dimensions of a grating explored with the fingers are represented in the responses of cortical neurons remains to be determined.

Preliminary examination of neurons in area 2 (posterior to area 1) indicates that these cells differ substantially from those described above. Many area 2 cells had cutaneous receptive fields that responded to brushing of two or more adjacent finger pads but not of the intervening skin. A high proportion of these neurons responded vigorously to movement of the finger pads in one direction across a grating but not to movement in the reverse direction (*8*), and a further group of cells, although responding to repetitive, gentle brushing of the pads, did not respond when the pads were rubbed to and fro across the grating.

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6. The subjective roughness of a periodic grating increases with its spatial period and the width of its grooves (Fig. 1) and decreases with increases in the width of its ridges [S. J. Lederman, *Percept. Psychophys.* **16**, 385 (1974); M. M. Taylor and S. J. Lederman, *ibid.* **17**, 23 (1975)].
7. The methods used for recording single neurons in the cerebral cortex of the alert macaque were similar to those used by several other laboratories [E. V. Evarts, *Methods Med. Res.* **11**, 241 (1966); V. B. Mountcastle, J. C. Lynch, A. Georgopoulos, H. Sakata, C. Acuna, *J. Neurophysiol.* **38**, 871 (1975)]. Recordings for the first few weeks were in the left hemisphere. After the transfer of learned movements to the left hand, recordings were continued in the right hemisphere.
8. Directionally selective neurons of this type have been observed in area 2 [J. Hyvarinen and A. Poranen, *J. Physiol. (London)* **283**, 523 (1978); Y. Iwamura and M. Tanaka, *Brain Res.* **150**, 662 (1978); E. P. Gardner and R. M. Costanzo, *J. Neurophysiol.* **43**, 420 (1980)].
9. Supported by grants from the University of Melbourne, the National Health & Medical Research Council of Australia, the Ian Potter Foundation, and the Van Cleef Foundation.

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Manipulation of Event-Related Potential Manifestations of Information Processing Stages

Abstract. *The timing of two event-related potential components was differentially affected by two experimental variables. The earlier component (N_A) was affected by degradation of the stimuli and the later component (N2) by the nature of a classification task. The results support the hypothesis that N_A and N2 reflect sequential stages of information processing, namely, pattern recognition and stimulus classification.*

The identification of brain activity associated with sequential stages of information processing has broad significance for cognitive science. An assumption underlying the concept of sequential stages of processing is that the time required for an earlier stage to extract the information necessary for the operations of a later stage should affect the timing of the later stage. Also, the duration of a later stage may vary with the task being performed even though the durations of earlier stages remain constant (*1*). We report here a pattern of results compatible with these assumptions concerning the timing of two event-related potential (ERP) components believed to be associated with two sequential stages of processing.

There is now substantial evidence that the N2 component reflects discrimination and classification of stimuli (*2*). The peak latency of N2 varies as a function of the difficulty of discrimination and precedes and correlates with the timing of discriminative behavioral responses (*3*). In a recent study (*4*), we identified an earlier negative component (designated N_A) which seems to be associated with pattern recognition processes. On the basis of changes in the relative timing of these two components across experimental conditions, we inferred that the discrimination and classification processes associated with N2 depended on the prior stimulus identification processes associated with N_A.

We have now tested the hypotheses about the stages of information processing associated with N_A and N2 by differentially manipulating their timing. We predicted that degrading the stimuli would increase the peak (but not the onset) latency of N_A. Since we hypothesized that the processing associated with N2 depends on the processing associated with N_A, changes in the peak latency of N_A should affect the timing of N2. On the other hand, varying the time required to perform a classification task was expected to affect only the peak latency of N2, producing an increase in the interval between the peaks of N_A and N2.

Eleven adult subjects were given two

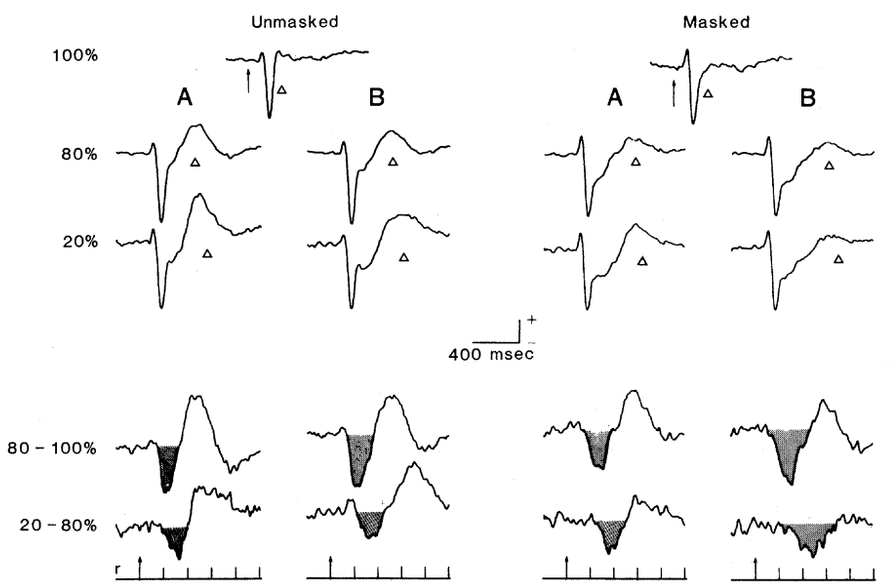

Fig. 1. Grand mean waveforms (11 subjects) at T5 to the unmasked and masked stimuli in tasks A and B during simple RT (100 percent) and choice RT (80 and 20 percent). Dark areas in difference waveforms below indicate N_A (80 − 100 percent) and N2 (20 − 80 percent). The bottom right waveform is from seven subjects for whom N2 could be measured in that condition. Arrows indicate stimulus onset and open triangles indicate mean RT. Voltage calibration is 5 μV for the upper three rows and 2.5 μV for the difference waveforms.

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visual classification tasks. In task A, a letter of the alphabet or a single digit was presented on each trial. Subjects differentially lifted either the right or left index finger as quickly as possible for the two classes of stimuli. All possible nonconfusable letters and digits were used. In task B, only letters were presented. Subjects memorized four letters of the alphabet and then responded differentially to whether the letter presented on a given trial was or was not a member of the memory set. Since N2 is larger for less probable stimuli (or classes or stimuli), the digits (task A) and the letters in the memorized set (task B) randomly occurred on 20 percent of the trials of their respective tasks. In another condition, the letter F was presented on each trial of a simple reaction time (RT) task.

The stimuli, which were black on a light gray background, were presented on a video monitor at the rate of one every 2.75 seconds. On half of the runs of each task, the stimuli were degraded with a mask consisting of a 4 by 3 matrix of small black x's presented concurrently with and superimposed on the letters and digits. Stimulus duration was constant for all conditions for a given subject, but ranged from 75 to 125 msec depending on the subject's ability to perform the classification tasks with a minimum of 90 percent accuracy for the degraded stimuli.

The ERP's were recorded from Cz, Pz, Oz, T5, and the left mastoid, all referred to the nose (including electrooculograms from above the right eye). Electroencephalographic amplification was down 3 dB at 0.1 and 100 Hz.

Difference waveforms were computed separately for the ERP's elicited by the degraded and undegraded stimuli. For each subject, the ERP obtained during simple RT was subtracted from that associated with the class of stimuli that occurred on 80 percent of the trials of each classification task. These difference waveforms delineated N_A, which is larger when stimuli randomly change from trial to trial during the classification tasks than when the same stimulus is constantly repeated during simple RT (*4*). Subtractions between the ERP's associated with the stimulus classes that occurred on 80 and 20 percent of the trials of each task were also computed to permit more effective delineation of N2.

The onset of N_A and the peak latencies of N_A and N2 were measured from the difference waveforms at T5, where both components were largest. In four subjects, N2 could not be measured for task B with degraded stimuli, probably because of its increased variability in latency from trial to trial. The results were tested with two-way analyses of variance for each peak and had P values of $< .01$, unless otherwise stated (*5*). In addition, a two-way analysis of variance was performed on the mean interval between the peaks of N_A and N2.

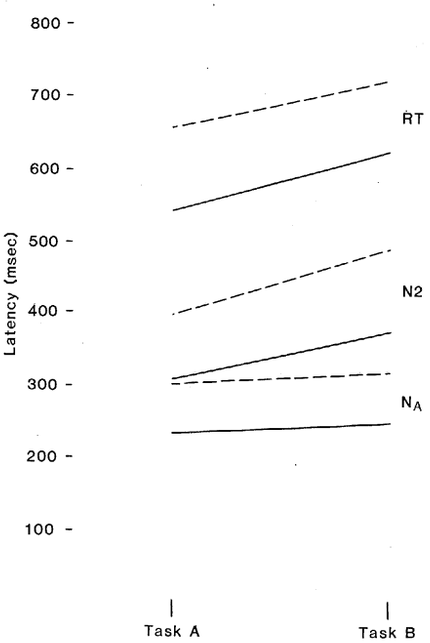

Fig. 2. Mean peak latencies of N_A and N2 measured at T5, and mean response times to the 20 percent stimuli, when masked (dashed lines) and unmasked (solid lines) in tasks A and B.

The ERP's averaged across subjects at T5 are presented in Fig. 1. During simple RT, the waveforms contain a positive and a negative wave peaking at 115 and 175 msec, respectively (P115 and N175). Neither simple RT (mean of 280 msec) nor the peak latencies of P115 or N175 changed with the introduction of the mask. Moreover, the latencies of P115 and N175 were not affected by any of the experimental conditions (rows 2 and 3). The 80 − 100 percent difference waveforms depict N_A, and the 20 − 80 percent difference waveforms depict N2.

The onset latency of N_A did not change across tasks ($P > .25$), but degrading the stimuli increased the peak latency of N_A about the same magnitude (70 msec) for the two classification tasks (Fig. 1). Figure 2 presents the peak latencies of N_A and N2 for tasks A and B with and without the mask, as well as mean RT's to the class of stimuli which occurred on 20 percent of the trials. The significant increase in the peak latency of N_A for the degraded stimuli was associated with significant increases in the peak latency of N2 and of RT. As predicted, the time interval between the peaks of N_A and N2 was not affected by the presence or absence of the mask ($P > .45$). By contrast, the interval between the peaks of N_A and N2 was affected by the nature of the classification task, being significantly longer for task B than for task A both with and without the mask. There were significant main effects of mode of stimulus presentation and nature of classification task on RT for the stimulus classes which occurred on both 20 and 80 percent of the trials. The RT was an additive for the frequent and infrequent stimulus classes, as suggested by the lack of interaction between task and mode of stimulus presentation (*6*).

In addition to differential affects on their latencies, N_A and N2 had different topographic distributions across the recording sites (*7*). Further differences between the N_A and N2 components, such as that they are morphologically distinct (N2 appears as a second inflection in the 20 − 100 percent subtraction waveform) and that their amplitudes vary differently as a function of stimulus probability, are detailed elsewhere (*4*).

The data support the interpretation that N_A and N2 reflect two sequential stages of information processing. The timing of N_A was affected by the difficulty in perceiving the stimuli, whereas the timing of N2 was differentially affected by the nature of the classification task. Moreover, in combination the changes in peak latency of N_A and N2 largely account for the variations in RT across the four conditions (Fig. 2). This result suggests that ERP components may be useful in identifying physiologically which stages of processing are affected by particular cognitive variables (*8*).

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5. Statistical details are available on request.
6. The peak latency of P3 (P300), measured at Pz for the ERP's to the infrequent classes of stimuli, yielded the same main effects, and lack of interaction, as RT. The increase in the peak latency of P3 with the introduction of the mask supports the findings of G. McCarthy and E. Donchin [*Science* **211**, 77 (1981)] in an experiment that crossed two independent variables in a 2 by 2 design according to logic similar to ours. The possibility that the changes in the peak of N_A across conditions were due to alterations of motor potentials or the latency of P3 is remote because the nature of the classification task had a significant main effect on RT and P3 latency but not on N_A peak latency.
7. The average percentages of maximum amplitude of N_A and N2, derived separately for each subject and collapsed across the four choice RT conditions, were, respectively, 50 and 29 at Cz, 65 and 47 at Pz, 77 and 77 at Oz, 92 and 92 at T5, and 46 and 66 at the mastoid. By the more conservative method outlined by J. R. Jennings and C. C. Wood [*Psychophysiology* **13**, 277 (1976)], analysis of variance yielded a significant interaction between components and electrodes ($P < .004$). The topography of N_A seems to differ from the selective attention effects on visual ERP's found by S. Van Voorhis and S. Hillyard [*Percept. Psychophys.* **22**, 54 (1977)], which comprised a positive enhancement around 220 msec at O1 and a negative enhancement around 155 msec at Cz.
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Impulse Conduction in the Mammalian Brain: Physiological Properties of Individual Axons Monitored for Several Months

Abstract. *Microelectrode recordings were used in conjunction with antidromic activation to monitor impulse conduction along individual mammalian cerebral axons for periods of up to 165 days. Approximately half of the axons studied showed a stable conduction velocity and stable aftereffects of impulse activity. The remaining axons showed slow and progressive increases or decreases in conduction velocity overtime. In these latter axons, changes in the magnitude of the aftereffects of impulse conduction were far less pronounced than were changes in axonal conduction velocity.*

Little is known about the long-term stability of axonal conduction properties in the central nervous system. It is not known, for example, whether the velocity of impulse conduction and the aftereffects of impulse activity in individual axons of adult mammals remain stable for long periods of time. The paucity of such information is primarily attributable to the lack of a method with which to study individual axons for more than a few hours. Such information is important for the understanding of progressive pathological influences on impulse conduction. In the study reported here, extracellular microelectrode recording methods were combined with antidromic activation of axons at one or several sites. These methods enabled the continuous study of conduction properties of individual axons in the mammalian brain for several months.

Recordings were obtained from the cell bodies of visual callosal axons in adult Dutch Belted rabbits. This axonal system consists of both nonmyelinated and small myelinated axons (*1*) which conduct impulses relatively slowly (*2–4*). The aftereffects of impulse conduction in this system have been documented (*2*, *4*, *5*). Microelectrodes were permanently implanted near the border of visual areas I and II (Fig. 1) (*6*). Stimulating electrodes activated the callosal axon near the midline (*7*) and in some cases near the terminals in the contralateral hemisphere. Recording sessions began approximately 1 week after surgery (*8*). Antidromic activation was differentiated from synaptic activation by means of collision tests and other criteria (*9*). Brain temperature was monitored (*10*) to ensure that the observed variations in conduction properties were not temperature-dependent (*11*). At the end of the experiments the animals were killed and the tissue was prepared for histological analysis (*12*).

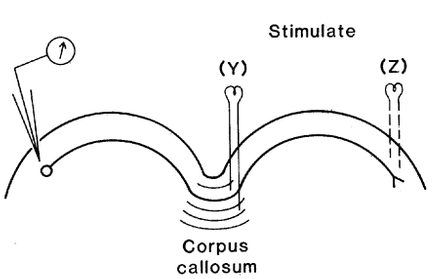

Fig. 1. Schematic illustration of the experimental setup. Microelectrodes were implanted into superficial cortical layers near the border of visual areas I and II. Banks of stimulating electrodes were implanted in the splenium of the corpus callosum (*Y*) and, in some cases, in the contralateral hemisphere (*Z*).

The present results are for 23 neurons studied for 20 to 165 days (mean, 48 days). An additional 40 neurons were studied for 5 to 19 days and provide confirmatory data. This report focuses on conduction velocity, supernormal conduction velocity, and the minimum interspike interval, since these three conduction properties were either very stable over time or showed progressive, systematic changes. Other measures studied were less stable over time (*13*).

Figure 2 shows oscilloscope tracings illustrating these measures. The records were obtained on the 60th day of recording from a cell that was studied for 165 days. Antidromic latency (25.3 msec) to a single antidromic test stimulus is shown in Fig. 2A. In Fig. 2B the supernormal conduction velocity that follows a single prior impulse is shown. The increase in conduction velocity is manifested as a decrease in antidromic latency to a test stimulus that follows a spontaneous spike or an electrically elicited spike at appropriate intervals (*14*). The minimum interspike interval (2.15 msec), shown in Fig. 2C, is the minimum interval between two conducted action potentials elicited by two stimuli presented at an interval near the absolute refractory period of the axon (1.3 msec).

Eleven of the 23 axons studied at length demonstrated a stable antidromic latency, with a variation of < 5 percent from the first to the last day of recording. In four of the remaining neurons, cumulative decreases in antidromic latency (increases in conduction velocity) of 8 to 14 percent occurred at a mean rate of 0.2 percent per day, while in eight neurons cumulative increases in antidromic latency (decreases in conduction velocity) of 6 to 81 percent occurred at a mean rate of 0.5 percent per day (*15*). Figure 3A shows data for three neurons studied for 78 days, 101 days, and 165 days. They demonstrated stable, decreasing, and increasing antidromic latencies, respectively.

The minimum interspike interval reflects the recovery processes of the entire axon between stimulating electrode and soma and is the reciprocal of the maximum firing frequency that may occur along this length of axon. Although the value of this measure varied significantly between cells (1.55 to 3.25 msec for the 23 cells studied at length), little

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