permit the use of signal enhancement techniques, this change is difficult to isolate but presumably starts when something seen in peripheral vision directs the eye scan toward the area of display containing the vehicle. It is similar in form to the contingent negative variation (*14*) and may indicate preparation for action.

However the main cortical sign of detection is the occurrence of a large positive potential maximum at the vertex and parietal electrodes shortly after the eyes have fixated in the region of the vehicle. This potential is not time locked to the large eye movement that brings the point of gaze into the region of the vehicle, although such potentials may be related to fine eye movements below the resolution of the oculogram. They occur when the observer has seen the vehicle and started to track it and could represent the changeover from the scanning to the tracking mode of the eye (*15*). However, as shown in Fig. 1, at 8 seconds after entry the reacquisition of the vehicle and the resumption of tracking after the eyes have flicked away for 500 msec is not accompanied by an obvious positivity. Nor are these potentials dependent on the execution of a motor action since they also occur when the observer sees a vehicle to which he must not respond by pressing the swtich. In this situation, however, it is still necessary for the observer to detect the vehicle in order to decide not to press. Thus it appears that the positivity occurs when the observer sees one of a class of events that he has been told to detect.

Both the role of these potentials, their distribution, and their positive polarity suggest that they might have common origins with the P300 component of the cortical evoked potential which occurs characteristically during discriminating and decision-making tasks (*16*). The amplitude of this component is usually less than 10 μv although Courchesne *et al.* (*7*) show a P300 component of 30 μv to the first presentation of the stimulus and Vaughan and Ritter (*17*) describe large potentials to infrequent events.

What the latency of 300 msec would mean in our experiments is difficult to tell because the vehicle is there for several seconds before it is seen; but often the occurrence of the P300 in other experiments is not accurately time locked to preceding events (*18, 19*). It may be related to the last microeye movement that finally confirms the existence of the vehicle, since the distribution of eye movements preceding the positivity (Fig. 4) is not normal and has a group of values of about 200 msec in most subjects. The hypotheses that have been advanced to describe the psychological nature of the P300 wave, such as the resolution of uncertainty (*20*), the orienting response to an unexpected stimulus (*21*), the stimulus-independent perceptual decision process (*19*), or the matching of a neural template (*22*) might have some relevance to the origin of the detection positivity and could lead to new experimentation that could advance our knowledge of what we mean by "seeing" a target.

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8. Contrast here is defined as $[(W - B)/(W + B)] \times 100$ where W and B are the brightness of the white and black parts of the display.
9. These electrode designations are part of the standardized placements known as the 10–20 system.
10. Amplifiers having an 8-second time constant were used to stabilize drift due to electrodes. This bandwidth is sufficient for determination of scanning pattern and the beginning of tracking. In three subjects, d-c amplification and very stable electrodes were used for scanning and for tracking the vehicle.
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12. By means of high-quality silver–silver chloride electrodes, the oculogram can be used to locate the point of gaze to about $\pm$ ½° for short periods of time (30 seconds). In our experiments long-term instability is corrected by positioning the two-dimensional display from the known position of the eyes and vehicle during tracking.
13. In subject S2, the average amplitude at the vertex decreased from 71 $\pm$ 10 μv to 55 $\pm$ 8 μv, $P < .002$.
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Reading Disability: An Information-Processing Analysis

Abstract. *In a task designed to separate perceptual processes from memory, 12-year-old children with reading disabilities showed no perceptual deficits as compared to their peers. However, they exhibited major deficiencies in memory skills for both labelable and nonlabelable visual information. Reading-disabled children in this age group appear to suffer from a basic information-processing deficiency.*

The nature of reading disability has been one of the most difficult and puzzling problems facing psychologists and educators. For years reading problems were thought to be a difficulty in the perception of written symbols. As recently as 1972 Cruickshank concluded that reading disabilities ". . . are essentially and almost always the result of perceptual problems based on the neurological system" (*1*). However, recent work has cast doubt on this "perceptual deficit" hypothesis and pointed instead to deficits in memory processes (*2*). One persistent problem in assessing reading disability has been the inability to devise experimental procedures for separating perception from memory (*3*). Recently techniques have been developed for isolating perceptual and memory processes by assuming them to be occurring sequentially in time (*4*). It has been demonstrated experimentally that initially large amounts of information are perceived by the visual system. This information persists in a raw perceptual form (called visual information storage or VIS) for about 0.25 second. During this period subjects are actively coding and transferring information into a more permanent storage (called short-term storage). The ability to temporally separate perception

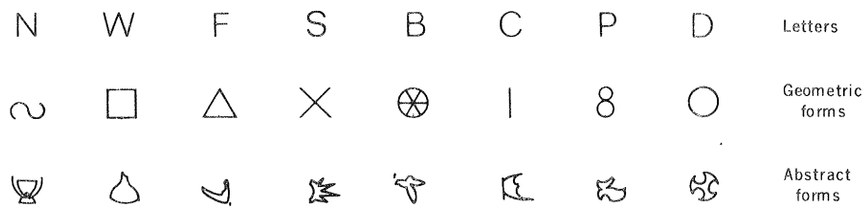

Fig. 1. Complete set of letters, geometric forms, and abstract forms used in the stimulus arrays.

from memory in the present study made possible a more fine-grained assessment of some factors potentially underlying reading disability. The specific procedure used was called the partial report technique. It involved presenting subjects with a circular array of eight visual forms for a very brief duration (150 msec) which prevented eye movements. Following offset of the array at varying intervals (0 to 2000 msec) a teardrop indicator appeared under one of the forms. The subject's task was to report the form to which the indicator had pointed by picking it out on a response card that contained all eight of the forms used in the original array. By noting the accuracy levels at the various delay intervals it was possible to estimate the amount of information initially perceived by the subject (0-msec delay), the trace duration of information in VIS (0 to 300 msec), and the amount of information encoded and transferred to more permanent storage (300 to 2000 msec). It was hypothesized that if reading disability were a perceptual deficit, then performance of reading-disabled children would be inferior to that of normal readers at short delays (0 to 300 msec) for which information was still held in a raw perceptual form. However, if reading disability involved an encoding or memory deficit, it was believed that performance of poor readers would be inferior only at later intervals (after the 300-msec delay). Also included in this study was a test of whether reading disability was a specific deficit, limited to primarily verbal materials (letters), or whether it might be a more general processing deficit. Accordingly three sets of figures that had been shown to differ in degree of familiarity or labelability were used (*5*): letters, geometric forms, and abstract forms (Fig. 1). It was thought that if reading disability were limited to predominantly verbal materials, major differences in performance between normal and poor readers would occur only with the letter stimuli or possibly geometric forms. If, however, reading-disabled children suffered a more general processing deficit, major differences were expected on all three sets of forms.

The children tested were all 12-year-old males in the sixth grade. Two groups of nine children each were identified as either normal readers (reading at grade level) or poor readers (reading at two levels or more below grade level) on the Comprehensive Test of Basic Skills (McGraw-Hill). The poor readers were average or above in intelligence and in other school subjects, and showed no gross behavioral problems or organic disorders. The poor readers were all in regular classes and had not received special instruction. A Gerbrands four-field tachistoscope, coupled with six Hunter timers which controlled stimulus durations and other intervals, was used in the experiment. After being familiarized with the equipment and procedure, each subject went through the following sequence: he looked inside the tachistoscope at a small fixation dot in the center of the visual field; a "ready-go" signal was given verbally and, after a 750-msec delay, the stimulus array flashed on for 150 msec. Following offset of the array the indicator appeared under one of the figures for 50 msec followed by reappearance of the fixation field. The subject then chose the indicated figure on the response card. Each stimulus card contained eight forms (each subtending 33′ of visual angle) in a circular array 1.30° in radius from central fixation. The teardrop indicator, subtending 26′ of visual angle, was placed approximately 25′ of visual angle from the stimulus figure. Illumination levels for the stimulus field, indicator field, and fixation field were approximately 4.0 foot-lamberts. Ten delay intervals were sampled: 0, 50, 100, 200, 300, 500, 800, 1000, 1500, and 2000 msec. A total of eight cards per delay interval was presented. Each position in the array served once as target position and each stimulus served as target stimulus once per interstimulus interval. All subjects were tested on the three familiarity sets on three consecutive days. For half the subjects the order of presentation was letters, geometric forms, and abstract forms while the other half viewed abstract forms followed by geometric forms and letters. A total of 80 trials per familiarity set was presented.

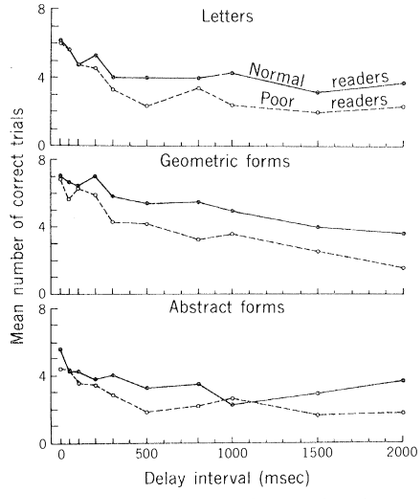

Fig. 2. Accuracy levels of normal and poor readers on the three sets of figures across all delay intervals.

The mean number of correct responses made by the normal and poor readers was compared across two sets of delay intervals: the perceptual phase, 0 to 300 msec and the encoding-memory phase, 500 to 2000 msec. Four major findings stood out. (i) There was no difference in performance between normal and poor readers across the 0- to 300-msec delay interval. As shown in Fig. 2, poor readers performed as well as normal readers while the information was present perceptually. (ii) No differences were found between the two groups during this phase on any of the sets of visual forms. (iii) Normal readers performed significantly better than poor readers during the encoding-memory phase ($P < .0001$). (iv) This superiority held for all three sets of forms (*6*).

Although it was clear from the first two results that the quantity of information initially perceived by poor readers was identical to that of normal readers, the quality of information might have been different in the two groups. At short delay intervals the poor readers might still have had enough high-quality information to perform adequately but at later intervals when information was fading rapidly the lower quality of information perceived by the poor readers could have produced lower levels of performance. In order to test this possibility a stimulus-response confusion matrix was constructed for each reading group and set of forms separately for each of the two sets of delay intervals. On the basis of these group matrices, a configuration of items representing their judged similarity was determined by a multidimensional scaling analysis (*7*). It was thought that by observing the kinds of

confusions made by the two groups of readers, an assessment could be made of whether they differed in the kind or quality of information perceived. Across all sets of figures and delay intervals, normal and poor readers showed similar confusion error patterns. Both groups tended to choose an incorrect form that was visually confusable with the correct form.

Taken together, the results of this study showed that poor readers were not deficient in the quantity or quality of information they initially perceived or in the trace duration of that information in a raw perceptual form (VIS). Poor readers did show a striking deficit during the 300- to 2000-msec interval, which argues that reading disability involves some problem in the processing of information in stages following initial perception, perhaps in encoding, organizational, or retrieval skills. Also, reading disability is not limited to verbal materials since poor readers performed equally poorly compared with normal readers on the geometric and abstract forms. The real difficulty may involve a more abstract ability which underlies processing of both labelable and unlabelable forms. The conclusions drawn from this study must at present be restricted to older children since beginning readers were not included. Further research is needed to assess the generality across age of the processing deficit discovered here. Nevertheless, the fact that poor readers were found to be deficient in a form of processing that is not primarily verbal is important especially given a recent tendency to tie reading problems in older children to verbal and linguistic processes (*4*). The development of techniques that can tease apart component processes acting on information represents an important step toward clarifying the complex nature of reading disability.

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6. Additional significant results included superior performance on the geometric forms over the letters and abstract forms [$F(2, 32) = 55.2$, $P < .0001$]; decrease in performance across delay intervals in the perceptual phase [$F(4, 80) = 60.2$, $P < .0001$); and an interaction between delay interval and type of figure, namely performance on the abstract forms and letters decreased more sharply in the 0- to 300-msec interval than did performance on the geometric forms [$F(10, 160) = 3.47$, $P < .0004$].
7. The output of this analysis provided a spatial configuration of items representing their judged similarity such that distance was monotonically and negatively related to similarity. A measure of the number of dimensions critical to task performance was obtained in the form of work level. This indicated the degree of difficulty incurred in accounting for data constraints within the respective number of specified dimensions, work level being inversely related to goodness of fit. Performance for both reading groups exhibited the best fit in a two-dimensional space with the work level for normal and poor readers being very similar: for example, 17.9 and 16.0, respectively, for the normal and poor readers on the abstract forms during the perceptual phase.
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Homeostasis During Hypoglycemia: Central Control of Adrenal Secretion and Peripheral Control of Feeding

Abstract. *Intravenous infusions of mannose or β-hydroxybutyrate, metabolic fuels which can be oxidized by brain, abolished adrenal discharge of epinephrine in rats during insulin-induced hypoglycemia, whereas infusion of fructose, a sugar which does not cross the blood-brain barrier, did not. In contrast, increased feeding behavior during hypoglycemia was prevented both by the sugars and by β-hydroxybutyrate. Thus, while the sympathetic response during marked hypoglycemia may have been initiated by alterations in cerebral metabolism, the feeding response evidently was not, and a decrease in the utilization of glucose per se does not appear to be the critical stimulus in either case.*

Compensatory responses help to maintain caloric homeostasis during insulin-induced hypoglycemia. For example, the discharge of epinephrine from the adrenal glands (*1, 2*) promotes the mobilization of internal fuel reserves, while increased feeding behavior (*3*) provides a fresh supply of nutrients from extracorporeal sources. It has long been assumed that both of these reactions are triggered by central glucoreceptor cells, in response to the decreased supply of glucose that reaches the brain (*2, 4*). Recently, however, Flatt *et al.* (*5*) have shown that intravenous infusion of ketone bodies (β-hydroxybutyrate and acetoacetate), metabolic fuels which can be oxidized by brain (*6*), abolishes the increased adrenal medullary secretions of anesthetized dogs during insulin-induced hypoglycemia. We have extended those findings to conscious rats, and demonstrate that intravenous infusions of mannose also lower circulating catecholamine levels despite continued hypoglycemia, whereas infusions of fructose, a carbohydrate which is not readily utilized by rat brain (*7*), do not. Furthermore, in contrast with their differential success in affecting adrenal medullary secretion, we have found that each of these metabolic fuels suppressed the feeding response to exogenous insulin.

Male albino rats of the Sprague-Dawley strain (Zivic-Miller), weighing 300 to 350 g, were housed in individual wire-mesh cages and allowed free access to Purina Chow pellets and tap water, both in their home cages and during testing, except as noted. Forty-five rats were fitted with intracardiac catheters (*8*). Beginning 3 days later, when food and water intakes had returned to normal, the rats were placed in cylindrical testing chambers (42 cm high, 28 cm in diameter). Several hours were allowed for adaptation to these surroundings. At about noon, rats were given a single injection of regular insulin (3 units per kilogram of body weight) through their catheters to produce hypoglycemia. They were then connected by means of a watertight swivel and polyethylene tubing to an infusion pump and immediately began to receive either ketone bodies (1.2*M* sodium DL-β-hydroxybutyrate in 0.15*M* NaCl, at the rate of 3 ml/hour), sugar (1.2*M* solutions of glucose, fructose, or mannose, in water, at 2 ml/hour), or control NaCl solutions (0.15*M*, 0.75*M*, or 1.2*M*). Supplemental injections (2 ml/kg) of the various infusates were given by way of the catheters 10, 30, and 60 minutes later. Food and water intakes were measured every 30 minutes for 2 hours. Individual rats were usually tested with two or three different infusates, once every few days, until six to ten animals had been tested with each solution.

The effects of the various infusions on insulin-induced feeding are summarized in Table 1. Rats infused with 0.15*M* NaCl solution usually began to eat within 20 to 30 minutes after insulin treatment and continued to feed periodically throughout the 2-hour test (Table 1). Infusions of β-hydroxybutyrate, glucose, mannose, or fructose virtually abolished feeding af-