Dyslexia--Is There Such a Thing.

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The theory of developmental dyslexia is contrasted with the theory of developmental immaturity to explain extreme reading retardation. Dyslexia is defined as an organically based reading disability which is determined indirectly since no anatomic lesion, chromosome, or gene has yet been found responsible. Dyslexia is usually deduced from (1) genetic findings (frequency of occurrence within families, greater incidence among boys), (2) by a process of eliminating other possible causes, (3) by the identification of soft neurological signs, and (4) by the effectiveness of special methods of instruction. The theory of developmental immaturity is based on four interrelated aspects of individual differences--normality, variability, covariability, and velocity. In this view, reading retardation is explained as an accumulation of specific deficits in trait performance. The absence of a comprehensive, individualized instructional program is considered basic in accounting for reading retardation. It is noted that sufficient evidence is not available to enable us to accept or reject either theory. Fifteen references are included. This paper was presented at the International Reading Association Conference (Boston, April 24-27, 1968).

(RJ)
"I am inclined... to agree... that the diagnosis 'specific dyslexia' is suitably obscure, adequately descriptive, and carries no theoretical implications."

(J. McFie, National Hospital, London, England)

It is a semantic axiom that words represent things and ideas in the real world. And our effectiveness in communicating with each other depends upon the extent to which we have shared directly or vicariously in these ideas and things. Dyslexia is a case in point. In fact, the title of the session asks "Is there such a thing?" If there is, we should now or soon be able to define it with minimal equivocation in such a way that we could then identify this phenomenon with considerable reliability.

At the present time many problems confront us in defining dyslexia. In the first place the term is used differently by different specialists. Further, some specialists deny its existence. A confounding problem is the proliferation of synonyms for dyslexia including: remedial case, developmental dyslexia, congenital dyslexia, perceptual handicap, minimal brain dysfunction, specific language disability, neurological impairment, etc.

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Since many states and local school systems offer special programs for pupils who are alleged to have dyslexia and since parents have to accept such labels for their children, (frequently bewildered at their meaning), it seems essential that every effort be made to clearly identify whether dyslexia is an entity and to define it specifically.

One of the many ways to consider this problem is to analyze two of the major and apparently different theories of extreme reading retardation: The Theory of Developmental Dyslexia and The Theory of Developmental Immaturity. This is the approach I will take in this paper.

Developmental dyslexia as a clinical entity was first postulated about 70 years ago (2). Some 30 years ago Orton (11) suggested that serious reading retardation might be physiological in origin.

THE THEORY OF DEVELOPMENTAL DYSLEXIA

Among the most distinguished users of the term dyslexia today are Ralph D. Rabinovitch, M.D., Neuropsychiatrist and Director of the Hawthorne Center, Northville, Michigan, and Macdonald Critchley, M.D., former Dean, Institute of Neurology, Queen Square, London, England; former Doyne Lecturer on Dyslexia; and President, World Federation of Neurology. In fact, most medical practitioners as well as other professionals whose orientation is primarily clinical have followed the lead of these two men in their uses of the term.

At the National Conference on Dyslexia in Philadelphia, 1966,
Rabinovitch (14) postulated "that the syndrome called 'dyslexia' is a separate entity, discretely definable from many causes of reading disability." This entity "...reflects a definitive neurological dysfunction in the absence of history or signs of brain injury." "The problem appears to reflect a basic disturbed pattern of neurological organization." (14) Thus he proposes a classification scheme of reading retardation based on etiology which includes two basic categories: (1) primary retardation or developmental dyslexia and (2) secondary retardation which includes all other causes of reading retardation, including brain damage.

Reading retardation resulting from brain damage is classified as secondary. In the case of brain damage, we have clear neurological deficits resulting from prenatal toxicity, birth trauma, or anoxia, encephalitis, head injuries, etc.

Other causes of reading retardation classified as secondary include emotional and motivational factors, poor learning opportunities, poor vision and hearing, and other physical problems.

Critchley agrees with Rabinovitch fundamentally. He differs only in that he uses "dyslexia" in both a general and specific way in the characteristically British fashion. Notice his uses in three consecutive sentences where I have underlined key words: "Most neurologists believe that a form of dyslexia exists which is organic in nature. This is not to say that other types of reading retardation do not exist, but the neurologic conception of dyslexia that exists in its purest form... Dyslexia is constitutional for two reasons..." (3). Thus, we see that whereas Rabinovitch reserves the term dyslexia for primary
retardation, Critchley sometimes does the same but sometimes uses the term as a generic term for all forms of reading retardation.

The Random House Dictionary of the English Language (Unabridged Edition, 1967), defines dyslexia as "an impairment of the ability to read due to a brain defect." This use presumably includes the primary dyslexia of Rabinovitch and brain damage.

Many regard dyslexia not as a term indicating pathology, but rather simply as the inability to read up to capacity or up to grade level without reference to etiology.

Clearly, in any discussion we must know in which way dyslexia is being used. In this paper, I will follow the use of Rabinovitch, using dyslexia to refer only to reading retardation caused by organicity other than brain damage.

THE LACK OF BIOLOGIC OR ANATOMIC EVIDENCE

With respect to more basic biologic evidence in the study of persons with severe reading retardation, Buchanan has stated that there is no anatomic locus of the brain which has been recognized with certainty as being functionally related to reading though several reports suggest the probability of the angular gyrus as the region. (1) At the conclusion of his extensive survey of neuroanatomy and neurophysiology specific to reading retardation he summarized:

"Knowledge of representation of intellectual functions in the cortex is still vague, contrast studies of the brain are crude and electroencephalographic tracings are complicated
and variable. Because of these difficulties there is yet no objective test that can display an anatomic or physiologic lesion underlying dyslexia." (1)

He further noted that "...no one has recognized a chromosome or gene that is responsible for the presence of dyslexia." (1)

Interestingly enough, despite the lack of biologic evidence, Buchanan nevertheless says that "...those trained in biology believe that dyslexia springs from a biologic fault. Although a specific gene has not been recognized, the available evidence supports the biologic explanation." (1) In other words, he supports the notion that there is likely a biologic fault of some specific type, even though it has not been identified or isolated according to his own review of evidence.

In the absence of identifiable biologic evidence, how then is the case made for dyslexia? It is, in fact, deduced from one or a combination of lines of argument including: (1) genetic findings, (2) by exclusion, (3) by the identification of so-called neurological soft signs, and (4) by the effectiveness of special methods.

(1) Genetic Findings

To support the theory that dyslexia is genetically determined, two kinds of findings are offered: (1) studies that show more than one case of reading retardation in families, and (2) studies that show boys to have the "pure type" of dyslexia more often than girls. From these studies alone, Critchley in one discussion of the subject concluded: "Hence the neurologic position is that specific developmental dyslexia is a genetically determined constitutional disorder. This is extremely important because it means that developmental dyslexia arises
independent of environmental factors." (3)

The work of Hallgren is cited by Critchley as the most definitive in showing the genetic factor in dyslexia. In 276 cases, Hallgren is reported as having found reading problems in the families of 88% of his cases.

Of course, use of the studies of genetic occurrence within families and male vs. female incidence (2,3) as a basis for establishing the existence of dyslexia has to be evaluated against alternative and reasonable hypotheses such as the following:

(a) That the co-occurrence of reading retardation within a family is a function of a more generally shared limitation of experience, instructional opportunities, verbal capacity, or personality type.

(b) That variability among boys is typically greater than among girls in many psychological and physiological characteristics. (15)

(c) That girls mature faster than boys.

(d) That myelinization in the cortex of the angular gyrus of the brain is more likely to be unduly delayed for boys as compared with girls.

(e) That sex differences in average performance and variability are culturally determined in part. For example, Preston (13) found German boys to be superior in reading to German girls in grades 4 and 6 and German girls showed more variability than boys.

(2) The Exclusion Definition

Another basis used to support the existence of developmental dyslexia is a definition of this term by exclusion. "By definition," Critchley says, "neurologists identify developmental dyslexia by eliminating all those children who are emotionally disturbed, who
have perceptual defects, or who have low intelligence." (2) In other words, if we can eliminate the so-called secondary causes as not relevant to the extent of reading retardation, developmental dyslexia must then be regarded as accounting for the problem. It is interesting to note the lack of reference in Critchley's definition to poor instruction. In his more comprehensive treatment of the subject of developmental dyslexia Critchley shows further his little concern for the influence of instruction when he accepts Eisenberg's definition of a dyslexic as one who "...is unable to learn to read with proper facility despite normal intelligence, intact senses, proper instruction, and normal motivation." (2) Then Critchley says: "Eisenberg's definition would be improved if for "proper" instruction he substituted the adjective "conventional." (2) Presumably Critchley's acceptance of "conventional" comes from the fact that a great majority of pupils do learn to read under usual circumstances.

The major weakness of this definition-by-default comes from the difficulty, if not impossibility, of eliminating the significant influences of psychogenicity, poor instruction, and other environmental factors (other than clear brain damage) from the picture. Since dyslexia cannot be observed directly we are left to see it as if through a lens which is occluded in some indeterminate measure by these other causative and complicating factors.

(3) Soft Neurological Signs

Critchley (4) has recently reported, contrary to the speculation of many, that there are no gross abnormalities to be detected in the dyslexic reader typically, i.e., no spasticity, no increased reflexes,
no conspicuous abnormality in growth or physical habitus, no defects in constitutional tasks, no evidence of Gerstmann's syndrome, no clumsiness or lack of manual dexterity, etc. On the other hand, he has found in "many cases" upon deeper probing "subtle defects" or soft neurological signs including: lack of cerebral dominance, confusion regarding space and time, mild electroencephalographic dysrhythmias, abnormal eye movements when reading, minor color blind defects, and abnormal preferred direction of lateral gaze.

Samuel Orton (11) believed that there was a state of ambiguity in the cerebral dominance of the brain of some retarded readers. It was this underlying condition, he thought, that accounted for mixed laterality, left-handedness, and reversals in reading and spelling. Orton's term for dyslexia was "strephosymbolia" (twisted symbols) but it never caught on.

The fact that some retarded readers exhibit directional confusion, mixed dominance patterns, etc., represents possibly the most solid base in making a case for the existence of dyslexia. Presumably, immediately underlying reading retardation are measurable substrata factors (to use Holmes' term) and a general integrative ability which are the foundation traits for success in reading, spelling, and writing. Soft signs are represented as symptomatic of the organic basis of reading disability. Now, if we were to postulate the existence of such soft signs without first observing them, I believe we should expect them to be qualitatively different from similar measurements taken with respect to the performance of the non-dyslexic pupil. Further, we should not expect to find major differences in these characteristics among pupils in various school systems whose instructional quality differs. One of my colleagues in the Reading Clinic at the University of Pennsylvania, Mrs. Irene
Gaskins, in her study of directional confusion of pupils retarded in reading seems to be finding great differences in pupil responses from school system to school system. Her findings will be reported more fully by this summer.

In other words, if pupils who were severely retarded in reading merely differed in the degree to which they exhibited this soft sign behavior, would we not have good reason to believe that we were merely looking at the low end of an expected distribution of the measured traits?

On the other hand, in an interesting study of the normality of distributions of reading ability, Carl A. Larsen, in Denmark, found no normal curve in any grade. Instead he found curves which he construed to result from two separate distributions, one the normal reader, the other the word-blind (7). Replication of this research is needed. In any event, it will have to be determined whether such a curve truly represents two populations or some paradox.

(4) Effectiveness of Special Teaching Methods

There seems to be no agreement at the present time among outstanding leaders as to the best method for teaching seriously retarded readers. Fernald developed a technique and successfully used the method which put emphasis on the student's own language patterns. In this method words are learned as wholes by a strategy which combines visual, auditory, tactile, and kinesthetic modalities. The Gillingham-Stillman method is heavily oriented toward learning individual letter-sound correspondences with emphasis on tactile kinesthetic learning. Cruikshank insists that such pupils need a program which feeds small doses of material to the pupil in a minimally distracting physical setting. Each of these and other methods claim success. When
analyzed, the common element in each of the many alternative methods of treatment seems to be intensive, individualized instruction by well trained teachers who help pupils experience success.

THE THEORY OF DEVELOPMENTAL IMMATURITY

An alternative theory to the existence of a discrete specific syndrome of dyslexia might be entitled Developmental (or Behavioral) Immaturity. Central in this view are the four known characteristics which affect individual differences: (1) normality, (2) variation, (3) covariation, and (4) velocity (12). We have already illustrated the characteristic of normality by pointing out that traits which distribute themselves according to the normal curve, differing from one point to another only in degree, can hardly be regarded at any particular point as signifying some underlying biologic fault manifest as a specific dysfunction. Further we know that individuals vary from one another and within themselves from trait to trait. Moreover these individual traits covary or interact with each other and with environmental forces as the individual functions as a total being, integrating these elements in complex ways. Finally, with respect to velocity, we know that individuals mature at rates which vary from time to time, providing periods of steady growth, plateaus, and spurts.

Thus, given the characteristics of individual differences it is postulated that the retarded reader is one whose accumulation of specific deficits or "lows" in trait performance are interacting as a
delaying force in his maturation. Such a pupil's patterns are not regarded as symptoms of pathological signs. He manifests a syndrome in the sense of the coincidental occurrence of "lows" in traits underlying reading, spelling, and writing. In this view of things his specific performances in relevant behavior are described and respected. The description must be comprehensive enough so that we can be reliably aware of all of his unique characteristics as indicated above. Respect for these differences means that instructional programs must be comprehensive in scope and sensitive to the learner's level, rate, modality preferences, motivation, etc. Failure to accommodate instruction to the uniqueness of the pupil would result in retardation. Such retardation is regarded as a function of lack of readiness on the part of the pupil and of poor instruction with consequent inefficiency in learning to read up to capacity.

Apparent differences in the two theoretical positions described may in part come from the different orientations and modus operandi of the investigators in the field. For example, the ideas supporting the developmental dyslexia hypothesis come largely from clinical specialists in medicine, psychology and remedial education. Since retarded readers who come to these centers have complex problems, the study of these clients in depth in the cross sectional sense may reveal many correlated characteristics of low performance. Seeing so many of these clearly invites speculation as to the probable existence of a unique syndrome, one due to a specific rather than general biologic or constitutional element.

On the other hand, the developmental immaturity hypothesis derives from the work of specialists in medicine, education, and
psychology who have studied their populations both cross-sectionally and longitudinally. In such studies the interaction of the child's internal and external environment are more manifest.

Five research projects of the latter type which are concerned with describing the comprehensive longitudinal behavior of a representative population of pupils will illustrate the promise that might come from such studies in understanding extreme retardation in reading.

These five studies may be further delineated as comprehensive assessment studies and comprehensive and personalized reading programs.

**COMPREHENSIVE ASSESSMENT STUDIES**

Comprehensive assessment studies are concerned with describing the learner rather fully in terms of major variables which relate to present and future learning performance. The work of Ilg and Ames (8) and that of de Hirsch (6) and her associates illustrate this kind of research. The Developmental Placement Test of Ilg and Ames comes from their research in observing the changing developmental patterns of children at various ages and include such subtests as: writing name, date, address and numbers; copying geometric forms including circle, cross, square, triangle, divided rectangle and diamond; the completion of the incomplete man figure; right and left discrimination, visual discrimination and visual memory; the number of animals named in one minute, and responses to the question -- what do you prefer to do at home and at school? These tests yield a general developmental age score which is to be regarded as more significant than chronological age in assessing readiness for success in all academic learning.
including reading.

In the de Hirsch study a generally representative group of 53 kindergarten age children were tested on 37 variables thought to be related to success in reading, spelling, and writing. Ten of these (together called the Predictive Index) were found to be highly predictive of success in reading, writing and spelling at the end of second grade. They include such subtests as holding and manipulating a pencil, the Bender-Gestalt Visuo-motor tests, Wepman auditory discrimination test, the number of words used in telling a story of the three bears, a category test in which the child is asked to produce the class names for three groups of words, the Horst reversals test, the Gates word-matching subtest, and word recognition and word reproduction tests.

Both the test batteries include tests of perceptual, motor, cognitive, and linguistic functioning. Further validation of these tests and similar test batteries is needed at two levels. First, with respect to their general ability to predict relative success of pupils in reading and other academic functioning. And second, with respect to their power to diagnose and prescribe differential instructional strategies.

COMPREHENSIVE AND PERSONALIZED READING PROGRAMS

Studies of the achievement of pupils who have experienced comprehensive and personalized reading programs represent another needed type of research.

I reported in November 1966 at the National Conference on Dyslexia
(1), a study of this type which was conducted in the Pennridge Schools, Bucks County, Pennsylvania. Among other purposes of this study, we were trying to determine if any pupils who have experienced five years of a comprehensive reading program of high quality would still be reading below the fourth reader level. We found that no beginning sixth-grader with an IQ above 80 scored below the high third grade level. Only two per cent of the pupils read below fourth-grade level. This was based on the results of the Botel Reading Inventory, standardized reading tests, and the actual successful performance in readers used in the schools. In the latter criterion pupils had to be performing fluently in oral reading (95 per cent or better on the average) and in comprehension (75 per cent or better on the average). The average IQ of these pupils was 106. In a continuation of the study in the same school this year, last year's findings were confirmed. In addition, pupils were tested this year on an informal spelling inventory. Only two per cent of the pupils scored as low as the third grade level. Thus we have found no pupils "impaled on a primer" (as my colleague Ralph Preston once characterized the dyslexic child) when continuously offered an outstanding instructional program over a five year period. In fact, no pupil was "impaled" even on a beginning third reader level by the beginning of sixth grade.

We are now planning our next year's study. One of my doctoral candidates, Mrs. Patricia Guth, Director of Elementary Education for the Pennridge Schools, will study next year's classes more intensively to determine if there are some specific qualitative patterns of performance which differentiate the retarded reader from the average and the more able reader. Further, Mrs. Guth will compare the performance
of Pennridge pupils with those from a similar socio-economic community whose reading program has been more typical of American schools, i.e. less comprehensive and less sensitive to individual differences. In this way we shall have an additional control on the influence of instruction as a factor in reading retardation.

In two monumental studies (2,10), in which the characteristics and correlates of reading ability were analyzed for large samples of pupils who experienced excellent comprehensive reading programs, no support was found for the notion of dyslexia by these researchers.

The Morris study of over 8000 pupils in Kent County, England, concluded "...that the poorest readers were not in any reasonable interpretation of the term a neurological problem, and that the study as a whole lends little support to the idea that 'specific developmental dyslexia' is an identifiable syndrome distinct from 'reading backwardness'. In other words, if word blindness exists as a condition which cannot be treated by good teaching within the state educational system it must be a rare condition indeed." (10)

The Malmquist study of first and fourth grade children in Sweden reports as follows:

"The results of the investigation show that none of the errors in reading recorded was made by only one group of readers. Every type of reading error is found among poor, medium and good readers. Consequently we consider that our hypothesis that differences between poor, medium, and good readers, with regard to errors in reading, are rather of a quantitative than a qualitative character, has been verified in our study." (2)
SUMMARY

This paper explored two theories of severe reading retardation: The Theory of Developmental Dyslexia and The Theory of Developmental Immaturity as the basis for considering the topic of the session: Dyslexia: Is There Such a Thing?

It was first noted that dyslexia as a term has many synonyms and that it is used in a great variety of ways. This leads to much confusion if those communicating about the problem do not clearly define the way in which they are using the term. For the purpose of this paper, dyslexia was defined to mean a postulated organically based reading disability.

The Theory of Developmental Dyslexia implies organicity. The case made for such a biological or anatomic defect underlying serious reading disability is made indirectly since there is no anatomic lesion nor chromosome or gene which has been found to be responsible for dyslexia as yet. Instead dyslexia is usually deduced from (1) genetic findings, specifically the occurrence within families and the greater incidence of boys with serious reading disabilities, (2) by exclusion of other causes of reading retardation, (3) by the identification of soft neurological signs and (4) by the effectiveness of special methods.

The Theory of Developmental Immaturity is based upon the four interrelated aspects of individual differences: normality, variation, covariation and velocity. In this view, reading retardation is explained as a function of a syndrome of specific deficits or "lows" in trait performance which are interacting as a delaying force in maturation. The lack of a comprehensive, individualized instructional
program sensitive to these aspects of individuals is regarded as basic in accounting for reading retardation.

It was noted that evidence is not yet available to enable us either to accept or reject either theory.

Research that is both cross sectional and longitudinal was proposed to give us more insight into the problem of serious reading retardation. These studies are needed in the areas of assessment and developmental or preventive reading programs.

In addition we need studies in which particular methods are related to syndromes of personality and performance characteristics of retarded and normal readers. One such study is now being conducted at the Reading Clinic, University of Pennsylvania by our colleague, Mrs. Margaret Willson. She is testing the hypothesis that the following matches are desirable between primary cause of reading disability and instructional mode: 1. educational factors predominate -- basal reader; 2. psychological factors predominate -- linguistic readers; 3. neurological factors predominate -- Fernald language experience approach.

A FINAL NOTE

In the fall of 1968, I shall have completed a monograph on dyslexia--a "state of the art" publication commissioned by ERIC/CRIER and IRA. This research report will analyze (1) the classical literature, (2) the relevant research literature indexed by ERIC/CRIER from 1950-1966, and (3) the periodical literature for the years 1963-4-5 on this subject. The latter group of several hundred items have been located,
abstracted, and indexed by my colleague, Mrs. Jane Levine at the University of Pennsylvania Reading Clinic. Her abstracts will be included in the monograph.
REFERENCES


