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AUTHOR Satz, Paul; And Others
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ABSTRACT

Hypotheses accounting for the pattern of deficits in specific developmental dyslexia were examined. The theory postulates that the disorder reflects a lag in maturation of the central nervous system, particularly the left cerebral hemisphere. The hypotheses in question predicted that skills developing ontogenetically earlier (visual-motor and auditory-visual integration) will be more delayed in younger dyslexic children (ages 7-8 years), and that skills developing later (language and formal operations) will be more delayed in older dyslexic children (ages 11-12 years). Subjects were 20 disabled male readers and 20 control males, all of average or above average intelligence. Each group was subdivided by age: younger (ages 7-8) and older (ages 11-12). Three tests classified as nonlanguage or perceptual and three classified as language tests were administered. The nonlanguage tests were postulated to represent skills developing ontogenetically earlier. The nonlanguage tests partially discriminated between younger dyslexics and controls, while none of the nonlanguage tasks discriminated between the older groups. By contrast, the language tasks revealed significant differences between older dyslexics and controls, while only one language measure discriminated between the younger groups, thus substantially supporting the hypotheses under evaluation. (KW)

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AN EVALUATION OF A THEORY

OF

SPECIFIC DEVELOPMENTAL DYSLEXIA

Paul Gatz, Donald Rardin and John Ross

Neuropsychology Laboratory and Department of Pediatrics

University of Florida

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SATZ

Abstract

The present study examined a number of hypotheses advanced by Satz and Sparrow (1970) to account for the pattern of deficits in developmental dyslexia. The theory postulates that the disorder is not a unitary syndrome but rather reflects a lag in the maturation of the CNS which delays the acquisition of those skills which are in ascendancy at different developmental ages. The hypotheses predicted that skills which develop ontogenetically earlier (e.g., visual-motor and auditory-visual integration) would be more delayed in younger dyslexic children (Ages 7-8) while skills which develop later (e.g., language and formal operations) would be more delayed in older dyslexic children (Ages 11-12). The results, based upon dyslexic and control children matched at two different age levels, were in substantial agreement with the theory.

Satz and Sparrow (1970) recently advanced a theory of specific developmental dyslexia which: (a) conceptualizes the pattern of deficits (nature of disorder), (b) postulates the CNS mechanisms which may underlie this disorder, and (c) generates a number of developmental hypotheses which predict differential behavioral patterns between dyslexic and control children at similar ages and between dyslexic groups at different ages. The present study is addressed to a test of these hypotheses.

Nature of Disorder (Language and/or left hemisphere function):

The theory, in brief, postulates that the behavioral pattern of deficits observed in dyslexic children is quite similar to adults who have sustained damage to the left cerebral hemisphere. Both clinical groups, while varying in age and etiology, have often shown a pattern of right-left confusion, finger agnosia, calculation difficulty, writing difficulty, visuo-constructional impairment and depressed verbal intelligence (Satz and Sparrow, 1970). The defect underlying this symptom pattern in left hemispheric-damaged adults is aphasia, although the symptoms involve both sensorimotor and language components.

More direct comparisons between the pattern of language deficit in brain-injured adults and dyslexic children was reported recently by Luria (1970). The author analyzed a number of functions which underlie the reading and writing process (e.g., evaluation of speech sounds, word recognition, coding of sound units, letter sequencing, etc.) and showed that these functions depend upon the integrity of specific areas in the left cerebral hemisphere.

An additional behavioral skill which dyslexic children have difficulty with is the capacity to form intermodal associations (Birch and Belmont, 1964). According to Butters and Brody (1968) ". . . reading and object-naming are viewed as psychological processes which depend heavily on visual-auditory or tactile-auditory intersensory associations. The written word and the visually or tactually presented object must arouse their appropriate auditory associates if they are to be successfully read or named (p. 328)." Using a group of left- and right-sided adult brain-injury cases,

demonstrated that lesions restricted to the left inferior parietal cortex differentially impaired performance on a number of cross-modal tasks, particularly auditory-visual. These same patients also showed impairment in reading ability (alexia)

Clue to CNS Mechanism. (Maturation Lag Hypothesis):

Despite the similar pattern of behavioral deficits in dyslexic children and brain-injured adults, studies have failed to document any structural alteration or damage to the left cerebral hemisphere in dyslexics. This presents a major problem in attempts to subserve the pattern of deficits in disabled readers and in left brain-injured adults under the rubric of a disturbance in language and/or left hemisphere function. A partial resolution of this problem is provided by Money (1966, p. 34): "The great majority of reading disability cases will be classifiable not on the basis of brain pathology, but simply as representative of a lag in the functional development of the brain and nervous system that subserves the learning of reading." This distinction between loss of language (structural alteration) and lack of language development (brain maturation lag) may be the key to the problem. If a functional lag in development is meant to imply a more diffuse or less complete differentiation of cerebral organization, then motor, somatosensory and language functions should be similarly affected. Thus, a delay in the lateral development of left hemisphere functions (sensorimotor and language) might affect the acquisition rather than the loss of those skills which require concepts of right-left discrimination, calculation, finger differentiation, visual-motor integration, auditory-visual integration, and the like.

The postulation of a correlation between delayed CNS maturation (left-hemisphere) and behavioral immaturity still lacks complete verification. Nevertheless, neuroanatomic studies of the cerebral cortex have shown that the growth of the brain undergoes enormous structural, electrophysiological and biochemical changes during the first two years of life and that this growth pattern comes to

a close around puberty (Lenneberg, 1967). Moreover, these growth phases tend to correlate with developmental milestones in motor, somatosensory and language function. A relationship between brain maturation and ontogenetic development has recently been discussed by Geschwind (1968, p. 103): "The early myelinating zones include all of the classic motor and sensory zones, i.e., the classical motor cortex (area 4), and the primary somesthetic, visual and auditory cortices." These early myelinating or "primordial" zones have the most efferent and afferent connections with subcortical structures and the fewest long connections with other cortical areas. By contrast, those zones which myelinate latest, the "terminal" zones (i.e., left angular gyrus), have prominent intercortical connections which are necessary in the mediation of more complex language and cross-modal integration skills. The hemispheric organization of speech has already been shown to evolve from a state of diffuse and bilateral representation in infancy to one of increased lateralization by puberty (Lenneberg, 1967).

More recently, Semmes (1968) has suggested that this development in hemispheric speech lateralization might stem from a basic difference in sensorimotor organization which has already been differentiated within the left hemisphere. This hypothesis attempts to account for differences in the hemispheric organization of a complex function (e.g., language) as an outgrowth or synthesis of elementary sensorimotor functions whose neural organization already favors specialization on the left.

Hypotheses. (Developmental Predictions):

Semmes' data suggests an alternative and more general formulation to the language disorder previously advanced to account for the pattern of deficits in dyslexia. It involves the concept of a maturation lag in the lateralization and differentiation of motor, somatosensory and language functions subserved by the dominant left hemisphere. This alternative formulation presupposes, in normal children, an orderly and hierarchical development of functions within the left hemisphere beginning with motor, then somatosensory and finally, speech lateralization.

Two premises are advanced:

The first premise (P_1) postulates that hemispheric specialization in language stems from a basic difference in sensorimotor organization in the brain which developmentally precedes the lateral differentiation of speech and language.

The second premise (P_2) postulates that, in normal development, behavior proceeds from grossly diffuse and unmodulated operations to greater differentiation and hierarchical integration of motor, somatosensory and symbolic language function.

The first premise is postulated to represent the underlying neural substrate for the behavioral counterparts in Premise II. Further, it is assumed that this maturation process, in normal children, is essentially an age-linked process, i.e., that maturation level is a function of chronological age (Gesell, 1945). A maturational lag, therefore, is defined as slow or delayed development of those brain areas (left hemisphere) which mediate the acquisition of developmental skills which are fundamentally age-linked. Thus, the pattern of deficits observed in dyslexic children, rather than representing a unique syndrome of disturbance, should resemble the behavioral patterns of chronologically younger normal children who have not yet acquired mastery of skills which develop ontogenetically later. In other words, it is postulated that the level of brain maturation in both younger normal and older dyslexic children is less mature and differentiated. On this basis, the pattern of deficits within dyslexic groups should vary as a function of the age at which certain skills are undergoing primary development. Because visual-motor skills are established ontogenetically earlier (Ages 7-8), one might expect to find this pattern of difficulty in the younger dyslexic child (Piaget and Inhelder, 1969). Conversely, those functions which develop ontogenetically later (e.g., language and formal operations) might be expected to occur in much older dyslexic children (Ages 11-12) who are assumed to be maturationally delayed (Piaget and Inhelder, 1969).

The present study represents an attempt to examine some of the hypotheses generated by the theory. The primary test concerns the prediction of differences between younger (Ages 7-8) and older (Ages 11-12) dyslexic children. Lacking

longitudinal data, it is not possible, within the present study, to examine differences in skills which are acquired much earlier in development (e.g., manual laterality). The following hypotheses are proposed.

Hypothesis 1. Younger dyslexic children will be more delayed in visual-motor integration and auditory-visual integration than older control children.

Hypothesis 2. Older dyslexic children will not be more delayed in visual-motor integration and auditory-visual integration than older control children.

Hypothesis 3. Older dyslexic children will be more delayed in language integration skills than older control children.

Hypothesis 4. Younger dyslexic children will not be more delayed in language integration skills than younger control children.

Method

Subjects

Disabled readers (D). Disabled readers were selected from teacher recommendations. At a meeting with the teachers it was requested that names of children be submitted who manifested a severe reading problem but who showed no gross physical, sensory or neurological handicap. Additional criteria for selection were that the children be Caucasian males of normal intelligence (teacher judgment) who were of ages 7-8 (younger) and 11-12 (older). Approximately 20 children were selected from each age group.

The Performance Scale of the WISC (PIQ) was then administered to all 40 children in order to select a sample of 10 children at each age level (7-8 and 11-12) who revealed average-above average intelligence. The WISC Verbal Scale (VIQ) was not administered during the pre-experimental selection phase.

Control Readers (C). The procedure for selecting control subjects (Ss) was exactly the same as for the disabled readers with the exception that each child be reading at grade level or above. Again, approximately 40 Caucasian male children were selected at ages 7-8 and ages 11-12. After administration of the Performance Scale, 20 children were selected, 10 at each age level, to match

the disabled readers on PIQ.

This matching procedure therefore yielded a total of 40 male children consisting of 20 disabled readers and 20 control readers with each group divided into two different age groups of 10 children: Younger (Ages 7-8), Older (Ages 11-12). All 40 children were selected from the same middle-income class school.

After the final selection of Ss was obtained, each child was administered the word recognition part of the Wide Range Achievement Test (WRAT) in order to obtain an independent measure of reading ability. Table 1 presents the general characteristics by group and age for each of the control variables. Although the mean WRAT scores were generally high overall, the differences between dyslexic and control groups were substantially different at both ages: Ages 7-8 ($\bar{X}_D = 2.35$, $\bar{X}_C = 3.97$, $t = 3.88$, $df = 19$, $p < .001$); Ages 11-12 ($\bar{X}_D = 4.80$, $\bar{X}_C = 7.51$, $t = 5.36$, $df = 19$, $p < .001$). In fact, there was no overlap between groups at either age level on this test which lends independent support for the validity of teacher recommendations. By contrast, the groups revealed similar mean PIQ values at both ages: Ages 7-8 ($\bar{X}_D = 99.9$, $\bar{X}_C = 104.4$, $t = 1.33$, $df = 19$, $p > .10$); Ages 11-12 ($\bar{X}_D = 104.4$, $\bar{X}_C = 103.3$, $t < 1.00$, $df = 19$, $p > .10$).

Tests

Six different tests were administered, three of which were classified as nonlanguage or perceptual and three of which were classified as language. The three nonlanguage measures were postulated to represent skills which develop ontogenetically earlier.

Nonlanguage Skills. The three measures of nonlanguage skills were the Bender-Gestalt Test, the Recognition-Discrimination Test (Small, 1968) and the Auditory-Visual Test (Birch and Belmont, 1964). Scores on the Recognition-Discrimination (R-D) and Auditory-Visual (A-V) tests were converted into percent correct. In order to obtain greater objectivity for the Bender-Gestalt Test (B-G) the protocols of each child were independently rated by four examiners

on the basis of poor (Score=1), medium (Score=2) and good (Score=3). The judgments on each child were then summed across the examiners yielding a base score of 4 (poor) and a maximum score of 12 (good). Judgments were made separately for each age group of 20 children without knowledge of S's name, group classification (D or C) or intelligence.

Language Skills. This group of tests was comprised of the Verbal Scale (VIQ) of the WISC, the Verbal Fluency Test (Spreen, 1965) and the Dichotic Listening Test (Satz, 1963). The Verbal Fluency Test (V-F) assesses the child's ability to name as many words beginning with the letters F, A, and S. The child is given one minute for each letter. Scores on this test were converted into percentile scores developed by Spreen (1965). The test provides a fairly useful and economical measure of oral language productivity in children. The Dichotic Listening Test (D-L) is described in detail elsewhere (Satz, 1963). Briefly, ss are presented with disparate pairs of numbers which arrive simultaneously via stereo headphones every half-second. In the present study, ss were presented with 25 trials of three pair digit sequences; each pair, within interval trials, was presented at a rate of two pairs per second with an intertrial/of 10 seconds for recall. Stimulus onset was synchronized between dichotic pairs by means of a computer program recently developed by our laboratory. Previous studies (adults) have demonstrated superior recall for verbal stimuli presented to the right ear presumably because of the more direct connections between the right ear and the speech "processor" in the contralateral left temporal lobe (Satz, 1963). Thus, the D-L procedure was felt to provide a fairly valid behavioral measure of cerebral lateralization of speech in children. Scores were converted into percent of correct recall for each ear (R,L) and for total recall (R+L).

Results

Nonlanguage Skills. (Hypotheses 1 and 2)

Bender-Gestalt Test. (Visual-Motor Integration):

Inspection of Table 2 reveals a much lower mean value on the Bender-Gestalt Test for the younger dyslexic than for the younger control group ($\bar{X}_D = 5.56$, $\bar{X}_C = 8.60$, $p < .001$). This finding lends support for Hypothesis 1. Conversely, there was no difference on this test between the older dyslexic and control groups ($\bar{X}_D = 7.10$, $\bar{X}_C = 8.50$, $p > .10$) which lends support for Hypothesis 2. With respect to Hypothesis 1, not one of the younger experimental Es had a higher score than their age-matched controls, which further illustrates the differential delay in the younger dyslexics on this test.

Auditory-Visual Test. (Auditory-Visual Integration):

Table 2 also reveals a lower mean correct performance on this task for the younger dyslexic than for the younger controls ($\bar{X}_D = 76\%$, $\bar{X}_C = 91\%$, $p > .10$). The difference, however, was not significant; this was largely due to small N and to four experimental Es who obtained scores greater than their matched controls. Thus, Hypothesis 1, while in the predicted direction, was not supported with this measure. The corollary hypothesis (Hypothesis 2), however, was confirmed and again revealed no group difference on this task at the older ages ($\bar{X}_D = 92\%$, $\bar{X}_C = 99\%$, $p > .10$).

Recognition-Discrimination Test. (Visual Perception):

Inspection of Table 2 reveals no difference in mean correct performance between the younger experimental and control groups on this lower level discrimination task ($X_D = 91.1\%$, $X_C = 94.4\%$, $p > .10$). This finding neither confirms nor rejects Hypothesis 1 because it represents a skill which is developed ontogenetically earlier (Ages 5-6) than cross-modal integrative functions (Small, 1963). There was also no difference between the older groups on this task ($X_D = 94.4$, $X_C = 95.3$, $p > .10$) which again lends support for Hypothesis 2.

Language Skills. (Hypotheses 3 and 4):

WISC Verbal Intelligence:

In order to examine the degree of language delay on this test, analyses were made on: (1) the mean Verbal IQ scores and (2) the difference between Verbal and

Performance IQ scores (VIQ - PIQ). Thus, a minus score on the latter analysis reflects a lower or depressed verbal score.

VIQ Scores:

Table 2 reveals the mean VIQ scores for the younger and older experimental and control groups. Inspection of this table reveals a lower mean VIQ for the older dyslexic than for the older control groups ($\bar{X}_D = 101.7$, $\bar{X}_C = 117.6$, $p < .001$). This finding lends support for Hypothesis 3. However, the mean VIQ scores were also lower for the younger dyslexic than for the younger control groups ($\bar{X}_D = 103.4$, $\bar{X}_C = 112.6$, $p < .01$) which contradicts Hypothesis 4. Nevertheless, the trend was in the predicted direction with older groups showing a greater difference on this task. The fact that the WISC Performance IQ scores were slightly, though non-significantly, lower in the younger dyslexic group may have produced an artificial depression in Verbal IQ in these same children. For this reason, discrepancy scores were used to obtain a more valid estimate of level of Verbal intelligence.

VIQ - PIQ Discrepancy Scores:

The results of this analysis were slightly different. Table 2 indicates that the mean discrepancy scores were much smaller in the older dyslexic than in the older control groups ($\bar{X}_D = -2.7$, $\bar{X}_C = 14.3$, $p < .001$) which again points to depressed verbal-integrative functioning in the older dyslexic Ss (Hypothesis 3). By contrast, the mean discrepancy scores were not different between the younger dyslexic and control groups which is in direct support of Hypothesis 4 ($\bar{X}_D = 3.5$, $\bar{X}_C = 7.7$, $p > .10$).

Verbal Fluency Test:

Mean percentile scores for groups and ages on this test are presented in Table 2. The mean percentile score was significantly lower in the older dyslexic than in the older control groups ($\bar{X}_D = 52.8\%$, $\bar{X}_C = 76.2\%$, $p < .01$) which again lends support for Hypothesis 3. By contrast, no difference in mean percentile score is found between the younger dyslexic and control groups on this task ($\bar{X}_D = 21.7\%$,

$\bar{X}_C = 27.6\%$, $p > .10$) which again lends support for Hypothesis 4. With respect to the older Ss, only one dyslexic S obtained a higher percentile score on this task than his matched control.

Dichotic Listening Test. (Ear-Speech-Brain Asymmetry):

The critical analysis on this measure was to determine whether the percent of correct recall for digits presented to the right channel was less in the older dyslexic than older control group. This finding would support Hypothesis 3 which predicts a delay in left hemisphere language development in older dyslexic children. The corollary hypothesis (Hypothesis 4) predicts no difference on this measure between younger dyslexic and control children. In order to test the preceding hypothesis, an analysis was first computed on total recall (RC + LC) for ages and groups (Table 2). No difference was observed in total recall between dyslexic and control groups at either the younger age ($\bar{X}_D = 47.6\%$, $\bar{X}_C = 43.3\%$, $p > .10$) or the older age ($\bar{X}_D = 51.7\%$, $\bar{X}_C = 56.4\%$, $p > .10$). Inspection of Table 2 also shows that significantly more digits were correctly recalled from the right channel in each group at both ages: Ages 7-8 ($RC_D = 57.2$, $LC_D = 33.0$, $t = 4.30$, $df = 19$, $p < .001$; $RC_C = 61.6$, $LC_C = 34.9$, $t = 5.20$, $df = 19$, $p < .001$); Ages 11-12 ($RC_D = 64.1$, $LC_D = 39.2$, $t = 4.75$, $df = 19$, $p < .001$; $RC_C = 74.0$, $LC_C = 33.3$, $t = 5.01$, $df = 19$, $p < .001$). In other words, the tendency to left cerebral dominance for speech was evident in both the dyslexic and control groups at both ages.

The critical test, however, was whether the degree of right channel dominance (or left speech-brain specialization) was significantly less in the older dyslexic than in the older control group. Table 2 indicates that the mean percent recall for the right ear was significantly less in the older dyslexic group ($\bar{X}_D = 64.1\%$, $\bar{X}_C = 74.0\%$, $p < .01$) but that no difference in right channel recall was observed between the younger dyslexic and control groups ($\bar{X}_D = 57.2$, $\bar{X}_C = 61.6$, $p > .10$). These findings lend additional support

Hypotheses 3 and 4. An analysis of frequency data in the older groups

revealed that only one dyslexic S had a higher percent recall score for the right ear than his age-matched control.

Discussion

The present results lend substantial support for the directional hypotheses predicted by the theory. With respect to the nonlanguage tests, the younger dyslexic children (Ages 7-8) produced more immature drawings on the Bender-Gestalt than did their matched controls. A similar trend in the younger groups was observed on the Auditory-Visual task, although the group difference was not significant. Thus, Hypothesis 1 was partially confirmed. On the other hand, not one of the three nonlanguage tasks (Recognition-Discrimination, Bender, Auditory-Visual) discriminated between dyslexic and control children in the older groups (Ages 11-12). This finding lends substantial support for the corollary hypothesis (Hypothesis 2).

By contrast, the language tasks primarily discriminated between groups in the older children (Ages 11-12). Each one of the language measures (VIQ, VIQ-PIQ, Verbal Fluency and Dichotic Listening) revealed a significant difference between dyslexic and control groups in the older children. Thus, Hypothesis 3 was substantially confirmed. Conversely, only one of the four language measures discriminated between the younger dyslexic and control children (i.e., VIQ) which lends fairly good support for the corollary hypothesis (Hypothesis 4). This finding (Hypothesis 4) limits the generality of a language deficit as the primary disturbance in developmental dyslexia. Consistent with the theory, the results tend to restrict this type of disorder to older disabled readers. Nevertheless, there are reports in the literature which suggest that delays in preschool language development may forecast later reading disability. Such children, however, may represent a quite distinct group with varying etiologies and environmental background. Only a longitudinal study could adequately examine this possibility.

On the basis of these findings certain conclusions and problems deserve

mention. First, the age factor is a critical independent variable which should be examined in investigations of specific reading disability. Failure to control for this variable could easily mask differences between dyslexic and control children. The long controversy concerning visual-motor disturbances in these children is an example. Benton's review of this problem (1962) suggested that a deficit in visual-motor performance may be associated with younger dyslexic children but that the deficit tends to attenuate with age. The theory advanced in this paper predicts that those developmental patterns which have an earlier ontogenetic development (e.g., visual-motor integration) are more likely to be observed in younger children who are delayed in maturational development. Conversely, those developmental patterns which have a later ontogenetic development (e.g., language and formal operations) are more likely to be observed in older children who are maturationally delayed. Thus, the nature of the disorder varies as a function of the age of the child. This differential prediction was largely confirmed by the present results (Hypotheses 1-4).

A second conclusion suggested by these findings is that an earlier delay in maturation may forecast behavioral immaturity at each successive stage of hierarchical development. Thus, while a child who lags in visual-motor integration age Age 7-8 may eventually "catch up" by Age 11-12, he may now lag in those skills (e.g., symbolic language) which have a later ontogenetic development. The present results indirectly support such a possibility. Not one of the nonlanguage measures differentiated between the groups at the older ages (Hypothesis 2) whereas all of the language measures revealed a decrement in the older dyslexic children (Hypothesis 3). Sparrow and Satz (1970), using only older children (Ages 9-12), demonstrated significant differences between dyslexic and matched control Ss on all measures of language skill (e.g., VIQ, Right-Left Discrimination, Finger Differentiation, Dichotic Listening); however, with the exception of one test of manual laterality, none of the sensorimotor tasks revealed differences between the older groups (e.g., manual laterality,

visual laterality). Similarly, Sabatino and Hayden (1970), using a factor analysis of psycholinguistic and perceptual tests on older (Age 11-4) and younger (Age 7-7) disabled learners, recently demonstrated a primary loading on perceptual deficits in the younger children and a psycholinguistic deficit in the older children. Consistent with the developmental hypothesis, the authors concluded "...that six years to nine years is the maximum growth period for perceptual functional performance. After age ten, integrated language skills become of prime importance" (p.411).

Although the preceding findings suggest that earlier delays in maturation may forecast a different pattern of deficits in later childhood, it should not imply that all such children will be delayed at each successive stage of hierarchical development. In order to examine this question, a longitudinal design would be required in which measures were available on the same ss at different developmental ages. Furthermore, the exploratory longitudinal study conducted by de Hirsch, Jansky and Langford (1966) identified a small number of children (slow starters) during the second longitudinal year (Grade 1) who, while delayed maturationally and behaviorally, managed to achieve grade equivalent reading scores at the end of the project (Grade 2).

An additional conclusion related to the present findings is that the identification of delays in maturation at earlier ages (e.g., pre-school) may provide valid predictors of later reading disability. The advantage of obtaining early indices of subsequent reading disability is that remedial programs may be introduced at a time when the child's central nervous system is more plastic and responsive to change and at a time when the child is more likely to be free of psychological conflict over his handicap. Again, a longitudinal study would more effectively identify these possible pre-school indices. The senior author is now engaged in a large-scale longitudinal investigation of male kindergarten children in order to obtain a more reliable test of the above problems.

The present findings, in summary, lend support for the concept of a maturational

tion lag as a possible mechanism underlying the reading disorder in these children. The central brain mechanism (lag in maturation of left hemisphere), which was postulated to underlie the disorder, lacks direct verification at this time. Nevertheless, it provided a framework in which to conceptualize the developmental-behavioral hypotheses. Confirmation of these hypotheses, therefore, provided more than heuristic value for the theory. The concept of a maturation lag, if valid, eliminates the need to involve labels such as brain damage to describe these children; the label, moreover, tends to have pernicious implications for the child. The theory merely states that many of these disabled readers are not maturationally or developmentally ready to cope with the reading process. Interestingly, the vast majority of these children are boys. Boys have long been known to mature at a slower rate than girls, particularly in perceptual-motor development between four and seven years of age (Beery, 1967).

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FOOTNOTE

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Table 1
Subject Characteristics by Group and Age

Group	WRAT Grade Reading Level	Mean Age	N	Mean PIQ
Dyslexic Young	2.35	3-2	10	99.9
Control Young	3.97	7-3	10	104.9
Dyslexic Old	4.80	11-4	10	104.4
Control Old	7.51	11-7	10	103.3

Note.-All subjects were white males from the same school

Table 2

Mean Scores and t-tests for each Variable by Age and Group (D,C)

TESTS	Age 7-8			Age 11-12		
	\bar{X}_D	\bar{X}_C	t	\bar{X}_D	\bar{X}_C	t
Nonlanguage Tests						
B-G	5.6	8.6	4.21**	7.1	8.5	1.41
A-V ^a	74.0	91.0	1.10	92.0	99.0	1.00
R-D ^a	91.1	94.4	1.00	94.4	95.8	1.00
Language Tests						
VIQ	103.4	112.6	2.74*	101.7	117.6	3.12**
VIQ-PIQ	3.5	7.7	1.00	-2.7	14.3	3.30**
V-F ^a	21.7	27.6	1.00	52.3	76.2	2.86*
RC ^a	57.2	61.6	1.00	64.1	74.0	2.15*
LC ^a	38.0	34.9	1.00	39.2	33.8	1.00
Total RC+LC ^a	47.6	48.3	1.00	51.7	56.4	1.00

^aScores in percent or percentile

*df=19 p. < .01.

**df=19, p. < .001.