THE LEARNING DISABLED CHILD: GENETICALLY DEFICIENT OR BRAIN INJURED?

BY

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THE LEARNING DISABLED CHILD: GENETICALLY DEFICIENT OR BRAIN INJURED?

A Research Paper

Presented to

the Graduate Council of

Austin Peay State University

In Partial Fulfillment

of the Requirement for the Degree

Master of Arts

in Psychology

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by

Ruth Fuller Lature

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To the Graduate Council:

I am submitting herewith a Research Paper written by Ruth Fuller Lature entitled "The Learning Disabled Child: Genetically Deficient or Brain Injured?" I recommend that it be accepted in partial fulfillment of the requirement for the degree of Master of Arts, with a major in Psychology.

Major Professor

Accepted for the Graduate Council:

Dean of the Graduate School

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CHAPTER I

INTRODUCTION

Although learning disabilities is relatively new as a comprehensive field of study, the problem of learning disabilities in children is not new (Lerner, 1972). The history of dyslexia, one form of a learning disability, dates back to 1887 and Dr. Berlin of Stuttgart, Germany (Rawson, 1971). In the late 1800's, an English opthalmologist, described a condition he called "word blindness," which basically involved an inability to read. In the 1900's, other specialists such as Orton, a neuropathologist, and McGinnis, a speech pathologist, became interested in speech and language disorders and their relationship to learning. Learning disabilities as a specialized field is considered to have begun in 1947 with the publication of Psychopathology and Education of the Brain Injured Child by Alfred Strauss and Laura Lehtinen. What is new in our present age is the increasing recognition of these learning difficulties and the movement by parents, schools, and the Federal government to offer services to children who are not developing and progressing adequately due to learning disabilities (Lerner, 1972).

Estimates of the prevalence of children who suffer from learning disabilities range from 1% to 30% of our school population (Lerner, 1972). Feingold (Note 1) states that some 10,000,000 children in the United States are

considered deficient in their learning abilities. Hanby and Stiles (Note 2) found the incidence of learning disabilities in the state of Washington to range from a low of 1.5% at the sixth grade level to a high of 5.4% at third grade. Thompson (1966) reports several recent estimates to be over 10%. The National Advisory Committee on Handicapped Children recommended to Congress that 1 to 3% of the school population be considered learning disabled (Lerner, 1972).

Any estimate of incidence will depend upon the criteria used in determining who is learning disabled. A survey of the literature indicates no clear professional unanimity in determining what constitutes a learning disability. The official definition of learning disabilities, formulated by the U. S. Congress when it passed the Children with Specific Learning Disabilities Act of 1969, incorporates views from several perspectives:

> Children with specific learning disabilities exhibit a disorder in one or more of the basic psychological processes involved in understanding or in using spoken or written language. These may be manifested in disorders of listening, thinking, talking, reading, writing, spelling, or arithmetic. These include conditions which have been referred to as perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, developmental aphasia, etc. They do not include learning problems which are due primarily to visual, hearing, or motor handicaps, to mental retardation, emotional disturbance or to environmental disadvantage (Lerner, 1972, p. 321).

In addition to the term, "learning disabilities," and terms mentioned in the above definition, this condition

has been referred to as analfabetica partialis, congenital word blindness, congenital dyslexia, alexia, strephosymbolia, severe learning disability, minimal brain damage, reading disability, neurological impairment, hyperkinesis, and specific-perceptual motor disability. These terms are often used interchangeably. In a survey of 814 references, Thompson (1971) found 60% of the references used the term, "reading disability." The second most frequently used term was "dyslexia," followed by "learning" and/or "language disabilities" in third place, and "brain damage" in fourth place.

McCarthy and McCarthy (1971) feel that causation is generally considered the most controversial issue in the field of learning disabilities. They speak of "professional convictions," but they feel there is little in the way of firm data to support a given point of view.

Frierson and Barbe (1967) propose that learning disabilities are due to a known impairment in the nervous system. According to these writers, this impairment may be the result of "genetic variation, biochemical irregularity, perinatal brain insult, or injury sustained by the nervous system as a result of disease, accident, sensory deprivation, nutritional defect, or other direct influence" (p. 4).

Critchley (1964) further points out that as long ago as 1905 it was observed that congenital word-blindness

might involve more than one member of a family. Further summarizing some of the thinking relating genetics to learning disabilities, Critchley states:

> The pioneer here was C. J. Thomas who found six patients within two generations of a single family. In the same year (1905) F. Herbert Fisher recorded congenital word-blindness in an uncle and a nephew. The following year S. Stephenson went so far as to postulate a recessive mode of inheritance on the basis of six cases cropping up in three generations (p. 63).

Hallgren (1950) studied 276 cases of reading disability, along with 212 controls. He found reading disabilities in 88% of the families of his reading disabled group and concluded that "reading problems are genetically determined and follow a dominant mode of inheritance" (p. 285).

Lending support to the theory that learning disabilities are genetically determined is the research of Hermann and Norrie (1958) who report that in a study of 45 sets of twins, at least one twin having a reading disability, there was a 100% concordance among identical twins. Of the 33 sets of non-identical twins studied, the incidence of learning disability was only 33%.

While there does appear to be a genetic basis for at least some cases of learning disabilities, a survey of the literature provides no clear connection between the two. According to Critchley (1964) "no connection has yet been found between chromosomal aberrations, as based upon recent techniques of intra-cellular chromosome counting" (p. 65).

Glaser (1974) feels that learning disabilities may result from biochemical irregularities although he admits that such irregularities have not been proven. For the most part, Glaser generalizes from biochemical abnormalities in animals to human organisms. It is his contention that certain varieties of mental deficiency are based on neurochemical disorders associated with amino acids, carbohydrates, and lipid disorders and that these same factors may be causative factors in learning disabilities.

Allen (Note 3) studied 135 hospitalized, brain damaged children. From studying samples of skin, muscle tissue, and blood of these children, she found that over 90% showed deficiencies of metabolic function. She concluded that "Enzyme imbalance has been found to play a part in many types of diseases, including hypoglycemia, diabetes, PKU, galactosemia, schizophrenia, muscular dystrophy, learning disorders, so-called brain damage, gout and others." Dr. Allen feels this intracellular, metabolic function is genetically linked.

Shedd and Mercke (Note 4) report remarkable improvement in the ability to attend and concentrate in learning disabled children who have been placed on a low carbohydrate diet (60 to 120 grams per day). They, therefore, conclude that learning disabled children are different from normal children because of a neurochemical malfunctioning associated with carbohydrate metabolism.

Prenatal environment has been associated with learning disabilities by some researchers. Di Leo (1969) relates prenatal causes to learning disabilities in his statement:

> Intrauterine development may be disordered, impaired, or impeded by noxious agents. Potentially damaging are radiation, viruses, drugs, and oxygen deprivation. Most cases of minimal cerebral dysfunction properly fall into this etiologic grouping (p. 6).

Critchley (1964) credits A. A. Kawi and B. Pasamanick as being the most articulate exponents of a maternal aetiology. Critchley refers to a study by Kawi and Pasamanick in which they found 16.6% of 205 children with reading problems had mothers with complications during pregnancy such as pre-eclampsia, bleeding, or hypertension. Of a control group of normal readers, maternal incidents of this kind occurred in only 1.5%.

Prechtl (1962) studied 50 cases of children with learning disabilities. According to parent reports, in 50% of these cases pregnancy was complicated by "toxemia, severe bleedings, etc." (p. 192).

Conan Kornetsky (1975) also feels that "There is reason to believe that specific toxic substances ingested during pregnancy or during early infancy could be responsible for some of the later behavioral manifestations" (p. 458). Kornetsky refers to one study in which prenatal administration of chlorpromazine resulted in a lower convulsive threshold in the offspring.

Another theory of the possible cause of learning disability has been birth injury. Critchley (1964) notes that J. H. Fisher in 1910 was the first person to suspect that birth injury might constitute a factor in the genesis of dyslexia.

Frierson and Barbe (1967) feel that anoxia, or oxygen deprivation at birth, is commonly recognized as one of the prominent causes of learning disabilities. They recognize that anoxia is often associated with premature births. In premature births, the breathing reflex may be intermittent or shallow because of neural underdevelopment. They emphasize the need for controlled oxygen supply inasmuch as excessive oxygen or irregularities in oxygen supply also has been associated with neural impairment. Furthermore, they postulate:

> Birth injury may be due to factors involved with the labor or, in a minor percentage of cases, it may arise in the use of obstetrical instruments. If there is an unusual presentation in delivery and prolonged labor, there may be a compression of the infant's skull. The damage to the brain in such a case may stem from a cerebral hemorrhage or from a direct injury to the brain tissue. The probability for injury in post-mature births appears to be higher than for normal-term deliveries (p. 113).

Koupernik, MacKeith, and Williams (1975) agree with Frierson and Barbe that to be born early puts a baby's brain at risk, but they feel that with intensive care 80% of preterm babies will be normal. They also feel that a lack of oxygen, both from placental disorders and from the hazards of delivery, could be related to learning disabilities.

Just as there is risk with the premature, Koupernik, MacKeith, and Williams feel the post-term infant is liable to intrauterine anoxia from placental insufficiency. They express no doublt that there will be some anoxia as the head of the baby is molded through the birth passage, but they point out that Caesarean section does not evade all the risks of delivery.

In Prechtl's (1962) case reports from 50 parents, he found neonatal disturbances in 46% of a selected group of infants: 26% of the infants had been treated for asphyxia, 14% had difficulties in sucking and had a low body temperature, and 8% were premature.

Schubert (1969) summarizes the complications of birth or labor which may be associated with learning disabilities: (a) Caesarean section; (b) premature birth; (c) prolonged labor--breech presentation, high forceps delivery; (d) dry birth; (e) precipitious birth; (f) improper use of anesthesia; (g) asphyxia from various causes.

Frierson and Barbe (1967) consider head injuries in infancy and childhood to be an important cause of brain injury. They feel that automobile accidents, bicycle accidents, and sidewalk falls contribute to learning disabilities, although the percentage of children who suffer permanent damage is small.

Of the 50 children that Prechtl (1962) studied, 60% were found to have a history of illness in their postnatal development: 28% had episodes of cyanosis in early life,

complicated pneumonia or pertussis infections; 12% had frequent epileptic attacks; 38% had a history of accidents with concussion. The fact that 38% had a history of accidents lead Prechtl to call learning disabled children "accident-prone."

The importance of nutrition during the first years of life has been emphasized. Glaser (1974) points out that malnutrition during the first years of life will affect brain size and DNA content of the brain. Montagu (1972) reports on the amount of DNA in the brains of children who died of malnutrition as compared with the amount in the brains of normally nourished children:

> They found the amount of DNA in the brains of the malnourished children to be significantly less, indicating the presence of a substantially smaller number of brain cells. Head circumference, brain weight, and protein content, were all reduced (p. 1050).

Concerning nutrition and brain development, Montagu (1972) also speaks of "sociogenic brain damage." He discusses nutrition and the developing fetus as well as in the young child:

> It is generally agreed that the most important factor in the healthy development of the conceptus is nutrition--not merely the nutrition derived from the mother, but also the nutrition of the mother's mother, and probably also of the mother's father, not to mention the child's own father (p. 1046).

Montagu goes on to state that:

> ...The evidence clearly indicates that during the first three years, when the basic foundations and organization of the brain are in process

> of construction, inadequate provision and poor quality of experience may seriously affect the fabric of the brain.... In such cases, the brain and mind are rendered incapable of later organization at levels of cognitive integration matching those achieved by others who have not suffered such sociogenic damage (p. 1058).

Koupernik, MacKeith, and Williams (1975) describe postnatal trauma from traffic collisions and from falling as causes in learning disabilities. Non-accidential injury of child abuse is also included. They also list infections such as meningitis. They feel that meningitis occurring in the first year of life is more likely to be followed by lasting sequelae such as epilepsy, cerebral palsy, or mental handicap. According to these writers, encephalitis is often blamed but they feel it is probably a rarity.

Rocky Mountain spotted fever has been investigated as a possible cause of learning disabilities. Wright (1972) tested 12 post-Rocky Mountain spotted fever patients and 12 matched controls. Significant differences were obtained on seven of 13 variables included in the Wechsler Intelligence Scale for Children and on two of five Frostig subtests. No differences were obtained on the Bender-Gestalt. Wright summarizes, "It was concluded that RMSF exerts a mild but consistent effect on intellectual functioning. This in turn suggests a higher probability of learning disability..." (p. 315).

In a summary of postnatal influences, the following designations are made by Schubert (1969): (a) encephalitis

(particularly measles encephalitis); (b) meningitis; (c) high fever with delirium; (d) head injury involving unconsciousness (particularly before the age of three); (e) poisons resulting in unconsciousness; (f) burns involving large areas of the body surface; and (g) excessive crying or head banging during the first year of life.

The aforementioned studies clearly indicate a difference of opinion among researchers and writers regarding causation in learning disabilities. One group of writers and researchers propose that learning disabilities are inherited. At the same time, others speculate that learning disabilities are due to abnormalities or injuries prenatally, perinatally, or postnatally. By understanding the cause or causes of learning disabilities, a program of prevention can best be instituted.

Inasmuch as the cause of learning disabilities is such a controversial issue, the present study was directed toward determining what percentage of learning disabilities might be linked to inheritance and what percentage might be associated with abnormalities prenatally, perinatally, and postnatally in a selected sample of learning disabled children.

As has been previously indicated, much confusion exists concerning children who cannot learn commensurate with their intellectual capabilities. This confusion is most evident in regard to definition of the learning disabled. For the purpose of this study, a learning disabled child is one

who shows evidence of the following characteristics: (a) spotty performance on IQ tests--achievement high in some areas, low in others; (b) below mental age on tests of drawing a person; (c) poor visual motor Gestalt tests for age and indicated intelligence; (d) poor performance on group tests which require reading and writing; (e) impaired temporal orientation; (f) impaired right-left discrimination; (g) poor spatial orientation; (h) field dependent perception; (i) frequent perceptual reversals in reading and in writing numbers beyond age and instructional level; (j) impaired reproduction of tonal pattern; (k) impaired auditory discrimination; (l) impaired reproduction of rhythmic patterns; (m) frequent mild speech irregularities; (n) nonspecific motor awkwardness; (o) periodic loss of fine motor skills; (p) reading disabilities; (q) spelling disabilities; (r) writing disabilities; (s) variability in performance; (t) poor ability to organize work; (u) slowness in finishing work; (v) short attention span for age; and (w) impaired concentration ability.

These 23 characteristics used as criteria for diagnosis of learning disability were assessed through the use of the following tests: Slosson Intelligence Test, Peabody Picture Vocabulary Test, Goodenough-Harris Draw-a-Man Test, test of directionality developed by Shedd and Drake (Jones, 1969), Berea Gestalt test developed by Shedd and Drake (Jones, 1969), Gilmore Oral Reading Test, and the Johnson

Handwriting Test. In addition, parents were asked to fill out a questionnaire, covering 83 items.

Other terms in this study are defined as follows: (a) prolonged labor--labor continuing for longer than 24 hours; (b) precipitious labor--labor continuing for less than four hours; (c) high forceps--forceps reaching high into the birth canal; and (d) extremely high fevers--fevers over 104 degrees which continue for more than three hours.

CHAPTER II

METHOD

Subjects

Subjects were 98 students, ranging in ages from 5 to 20, who had been referred to the Christian County Association for Specific Perceptual Motor Disability in Hopkinsville, Kentucky. All subjects had been diagnosed as learning disabled according to the criteria previously listed on pages 11-13. The diagnosis of learning disability was made by the late Dr. Charles L. Shedd, psychologist, or by another psychologist on his staff.

Instrumentation

The instrument used in this study was a Yes-No Questionnaire devised by the late Dr. Shedd (see Appendix A). Each subject's parent completed the Questionnaire.

Procedure

Case files of the Christian County Association for Specific Perceptual Motor Disability were surveyed. All cases with a diagnosis of learning disability were chosen for this study. Yes-No answers to selected questions on a parent Questionnaire were tabulated. A percentage of Yes answers to each selected item was tabulated. A Yes answer indicated a positive relationship between the factor in question and the learning disabled child being assessed.

In figuring percentages, the total Yes-No response was used, rather than the total N of 98, as in a few instances, a parent chose not to answer a particular item.

CHAPTER III

RESULTS

Parental responses to selected items on the Questionnaire are summarized in the following table in terms of the total number of responses to each question and percentage of cases in which the factor was associated with a learning disability.

Table 1

Total Number of Responses and Percentage of Responses Associated with each Abnormality

	Question	N	%
1.	Did any of the siblings have any physical or learning disabilities?	77	30%
2.	Was the mother's health poor during pregnancy?	93	10%
3.	Did the mother have difficulty carrying child to term?	95	20%
4.	Did the mother incur accident or injury during pregnancy?	95	3%
5.	Was labor prolonged?	87	21%
6.	Was labor precipitious?	77	27%
7.	Were high forceps used?	78	22%
8.	Was the birth Caesarian?	92	8%
9.	Was the child discolored at birth?	86	6%
10.	Was this a breech presentation?	86	5%
11.	Did the child require oxygen at birth?	86	12%
12.	Has the child ever been knocked unconscious?	92	10%
13.	Has the child had convulsions?	95	9%
14.	Has the child had extremely high fevers for prolonged periods?	96	18%
15.	Is there any history of epilepsy in either family?	93	13%

CHAPTER IV

DISCUSSION

In the present study, the percentage of siblings with learning disabilities (30%) differed from findings of Hallgren (88%), previously reported. However, incidence of learning disability among siblings in the present study did not differ from the findings of Herman and Norrie (1958) who found that 33% of the non-identical twins they studied had reading problems. On the other hand, a higher percentage was found in the present study than reported by Shedd (1967) who indicated that from his research "Dyslexia appears in siblings in 17-20% of the cases" (p. 160). It is not known whether Hallgren included only siblings or siblings and relatives in his data. As learning disabilities follow no definite hereditary pattern, they may be expressed at the grandparental or great-grandparental level without being present in the subject's immediate family (Note 4). If Hallgren traced the hereditary trend beyond the child's immediate family, this could influence the percentage results. To obtain an accurate representation of a possible genetic link, both paternal and maternal parents and their offspring should be considered. A weakness of the present study is that siblings alone were considered in the family history. Another limitation of this aspect of the present study is that siblings may have had an undiagnosed learning disability. In collecting data for this study, the researcher

observed situations in which a parent would indicate on the Questionnaire that no other sibling in that family had a learning disability. Then later another child or children from that same family would be diagnosed as learning disabled. Therefore, because of unknown cases of learning disabilities among siblings at the time the parent made his response on the Questionnaire, the inherited incidence of learning disabilities may be greater than the 30% incidence obtained in this study.

Data regarding prenatal, perinatal, and postnatal abnormalities associated with learning disabilities were compared with the findings of Shedd (1967), Paine (1965), and Prechtl (1962). It will be noted that Prechtl obtained higher percentages than the present study or Shedd and Paine on some abnormalities. Prechtl's subjects differed in that they all showed choreiform movements. By choreiform movement he means "slightly jerky movements, occurring quite irregularly and arhythmically in different muscles" (p. 189). It could be that Prechtl's subjects were a select group, rather than a representative sampling of learning disabled children, thus accounting for his higher percentages.

In comparing percentages of prenatal factors associated with learning disabilities, a study by Kawi and Pasamanick (Critchley, 1964) should be used as a guide. These researchers found that maternal incidents occurred in 1.5% of children experiencing reading problems. The present

writer also studied the relationship of maternal abnormalities occurring in the learning disabled children included in this sample. However, these incidents are broken down into specific maternal complications.

Concerning the mother's poor health during pregnancy, Shedd (1967) found poor health in 2% of 99 cases; Paine (1965) reported bleeding during pregnancy in 4% of 48 cases; and Prechtl (1962) wrote that 50% of his 50 cases had mothers with complications of "toxemia, severe bleeding, etc." (p. 192). In the present study, 10% reported the mother's health as poor during pregnancy.

In regard to prematurity, Prechtl described 8% of his cases as falling into this category; Paine reported 10%; and Shedd found only 2%. Twenty percent of mothers in the present study had difficulty carrying the child to term.

Shedd reported none of the mothers in his study to have experienced accidents or injuries during pregnancy. In the present study, 3% reported accidents or injuries during pregnancy.

Paine listed 10% of his mothers as having prolonged and difficult labor (48 hours). Shedd found prolonged labor in 10% of his cases. Prolonged labor is reported in 21% of the cases in the present study.

In regard to precipitious labor, Shedd reported this factor present in 10% of his cases. Precipitious labor is associated with 27% of the cases in the present study.

Shedd reported high forceps used in 10% of the cases in his study. In the present study, high forceps were used in 22% of the cases.

Paine found Caesarian delivery in 6% of his cases; Shedd reported 2% to have had Caesarian delivery; and in the present study, 8% were reported.

In Shedd's 99 cases, none were reported to have been discolored at birth. However, in the present study, 6% of the subjects were reported to have been discolored at birth.

Prechtl reported that 26% of his cases had been treated for severe anoxia. Shedd observed that oxygen was required at birth in 3% of his cases. In the present study, 12% of the infants were reported to have required oxygen at birth.

In the present study, breech presentation was reported in 5% of the cases. The only figures available for comparison is Paine's "abnormal presentation, difficult delivery" category in which he lists 15% of his subjects.

Prechtl reported a history of accidents with concussions in 38% of his cases. Paine recorded severe head injury in only 2% of his subjects. Shedd reported that 10% of his subjects had been knocked unconscious. In comparison, 10% of the subjects in the present study were reported to have been knocked unconscious.

Concerning convulsions, Shedd reported that 1% of his subjects once had convulsions. Prechtl found that 12%

of his subjects had frequent epileptic attacks. In the present study, 9% of the parents said the subject had experienced convulsions. In addition, 13% reported a history of epilepsy in either the father's or mother's family.

Shedd found 10% of his subjects to have experienced extremely high fevers for prolonged periods. In the present study, 18% of the subjects had suffered extremely high temperatures for extended periods of time.

A possible limitation of the present study was a lack of guidance in completing the Questionnaire. An effort was made to have someone present to answer questions as the Questionnaire was being filled-in. However, in some instances there may not have been anyone easily accessible to give assistance. In addition, parents may be mistaken concerning details surrounding the birth process. A number of questions were not answered, indicating that the parent simply did not know the answer.

Conclusions

On the basis of the present study as compared with the work of Shedd (1967), Paine (1965), and Prechtl (1962), one cannot conclude that learning disabilities are an inherited entity. Neither can one conclude that some abnormality before, during, or after birth is associated with, as a possible cause, all cases of learning disabilities. Causation in learning disabilities appears complex. The data would suggest that some cases are inherited; however

in other cases, there appeared to be an abnormality prenatally, perinatally, or postnatally which could have caused the learning disability.

Recommendations

Recommendations for further research in the area are:

1. It is doubtful that accurate information can be obtained with the direct questionnaire. Concerning this Bryant (1966) says:

> Frequently, a family will not volunteer that the father, two uncles, and all male siblings have shown a learning problem similar to the one exhibited by the child. So the social worker must actively seek such information (p. 270).

A better approach might be the written questionnaire and a personal interview. Therefore, where a parent seems uncertain of information, that information can be further investigated or thrown out.

2. A control study should be done to determine what percentage of children without learning problems have abnormalities prenatally, perinatally, and postnatally.

3. In inherited cases of learning disabilities, the same unknown factor or factors causative in the learning disability may be causative in some of the abnormalities prenatally and perinatally such as prolonged or precipitious labor, prematurity, or the need for Caesarian delivery. An investigation should be made of cases of learning disabilities where there is a family history of learning disability to see if there are also present indications of prenatal or perinatal irregularities.

Reference Notes

1. Feingold, B. F. Hyperkinesis and learning disabilities (H-LD) linked to the ingestion of artifical food colors and flavors. Paper presented before the Senate Subcommittee on Health, Education, and Welfare, Washington, D. C., September 1975. (Available from Sen. Ted Kennedy, Washington, D. C.)

2. Hanby, V. L. & Stiles, R. L. An estimate of the incidence of learning disabled students in the state of Washington and the effectiveness of teachers in identifying that population. Paper presented at the International Reading Association Convention, Anaheim, California, May 1976.

3. Allen, M. Personal communication, September 1975.

4. Shedd, M. & Mercke, M. L. Personal communication, September 1975.

5. Shedd, C. Personal communication, October 1973.

References

Critchley, M. Developmental dyslexia. London: William Heinemann Medical Books, Limited, 1964.

Bryant, N. D. Clinic inadequacies with learning disorders--the missing clinical educator. In Learning disorders, Vol. 2, ed. Jerome Hellmuth. Seattle, Washington: Special Child Publications, 1966.

Di Leo, J. H. Early detection of developmental disorder. In Learning disabilities ed. Doreen Kronick. Chicago: Developmental Learning Materials, 1969.

Frierson, E. C. & Barbe, W. B. Educating children with learning disabilities. New York: Appleton-Century Crofts., 1967.

Glaser, K. Learning difficulties: causes and psychological implications. Springfield, Illinois: Charles C. Thomas, 1974.

Hallgren, B. Specific dyslexia. Acta Psychiatric Neurology, Supplement No. 65, 1959, pp. 1-287.

Hermann, K. & Norrie, E. Is congenital word-blindness a hereditary type of Gerstman's syndrone? Psychiatric Neurology, Vol. 136 (1958), p. 59.

Jones, J. Dyslexia: identification and remediation in a public school setting. Journal of Learning Disabilities, Vol. 2 (1969), pp. 44-49.

Kornetsky, C. Minimal brain dysfunction and drugs. In Perceptual and learning disabilities in children, Vol. 2, ed. W. M. Cruickshank. Syracuse University Press, 1975.

Koupernik, C., MacKeith, R., & Williams, L. F. Neurological correlates of motor and perceptual development. In Perceptual learning disabilities in children, Vol. 2, ed. W. M. Cruickshank, Syracuse University Press, 1975.

Lerner, Janet W. Learning disabilities: a school health problem. Journal of School Health, Vol. 42 (1972), pp. 320-325.

McCarthy, J. J. & McCarthy, J. F. Learning disabilities. Boston: Allyn & Bacon, Inc., 1971.

Montagu, A. Sociogenic brain damage. American Anthropologist, Vol. 74 (1972), pp. 1045-1061.

Paine, R. S. Organic neurological factors related to learning disorders. In Learning disorders, Vol. 1, ed. J. Hillmuth. Seattle, Washington: Special Child Publications, Inc., 1965.

Prechtl, H. F. R. Reading difficulties as a neurological problem in childhood. In Reading disability, progress and research needs in dyslexia, ed. John Money. Baltimore: The Johns Hopkins Press, 1962.

Rawson, M. B. Perspectives of specific language disability: I. the past--what has been learned? Bulletin of the Orton Society, Vol. 21 (1971), pp. 22-34.

Schubert, D. G. Diagnosis in severe reading disability. In Reading disability and perception, ed. George Spache. Newark, Delaware: International Reading Association, 1969.

Shedd, C. L. Some characteristics of a specific perceptual-motor disability--dyslexia. The Journal of the Medical Association of the State of Alabama, Vol. 37 (1967), pp. 150-162.

Thompson, L. J. Reading disability, developmental dyslexia. Springfield, Illinois: Charles C. Thomas Publishers, 1966.

Thompson, L. J. Remarks by Dr. Thompson. Bulletin of the Orton Society, Vol. 21 (1971), p. 8.

Wright, L. Intellectual sequelae of Rocky Mountain spotted fever. Journal of Abnormal Psychology, Vol. 80 (1972), pp. 315-316.

APPENDIX A

QUESTIONNAIRE

1. Do any of the siblings have any physical or learning disabilities?
2. Was the mother's health poor during pregnancy?
3. Did the mother have difficulty carrying child to term?
4. Did the mother incur accident or injury during pregnancy?
5. Was labor prolonged?
6. Was labor precipitious?
7. Were high forceps used?
8. Was the birth Caesarian?
9. Was the child discolored at birth?
10. Was this a breech presentation?
11. Did the child require oxygen at birth?
12. Has the child ever been knocked unconscious?
13. Has the child had convulsions?
14. Has the child ever had extremely high fevers for prolonged periods?
15. Is there any history of epilepsy in either family?