THE PERFORMANCE OF CHILDREN DIAGNOSED AS HAVING MINIMAL BRAIN DYSFUNCTION ON AN AUDITORY DISCRIMINATION TASK

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CHAPTER I

PROBLEM DEFINITION

Introduction

The concept of brain dysfunction as a primary causative factor in learning and behavior disorders of children has received increasing attention over the past 20 years, especially in the fields of medicine, psychology, education, and the language specialties.

Brain dysfunction can manifest itself in varying degrees of severity and can involve any or all of the more specific areas, e.g., motor, sensory and intellectual. The term minimal brain dysfunction (MBD) is often used to describe this condition in children and refers to the child whose symptomatology appears in one or more of the specific areas of brain function but which is in a mild form and which does not reduce overall intellectual functioning to the subnormal ranges (Clements, 1966). It has been used to describe a group of abnormalities which are believed to comprise a childhood syndrome. The principal manifestations of this syndrome include: reading disability (dyslexia), short attention span, hyperactivity, history of or presence of right-left confusion in writing, poor motor coordination and impulsiveness (Peters, 1974). At present this is an area in which there is much confusion and disagreement among professionals. At one extreme there are those who believe in the concept of MBD (e.g., Clements and Peters, 1962; Dykman, 1970) and at the other there are those (Cruickshank, 1971; Schrag and Divoky, 1975) who believe that this is a term that has no scientifically demonstrable meaning and is used by professionals to describe those children whose behavior is regarded as troublesome to adults.

The present study will concentrate on one area of deficit thought by both medical and educational specialists to be common in MBD children---Auditory Perception. Within this area the present study will concentrate on one parameter---Auditory Discrimination.

Perception has been defined by the Arkansas State Department of Education (1964) as the process by which the brain receives various stimuli from the sensory organs and arranges these into meaningful mental images and concepts. Auditory Perception deals with the stimuli received by the brain through the ears and arranged into organizational patterns and then into concepts by the brain. These concepts are presumed to be stored in the brain for later recall or transmitted to the vocal or motor mechanism of the body for immediate action. Any breakdown or weakness in this system, therefore, will impair the reception, storage, or expression of spoken symbols. Van Riper (1963) hypothesizes that our own inability to remember the happenings of the first two years of life is due to the fact that then we had no language symbols with which to file them in storage.

The present study will not be concerned with the loss or lack of acuity but rather with the child who as Hardy and Pauls (1959, p. 14) state "can hear but is deaf". Such a child can respond to sound but cannot discriminate or remember what he hears. The child who is developing language and who cannot, because of his impairment, perceive words does not have aphasia but rather auditory agnosia, defined by Clements (1966, p. 3) as "a condition in which sound is received by the brain but not interpreted and so has no meaning attached". After the reception of an auditory stimulus the first step in interpretation of this stimulus is auditory discrimination which is defined by Wepman (1960) as the ability to recognize or distinguish between individual sounds in speech. He further states that this ability is not to be confused with the gaining of meaning from words. It is the distinguishing of sounds of a spoken language, even when the sound wave patterns are highly similar. Auditory discrimination as defined by Lerner (1971) is the ability to recognize a difference between words and to identify words that are the same and words that are different. Auditory discrimination in MBD children would therefore be the ability of these children to distinguish between sounds of a spoken language that are highly similar and the ability to identify words that are the same and words that are different. It has been assumed, by medical and educational specialists that many of the children diagnosed as having MBD have auditory perceptual deficits which cause both communication and academic difficulties. In reviewing the literature on auditory discrimination however, there is not a single study to either support or dispute this assumption. This indicates an obvious need for further research in this area.

Statement of the Problem

The primary purpose of the study then, is to compare auditory discrimination in children diagnosed as having minimal brain dysfunction to auditory discrimination in school children of the same age who appear to be "normal". To study this twenty-six MBD children were matched to twenty-six normals on the basis of age, sex, race, I.Q., socioeconomic status and the absence of any known debilitating emotional disturbance. All children had normal hearing. The Wepman Auditory Discrimination Test was used which has thirty similar word pairs, e.g., "cat - cap" and ten identical word pairs, e.g., "tall - tall". Ten dissimilar word pairs were developed by the author, e.g., "lake - girl". These word pairs were put on auditory tape and presented to both groups of children. The children listened to these word pairs through head phones and responded by pressing a telegraph key marked "same" and "different".

Two experimental conditions were presented: The first condition was the effect of lengths of time between the presentation of the first word and the second word in the word pairs. Two time delay conditions were used:

(1) Delay one, which had a time lag of .5 of a second between the presentation of the first word and the second word.

(2) Delay two, which had a time lag of 5 seconds between the presentation of the first and second word.

The second condition was concerned with the effects of fatigue. To study this, the two delay conditions were placed into three replicates. Each replicate contained one set of fifty word pairs with .5 of a second delay and one set of fifty word pairs with 5 seconds delay. This made a total of three hundred word pairs for each child to discriminate. This condition investigated whether or not there was a significant difference in response errors and nonresponses to the word pairs across replicates between these two populations.

CHAPTER II

REVIEW OF THE LITERATURE

History of the Concept of MBD

Clements (1966) in a review of the literature on minimal brain dysfunction (MBD) notes that prior to 1920 the literature is sparse and is generally concerned with observations on individuals who sustained damage to the brain after reaching adulthood. Early references, e.g., Burr (1921) and Miles (1921), describe "nervous conditions" in children which affect learning and behavior. Many papers appearing between World War I and World War II were descriptive forerunners of minimal brain dysfunction. A large number of these studies, e.g., (Blau 1937; Bond 1932; Hohman 1922) were devoted to the linkage between specific causative agents and resulted changes in behavior.

The work of Gesell and Amatruda (1941), Werner and Strauss (1941), Werner and Thuma (1942), Werner and Weid (1956), Strauss and Werner (1942), and Strauss (1944) set the stage for the concept of minimal brain dysfunction that is employed today. From Strauss and Lehtinen's classic work, <u>Psychopathology and Education of the Brain Injured Child</u> (1947) came the first comprehensive presentation on the topic. It represented the essence of 20 years of previous research and is still the reference that is most frequently cited by researchers. It has been influential in the production of new considerations (e.g., hyperactivity, dyslexia, impulsiveness, intelligence, etc.) in the areas of pathology, diagnosis, education, and investigation of children with learning and behavioral disabilities. It revitalized interest in the neglected areas of individual differences among children. Since 1950, the literature has shown a steady increase in the number of clinically oriented studies of these disabilities under the general concept of minimal brain dysfunction in children.

Clements (1966, p. 9) in a selected review of the literature revealed a total of 37 terms used to describe or distinguish the conditions grouped as minimal brain dysfunction. He grouped these terms into two categories:

Group I --- Organic Aspects

Association Deficit Pathology
Organic Brain Disease
Organic Brain Damage
Organic Brain Dysfunction
Minimal Brain Damage
Diffuse Brain Damage
Neurophrenia
Organic Drivenness
Cerebral Dysfunction
Organic Behavior Disorder
Choreiform Syndrome
Minor Brain Damage
Minimal Brain Injury
Minimal Cerebral Injury
Minimal Chronic Brain Syndromes
Minimal Cerebral Damage
Minimal Cerebral Palsy
Cerebral Dys-synchronization Syndrome

Group II --- Segment or Consequence

Hyperkinetic Behavior Syndrome
Character Impulse Disorder
Hyperkinetic Impulse Disorder
Aggressive Behavior Disorder
Psychoneurological Learning Disorders
Hyperkinetic Syndrome
Dyslexia
Hyperexcitability Syndrome

Perceptual Cripple
Primary Reading Retardation
Specific Reading Disability
Clumsy Child Syndrome
Hypokinetic Syndrome
Perceptually Handicapped
Aphasoid Syndrome
Learning Disabilities
Conceptually Handicapped
Attention Disorders
Interjacent Child

Clements (1966) noted that the most striking omission throughout the literature was the lack of a definition of the terms used or the conditions discussed. Previously, Clements and Peters (1962) had developed a 38th term, which is the one that has now had widespread use---minimal brain dysfunction. Their 1962 article "Minimal Brain Dysfunctions in the School-Age Child" was influential in establishing the concept of and widespread use of the term.

The group of symptoms included by Clements and Peters (1962) under the term minimal brain dysfunction stems from disorders which may manifest themselves in severe forms. The child with minimal brain dysfunction may exhibit the following minor symptoms in varying degrees and in varying combinations (Clements 1966, p. 10):

Minimal (minor; mild)	Major (severe)
1. Impairment of fine movement or coordination.	1. Cerebral palsies.
2. Electroencephalographic abnormalities without actual seizures, or possible sub-clinical seizures which may be associated with fluctuations in behavior or intellectual function.	2. Epilepsies.
3. Deviations in attention, activity level, impulse control, and affect.	3. Autism and other gross disorders of mentation and behavior.

4. Specific and circumscribed perceptual, intellectual, and memory deficits.	4. Mental subnormalities.
5. Nonperipheral impairments of vision, hearing, haptics, and speech.	5. Blindness, deafness, and severe aphasias.

In December of 1972 Peters defined MBD operationally and in behavioral terms (p. 1):

> ...it is the presence of a chronic history of poor control of attention, poor organization of activity, poor control of impulses to act and speak, poor modulation of the expression of emotions, deviations in the control of integrated movements and tonic positions, and circumscribed deficits in cognitive functioning which are inconsistent with the child's overall intelligence.

In 1971 Dykman et al. found a high incidence of "soft signs" (as indicated by a special neurological examination) in their MBD study group. They concluded that a neurodevelopmental lag may be the etiological explanation in most of these cases and that neurological immaturity could explain the attentional deficits of SLD or MBD children.

In a follow-up study (Mendelson, et al., 1971) it was found that a higher incidence of antisocial behavior was present in teenagers who had earlier been diagnosed as hyperactive children. This indicates that the inability to foresee consequences or poor impulse control still lags into the teens.

In another follow-up study Dykman et al., (1973) questioned his earlier hypothesis. They found that MBD children continued to lag at least into their mid teens. The MBD teenagers were as far behind their agemates scholastically as when they were seen initially.

Dykman and Ackerman (1974) state that there is no one single etiologic explanation of MBD. Etiological explanations of MBD have included prenatal and postnatal insults (Rosenfeld and Bradley, 1948; Laufer and Denhoff, 1957); inadequate parenting (Bereiter and Engelman, 1966; Dentsch, 1964); heredity (Critchley, 1964); delayed lateralization of the brain functions (Satz and Sparrow, 1970); delayed neural maturation (Dykman et al., 1971; Lucas et al., 1965; and Solomons, 1965); chemical malfunctions of the brain and/or the brain stem (Shetty, 1971; Stewart, 1970; and Wender, 1972). MBD has also been attributed to allergies as a result of food additivies; (Conners et al., 1973; Feingold, 1973 and 1975); and to florescent lighting and radioactivity from television sets (Arehart-Treichel, 1974).

Dykman and Ackerman (1974) reviewed some of the theorizing concerning the brain mechanisms that underlie MBD. They state that Laufer and Denhoff (1957) espouse essentially an attentional defect theory, i.e., a failure of some essential inhibitory control or filtering mechanism along with a lack of coordination between cortical and subcortical structures. This would mean that the cortex would have insufficient control over the lower regions. Several investigators (Laufer and Denhoff, 1957; Dykman et al., 1971; Satterfield and Dawson, 1971; Stevens et al., 1967; Stewart, 1970; Werry and Spragne, 1969) have implicated the reticular activation system. Some of these researchers hypothesized overarousal and some underarousal in the MBD child.

Dykman and Ackerman (1974) state that they have come more and more to accept Critchley's (1964) position, i.e., at least for specific language disability, heredity is the main etiological factor. Bakwin (1973) found an 84% concordance for reading disability in monozygotic twins compared to a 29% concordance in like sexed dyzygotic pairs.

MBD children have to discover ways of working around their deficits and to develop strategies for recognizing words and associating these words with their speaking (Dykman and Ackerman, 1974). This may entail developing a system of symbols as, for example, the blind person has developed that enables him to recognize words.

Dykman and Ackerman (1974) state that children are not born with an equal potential for developing MBD any more than they are born with an equal potential for becoming schizophrenic (Meehl, 1972). They state that people often react strongly to a genetic interpretation because this leads to a do nothing attitude. However a genetic point of view allows you to recognize a child's limitations and attempt to work around them to produce a productive citizen. To believe that any child can become a doctor, given appropriate experience, is a disservice to the child, his parents, and his teachers.

Opposition to the Concept of MBD

In less than a decade the concept of minimal brain dysfunction (and other names that have been synonymously associated with it, e.g., learning disabilities, hyperkinesis, impulse disorders, etc.) has gone from virtual obscurity to the leading childhood disorder. Before 1965 almost no one had heard of the "disease" and today it is said to afflict as many as forty percent of all American children and be the cause of nearly all school failure, most juvenile delinquency, be a major contributor of broken marriages, and have some part in practically every other social affliction (Schrag and Divoky 1975). In some societies, however, this concept is regarded as extremely rare. Rutter, et al. (1970) studied two thousand London children and identified nine as having neuroepileptic disorders and one as being hyperactive. Bax (1972) studied all five-year-olds on the Isle of Wright (some 1,200 children) and did not find a single MBD or hyperactive child.

In 1966 Clements published a monograph which purported to eliminate professional disagreement in the area of MBD Of the 38 terms which he and his colleagues identified as previously describing the conditions, they chose minimal brain dysfunction as the new official label. The "minimal" suggested an absence of extreme behavior and "dysfunction" eliminated the need to find organic causality (Schrag and Divoky, 1975). The monograph also lists 99 of the most common symptoms found in MBD. The results of this "clarification" was once again confusion and the Clements' definition has been regarded by some as a sophisticated statement of ignorance. The message however is simple, almost any troublesome behavior can be a sign of MBD (Schrag and Divoky, 1975).

Another area of disagreement among professionals is the use of medication for children diagnosed as having MBD or hyperkinesis. Walker (1974) states that the history of medicine is full of treatment fads that have been proven to be not only inappropriate but ridiculous. For example, for centuries physicians used to bleed their patients as the treatment of choice for many diseases, and Pernicious Anemia, a disease which is caused by a vitamin B-12 deficiency, was treated by pulling out all of the patients' teeth. It is Walker's (1974) view that the use of Ritalin and amphetamines to calm hyperactive and MBD children is another disastrous fad, in our own time, that will be recorded in history. In 1971 Ritalin was put under restrictions by the

Bureau of Narcotics and Dangerous Drugs. This prohibited prescription refills. However, according to the National Disease and Therapeutic Index (1974), 623,000 prescriptions were written in 1973 (year ending in September) for Ritalin to treat MBD and hyperkinesis in children. Due to the restrictions on prescription refills, many physicians wrote larger prescriptions, some for as many as 1,000 tablets (Schrag and Divoky, 1975). In an Iowa study, Solomons (1973) found that for any six months period, nearly half the cases sampled were followed up with less than two patient visits or phone calls and that the average term for medication was almost three years.

Grinspoon and Singer (1973) state that before scientists have had a chance to study and refine the issues regarding medication, the field had become the domain of educators and the drug industry. A large proportion of teachers are, as Eric Hoffer (1951 p. 10) states, "true believers". Between 88 percent and 96 percent of teachers believe they can diagnose a hyperkinetic or MBD child and often ask that the child be put on medication (Robin and Bosco, 1973). Boys diagnosed as having MBD outnumber girls with the same diagnosis by a margin of four to one. The explanations for this disproportion (e.g., boys are neurologically more immature, they are more active, they are genetically different, etc.) are as vague as the definitions of the ailment itself. The possibility that all this may be culturally determined does not appear to be serious enough for the diagnosers to reexamine their premises and it appears to be out of the awareness of the true believers (Schrag and Divoky, 1975). Eisenberg (1972) has claimed that the certainty with which convictions are held often varys inversely with the depth of knowledge on which they are based.

Walker (1974) states that a causal relationship has not been established but a suppression of weight and height have been reported with long term use of stimulants in children. It is possible that stimulants produce greater harm in the long run than the symptoms they were meant to control. Insufficient knowledge concerning a medical basis for "brain dysfunction" led the Federal Drug Administration, in 1975, to declare that MBD could not be associated with the prescription of drugs. Therefore, the term MBD would be too vague to be used as the "disease" for which drugs could be used. The symptoms, e.g., short attention span, hyperactivity, etc., which once constituted the separate elements of MBD, would be the behavior that indicated the use of certain drugs. These symptoms had to appear on the labels for Dexedrine, Ritalin, and Cylert (Schrag and Divoky, 1975).

Stroufe and Stewart (1973, p. 409) state that although the concept of MBD has now been widely accepted, the reasoning behind it was circular. That is, "authors have assumed that behaviors such as hyperactivity were signs of brain damage independent of neurologic indexes, and, therefore, that many behavior problem children had brain damage." Walker (1974) states that minimal brain dysfunction is not a disease but merely a label for a constellation of signs and symptoms that can occur for various reasons. Freeman (in Schrag and Divoky, 1975) states that there is simply no such thing as MBD and that everytime this diagnosis is made there is a possibility that it will only mask another ailment.

The preceeding sections have dealt with two very different ways of looking at MBD. Both have merrit and both can contribute to our knowledge and understanding of children who are diagnosed as having minimal brain dysfunction. Questions may be raised as to a reasonable stance that one might take in working with these children and in evaluating information in this general area. Ross (1976, p. 168) and the present author make the following suggestions:

(1) Be skeptical. Don't accept as true the things "everybody knows". Don't ask "Who said so?" but "How do they know?" i.e., ask about their facts not their reputations. So-called authorities can be wrong.

(2) Consider a child who carries the label of MBD or learning disability, as a child who has the ability to learn. If a child fails to learn, look for methods of improving teaching, don't look for something that is wrong inside the child. Many times this has been used as an excuse for poor teaching methods.

(3) Consider terms like dyslexia and hyperactivity as labels that describe behavior and not explanations for the behavior per sa.

(4) Be aware that perceptual and learning deficits are problems for the child and that hyperactivity is primarily a problem for the adults who are involved with the child. The acquisition of knowledge that can be behaviorally demonstrated would better be the goal of effective teaching not sitting still and being quiet.

(5) Since there are many unanswered questions about the effectiveness and long term consequences of behavior altering drugs, consider all possible alternatives before making the decision to use or ask for medication for a specific child.

Auditory Discrimination

In reviewing the literature on auditory discrimination, it was found that there was not a single study in the area of auditory discrimination and children diagnosed as having minimal brain dysfunction. This section will therefore briefly discuss auditory discrimination and a few related studies.

Dysfunction in the auditory modality has been relatively neglected by researchers. Lauretta Bender (1963, p. 25) states that auditory perception "is a much more difficult field to explore than visual perception." Flower (1965) reinforces this in pointing out that we know little of the precise stages or steps the normal child pursues in the development of mature auditory skills. After reviewing clinical data, Sabatino (1969) suggested that the routine assessment of visual perception may contribute to the overlooking of children with serious auditory perceptual impairment.

For auditory stimuli to be meaningful, there must be an intact auditory perceptual system which receives, processes, stores and retrieves information provided by the hearing mechanism (Arkansas State Department of Education, 1964; Mencher and Stick, 1974).

It is this system which permits focusing on words while at the same time blocking out irrelevant stimuli. It enables identification of a single voice, and allows recognition of "cat" as one of the "at" words that rhymes with "hat" (Mencher and Stick, 1974, p. 978).

Hardy (1959) has suggested that hearing, language and speech are not unrelated operations and that they are bound together in a kind of feedback loop of relations between an individual and his environment.

If there is any interference or breakdown in this selfmonitoring loop, it may affect the other areas. Lewis (1960) states that impaired auditory reception makes it difficult to detect variations in sounds that are alike, such as a and e, ch and sh, etc., and to hear final consonants so that "bold" is confused with "bolt". Myklebust (1954) suggests that the types of language which must develop before useful communication are: (1) inner language, which is used to think and organize our thoughts; (2) receptive language, which is used to receive and understand communication from others by speech or reading; and (3) expressive language, which is man's method of expressing his thoughts to others in speech or in writing. Clark (1962) emphasizes that ineffective expression of language follows poor reception and that children in this group are often slow in beginning to talk, have much difficulty in discriminating sounds and words and typically show many sound substitutions. Disturbances in auditory perception include difficulty in auditory discrimination, not only in consonant discrimination but also in the discrimination of environmental sounds (Rampp, 1972). Children with disturbances in auditory perception have difficulty in going from parts-to-wholes, i.e., auditory synthesis. These children find it difficult to synthesize a consonant-vowel-consonant combination when presented with a one-syllable word. Therefore, synthesizing a complete sentence or sentences is for them a monumental task if not an impossibility (Rampp, 1972). Regardless of the types of tests given, these children manifest deficiencies. Most all children, aged seven and eight, with auditory perceptual disturbances have deficiencies in serial memory, e.g., the months of the year, the alphabet, their home address, their telephone number, and their birthdate (Rampp, 1972). These deficits have far-reaching consequences and may involve the entire communication cycle (Mencher and Stick, 1974). DeHirsch (et al., 1966) and Wepman (1968) claim that inadequate auditory perceptual skills ultimately lead to scholastic difficulties and to behavior problems associated with these difficulties.

At present, more tests and teaching materials have been designed for evaluating or improving visual perception than for auditory perception (Lerner, 1971). Silver (1963) list the following four types of tests used in measuring different aspects of auditory perception: (1) auditory word discrimination, (2) auditory blending, (3) sound matching, and (4) word meaning. The present study employs the first type of test that Silver mentions, i.e., auditory word discrimination which was defined earlier as the ability to recognize or distinguish between individual sounds in speech (Wepman, 1960) and the ability to identify words that are the same and words that are different (Lerner, 1971). Phoneticians may often disagree as to the relative importance of the physiologic and acoustic properties of the phonemes in the perception of sounds (e.g., Ladefoged, 1962 and Peterson, 1966) but there is little disagreement as to the basic necessity of being able to differentiate between the phonemes (Halle, 1967).

The alphabet of sounds that make up the words of any language are composed of the phonemes. These sounds are learned by children as they become discriminated from each other (Wepman, 1968).

In the child with an auditory discrimination deficit, sound and letter combinations are either meaningless or confused (Heilman, 1968 and Zigmond, 1968). Deficits in distinguishing between these sounds create problems in developing and using the words of a given language.

Evans (1969) states that auditory discrimination abilities may be particularly important in the development of a working vocabulary.

As previously mentioned, the stimulus word pairs in the Wepman Test of Auditory Discrimination (Wepman, 1958) was used in the present study. Snyder and Pope (1972) claim that the Wepman shows considerable potential for expanded use as a screening device for auditory discrimination. Several studies (Wepman, 1960; Clark and Richards, 1966; Dentsch, 1964; Oakland, 1969; Golden and Steiner, 1969) have shown that the Wepman supports earlier studies (Crossley, 1948; Nila, 1953; Harrington and Durrell, 1955) in that children with poor phonemic auditory discrimination ability tend to be poorer readers than children without this deficit. Clark and Richards (1966) investigated the relationship between auditory discrimination and social class membership and found that economically disadvantaged children made significantly more errors on the Wepman than did the nondisadvantaged children. Berlin and Dill (1967) studied the effects of feedback and positive reinforcement on the Wepman for lower-class Negro and white children. They found that positive reinforcement and feedback improved the discrimination scores of the Negro subjects only. There was no change observed in the control group. In another study in which the Wepman was used, Okada (1969) found that the simultaneous training of the visual and auditory modalities was effective in raising the language performance as well as the perceptual performance of institutionalized educable mental retardates regardless of their individual strengths and weaknesses. Dahle and Daly (1972), in a replication of the Berlin and Dill study, found that verbal feedback on the Wepman did not significantly alter the test scores of retarded children. They also concluded that the Wepman was a reliable measure of auditory discrimination for educable mentally retarded children. In another study Dahle and Daly (1974) investigated the performance of educable mentally retarded children on the Wepman when they received tokens and tangible rewards for correct responses. They found that tangible rewards also did not affect overall performance.

It is essential when using a test of auditory discrimination that the child be able to distinguish between the concepts of "same" and "different" (Irwin and Hammill, 1965; Blank, 1968; Baldes, et al., 1969). Beving and Eblen (1973) tested thirty children between the ages of four and eight years old with two speech-sound discrimination task. In one they asked the subjects to identify pairs of nonsense syllables as "same" or "different" and in the other they were asked to repeat the syllable pair. They found that the younger children scored better on the imitation task than they did on the "same - different" task. The older children did not differ in their ability to perform either task. They concluded that preschool-age subjects were probably unable to make the cognitive judgment "same" or "different", and that since these concepts are taught in the lower primary grades, that this may have accounted for the differences in performance between the preschool and school-age child. They suggest that clinicians using "same - different" tasks with preschool-age subjects must ascertain if these subjects understand these concepts before testing.

Aten and Davis (1968) examined disturbances in the perception of auditory sequence in children with minimal cerebral dysfunction. They administered three nonverbal and seven verbal recorded tests to a group of twenty-one children with minimal cerebral dysfunction and learning difficulties and compared their scores to twenty-one normal children on the same tests. They found that the children with minimal or mild cerebral dysfunction performed more poorly as evidenced in shorter perceptual spans, reduced number of stimuli retained and less accurate reproduction of sequential information than did the normal children. The children in the minimal cerebral dysfunction group were shown to be significantly impaired in their ability to perceive nonverbal auditory stimuli which varied in rhythm or duration. They were also deficient in their ability to reproduce nonverbal stimuli in proper sequence. These results support theories which state that the perception, storage, and reproduction of sequential stimuli are deficient in children with minimal cerebral dysfunction.

Doehring and Rabinovitch (1969) suggest that since auditory stimuli are usually presented over time, that auditory memory may be an intrinsic factor in most auditory perceptual abilities. In a study that involved a speech reception threshold test, a speech discrimination task presented at a comfortable listening level, and a speech discrimination task in white noise, they found that there was a tendency for children with learning problems to be deficient in all abilities involving auditory discrimination and perception.

Hypotheses

The following experimental hypotheses were made for the present study:

Hypotheses Related to Errors:

(1) Children diagnosed as having MBD will make significantly more errors than the controls on an auditory discrimination task, i.e., on

similar, identical and dissimilar word pairs.

(2) Both controls and children diagnosed as having MBD will make more errors on similar word pairs, less errors on identical word pairs and the least amount of errors on dissimilar word pairs.

Hypotheses Related to Duration:

(3) Children diagnosed as having MBD will make significantly more errors than the controls on a short duration between word pairs and on a long duration between word pairs.

(4) Children diagnosed as having MBD will make significantly more errors on a long duration between word pairs than they will on a short duration between word pairs.

(5) Control subjects will show no significant difference between a short and a long duration between word pairs.

Hypotheses Related to Replicates:

(6) It is assumed that the replications factor will show a fatigue effect for the experimental group, therefore children diagnosed as having MBD will make significantly more errors than the controls as the auditory discrimination task increases in time.

(7) It is assumed that the replications factor will show a fatigue effect for the experimental group, therefore children diagnosed as having MBD will make more errors as the auditory discrimination task increases in time.

(8) It is assumed that the replications factor will not show a fatigue effect for the control group, therefore control subjects will show no significant increase in the number of errors as the auditory discrimination task increases in time.

Hypotheses Related to Nonresponses:

(9) Children diagnosed as having MBD will show significantly more nonresponses than the controls.

(10) Children diagnosed as having MBD will show more nonresponses as the auditory discrimination task increases in time.

(11) Control subjects will show no significant increase in the number of nonresponses as the auditory discrimination task increases in time.

CHAPTER III

METHODS AND PROCEDURES

Subjects

There were 52 children chosen for this study. Twenty-six children were previously diagnosed by the Child Study Center at the University of Arkansas Medical Center as having minimal brain dysfunction (see Appendix I). These children were matched with twenty-six children from the public school system of Little Rock, Arkansas according to age (8 - 12); sex (boys); race (Caucasian); I.Q. (85 - 110); socioeconomic status; and absence of any known debilitating emotional disturbance. Children who had not had a hearing test within the past year were tested for hearing by an audiologist. Several children who were diagnosed as having MBD were taking Ritalin. Since the onset of Ritalin takes within an hour and washes out of the body in twenty-four hours, these children were taken off their daily dosage of Ritalin at least two days before testing to rule out any effects of medication on their performances. The above information was obtained from physicians, teachers, medical and school records.

Experimental Set Up

Subjects were tested individually in an eight foot by eleven foot room set up for testing in the Child Study Center at the University of

Arkansas Medical Center. Prior to testing the research project was cleared through the State Department of Education and with the school principals. Each child's parents were contacted both by phone and by letter. The research project was explained to them and other questions they had were answered. Both verbal and written permission was received from each parent to use their child in the study. All MBD children except one were tested while they were in the therapeutic day school at the Child Study Center. These children did not have to leave the building in order to be tested. The research criteria exhausted the population of MBD children at the Child Study Center and one child previously diagnosed by the Child Study Center as having MBD was taken from a private school in the Little Rock area. This child along with all the control subjects were picked up at school by the examiner, brought to the Medical Center, tested, and returned to school. They were paid two dollars for their participation in the experiment. Each was told how long the experiment would last (approximately 45 minutes).

The word pairs (see Appendices J - O) were made beforehand by a speech pathologist and recorded on auditory tape. The examiner, using two separate tape recorders, a counter and an audiometer, placed the word pairs and an audible "beep" on auditory tape at designated distances. An Akai professional model GX280D-SS tape recorder with glass and crystal ferriate heads was used to get the best sound production possible.

Each child sat before a table adjusted to their height, in a comfortable straight back chair that allowed their feet to touch the floor. They listened to the word pairs through Koss Stereo head phones. Each subject was given (by auditory tape) instructions for the test and a practice trial consisting of five word pairs from the short delay period (s) and five word pairs from the long delay period (S) (see Appendix I). He responded by pressing one of two telegraph keys marked "same" or "different" on the table in front of him. Before the practice session each child was asked the meaning of the words same and different. If he could not understand these concepts or the practice session, he was eliminated from the experiment. Two of the children diagnosed as having MBD were omitted because they did not understand the concepts of same and different.

There was a shield between the subject and the examiner, so the subject would not be distracted by what the examiner was doing. There were two lights which corresponded with the telegraph keys on the examiner's side of the table. These lights were marked S and D for same and different. The examiner listened to the word pairs being presented to the child through Koss Stereo head phones. This set up allowed the examiner to listen to the word pairs being presented to the subject, see the response made by the subject and record this response on an answer sheet. Six different answer sheets were used (see Appendices P - U), one for each of the six sets of word pairs.

Definition of the Test

A. Content of the word pairs (fifty):

There were thirty similar word pairs and ten identical word pairs taken from the Wepman Auditory Discrimination Test. Ten dissimilar word pairs were composed by the author, making a total of fifty word pairs. These word pairs were arranged into six sets (see Appendix P) making a total of 300 word pairs for each child to discriminate.

B. Delay within the word pairs (two):

There were two delays within the word pairs:

(1) A short delay, designated by s, of .5 of a second.

(2) A long delay, designated by S, of five seconds.

C. Delay between the word pairs:

There was a three second delay between the word pairs. This gave the subject time to respond "same" or "different" and for the investigator to score his response.

There was an audible "beep" on the tape .5 of a second before the next pair was presented. There were two reasons for this: (1) To obtain optimal attention from the child to measure the delay and fatigue factors and (2) Since there was a five second delay within the word pairs on the long duration and a three second delay before the presentation of the next pairs, the last word in the long duration and the first word in the next pair may have been confused.

D. Order and sequence of the six sets of fifty word pairs:

(1) There were six sets of fifty word pairs as follows:

(a) Three sets of fifty word pairs had a short duration within the pairs of .5 of a second and were designated by a small s.

(b) Three sets of fifty word pairs had a long duration within the pairs of five seconds and were designated by a large S.

These six sets of fifty pairs of words were arranged into three replicates in order to estimate the effects of fatigue. Each replicate consisted of two sequences of fifty words (s , S), one of short duration (s) and one of long duration (S). The first replicate was designated as $s_1\ S_2$, the second as $s_3\ S_4$, and the third as $s_5\ S_6$.

(2) Orders (two):

There were two orders in which the sequences were arranged:

(a) $s_1\ S_2\ ;\ s_3\ S_4\ ;\ s_5\ S_6$

(b) $S_2\ s_1\ ;\ S_4\ s_3\ ;\ S_6\ s_5$

(3) Order within pairs:

Order within the pairs (i.e., which word came first at each repetition) was randomized for all six sequences to minimize the learning effect.

E. Assignment to treatment groups:

Individuals within each of the two populations were randomly assigned to one of the two orders:

$s_1\ S_2\ ;\ s_3\ S_4\ ;\ s_5\ S_6$

$S_2\ s_1\ ;\ S_4\ s_3\ ;\ S_6\ s_5$

(1) Each control was numbered as follows: 1, 2, 3, ...26.

(2) A sequence of 26 random numbers was drawn from a table of three digits of random numbers and matched to individuals in the order drawn.

(3) The 26 individuals were ranked by their random number. The first 13 received the order $s_1\ S_2\ ;\ s_3\ S_4\ ;\ s_5\ S_6$ The second 13 received the order $S_2\ s_1\ ;\ S_4\ s_3\ ;\ S_6\ s_5$

(4) Each MBD was number as follows: 27, 28, 29, ...52. The same sequence was followed as for the controls above

in 1, 2, and 3.

Measurements

There were six measurements on each subject, one for each set of fifty word pairs. The percent of incorrect responses was used in the analysis. For each subject there were six sets of fifty word pairs divided as follows:

1. There were six sets of thirty similar word pairs.
2. There were six sets of ten identical word pairs.
3. There were six sets of ten dissimilar word pairs.

Data Layout

The data layout was as follows:

TABLE I

REPLICATE

Group	1	2	3
G_1: Controls	s_1 S_2 S_2 s_1	s_3 S_4 S_4 s_3	s_5 S_6 S_6 s_5
G_2: MBD	s_1 S_2 S_2 s_1	s_3 S_4 S_4 s_3	s_5 S_6 S_6 s_5

There are a total of 24 cells in this layout with four factors in each cell. The four factors are:

1. Group
2. Order
3. Duration
4. Replicate

In order to test the experimental hypotheses, a mixed model design with the first two factors being independent groups and the latter being repeated measures was decided upon. There are three factors at two levels each (group, order, and duration) and one factor at three levels (replicate) giving a basis of 24 means that were expressed as a mean percentage. From this basis an overall mean, a group mean, an order mean, and a replicate mean was obtained to make the overall comparisons.

The order factor may be considered to be a nuisance factor, but it was included to rule out the possible effects of ordering when using both short and long durations with each subject. There was not expected to be a significant difference in the orders both groups received.

There were two analyses on the three different word pairs. There was an analysis on the incorrect responses (I) and another analysis was made on the nonresponses (N) to the word pairs.

An analysis of variance mixed model, that corresponded with the design was used to analyze incorrect responses (I) for similar, dissimilar, and identical word pairs. This made a total of three analyses on incorrect responses. Since there was an uneven number of various word pairs for similar (30), dissimilar (10), and identical (10), transformed values in the form of percents, were used in the analysis of variance. The main reason for using the transformed data was to make the analysis comparable for the purpose of comparing means and for the purpose of illustration.

The Fisher's Exact Test was used for the analysis of nonresponses (N) to the word pairs. Six separate analyses were made on each of the similar, dissimilar, and identical word pairs, making a total of eighteen analyses on no responses. In each of the word groups, three separate analyses were for the short duration (s) between the word pairs (one for each replicate) and three separate analyses were made for the long duration (S) between the word pairs.

The analysis of variance was not used for nonresponses because the controls responded to essentially every item. That is, it was felt that the assumptions of normality and homogeneity of variance would be stretched to its limits. Therefore, the Fisher's Exact Test was chosen for the analysis of nonresponses. The decision to use this statistic was based on two factors: (1) It gives the exact probabilities that a given set of events will occur and is therefore a much more powerful test than is, for example, the chi square approximation; (2) When a two by two contingency table is used, the expected values of chi square should be greater than ten. For the control group in the present study there was a zero in one of the quadrants in these tables. This made the expected value of chi square much less than ten and made the computation of the exact probabilities much less complicated.

The conventional .05 level of significance was chosen for this study.

CHAPTER IV

RESULTS

This chapter presents the results yielded by the analysis of the data. The first section concentrates on the analysis for incorrect responses (I) on similar, dissimilar, and identical word pairs. The second section concentrates on the analysis for nonresponses (N) to the word pairs on these three different word groups.

Incorrect Responses (I)

Those children diagnosed as having MBD made significantly more errors ($P < .01$) than the controls on both the short and long duration between the word pairs, on similar, dissimilar and identical word pairs, and on all three replicates. Figure 1 represents the total number of errors made by both groups on similar, dissimilar, and identical word pairs.

Both groups made the least amount of errors on the dissimilar word pairs, more errors were made on the identical word pairs, and the most errors were made on similar word pairs. Children diagnosed as having MBD made significantly more errors ($P < .01$) on the similar word pairs than they did on the identical and dissimilar pairs. They also made significantly more errors ($P < .05$) on the identical word pairs than they did on the dissimilar pairs. Controls also made significantly more errors ($P < .01$) on the similar word pairs than they did on the identical and dissimilar pairs, however, they did not have a significant difference in the number of errors between the identical and dissimilar pairs. The analysis for incorrect responses (I) on dissimilar word pairs (see Appendix B, ANOV Table) showed that groups were highly significant ($P < .01$), replicates were significant ($P < .05$) and replicate by group interaction was highly significant ($P < .01$). Table II is a summary of incorrect responses on dissimilar word pairs.

TABLE II

SUMMARY OF INCORRECT RESPONSES (I)
ON DISSIMILAR WORD PAIRS

	Mean	Percentage
Both Groups	.10089	1.0
Controls	.00206	0.0
MBD's	.19972	3.9

This table shows that minimal brain dysfunction plus controls under all conditions thrown together had a 1.0 percent error rate. The controls error rate was zero, i.e., they made essentially no errors on the dissimilar word pairs compared to a 3.9 percent error rate for minimal brain dysfunctions.

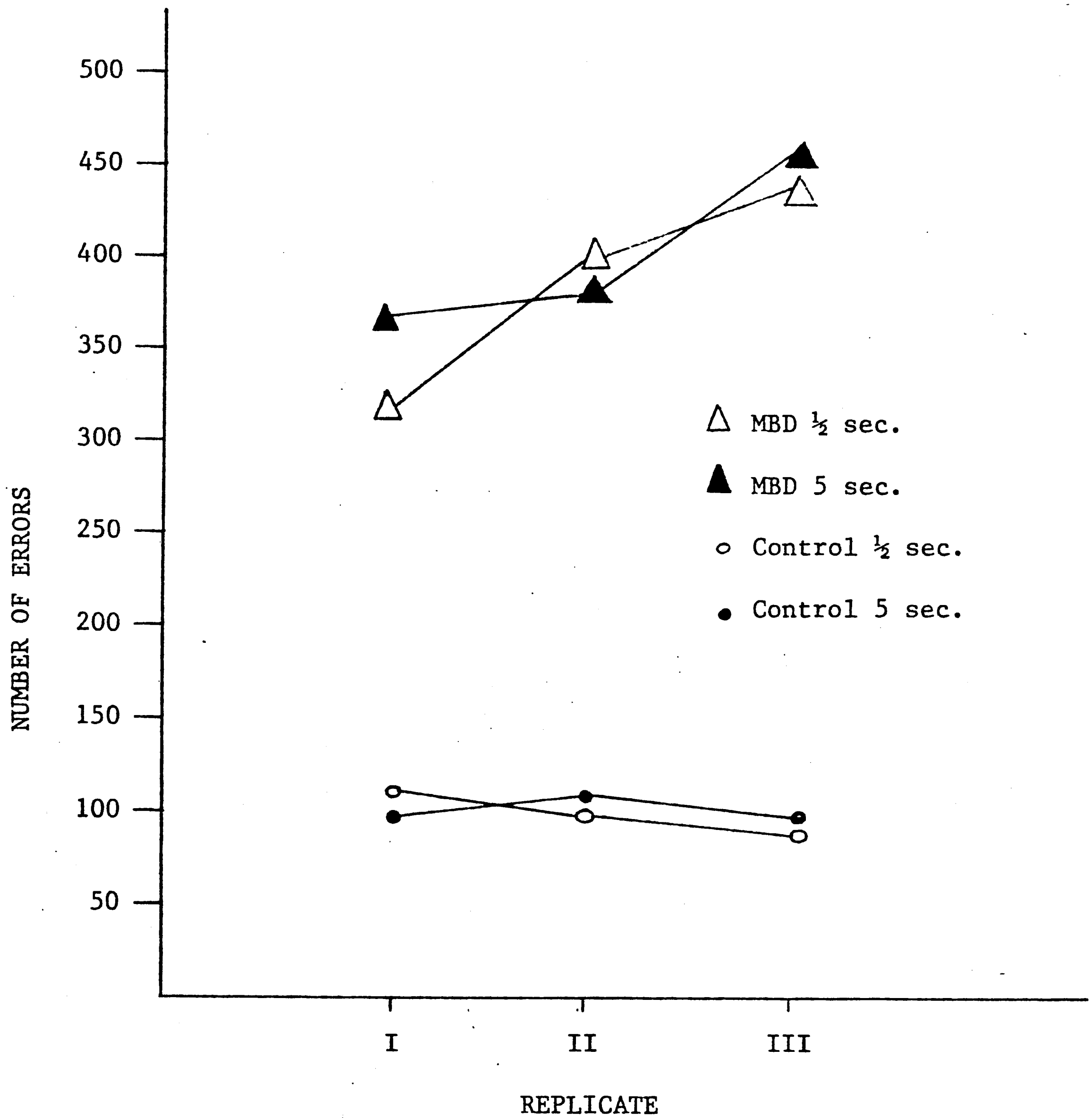

Figure 1. Total Errors for MBD's and Controls on Similar, Dissimilar and Identical Word Pairs

Table III shows that controls were essentially zero across all replicates, i.e., there was no significant differences across replicates for the control group. There is, however, an upward trend in the number of errors across replicates for minimal brain dysfunctions. The standard error of the differences between the two groups in replicates was .0491, which means that minimal brain dysfunctions made significantly ($P < .01$) more errors than controls in all replicates. By the least significant difference method, replicate three was greater ($P < .05$) than replicates one or two for minimal brain dysfunctions.

Figure 2 is a summary of the total number of errors made by both groups on dissimilar word pairs.

The analysis for incorrect responses (I) on identical word pairs (see Appendix C, ANOV Table) showed that groups were still highly significant ($P < .01$), replicates were still significant ($P < .05$) and delay by group interaction was significant ($P < .05$). Table IV is a summary of incorrect responses on identical word pairs. Both groups combined went from an overall error rate of 1.0 percent on dissimilar word pairs to a 3.5 percent on identical word paris. Table IV shows that the minimal brain dysfunctions were primarily responsible for this increase in the overall percentage of errors. The error rate for controls was once again essentially zero (0.6) as compared to a 8.6 percent error rate for minimal brain dysfunctions.

The replicate by group interaction dropped out on identical word pairs, which indicates that the replicate effect was the same for both groups. However, there was a group effect within delays.

TABLE III

SUMMARY OF REPLICATE BY GROUP INTERACTION ON INCORRECT RESPONSES (I) FOR DISSIMILAR WORD PAIRS

Group	Replicate I	Replicate II	Replicate III
Control	.00620	0	0
MBD	.15795**	.18340**	.25778**

** $P < .01$

TABLE IV

SUMMARY OF INCORRECT RESPONSES (I) ON IDENTICAL WORD PAIRS

	Mean	Percentage
Both Groups	.18889	3.5
Controls	.07904	0.6
MBD's	.29874	8.6

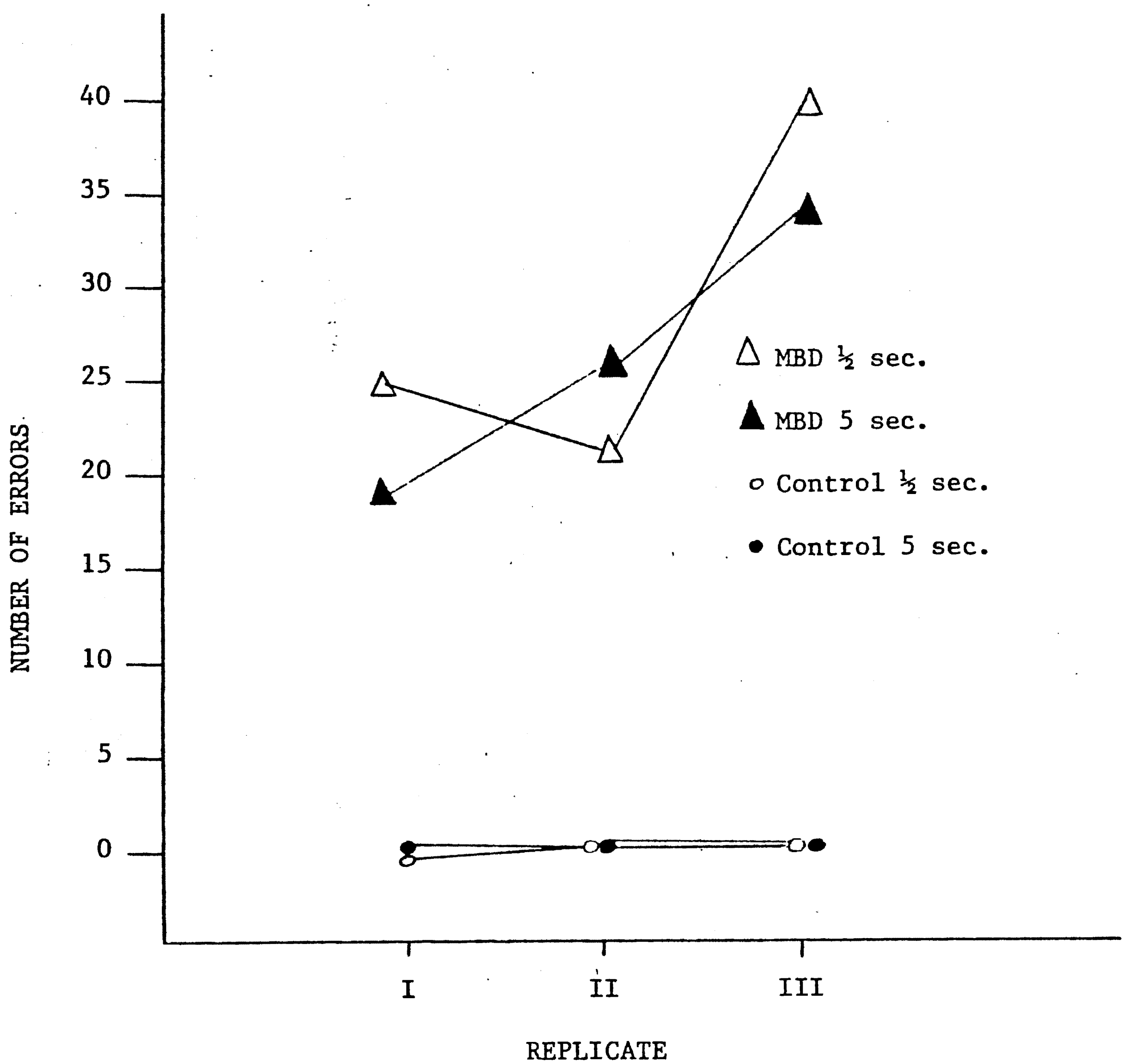

Figure 2. Total Errors for MBD's and Controls on Dissimilar Word Pairs

Table V shows that within delay one, which is the short .5 second delay, there was a smaller effect between minimal brain dysfunctions and controls than for delay two. Both delays were significant beyond the .05 level. The difference between groups one and two on delay one was .18007 which yielded a t of 2.01 and a probability of less than .05. The difference between groups one and two on delay two was .25930 which yielded a t of 2.9 and a probability of less than .01. Therefore, there was a larger group effect for the longer delay (5 seconds) than for the shorter delay (.5 second). Minimal brain dysfunctions made more errors on the long delay than they did on the short delay. The opposite was true for the controls. They made more errors on the short delay than they did on the long delay. This difference, however, was not significant. On a percentage basis, the minimal brain dysfunctions went from seven percent incorrect responses to ten percent incorrect responses, from a short to a long delay between the word pairs. The controls made less than one percent incorrect responses in both cases.

By the least significant difference application, replicate one (.18559; 3.4 percent incorrect) compared to replicate two (.15953; 2.5 percent incorrect) was not significant and replicate one compared to replicate three (.22158; 4.8 percent incorrect) was not significant. However, replicate two when compared to replicate three was significant beyond the .05 level.

Figure 3 is a summary of the total number of errors made by both groups on identical word pairs.

The analysis for incorrect responses (I) on similar word pairs (see Appendix D, ANOV Table) showed that groups were still highly significant ($P < .01$), replicates were still highly significant

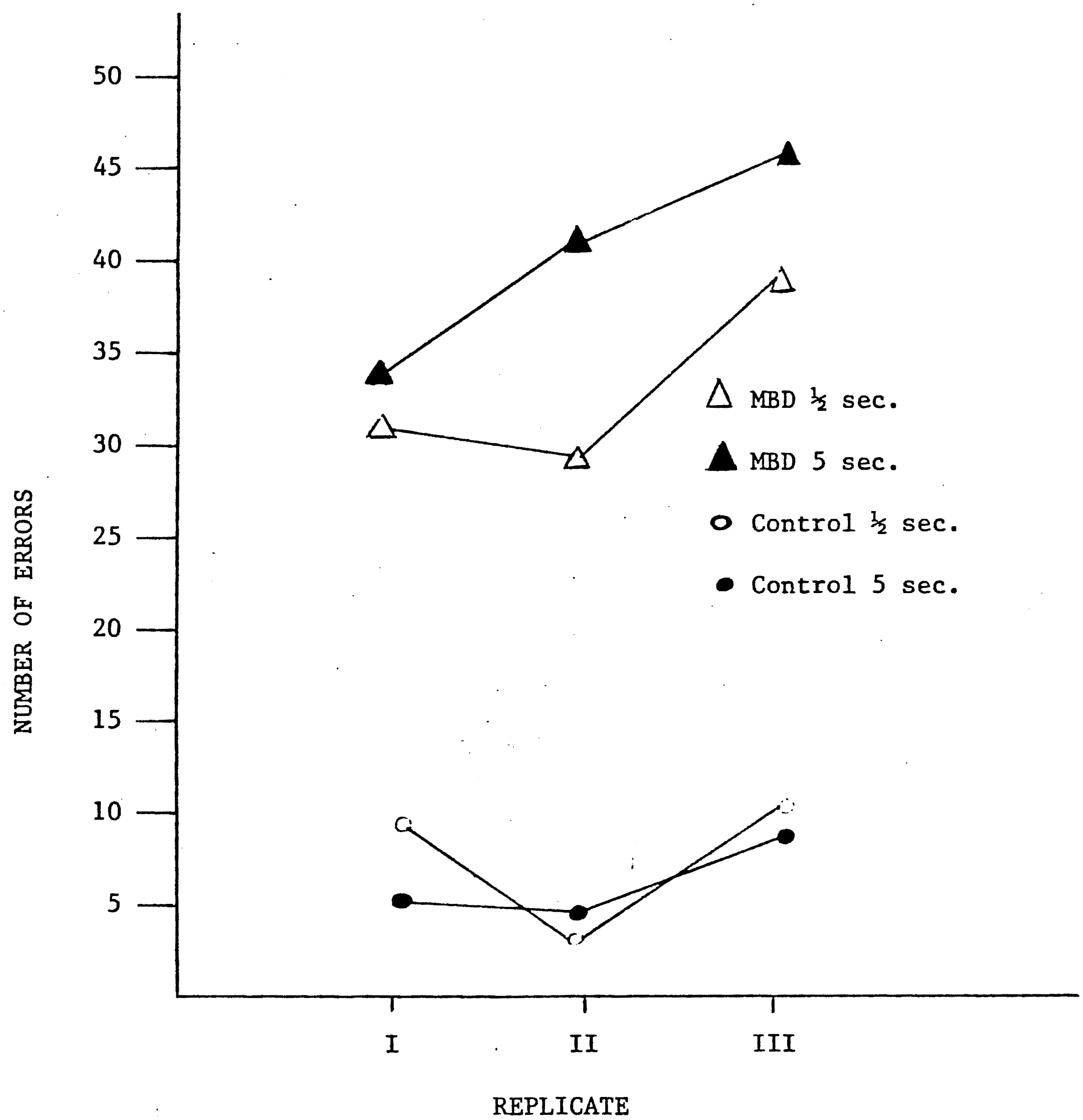

Figure 3. Total Errors for MBD's and Controls on Identical Word Pairs

TABLE V

SUMMARY OF DURATION BY GROUP INTERACTION ON
INCORRECT RESPONSES (I) FOR
IDENTICAL WORD PAIRS

Group	Delay I	Delay II	Mean
Controls	0.9	0.4	0.6
MBD's	7.4	10.0	8.7
Mean	3.4	3.7	3.5

($P < .01$), and there was a delay by replicate by order interaction that was highly significant ($P < .01$). Table VI is a summary of incorrect responses on similar word pairs. As found previously, there was a highly significant difference between controls and minimal brain dysfunctions.

Both groups went from an overall error rate of 1.0 percent on dissimilar word pairs, to a 3.5 percent on identical word pairs, and to a 22.9 percent on similar word pairs. The error rate for controls went from essentially zero on dissimilar and identical word pairs, to 13.5 percent on similar word pairs. In comparison, the error rate for minimal brain dysfunctions went from 3.9 percent on dissimilar word pairs, to 8.6 percent on identical word pairs and to 34 percent on similar word pairs.

There was no significant order by replicate or delay by replicate interaction on similar word pairs. The third order interaction was

TABLE VI

SUMMARY OF INCORRECT RESPONSES (I) ON
SIMILAR WORD PAIRS

	Mean	Percentage
Both Groups	.49953	22.9
Controls	.37638	13.5
MBD's	.62269	34.0

TABLE VII

SUMMARY OF PERCENT OF INCORRECT RESPONSES IN DXRXO
INTERACTION ON INCORRECT RESPONSES (I)
FOR SIMILAR WORD PAIRS

Order	Delay	Rep. I	Rep. II	Rep. III
1sS	1s	26.0	22.4	17.5
	2S	21.2	21.3	20.2
2Ss	1s	22.8	24.7	23.5
	2S	27.2	24.8	24.7

caused by the occurrence of more errors on the first sequence of word pairs presented to the groups regardless of the order they received. This was perhaps due to a lack of practice. Table VII shows that order one, delay one, replicate one (26.0) and order two, delay two, replicate one (27.2) were the cause of the third order interaction.

Figure 4 is a summary of the total number of errors made by both groups on similar word pairs.

Figure 5 combines both the half second delay and the five second delay for both groups and represents a summary of the total number of errors across all word groups. This figure shows that the controls actually improved their performance across replicates, i.e., they made fewer errors as the test increased in time. In comparison, minimal brain dysfunctions made more errors as the test progressed, i.e., across replicates.

Nonresponses (N)

Figure 6 represents the total number of nonresponses (N) made by both groups on similar, dissimilar and identical word pairs.

The analysis for nonresponses (N) on dissimilar word pairs (see Appendix E, Table) showed that there was no significant difference between controls and MBD's on the first replicate they received. However in the second replicate there was a significant difference (P = .005) in the number of nonresponses for MBD's on the short duration between the word pairs and in replicate three there was a significant difference in nonresponses for MBD's for both the short (P = .001) and the long (P = .000) duration between the word pairs. Table VIII is a summary of nonresponses on dissimilar word pairs. This table shows

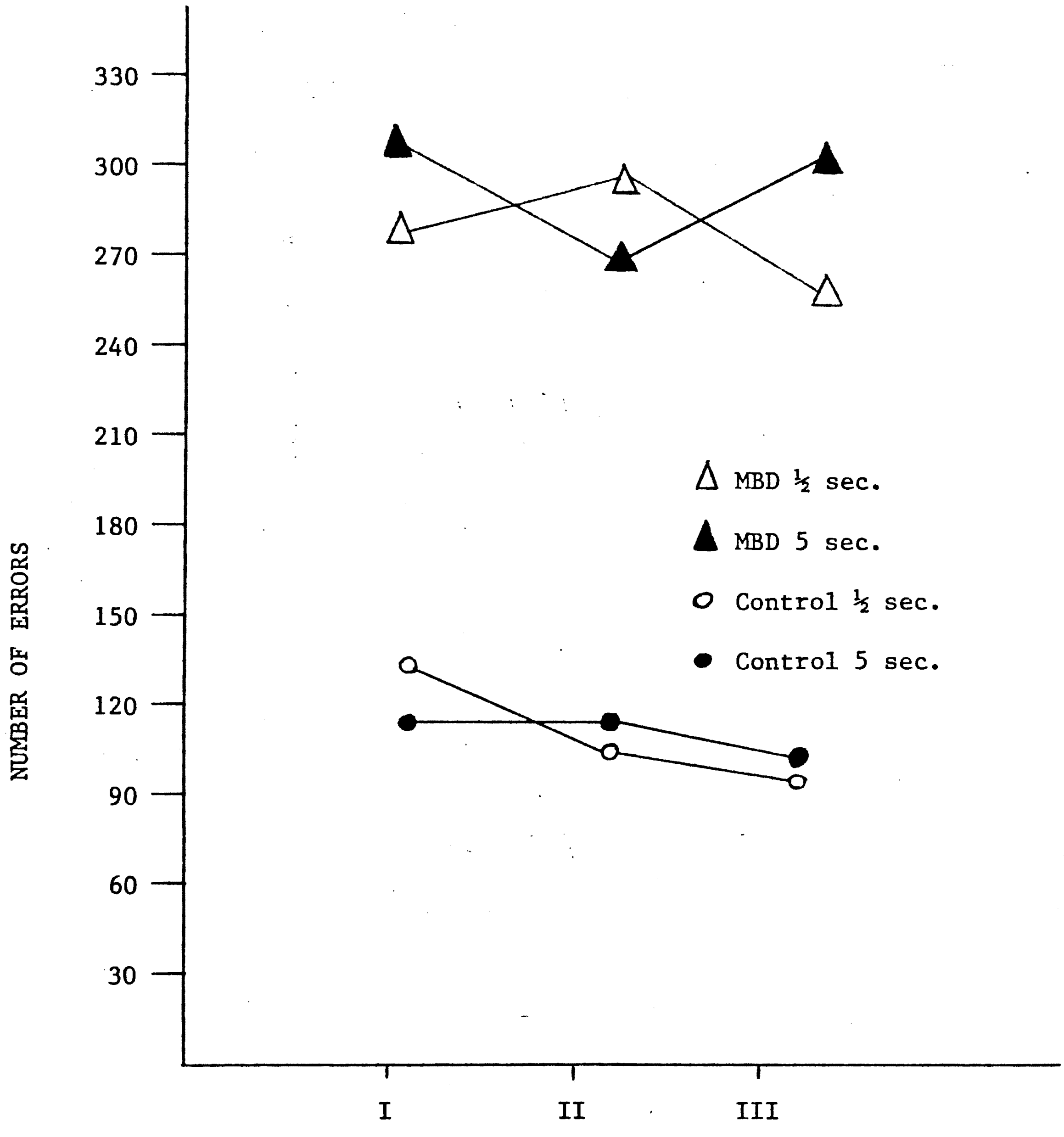

Figure 4. Total Errors for MBD's and Controls on Similar Word Pairs

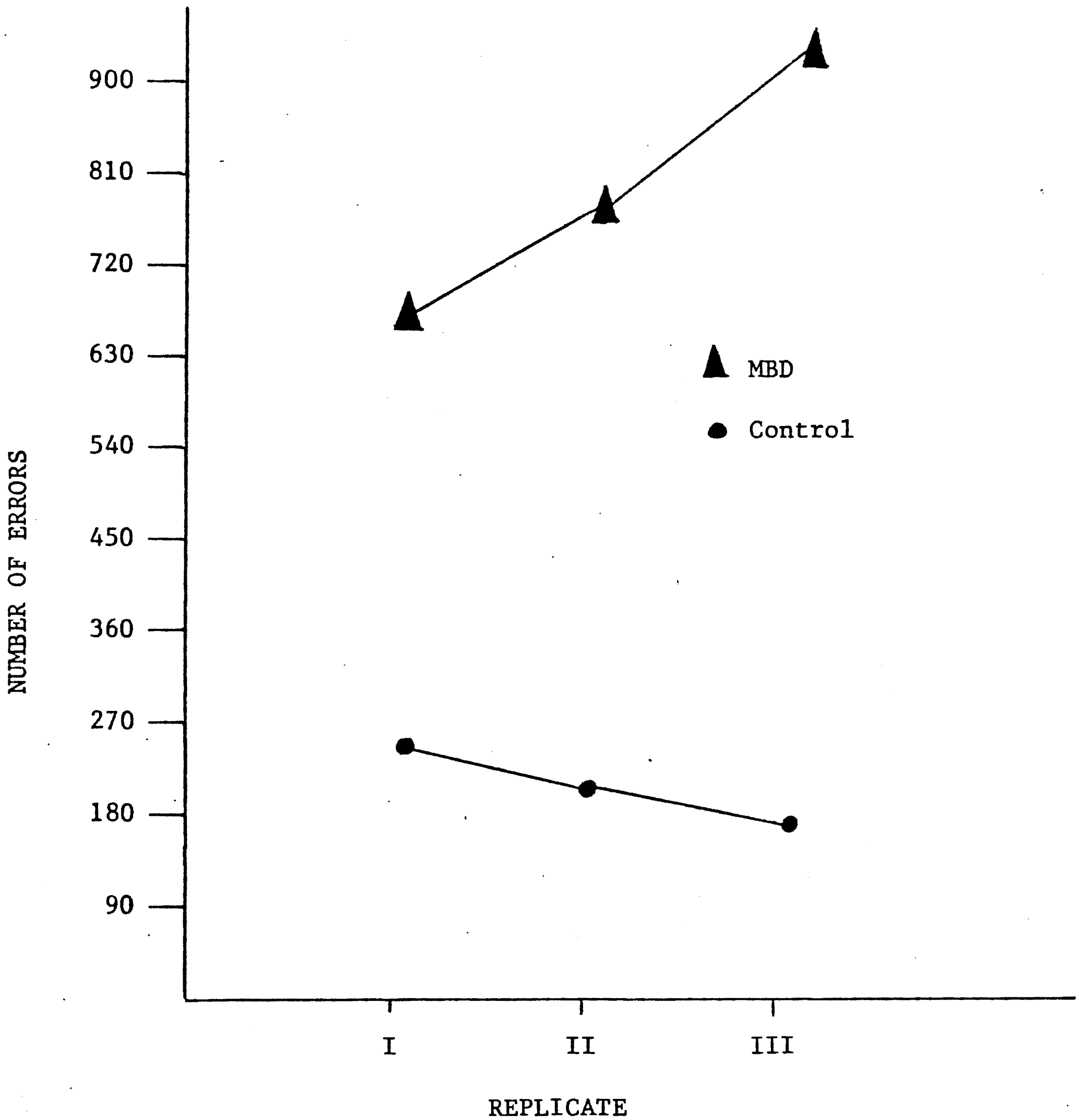

Figure 5. Total Errors for MBD's and Controls with Delays Combined Across All Word Groups

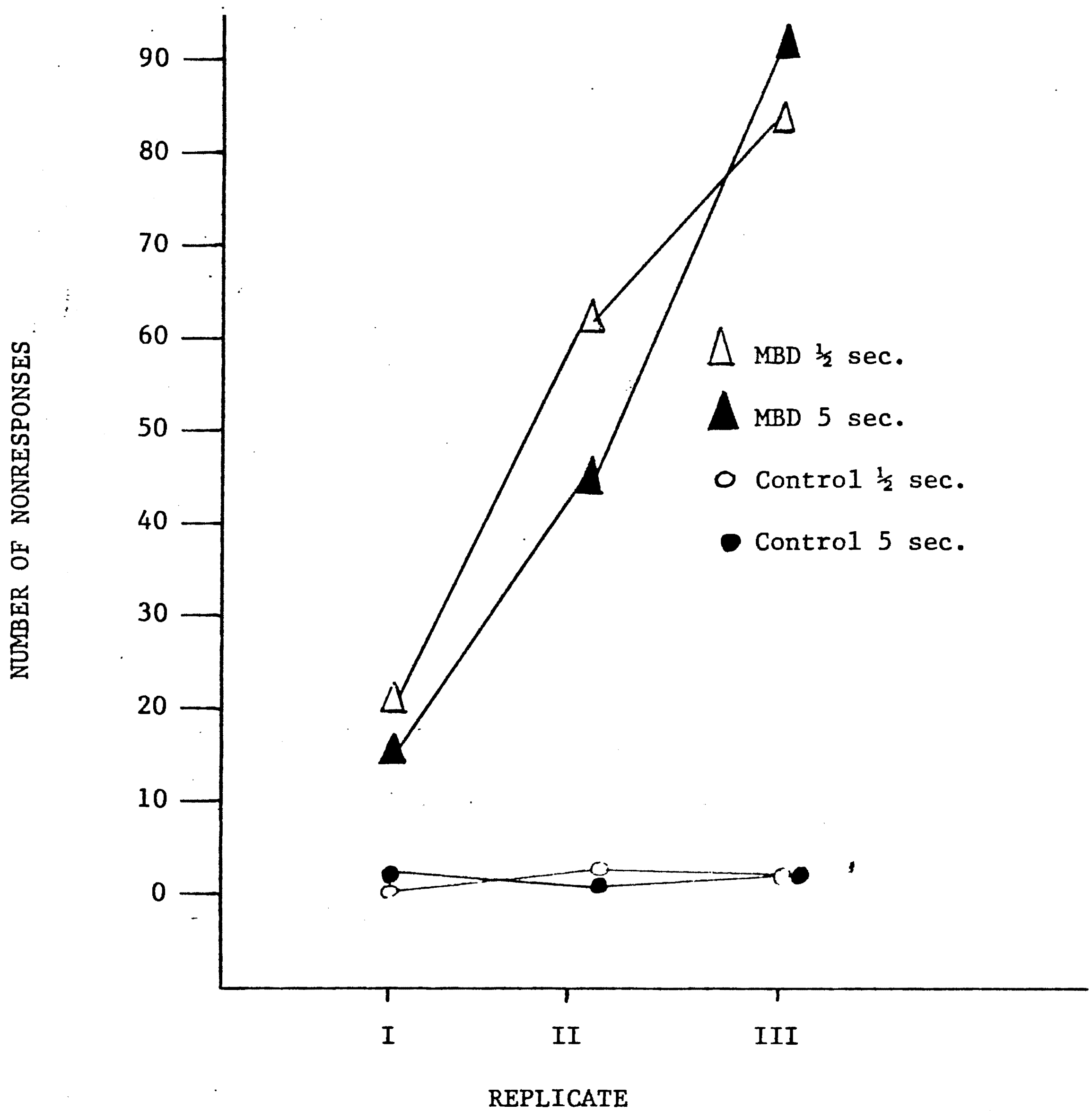

Figure 6. Total Nonresponses (N) for MBD's and Controls On Similar, Dissimilar and Identical Word Pairs

that minimal brain dysfunctions plus controls under all conditions had a .11 percent nonresponse rate. The controls nonresponse rate was zero on the dissimilar word pairs compared to a .46 percent nonresponse rate for minimal brain dysfunctions.

Figure 7 is a summary of nonresponses made by both groups on the dissimilar word pairs.

TABLE VIII

SUMMARY OF NONRESPONSES (N) ON DISSIMILAR WORD PAIRS

	Mean	Percentage
Both Groups	.03390	0.11
Controls	0	0
MBD's	.06779	0.46

The analysis for nonresponses (N) on identical word pairs (see Appendix E, Table) showed, as in the dissimilar word pairs, that there was no significant difference between controls and MBD's on the first replicate they received. In the second and third replicates, minimal brain dysfunctions had significantly more nonresponses than the controls on both the short and long duration between the word pairs.

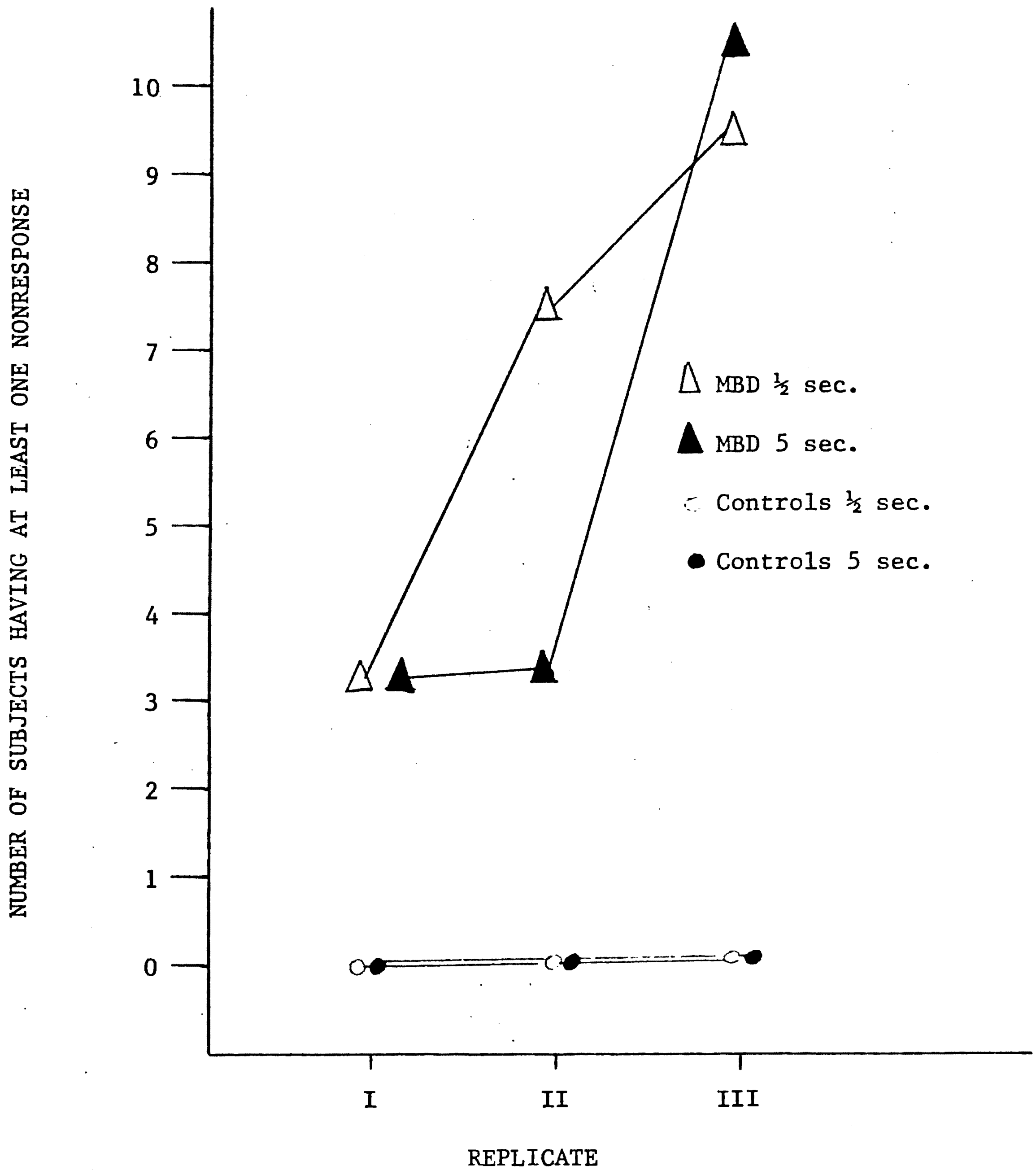

Figure 7. Summary of Nonresponses (N) for MBD's and Controls On Dissimilar Word Pairs

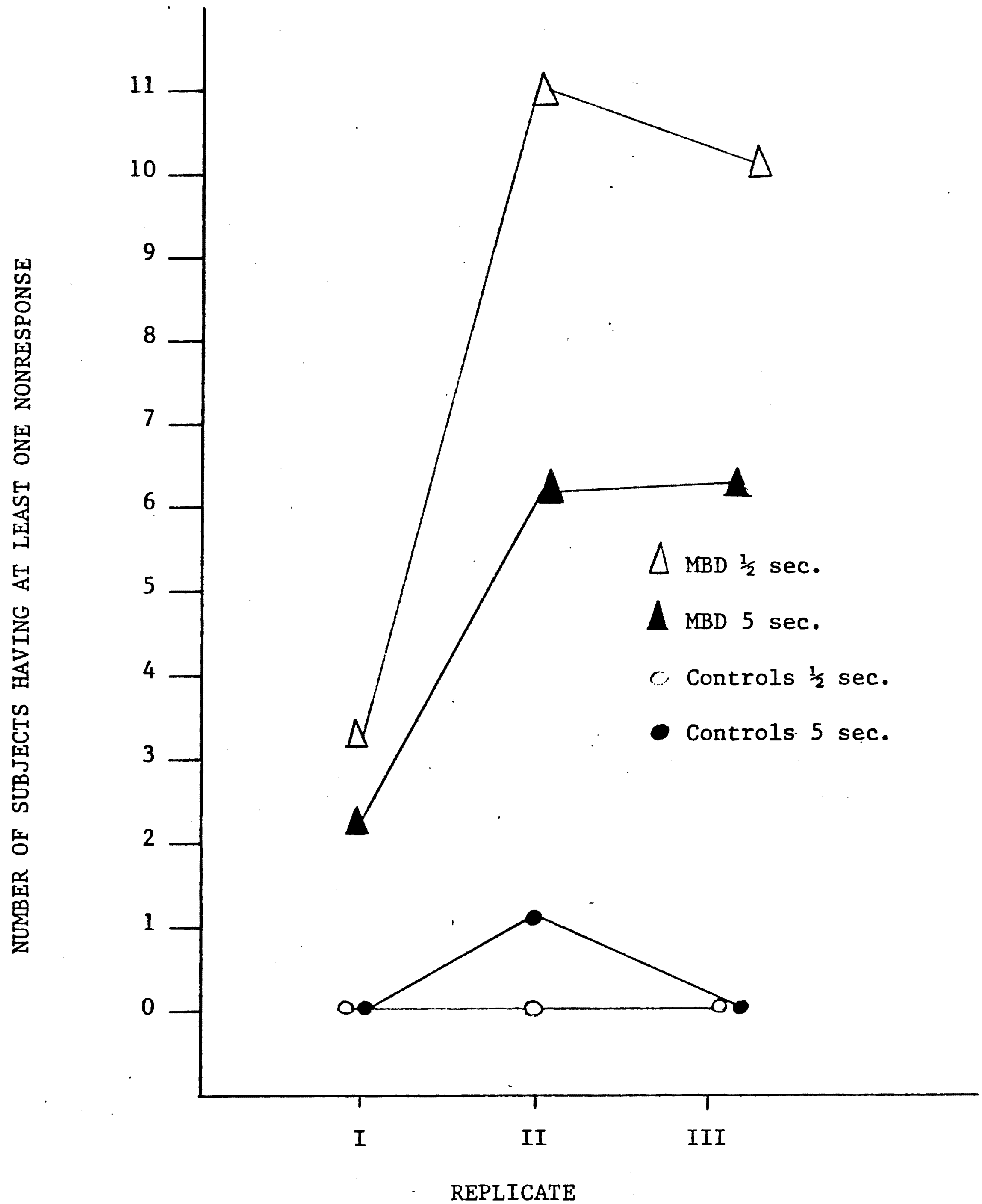

Figure 8. Summary of Nonresponses (N) for MBD's and Controls On Identical Word Pairs

Table IX is a summary of nonresponses on identical word pairs. This shows that minimal brain dysfunctions plus controls under all conditions had a .16 percent nonresponse rate. Again controls were essentially zero compared to a .62 percent nonresponse rate for minimal brain dysfunctions. Figure 8 is a summary of nonresponses made by both groups on the identical word pairs.

TABLE IX

SUMMARY OF NONRESPONSES (N) ON
IDENTICAL WORD PAIRS

	Mean	Percentage
Both Groups	.04054	.16
Controls	.00206	.0004
MBD's	.07903	.62

The analysis for nonresponses (N) on similar word pairs (see Appendix E, Table) showed, that once again, MBD's had significantly more nonresponses than the controls, in replicates two and three, on both the short and long duration between the word pairs. On similar word pairs, MBD's also had more nonresponses than the controls on the short duration between the word pairs in replicate one. On dissimilar and identical word pairs, there was no significant differences between MBD's and controls on replicate one. Table X is a summary of nonresponses on similar word pairs. This table shows that MBD's plus controls under all conditions had a .46 percent nonresponse rate. Controls were once again essentially zero (.003) compared to a 1.7 percent nonresponse rate for MBD's. The overall nonresponse rate went from a .11 percent on dissimilar word pairs, to a .16 percent on identical word pairs, to a .46 percent on similar word pairs. MBD's were essentially the cause of this increase in the nonresponse rate and this was due primarily to their increase in nonresponses on the second and third replicates.

TABLE X

SUMMARY OF NONRESPONSES (N) ON SIMILAR WORD PAIRS

	Mean	Percentage
Both Groups	.06760	.46
Controls	.00588	.003
MBD's	.12931	1.7

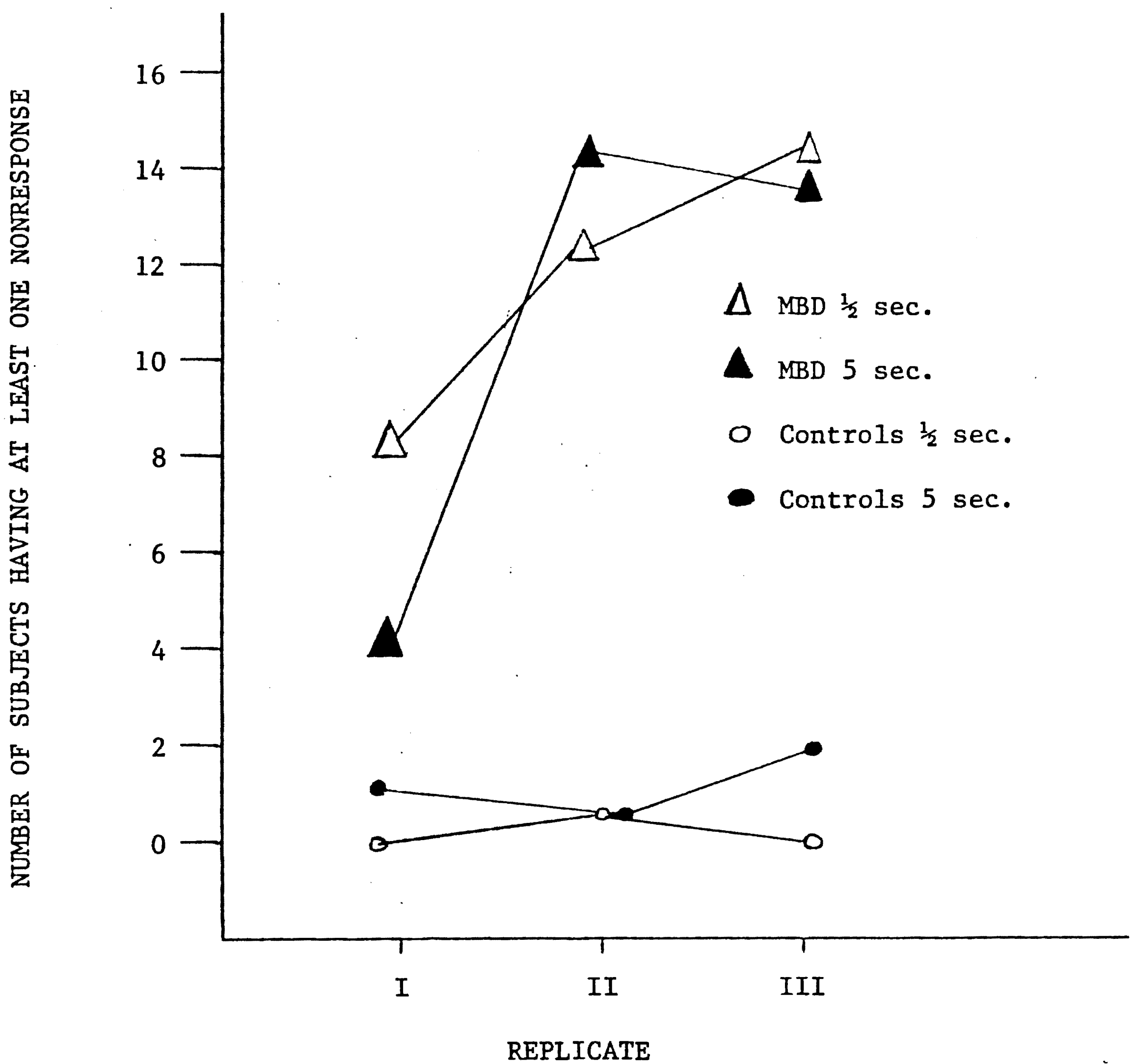

Figure 9. Summary of Nonresponses (N) for MBD's and Controls On Similar Word Pairs

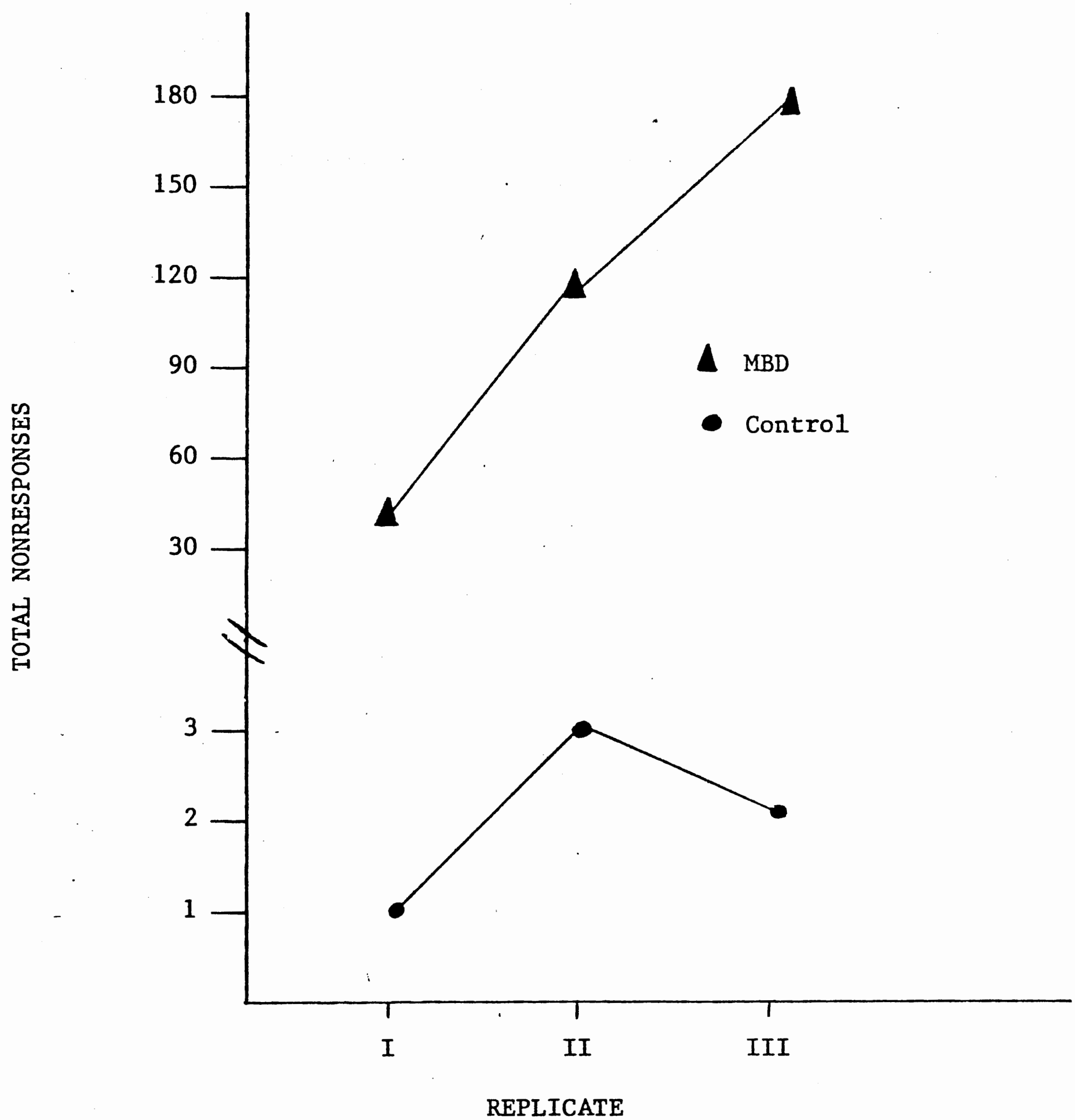

Figure 10. Total Nonresponse (N) for MBD's and Controls with Delays Combined Across All Word Groups

Figure 9 is a summary of nonresponses made by both groups on the similar word pairs. Figure 10 combines both the half second delay and the five second delay for both groups and represents a summary of the total number of nonresponses across all word groups. There were no significant differences in the number of nonresponses across replicates for the control group. The minimal brain dysfunctions, on the other hand, had significantly more nonresponses as the test increased in time.

CHAPTER V

DISCUSSION AND IMPLICATIONS FOR FURTHER RESEARCH

Statistical support was found for the following experimental hypotheses:

Hypothesis 1. Children diagnosed as having MBD made significantly more errors than the controls on the auditory discrimination task, i.e., on similar, identical, and dissimilar word pairs.

Hypothesis 2. Both controls and children diagnosed as having MBD made more errors on similar word pairs, less errors on identical word pairs and the least amount of errors on dissimilar word pairs.

Hypothesis 3. Children diagnosed as having MBD made significantly more errors than the controls on a short duration between word pairs and also on a long duration between word pairs.

Hypothesis 5. Control subjects had no significant difference between a short and a long duration between word pairs.

Hypothesis 6. Children diagnosed as having MBD made significantly more errors than the controls as the auditory discrimination task increased in time.

Hypothesis 7. Children diagnosed as having MBD made more errors as the auditory discrimination task increased in time.

Hypothesis 8. Control subjects showed no significant increase in the number of errors as the auditory discrimination task increased in time.

Hypothesis 9. Children diagnosed as having MBD had significantly more nonresponses than the controls.

Hypothesis 10. Children diagnosed as having MBD had more nonresponses as the auditory discrimination task increased in time.

Hypothesis 11. Control subjects showed no significant increase in the number of nonresponses as the auditory discrimination task increased in time.

Hypothesis four stated that children diagnosed as having MBD would make significantly more errors on a long duration between word pairs than they would on a short duration between word pairs. This was true only for the identical word pairs. On the similar and dissimilar word pairs there were no significant differences between a short and a long delay and there were also no significant "over all" differences (see Figure 1) between a short and a long delay for children diagnosed as having MBD. Therefore the statistically significant difference found on the long delay for the identical word pairs was not viewed as being of any practical significance.

Four major findings can be summarized from the results in Chapter IV:

First, children diagnosed as having minimal brain dysfunction made significantly more errors than the controls on both the short and long duration between the word pairs, on similar, dissimilar and identical word pairs, and on all three replicates.

Second, children diagnosed as having minimal brain dysfunction made more errors as the test progressed, i.e., across replicates. In comparision, the controls actually improved their performance across replicates.

Third, children diagnosed as having minimal brain dysfunction had significantly more nonresponses as the test increased in time. There were no significant differences in the number of nonresponses across replicates for the control group.

Fourth, children diagnosed as having minimal brain dysfunction had no significant "over all" differences in the number of errors and the number of nonresponses on the half second and five second delay between the word pairs. This was an interesting and unexpected finding.

Another interesting finding (on which no measurements were taken) was that when the MBD children returned to the classroom, the teachers reported that they were totally "wiped out" for the rest of the day. That is, they were physically exhausted and the teachers stated that they would allow the child to rest his head on his desk. Teachers reported no difference in the controls behavior before and after testing. Apparently the experimental procedure exhausted the MBD child but had no observable effects on the controls. This is also commensurate with the linear progression of errors and nonresponses across replicates for MBD children.

Within the limits imposed by the research design the following conclusions appear warranted: These results strongly suggest that children diagnosed as having minimal brain dysfunction do much poorer than normal children on an auditory discrimination task. Apparently there was a learning and a fatigue factor operating within these populations. There appears to be a learning factor associated with the control's performance because their performance improved over trials. That is, they made fewer errors as the test increased in time. The performance of children diagnosed as having minimal brain dysfunction deteriorated over trials, i.e., they made many more errors as the test progressed. They also had a progressive increase in the number of non-responses across replicates. This indicates that there was a fatigue factor associated with their performance that was not evident in the control group. Teacher reports on the children's behavior after they returned to the classroom further suggests that this factor influenced the performance of MBD children.

A question may be raised as to the role motivation played in the present procedure and if this factor could account for the results. This does not appear likely because all children were given two dollars after their participation in the experiment and all seemed eager to earn this money. On the school grounds and on the first floor of the Child Study Center children would ask if they could take part in the study. Comments such as "take me", "I want to go", and "let me help you" were common.

This leaves two other factors to consider---attention and auditory discrimination. The child has to be able to attend to the words he is going to discriminate. Separating attention from true deficits in auditory or visual perception has been, so far, an insurmountable problem for researchers. It is very difficult, therefore, to ascertain whether or not actual physiological deficits exists in the various modalities between MBD and normal children. A rapid decay of attention has been one of the key signs in diagnosing children as having minimal brain dysfunction. Peter's (1974) states that the concentration mechanism seems to fatigue as rapidly in a nine-year-old MBD child as it does in a normal three-year-old child. Therefore the question may be raised as to whether or not attention was a primary factor that contributed to the poor performance of MBD children. Attention does appear to be a primary factor. Kahneman (1973), in Attention and Effort, indicates that attention and fatigue are linked together conceptually, i.e., you couldn't have a fatigue effect without suspecting to also have an attention effect. They are intricately related. The most important finding was that the normal child did not appear to have any problems with fatigue or attention. Their performance improved across replicates and they responded to essentially every item.

The present results suggest that auditory discrimination was also an important factor that contributed to the poor performance of MBD children. Three indicators were: (1) On the first replicate where attention would be at an optimal level and fatigue was at best minimal, children diagnosed as having MBD made almost three times as many errors as normal children. With these two factors in check, it is highly probable that the only other factor that could account for their poor performance was auditory discrimination. (2) MBD's made more errors across replicates on identical and dissimilar word pairs and less errors across replicates on similar word pairs. This may, at first, appear that auditory discrimination would be counterindicated. However, MBD's had a much higher error rate and many more nonresponses on similar word pairs across replicates. This suggests that as the test progressed they became tired and had more difficulty in discriminating between pairs of words that were similar. If incorrect responses and nonresponses were combined, similar word pairs would have also increased across replicates. (3) There was no significant "over all" difference between the .5 second and the five second delay between the word pairs. If attention and fatigue had been the only factors, MBD's should have made many more errors on the five second delay. This indicates that auditory discrimination was a key factor in their over-all performance. MBD's appear to have difficulty discriminating between pairs of words regardless of the time of presentation.

An unexpected finding was that there were no significant "over all" differences between the .5 second and the five second delay between the word pairs for children diagnosed as having MBD. These children are often described as having a short attention span. If this had been a key factor in their performance they should have made many more errors on the five second delay. Deficits in short-term memory have also been implicated (Clements 1966, Dykman 1971, and Peters 1974). These results suggest that MBD children do not show deficits in short-term auditory memory within the limits of the present procedures.

In summary, it appears that three factors contributed to the results: (1) fatigue, (2) attention, and (3) auditory discrimination ability. Fatigue and attention appear to be the primary factors contributing to the decline in performance across replicates for MBD children. Children diagnosed as having MBD made many more errors than the controls on all three sets of word pairs and on all three replicates which indicates deficits in their auditory discrimination ability.

The present study does not prove that MBD children have auditory discrimination deficits. It does however indicate that children diagnosed as having minimal brain dysfunction do much worse than normal children on auditory discrimination tasks and that fatigue and attention appear to be significant factors.

The results from the present study support the findings from earlier studies (e.g., Hardy, 1959; DeHirsch, 1966; Aten and Davis, 1968; Heilman, 1968; Wepman, 1968; Zigmond, 1968; Doehring and Rabinovitch, 1969; Spring, 1971; Rampp, 1972) and represents the first study on auditory discrimination in children diagnosed as having minimal brain dysfunction.

From the findings in the present study the following recommendations appear to be in order:

1. The present study needs to be replicated to substantiate these findings and to establish a stronger link between auditory discrimination deficits and children diagnosed as having minimal brain dysfunction.
2. Groups could be set up to partial out, as much as possible, the three factors indicated as important.
3. Longer intrastimulus intervals could be devised to measure the reaction times of minimal brain dysfunctions as compared to normal children.
4. Various interstimulus intervals could be employed to investigate possible deficits in short-term memory.
5. Various schedules of reinforcement could be employed to determine what effects this might have on the child's performance.

6. A Lykert type questionnaire could be devised and given to the teacher so that measurements could be taken on the child's performance after he returned to the classroom.
7. Different groups could receive various stimulant drugs to see what effect this might have on the child's performance.
8. Different groups of children could be used to determine their proficiency in auditory discrimination, e.g., minimal brain dysfunction, normal, mentally retarded, and gifted children.
9. A portable tape unit with headphones could be used by the researcher to take to the various schools where the children were to be tested. This would economize on the time required for gathering the data.
10. A follow-up study on these children would be interesting to see if these results remained constant in adolescence and adulthood.

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APPENDIXES

APPENDIX A

SIGNS OF MBD AS USED BY THE UNIVERSITY OF ARKANSAS MEDICAL CENTER

1. Rapid Decay of Attention:

   The child quickly tires of a new object. The concentration mechanism seems to fatigue as rapidly in a nine-year-old MBD child as it would in a normal three-year-old child.

2. Distractibility:

   Very slight or ordinary stimuli will deflect the MBD child from what he is doing, saying or thinking.

3. Pattern Perception:

   Out of a welter of stimuli, proprioceptive, visual, auditory, or social, the MBD child has trouble locating the pertinent cues and patterns.

4. Holding a plan or Pattern:

   This is related to that of pattern perception but emphasizes the ability to keep a plan in mind as it is carried out.

5. Hyperactivity:

   The MBD child fidgets, moves, or talks excessively.

6. Impulsiveness:

   The MBD child often immediately blurts out whatever he is thinking or touches what he is looking at. He impulsively jumps to conclusions.

7. Labile Emotions:

The MBD child seems to lack the inhibitory circuitry required to hold emotions within well-modulated bounds.

8. Disorders in Language Development:

Slowness in acquiring the use of sentences is a sensitive, early sign of MBD. Another useful sign is slowness in learning to detect and use the different vowel sounds and blends of sounds.

9. Disturbance in Directionality:

This problem often includes confusion of right and left, up and down, in front or behind, inside - outside and before - after.

10. Motor Incoordination:

In most cases the principal motor manifestations are fine movement defects of fingers and hands. The most useful test of fine motor functions are: (a) touching fingers to thumb in sequence, over and over; (b) alternating movements of hands; and (c) writing to dictation.

For a diagnosis of MBD, the child must have three or more of these signs and an I.Q. in the near average range or above, as determined by an individually administered I.Q. test.

APPENDIX B

SUMMARY OF ANALYSIS OF VARIANCE FOR INCORRECT
RESPONSES (I) ON DISSIMILAR WORD PAIRS

Source	d.f.	S.S.	M.S.	F	P
Total	311	21.56601			
Among Subjects	51				
G	1	3.04733	3.04733	10.37	<.01
O	1	0.00003	0.00003	1	ns
G X O	1	0.00098	0.00098	1	ns
S/(G,O)	48	14.10916	0.29394		
Within Subjects	260				
D	1	0.00023	0.00023	1	ns
R	2	0.12716	0.06358	3.87	<.05
D X R	2	0.02170	0.01085	1	ns
D X G	1	0.00267	0.00267	1	ns
D X O	1	0.00225	0.00225	1	ns
D X G X O	1	0.00012	0.00012	1	ns
R X G	2	0.15404	0.07702	4.69	<.01
R X O	2	0.02329	0.01165	1	ns
R X G X O	2	0.01086	0.00543	1	ns
D X R X G	2	0.03519	0.01759	1.07	ns
D X R X O	2	0.04610	0.02305	1.40	ns
D X R X G X O	2	0.04471	0.02236	1.36	ns
Remainder	240	3.94010	0.01642		

APPENDIX C

SUMMARY OF ANALYSIS OF VARIANCE FOR INCORRECT RESPONSES (I) ON IDENTICAL WORD PAIRS

Source	d.f.	S.S.	M.S.	F	P
Total	311	19.69464			
Among Subjects	51				
G	1	3.76485	3.76485	21.94	<.01
O	1	0.42974	0.42974	2.50	ns
G X O	1	0.02752	0.02752	1	ns
S/ (G,O)	48	8.23753	0.17162		
Within Subjects	260				
D	1	0.00491	0.00491	1	ns
R	2	0.20194	0.10097	3.59	<.05
D X R	2	0.05038	0.02519	1	ns
D X G	1	0.12234	0.12234	4.35	<.05
D X O	1	0.00516	0.00516	1	ns
D X G X O	1	0.00850	0.00850	1	ns
R X G	2	0.02255	0.01128	1	ns
R X O	2	0.00179	0.00090	1	ns
R X G X O	2	0.03681	0.01841	1	ns
D X R X G	2	0.00024	0.00012	1	ns
D X R X O	2	0.01476	0.00738	1	ns
D X R X G X O	2	0.01145	0.00572	1	ns
Remainder	240	6.75417	0.02814		

APPENDIX D

SUMMARY OF ANALYSIS OF VARIANCE FOR INCORRECT

RESPONSES (I) ON SIMILAR WORD PAIRS

Source	d.f.	S.S.	M.S.	F	P
Total	331	13.06108			
Among Subjects	51				
G	1	4.73231	4.73231	36.5	$<$.01
O	1	0.11434	0.11434	1	ns
G X O	1	0.0004	0.0004	1	ns
S/ (G,O)	48	6.22362	0.12966		
Within Subjects	260				
D	1	0.00184	0.00184	1	ns
R	2	0.06308	0.03154	4.41	$<$.01
D X R	2	0.01484	0.00742	1.03	ns
D X G	1	0.02129	0.02129	2.97	ns
D X O	1	0.02265	0.00265	3.16	ns
D X G X O	1	0.00317	0.00317	1	ns
R X G	2	0.00546	0.00273	1	ns
R X O	2	0.02875	0.01438	2.01	ns
R X G X O	2	0.01653	0.00827	1.16	ns
D X R X G	2	0.02575	0.01288	1.80	ns
D X R X O	2	0.05759	0.02880	4.02	$<$.01
D X R X G X O	2	0.01152	0.00576	1	ns
Remainder	240	1.71830	0.00716		

APPENDIX E

SUMMARY OF FISHER'S EXACT TEST

FOR NONRESPONSES(N)

Dissimilar Word Pairs

		Gave a Response to all 10			Nonresponse to at least one pair		
		R_1	R_2	R_3	R_1	R_2	R_3
s	C	26	26	26	0	0	0
	MBD	23	19	17	3	7*	9 Δ
S	C	26	26	26	0	0	0
	MBD	23	23	16	3	3	10 ∞

* P = .005
Δ P = .001
∞ P = .000

Identical Word Pairs

		Gave a response to all 10 items			Nonresponse to at least one pair		
		R_1	R_2	R_3	R_1	R_2	R_3
s	C	26	26	26	0	0	0
	MBD	23	15	16	3	11∞	10 ∞
S	C	26	25	26	0	1	0
	MBD	24	20	20	2	6*	6**

* P = .05
** P = .01
∞ P = .000

Similar Word Pairs

		Gave a response to all 30			Nonresponse to at least one pair		
		R_1	R_2	R_3	R_1	R_2	R_3
s	C	26	25	26	0	1	C
	MBD	18	14	12	8°	12^{∞}	14^{∞}
S	C	25	25	24	1	1	2
	MBD	22	12	13	4	14^{∞}	13^{Δ}

$^{\circ}$ P = .002
$^{\Delta}$ P = .001
$^{\infty}$ P = .000

APPENDIX F

SUMMARY OF CONTROLS

Total Number Missed 711.

Number missed in replicate 1 256; rep. 2 234; rep. 3 221.

Replicate I: ½ sec. missed 137. 5 sec. missed 119.

Replicate II: ½ sec. missed 116. 5 sec. missed 118.

Replicate III: ½ sec. missed 109. 5 sec. missed 112.

Errors on Identical Pairs 40.

IP missed in rep. 1 15; rep. 2 7; rep. 3 18.

Replicate I: ½ sec. IP missed 9. 5 sec. IP missed 6.

Replicate II: ½ sec. IP missed 3. 5 sec. IP missed 4.

Replicate III: ½ sec. IP missed 10. 5 sec. IP missed 8.

Errors on Similar Pairs 664.

Sp missed in rep. 1 239; rep. 2 224; rep. 3 201.

Replicate I: ½ sec. SP missed 128. 5 sec. SP missed 111.

Replicate II: ½ sec. SP missed 111. 5 sec. SP missed 113.

Replicate III: ½ sec. SP missed 98. 5 sec. SP missed 103.

Errors on Dissimilar Pairs 1.

DP missed in rep. 1 1; rep. 2 0; rep. 3 0.

Replicate I: ½ sec. DP missed 0. 5 sec. DP missed 1.

Replicate II: ½ sec. DP missed 0. 5 sec. DP missed 0.

Replicate III: ½ sec. DP missed 0. 5 sec. DP missed 0.

Nonresponses 6.

NR in rep. 1 1; rep. 2 3; rep. 3 2.

Replicate I: ½ sec. NR 0. 5 sec. NR 1.

Replicate II: ½ sec. NR 2. 5 sec. NR 1.

Replicate III: ½ sec. NR 1. 5 sec. NR 1.

APPENDIX G

SUMMARY OF EXPERIMENTAL GROUP

Total Number Missed 2,317.

Number missed in replicate 1 699; rep. 2 763; rep. 3 855.

Replicate I: ½ sec. missed 335. 5 sec. missed 364.

Replicate II: ½ sec. missed 386. 5 sec. missed 377.

Replicate III: ½ sec. missed 409. 5 sec. missed 446.

Errors on Identical Pairs 213.

IP missed in rep. 1 62; rep. 2 68; rep. 3 83.

Replicate I: ½ sec. IP missed 30. 5 sec. IP missed 32.

Replicate II: ½ sec. IP missed 28. 5 sec. IP missed 40.

Replicate III: ½ sec. IP missed 38. 5 sec. IP missed 45.

Errors on Similar Pairs 1,632.

SP missed in rep. 1 559; rep. 2 542; rep. 3 531.

Replicate I: ½ sec. SP missed 269. 5 sec. SP missed 290.

Replicate II: ½ sec. SP missed 275. 5 sec. SP missed 267.

Replicate III: ½ sec. SP missed 250. 5 sec. SP missed 281.

Errors on Dissimilar Pairs 164.

DP missed in rep. 1 44; rep. 2 48; rep. 3 72.

Replicate I: ½ sec. DP missed 25. 5 sec. DP missed 19.

Replicate II: ½ sec. DP missed 22. 5 sec. DP missed 26.

Replicate III: ½ sec. DP missed 38. 5 sec. DP missed 34.

Nonresponse 308.

NR in rep. 1 34; rep. 2 105; rep. 3 169.

Replicate I: ½ sec. NR 18. 5 sec. NR 16.

Replicate II: ½ sec. NR 61. 5 sec. NR 44.

Replicate III: ½ sec. NR 83. 5 sec. NR 86.

APPENDIX H

LETTER SENT TO THE PARENTS OF MBD CHILDREN

Dear Parent,

I am writing a dissertation on Auditory Discrimination in Children with learning problems. I would like your permission to include your child in this study. This will take place while he is in the Therapeutic Day School. The test will last approximately one hour and he will be paid $2.00 for his participation in the study.

If your child is on medication, he will have to be taken off two days prior to his being tested. After testing he will be placed back on his regular schedule.

It is hoped that through this study we will learn not only more about your child but more about children with learning disabilities.

Thank you,

Vincent E. Parr
Doctoral Student in
Clinical Psychology

This research study has our approval and will be done under our supervision.

Sam D. Clements, Ph.D
Professor, Department of Psychiatry
Executive Director
Child Study Center

John E. Peters, M. D.
Professor & Head Division
Child-Adolescent Psychiatry

(Miss) Jean E. Lukens, M.S.
Instructor in Psychiatry
Educational Director
Therapeutic Day School

I, the undersigned, give my consent for my child to participate in the research study being conducted at the Child Study Center by Vincent E. Parr.

____

Parent's Signature

APPENDIX I

TEST INSTRUCTIONS ON AUDITORY TAPE

Hello! ---You will be paid $2.00 for your participation in this experiment.

I want to see if you can tell the difference between words that are the same and words that are different.

Listen carefully to the following instructions:

You will hear a "beep" on the tape. After this two words will be presented. I want you to respond as fast as possible by pressing one of the buttons on the table in front of you.

If the words are the same, press the button marked "same". If they are different, press the button marked "different".

Some of the words will be further apart than others. However, there will always be a "beep" before the two words that you will respond to as same or different.

There will be a practice session before the actual experiment begins. This is the time to ask questions if you do not understand what you are to do.

After this practice session I will tell you that the experiment is about to start.

Once the experiment starts, there can be no further questions.

The experiment will last approximately one hour.

The following is the practice session:

1. bold - cold
2. pile - pile
3. mark - mark
4. free - tree

5. thin – zest
6. thick – thick
7. gap – gap
8. fun – net
9. gag – tie
10. blot – pot

APPENDIX J

THE FIRST SET OF FIFTY WORD PAIRS (s_1)

1.	shake	-	shape	26.	fie	-	thigh
2.	book	-	date	27.	pat	-	pet
3.	tub	-	tug	28.	bass	-	bath
4.	slate	-	hook	29.	sought	-	fought
5.	zest	-	zest	30.	wretch	-	wretch
6.	thimble	-	symbol	31.	rake	-	doll
7.	tin	-	pin	32.	match	-	read
8.	house	-	dial	33.	jam	-	jam
9.	pat	-	pack	34.	tree	-	hoe
10.	man	-	cat	35.	vow	-	thou
11.	web	-	wed	36.	badge	-	badge
12.	bale	-	gale	37.	lack	-	lack
13.	king	-	king	38.	leg	-	led
14.	coast	-	toast	39.	din	-	bin
15.	shack	-	sack	40.	dog	-	strike
16.	tall	-	tall	41.	phone	-	bait
17.	dim	-	din	42.	moon	-	noon
18.	gum	-	dumb	43.	shoal	-	shawl
19.	cat	-	cap	44.	pose	-	pose
20.	clothe	-	clove	45.	muff	-	muss
21.	girl	-	lake	46.	par	-	par
22.	lease	-	leash	47.	pork	-	cork
23.	sheaf	-	sheath	48.	chap	-	chap
24.	lath	-	lash	49.	pen	-	pin
25.	thread	-	shread	50.	bum	-	bomb

APPENDIX K

THE SECOND SET OF FIFTY WORD PAIRS (S_2)

1.	zest	-	zest	26.	par	-	par
2.	slate	-	hook	27.	web	-	wed
3.	lack	-	lack	28.	king	-	king
4.	clove	-	clothe	29.	wretch	-	wretch
5.	thread	-	shread	30.	muff	-	muss
6.	toast	-	coast	31.	leash	-	lease
7.	lake	-	girl	32.	bass	-	bath
8.	house	-	dial	33.	dumb	-	gum
9.	bin	-	din	34.	strike	-	dog
10.	sack	-	shack	35.	thou	-	vow
11.	sought	-	fought	36.	leg	-	led
12.	pin	-	tin	37.	bale	-	gale
13.	pose	-	pose	38.	dim	-	din
14.	rake	-	doll	39.	pin	-	pen
15.	date	-	book	40.	lash	-	lath
16.	thigh	-	fie	41.	phone	-	bait
17.	tree	-	hoe	42.	pork	-	cork
18.	cap	-	cat	43.	tug	-	tub
19.	pet	-	pat	44.	pat	-	pack
20.	thimble	-	symbol	45.	shoal	-	shawl
21.	sheaf	-	sheath	46.	shape	-	shake
22.	bum	-	bomb	47.	tall	-	tall
23.	cat	-	man	48.	match	-	read
24.	chap	-	chap	49.	jam	-	jam
25.	moon	-	noon	50.	badge	-	badge

APPENDIX L

THE THIRD SET OF FIFTY WORD PAIRS (s_3)

1.	hook	-	slate		26.	sack	-	shack
2.	muss	-	muff		27.	bass	-	bath
3.	pat	-	pack		28.	noon	-	moon
4.	zest	-	zest		29.	man	-	cat
5.	leg	-	led		30.	read	-	match
6.	fie	-	thigh		31.	tub	-	tug
7.	dial	-	house		32.	pork	-	cork
8.	coast	-	toast		33.	king	-	king
9.	bale	-	gale		34.	clothe	-	clove
10.	par	-	par		35.	gum	-	dumb
11.	tree	-	hoe		36.	din	-	dim
12.	bin	-	din		37.	pose	-	pose
13.	leash	-	lease		38.	tall	-	tall
14.	thou	-	vow		39.	date	-	book
15.	lash	-	lath		40.	thimble	-	symbol
16.	shake	-	shape		41.	badge	-	badge
17.	lack	-	lack		42.	tin	-	pin
18.	chap	-	chap		43.	sheaf	-	sheath
19.	bum	-	bomb		44.	dog	-	strike
20.	girl	-	lake		45.	doll	-	rake
21.	fought	-	sought		46.	shread	-	thread
22.	pin	-	pen		47.	wretch	-	wretch
23.	pet	-	pat		48.	bait	-	phone
24.	jam	-	jam		49.	cat	-	cap
25.	shawl	-	shoal		50.	wed	-	web

APPENDIX M

THE FOURTH SET OF FIFTY WORD PAIRS (S_4)

1.	dim	-	din	26.	sheaf	-	sheath
2.	tall	-	tall	27.	girl	-	lake
3.	strike	-	dog	28.	badge	-	badge
4.	tree	-	hoe	29.	vow	-	thou
5.	thimble	-	symbol	30.	bin	-	din
6.	dumb	-	gum	31.	bait	-	phone
7.	tub	-	tug	32.	pen	-	pin
8.	wretch	-	wretch	33.	fought	-	sought
9.	jam	-	jam	34.	pin	-	tin
10.	bum	-	bomb	35.	zest	-	zest
11.	slate	-	hook	36.	sack	-	shack
12.	wed	-	web	37.	leg	-	led
13.	pet	-	pat	38.	pack	-	pat
14.	pose	-	pose	39.	pork	-	cork
15.	bath	-	bass	40.	par	-	par
16.	cap	-	cat	41.	lease	-	leash
17.	doll	-	rake	42.	dial	-	house
18.	read	-	match	43.	cat	-	man
19.	thigh	-	fie	44.	king	-	king
20.	book	-	date	45.	noon	-	moon
21.	coast	-	toast	46.	shoal	-	shawl
22.	shape	-	shake	47.	chap	-	chap
23.	lack	-	lack	48.	muff	-	muss
24.	clove	-	clothe	49.	thread	-	shread
25.	lath	-	lash	50.	gale	-	bale

APPENDIX N

THE FIFTH SET OF FIFTY WORD PAIRS (s_5)

1.	bum	-	bomb	26.	bale	-	gale
2.	shread	-	thread	27.	sack	-	shack
3.	led	-	leg	28.	muff	-	muss
4.	bin	-	din	29.	tree	-	hoe
5.	lease	-	leash	30.	thigh	-	fie
6.	pet	-	pat	31.	zest	-	zest
7.	date	-	book	32.	pen	-	pin
8.	vow	-	thou	33.	shoal	-	shawl
9.	bass	-	bath	34.	cork	-	pork
10.	pack	-	pat	35.	man	-	cat
11.	badge	-	badge	36.	sheaf	-	sheath
12.	read	-	match	37.	par	-	par
13.	tall	-	tall	38.	wretch	-	wretch
14.	wed	-	web	39.	moon	-	noon
15.	symbol	-	thimble	40.	clothe	-	clove
16.	tub	-	tug	41.	dumb	-	gum
17.	king	-	king	42.	tin	-	pin
18.	coast	-	toast	43.	lack	-	lack
19.	chap	-	chap	44.	shape	-	shake
20.	house	-	dial	45.	pose	-	pose
21.	din	-	dim	46.	phone	-	bait
22.	doll	-	rake	47.	cat	-	cap
23.	strike	-	dog	48.	fought	-	sought
24.	slate	-	hook	49.	jam	-	jam
25.	lash	-	lath	50.	lake	-	girl

APPENDIX O

THE SIXTH SET OF FIFTY WORD PAIRS (S_6)

1.	dog	-	strike	26.	jam	-	jam
2.	shread	-	thread	27.	zest	-	zest
3.	din	-	dim	28.	thou	-	vow
4.	pet	-	pat	29.	chap	-	chap
5.	bass	-	bath	30.	lake	-	girl
6.	muff	-	muss	31.	pose	-	pose
7.	slate	-	hook	32.	tall	-	tall
8.	shawl	-	shoal	33.	web	-	wed
9.	bale	-	gale	34.	rake	-	doll
10.	shack	-	sack	35.	shape	-	shake
11.	cat	-	cap	36.	gum	-	dumb
12.	clothe	-	clove	37.	bin	-	din
13.	dial	-	house	38.	date	-	book
14.	read	-	match	39.	lash	-	lath
15.	lack	-	lack	40.	lease	-	leash
16.	pat	-	pack	41.	toast	-	coast
17.	pen	-	pin	42.	badge	-	badge
18.	pork	-	cork	43.	wretch	-	wretch
19.	bum	-	bomb	44.	tub	-	tug
20.	sheaf	-	sheath	45.	tree	-	hoe
21.	noon	-	moon	46.	tin	-	pin
22.	par	-	par	47.	bait	-	phone
23.	cat	-	man	48.	thimble	-	symbol
24.	led	-	leg	49.	king	-	king
25.	thigh	-	fie	50.	sought	-	fought

APPENDIX P

SCORE SHEET FOR FIRST SET OF

FIFTY WORD PAIRS (s_1)

Order Received: 1______ or 2______. MBD______, Control______.

Name of Child________________________. Age___. I.Q.___. Date___.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(G)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

1	S	D	26	S	D
d 2	S	D	27	S	D
3	S	D	28	S	D
d 4	S	D	29	S	D
* 5	S	D	* 30	S	D
6	S	D	d 31	S	D
7	S	D	d 32	S	D
d 8	S	D	* 33	S	D
9	S	D	d 34	S	D
d 10	S	D	35	S	D
11	S	D	* 36	S	D
12	S	D	* 37	S	D
* 13	S	D	38	S	D
14	S	D	39	S	D
15	S	D	d 40	S	D

* 16	S	D
17	S	D
18	S	D
19	S	D
20	S	D
d 21	S	D
22	S	D
23	S	D
24	S	D
25	S	D

d 41	S	D
42	S	D
43	S	D
* 44	S	D
45	S	D
* 46	S	D
47	S	D
* 48	S	D
49	S	D
50	S	D

APPENDIX Q

SCORE SHEET FOR SECOND SET OF

FIFTY WORD PAIRS (s_2)

Order Received: 1______or 2______. MBD_______, Control________.

Name of Child_____________________. Age___. I.Q.____. Date____.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(C)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

* 1	S	D	* 26	S	D
d 2	S	D	27	S	D
* 3	S	D	* 28	S	D
4	S	D	* 29	S	D
5	S	D	30	S	D
6	S	D	31	S	D
d 7	S	D	32	S	D
d 8	S	D	33	S	D
9	S	D	d 34	S	D
10	S	D	35	S	D
11	S	D	36	S	D
12	S	D	37	S	D
* 13	S	D	38	S	D
d 14	S	D	39	S	D

d	15	S	D
	16	S	D
d	17	S	D
	18	S	D
	19	S	D
	20	S	D
	21	S	D
	22	S	D
d	23	S	D
*	24	S	D
	25	S	D

	40	S	D
d	41	S	D
	42	S	D
	43	S	D
	44	S	D
	45	S	D
	46	S	D
*	47	S	D
d	48	S	D
*	49	S	D
*	50	S	D

APPENDIX R

SCORE SHEET FOR THIRD SET OF

FIFTY WORD PAIRS (s_3)

Order Received: 1______ or 2______. MBD________, Control__________.

Name of Child____________________. Age_____. I.Q._____. Date_____.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(G)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

d	1	S	D		26	S	D
	2	S	D		27	S	D
	3	S	D		28	S	D
*	4	S	D	d	29	S	D
	5	S	D	d	30	S	D
	6	S	D		31	S	D
d	7	S	D		32	S	D
	8	S	D	*	33	S	D
	9	S	D		34	S	D
*	10	S	D		35	S	D
d	11	S	D		36	S	D
	12	S	D	*	37	S	D
	13	S	D	*	38	S	D
	14	S	D	d	39	S	D

15	S	D
16	S	D
* 17	S	D
* 18	S	D
19	S	D
d 20	S	D
21	S	D
22	S	D
23	S	D
* 24	S	D
25	S	D

40	S	D
* 41	S	D
42	S	D
43	S	D
d 44	S	D
d 45	S	D
46	S	D
* 47	S	D
d 48	S	D
49	S	D
50	S	D

APPENDIX S

SCORE SHEET FOR FOURTH SET OF

FIFTY WORD PAIRS (s_4)

Order Received: 1______or 2______. MBD________, Control___________.

Name of Child_____________________. Age____. I.Q._____. Date_______.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(G)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

1	S	D	26	S	D
* 2	S	D	d 27	S	D
d 3	S	D	* 28	S	D
d 4	S	D	29	S	D
5	S	D	30	S	D
6	S	D	d 31	S	D
7	S	D	32	S	D
* 8	S	D	33	S	D
* 9	S	D	34	S	D
10	S	D	* 35	S	D
d 11	S	D	36	S	D
12	S	D	37	S	D
13	S	D	38	S	D
* 14	S	D	39	S	D

	15	S	D
	16	S	D
d	17	S	D
d	18	S	D
	19	S	D
d	20	S	D
	21	S	D
	22	S	D
*	23	S	D
	24	S	D
	25	S	D

*	40	S	D
	41	S	D
d	42	S	D
d	43	S	D
*	44	S	D
	45	S	D
	46	S	D
*	47	S	D
	48	S	D
	49	S	D
	50	S	D

APPENDIX T

SCORE SHEET FOR FIFTH SET OF

FIFTY WORD PAIRS (s_5)

Order Received: 1______or 2______. MBD_______, Control____________.

Name of Child_____________________. Age____. I.Q._____. Date_______.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(G)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

1	S	D	26	S	D
2	S	D	27	S	D
3	S	D	28	S	D
4	S	D	d 29	S	D
5	S	D	30	S	D
6	S	D	* 31	S	D
d 7	S	D	32	S	D
8	S	D	33	S	D
9	S	D	34	S	D
10	S	D	d 35	S	D
* 11	S	D	36	S	D
d 12	S	D	* 37	S	D
* 13	S	D	* 38	S	D
14	S	D	39	S	D

15	S	D
16	S	D
* 17	S	D
18	S	D
* 19	S	D
d 20	S	D
21	S	D
d 22	S	D
d 23	S	D
d 24	S	D
25	S	D

40	S	D
41	S	D
42	S	D
* 43	S	D
44	S	D
* 45	S	D
d 46	S	D
47	S	D
48	S	D
* 49	S	D
d 50	S	D

APPENDIX U

SCORE SHEET FOR SIXTH SET OF

FIFTY WORD PAIRS (s_6)

Order Received: 1______or 2______. MBD________, Control___________.

Name of Child____________________. Age_____. I.Q.______. Date_____.

EXAMPLES

(A)	S	D	(F)	S	D
(B)	S	D	(G)	S	D
(C)	S	D	(H)	S	D
(D)	S	D	(I)	S	D
(E)	S	D	(J)	S	D

****

d 1	S	D	* 26	S	D
2	S	D	* 27	S	D
3	S	D	28	S	D
4	S	D	* 29	S	D
5	S	D	d 30	S	D
6	S	D	* 31	S	D
d 7	S	D	* 32	S	D
8	S	D	33	S	D
9	S	D	d 34	S	D
10	S	D	35	S	D
11	S	D	36	S	D
12	S	D	37	S	D
d 13	S	D	d 38	S	D
d 14	S	D	39	S	D

* 15	S	D
16	S	D
17	S	D
18	S	D
19	S	D
20	S	D
21	S	D
* 22	S	D
d 23	S	D
24	S	D
25	S	D

40	S	D
41	S	D
* 42	S	D
* 43	S	D
44	S	D
d 45	S	D
46	S	D
d 47	S	D
48	S	D
* 49	S	D
50	S	D

VITA

Vincent Ernest Parr

Candidate for the Degree of

Doctor of Philosophy

Thesis: THE PERFORMANCE OF CHILDREN DIAGNOSED AS HAVING MINIMAL BRAIN DYSFUNCTION ON AN AUDITORY DISCRIMINATION TASK

Major Field: Psychology

Biographical:

Personal Data: Born in Jacksonville, Florida on September 26, 1943, the son of Mr. and Mrs. Vincent Theodore Parr.

Education: Graduated from S. R. Butler High School, Huntsville, Alabama, in May, 1962; received the Bachelor of Arts degree in psychology from Auburn University, Auburn, Alabama in June, 1967; received the Master of Science degree in counseling psychology from Auburn University in August, 1969; completed requirements for the Doctor of Philosophy degree at Oklahoma State University in July, 1976.

Professional Experience: Internship in Clinical Psychology, University of Arkansas Medical Center, 1972-1973; Psychologist, Department of Psychiatry, University of Arkansas Medical Center, 1973-1975; Coordinator of Clinical Services, Ozark Regional Mental Health Center, Harrison, Arkansas, 1975 - present; Will join the staff of The Institute for Rational Living (Florida Branch), Clearwater, Florida, in September, 1976.