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Dyslexia

Mirror writing and similar problems are usually blamed on defects in visual perception, but in truth dyslexia seems to be a complex linguistic deficiency. The remedy is proper instruction in reading

by Frank R. Vellutino

Dyslexia is a generic term that has come to refer to an extraordinary difficulty experienced by otherwise normal children in learning to identify printed words, presumably as the result of constitutional deficiencies. The condition is commonly believed to originate in the visual-spatial system. Its presence is considered to be signaled by mirror writing and letter reversal. Dyslexics, it is believed, show uncertain hand preference. Children whose first language is based on alphabetic rather than pictographic or ideographic characters are said to be particularly susceptible to the condition. Finally, dyslexia is widely considered to be correctable by means of therapies aimed at "strengthening" the visual-spatial system. Each of these perceptions, contemporary research shows, is seriously flawed.

It was through the work of the U.S. neuropsychiatrist Samuel Torrey Orton in 1925 that the deficiency first came to be perceived as lying in the visual system. Orton suggested that an apparent dysfunction in visual perception and visual memory, characterized by a tendency to perceive letters and words in reverse (*b* for *d* or *was* for *saw*), causes dyslexia. Such a disorder would also explain mirror writing. Orton further suggested that the disorder is caused by a maturational lag: the consequence of a failure of one or the other hemisphere of the brain to dominate the development of language. This last proposal, at least, was and is still a viable hypothesis.

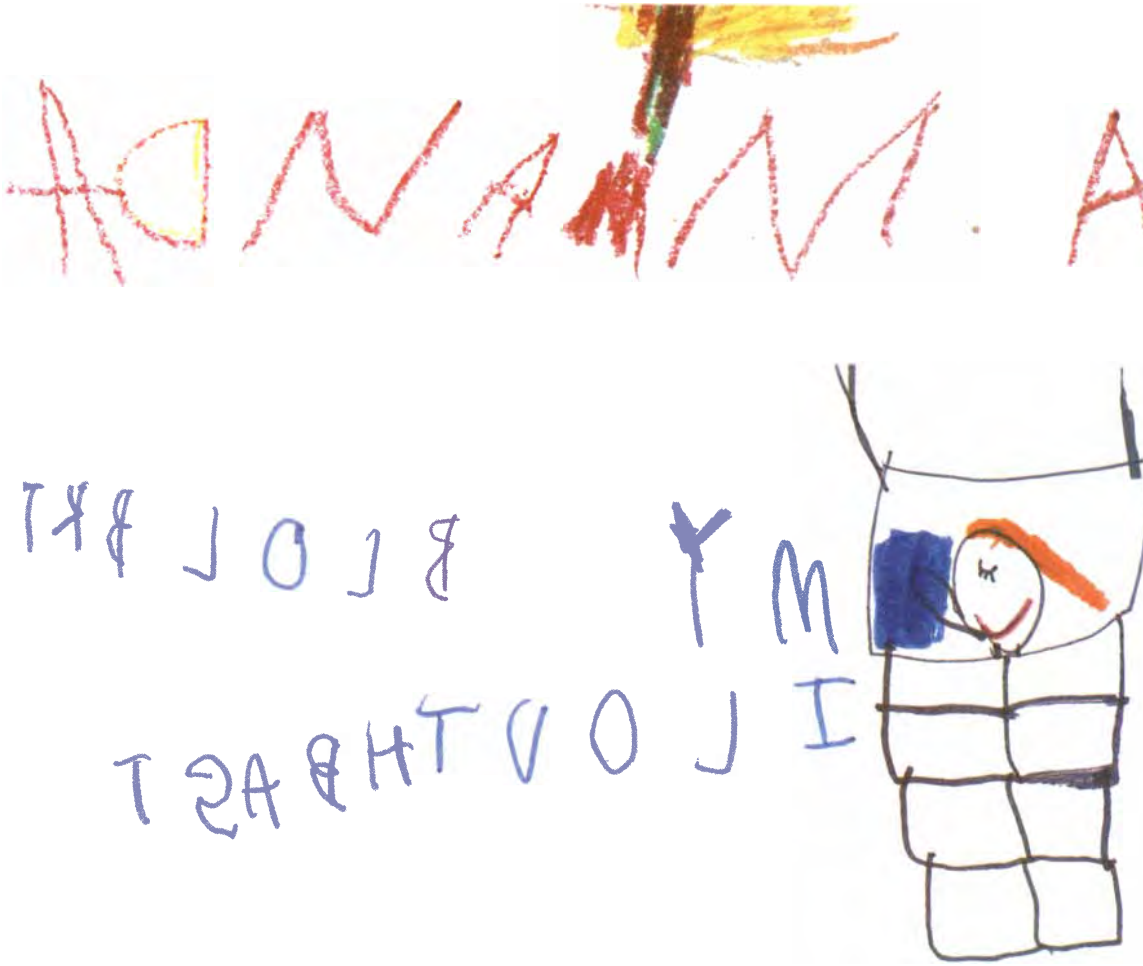
Related hypotheses, not attributable to Orton, are that dyslexia may somehow be caused either by motor and visual defects or by eye-movement defects affecting binocular coordination, eye tracking and directional scanning. Both the concept of dyslexia as a visual problem and its presumed association with uncertain cerebral dominance still underlie many therapeutic approaches to the condition.

Working at the Child Research and Study Center of the State University of New York at Albany, my colleagues and I have begun to examine, and to challenge, common beliefs about dyslexia, including the notion that the condition stems primarily from visual deficits. Along with other researchers in this country and abroad, we have been finding that dyslexia is a subtle language deficiency. The deficiency has its roots in other areas: phonological-coding deficits (inability to represent and access the sound of a word in order to help remember the word); deficient phonemic segmentation (inability to break words into component sounds); poor vocabulary development, and trouble discriminating grammatical and syntactic differences among words and sentences. Far from being a visual problem, dyslexia appears to be the consequence of limited facility in using language to code other types of information.

To understand this definition of the disorder, one can conceive of the mind as an extremely sophisticated reference library. The library model is appropriate because recent studies seem to indicate that dyslexia is as closely aligned with the cross-referencing and retrieval of coded information already stored in memory as it is with the storing and coding of new information.

The library model is based on the assumption that the processing of information to be stored in memory proceeds in stages. The first stage of processing takes place in a sensory storage system, where a replica of a given stimulus is held briefly. The second stage is believed to take place in a short-term "working" memory: a limited-capacity system in which an encoded (transformed) version of the stimulus is available for no longer than 30 seconds. In this working memory, physical information is transformed into a more abstract symbolic representation for storage in long-term memory, which is thought to have an unlimited capacity. During the final stage of memory processing, the encoded form of the stimulus is either categorized and stored in long-term memory, discarded or inadvertently lost from working memory.

In research based on this model we have found that dyslexia is more a symptom of dysfunction during storage and retrieval of linguistic information than it is a consequence of a defect in the visual system. In one experiment poor readers in the second through sixth grades, who frequently made reversal errors, were asked to copy designs, words, scrambled letters and numerals after brief visual presentation. Afterward they were asked to name the stimuli that were actual words. We found that the poor readers could reproduce the letters in a stimulus word in the correct orientation and sequence even when they could not name the word accurately. For example, they typically copied *was* correctly but then often called it "saw." When asked to read out the letters of the words right



PRESUMED SIGNS OF DYSLEXIA such as mirror writing are often seen in the early stages of normal development of writing skills. At three Amanda mirror-writes her name (*top*). The habit persists when, at four, she writes about "my blanket I love the best" (*middle*). At five she writes about playing with her friends Tracy, Daniel and Alex. Mirror writing has all but disappeared; there is a clear ability to encode a desired sound by means of phonologically appropriate (if not always correct) letters (*bottom*).

after naming the words as wholes, they could name the letters of most words in the correct order even when they named the words incorrectly.

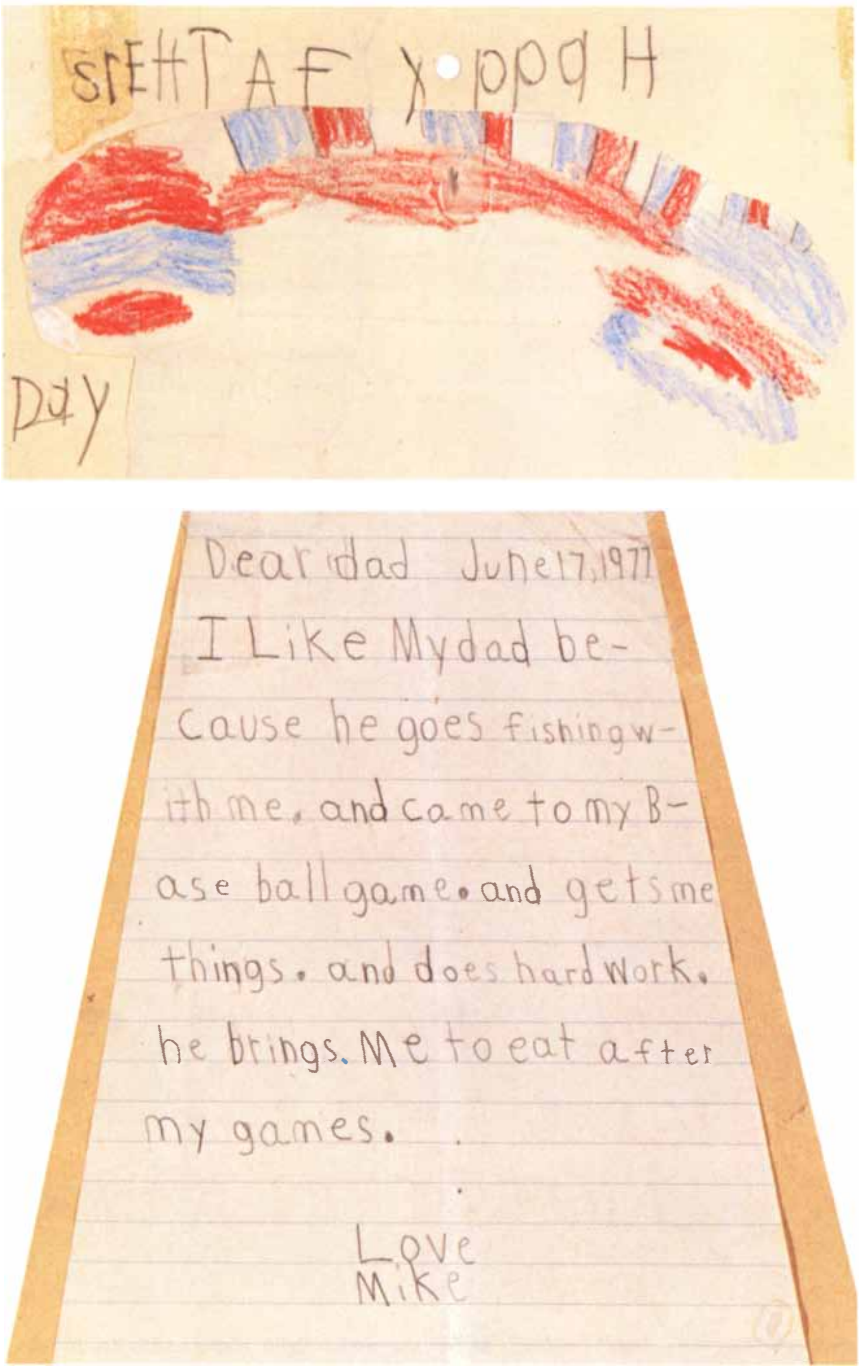
The inference from this experiment was clear. Errors such as calling *was* "saw" are the result of difficulties in storing and retrieving the names of printed words rather than of a dysfunction in visual-spatial processing.

This inference was reinforced by the results of a series of studies of children's ability to reproduce, from visual memory, words from an unfamiliar writing system. Groups of dyslexic and normal readers were asked to print Hebrew words and letters in the proper sequence and orientation after brief exposure. Some children who were already learning to read and write Hebrew were also tested to see how they would compare with the first two groups.

The important finding here was that the dyslexic readers did as well as the normal ones on this task—although, to be sure, neither group did as well as the children who were learning Hebrew. The result seemed to underscore the fact that when complex, wordlike symbols lacked any linguistic associates—had no meaning or sound, in effect—the visual recall of those symbols was no less difficult for the normal readers than it was for the poor readers. This implies that memory for visual symbols representing words is mediated by the linguistic properties of those words, particularly their meanings and sounds.

We found too that both groups of children who were not familiar with Hebrew manifested identical tendencies to process the Hebrew letters from left to right, suggesting that dyslexics are not inherently impaired in their ability to maintain left-to-right directionality. In other words, if dyslexics have difficulty maintaining proper directionality, it is a symptom of a reading disorder rather than the cause of the disorder.

A secondary inference from this study (one that has been documented by other studies directly evaluating sensory storage in dyslexic and normal readers) is that the dyslexic readers were able to hold a memory trace for as long as the normal readers. That is to say, visual traces dissipated no more rapidly in a dyslexic's sensory memory than in a normal reader's: visual form perception seems to be comparable in the two groups.



NORMAL DEVELOPMENT is evident in Father's Day cards written by a boy in first grade (*top*) and then in second grade, after he had a year of reading instruction (*bottom*).

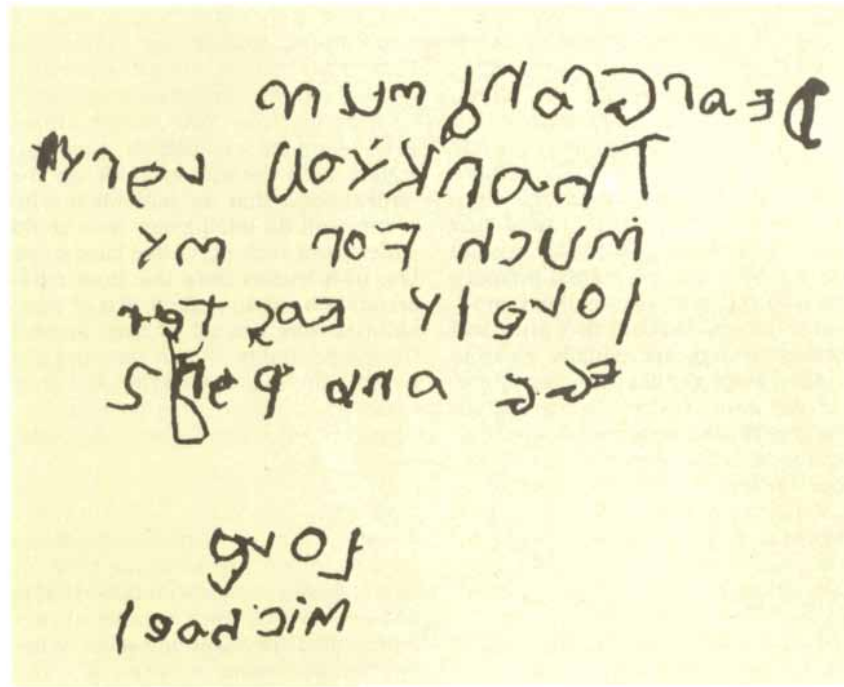
If dyslexic readers are at least capable of perceiving and reproducing letters at roughly the same level of accuracy as normally developing readers, then the problem is again thrown back on linguistic rather than visual coding systems. Printed words can be identified either through whole-word processing based on their salient visual features, their meanings and the context in which they appear, or through "part-whole" processing based on alphabetic mapping: breaking down words into letter sounds.

Because learning to read is inherently difficult, the beginning reader must be able to adopt both strategies to identify words. If the child leans too heavily on a whole-word strategy—and does not use the sounds associated with alphabetic characters to help decode new words—visual memory is inordinately taxed; errors such as *was/saw* and *lion/loin* result. On the other hand, children who rely exclusively on alphabetic mapping—and do not use salient visual features, word meanings and context to facilitate word identification—find it hard to read fluently and have trouble understanding what they read.

The implication is clear that dyslexia may be caused, on the one hand,

by highly specific linguistic deficiencies (such as vocabulary weakness or sound-mapping deficits) affecting only certain of the subskills that are necessary components of the ability to read. On the other hand, it is equally plausible that more general language deficiencies are present, which affect all subskills. A hypothesis supporting the first view has been put forward by Isabelle Y. Liberman and Donald P. Shankweiler of the Haskins Laboratories, Inc., in New Haven.

According to Liberman and Shankweiler, poor readers are not explicitly aware that spoken and printed words can be segmented into individual phonemes; this makes it hard for them to learn to identify words through alphabetic mapping and letter-sound synthesis, or what is termed phonetic decoding. Poor phoneme segmentation is said to be a manifestation of a more general problem in phonological coding, characterized by the storage in memory of impoverished representations of letter sounds and word names.



TYPICAL MIRROR WRITING is displayed in a note from a five-year-old boy to "Grandmum." This child's development was not normal: when seen at 11, he was dyslexic, according to the English investigators Macdonald Critchley and Eileen A. Critchley.

Such a dysfunction could theoretically lead to difficulty not only in learning the sounds associated with given letters and combinations of letters but also in learning the names of printed words as whole entities. Words are therefore stored without complete phonological codes—file cards, in the library model. Asked to call up the proper word, the child finds that he or she has not retained enough clues to the name of the word.

Results of other studies done in our laboratory and elsewhere support the concept that deficiencies in alphabetic mapping and phonetic decoding are major factors in reading difficulties. The studies show consistently that severely impaired readers are much less proficient than normal readers in learning to use letter sounds to decode pseudowords (meaningless wordlike letter assemblages used in testing) and words they have never seen before. Such deficiencies seem to be the result of a poor grasp of phoneme values. It has also been found that kindergarten and first-grade children who have some ability to segment spoken words into syllables and phoneme-size units learn to read better than children who have little or no such ability. Perhaps the most impressive support comes from studies showing that children trained to identify phonemes have an increased ability to map alphabetically and therefore an enhanced capacity to identify printed words.

If relative lack of awareness of phonemes and deficiency in phonetic decoding are rooted in more basic difficulties in phonological coding, one might expect to find that poor readers have trouble remembering words they hear. This turns out to be the case. In a large number of studies done in our laboratory, at the Haskins Laboratories and elsewhere, poor readers did less well than normal readers when they were asked to recall lists of words they had just heard.

A number of investigators—notably Martha B. Denckla and the late Rita G. Rudel of the Columbia University College of Physicians and Surgeons—have also found that dyslexics tend to be slower and less accurate than normal readers not only in naming letters and words but also in naming common objects, colors and numerals. The performance of many dyslexics in these studies was often characterized by severe blocking: circumlocutions, long hesitations and such substitution errors as saying "dog" when confronted with a picture of a cat.

Deficiencies in vocabulary development and semantic ability in general also seem to make the identification of words difficult, as several studies have suggested. A deficiency in syntactic competence may be yet another factor. Studies in our laboratory and elsewhere show that poor readers seem to be less proficient than normal readers in comprehending sentences, particularly those that are syntactically complex; in making use of inflectional morphemes (such as *-ed* and *-ing*) to specify such things as tense and number; in distinguishing between grammatical and ungrammatical sentences; in using complex sentences in a grammatically correct way, and in making fine distinctions among abstract words, particularly such "noncontent" words as *if, but* and *their*.

In fact, considerable evidence suggests that poor readers are more deficient (compared with normally developing readers) in their ability to identify noncontent words than they are in their ability to identify content words such as *dog* or *cat*. Poor readers also seem to have difficulty using sentence context to help them identify printed words. It should be pointed out, however, that a causal connection between reading disability and deficiencies in processing the semantic and syntactic attributes of printed words has not yet been established.

The possibility that dyslexics may be impaired by deficiencies in language raises the question of whether or not they are basically impaired in auditory processing. One possibility is that their auditory sensory, or echoic, memory is deficient, which would mean the auditory trace is dissipated faster in poor readers than in normal readers. This possibility has been evaluated and dismissed by Randall W. Engle and his associates at the University of South Carolina. We have obtained similar results.

A second possibility is that poor

readers are generally limited in their ability to store acoustic information in permanent memory. Susan Brady of the Haskins Laboratories evaluated this question by comparing the ability of dyslexic readers and of normal readers to remember verbal and nonverbal information (words and environmental sounds). They found that dyslexics performed below the normal readers only on the verbal-memory tasks. In terms of our memory model such results indicate that poor and normal readers are equally capable in initial-stage auditory processing and that the poor readers do not sustain any generalized deficiencies in memory for material presented auditorily. Poor readers do, however, appear to be deficient in their ability to retrieve linguistic representations stored in long-term memory. Such results seem to be consistent with linguistic-coding theories of reading disability.

Several other hypotheses propose that the cause of dyslexia lies in nonlinguistic functions, but none of them is very persuasive. One, the "attention deficit" hypothesis, relates difficulty in reading to a generalized inability to concentrate and pay attention. Some workers have found evidence associating this pattern with physiological abnormalities. Children exhibiting it, however, have difficulty with subjects other than reading and may not be representative of those whose problems are limited to reading.

Another theory relates dyslexia to deficits in "cross-modal transfer," or an inability to relate stimuli perceived through one sensory system to stimuli perceived through another system. The theory suffers both from lack of experimental support and from logical consistency; it would seem improbable that a child whose intelligence is average or above average (as is true of dyslexics) could suffer from cross-modal-transfer deficits, given the degree of cross-modal learning required to score in at least the average range on any test of intelligence.

Some other theories deserve mention. One posits deficiencies in associative learning. Another argues that dyslexics have trouble detecting patterns and learning "invariant relationships," such as the rules that govern alphabetic mapping or number concepts. Again, both theories stumble on the unlikelihood that an individual who scores well on intelligence tests could suffer from such pervasive handicaps. Our own studies show that poor readers who do not do well on tests of these abilities were limited by their linguistic-coding ability. When the tests did not rely on linguistic coding, the poor readers were able to improve their performance on association and rule-learning tasks.

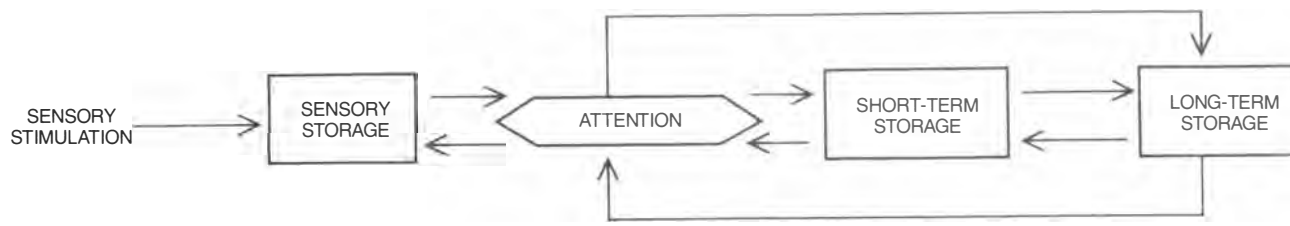
For example, in one such study we asked a group of poor and normal readers to learn to associate pairs of novel visual symbols with two-syllable nonsense words; each symbol always represented the same nonsense syllable [*see illustration on page 40*]. The child's task was to learn to say the two-syllable nonsense word when presented with the pair of symbols representing that word. To help remember the whole word the child was told to try to remember the individual symbol that represented a given syllable. After practice on this task the symbols were rearranged; the child was then shown the rearranged set of symbols to see if he or she could transfer, or generalize, the symbol-syllable units learned initially to associate the rearranged pairs. This is analogous to learning letter sounds in *cat, ran* and *fan* and relying on the sounds to decode "new" words such as *fat, rat* and *can*.

A second group of poor and normal readers was given a similar transfer-learning task, but instead of learning visual-verbal associates these subjects learned to associate and transfer visual pairs; again one of the symbols in a stimulus pair always represented one of the symbols in a response pair. One significant finding from this study was that poor readers did less well than normal readers on the visual-verbal learning tasks. The implication was that they had difficulty with both initial-association and transfer learning because they were impaired by inability to remember the nonsense syllables, not by inability to associate or generalize. This conclusion was verified by a second finding, which was that poor readers did as well as normal readers on the visual-visual association and transfer-learning tasks. Poor readers, then, appear to have difficulty with association and rule-learning tasks only when the tasks require them to store and retrieve the auditory representation of words and syllables.

I come, finally, to what might be called the serial-deficit theory. It holds that an inability to remember the sequence of a series of items or events underlies dyslexia. The theory presumes that the brain is equipped with a ubiquitous ordering ability, a supposition that strikes me as highly unlikely. I suspect instead that different cognitive systems have their own rules and algorithms for establishing order and sequence. If this is the case, then such theories are ruled out.

A number of investigators have taken an approach somewhat different from the ones discussed so far. They suggest that reading disability may be associated with a number of different neurologic disorders, each of which underlies one or another of the basic processes involved in learning to read. Such a view implies that there is an array of neurologic disorders characterized respectively by visual deficits, language deficits, deficiencies in cross-modal transfer and so forth. Although reading disability may result from a number of different factors, the cause in an otherwise normal child would seem to be more circumscribed. As I have argued, we believe the problem lies in the linguistic domain. The question remains open, however.

Our research and the work of investigators elsewhere has called into question other perceptions of dyslexia. First, it is important to point out



INFORMATION-PROCESSING MODEL of the stages of memory and their interrelations is diagrammed. A literal copy of a visual or auditory stimulus is held in sensory storage briefly. If the subject attends to the stimulus, it enters a short-term memory system, where it is encoded into a representation appropriate for storage in long-term memory; a stimulus that is not attended to is not encoded and is lost from memory. The long arrows suggest some of the interactions among the memory systems. For example, a person is more likely to attend to a familiar stimulus because a representation of it has been filed in long-term memory.

that there are no well-defined reading behaviors that can clearly distinguish a dyslexic from other poor readers whose difficulties stem, for example, from limitations in experience; nor are there distinguishing clinical patterns. All poor readers have difficulty learning to identify and spell printed words, but not all would qualify as dyslexics—if the term dyslexia is used to define a very specific reading disability in an otherwise normal child. Moreover, the reversal errors said to be characteristic of dyslexics account for no more than between 20 and 25 percent of their reading errors, most of which are generalizations promoted by an imperfect knowledge of linguistic associates (such as *cat* for *fat, cat* for *kitty, bomber* for *bombardier*).

Moreover, reversal errors can be plausibly explained without invoking spatial confusion. If, for example, a child attempts to remember the words *pot* and *top* only as whole entities and does not know the sounds of the individual letters, there is a stronger inclination to reverse them. We have verified this in experiments in which two groups of dyslexic and normal readers (in the second and sixth grades) learned to identify pseudowords constructed from a novel alphabet, which were designed to prompt reversal errors of the *was/saw* type. Children taught to identify these pseudowords by a whole-word method made many more reversal errors than children who were taught to use alphabetic mapping, but the poor readers made no more reversal errors than the normal readers. It seems clear that the spatial-confusion interpretation of reversal errors is incorrect.

The clinical significance of mirror writing is also commonly misunderstood. Some degree of mirror writing can be observed in normally developing readers as well as in poor ones. The tendency is quite likely a vestige of an earlier stage of development, which some poor readers take more time to transcend. It persists in these children, I think, because they find it difficult to remember both the visual-linguistic clues and the clues provided by sentence context that foster accurate judgment about the relative positions of words and letters and the direction in which they run. What causes the habit of mirror writing to persist is a lack of correct practice in writing and spelling that actually results from a child's reading problems. I suggest, in other words, that when mirror writing is observed in poor readers, it is a consequence of their reading difficulty rather than a benchmark of visual-spatial confusion.

Another common misconception is that reading problems are caused by perceptual deficits associated with motor and visual-motor defects or with ocular defects other than loss of visual acuity. If deficiencies in motor and visual-motor development or defects in eye movements caused perceptual impairment and reading problems, one would be at a loss to explain how so many children with cerebral palsy and various visual-tracking defects become literate.

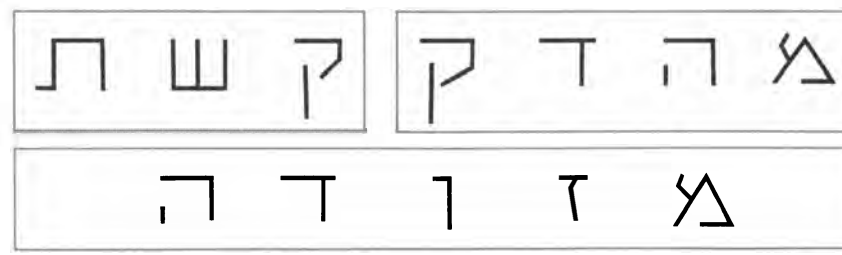
Still another misconception is that dyslexia is more prevalent in countries where the writing systems are based on an alphabet than it is in countries where the writing systems either are pictographic or are phonetically less complex. A study done by Harold W. Stevenson and his colleagues at the University of Michigan has yielded some evidence against this idea. The investigators evaluated schoolchildren in comparable cities in the U.S., Japan and Taiwan. The three groups were compared on a large battery of tests measuring school achievement as well as language and cognitive ability. The results yielded no evidence that the writing systems of Japan and China preclude reading disability. The fact that the Japanese and Chinese languages respectively contain characters representing syllables and entire words seemed to make no difference.

Since dyslexia appears to be commonly associated with brain dysfunction and the brain's ability to store and retrieve information, it is necessary to consider whether constitutional factors—genetic and/or neurologic—contribute to the condition. There are actually no diagnostic criteria that enable one to distinguish clearly between constitutionally and experientially derived origins of reading disability. There is one highly suggestive piece of evidence, however: boys who are impaired in reading outnumber girls who are so impaired by ratios ranging from 4 : 1 to 10 : 1.

Taken together with results from developmental studies showing that boys are in general less capable than girls on language and language-related tasks, such ratios could be taken as support for both constitutional and language-

REAL WORDS		
THREE-LETTER	FOUR-LETTER	FIVE-LETTER
was	**loin**	**blunt**
SCRAMBLED LETTERS		
THREE-LETTER	FOUR-LETTER	FIVE-LETTER
dnv	**jpyc**	**ztbrc**
NUMBERS		
THREE-DIGIT	FOUR-DIGIT	FIVE-DIGIT
382	**4328**	**96842**

VERBAL AND NONVERBAL STIMULI were presented for half a second to poor and normal readers in the second and sixth grades; examples of each are given here. In one phase of the experiment subjects were asked to write down the words, scrambled letters or numbers from memory; in a second phase they were asked instead to name each character of a stimulus in the right order—in the case of the words, after pronouncing them. Poor readers did about as well as normal readers on the copying task but not on the naming; their problems seem to arise from deficiencies in verbal, not visual, processing.



HEBREW WORDS in simplified block form were presented one at a time to three groups (second through sixth grade): poor readers and normal readers unfamiliar with Hebrew and children currently studying it. Three-, four- and five-letter words (sampled here) were shown for three, four and five seconds respectively. Subjects had to reproduce the words on paper from memory. Among those unfamiliar with Hebrew, poor readers did as well as normal readers. (Neither, of course, did as well as children studying Hebrew.)

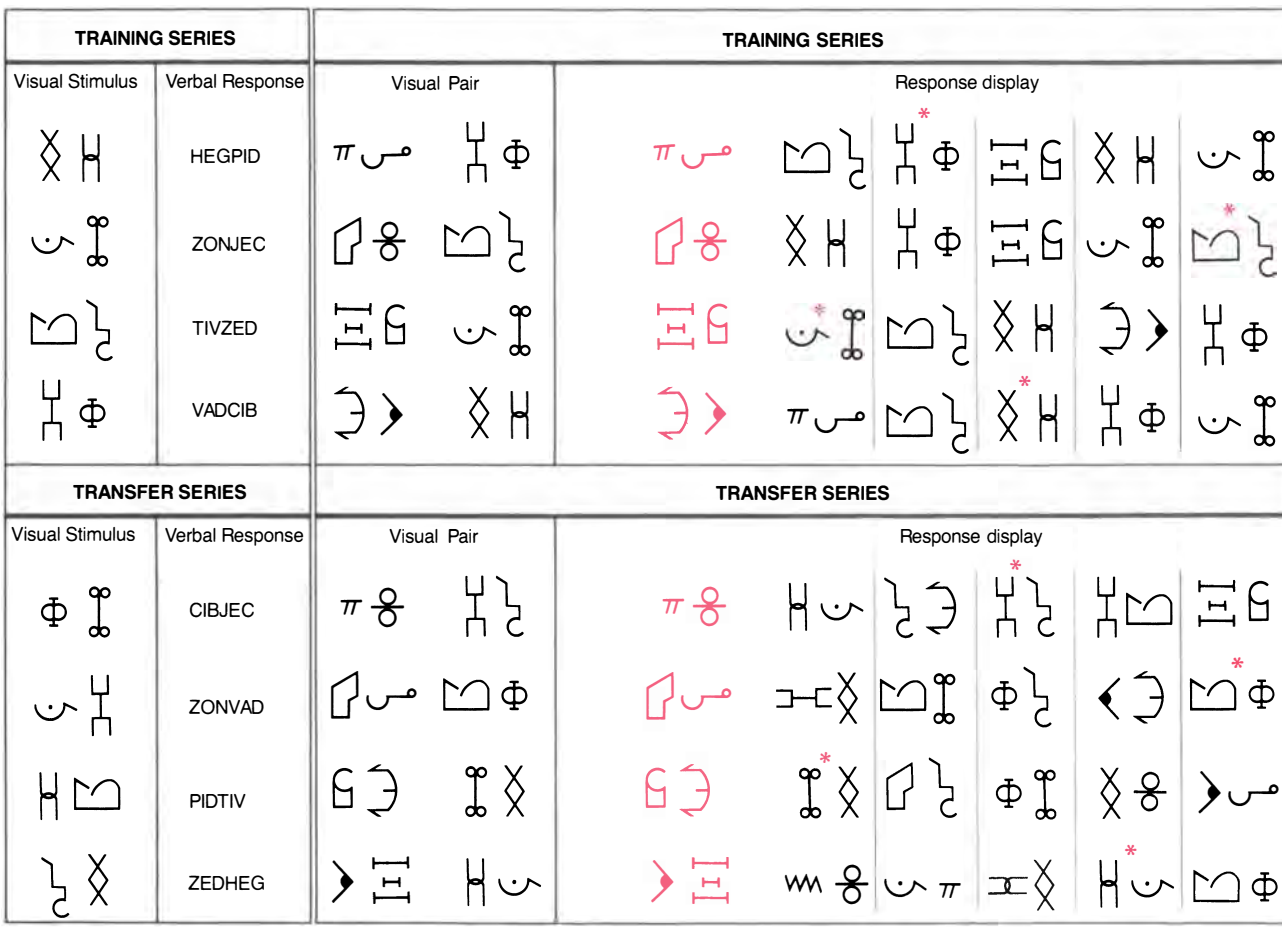
deficit theories of dyslexia. Boys may be either genetically less well endowed with linguistic capabilities than girls are or more vulnerable to neurologic defects affecting language development than girls are. Then, if it is true that reading disability is caused by limitations in language ability, constitutionally derived language deficits would result in a higher incidence of the disorder in boys than in girls.

Support for a genetic basis for dyslexia comes from a small number of familial and twin studies carried out over the years. The early studies in the literature were poorly controlled, but they consistently showed that reading disability occurs more often in near relatives than in the population at large, that reading disability occurs more often in twins than in siblings and that reading disability has a much higher concordance rate in monozygotic (identical) twins than in dizygotic twins. These findings have been verified recently in a more highly controlled study conducted by John C. DeFries and his associates at the Institute for Behavioral Genetics of the University of Colorado at Boulder.

Perhaps an even more exciting finding, from research done by the Boulder group with Shelly D. Smith, is the tentative localization of a particular gene on chromosome 15 in members of families in which there is a history of reading disability. Once a gene that may be responsible for a specific attribute has been localized on a specific chromosome, geneticists are in a position to find the mechanisms whereby the gene gives rise to the attribute. The finding could be a significant breakthrough in the study of dyslexia, but it has not yet been replicated.

A number of investigators of brain function have also begun to study the etiology of dyslexia, and their early findings are promising. A Boulder team headed by David W. Shucard compared dyslexic and normal readers on measures evaluating electrophysiological responses to auditory and visual stimulation. The group's major finding has been that electrical activity in response to reading is characterized in dyslexics by greater amplitude in the left hemisphere than in the right hemisphere, whereas the opposite pattern emerges in normal readers.

A novel technique recently exploited in this area of inquiry is "brain electrical activity mapping" (BEAM), developed by Frank H. Duffy and his colleagues at Children's Hospital in Boston. The BEAM technique produces topographic maps that are based on brain functions, not brain structures. Duffy and his associates have now obtained evidence that left-hemisphere functioning in dyslexics is qualitative-



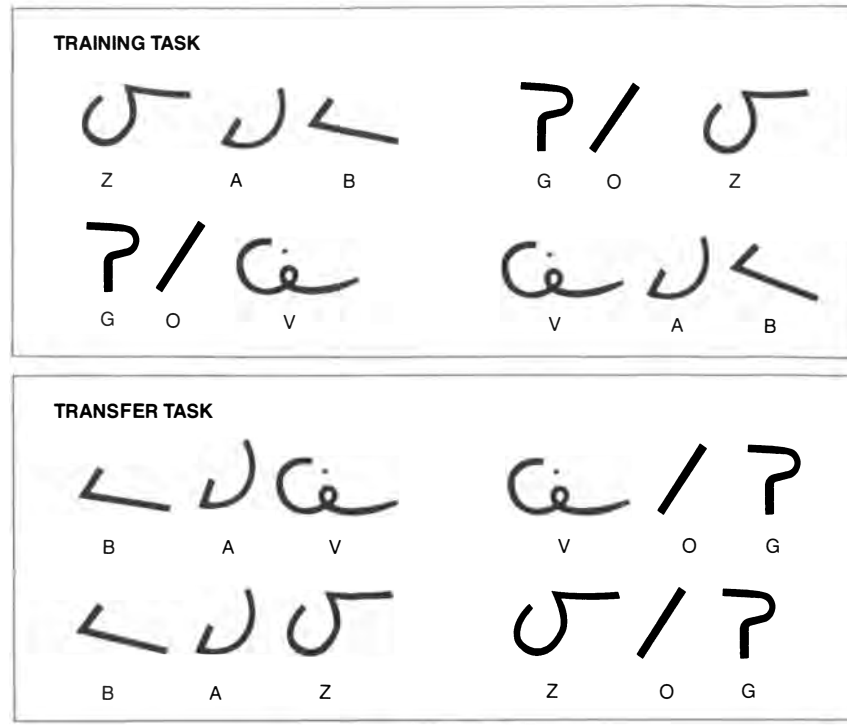
RULE-LEARNING TASKS were given to poor and normal readers in the fourth, fifth and sixth grades. The visual-verbal task (*left*) involved novel visual symbols, each representing a syllable. Subjects were trained (*top*) to match each pair of symbols (stimuli) with the correct response, a two-syllable nonsense word. Then they were shown a transfer series (*bottom*), in which the symbols and words were reordered, and were asked to say the new words. In the visual-visual task (*right*) visual responses were substituted for the verbal ones. Now each stimulus-response pair was a pair of two-symbol "words." The response display presented the stimulus (*color*); the subject was trained to select the correct response from among a series of five pairs. The colored asterisks designate the correct responses. Again the symbols had been reordered in the transfer series, but the correspondence between individual stimulus and response symbols was maintained. The poor readers failed to perform as well as the normal readers only on the visual-verbal task, which suggests that they had difficulty learning rules only when the experiment required them to make a verbal response.

ly different from that in normally developing readers. The differences were particularly prominent in adjacent regions of the left parietal and temporal lobes, areas of the brain known to support speech, language and related linguistic activities.

Finally, I should mention the neuroanatomical studies conducted by Albert M. Galaburda and his colleagues at the Harvard Medical School. Two types of anatomic anomalies were disclosed by postmortem analysis of the brains of several male dyslexic subjects. First, there was a consistent absence of the standard pattern of brain asymmetry in language regions: atypically, the left-hemisphere regions were no more highly developed than the right-hemisphere regions. Second, in the cerebral cortex of the language-related areas there were multiple sites where the microarchitectural arrangement and position of neurons, or nerve cells, was distorted, particularly in the left hemisphere.

Since the absence of asymmetry resulted from the excessive development of right-hemisphere areas that are normally smaller, Galaburda has argued that both the anatomical anomaly and the anomalous neuronal architectures may reflect interference with the normal developmental process whereby undesirable neurons and their connections are eliminated. Presumably the oddity in microanatomical organization of the language areas of the brain would explain some linguistic deficits seen in the reading disorder.

The late Norman Geschwind hypothesized that the variations from the standard pattern of cerebral lateralization observed by Galaburda were significantly linked to disorders involving an immune dysfunction. He suggested that common developmental mechanisms acting during fetal life might lead to the abnormal development of parts of the immune system and also to anomalous asymmetrical development of the brain; he proposed an important role for the male sex hormone testosterone in these mechanisms. Although testosterone was postulated to interfere with the normal development of language areas, Geschwind also suggested that it may lead to the superior development of certain brain areas involved in spatial visualization and visual-motor coordination. If this suggestion is verified, the failure of investigators to obtain evidence for visual-deficit theories of dyslexia would be at least partly explained.



PSEUDOWORDS were shown to two groups of second- and sixth-grade children (both poor and normal readers) who had been given a week's intensive instruction in reading by one of two methods: training in "alphabetic mapping," designed to make them sensitive to the sounds of individual letters, or training in "whole-word meaning," in which pictures of imaginary animals were associated with such nonsense syllables as "zab." For the experiment both groups were shown pseudowords (*top*), made up of novel alphabetic characters, representing the nonsense syllables presented in the whole-word-meaning training. Children learned to say the right nonsense word when it was presented with its pseudoword—just what a child learning to read new words must do. In essence, the alphabetic mappers had to learn to read the pseudowords without benefit of meaning; the children trained in whole-word meaning had to learn to read them without benefit of alphabetic mapping. After several learning trials, subjects were presented with the same characters in reverse order (*bottom*) and asked to learn the names of the new pseudowords. Both the poor and the normal readers who had been trained in alphabetic mapping made very few reversal errors, whereas children trained in whole-word meaning made many such errors.

The nonlinguistic theories of dyslexia discussed here have each given rise to remedial techniques designed to correct the cognitive deficits implicated by the theories. For example, visual-deficit theories have spawned remedial approaches designed to improve visual perception, such as optometric training to facilitate binocular coordination, eye tracking and so on. Similarly, cross-modal and serial-memory theories have given birth to a variety of remedial exercises designed to improve these functions, presumably through the direct stimulation of certain brain centers.

If the logical and empirical arguments against the theories supporting these approaches are valid, then the approaches themselves must be seriously questioned. The fact is that research evaluating the efficacy of these approaches for the remediation of reading disabilities has produced no convincing evidence to support them—and a considerable amount of evidence against them. In any case, not enough is yet known about how the brain works to enable anyone to devise activities that would have a direct and positive effect on neurologic functions responsible for such basic processes as visual perception, cross-modal transfer and serial memory.

More conventional approaches to remedial instruction have had greater success, particularly in educational settings equipped to provide dyslexics with the type and amount of help they need. We have found that early remediation of reading difficulties is indicated. It should be based on intensive one-to-one tutoring and a balanced reading program—one that makes generous use of both the holistic/meaning and the analytic/phonetic approaches. The training in reading should be supplemented with enrichment activities to foster language development. Such a program can help a child to develop functional and independent reading skills and so remove him or her from the disabled list. A consensus is emerging among investigators that there is no substitute for direct remedial instruction in reading.