dramatic changes in their contractile characteristics (*13, 14*). Furthermore, the optical axes are not fully aligned until after the second month of life (*14, 15*). Thus, the same neural output from the colliculus may produce large, coordinated movements between the two eyes in adults when the eyeballs and extraocular muscles are mature and smaller movements, which seem less coordinated, in the neonate. In addition, younger kittens will detoxify anesthetics more slowly than adults and some of these discrepancies might reflect the effects of the anesthetic (*16*). Consequently, the neural substrate for eye movements in the neonatal superior colliculus might be even more highly developed than could be demonstrated in these experiments.

The observation that eye movements can be elicited from the superior colliculus before cells in the colliculus can be activated by visual stimuli is consistent with the hypothesis advanced earlier: the development of eye-movement control systems precedes maturation of visual responses even in an area of the brain concerned with visuomotor integration. Since the motor responses seem to be necessary to "make sense" of the visual input (*1*), this developmental sequence seems appropriate. It is intriguing to consider what produces eye movements in these young animals in order to initiate the process of associating visual stimuli with motor responses, an association critical for normal visuomotor integration. This process may occur as the result of spontaneously generated eye movements or of eye movements initiated by vestibular stimulation (*17*). Another possibility is that tactile or auditory stimuli initiate eye movements via the superior colliculus. The different sensory modalities represented in the colliculus [visual, auditory, somatic, vestibular (*18*)] have been hypothesized to have access to the same motor outputs (*19*) that orient receptor organs (*11, 12*). Thus, stimuli from any of these sensory modalities may evoke organized eye, ear, or head movements. Since cells in the intermediate and deeper layers of the superior colliculus are responsive to tactile stimuli at birth and auditory stimuli at 5 days of age, these stimuli might initiate the eye movements, which, in turn, set the stage for later visual-motor associations.

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9. The testing session was variable in length. Despite the absence of painful wounds or pressure points, some kittens reacted to restraint by struggling or emitting stress vocalizations (more than a single meow). When such signs were exhibited, the experiment was abruptly ended. In some cases, only a single electrode penetration was made, in others two or more could be completed; young animals were studied for as little as 10 minutes and occasionally for well over 1 hour.
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26 February 1980; revised 4 June 1980

Brain Events Underlying Detection and Recognition of Weak Sensory Signals

Abstract. *Through the use of a quantitative extension of signal detection theory, the brain events associated with the detection and recognition of weak acoustic signals were examined by recording brain event-related potentials. The early N100 component of the event-related potential varied only with detection, whereas the late P300 component varied with both detection and recognition. P300 amplitude accurately predicted recognition performance on a trial-by-trial basis. The results suggest that detection and recognition are partially concurrent processes in perception and demonstrate that the electrocortical events occurring during the perception of sensory stimuli are closely associated with both detection and recognition of these stimuli by the nervous system.*

Human observers are often required to detect the presence of weak sensory signals in the environment. Examining an x-ray for a suspected tumor, listening to sonar returns for the presence of a ship, and noticing a hidden obstruction on the highway are examples of signal detection, a phenomenon that has been extensively analyzed from the perspective of signal detection theory, or SDT (*1*). However, detection is only one aspect of the normal perception of weak sensory signals; the other is recognition or identification. A driver at night will not only notice a weak visual signal but will attempt to identify it as a relevant environmental stimulus, such as a tree, a child, or another automobile. Similarly, the radiologist and the sonar operator must also recognize as well as detect their targets. The relationship between detection and recognition, however, and the brain events mediating this relationship are not well understood. Two important questions are whether detection and recognition are independent processes and whether recognition can be predicted from detection or from the brain events subserving these aspects of perception. A related point of interest is whether detection and recognition are sequential or concurrent temporal processes.

We approached this problem by examining the event-related potentials (ERP's) of the brain, which provide a unique method for assessing dynamic properties of human cerebral function during cognitive processing. In pure signal detection, the amplitudes of both an early negative component, N100, and a late positive component of the ERP, P300, increase monotonically with in-

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creases in the a posteriori likelihood ratio that a signal was presented (*2*). We now report what are, to our knowledge, the first studies of the brain events underlying detection and recognition of weak sensory signals. We used a recent extension of SDT that provides a quantitative theoretical means for interrelating the qualitatively disparate processes of detection and recognition (*3*). In two experiments of ERP's elicited by weak acoustic signals in noise, we show that whereas the N100 component varies only with detection, the P300 component varies with both detection and recognition; this result permits accurate prediction of recognition performance on a trial-by-trial basis and indicates that detection and recognition are partially concurrent processes in perception.

Ten paid subjects with normal hearing participated, six in experiment 1 and four in experiment 2. Subjects sat at a computer display and listened through binaural headphones (Superex) to pure tones presented against continuous wide-band noise (60 dB sound pressure level). Each trial began with a 100-msec exposure of the word "Ready" followed 500 msec later, on half the trials, by a 50-msec tone drawn equiprobably from a set of two (experiment 1) or four (experiment 2) targets. On the remaining trials noise alone was presented. At 1500 msec, a response cue directed subjects to use a four-category confidence rating scale (1, yes-sure; 2, yes-unsure; 3, no-unsure; and 4, no-sure) to indicate whether they had detected a target and to depress one of two or four recognition response keys to indicate target type. A recognition response was always required. Intertrial interval varied randomly from 2 to 6 seconds. The targets were 900- and 1400-Hz tones in experiment 1 and 600-, 1100-, 1700-, and 2200-Hz tones in experiment 2 (*4*, *5*). Each subject served in three to five 2-hour sessions. About 3 hours of practice preceded data collection. Each subject received between 1200 and 1300 trials.

The electroencephalogram (EEG) was recorded from Fz, C3, Cz, C4, and Pz (*6*), referred to linked earlobes, in experiment 1 and Fz, Cz, and Pz in experiment 2. The electrooculogram (EOG) was recorded from infra- and supraorbital electrodes placed about the left eye. The EEG and EOG were amplified (Grass model 8, 0.1 to 35 Hz) and digitized at 12 bits (PDP-11/34). A 1600-msec epoch beginning 100 msec before the warning stimulus and ending at the response cue was sampled at 80 Hz and stored on magnetic tape. A root-mean-square voltage criterion and a moving-window algorithm designed to detect sharp transients searched for ocular and muscle artifacts. Artifact-contaminated trials (5 to 15 percent) were replaced to give a minimum of 1000 artifact-free trials, which were analyzed in two stages. (i) Conventional ensemble averages were computed for target trials. (ERP's for noise trials are not reported here.) (ii) Single-trial ERP's were extracted to validate the ensemble averages and to obtain single-trial ERP measures (*7*).

Figure 1 shows the vertex ERP's for correctly and incorrectly recognized targets at each confidence rating. The P300 component was larger (peak amplitude relative to a 100-msec prestimulus baseline) for correct recognitions than for incorrect recognitions for ratings 1, 2, and 3 only [simple effects, experiment 1: $F(1, 5) > 19.1$, $P < .05$; experiment 2: $F(1, 3) > 34.1$, $P < .01$]. The amplitude of P300 decreased [experiment 1: $F(3, 5) = 59.4$, $P < .001$; experiment 2: $F(3, 3) = 39.8$, $P < .01$] and its latency increased [experiment 1: $F(3, 5) = 29.8$,

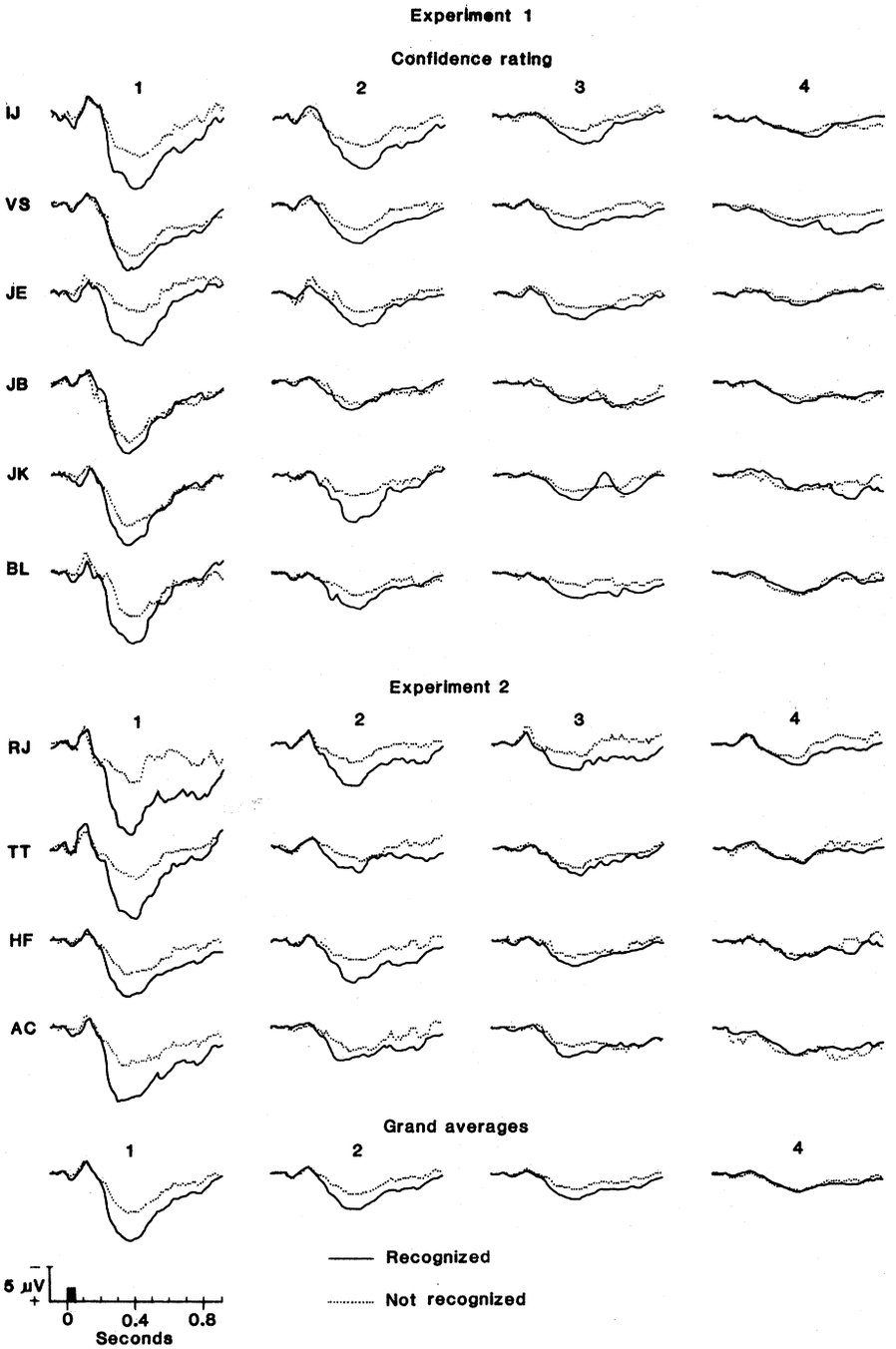

Fig. 1. Event-related potentials (ERP's) averaged separately for correctly and incorrectly recognized targets at each of the four ratings of detection confidence (1, yes-sure; 2, yes-unsure; 3, no-unsure; and 4, no-sure). The ERP's were recorded from Cz and are shown for each of the ten subjects in experiments 1 and 2; ERP's averaged across subjects are also shown.

$P < .01$; experiment 2: $F(3, 3) = 18.2$, $P < .025$] across ratings 1 to 4.

In contrast, only detection affected the early negative component N100; its amplitude decreased [experiment 1: $F(3, 5) = 14.3$, $P < .01$; experiment 2: $F(3, 3) = 5.8$, $P < .10$] and latency increased [experiment 1: $F(3, 5) = 23.8$, $P < .01$; experiment 2: $F(3, 3) = 20.1$, $P < .025$] across ratings 1 to 4. Thus, while detection was related to the amplitude and latency of both N100 and P300, recognition was related only to P300 amplitude.

An amplitude difference in an averaged ERP component can result from changes in its latency variability across trials. However, although both the mean latency [$F(3, 9) = 18.6$, $P < .01$, for both experiments] and the latency variability of P300 across single trials [$F(3, 9) = 61.7$, $P < .001$] increased across ratings 1 to 4, there were no differences in these measures between recognized and unrecognized targets. It was confirmed that the amplitude of P300 on single trials was greater for recognized than for unrecognized targets for ratings 1, 2, and 3 [simple effects, $F(1, 9) > 45.3$, $P < .001$] but not for rating 4 (*8*).

The ERP's in Fig. 1 were averaged without respect to target type. Figure 2 shows the grand average ERP's for each cell of the "confusion matrix" between target type and recognition response. Because of the small number of responses in some cells, ERP's were averaged to targets given either a confident or doubtful positive response (ratings 1 or 2). The ERP's show a striking similarity of waveshape and amplitude for cells along the diagonal (correct recognitions) with decreasing amplitude for off-diagonal cells (incorrect recognitions). There was a significant interaction between target type and recognition response for P300 amplitude [experiment 1: $F(1, 5) = 36.3$, $P < .005$; experiment 2: $F(9, 27) = 30.9$, $P < .001$], indicating that P300 amplitude was dependent on the relative distance between presented and perceived targets in the confusion matrix (*9*). Targets likely to be confused with each other showed a lower P300 amplitude difference than targets less likely to be confused with each other.

Table 1. Mean probabilities (based on 600 to 650 target trials per subject) of detection (D), recognition (R), and detection plus recognition (D + R) for each confidence rating. The chance level of correct recognition was .50 in experiment 1 (two targets) and .25 in experiment 2 (four targets).

Rating	Experiment 1			Experiment 2		
	D	R	D + R	D	R	D + R
1	.41	.87	.35	.46	.81	.37
2	.22	.74	.16	.19	.56	.11
3	.15	.62	.10	.13	.52	.07
4	.22	.42	.12	.22	.35	.08
All	1.00	.73	.73	1.00	.63	.63

Table 1 shows the mean response probabilities for each confidence rating. Infrequency of response outcome, which is associated with increased P300 amplitude (*10*), cannot account for the finding that P300 was larger for correct recognitions than for incorrect recognitions, since, at a particular confidence rating, a correct recognition outcome was more probable than an incorrect recognition for ratings 1 through 3. The probability of correct recognition was above the level of chance for target-absent rating 3 [for nine of ten subjects; $\chi^2(1) = 4.9$ (corrected for small sample), $P < .05$], and at chance level for target-absent rating 4 (Table 1). This suggests that recognition is only partially contingent on detection, and is consistent with the finding that P300 was larger for recognized targets than for unrecognized targets for rating 3 but not for rating 4. Thus, P300 amplitude indexed whether a target would be recognized reliably even if the target was not detected reliably.

Finally, two methods for predicting recognition were assessed. The first used a recent theorem by Starr *et al.* (*11*) to predict recognition probabilities from detection performance. The theorem assumes that targets are orthogonal and equally detectable. The predicted and obtained recognition probabilities at each rating were monotonically related, but the theorem overestimated observer performance by, on average, 5, 8, 10, and 15 percent for ratings 1 to 4, respectively. The second method utilized the amplitude of P300 (at Cz) on single trials to predict recognition on individual trials. A binary classification algorithm that maximized the correct classification of targets as correctly or incorrectly recognized on the basis of P300 amplitude gave mean classification percentages of, for ratings 1 to 4, 90, 77, 68, and 54 percent (recognized targets), and 87, 77, 64, and 55 percent (unrecognized targets).

Several aspects of these data deserve emphasis. (i) The quantitative SDT model provides a basis for investigating more complex aspects of perception when its domain is appropriately expanded and a single theoretical framework for analyzing two qualitatively different aspects of perception, detection and recognition. (ii) Inspection of the ERP confusion matrix shows that P300 amplitude varies as a decreasing function of the physical similarity between presented and perceived stimuli. It may be possible to use correspondences between physical, perceptual, and neurophysiological dimensions to develop ERP measures of classificatory dimensional scaling for complex auditory stimuli. (iii) The brain events

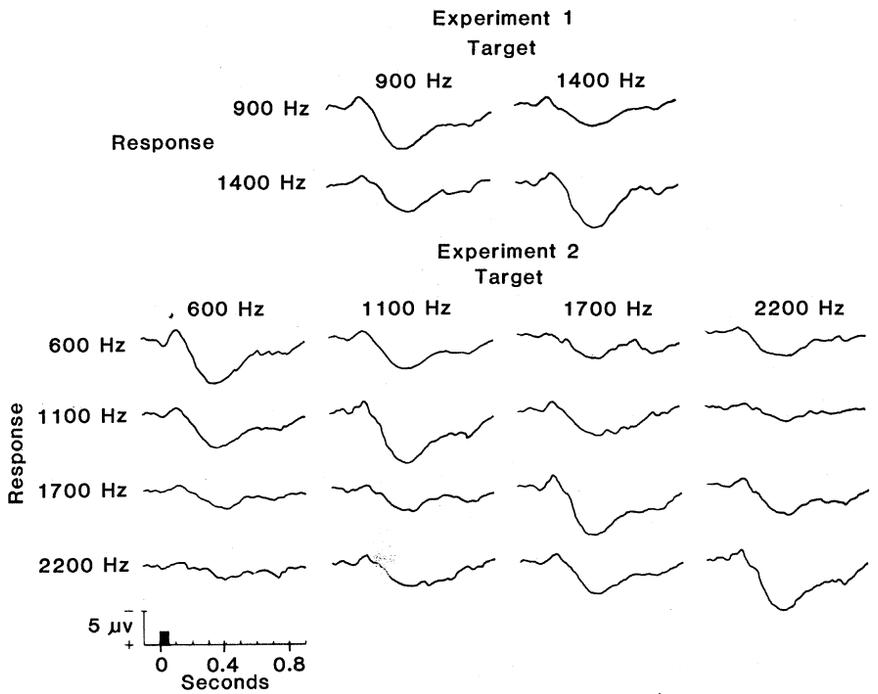

Fig. 2. Grand average ERP's (averaged over the six subjects in experiment 1 and over the four subjects in experiment 2) for each cell of the confusion matrix between target type and recognition response (four cells for the two targets in experiment 1, 16 cells for the four targets in experiment 2).

associated with recognition are faithful accompaniments of perceptual processing; they are sufficiently robust to permit accurate electrophysiological prediction of recognition performance on individual trials.

Finally, these results have implications for neurophysiological theories of perceptual processing and for the interpretation of brain ERP components. The finding that the early N100 component of the ERP varies only with detection, whereas the late P300 component varies with both detection and recognition, parallels the differential involvement of these components with stimulus selection and target detection in selective information processing (*12*). The process of detecting a stimulus may thus begin before the process of recognition or identification, but at later stages detection and recognition seem to be concurrent processes. This view is in accord with recent theoretical elaborations of the SDT model (*13*) and with the hypothesis that detection and recognition proceed together as the nervous system develops probabilistic information of the sensory signal, which forms the basis for perceptual decisions of either type (*13, 14*). The finding that P300 amplitude indexes whether a stimulus will be recognized reliably suggests that P300 may reflect the quality of this information relative to some internal criterion. One interpretation of this finding is that the processes manifested in the P300 component directly influence stimulus recognition. An alternative interpretation is that this brain ERP component is a sign (*15*) of the interaction between more fundamental neurophysiological events and behavioral events. The direction of causality between brain events and behavioral events cannot be determined from these data, and is not presumed. Nonetheless, these results provide what we believe to be the first demonstration that the central electrophysiological events occurring during the perception of sensory stimuli are closely associated with the recognition of these stimuli by the nervous system.

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7. The single-trial EEG epochs were filtered with a minimum mean-square error digital filter. Filter coefficients were chosen individually for each subject to remove high-frequency EEG and EEG components uncorrelated with the ERP. A peak selection and detection routine modified from one developed by C. McGillem and J. Aunon [*IEEE Trans. Biomed. Eng.* **24**, 232 (1977)] searched for and obtained estimates of ERP components.
8. These data should be interpreted in conjunction with the data on the percentage of P300 peaks detected. For ratings 1 to 4, the percentages of peaks detected on single trials (at a 95 percent confidence level) were 85, 90, 86, and 75 percent (recognized targets) and 88, 85, 86, and 76 percent (unrecognized targets). There were no significant differences in the proportions of peaks detected between recognized and unrecognized targets. Thus, the P300 amplitude difference between recognized and unrecognized targets did not depend on the number of peaks reliably associated with these two categories of response.
9. The distance between target type and response in experiment 2 was given a value from 0 to 3, depending on the position of the cell relative to the diagonal in the confusion matrix (for example, correctly recognized targets = 0; 600-Hz target, 1700-Hz response = 2). The Spearman rank-order correlations between distance and P300 amplitude for subjects R.J., T.T., H.F., and A.C. were −.71, −.74, −.88, and −.81 (N = 16, $P < .01$ in each case).
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$$Pc_i(D + R/s) = Pc_i(D/s) - \left(\frac{M - 1}{M}\right)\int_0^{c_i} \frac{1 - Pc(D/s)}{1 - Pc(D/n)}\, dPc(D/n)$$

where M is the target set size, s and n refer to signal plus noise and noise alone, respectively, and $Pc(D/s)$ and $Pc(D/n)$ are the hit and false alarm probabilities, respectively, at the criterion c. The integral was estimated by an area measure

$$\sum_{j=1}^{i} \frac{1}{2}\left[\frac{1 - Pc_j(D/s)}{1 - Pc_j(D/n)} + \frac{1 - Pc_{j-1}(D/s)}{1 - Pc_{j-1}(D/n)}\right] \times [Pc_j(D/n) - Pc_{j-1}(D/n)]$$

where $[1 - Pc_j(D/s)]/[1 - Pc_j(D/n)] \rightarrow 1$ as $Pc_j(D/n) \rightarrow 0$.
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27 February 1980; revised 9 June 1980

Acoustic Responses After Total Destruction of the Cochlear Receptor: Brainstem and Auditory Cortex

Abstract. *Acoustically evoked neural activity has been recorded from the brainstem and auditory cortex of guinea pigs after complete destruction of the organ of Corti by the aminoglycosidic antibiotic amikacin. These responses to sound differ in important respects from the evoked potentials normally recorded from the auditory pathways. At the brainstem level they resemble the potentials reported by others after stimulation of the vestibular nerve.*

Injury to the organ of Corti by aminoglycosidic antibiotics is a well-documented phenomenon (*1*). It results in a loss of sensitivity of the cochlea to acoustic stimulation beginning with the high frequencies; the functional loss increases as the destruction proceeds from the basal end toward the apex and is proportional to the amount of anatomical damage. Recently we reported examples of complete destruction of the organ of Corti in guinea pigs treated with massive doses of the antibiotic amikacin (*2*). In these animals, in which all sensory cells had disappeared except for a very few outer hair cells at the extreme apex, well-defined, short-latency responses to clicks could still be recorded. Such observations were later repeated in another group of animals, and a more detailed analysis of the characteristics of the responses strongly suggested that they were neural (*3*). On the basis of these data, the site of the acoustico-neural transduction remains unclear. The hypothesis of the stimulation of some nerve fibers still remaining within the cochlea is not easily tenable, whereas the implication of the vestibular system, whose structure and function were preserved, seems more likely. In particular, the possibility of electrical synapses (*4, 5*) in the vestibular neuroepithelia would explain the very short latency and the lack of adaptation of the responses.

This report is concerned with the structures of the central nervous system excited by this peripheral acoustico-neural transduction in the absence of the organ of Corti. It describes the results of far-field recordings of acoustically evoked brainstem potentials together with responses obtained with differential recordings from the auditory cortex in animals for which the peculiar round-window responses and the total cochlear hair cell destructions were also confirmed.

Adult tricolor guinea pigs were used in

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