasted for 500 msec. Stimulus presentations, response scoring, and AEP averaging were controlled automatically by a PDP-9 computer. In all, AEP's were obtained from each observer for at least four to eight



blocks of 100 trials at each signal intensity, for a total of 60 to 80 blocks administered over several weeks.

Figure 1 shows typical AEP's from each observer, averaged over 600 to 700 trials on which detection performance was at or near the indicated levels. The small negative baseline shift in the first epoch, the "contingent negative variation" (CNV), was reliably larger prior to the correct responses to low intensity signals, a finding consistent with previous reports (5, 19). In the epoch that followed the signal, the AEP had a large P300 component only when the signal was delivered and correctly detected (hits). On the remaining three types of trials, the P300 wave was either nonexistent or greatly attenuated.

The relationship of P300 magnitude on hit trials to the level of detection performance over a range of signal intensities is shown in Fig. 2. An area measure of P300 (20) increased monotonically as a function of both the percentage of correct detections and the criterion-free measure d', as performance grew from chance level up to about 90 percent correct (21). At the highest performance levels, there was a significant decline in P300 area (22).

In a further series of observations AEP's were recorded from all three observers while they were sitting passively with no requirement either to listen to or respond to the signals. There was no evidence of an enlarged P300 area under passive conditions when the signal intensities used were identical to those that evoked a substantial P300 wave on hit trials. In Fig. 3, AEP's taken under active (detecting) and passive conditions are compared for observer S.S. In fact, in none of the observers was an AEP discernible in the passive state at signal levels below those that produced about 80 percent correct performance on active trials. This demonstrates a dissociation between the passively recorded AEP and threshold detection performance. The P300 wave on hits was thus some 6 to 8 db more sensitive than any component in the passive vertex AEP typically used for clinical audiometry (23).

Finally, to ensure that the diminished P300 wave on trials other than hits was not merely a side effect of unstable time locking of the sensory decision to the computer-averaging epoch (24), we repeated the entire detection experiment in all observers with the addition of a 50-msec "cue light" flashed concurrently with the signal observation

SCIENCE, VOL. 172

interval on all trials. As shown in Table 1, the P300 wave was still substantially larger on hit trials than on any other type of trial. In fact, at low performance levels, the cue light considerably enhanced the differences in the P300 wave between hit trials and the others. The small but consistent positive deflections in the AEP's on trials other than hits (Fig. 2 and Table 1) may be attributed to the slow return to baseline of the negative CNV that developed in the period before the signal.

These findings demonstrate the critical importance of separating AEP's on the basis of both stimulus conditions and perceptual responses. The P300 wave was enlarged only when stimulus information was being actively processed and was uniquely associated with the occurrence of a signal and its correct detection. Moreover, the P300 component did not develop until actual signal information began to be utilized as a basis for judgments (that is, until d'exceeded zero). These results, plus the strong dependence of P300 magnitude upon hit rate, suggest that the P300 wave is closely linked to cerebral events that underlie the sensory analysis and subsequent correct decision of signal occurrence. It does not simply reflect a general shift of attention arousal, or activation that is independent of sensory processing.

These results bear upon certain theoretical interpretations of the P300 wave. Ritter and Vaughn (4) have suggested that the P300 component represents a "cognitive evaluation of stimulus significance" and appears whenever a difficult decision is made. This proposal does not, however, account for our findings that P300 amplitude depends on the specific stimulus-response outcome and that it declines as performance nears chance levels.

A second hypothesis, that the P300 wave represents a "reactive change of state" which follows task completion (for instance, from "readiness, vigilance or arousal" to relaxation) (25), also seems inconsistent with the absence of the P300 wave on trials other than hits (especially correct rejection trials when d' was high). Moreover, the P300 wave does not appear to be simply a "reactive" return to baseline of a preceding CNV, since the amplitude of CNV was nearly always several times smaller on hit trials than was the amplitude of the P300 component (Fig. 1). Although the P300 wave may often be superimposed upon and confounded with the positive terminal phase of the CNV, our results indicate that the CNV and the P300 wave are separate phenomena (26).

Although no theory yet proposed gives an adequate account of the behavior of the P300 wave in all situations, the hypothesis most compatible with our results states that the P300 wave indexes the observer's degree of confidence or certainty in his decision that a signal has occurred (12). This would explain why the P300 wave was absent on false alarm trials, since, in contrast to the hit trials, those judgments were based on the average on lower signal-to-noise ratios and hence should be made with less certainty. The growth of the P300 wave over the range of 50 to 90 percent of correct responses may also be attributed to the increasing certainty of hearing the more easily detectable signals. Finally, our work now in progress shows that on hit trials the P300 wave increases monotonically with the observer's numerical rating of his confidence in his report. This indicates that the P300 wave grows not only as signals become more detectable but also as a higher criterion is adopted for the hit decisions, thereby increasing the subjective (and objective) certainty of those decisions (27).

The decline of the P300 wave on hit



Fig. 2. Dependency of P300 magnitude on hit trials upon signal detectability, measured in terms of both d' and the percentage of correct responses. The ordinate gives an area measure of P300 magnitude, the net positive area within the interval of 260 to 340 msec after the signal, relative to the baseline voltage measured during the 50-msec signal. Each point is based on AEP's averaged over four to eight blocks from each observer, when detection performance was within  $\pm$  5 percent of the plotted value; *F.A.*, false alarms; *C.R.*, correct rejections.



Fig. 3. Comparison of AEP's on active hit and miss trials with those recorded from the same observer (S.S.) in the passive condition, at identical signal intensities. These AEP's were averaged on the basis of signal level (in decibels) rather than on the observer's performance level, as in Fig. 1. The percentage of correct responses at the left are means obtained from all active trials in which the signal level indicated to the right was used. The number of trials in each AEP were: hits, 200 to 400; misses, 50 to 200; passive, 200. The AEP's on misses are not shown for the higher signal levels because of an insufficient number of trials.

trials for signals beyond 90 percent correct (Fig. 2) indicates, however, that the certainty of the decision per se cannot be the only factor governing the P300 component. It has also been shown that the a priori improbability of an event, and hence its unexpectedness or prior subjective uncertainty, is a determinant of P300 (28). If the effect of increasing the detectability of signals (d') is both to enhance P300 because the hit decisions are more confident and at the same time to reduce P300 because the a priori uncertainty of hearing a signal is less, its amplitude should in fact be at a maximum at some intermediate signal level, whereupon the "resolution of uncertainty" or "delivery of information" (24) would be at a maximum. Most puzzling, however, are the minimum P300 components on the correct rejection trials, on which decisions are confident, uncertainty is resolved, and information is delivered. A partial explanation might be that P300 activity is triggered by a definite match between an expected sensory event (which is not wholly predictable) and a neural "template" or "model" (29) of the stimulus. On the assumption that the observer's detection strategy is to match sensory input against a template for the signal, there would be no P300 wave for a correct rejection.

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- 14. For example, a very "conservative" (high criterion) observer would only report those signals of which he was absolutely certain; signals somewhat less intense than this high criterion may still evoke AEP's, however, which would result in an artificial raising of behavioral threshold in relation to electrical threshold. Donchin and Sutton (9) levied this criticism at the results of Libet *et al.* (7) and a converse argument to the results of Clark et al. (8). Clark et al. also failed to obtain behavioral and AEP measures concur-
- rently. The d' measure is derived from the signal 15. detection model that treats the threshold signals in noise as a statistical decisicn problem [see D. M. Green and J. A. Swets, Signal Detection Theory and Psychophysics (Wiley, New York, 1966)]. The model assumes that the sensory inflow upon which decisions are based varies in magnitude fr.m trial to trial according to a Gaussian distributhat to that according to a Gaussian distribu-tion. Addition of a signal to a background noise simply increases the mean of the hypothetical noise distribution by an amount d' (expressed in units of standard deviations of the distribution of noise alone). Thus d' (ranging from zero to infinity) indicates the amount of separation between noise and signal-plus-noise distributions; to the extent that these two distributions overlap, a higher proportion of incorrect responses results. In other words, d' is determined by the per-centage of hits in relation to the percentage d' is determined by the perof false alarms and is independent of the criterion level of sensory magnitude chosen by the observer.
- 16. Prior to each block, several "sample" signals were delivered. A rest peri minutes followed each block. period of 5 to 10
- 17. Nonpolarizable biopstential electrodes (Beckman Instruments).
- 18. Those trials in which eye movements were large enough to contribute artifact to the vertex-mastoid channel were rejected from the mputer analysis.
- The mean CNV amplitudes (measured over 75 msec prior to the signal) of all subjects, averaged across performance levels between 55 and 65 percent correct, were significantly 55 and 65 percent correct, were significantly larger on correct trials (hits, 4.1  $\mu$ v; correct rejections, 4.8  $\mu$ v) than on incorrect trials (misses, 2.0  $\mu$ v; false alarms, 2.4  $\mu$ v) [t = 2.51, d.f. = 10, P < .02 (one-tailed)]. For higher performance levels, CNV amplitude was not related to correctness of response. We at-tribute the low CNV amplitudes (1 to 6  $\mu$ v) and pcor correlations with behavior to the and poor correlations with behavior to the brevity of the intertrial and warning time intervals.
- The magnitude of P300 was taken as the 20. total area (in microvolts × milliseconds) contained in the positive deflection within the time interval between 260 and 340 msec after the signal onset, relative to the mean base-line voltage within the 50-msec signal interval. This area measure was chosen to reduce the variability inherent in determining a single peak on the broad P300 wave and to calculate

the magnitude of changes in duration and breadth of the P300 wave (as well as amplituce changes). However, the shapes of the curves on Fig. 2 were little changed by plotting them in terms of baseline to peak voltages

- ages. 21. The percentage of correct detections and d'may be plotted on equivalent axes in Fig. 2 because observers' criteria were relatively stable from one block to the next. When criteria are stable, the percentage of correct detections is a monotonic function of d', the form of which is implied by the scaling of upper and lower axes of Fig. 2. Quantitatively, criterion level may be expressed as a " $\beta$ alue," equal to the ratio of the probability value," densities of noise and signal-plus-noise distri-bution functions at the criterion point. For our observers, mean  $\beta$  values over all blocks of trials were:  $1.09 \pm 0.42$ ,  $1.11 \pm 0.33$ , and  $0.87 \pm 0.10$ . These standard deviations were small enough to preserve the monotonic relation between d' and the percentage of correct detections. The significance of the growth of P300 area on hit trials was assessed by linear-Provide a consistent of the trans was assessed by inhear-trend analyses of variance: over the range of 50 to 90 percent correct, F(1, 47) = 61.5, P < .001; over the range of 50 to 100 percent, F(1, 47) = 45.4, P < .001. On the other three types of trials, the P300 area did not relate considerative to performance ignificantly to performance.
- Significantly to performance. The significance of this decline in P300 area was assessed by a quadratic trend analysis of variance [F(1, 47) = 30.79, P < .001]. Part of this decline results from the use of the area measure (between 260 and 340 msec), since P300 latency was shortened at higher signal levels. However, the baseline to peak ampli-tu<sup>A</sup>e of the P300 wave was also reduced at the highest signal levels. 23. This conclusion is based on AEP's averaged
- over 200 to 400 signals. The N1 and P2 components of the vertex AEP (negative wave at 120 msec and positive wave at 200 msec) were also enhanced in the active condition relative to the passive condition, but the P300 component was more discernible from  $n{\sim}ise$ levels at low signal intensities and was more closely correlated with performance than these earlier vertex potentials
- 24. Our observers had over 50 hours of experience in the task and hence could estimate quite precisely the timing of the signal interval. This is far more experience than was received by subjects in previous studies in which large P300 waves were triggered by stimulus absence. See, for example, S. Sutton, P. Tueting, J. Zubin, E. R. John, Science 155, P. Iueing, J. Zubin, E. R. Joini, Science 155, 1436 (1967); R. Klinke, H. Fruhstorfer, P. Finkenzeller, *Electroencephalogr. Clin. Neuro-physiol.* 25, 119 (1968).
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## **Neuronal Thermosensitivity**

Barker and Carpenter, in reporting thermosensitive neurons in the cerebral cortex of cats (1), conclude that "thermosensitivity of a neuron does

not necessarily indicate a ro'e for that neuron . . . in thermoregulation, as has been implied in previous studies." The first part of this conclusion is mani-