Reviewed are three types of etiological theories of dyslexia: theories of visual perceptual deficit, theories of difficulties with sensory integration, and theories of verbal deficiencies. The author concludes that recent findings weigh heavily against perceptual deficit theories of reading disability as suggested by S. Orton and others; that the equivocal findings relating to the sensory integration theory of H. Birch suggest the need for further research; and that the author's research and that of others support the verbal or linguistic deficit theory. Noted are practical implications of the findings such as the importance of including all (graphic, phonologic, semantic, and syntactic) aspects of words and sentences in remedial reading instruction. (DB)
Psychological Factors in Reading Disability

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Dyslexia is a medical term referring to disorder in reading, presumably due to some form of neurological dysfunction. It is also known as specific reading disability and the terms are employed interchangeably. The literature dealing with the problem, is uniform in its suggestion that dyslexia is an intrinsic developmental anomaly, the etiology of which is qualitatively different from reading difficulties arising because of extrinsic or environmental factors. The fact remains, however, that dyslexia is not a well defined entity and its characteristics are not easily distinguished from any other form of reading deficiency. Indeed, some believe that there is little point in attempting to differentiate neurologically based reading disorder, from reading problems caused by other factors, especially since its remedy lies almost exclusively upon re-education and other behavioral treatment methods (International Reading Association, 1972).

Yet in spite of this attitude, one, that from a practical standpoint is reasonable and justified, we are puzzled by children, apparently normal in other respects, who have inordinate difficulty learning to read. Such children have attracted the attention of researchers as well as practitioners, and prompted them to circumscribe their definition of the disorder to exclude probable extrinsic causes, and consider the possibility of basic developmental deficiency in the population so defined. Thus it is suggested that dyslexia occurs in the child with average or
above intelligence, who sustain no peripheral sensory deficits, severe brain damage or other debilitating physical problems, who has not been hampered by serious emotional and social disorders, or by cultural disadvantage, and who has had adequate opportunity to learn (Rabinovitch, 1958; Johnson and Myklebust, 1967).

With respect to their reading behaviors, dyslexics are described as children who have unusual difficulty identifying words as wholes, as well as in segmenting them into their component sounds. They also have apparent difficulty abstracting and generalizing the common constituents of given words, and are inclined to treat those (words) containing redundant elements (cat vs fat) as discrete entities. In addition, dyslexics are said to be characterized by a prolonged tendency to make orientation and sequencing errors in reading and written language (e.g. calling b/d or was/saw). Such children also tend to be poor spellers, and their written language is markedly deficient in all respects.

There are also a number of other characteristics, not uniformly apparent in dyslexics, but which are said to occur often enough to be examined for their possible significance (Bryant, 1965; Critchley, 1971). Among those mentioned in the literature are the following: (1) boys are observed to have reading problems more often than girls; the ratio generally exceeding 4:1 (Eisenberg, 1966); (2) frequently, there is a significant incidence of reading difficulties in the families of dyslexics (Hermann, 1959; Hallgren, 1950); (3) there may be apparent difficulty in other forms of representational learning, such as telling time, naming the months and seasons of the year, left-right identification, etc.; (4) the occasional appearance of neurological "soft" signs such
as abnormal reflexes, or minor coordination problems, and (5) not infrequently, a history of developmental problems, particularly in one or more aspects of language (Kawi and Pasamanick, 1954; Lyle, 1970).

It should be apparent that the criteria delimiting dyslexia are not very definitive with respect to its nature and origin. Yet there is enough suggestive evidence from research done to date, and from clinical practice, to warrant further study of the problem. The remainder of this paper will be devoted to a brief outline of current conceptualizations of the etiology of specific reading disability. Relevant research will be reviewed and integrated with the results of research issuing from our own laboratory. Each of the studies we will mention has employed the foregoing criteria of dyslexia, as operationalized in standardized tests of intelligence and reading ability, as well as in screening measures to control for extrinsic causes of reading difficulty. Thus the results are generalized only to this population.¹

The literature in the area of reading disability has provided us with three major theoretical viewpoints. By far the most popular of these is the suggestion that reading disability is caused by visual-spatial confusion stemming from neurological disorder. This position was given initial impetus by Orton (1925, 1937) who attached particular significance to the orientation and sequencing problems observed in letter and word identification (e.g., b/d, was/saw). Such disturbances were thought to be a manifestation of delayed development of laterial dominance, resulting in the failure to suppress "mirror images" of visual events, believed to be stored in each of the hemispheres. Several variants of Orton's hypothesis have appeared subsequently, but all
have in common, the view, that dyslexia is primarily the result of visual organization and memory problems. Thus, the disorder has been attributed to inherited directional confusion (Hermann, 1959); figure ground difficulties (Bender, 1959; Birch, 1962); dysfunction in visual analysis and synthesis (Birch, 1962); perceptual motor problems (Kephart, 1960; Cruickshank, 1968; Frostig, 1967); and optical deficiencies (Getman, 1962; Anapolle, 1967).

The perceptual deficit hypothesis has enjoyed a surprising longevity in spite of the fact that research evidence supporting it is, at best, meagre. Benton (1962) makes note of methodological weaknesses and conflicting results in most of the studies appearing in the literature, and concludes that deficient form perception and impaired directional functioning are not significant correlates of reading disability. However, he allows for the possibility that perceptual problems may exist in children, younger than those employed as subjects in the investigations reviewed (i.e., 9 years and above). He also suggests that reading problems in older children are, most likely, associated with dysfunction in some aspect of "verbal mediation."

Some research recently completed in our own laboratory supports Benton's suggestion that reading difficulties may be associated with verbal mediation problems rather than visual-spatial disorder. In an unstratified sample of children between the ages of 9 and 14, it was found (Vellutino, Steger and Kandel, 1972) that poor readers performed considerably better in the visual reproduction of 3, 4 and 5 letter words, presented tachistoscopically, than they did in pronouncing those same words. In addition, their performance was comparable to that of
normal readers on the reproduction task, except for those configurations which taxed short term visual memory (i.e., the five letter words). Yet, the poor readers pronounced and spelled all of the stimulus words less accurately than the normals.

In order to assess Benton's (1962) suggestion that poor readers at younger age levels may sustain perceptual disorder, a sequel to the above study (Vellutino, Smith, Steger and Kaman, 1974) compared the performance of poor and normal readers at ages 7 and 11 respectively. The major findings of the previous investigation were replicated in this study, the results of both clearly indicating that the visual perception of a word does not necessarily parallel its oral encoding. This was especially evident in the fact that poor readers in both studies copied correctly, even those words on which they made a large number of apparent spatial and sequential errors in oral reading (e.g., din/bin; cob/cod; sung/snug; lion/loin). Thus it appears that the positional inaccuracies, so often observed in the reading and written language of poor readers may, in fact, be linguistic intrusion errors rather than visual spatial distortions. In simpler terms, our results suggest that when dyslexics call a "b" "d" or "was" "saw," it isn't because they perceive ("see") these items differently than normal readers, but because they can't name them correctly. A similar conclusion was reached on the basis of research done elsewhere (Liberman, Shankweiler, Orlando, Harris, and Berti, 1971).

Additional support for the suggestion that poor readers sustain no basic disorder in visual perception and visual memory is provided by the results of several other studies conducted at the Child Research Center.
In two separate investigations (Vellutino, Fruzuk, Steger and Meshoulam, in press; Vellutino, Steger, Kaman and DeSetto, in press) poor readers performed as well as normals in the immediate visual recall of varying length words printed in Hebrew, an unfamiliar orthography. However, the performance of both groups was inferior to normal readers learning to speak, read, and write Hebrew, owing to the latter children's familiarity with both the orthographic and linguistic characteristics of the stimuli presented. In another study, employing a similar format (Vellutino, Steger, DeSetto and Phillips, in press), poor and normal readers were comparable in the visual recognition of randomly arrayed Hebrew letters, presented immediately after initial exposure as well as twenty-four hours and six months later. In this investigation, as in the others, the non-Hebrew groups did not perform as well as the Hebrew groups. Thus, it would appear that both short and long term visual memory are comparable in poor and normal readers, an inference which can be generalized to both younger and older children, since our samples in two of the studies (Vellutino, Steger, DeSetto and Phillips, in press; Vellutino, Steger, Kaman and DeSetto, 1974) were stratified at the second, fourth and sixth grades.

Finally, there is a substantial body of evidence that reading disability is not attributable to optical deficiencies as suggested by some authors (Getman, 1962; Anapolle, 1967). Lawson (1964) in reviewing a large number of studies spanning a period of over seventy-five years, observed that the incidence of visual difficulties was no greater in poor readers than in normals. He thereby concluded, that specific reading disability is a symbolic learning disorder caused by central rather than
peripheral dysfunction. These findings coupled with the results of the studies cited above, seriously undermine the perceptual deficit theories of reading disability, and strongly suggest that the origin of the problem will be found elsewhere.

The second most popular explanation of specific reading disability appearing in the literature, is the suggestion that the disorder is caused by deficient integration of the sensory systems. This hypothesis was initially proposed by Birch (1962), and later given research support by Birch and Belmont (1964), as well as by several other studies which appeared subsequently (Muehl and Kremenak, 1966; Beery, 1967; Zurif and Carson, 1970). In all these investigations, poor readers were less accurate than normal readers in matching simple rhythmic patterns with their visual representations. Similar results were obtained in a number of other studies (Senf, 1969; Bakker, 1970; Zurif and Carson, 1970) reporting significant differences between poor and normal readers in the temporal organization of auditory and visual stimuli, presented simultaneously. However, Blank and her associates provide an alternate explanation of results supporting the intersensory deficit hypothesis. In two separate experiments (Blank and Bridger, 1966; Blank, Weider and Bridger, 1968), it was found that poor readers' difficulties in sensory matching and temporal ordering tasks were due to their limited ability to employ a verbal coding system in the serial organization of stimuli presented to them, as compared with normal readers who were apparently more effective in utilizing verbal mnemonics to aid recall. The authors conclude, from these findings, that reading disability may be attributable to verbal concept deficiencies rather than dysfunction in cross-modal transfer.
We might also point out, with respect to the theory in question, that in none of the studies finding reader group differences in intersensory functioning was there any attempt to control for the possibility of group differences in intrasensory functioning. Thus, in two investigations (Zigmond, 1966; Vande Voort, Senf and Benton, 1969), it was found that poor readers were inferior to normals on auditory-auditory as well as auditory-visual integration tasks (i.e., matching and association), although comparable to the normals in visual-visual integration. And in our own study of the problem, we found that poor and normal readers who were equivalent in paired associates learning within given modalities, were also comparable in learning between modalities (Steger, Vellutino and Meshoulam, 1972; Vellutino, Steger and Fruzek, 1973).

It has also been suggested by some authors that reader group differences in sensory integration may be due to attention and memory factors (Senf and Freund, 1971; Vande Voort and Senf, 1973), but the possible nature of such disorder was not made explicit.

It is apparent that research findings in support of the intersensory deficit explanation of dyslexia are, at best, equivocal. Yet in spite of conflicting results, it would be premature to reject this theory and continued research in the area is certainly warranted.

The third and final hypothesis, to be discussed, is the suggestion that reading disability is specifically associated with verbal learning deficiencies. Several variations of this idea have appeared in the literature, each relating to somewhat different aspects of linguistic functioning. For example, Rabinovitch (1954, 1959, 1968) suggests that
dyslexics are characterized by subtle language defects which can be observed not only in poor reading ability, but also in expressive language problems, word finding difficulties, deficient concept formation, and difficulties in symbolic learning generally.

Research supporting Rabinovitch's suggestion is provided by a number of studies (e.g., Neville, 1961; McLeod, 1965; Belmont and Birch, 1966; Lyle and Goyen, 1969) comparing poor and normal readers on measures of verbal and non-verbal "intelligence." In all of these investigations, poor readers were significantly below the normals on the WISC Verbal subtests, and were found to be particularly deficient on measures of verbal expression and categorization. In contrast the groups were comparable on the non-verbal (Performance) subtests. That these results were not simply the cumulative effect of prolonged reading disability is suggested in the finding of one study (Lyle and Goyen, 1969) that differences between reader groups at the first grade level were of the same magnitude as differences at sixth grade.³

Two other studies (Fry, 1967; Schulte, 1967) attempted a more refined analysis of the oral language productions of poor and normal readers. In carefully selected samples of second graders, normal readers were found to be linguistically more sophisticated than poor readers, as manifested in greater verbal fluency, larger speaking vocabularies, better organizational and integrative skills, more abstract usages, and syntactic differences in sentence structure. The authors, in a later review, (Fry, Johnson and Muehl, 1970), infer no cause-effect relationship between oral language patterns and reading disability. However, they suggest that such deficiencies as those observed could
impair both word recognition and comprehension by limiting the number and variety of (verbal) labels and mediators available for learning grapheme-phoneme associations, and for abstracting meaning from running text. The research by Blank and her associates mentioned earlier (Blank and Bridger, 1966; Blank, Weider and Bridger, 1968) supports this suggestion, in that poor readers were differentiated from normal readers in integrating spatial and temporal patterns as a result of verbal labeling problems.

Additional evidence for linguistic deficiencies in poor readers is derived from a longitudinal study by de Hirsch, Janisky and Langford (1966) in which pre-schoolers, sustaining a variety of language deficits, were found later to have reading problems. However, these data can only be suggestive, since there was no control for the early school experience of the subjects in the sample; thus, it is difficult to be certain of the degree to which subjects received adequate reading instruction.

The studies discussed thus far have focused upon semantic and syntactic deficiencies as possible causes of reading disability, but some authors have suggested that dysfunction in phonemic analysis (i.e., analyzing the sounds in words) may contribute to this disorder. For example, Shankweiler and Liberman (1972), found that poor readers made more errors in reading given words than they did in repeating the same words read to them; and, further, that the types of errors made in each instance differed. In reading, most of the errors were made in the medial and final positions, and more often on vowels than on consonants. However, in oral repetition of the words, errors were evenly distributed
across the respective positions, and fewer errors occurred on vowels than on consonants. It was also found that poor readers substituted "real" words for nonsense syllables, they were asked to read, more often than the normal readers. The authors suggest, on the basis of these findings, that phonemic segmentation in speech perception is quite different from phonemic segmentation in decoding written language, and that poor readers may not have developed a conscious awareness of this distinction. As a result, they are inclined to treat all words as unit syllables, which, as the authors point out, becomes problematic, considering the orthographic and phonetic complexities involved in mapping alphabetic symbols to sound. A similar suggestion is made by a number of other authors (e.g., Mattingly, 1972; Savin, 1972; Rozin, Poritsky, and Sotsky, 1971), although none were specifically concerned with the population being considered herein.

Parenthetically, we might also note, the work of Wepman (1960, 1961) who suggests that reading difficulties, in some poor readers, may be the result of maturational differences in the discrimination of speech sounds. Such problems, if they exist in this population, may be more basic than the difficulties in phonemic analysis, described by Shankweiler and Liberman (1972). However, the evidence supporting this possibility is not impressive (Vernon, 1971; Vellutino, DeSetto and Steger, 1972).

Support for a possible relationship between verbal learning problems and reading disability is also provided by a large number of studies investigating paired-associates learning, in poor and normal readers. We may synthesize research findings (Brewer, 1967; Zigmond, 1966) by pointing out, that in most of the studies conducted, these two groups
were more often differentiated on associative tasks involving verbal components, than they were in the learning of non-verbal relationships. Similar results were obtained in studies recently completed in our laboratory (Vellutino, Harding, Phillips and Steger, in press; Vellutino, Steger, Harding and Phillips, in press). Of particular interest is our observation (Vellutino, Steger, Harding and Phillips, in press) that poor readers, in learning to associate novel visual stimuli with pronounceable nonsense syllables, were inclined to substitute "real" words for these syllables more often than normals. As mentioned earlier, this tendency was observed by Shankweiler and Liberman (1972) and may be a reflection of phonemic analysis problems in poor readers, as suggested by these authors.

A variant of the linguistic deficit view of reading disability is the suggestion of some (Rabinovitch, 1959, 1968; Blank and Bridger, 1966; Blank, Weider and Bridger, 1968) that poor readers sustain basic disorder in verbal concept formation. Such disorder is said to be particularly evident in the poor reader's difficulties in abstracting phonic generalizations (e.g., cat, rat, can → ran). However, the results of a recent study (Vellutino, Harding, Phillips and Steger, in press) suggest that such difficulties are the result of dysfunction in verbal labeling and integration rather than basic disorder in categorical processing. In this investigation, poor readers performed as well as normals on visual-visual association and transfer tasks, but were less proficient than the normals in the initial learning and transfer of visual-verbal relationships similar to those involved in learning to read. Furthermore, covariance analyses controlling for group
differences in (visual-verbal) training eliminated observed differences in transfer, thereby implicating visual-verbal integration rather than categorical functioning as a basic disorder. Thus, it is unlikely that poor readers' difficulties in abstraction and generalization tasks is a result of conceptual disorder in the strict sense.

Finally, a more exotic explanation of reading disability, one also unique to verbal learning, is the suggestion that disturbances in reading and other verbal skills may issue from dysfunction in the transfer of information between brain hemispheres containing visual and verbal associates. Based on the split brain studies of Sperry and his colleagues (Sperry, 1964; Gazzaniga, Bogen and Sperry, 1965) as well as on the clinical findings of Geschwind (1962) studying brain-injured adults, Gazzaniga (1970) suggests that learning problems in some children may be the result of "a disconnected or partially disconnected brain." We know of no studies which attempted to assess inter-hemispheric transfer problems in poor readers and it may be fruitful to do so.

To summarize the foregoing, we have outlined three major etiological hypotheses, in explanation of dyslexia and have presented research findings relating to each. We have seen that, in spite of conflicting results which appeared in the literature (Benton, 1962; Vernon, 1971) recent findings weigh heavily against perceptual deficit theories of reading disability, as suggested by Orton (1925, 1937) and others (e.g., Hermann, 1959; Birch, 1962). Research data relating to the sensory integration hypothesis of Birch (1962) are somewhat more equivocal, and continued exploration of this theory is clearly indicated. In particular, there is need of additional experimentation controlling for possible confound-
ing by virtue of reader group differences in intrasensory and/or verbal encoding ability.

There seems no reason to believe that reading disability is caused by any basic differences between poor and normal readers in paired associates learning (Brewer, 1967). Rather, the evidence suggests that these two groups are most often differentiated when such learning involves a verbal component. In fact these, and research findings cited earlier, are consonant with suggestions, in the literature, that difficulties in reading may be attributable to dysfunction in one or more aspects of verbal learning. That reading problems may be associated with specific language disorder, is an especially attractive hypothesis in view of recent research which indicates that, whatever else reading is, it is a decidedly linguistic function. Indeed, several authors (e.g., Goodman, 1970; Smith, 1971) have made an excellent case for conceptualizing reading as an information gathering process which leans heavily upon linguistic ability, in the general sense. However, given the possibility that reading disability is the result of language disorder, there remains the problem of determining the nature of such disorder and its relationship to the reading process.

As we have seen, the hypotheses advanced have encompassed the semantic, syntactic and phonologic aspects of reading, but there are as yet, no definitive data which give any of these functions etiologic prominence. Perhaps this is because they are not easily separated, either in the chronically impaired reader, or in the fluent reader. For example problems in learning whole words may well reflect specific difficulties in verbal labeling and mediation as a result of semantic and/or syntactic
deficiencies, as suggested. Conversely, select disorder in phonemic analysis may occur in the absence of semantic and syntactic problems, but difficulties in any one of these areas certainly lead to difficulties in the others, owing to their interdependent nature. Thus, the linguistic skills of chronically impaired readers are typically fragmented and unreliable and their reading behaviors may appear to be similar, even though their problems may vary as to basic origin. In contrast, proficient readers, are able to make efficient and economical use of all of their linguistic abilities and it is difficult to be certain of those they employ, or of the priorities they set in deciphering any bit of printed material (Gibson, 1971). Consequently, greater specificity with respect to the linguistic correlates of reading disability is dependent upon refinement of both our conceptualizations and our measuring instruments, but continued research in the area would appear to be a useful course. We might add, in this connection, that language deficits of the types proposed could theoretically accrue, either as a result of extrinsic-experiential factors, or because of intrinsic developmental disorder of neurologic origin. These causes are not mutually exclusive and could interact to obscure basic etiology. However, in our opinion it would be counterproductive for researchers to defer from making such distinctions, especially in view of the possibility that doing so could lead to significant differences in the remediation of children so impaired.

The possibility that reading disability may be a function of deficiencies in inter-hemispheric transfer is, at this point, hypothetical, since the research supporting this idea has been conducted exclusively with adults and is, at best, suggestive. However, the hypothesis merits consideration, and we are currently exploring it with children.
With respect to their practical implications, the research findings strongly suggest that corrective measures issuing from visual deficit explanations of reading disability may have little utility whatsoever. Our own findings are particularly indicting for they suggest that apparent perceptual problems in poor readers are a secondary manifestation of verbal encoding difficulties. Thus visual training and discrimination activities, so highly touted by clinicians and educators of late, would most likely have only the remotest relationship to progress in reading, unless they are designed to help correct specific inaccuracies in word decoding, within the context of remedial reading. These inferences are supported by recent research findings demonstrating no significant relationship between visual-motor training and improvement in reading (Robinson, 1971; Hartman and Hartman, 1973; Goodman and Wiederholt, 1974).

The equivocal nature of research findings relating (inferred) deficiencies in cross-modal transfer, to reading problems, dictates that the use of diagnostic and remedial measures based on this hypothesis is, at best, premature. Yet we would not be surprised to find widespread use of auditory-visual matching tasks among clinicians and educators, given the popularity of the theory in question.

Finally, research data support the possible utility of remedial activity designed to improve the verbal skills of poor readers, but the evidence is yet too scant and tentative to provide us with definitive direction as to degree and kind of such remediation. However, we have little doubt that, whatever the activity, it would be most effective if implemented in direct relationship to specific aspects of the reading
process. Thus discrimination training to improve phonemic analysis may have little transfer value, unless an effort is made to teach the child something of the similarities and differences in spoken and written words (e.g., Shankweiler and Liberman, 1972). Similarly, enrichment activities designed to improve linguistic comprehension and expression in oral language, are bound to help improve reading in a variety of ways, but such activity would no doubt be more effective if integrated with other remedial procedures designed to correct specific reading skills deficiencies.

Perhaps the most significant factor emerging from research in both normal and abnormal reading development, is that reading may be best viewed as a categorical and synthetic function that necessitates economical and efficient use of all of the child's cognitive skills, but most especially his linguistic abilities. In fact the fluent reader may be described as a verbal gymnast, who can employ a variety of linguistic devices for sampling the text selectively, or to use Goodman's (1970) terms, for "predicting and reconstructing" the information contained therein. The severely impaired reader does not have such an armamentarium available to him, and must be provided with alternate means of deciphering a message, when one or more of his decoding skills fail him. Thus the child who misreads the word PLUS and responds ADD, may not seriously interrupt the intended meaning of the passage, but if his response is SUBTRACT or DIVIDE, he may need some other decoding mechanism that mediates to the correct meaning; for example, knowledge of the component sounds of the stimulus word. The latter implies a total and balanced approach to remedial reading, with
appropriate emphasis on activities that sensitize the reader to all of
the features of words and sentences (i.e., graphic, phonologic, semantic
and syntactic; Gibson, 1971), in relation to given skills deficiencies,
whatever their origin. We suspect that the successful teacher of read-
ing adopts such an approach, whether she is aware of it or not.
In most of the studies which have recently appeared in the literature, research samples have been more carefully selected than in many of the early studies conducted (Benton, 1962). Our own sample selection has employed individually administered tests of intelligence (WISC); and oral reading, and phonics skills, as well as screening measures to exclude children with gross physical and neurological defects, sensory acuity problems, severe emotional disorder, and frequent absences from school. In addition all subjects attended public and parochial schools located in middle to upper middle socio-economic areas, and none were clinic cases. Comparisons between poor and normal readers were also characterized by equivalent age and sex ratios.
Footnote 2

Also noteworthy, is the disparity between second and sixth grade poor readers observed in the second study (Vellutino, Smith, Steger and Kaman, 1974). As expected, the second graders’ performance on letter reproduction declined as the length of a word increased; however, poor readers in sixth grade both named and copied, from memory, all stimulus words as well as normals. This was particularly impressive in the case of the naming task, considering that the letters were named directly after pronunciation of the words, and in view of the fact that these subjects pronounced most of the words incorrectly. These data suggest that the poor readers were sufficiently well acquainted with the orthographic structures of the words to reproduce their letters in correct sequence, in spite of the fact that they did not identify them verbally, as whole words. The latter obviously implies intact (visual) perception and memory. Perhaps as important, it supports the contention of those (e.g., Kolers, 1970; Smith, 1971) who suggest that word identification is not accomplished by serial letter processing.
Footnote 3

In nine studies conducted in our own laboratory (see references), poor readers were significantly below normals on the WISC Verbal I.Q., but equivalent to the normals on the Performance I.Q. These results were obtained in children whose ages ranged from 7 to 14 years.
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