THE EFFECTS OF METHYLPHENIDATE (RITALIN) ON THE NEUROPSYCHOLOGICAL STATUS OF LEARNING DISABLED-MINIMAL BRAIN DYSFUNCTION CHILDREN

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in partial fulfillment of the requirements
for the Degree of
DOCTOR OF PHILOSOPHY
December, 1977

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ACKNOWLEDGMENTS

As I complete the final tasks associated with the fulfillment of years of academic effort, I feel that I, like the star who has just received the academy award for her part in a movie with a "cast of thousands," owe my triumph to the assistance of innumerable individuals. I could not have completed this dissertation without the editorial assistance of Dr. William Jaynes, final chairman of my doctoral committee; Dr. Philip Murphy of my doctoral committee; and Dr. Leslie Ruthven, my husband. Drs. Robert Schlattmann, Joseph Pearl and David Perrin, of my doctoral committee, gave much appreciated support of my efforts. Dr. Kenneth Sandvold, a former doctoral committee member, gave hours of moral support and encouragement, as did Dr. Richard Prawat, former chairman of my doctoral committee.

I also wish to express my appreciation for the review of specific sections of my dissertation and their helpful comments to two men I never met personally. Dr. Frank Holloway from the Health Sciences Center in Oklahoma City, and Dr. Robert Gholson from the Biochemistry Department at Oklahoma State University, gave me the benefit of their expertise. This assistance was greatly appreciated.

I owe untold gratitude to many members of the Hays and Wichita public schools, without whose help this research would never have been possible. I wish to express my especial gratitude to

Mrs. Ester Spomer, school psychologist from Hays School District; Mrs. Lorenne Gurley, Wichita Public Schools; Dr. A. W. Dicks, Director of Research, Planning and Development Services for the Wichita schools; Dr. Lynn Silvertooth, Principal, Enterprise Elementary School, Wichita; and also to Dr. James Ryabik, Director of the Fort Hays State University's Psychology Clinic, who so generously gave me the use of the clinic facilities for testing.

I wish to thank the parents and children who assisted me in my efforts to gain some greater understanding of the mechanisms of the LD-MBD syndrome. Without their help there would have been no research.

I cannot fail to thank three of those women without whom the business of America would grind to a halt, the secretaries. To Barbara Moore, whose unfailing good humor and ingenuity kept me in contact with Dr. Jaynes; to Diane Deeble, my husband's secretary who typed and re-typed many sections of this work and worked around my preoccupations with it when I was needed for other duties, and to Sandy Harris, who patiently typed and waited, and typed some more to produce the final draft, I extend my undying gratitude.

Last, I wish to thank those individuals without whom I could never have completed my schooling or research, my family. I fondly dedicate this dissertation to them. They helped and supported me even when their lives were less orderly and comfortable. My husband, Les Ruthven, gave much technical assistance with my research and writing, for which I am deeply grateful, but even more importantly, he wanted me to succeed. Our children, Beth, Leslie and Laura,

gave in so many ways that I cannot enumerate them, but, again, they wanted me to succeed in what I wanted and I shall always be grateful.

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CHAPTER I

INTRODUCTION

The inability of a school child with normal intelligence to perform academically at the level expected of him has been a perplexing problem for educators and parents alike. Parents and educators have varied between blaming themselves, each other or the child. Along with the knowledge that some children are not learning as expected has come the recognition that many of these children also fail to keep their activity level at a point which the teacher or parent can tolerate comfortably. Professionals and parents have sought the responsible agent for the hyperactivity and learning impairment of these children. As will be discussed later, a minimal dysfunction of the cerebral cortex has been postulated to account for these deficits along with a cluster of other symptoms often associated with hyperactivity and learning problems.

The treatment for these children which is in most widespread use at this time is the administration of one of the stimulant drugs. How these drugs modify the symptom complex in these children is far from understood but the most prevalent thought is that these drugs alter neural response patterns (see literature review). Despite the lack of understanding on the specific neurophysiology involved, there is a general belief among many professionals that such drug therapy reduces unacceptable behavior for many of these children. Anecdotal evidence

suggests that stimulant medication often does produce favorable gross behavioral changes in these children, but an open question remains as to whether or not stimulants alter the basic neuropsychological functioning of these children.

The major focus of this study will be to assess the effects of a major stimulant drug, methylphenidate (Ritalin), on the neuropsychological status of these children. The efficacy of Ritalin on school performance is not the issue here. The question which is being considered is whether or not the drug alters neuropsychological functioning as measured by the Halstead-Reitan Neuropsychological Battery for older children. Two groups of children with learning disabilities, one group receiving Ritalin therapy and the other group without current Ritalin treatment, will be assessed neuropsychologically to determine if the drug alters basic neuropsychological status. All children in the study will be tested on the Halstead-Reitan Neuropsychological Test Battery for Older Children, which will be used to assess neuropsychological functioning. The test performance of the two groups (Ritalin vs. non-Ritalin) will be analyzed statistically to determine whether any drug associated differences in neuropsychological functioning can be shown.

CHAPTER II

LITERATURE REVIEW

George Kaluger and Clifford Kolson (1969) reported in their book on reading and learning disabilities that:

> Since the middle 1950's educators have been developing quality, subject-centered educational programs for the schools. Approximately eighty-five percent of all school children have been able to succeed in these programs. However, about fifteen percent of the children have not been able to master fundamental reading skills and, so, have become educationally retarded in other subjects which are primarily dependent upon reading ability (p. 1).

The children about whom they speak are presumed by the authors to have no obvious intellectual, physical, emotional or environmental handicaps. Yet they fail. The existence of this type of failure to learn has been known for many years. Thompson (1973) reported that as early as 1896 an English physician, W. Pringle Morgan, cited the case of a 14 year-old boy who could not read or spell, but who showed no evidence of brain injury, eye trouble, or low intelligence. Other individual cases have been reported in the intervening years, but they were treated as just that "individual case" until the 1950's when the large number of such failures began to make an impact on educators and parents.

It was noted that other behaviors often accompanied the child's failure to learn. The most striking of these was excessive motor activity. Short attention span, marked distractibility, motor incoordination and impulsivity were also often associated with this failure to

learn. Numerous studies were done, and countless articles written from which came a variety of terms. Among the terms used were "brain-damaged-behavior syndrome," "hyperkinetic syndrome," "Strauss syndrome," "post-encephaletic behavior disorder," "learning disorders," "learning disabilities," "minimal brain dysfunction syndrome" and others. The definitions for these terms are also varied (see Appendix A), but the one offered by Clements (1966) exemplifies the best of them:

> The term 'minimal brain dysfunction syndrome' refers in this paper to children of near average, average, or above average general intelligence with certain learning or behavioral disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system. These deviations may manifest themselves by various combinations of impairment in perception, conceptualization, language, memory, and control of attention, impulse, or motor function.
>
> These aberrations may arise from genetic variations, biochemical irregularities, perinatal brain insults or other illness or injuries sustained during the years which are critical for the development and maturation of the central nervous system, or from unknown causes (pp. 9-10).

Since the names given this syndrome are often used interchangeably, descriptive characteristics found or implied by most definitions should be examined. Burks (1960), Laufer, Denhoff and Solomons (1957), Strauss and Lehtinen (1947) and Wender (1962) were among the many authors who described such a syndrome. All of the definitions have common elements as well as distinctive features. Each definition includes defects in one or more of the following areas: the process of thinking, learning, memory, speech, conceptualization, language, perception, emotional control, attention, neuromuscular or motor control. Discrepancies between measured achievement level and achievement potential in reading, writing and

arithmetic are always included. Some definitions include references to etiological correlates (Clements, 1966; and Strauss and Lehtinen, 1947) and some definitions specifically include statements about excluded states. For example, the author(s) may explicitly state the underlying assumption that the child has no obvious intellectual, physical, emotional, environmental or educational handicap which could account for the failure to learn (Kirk, 1962; Bateman, 1965; and Clements, 1966).

Not all authors agree that the various terms can be used as if they are equivalent. Safer and Allen (1976) argued that the interchangeable use of terms like hyperactivity (HA), minimal brain dysfunction (MBD), and learning disabilities (LD) is inappropriate. They said that although each of the core behaviors, hyperactivity, brain dysfunction and learning difficulties is often associated with the other, each syndrome can occur without the others. For example, MBD is generally based upon learning and perceptual disorders, and it is usually associated with hyperactive behavior. Hyperactivity is not always associated with learning deficits, although the association is very common. In fact, several authors, including Luria (1966) and Wender (1971) have reported that some MBD children are hypoactive but these children are rarely brought to the attention of psychologists and physicians as MBD children. Because of this failure to identify hypoactivity children as MBD children, most authors in the field disagree with Safer and Allen and continue to include all three syndromes, HA, MBD and LD, within the definition of Learning Disability-Minimal Brain Dysfunction (LD-MBD).

Symptomology of LD-MBD

Clements and Peters (1962) described a typical symptom cluster for the LD child. They listed: (1) specific learning deficits; (2) impulsivity; (3) emotional lability; (4) short attention span and marked distractibility; (5) hyperkinesis or hyperactivity; (6) equivocal or soft neurological signs; (7) perceptual motor deficits; (8) general coordination deficits; and (9) borderline abnormalities in EEG records. A regrouping of these characteristics under learning disabilities indicators, hyperactivity indicators, and minimal brain dysfunction indicators will make the discussion of the symptom cluster more comprehensible.

The learning disabilities indicators correspond to Clements' and Peters' symptom cluster of specific learning deficits. This term is best defined as achievement in one or more of the major academic subjects, spelling, reading and arithmetic, which is markedly below the expectations for the child based on his age, intellectual level and grade placement. There is a clear discrepancy between the child's mental and/or chronological age and his academic achievement. As a rule, educators require a 10% or two year (whichever is less) deficit for a child to be considered educationally retarded (Weiss, Kruger, Danielson and Elman, 1974; and Kaluger and Kolson, 1969).

In establishing the nature of the learning difficulties the educationally retarded child may have, most school psychologists appraise the three areas of information processing: receptive, integrative and expressive. The receptive information processing refers to the child's ability to grasp sensory details; it is the intake of information. Integrative information processing refers

to the organization of the input into understandable units. Expressive information processing is the expression of information, or the appropriate response to the information, or the appropriate response to the information perceived and processed. Defects in these areas are referred to as perceptual-cognitive disorders. The ITPA (Illinois Test of Psycholinguistic Abilities) is commonly used to measure perceptual-cognitive skills for LD-MBD diagnosis in most schools. This test is used because the majority of children with marked academic deficits are believed to have perceptual-cognitive deficits (Myklebust and Boshes, 1969; and Kass, 1966).

As a rule, LD-MBD children experience a qualitative as well as a quantitative difference in learning. The LD-MBD child is not only slower to learn what other children of equal age, experience and ability learn, but his learning is qualitatively different from the normal child (Campbell, Douglas and Morgenstern, 1971). Generally, these children can deal more successfully with concrete tasks than with abstractions. They have trouble learning and applying general principles. They often need to count on their fingers in order to perform arithmetic operations, and even with this aid they do poorly when using paper and pencil to add or subtract. They may be able to repeat multiplication tables by rote, but they are unable to competently perform multiplication or division problems. Another example of their failure to understand abstractions is their difficulty with phonics. Most LD-MBD children can name the letters of the alphabet, but these children have great difficulty in remembering how to pronounce letters correctly. They also fail to grasp the rules for spelling, and their spelling, which is generally quite poor, is often

an idiosyncratic "spelling by ear." For the LB-MBD child, general rules when learned are treated as isolated facts and are not transferred to related problem solving situations (Safer and Allen, 1976).

The second major grouping of deficits which characterize the LB-MBD child consists of Clements' and Peters' symptom cluster of impulsivity, emotional lability, short attention span or marked distractibiity and hyperkinesis or hyperactivity indicators. The term impulsivity refers to the inability or failure to inhibit acting even when the action is inappropriate or disruptive to the child and others. The stimulus for the action may be internal, that is, some wish, thought, or appetite which is aroused, or it may be external, something seen or heard which impels the child to activity. The impulsivity of LD-MBD children often brings them into conflict with their families. Their behavior often leads to punishment because of destruction of property, or fights with siblings, or disobedience to parents. Their actions are taken without apparent regard for the other person's reaction to their behavior (Safer & Allen, 1976). In school their impulsive answers are more often wrong or silly than right or appropriate. Their teachers are angered by their apparent lack of effort and their peers laugh at them because they are aware that the teachers are angry with the LD-MBD children and at their seeming foolishness (Ross & Ross, 1976). The older LD-MBD child may get into trouble with law enforcement agencies because they are easily led into impulsive acts, and because they may lack the ability to plan or look ahead to the consequences before they act (Huessy, Metoyer & Townsend, 1974).

The LD-MBD child's emotions are labile, that is, they are variable and extreme. The child may appear happy and cheerful one minute

and exhibit a temper tantrum or tears the next. Happy moods often have a false or brittle quality to them and they may change from moment to moment. The unpredictable behavior and moods of these children further interfere in their social relationships with adults and peers alike (Ross, 1961).

The inability to resist distractions and the short attention span are, of course, closely related to the impulsivity and unpredictable behavior of these children. In fact, this defect in attending ability may underlie the impulsivity, hyperactivity and learning failure (Rourke, 1975; Hernandez-Peon, 1975; Magoun, 1963; and others).

The hyperkinesis, mentioned above, is the most easily seen and the most compelling characteristic of the LD-MBD child, and hyperkinesis is usually noted by others before any other problem, including the learning problem, comes to the surface. The term hyperkinesis refers to a disruptive activity level. The hyperkinetic child is one who has been described as appearing to be unable to remain at rest for any period of time. The child is said to be driven to constant motion, going from one activity to another, fidgeting, fumbling, or playing with various objects at inappropriate times (Ross & Ross, 1976). The above definition is very subjective, and the subjective nature of the definition of hyperkinesis is one of the major difficulties that researchers have encountered in studying this area. As Kaspar (1974) pointed out, the concept of hyperkinesis is based upon the child's presumed inability to control his interactions or exchanges with his environment due to a biological defect. A presumed biological deficiency has been converted into a socially

measured one. Thus, hyperkinesis, the product of some unknown internal defect, is determined externally to the child, usually by adults in control of the child who find the child's behavior unacceptable. Kaspar cautions that the history of the adults concerned, as well as the actual behavior of the child, may be very important in the determination of the existence of this condition.

The research literature on HA shows the confusion which can result when there is no objective definition of an experimental variable which is agreed upon by the investigators in the field. Attempts to measure the difference in activity level of "normal" and "hyperactive" children have been many and varied in the techniques employed and in the results. Lapouse and Monk (1958) carried out an epidemiological study of 6 to 12 year-old children and found that 50% of their mothers thought that their boys were hyperactive, although the children studied were chosen from the general population. Kenny et al. (1971) did a study to ascertain the reliability of the diagnosis of HA in a large number of children referred to them. They found that trained observers agreed with the diagnosis in only 13% of the cases. McConnell et al. (1964) studied the agreement between the ratings of HA made by nurses observing the children and an objective measure of activity. They found very poor agreement between these two independent measures.

Werry and Sprague (1970) and Martin and Powers (1967) agreed with Kaspar's conclusion that the assessment of hyperkinesis is a very unreliable activity. These authors enumerated some of the factors which influence the perception of hyperkinesis. The factors were frequent goal changes, meaningless activity, dependency demands and the social nuisance value of the behavior.

For many researchers the solution to the problem of definition of hyperkinesis appeared to be to objectify it by using quantifiable measures of activity. Numerous devices were invented and used. Checklists of motor activity (Werry and Sprague, 1970; and Shaffer and Pincus, 1974), sensitized platforms or stabilmeters (Shaffer and Pincus, 1974, and Sprague and Toppe, 1966), pedometers (Bell et al., 1971) and accelerometers (Schulman and Reisman, 1959), among others, have been used with varying success. (See the following literature review of etiology and drug treatment for a more detailed review of this literature.) Despite the difficulties of reliable assessment, few children are referred for psychological or physiological help for learning problems without the mention of the hyperkinetic component of the HA syndrome.

The third major grouping of indicators of LD-MBD consists of a cluster of symptoms often considered MBD indicators. These are soft neurological signs and abnormalities in EEG records. Clumsiness is often subsumed under soft neurological signs and will be so treated here. These indicators correspond to Clements' and Peters' symptom cluster of equivocal or soft neurological signs, perceptual motor deficits, general coordination deficits and borderline abnormalities in EEG records.

Werry (1968) reported that LD-MBD children showed more minor or soft neurological signs than the average child, while the number of classical or hard signs is approximately the same for the LD-MBD children and normal children. Soft signs are slight variations which are not always present and are not positively associated with documented lesions in the brain, while the so-called hard signs of

paralysis, anesthesia, paresis and changes in reflexes are associated with specific brain lesions.

The exact number and definition of soft neurological signs vary somewhat from author to author, but they tend to include the following: clumsiness, poor balance, mixed dominance, athetoid or choreiform movements, perceptual-motor difficulties, fine coordination difficulties, transient strabismus, dysdiadochokinesis or hand-finger dyskinesia, astereognosis, graphesthesia, and finger localization difficulties. The terms used for the soft neurological signs are a combination of everyday language and neurological terminology; therefore, the less well-known terms may need definition. Athetoid or choreiform movements are stereotyped, uncontrolled, jerky movements of the arms and are often seen in individuals with documented damage to the subcortical structures of the brain. Athetoid movements may also appear in individuals without documented brain damage, however. The perceptual motor difficulties referred to above are estimated from an evaluation of the handwriting, Bender-Gestalt design performance, or performance on the Coding (Digit Symbol) subtest of the WISC. Transient strabismus is the uncontrollable pulling of the eyes to one side or the other under specific circumstances. Difficulties in controlling the degree of convergence of the eyes is also noted here. Dysdiadochokinesis or hand-finger dyskinesia is the difficulty in rapid, smooth oscillation of the finger or hand. Astereognosis is a defect in the ability to recognize objects from touch alone. Graphesthesia is the difficulty in recognizing numbers, letters, or other common symbols which are written on the skin. Finger localization is the ability to correctly name the finger touched when visual clues are excluded. Difficulty

in this area is called finger agnosia. Finger agnosia, astereognosis, and graphesthesia are considered to be present only when there is no primary loss of the sense of touch in the areas stimulated. When soft signs are found in the absence of the classical neurological signs, they are considered by LD-MBD theorists to be due to very mild brain damage or dysfunction. Soft neurological signs are an important theoretical correlate to establishing minimal brain damage or dysfunction as the legitimate source of the learning disability and hyperactive behavior exhibited by LD-MBD children.

Clumsiness has been found to be associated with poor school performance in the following studies: Denhoff and Sequeland (1968), Gubbay (1965), Rubin and Bakwin (1968), and Illingsworth (1963). Laufer et al.(1957) found that 75% of the MBD children that he studied were clumsy, inept and uncoordinated. As a group these children performed poorly on the Bender-Gestalt, auditory synthesis, transferring information from one sensory modality to another, orienting themselves in space, right-left orientation, and they showed reversals in reading and writing.

Not all the studies in the literature gave such strong support of the association of soft neurological signs with learning difficulties. In fact, one of the most long-held beliefs on this association has been challenged. Mixed laterality or mixed eye-hand preference has long been associated with reading difficulties (Orton, 1937, 1943). In his 1937 book on reading, writing, and speech problems, Orton developed a theory to explain the association he had postulated. He hypothesized that the reading deficits were the result of strephosymbolia or "twisted symbols" which resulted in the

reversal of letters and words. The strephosymbolia was hypothesized to be the result of a lack of complete dominance of either of the hemispheres of the brain. Both the hypothesized lack of dominance of one of the hemispheres over the other and the association between mixed eye-hand dominance with dyslexia have been challenged. Wada (1949) used the amytal test to determine the dominant hemisphere in a group of subjects. He found that the dominant hand was not necessarily contralateral to the dominant cerebral hemisphere. Cross dominance was found for 99% of the right-handed subjects studied; however, 67% of the left-handers had ipsilateral dominance, that is, their left hemisphere was also their dominant hemisphere. This study indicated that the left hemisphere is the dominant hemisphere for the majority of people whether they are predominantly right or left-handed. Orton's assertion that mixed eye-hand dominance is associated with dyslexia due to a lack of cerebral dominance was not supported by this research data. Many authors, among them Flescher (1962), Hoveston (1970), Towen (1972), Spritzer (1959), Witty and Kopel (1965) and Belmont and Birch (1965), also failed to find the relationship between mixed eye-hand dominance and reading disability which Orton had postulated.

Orton's (1937, 1943) belief that there was an association of cerebral dominance problems and reading problems has been supported by others, however. Ingram (1975) found that a high proportion of children with known central nervous system (CNS) involvement showed weak lateralization of eye, hand, and foot. Further, Ingram and Reid (1956) found that only 29% of the school children they studied who had reading difficulties were either firmly right or left-handed.

Eighteen percent were classified as definitely ambidextrous, and the remaining 53% of the children had not established a consistent reliance on one hand over the other, although they did not use the hands equally.

Goldstein (personal communication) may have provided some clarification of this issue. He reported, on the basis of long clinical experience, that eye-hand differences in dominance were not necessarily indicative of brain impairment; however, hand-foot differences are generally considered to be diagnostic of early brain damage. Thus, eye-hand mixed dominance may be asoociated with mixed dominance of hand-foot; this relationship could be considered a sign of early brain damage, according to Goldstein. Mixed eye-hand dominance in the presence of hand-foot lateralization to the same side would not be an indicator of early brain damage. Eye-hand mixed dominance is much more frequent than hand-foot mixed dominance, and the former may be produced by a number of conditions other than damage to one of the cerebral hemispheres. The use of eye-hand mixed dominance as an index of brain damage gives a spuriously high estimate of the incidence of brain damage. Thus far, the research data indicates that there is an association between mixed dominance and reading difficulties, although it is not the exact association hypothesized by Orton (1937, 1943).

Choreiform movements are another set of soft signs which are reported to be associated with learning difficulties in school (Prechtl, 1962, and Rutter and Birch, 1966). The lack of knowledge of the frequency with which choreiform movements occur in the unselected school population weakens their results. Wolff and

Hurwitz (1974) attempted to provide that information by studying the relationship of choreiform movements and behavior disturbances in a classroom of presumably normal children. Wolff and Hurwitz define choreiform movements as small jerky movements of short duration that occur irregularly and without regular rhythm. They described their method of observation which had not been done by the previous researchers and they took their children from a large sample of 1,300 children between 10 and 12 years of age. From this sample, they found 103 boys and 25 girls with choreiform movements, or approximately 8% of this population of "normal" children. The authors selected a control group of an equal number of children without choreiform movements from the remainder of the 1,300 children. Teachers' rating of behavior problems and academic achievement in reading and spelling were correlated with the presence or absence of choreiform movements. Boys with choreiform movements were found to have significantly lower academic achievement in the areas measured. However, no difference was found in the academic achievement of the girls. Choreiform movements were found to be positively correlated with clumsiness, distractibility, emotional lability, and for the boys, lower academic performance. None of these children had been diagnosed as LD-MBD, but they did show many of the signs associated with this syndrome. The Wolff and Hurwitz study found that approximately 8% of a large group of presumably normal children had not only choreiform movements but other components of the LD-MBD syndrome as well.

Numerous authors, aware of the problem caused by the lack of knowledge of the frequency of soft neurological signs in both the LD-MBD and the normal population of children, have attempted to

establish normative data with conflicting results. Meier (1971), in a study which examined 3,000 children from eight Rocky Mountain states, found that 90% of the children diagnosed as LD exhibited one or more soft signs, while 75% of the normal children showed one or more of these signs. Kennard (1966) studied a group of hospitalized "organic" patients and another group of hospitalized control subjects to determine the frequency of soft neurological signs. He found that 100% of his "organic" patients and 70% of the control subjects had at least one soft neurological sign. The similarity between the groups was not as great as it would at first seem because the average number of soft signs was five for the experimental group and the average number for the control group was one. Wikler, Dixon and Parker (1970) found similar results with non-hospitalized children. The authors found that 22 of the 24 MBD children showed one or more soft neurological signs and that 20 of the 24 matched control children also showed one or more signs. The total number of soft signs was three times as great for the MBD children. Lerer and Lerer (1976) reported that the majority of the children referred to their clinic because of learning problems also showed distractibility, short attention span, and soft neurological signs (40 out of 57). In contrast, Kenney et al. (1971) found that only 46% of 100 children referred for hyperactivity and learning problems showed any soft neurological signs.

The conflicting data from the above studies, as well as the high frequency of soft neurological signs in the general population found by so many authors, led Adams, Kocsis and Estes (1974) to attempt to establish the frequency of a number of soft signs in learning

disabled children, borderline learning disabled, and normal children. The authors were unable to differentiate between the three groups on the basis of the frequency of soft neurological signs alone.

The frequency of soft signs is known to decrease with age; therefore, Benton (1959) studied 158 normal children between the ages of 5½ and 9½ years to establish the normal frequency of difficulty in finger localization. He found that this ability did not mature until late childhood. From this finding, he cautioned that poor performance in finger localization does not necessarily indicate a pathological condition. Poor finger localization may be indicative of cerebral dysfunction, however, if it persists past the age of 9½ years. Grant et al. (1973) studied the frequency of dysdiadochokinesis. They found that the rapidity and consistency with which a child could oscillate the index finger is not well developed until 9 or 10 years of age. The above studies indicate that at our present stage of knowledge it is unwise to diagnose MBD on the presence of one or two soft signs alone. Thus, a single soft sign should never be used to establish the diagnosis, but the presence of one or more of these soft signs often adds credence to the diagnosis of LD-MBD when these signs are found in association with the other characteristics of the syndrome.

The EEG record is not generally used in the diagnosis of the LD-MBD child; however, EEG abnormalities are hypothesized to be present in a large number of these children. The presence or absence of EEG abnormalities is considered to be theoretically important to many authors (Clements, 1966). The EEG is a diagnostic tool which is known to be sensitive to the effects of even minor head injuries.

In addition, the EEG record can reveal alterations in the electrical activity of the brain even when no demonstrable structural lesions in the brain can be found. This knowledge encouraged many authors to look for EEG abnormalities in the records of LD-MBD children. Kahn and Cohen (1934) were the first to report EEG abnormalities in a syndrome which they called "organic driveness" and which they defined behaviorally as a hyperkinetic, impulsive behavior pattern. Since this early study, numerous workers have attempted to verify Kahn's and Cohen's findings and to establish the specific pattern or patterns of EEG abnormalities which would correlate with LD-MBD.

Since the brain dysfunction felt to be associated with learning problems was thought to be minimal or borderline, the abnormalities in the EEG records were expected to be of a borderline variety. There are a number of abnormalities reported by authors studying EEG tracings of the LD-MBD child. Among the most common EEG tracings in LD-MBD children are slow occipital waves or paroxysmal slow bursts (Cohn, 1961), diffuse slowing or immaturity (Predescu et al., 1968) and spike foci with 14 and 6 per second positive spike and wave activity (Kellaway, Crawley and Maulsby, 1965). The research studies in the area have resulted in a mass of conflicting and confusing data. A few of the reasons for this confusion are the lack of a consistent criterion for the definition of an abnormal or borderline abnormality in EEG records, various types of stimulation used during the recording of the EEG, and the variation in the state of the subject during the EEG recording (i.e., sleep vs. awake).

The first effort of the researchers was to establish the relationship between LD-MBD and EEG abnormalities. Knobel, Walman and Mason (1959) attempted to find a correlation between behavioral

ratings, psychological tests and EEG abnormalities in a group of HA children. The authors found that 62.5% of the children had EEG records which were judged to be abnormal. Most of the EEG abnormalities were defined as mildly abnormal (not further defined). No significant correlation was found between the psychological testing and the EEG records. HA was found to be least frequent in the children with the severely abnormal records and most common in the normal and mildly abnormal EEG records. Werry found in a 1968 study that 52% of the HA children he studied showed a slow-diffuse dysrhythmia on their EEG records during hyperventilation. Paine (1962) reported that 15 of the 17 EEG records obtained from HA children seen in his extensive private practice were abnormal. The high frequency of abnormalities in Paine's study was due to several methodological irregularities. All EEG's were obtained because of suspected cerebral dysfunction. Three of the children had known seizure disorders and showed the typical spike wave pattern characteristic of that disorder, and no effort was made to obtain EEG records from children who were not suspected of having cerebral disease. Prechtl and Stemmer (1962) found that only 14% of the 50 HA children in their study produced abnormal EEG records; three conditions of stimulation were used during the recordings. All of the abnormal EEG records were of the spike wave pattern, which is characteristic of petit mal seizures. None of the children had had any known seizures.

The methodological errors of the earlier studies reported above were corrected and some understandable relationships between EEG records and some behavioral aspects of the LD-MBD syndrome were reported during the later sixties and the seventies. Demerdash,

Eeg-Olofssor and Petersen (1968) studied the EEG records of almost 500 children with no known neurological, behavioral or learning problems to establish the frequency of the 14 and 6 per second spike wave pattern (petit mal epilepsy pattern) in normal children. The authors found that approximately 14% of the normal children studied had EEG records which contained this pattern. They also found the frequency of this spike wave pattern to be age dependent. The frequency increased between the ages of ten to fourteen, and then decreased in frequency after fourteen years of age.

Two types of EEG abnormalities were found to be most common in the LD-MBD children, the spike wave pattern reported above and the slow wave activity. Wikler, Dixon and Parker (1970) compared the EEG records of two groups of children with behavior problems. They found that slow wave activity and abnormal transient discharges were associated with hyperactive behavior, perceptual motor deficits and a high incidence of soft neurological signs. There was no age dependent change in EEG records for this group. The second identifiable group was also found to have excessive slow wave activity, but without the abnormal transient discharges. This pattern was found in the children who were in the non-hyperactive behavior problem group. Perceptual motor deficits and soft neurological signs were also present in this group, but slow wave activity was found to decrease with increasing age in this group. Stevens, Sachdev, and Milstein (1968) also found that EEG slowing was associated with hyperactive behavior and that the spike wave pattern on the EEG record was associated with finger agnosia, and disturbance in attention, time sense, and ideation. Stevens et al. did not consider the presence or absence of EEG abnormalities to be of predictive clinical value or an

etiological factor. Satterfield, Cantwell, Lesser, and Podosen (1972) reported on an EEG study in which a normal control group was compared with a hyperactive group of children. They found, as did Windler et al., that slow wave activity was significantly associated with hyperactivity. Satterfield, Cantwell, Saul and Yusin (1974) examined 120 hyperactive children extensively. They found that 52% had normal EEG readings during hyperventilation. Borderline abnormalities, defined as excessive slow wave activity, were seen in 29%, and definitely abnormal EEG records, defined as spike wave patterns or excessive slow wave activity plus other abnormalities, were found in 18% of the children. As in the Knobel et al. (1959) study, the children with the most abnormal EEG records were considered to be the least hyperactive. Klonoff and Low (1974) obtained EEG records from MBD children, normal controls, and children with known acute lesions of the cerebral hemispheres. The age range was 2 years, 8 months to 15 years, 11 months. The percentage of abnormal EEG tracings in the MBD children decreased with age. Diffuse slowing was the most common abnormality for both the children over 9 years and the children under 9 years of age, especially at the younger ages. The spike wave pattern was the second most common type observed for the older (9 to 15 year-old) child, and the frequency of this pattern increased with age. For the younger children (2 to 9 years) asymmetries and the spike wave pattern were the second and third most common EEG abnormality.

The above studies on the EEG records of LD-MBD children fail to firmly establish the association between EEG abnormalities and the LD-MBD syndrome. The same studies also fail to disprove the hypothesized association. EEG abnormalities, like soft neurological signs,

remain hypothesized but unproven components of the LD-MBD syndrome and taken alone they prove nothing, but in combinations with other aspects of the syndrome they add diagnostic credence.

Prevalence of LD-MBD

The prevalence of the LD-MBD syndrome has not been well established. Meier (1971) reported that 15% of the 3,000 second grade children he examined in eight Rocky Mountain states were showing disabilities in learning marked enough to be so classified. Werry (1968) places the occurrence of hyperactivity and associated learning problems at between four and ten percent by the second grade level. Yanow (1970) reported the incidence of such children to be between 15% and 20% for all elementary school children. Wender (1971) stated that the minimal brain damage syndrome (MBD) is very common in children referred to child guidance centers. He stated that MBD was so common that one would not go far wrong in diagnosing MBD from knowing that a child had been referred to such a center and also has had learning problems. Wender appears over-generous in his designation of the MBD child. He stated (1971, p. 69), "the use of the psychiatric interview of a child can be extremely useful in one subcategory of MBD children, the borderline schizophrenic child." From this statement, we can assume that Wender places all children with any type of learning problem in the MBD diagnostic classification. This is unlike most investigators in the field who use a more conservative definition. Prechtl and Stemmer (1962) reported that the "choreiform syndrome," their term for LD-MBD, is present together with "behavioral problems" in 20% of the school age boys in the Netherlands.

Five percent of these boys were reported to show severe manifestations of the syndrome. Prechtl and Stemmer reported that the frequency in girls was about half as common (or 10%) as in boys and less than 1% of the girls were considered to show the severe form of the syndrome. Prechtl and Stemmer also reported that 90% of the children so diagnosed had more or less severe reading difficulties. Stewart et al. (1966) reported that the presence of the "hyperactivity syndrome" is approximately 4% in the population of St. Louis grade school children between 5 and 11 years of age. Huessy (1967) placed the incidence of "hyperkinesis" at 10% in an unselected population of school children. Wender (1971) reported a study done in Montgomery County, Maryland, in which 20% of the elementary school population exhibited MBD signs to some degree. In that study, teacher ratings were used to identify the frequency of the presence of children with abnormal activity levels which were in the hyperactive direction. These ratings relied on the teacher's subjectively held expectations of normal activity level for a child in a classroom.

The research reported in the literature consistently found a higher incidence of LD-MBD in male children than in female children. The ratio ranges widely, from 2 to 1 (Prechtl & Stemmer, 1962) to a high of 9 to 1 (Werry, 1968). The reason for this difference in incidence between the sexes has not been studied specifically. Rubin et al. (1972) reported a study in which he offered one explanation. He found that even when academic performance was held constant, boys were much more frequently referred for learning problems. From this finding the authors hypothesized that teachers had less tolerance for boys with classroom difficulties than girls with similar problems.

The teachers' intolerance of the failure of male children, rather than differences in behavior between males and females, may be responsible for the higher incidence of LD-MBD diagnosis among boys.

Studies from the literature, such as the above, report an incidence of LD-MBD children that ranges from 4% to 20% of the school population. The studies in which the higher figures were reported (15% to 20%) appeared to define the syndrome too loosely and to include any child of average intelligence with hyperactive behavior who also had a level of academic functioning which was substantially below grade level in some area. This overly broad definition of the syndrome seems to go far beyond the concept of a minimal brain dysfunction etiology of LD-MBD, and such a definition would make it very difficult to establish specificity. It is obvious that the syndrome, however narrowly or broadly defined, includes a substantial number of children in the school age population and thus the problem has wide social significance.

Etiology of LD-MBD

The etiology of the LD-MBD syndrome has not been definitely established. The earliest hypothesized cause of learning disabilities (LD) and the associated behavior disorders was organic brain damage. Kahn and Cohen (1934) published an article describing the behavior of a number of children who had recovered from von Economo's encephalitis. The similarity between the behavior of the post-encephalitic children and the symptom complex which was associated with learning disabilities was highlighted as educators and other professionals began to try to develop a theory to explain LD-MBD. Thompson (1947)

described the symptom pattern of 30 post-encephalitic children and emphasized the parallel between the behavior of these children and that of the typical LD child. His data and observations lent support to structural brain damage as etiologically significant in the LD-MBD syndrome. Strauss and Lehtinen (1947) also emphasized the similarity between brain injured children who exhibited hyperactivity, distractibility, impulsivity, low frustration tolerance, and many children experiencing learning problems. The similarity between the behavior patterns of the LD-MBD child and the child with neurologically diagnosed brain damage has led many theorists to assume that the LD-MBD syndrome, although not accompanied by obvious stigmata of outright neurological disease, is the result of a very mild brain damage. Thus, Kirk (1962), Myklebust (1963), Bateman (1965) and Clements (1966), major theorists on LD-MBD, include brain damage or brain dysfunction as the causal factor--or at least one of the causal factors--in LD-MBD.

The evidence for a brain damage etiology of LD-MBD is conflicting. Stewart and Olds (1973) pointed out that less than ten percent of the referrals of children with hyperactive behavior have histories which suggest brain injury. Further, they find no greater frequency of birth complications in hyperactive children than in children of the general population. This finding is in contradiction to an earlier study by Knobloch and Pasamanick (1966) who reported on a large number of studies demonstrating a higher frequency of prematurity, prenatal difficulties and perinatal medical complications and a variety of behavioral and neurological difficulties in LD children than in the normal population. Learning Disabled children

constituted one of the groups studied for whom the above association was found. These apparent contradictions in findings may be due to an artifact of retrospective studies or the contradiction may lie in the definition of the term LD or to variations in the definition of "histories which suggest brain injury."

In an effort to circumvent these problems, Rubin, Rosenblatt, and Balow (1972) conducted a longitudinal study of 241 infants. Their aim was to evaluate the psychological and educational sequelas of prematurity, which has been reported to be associated with neurological and behavioral problems in the later life of children (Benton, 1940; Wiener, 1962; Harper and Wiener, 1965; and Caputo and Mandell, 1970). Rubin et al. (1972) restricted their sample to an almost exclusively Caucasian subject population (96.5%) whose socioeconomic status was typically distributed for the population of the North Central states. Prematurity was defined by birth weight and gestation period. Repeated neurological, achievement and behavioral measures were taken on these children from birth through age seven. The authors found that low birth weight was associated with impaired school progress, and increased behavioral and neurological problems. These deficits associated with low birth weight were much more pronounced in full term gestation children. Low birth weight boys had a higher proportion of school-identified educational problems than did girls of similar birth weight. Academic achievement, however, was found to be the same for both sexes. The Rubin et al. study, which helped to establish more definitive measures of prematurity and thus reduced a source of variability, has made a step toward clarifying possible etiological sources of LD-MBD. Nevertheless, Werry's (1968)

conclusion that there is no firm data to support the brain injury etiology of learning disabilities is generally still accepted by most investigators in the field.

Delayed maturation of the cerebral hemispheres has been postulated as an alternative to brain damage as the cause of LD-MBD. Thompson (1973) changed his view from one of a brain damage etiology, which he had earlier espoused, to the theory of delayed maturation. Loretta Bender (1957) also hypothesized the existence of such a delay, as did Werry (1969). Some experimental studies have been done which offer support for this view. Butter and Lapierre (1947) compared the performance of normal and hyperactive 6 to 12 year-old children on the Illinois Test of Psycholinguistic Abilities and found that the hyperactive group was from 18 to 24 months less mature on a number of the developmental measures than the control group. Peters, Romine and Dykman (1975) found that the magnitude of differences in performance on tests thought to measure central nervous system functioning of LD and normal children decreased with increasing age. Oettinger, Majovski, Limbeck, and Gauch (1974) measured the bone age of MBD children. They found that two-thirds of the subjects had bone calcification and densities which fell below the average for their age. Czudner and Rourke (1970 & 1972) found that younger brain-damaged children showed deficits in visual reaction time when compared to young normal children, but that older brain-damaged children did not differ from their normal age mates in visual reaction time. The same relationship was also found in a study of auditory reaction time (Rourke and Czudner, 1972). Shaffer, McNamara and Pincus (1974), in a study which compared the performance of brain-injured and non-brain

injured subjects on activity, attention and impulsivity while controlling for the presence of conduct disorders, found an inverse relationship between Mental Age, or IQ, and Chronological Age with almost all measures of hyperactivity, inattention and impulsivity. Thus, Shaffer et al. found that as mental age, intelligence or chronological age increased, hyperactivity, inattention and impulsivity decreased. These studies add support for a maturational lag theory of LD.

The evidence to support the maturational lag theory does not rule out the possibility of a brain dysfunction; such evidence only makes that brain dysfunction a time limited one. The dysfunction may represent a normal stage for the individual or it may be the result of some type of organic insult. A type of normal delay may be seen in the studies on hereditary hyperactivity and learning problems. Morrison and Stewart (1971) found a trend toward an association of hyperactivity in parents and their children. These investigators interviewed the parents of 59 hyperactive children and 41 non-hyperactive control children for evidence of parental hyperactivity and hyperactivity in second-degree relatives (aunts, uncles, or cousins) during the childhood of these relatives. Twenty percent of the parents of hyperactive children and only five percent of the parents of control subjects reported childhood hyperactivity ($p < .01$). When the frequency of hyperactivity in the childhood of the second-degree relatives was combined with the data for the parents, the difference between the two groups was significant at the .001 level. Cantwell (1972) found a similar relationship in his study of intergenerational hyperactivity. The above studies, which showed a <u>possible</u> genetic

role in hyperactive behavior, call for similar investigations into the role of heredity on other aspects of the LD-MBD syndrome.

Safer (1973) studied the siblings and half-siblings of MBD children to further assess the possibility of genetic factors. The sample consisted of 19 full siblings and 22 half-siblings of 17 children diagnosed as MBD. All of these children had been separated and assigned to foster homes at an early age, thus minimizing the influence of living in the home with an MBD child. There was a significantly higher incidence of signs of MBD in the full siblings than in the half-siblings. Only two of the 22 half-siblings were diagnosed MBD, but ten of the 19 full siblings had the MBD diagnosis as well.

Since monozygotic twins are identical genetically, differences in their development are assumed to be due to environmental factors, and fraternal dyzygotic twins can develop differently due to both genetic and environmental factors. Matheny, Dolan and Wilson (1976) used this twin method to study learning difficulties. They reported their findings on 46 twins with learning difficulties who were part of the Louisville Twin Study. The Louisville Twin Study was a longitudinal investigation of all twins born in the Louisville, Kentucky area from the late 1960's through the time of their report. In 1976, approximately 440 children had been followed from their birth through their early school years. The 46 learning disabled twins in the Matheny et al. study were taken from 31 pairs of twins in that study, and the control subjects were matched twins taken from the remaining non-learning disabled population of twins in the Louisville study. Two important environmental factors were found in

the comparison of the birth and developmental records of the two groups; breech presentations were significantly more common in the experimental (LD) than in the control group (48% vs. 28%), and although the two groups did not differ in birth weight, the experimental subjects failed to gain weight as rapidly during the first 24 months as the control subjects. Behavioral differences were also found. The experimental subjects were found to be more active, more prone to temper tantrums, to have more sleep disturbances and to have more speech articulation difficulties. The IQ's for the experimental subjects were also consistently lower than that of the control group. At the time of the report, the Full Scale IQ on the Wechsler Intelligence Scale for Children (WISC) averaged 102 for the control subjects and 94 for the experimental subjects. Matheny et al. also compared the data for identical and fraternal twins across groups. They found that the correlation of learning difficulties or lack of learning difficulties in a twin pair was significant at the .001 level for identical twins, but only at the .01 level for same sex fraternal twins. The authors felt that their study supported the hereditary etiology of LD. None of these studies gave definitive proof that there is an hereditary factor in the syndrome, but they do lend support to a genetic hypothesis.

A third theory of LD-MBD etiology, and one which is not necessarily incompatible with either the hereditary or the brain damage/dysfunction theory of LD-MBD etiology, is the physiological or biochemical theory. One of the earliest of these theories was proposed by Laufer, Denhoff and Solomon (1957). Laufer et al. monitored the EEG recordings of a group of hyperkinetic behavior disordered

children and those of a control group of non-hyperkinetic children during stroboscopic stimulation. The authors were attempting to determine the minimum amount of metrazol necessary to produce the EEG spike wave burst and associated myoclonic jerking of the forearm in each group of children. One-third of the hyperkinetic group of children had an unequivocal history of brain damage, but both they and the non-brain damaged hyperactive children required a lesser amount of metrazol to produce the myoclonic jerking than the control group of non-hyperactive children. From these findings the authors hypothesized that the HA syndrome was due to a dysfunction in the CNS. This dysfunction Laufer et al. localized in the diencephalon.

Wender (1969, 1971) agreed that there is a neurophysical origin in MBD. He formulated his theory on the basis that amphetamines and other stimulant drugs improve the behavior of MBD children by reducing their activity level (Bradley, 1937, 1950; Burks, 1964; Conrad and Insel, 1967; Eisenberg, 1966; and Zrull, Westman, Arthur, and Bell, 1963). Wender (1971) was also impressed by the effects of the stimulant drugs on "complex psychological functioning." He stated:

> ...One aspect of the drug action of certain stimulant drugs (amphetamine, methylphenidate) on MBD children is unique: they produce immediate psychological growth; while the drug is active children may demonstrate age-appropriate psychological functioning which they have never attained previously.....these children are, so to speak, psychologically retarded and the unique effect of these stimulants is to produce temporary psychological maturation (p. 163).

Wender stated that an examination of the data on stimulant therapy of MBD suggested a biochemical abnormality and a possible neurophysiological location of the biochemical "lesion." Wender hypothesized (1) that MBD children had a defect in their ability to metabolize one

of the monoamines, and (2) that the suspected biochemical abnormalities affect behavior by impairing either the reward mechanism of the brain or the activating system of the brain.

The monoamines are believed to be the major neurotransmitters of the central nervous system. The three major neurotransmitters are serotonin (5-HT), norephinephrine (NE), and dopamine (DA). The biochemical action of amphetamines and other stimulants is thought to be related to the action of NE and DA (Weiss et al., 1968; McRene and McCartney, 1961; and Moore, 1963). Amphetamines are known to stimulate the release of newly synthesized NE and DA at the presynaptic terminals and to block re-uptake of these transmitters (Kornetsky, 1967). Boakes, Bradley, and Candy (1972) performed a study which may help to clarify the mechanism of action of amphetamines and other stimulants. They studied the effects of d-amphetamine on single neurons of the brain stem of rats. They found that the amphetamine mimicked the excitatory and inhibitory action of NE. Thus, d-amphetamine acts directly upon the NE receptors. The stimulants also act on the presynaptic terminals to potentiate the release of NE and DA. In addition, stimulant drugs are known to block the re-uptake of the transmitter substances. Lesions of the lateral hypothalamus are associated with a decrease in 5-HT and NE in the whole brain stem and telencephalon (Heller and Moore, 1965). Coleman (1973) and Greenberg and Coleman (1973) found that 5-HT blood and platelet levels were low in a wide variety of childhood diagnostic categories including HA and LD children. The above supports the involvement of the monoamines in the regulation of LD-MBD behavior, although not necessarily in the mechanism of positive reinforcement.

Most theorists who have looked to the subcortical structures of the brain for the location of the basic dysfunction in the brain of the LD-MBD child have looked at the limbic system and the reticular activating system (RAS) for the site of the problem. The earlier theorists on LD-MBD favored the limbic system because of its action in mediating emotional behavior. The limbic system is no longer considered to be the primary focus of LD-MBD mediation; however, because recent research has turned the attention to the RAS since the classic study of Moruzzi and Magoun in 1949, researchers and theorists have become increasingly aware of the RAS and its major importance in mediating wakefulness, arousal and attention.

A brief description of the RAS and its functions may be useful at this point. The RAS consists of a diffuse system or network of fibers and cells in the brain stem, hypothalamus and thalamus. It receives stimuli from the specific afferent systems via collaterals, as well as directly from the spinothalamic and spinocerebellar tracts and various cranial nerves and peripheral nerves. Thus touch, pain, temperature, and other superficial sensibilities, muscle and deep tendon sensations, and afferent impulses from the viscera and other internal structures of the body contribute to the RAS input. There are a multiplicity of receptors in the RAS for the afferent impulses. The RAS appears to receive all afferent sensory impulses and it receives impulses from and transmits impulses to the cerebral cortex, cerebellum, hypothalamus, and neurons at all levels. The areas of the cerebral neocortex which contribute to the RAS include the orbital, oculomotor, sensorimotor, and parietal cortices, parts of the cingulate gyri, and the temporal poles.

The RAS is not a passive recipient of masses of information alone. It has a very wide range of functions. In 1932, W. F. Allen (see Thompson, 1967) theorized that the embryological and anatomical characteristics of the RAS suggested that it was suited to serve the general function of inhibition, excitation and integration of brain activity. It is now known that it can both facilitate and inhibit many areas of activity, including increasing or decreasing muscle tone, respiration, and circulation. It also plays a central role in sleep and arousal from sleep. Moruzzi and Magoun (1949) produced an arousal EEG pattern by the electrical stimulation of the RAS in a lightly anesthetized cat. The type of EEG pattern produced was one which is usually associated with behavioral arousal. Probably the most important functions of the RAS with respect to the problems of learning are those functions pertaining to the sorting and organization of information. The RAS mediates attention, memory and habituation, all of which are of major importance in learning. The RAS is hypothesized to enable the focus of attention by reducing the extraneous sensory information which might reach conscious awareness and thus compete with a specific stimulus (Frederiks, 1969).

The effects of amphetamine upon RAS reactivity and arousal support the hypothesis that the RAS or other closely related subcortical structures are involved in the LD-MBD syndrome. The Laufer, Denhoff and Solomons (1957) study revealed that hyperkinetic children who had received amphetamines required a larger dose of pentylenetetrazol (metrazol) under stroboscopic stimulation to give the pathological myoclonic jerking and EEG spike wave burst than children who had not received the amphetamines. It is unfortunate that Laufer

et al. failed to conduct the same study on non-hyperactive children. Bradley and Key (1958) used a preparation with a transection of the spine at the third cervical vertebrae to study the effects of amphetamines on the EEG response of the RAS under two conditions of stimulation. Direct electrical stimulation of the RAS and auditory stimulation were used. Amphetamines reduced the level of stimulation needed for both the electrical stimulation and the auditory stimulation to product EEG arousal and the associated behavioral arousal. The authors found that the EEG arousal in the auditory cortex was not changed by the administration of the amphetamines. The facts that the cortical evoked response remained unchanged while the RAS showed arousal suggested to Bradley and Key that the site of the action of the amphetamines and other stimulant drugs must be in the RAS rather than in the sensory-cortical pathways or the collaterals to the RAS.

Other supporting data for this theory was found in the failure of amphetamines to cause behavioral or EEG arousal in animals with a transection of the brain at the colliculi (cerveau isole) (Bradley and Elkes, 1957). Killam (1962) reported more data in support of the above theory. He found that amphetamines failed to produce EEG arousal when lesions had destroyed most of the mesencephalic RAS. These experiments suggest that the behavioral and EEG arousal of amphetamines is intimately related to or mediated by some activity of the brain stem RAS.

The RAS is not alone in the mediation of attention, however. There are two types of attention, "sensorial attention" and "ideational or intellectual attention," depending upon whether the attention is elicited by activity outside or inside the brain

(Hernandez-Peon, 1961, 1966). Sensorial attention is thought to be mediated by the RAS directly and ideational or intellectual attention is believed to be mediated by the cortex as well as the RAS. The experiments of Moruzzi and Magoun (1949), Linsley, Bowden and Magoun (1949) and French (1952) used external stimulation to the RAS to experimentally produce the first type of arousal. Experimental support for the ideational or intellectual attention and arousal has been demonstrated in the work of French, Hernandez-Peon, and Livingston (1955), Segundo, Arana-Iniguez and French (1955) and Segundo, Naquet, and Buser (1955). In each of the above studies electrical stimulation of various areas of the cortex in a sleeping or lightly anesthesized animal produced waking or EEG arousal. In the same studies, unrestrained chronically implanted resting animals showed signs of behavioral and EEG arousal when given electrical stimulation to selected cortical areas. These findings suggest that the cortex plays an important part in the second type of attention or arousal. These findings also suggest that a reverberating loop appears to exist between the RAS and the cortex. The frontal lobes are known to play an important role in maintenance of attention of the voluntary type (French et al., 1955). Distractibility is also a very common feature in patients after frontal lobotomies (Greenblatt, 1951). The frontal lobes are not the only cortical areas involved in ideational arousal. Any of the areas of the cortex contributing input to the RAS, the orbital, oculomotor, sensorimotor, parietal cortices, parts of the cingulate gyri, and the temporal poles, as well as the frontal lobes, may be important in the maintenance of voluntary attention.

Frederiks (1974) believed that the RAS played a prominent role in both the HA and MBD syndrome. He theorized that the attentional weaknesses, distractibility, overactivity, irritability, impulsiveness, low frustration tolerance, and poor learning typify the difficulties one should expect from a failure of the RAS to perform its screening functions adequately.

Kornetsky and Eliasson (reported in Kaplan, Sadock and Freidman, (1975) performed an experiment which gives very strong support for the RAS theory of LD-MBD. Rats with electrodes implanted in the RAS were trained on an attentional task in which errors of omission and commission were both recorded. After pre-training, the RAS was stimulated with an electrical current which was reported to be too low to produce changes in gross behavior. The animals repeated the attentional task during the low level electrical stimulation. Under this condition the animals were reported to show fewer errors of omission. The authors interpreted the results to mean that attention was improved. When stronger electrical stimulation was used the errors of omission increased without an increase in errors of commission. The relationship was interpreted to mean that a disruption of attention had occurred. The administration of amphetamines to these animals was reported to have reduced the level of stimulation needed to improve performance.

The Kornetsky and Elisson study, if replicable, could be used to provide strong support for the presence of a disruption of some central process in the brain in at least some cases of LD-MBD. The disruption may be at the level of the RAS, at the level of the cerebral cortex, or at both on the basis of the research findings to

date. The strongest support to date, however, is for a disruption of normal RAS functioning.

Drug Treatment of LD-MBD

The major drugs used in the treatment of children diagnosed as LD-MBD are the stimulant drugs such as the amphetamines. The use of these drugs emerged first from empirical data, and the theoretical rationale developed later. The history of the use of amphetamines in the treatment of "behavior disorders" goes back to the thirties. Von Economono's Encephalitis left many children with a residual brain damage and assorted behavior problems. Bradley (1937) reported remarkable therapeutic effects from the use of amphetamines on a heterogeneous group of children who were living in a residential treatment center. Among the positive therapeutic effects noted in these children treated with amphetamines was a marked improvement in school performance. Molitch and Sullivan (1937), in another study published the same year as the Bradley study, found that benzedrine also improved the school performance of a group of children treated with the drug. These and other early studies laid the foundation for the later use of the central nervous system stimulant drugs on a wide variety of school children with learning problems. Most of the relevant research on the use of amphetamines and amphetamine-like drugs with LD-MBD children was completed in the sixties and early seventies.

The stimulant drugs, methylphenidate (Ritalin) and the amphetamines, are the drugs which are considered at present to be the most useful in the treatment of LD-MBD children. Improvement in behavior

has been found to occur in two-thirds to three-fourths of the children treated with these stimulants, while 5 to 10 percent experience a worsening of their symptoms (Cantwell, 1975, and Millichap, 1973). Therapeutic properties and side effects are very similar, although Ritalin has been found to have somewhat fewer side effects. Both drugs seem to be effective by their action in potentiating NE and DA at synapses in the central nervous system (CNS) (Kety, 1967). The latency of action for stimulant drugs is approximately thirty minutes and the effects last for from three to six hours. Ritalin must be given twice a day to be effective throughout the school day. Since Ritalin has fewer toxic effects than the other widely used stimulants (Grinspoon and Singer, 1973), and since it is considered as effective as the amphetamines, Ritalin has become the drug treatment of choice for the LD-MBD child (Millichap, 1973).

Sprague and Sleator (1973) observed that the ability to maintain sustained attention is particularly impaired in the hyperkinetic child and that CNS stimulants either reverse or ameliorate this defect. Conners and Rothschild (1968) and Conners (1970) used the Continuous Performance Test in modified form to study the effects of d-amphetamine and Ritalin on attention or vigilence. They found both of these drugs effective in decreasing errors of omission and commission.

Sykes, Douglas and Morgenstern (1972) compared the performance of HA and control children on a continuous performance test. The investigators found that the HA children performed significantly less well than the controls on this task, and that Ritalin was effective in reducing the relative impairment. In this study, three conditions

were used, experimeter paced task, the continuous performance test, and a similar task in which the subjects could control the presentation of stimuli. The hyperkinetic children were most impaired on the experimenter paced task relative to the control children.

Sprague et al. (1970) compared the effectiveness of Ritalin and thioridazine on the ability of 12 boys with poor school histories to perform vigilance tasks. The performance in the testing situation was compared with classroom behavior in order to investigate whether the objective performance task was predictive of classroom behavior. The authors found that Ritalin increased correct responding, increased the speed of response in hyperkinetic children, and improved classroom behavior. The thioridazine, however, produced a greater number of errors and slowed response time.

The effect of Ritalin on learning and reaction time performance in LD-MBD children was studied by many authors. Reaction time was studied because adequate attention and concentration, which are disrupted in the LD-MBD child, are thought to be essential elements of rapid reaction to stimuli. Sykes et al. (1972) administered Ritalin to forty clinically HA children, 34 boys and 6 girls, all of whom had IQs of 80 or above. The authors excluded all children who were considered to be psychotic, epileptic, grossly brain damaged, and children whose behavior problems were thought to be on a clearly emotional basis. With these exclusions, the drug treated subjects were found to perform significantly better than the placebo group on a learning task. These children not only made more correct responses than the placebo children, but they also made fewer errors. Connor and Eisenberg (1963) reported that the acute, or one trial

administration of Ritalin, facilitated learning and reaction time. They also reported that stimulant therapy reduced hyperactive behavior in the children. These authors found no carry-over effect of the drug on learning or reaction time 24 hours after the drug had been administered. Sprague, Barnes and Werry (1970) also investigated the effects of stimulants on learning and reaction time. Sprague et al. compared the effects of Ritalin, thioridazine, and a placebo on the learning and HA behavior of 12 emotionally disturbed under-achieving boys in a special education class. The authors found that the greatest positive effects of the stimulant drugs was a positive influence on attention span. Eisenberg, Conners and Sharpe (1965) and Weiss et al. (1968) reported similar studies in which the subjects received dextroamphetamine, Ritalin or a placebo. Each group of authors found similar improvement rates for the two active drugs. The children's school teachers appeared especially impressed by improvement in each child's general behavior. In all of the studies reviewed above, learning performance and reaction time performance were improved in LD-MBD children treated with Ritalin and the amphetamine, but placebo and thioridazine groups did not improve on any of the measures. In four of the above studies, Ritalin therapy seemed to have an overall positive effect on the child's behavior.

The effect of Ritalin on unwanted behavior was directly tested in the following study. Conners, Eisenberg, and Sharpe (1964) administered Ritalin and placebos to disturbed children from a residential psychiatric treatment center and to similar children in a group foster home. Positive behavioral changes were found among those

children in the drug treated group who had a measured IQ of less than 94, but no such positive behavioral changes were found in those children whose IQ measured between 94 and 135. The authors postulated that there could be an interaction between measured intelligence and drug effectiveness. Epstein et al. (1968) performed a study with ten boys as subjects, five of whom had evidence of a history of injury to the CNS. All of the ten boys showed hyperkinesis, short attention span, poor concentration and poor school performance. These subjects were used as their own controls and each received dextroamphetamine or placebo for two weeks. The authors reported a marked improvement in behavior and school performance of children in the organic group, but not in the boys with a negative history for CNS injury. This study gave findings in direct contradiction to those of many other authors (Bradley, 1937; Molitch & Sullivan, 1937; Conners, Eisenberg & Sharpe, 1964; Millichap, 1973, etc.). In these other studies, a positive result from drug therapy was not dependent on a positive history of CNS involvement.

Hyperactive behavior was specifically investigated to determine the type of change and mechanism involved in the reduction of HA due to stimulant medications. Millichap et al. (1968) reported that both Ritalin and placebo reduced activity, as measured by an actometer for the HA children. In another study by Millichap and Boldrey (1967) an actometer measure of motor activity actually showed an increase in the movement of children on Ritalin, even though both parents and teachers reported that there was an improvement in motor coordination and a reduction in impulsivity.

The mechanism of action of these drugs on HA and the exact definition of the population on which the stimulant drugs should be used have not been definitively determined, but hypotheses concerning drug action and patient characteristics abound. Lytton (1958) stated that stimulant drugs not only decrease the absolute amount of motoric activity or hyperkinesis, but the drugs also increase the amount of activity involved in goal directed behavior. Recent studies (Millichap & Boldrey, 1967; Millichap et al. 1968) have brought into question Lytton's belief that stimulant drugs in LD-MBD reduce the total amount of motoric activity. Some studies concerned with the nature of HA (Pope, 1970; Kenny et al. 1971; Fish, 1971) question the assumption that HA children are indeed more physically active than normal children, which was an implicit assumption in Lytton's study. Conners (1966) further suggested that the calming effect of the stimulants on HA children might be an artifact of observation which reflects not the activity level itself, but the organization of the activity in relation to the social demands of the situation. Cromwell (1963) has suggested that the over-activity of the HA child may reflect the short attention span and rapidly changing goal direction of these children whose behavior is fragmented and disoriented, a state of affairs which gives the impression of a high activity level. These explanations stress not the change in motor activity level which may or may not be produced by drug therapy, but rather the change in the direction or appropriateness of the activity as judged by teachers, parents, and other adults in authority.

A theoretical construct was also felt to be needed to explain the improvements in learning ability of drug treated children. To

account for the apparent improved learning ability of these children under stimulant drugs, Knobel (1959), Knobel, Wolmer and Mason (1959), and Knobel (1962) hypothesized that the drugs produce greater integration in these children which allows them to perform better. This integration was believed to be due to changes in the CNS of the children being treated. Conners and Rothschild (1968) concurred with this belief that the stimulant drugs resulted in improved school performance because the drug enabled the child's responses to a given stimulus to be more appropriate to the particular task at hand. Conners (1971) hypothesized that such improvement was due to the enhancement of attention to the task combined with increased arousal and motivation to perform the task. These authors believed that CNS stimulants improve learning receptivity by decreasing non-task relevant responses.

Many educators and psychologists interested in the LD-MBD child felt compelled to attempt to explain the paradoxical effect that stimulant drug therapy appeared to have on the hyperkinetic behavior seen in many LD-MBD children. The paradoxical effect is that stimulant drugs have a calming influence on many hyperkinetic children, the reverse of the drug effects observed in the normal adult. This apparent paradox has long puzzled parents and educators. In an effort to explain this, Satterfield, Cantwell, and Satterfield (1974) hypothesized that the behavior disorder of hyperkinesis was characterized by a low CNS arousal which was correlated with low levels of inhibition in the CNS. The stimulant medications were thought to restore the CNS arousal to normal levels which then would allow normal inhibitory functioning, thus providing the child with better internal

control of his behavior. Satterfield, Cantwell and Satterfield (1974) conducted a four part study to test this hypothesis. First, they attempted to determine the relationship between skin conductance levels and CNS arousal levels as measured by EEG readings on non-medicated children who were considered to be hyperactive. Fifty percent of the children were found to have low skin conductance levels, while only 8% had abnormally high levels of skin conductance. Second, the introduction of Ritalin was found to increase skin conductance levels, although the authors reported a number of spontaneous fluctuations in the conductivity of the skin. The child who showed lower skin conductance levels, and a higher mean EEG amplitude with more energy in the low frequency bands while at rest, was found to respond best to medication. Further, these children showed a greater increase in CNS arousal than did the poor drug responders. The third study showed that teacher ratings correlated well with the skin conductance levels. Those children who had the lowest skin conduction levels had the most overall classroom behavior disturbance and these were also the children who obtained the best results from the Ritalin. In the fourth part of this study, EEG findings were used to divide the children into three categories, normal, borderline, and abnormal EEG tracings. The greatest improvement from the medication, as reflected by teacher ratings, was found in the group of children who had had the abnormal EEG readings. The findings of the Satterfield et al. (1974) study are compatible with the theory that there is a dysfunction of the RAS which is responsible for the behavioral and learning problems found in LD-MBD children.

The theories of Conners and Cromwell on the mechanism of the drug effects on hyperkinesis and Knobel and Conners on the theoretical explanation of learning improvement after medication, as well as Satterfield's explanation of the paradoxical calming effects of amphetamines were apparently made without extensive knowledge of physiological research on the RAS. They also appeared to be unfamiliar with Frederik's (1969) theory that the RAS, when functioning normally, enabled the focus of attention by reducing extraneous sensory information. However none of the theories are incompatible with the present knowledge of RAS functioning. Each of the theories appear to oversimplify the LD-MBD problem but none of these theories establish the exact biochemical and physiological actions of the stimulant drugs. The investigation of measurable behavior changes produced by the stimulant drugs need not, however, wait on knowledge of the neurochemical actions involved.

Neuropsychological Assessment of the LD-MBD Child

Neuropsychology most broadly defined is the study of brain-behavior relationships (Pribram, 1962). Restricting the area of the term neuropsychology to its meaning in human clinical neuro-psychology is more useful, however. Neuropsychology, so defined, is the application of psychological tests and procedures to the study of abilities dependent on brain functioning. Neuropsychology as defined for human clinical neuropsychology can be used in the assessment of brain dysfunction in the LD-MBD child.

Twenty years of research by various investigators have established the Halstead-Reitan Neuropsychological Test Battery and its

associated procedures as a highly effective instrument in the study of brain-behavior relationships (Reitan, 1966; Russell, Neuringer and Goldstein, 1970; Benton, 1974 and Boll, 1974) for adults and children. By utilizing the various research findings, and avoiding the dependence on a strictly "level of performance approach" with its many inadequacies (Reitan, 1974 and Rourke, 1975), psychologists are able to predict with reasonable accuracy the site and type of lesion in individual cases. With these assessment procedures, the psychologist is no longer limited to a probability statement with respect to the presence or absence of brain damage. One can offer a description of the underlying neurological processes which give rise to the patient's symptoms as well as the constellation of behavioral deficits observed. The type and extent of psychological deficits and remaining strengths in cognitive, perceptual and motor abilities can also be used to provide information helpful to the remediation process. Typically neurological inference is derived in a variety of ways through the analysis of the patterns and levels of performance on a number of cognitive, perceptual and motor tasks (Ruthven, Lewis and Goldstein, 1973).

Reitan developed the first battery of tests suitable for the use with children. This battery used the adaptations of the adult battery which was devised by Halstead (1947). Reitan and his colleagues began formal research with the battery for older children in 1953, but it was not until 1965 that Reed, Reitan and Kløve (1965) published the data from this long term project. The battery that they developed was a modification of the adult battery and was developed for the 9 to 15 year-old child. There is a battery for

younger children 5 to 9 years-old (Klonoff, Robinson, and Thompson, 1969), but it will not be further described here. In relation to the adult battery, the modifications made for the older children's battery are relatively minor and are ones of degree rather than kind. A brief description of the subtests in the battery and their uses in the description of cerebral dysfunction follows. A complete description of the tests and directions for their administration can be found in Appendix B.

The first two tests described are primarily measures of cognitive functioning. They are the Category Test and the Wide Range Achievement Test.

> The Category Test: This test measures the subject's ability to identify the concept or principle that rules a series of figures and geometric forms. Performance on this test requires the use of a number of complex functions and abilities. The category test appears, however, to be primarily a test of the capacity to abstract, to form concepts and to use organizational ability. Since one of the most frequent effects of brain lesions, regardless of the type or focus, is to impair the ability to conceptualize or to generalize from individual instances to some rule or principle, this test appears to be a good measure of the individual's capacity to cope in a general way with the complexities of a normal environment. Descriptively this test assesses the capacity of the individual to organize, plan, transcend the immediate stimulus, use judgmental capacity and to solve complex problems (Ruthven, Lewis & Goldstein, 1973).

The Category Test was found to be the best of the major tests in the battery for discriminating normal from brain damaged adults by Reitan (1973) and for children by Boll (1972). It does not appear to have any particular localizing or lateralizing value (Shure and Halstead, 1958; and Doehring and Reitan, 1962). People who have extremely poor Category Test scores often suffer from extensive brain dysfunction that is recently acquired or rapidly progressive in

nature (Ruthven, Lewis and Goldstein, 1973), and such a finding would be unlikely in children who are diagnosed as being LD-MBD. Mild to moderate impairment on the Category Test would, however, be expected from LD-MBD children. Good or relatively good performance on this test does not indicate the absence of brain dysfunction because the individual may have a discrete lesion or specific cognitive, perceptual or motor deficits which do not significantly impair more complex abilities.

> The Wide Range Achievement Test (WRAT): This test is used to assess academic achievement in reading, spelling and arithmetic (Jastak and Jastak, 1965).

The Reitan-Indiana Aphasia Screening Test is a test which measures both cognitive and perceptual elements.

> The Reitan-Indiana Aphasia Screening Test: This test is a modified and shortened version of an aphasia test developed by Halstead and Wepman (1949). This test measures language abilities and therefore may be viewed as an examination of left hemisphere functions. It is a screening test and it is not adequate for exact localization within that hemisphere based on the pattern of aphasic symptoms alone. It should not be used to diagnose the exact type of aphasia, since there are difficulties in clearly differentiating between expressive and receptive difficulties. Certain inferences, however, can be drawn by clinically correlating one item with another to decide among possible alternative or explanations.

The following tests measure primarily perceptual abilities. They are the Perceptual Disorders, the Speech Sounds Perception, and the Seashore Rhythm Test.

> Reitan-Kløve Sensory-Perceptual Examination: This part of the battery consists of a group of brief tests of tactile, auditory and visual perceptual skills. Additional lateralization information can be obtained from this series of tests since each involves a comparison of the sides of the body. Parietal lobe integrity is tested.

A. Tactile, Auditory and Visual Suppression: The underlying rationale for these tests is the fact that in some cases of brain damage single stimulation of the receptor can be correctly perceived, but a second simultaneously applied stimulus in the contralateral area cannot be perceived. This effect has been known for a long time, and it was carefully studied by Benton (1952). The visual field is established while testing for visual suppressions. Generally non-brain damaged subjects can perform these tasks without error, or with a very few initial errors. The production of errors in a consistent pattern can denote severe pathology of the cerebral hemispheres. These tests do not measure primary sensory functions, the defect appears to be more subtle, and may denote a defect in attention (Russell, Neuringer and Goldstein, 1970).

B. The Finger Discrimination: The purpose of this test is to detect a condition known as finger agnosia. Benton (1959) defined this deficit as the relative or absolute inability to name or show the individual finger stimulated tactually when no visual cues are allowed. Impairment on this test appears to represent a tactile discrimination deficit, particularly if it is unilateral.

C. The Fingertip Number Writing: This test provides a measure of tactile recognition ability. The impaired ability is called astereognosis or tactile agnosia. Tactile agnosia is probably the more correct of the terms, since astereognosia generally refers to the inability to recognize a three dimensional form from touch alone. The parietal lobes are the primary mediators of tactile discrimination; however, the deficit on this test is seen also in individuals with lesions in a variety of loci. Thus, interpretation of deficits on this test can be made only by evaluating the entire test battery.

The Speech Sounds Perception Test: This test assesses the subject's ability to make auditory discriminations, i.e., the ability to tell the difference among speech sounds, and to identify their written forms. The subject must be able to see, hear, and have minimal knowledge of English consonant's phonetic representation. The temporal lobes mediate hearing and speech and this test is generally considered to be a test of the intactness of the left temporal lobe.

Extreme deficits on the Speech Sounds Perception Test are seen in individuals with receptive or Wernicke's aphasia. Goodglass and Kaplan (1972) reported that this type of aphasia can be localized in the posterior portion of the first temporal gyrus of the left hemisphere. Their work was with adults, and we would not expect to see extreme dificits in most LD-MBD children. Deficits on the Speech Sounds Perception Test can be of a quite subtle nature, however. LD-MBD children may have difficulties in integration of sound in a normally precise and rapid manner and therefore may show milder deficits. Klonoff and Low (1974) found a difference ($p < .05$) between normal controls and children with learning problems on this test.

> The Seashore Rhythm Test: This test was taken from the Seashore Test of Musical Talent. This test requires alertness, sustained attention to the task and an ability to perceive differing rhythmic sequences (Reitan, 1966). This test is easily disrupted by an inability to sustain attention and by a defect in temporal sequencing and time perception.

The Seashore Rhythm Test is not used to lateralize brain dysfunction. This test does, however, discriminate very well between individual adults with brain damage from those without brain damage (Reitan, 1966). Reitan et al. (1965) found that the Seashore Rhythm Test was one of the tests in the battery which differentiated between brain damaged and non-brain damaged children at beyond the .005 level. This test is a good measure of the capacity to sustain task-oriented attention (Ruthven, Lewis and Goldstein, 1973). The Seashore Rhythm Test performance should be in the impaired range for the LD-MBD children, for subjects who do poorly on the Seashore Rhythm Test are characterized as being readily distractible, unable to concentrate, and as having a limited attention span, as are LD-MBD children.

The following tests measure primarily perceptual-motor elements, although their usefulness is not limited to this aspect of neuropsychological functioning. They are the Tactual Performance Test, the Trail Making Test and the Grooved Pegboard.

> The Tactual Performance Test: This test is a modification of the Sequin-Goddard formboard. It measures the individual's ability to use each hand independently. The first two trials give an opportunity to compare the functioning of the two sides of the body and to investigate the status of the contralateral cortical hemisphere. The third trial provides an opportunity to examine the subject's ability to coordinate the use of both hands. Normal individuals show improvement from trial to trial on the Tactual Performance Test. Inferentially, those subjects who show trial-to-trial improvement may respond more normally to learning situations than those who do not. Clinically, the right-left performance discrepancy is a lateralizing indicator that can be used with supportive data from other tests.
>
> After the third trial with the Tactual Performance Test board, the board is removed and the subject is asked to draw the shapes and location of the blocks on the board. The drawing of the board is evaluated to ascertain the subject's perceptual-motor coordination, spatial relations abilities, and some aspects of incidental memory.

The Tactual Performance Test has been found useful in discriminating brain damaged from non-brain damaged individuals and right hemisphere from left hemisphere damage (Reitan, 1958). Boll (1974) compared the performance of a heterogeneous group of brain damaged children with matched controls. The neurological impairments were due to both acute and chronic head injuries and included right hemisphere, left hemisphere and diffuse damage. The normal and brain damaged children were significantly different in their performance on all three timed executions of the Tactual Performance Test. Klonoff and Low (1974) found that children with minimal cerebral dysfunction

performed significantly less well than their matched controls on these measures also.

> The Trail Making Test: The Trail Making Test consists of two parts, Trails A and Trails B. The task in its total demands is rather more complex than it appears. At its most basic level, Trails A, it requires that the subject be able to interpret the stimuli, and know the sequence of numbers, while he performs the task rapidly. Trails B also requires psychomotor speed and perceptual acuity, but more importantly, it requires what Goldstein and Scheerer have referred to as a "simultaneous function" or the ability to carry on two activities concurrently. It involves thinking while in action, where decisions must be made as the task proceeds. The performance can be used to establish lateralization when used in combination with other data. When the performance on Trails A and B are equally impaired relative to their respective norms, the individual may have a relatively less functional right hemisphere or generalized dysfunction of both hemispheres, but if Trails B is impaired while the performance on Trails A is in the normal range, lesions in the left hemisphere can be suspected.

The research data supports the clinical interpretations quoted above. Reitan (1955, 1958) reported that the Trail Making Test was a valid indicator of brain damage in adults. Individuals with left hemisphere lesions did more poorly on Trails B relative to its norms than on Trails A relative to its norms (Reitan and Tarshes, 1959, and Yanni, MacDonald and Young, 1973). Davids et al.(1957) used the modified Trail Making Test for older children and found that brain-damaged children performed more poorly on it than did normal children. Reed et al. (1965) reported that for a group of heterogeneously brain damaged children, performance on both Trails A and B was significantly less adequate than for a matched control group with normal brain functioning ($p < .001$). Boll (1974) confirmed these results. Thus, the research data indicates that this test can be

used with children to indicate, with a significant level of confidence, both the presence of brain damage and lateralization.

> The Grooved Pegboard Test: This test is a measure of motor speed and accuracy of hand-eye coordination. It allows comparison of the sides of the body and can be used for lateralizing cortical damage in adults (Kløve, 1963; and Knights and Moule, 1968) and children (Rourke, Yanni, MacDonald, and Young, 1973). This test is one of the tests in the Kløve-Mathews Motor Steadiness Battery, which has been added to the Halstead-Reitan battery by many investigators (Goldstein, personal communications; Rourke, 1975; and others).

The Wechsler Intelligence Scale for Children (WISC) is a battery of subtests which assess many facets of functioning. There are subtests which are primarily cognitive. Other subtests assess cognitive-perceptual elements of behavior and still others assess perceptual motor skills.

> The Wechsler Intelligence Scale for Children (WISC): The WISC is not generally used to determine the presence or absence of brain damage. The verbal-performance relationship can be used as one of the many indicators of lateralized dysfunction however. Generally, if the IQ score obtained from the Verbal subtests is significantly lower than the IQ obtained from the Performance subtests, lesions in the left cerebral hemisphere can be suspected. The reversal of the verbal performance IQ difference usually indicates that there may be a dysfunction of the right cerebral hemisphere. Lack of a difference in the verbal-performance IQs does not necessarily mean no brain damage. Equal performance on the Verbal and Performance subtests may also be found in individuals with diffuse lesions. There is clinical evidence for the relationship between certain of the subtests and certain lobes of the cerebral hemispheres. Lower performance on the Block Design is thought to be a function of the parietal lobe. Picture Arrangement deficits are often found associated with right temporal lobe disease. Similarities deficits are often found in association with left temporal lobe dysfunction (Russell, Neuringer and Goldstein, 1970). These associations were found for adults, but should be considered as possibly valid for older children also.

The WISC should not be used by itself to determine the presence or absence of brain damage, for many other tests do that better for

adults (Reitan, 1959) and the older children (Boll, 1962). The IQ score is, however, very useful in interpreting neuropsychological test results. A score which is in the impaired range on a task of complex reasoning like the Category Test can have vastly different meanings for an individual with an IQ of 130 than for an individual with an IQ of 85. In general, the IQ has a tempering effect on the clinical interpretation of the performance on a number of tests of the battery, which assess more complex abilities (Klonoff and Low, 1974, and Matthew, 1974).

Specific features of the WISC are useful in the interpretation of the Halstead-Reitan Battery. The Coding (Digit Symbol) subtest is very sensitive to many kinds of dysfunction in both children and adults. Reed et al. (1965) reported that they found only a 4% overlap between normal and brain damaged children in their performance on the Digit Symbol subtest. Thus for children, performance on this subtest was the best single predictor in the battery for the presence of brain damage. The ratio of the Digit Symbol to Block Design, Picture Arrangement and Picture Completion is of particular importance. Rennick (cited in Russell, Neuringer and Goldstein, 1970) has developed a system for scoring the relationship between Block Design and the other subtests of the WISC named above. Rennick's index was developed for adult brain injured subjects; however, it has been used in clinical interpretation of the performance of children suspected of having brain dysfunction (Goldstein, personal communication). This use of Rennick's index for children is further supported by Reed et al. (1965) who found that Digit Symbol performance differentiated brain damaged from normal children.

The last major test in the Halstead-Reitan battery is the Finger Tapping Test, which is a test of pure motor ability.

> Finger Tapping Test: This is a test of pure motor speed. Brain damage of many kinds is accompanied by motor speed reduction. The finger tapping test provides an opportunity to compare the right and left sides of the body and the status of their corresponding hemispheres.

The Finger Tapping Test was found to be a good lateralizer of brain dysfunction in adults (Goldstein and Shelly, 1973) and should be for children also. Boll (1974) and Reed et al. (1965) found that this test failed to differentiate normal control children from older brain damaged children. Klonoff and Low (1974) found a significant difference when children of the same age were compared, but none when matched groups of children of varying ages from 9 to 15 years-old were compared. These results may be due to the great variability in motor speed of children of different ages which is due to developmental factors.

The above tests are the major ones used in the Halstead-Reitan Battery. The research on its development and use with brain injured adults and children is extensive (Reitan, 1971; Doehring and Reitan, 1962; Boll, 1974; Klöve and Matthews, 1974; and Davison, 1974; among others). The application of this battery and other neuropsychological measures to the examination of the LD-MBD child has only developed within the last five years, however.

A great deal of the work done in evaluating the LD-MBD child with the Halstead-Reitan has been done by Rourke and his co-workers at Windsor Western Hospital in Ontario and is reviewed by Rourke (1975). These researchers did not use the entire Halstead-Reitan test battery

in their work, instead they used selected tests. They investigated the relationship of the selected tests to the variables under study.

Rourke and his co-workers used one of the major underlying assumptions of LD as a starting point for their research. Their basic assumption was that these children are characterized by an extreme difficulty in maintaining attention. The LD-MBD children are distractible, unable to pay attention, or prone to focus on irrelevant aspects of the learning situation. Further, it has been postulated that such attentional deficits are greater in younger than older children and the attentional deficits were considered to be correlated with hyperactivity. In order to test these hypotheses, Czudner and Rourke (1970, 1972) used reaction time (RT) as a measure of attention in brain damaged children. The choice of RT to measure attention was also influenced by the earlier studies of Blackburn and Benton (1955) and De Renzi and Faglioni (1965), which indicated that simple RT can be used as a sensitive indicator of brain damage. Two age groups of children were selected for normal and brain damaged groups. Visual reaction time was measured. The younger brain damaged children were inferior in performance to the younger normal children, older brain damaged children, and older normal children. The older brain damaged children did not differ in performance from the older normal children. This study supported the contention that younger brain damaged children were more distractible than non-brain damaged children. It also showed a reduction in magnitude of the deficit as the child matured.

In order to establish the generalizability of the findings reported above, Rourke and Czudner (1972) repeated the study with

auditory RT in the place of the visual RT. The findings were similar. From these three studies, Rourke and Czudner concluded that the deficit in RT must be central, and therefore was much more likely to be related to attention.

Pulvermach (1973) attempted to treat the deficits of attention. He used positive reinforcement to shape attending behavior. He reported success in significantly modifying attending behavior. His results suggest that attention in the LD-MBD child can be improved by environmental influences, even if the deficit arises from CNS factors. The above studies dealt with frankly brain damaged individuals, not LD-MBD children, but they set the stage for connecting the two in a logically consistent manner.

In an attempt to establish whether the terms "cerebral dysfunction" or "neuropsychological" could be used to advantage with LD children, Rourke, Yanni, MacDonald and Young (1973) investigated the similarity in performance between these children and adults with known cerebral dysfunction. The authors divided these children, who had no documented cortical dysfunction, on the basis of presence or absence of lateralized motor deficits as measured by the Kløve-Mathews Motor Steadiness Battery. The degree and direction of the WISC Verbal-Performance difference was obtained for each child. The pattern of WISC subtest scores for each group was compared with the pattern of WAIS subtest scores previously found (Reitan, 1955; Benton, 1962; and Reed and Reitan, 1963) for adult subjects with known cerebral lesions. The results were similar for the adult brain damaged and the LD children. Impaired speed with the right hand combined with normal speed for the left hand was found in individuals

who had a significantly lower Verbal IQ than Performance IQ score as measured on the Wechsler. Impaired motor speed with the left hand, in the presence of normal performance with the right hand, was associated with lower Performance IQ than Verbal IQ on the Wechsler scales. The authors also noted that the pattern of slower right motor speed with normal left motor speed was found in LD children who performed less well on all verbal and academic tests than in children who had bilateral motor speed impairment. These findings gave added support to the hypothesis that there is a neurological deficit of some kind operating in many LD-MBD children.

Kinlayson and Rourke (reported in Rourke, 1975) found similar relationships when the above study was repeated with 6 to 8 year-old children with learning disabilities. The results, however, were said to be less striking than those found for the older children. This is in agreement with the results reported by Reitan (1974) that the WISC Verbal IQ and Performance IQ were among the most powerful discriminators between brain damaged and normal children in the early school years. From these studies, Rourke (1975) concluded that learning disabled children could also be separated into groups on the basis of lateralized deficits on the grooved pegboard.

To follow-up these findings, a series of studies was undertaken (Rourke, Dietrich, and Young, 1973; Rourke and Telegdy, 1971; and Rourke, Young and Flewelling, 1971). Again, Rourke and his co-workers found that LD children could be divided into groups on the basis of psychological test patterns similar to adults with known cerebral lesions, and that the children so divided performed on other tests similarly to the adults grouped in the same manner. Typically in

these studies LD children were formed into groups on the basis of WISC Verbal-Performance IQ disparities. These groups performed on other psychological tests and measures in a manner much like adults with cerebral lesions who were grouped on the same kind of Verbal-Performance IQ differences. Younger children of 6 to 9 years-old, however, fail to show the performance relationships found for the older, 9 to 15 year-old LD children. Rourke (1975) suggested that the difference in performance between the younger and older children on psychological tests taken mainly from the Halstead-Reitan Battery supported the contention of many authors and investigators (Boll, 1974; Werry, 1968; and Reed, Reitan and Kløve, 1965) that developmental factors are of critical importance when considering brain-behavior relationships. While it is no doubt true that there is an age related change in performance, it is also possible that the lack of consistency in findings for the younger and older children may be partly due to differences in the test battery itself for the younger and older children.

Rourke and Kinlayson (1975) reported the results of their use of the Trail Making Test to divide LD children into groups. One group of children was formed from those who had normal performance on Trails A of the Trail Making Test and lower than normal performance on Trails B. These children were assumed to have relatively deficient left cerebral hemispheres. The second group of children were impaired in their performance on both parts A and B. The latter children were assumed to have less efficient right cerebral hemispheres. The children so divided were tested on a number of verbal, auditory-perceptual, visual-spatial, and psychomotor

abilities. The performance on the above tests for each of the two groups of LD children was compared with the performance of adults with known brain-damage and similar performance patterns on the Trail Making Test. The authors found that the pattern of performance on the Trail Making Test was predictive of the pattern of performance on all the other tests of the battery for both the LD children and the brain injured adults.

Rourke (1975) reported an unpublished study done by himself and Finlayson in which LD children were divided into groups on the basis of their academic performance measured on the WRAT. The children, who were again in the age range of the Older Children Battery (9 to 15 years), were divided into groups which were relatively more adept at arithmetic skills than reading and spelling, a second group which was equally impaired in academic performance on all three subjects, and a third group in which the reading and spelling were average, but where the arithmetic skills were lower than expected for their age, intellectual level and grade. The most clear-cut finding was that a deficit in arithmetic, combined with good achievement in reading and spelling, was associated with a performance which is characteristic of individuals with a relatively dysfunctional right hemisphere. This finding supports the hypothesis of Diamond & Beaumont (1962). that arithmetic skills may be dependent on right hemisphere functioning. The results for the other two groups were less clear, but they were reported to resemble in many respects the performance expected of individuals with a relatively dysfunctional left hemisphere. Rourke (1975) concluded from these findings that the differential performance on tasks such as the Trail Making Test

is more accurate in predicting patterns of performance on other psychological tests than predictions of neuropsychological functioning taken from academic performance.

Klonoff and Low (1974) used a different approach than Rourke and his co-workers in their study of children with learning problems. Klonoff and Low reported the results of the entire Halstead-Reitan Battery for two groups of minimal cerebral dysfunction (MCD) children with comparison data from matched normal control subjects, two groups of acute brain injured children and two groups of chronic brain injured children. They found that all groups differed significantly from each other for most of the measures taken from the neuropsychological battery. Both younger MCD children (2 to 9 years-old) and the older MCD children (9 to 15 years-old) differed significantly from the matched normal controls, but younger MCD children were more impaired relative to the control group than the older children. The Klonoff and Low study was the only study reported in the literature in which the entire Halstead-Reitan Battery was given to 9 to 15 year-old children showing the LD-MBD symptom cluster.

From the review of the literature, it is apparent that the development of the theory of LD-MBD and the treatment of the children exhibiting the LD-MBD symptom cluster has been at times divergent and confusing. The multiplicity of terms used, the lack of agreement on definition and frequency of the syndrome, the uncoordinated multidisciplinary sources of the research and theories, and the equating of groups of children which are only superficially similar, have all been major factors in the creation of the confusion in the area.

Order, however, appears to be beginning to emerge. The following results are clear. Research is becoming more controlled and the definition of the syndrome is becoming more consistent. The three major theories of etiology are: (1) brain damage or brain dysfunction of a sub-clinical nature; (2) hereditary or genetic; and (3) biochemical or physiological dysfunction of the brain, none of which are necessarily mutually exclusive and any or all of which may be valid. The data indicates that there is at the very least a biochemical or physiological dysfunction of the brain which is of major importance in the LD-MBD syndrome. Whether the deficits in performance in LD-MBD children are due to cerebral dysfunction or dysfunctions of some type in the lower brain centers is not known. There is evidence, however, that LD-MBD children perform less well on tests which are known to measure neuropsychological functioning in both brain damaged adults and brain damaged children than do normal control children. Further, clinical and controlled research studies with LD-MBD children have shown that stimulant drugs are effective in improving behavior in many cases.

CHAPTER III

METHOD

This chapter consists of five major sections. They are: statement of the problem, subjects, instrument, procedure, and statistical analyses. The statement of the problem consists of a brief rationale for the study. The rationale precedes a statement of the major hypothesis. This statement is followed by a brief statement of minor hypotheses to be tested. The first minor hypothesis is based upon clinical experience concerning tests from the neuropsychological battery which would appear to be most likely to show changes due to Ritalin treatment. The last set of minor hypotheses is based on logically consistent groupings of the various tests of the battery. The three sections on subjects, instrument and procedure will consist of a brief description of each. The last section on statistical analyses provides a description of the data analysis.

Statement of the Problem

The LD-MBD syndrome is hypothesized to have some type of neurological basis, and recent research has shown that LD-MBD children do indeed perform more poorly than normal children on tests which traditionally have been used to assess neuropsychological status. Ritalin, which is the drug in most widespread use in the treatment of LD-MBD children, is reported to increase the screening activity

of the RAS. This increased screening which Ritalin produces is believed to enhance the attending ability of the child. The increase in attending ability facilitates academic and social learning. The increased ability to attend is also hypothesized to be directly associated with decreased distractibility, decreased activity, reduced irritability, improved impulse control, and improved receptivity to the learning process. At this time, the effects of Ritalin on the performance of LD-MBD children on tests of neuropsychological functioning have not been evaluated. This evaluation of neuropsychological functioning of LD-MBD children with and without Ritalin therapy is the major focus of the present research. The major hypothesis to be tested is as follows:

> Children who are receiving Ritalin therapy for their LD-MBD problems will be less impaired in their neuropsychological functioning than similar children who are not receiving Ritalin therapy.

The following minor hypothesis will be tested. The first hypothesis was developed from clinical experience with the Halstead-Reitan tests in assessing brain impaired individuals. The last hypotheses were made from a grouping of logically related clusters of skills. The following predictions concern the differential test performance on the neuropsychological test battery of LD-MBD children with and without Ritalin therapy. Appendix C contains a listing of the test measures compared in each hypothesis.

> 1. The experimental or Ritalin therapy children will be less impaired in their neuropsychological functioning than the controlchildren on selected tests measuring cognitive, perceptual and perceptual-motor skills. These tests were chosen on the basis of clinically determined sensitivity to changes in neuropsychological functioning.

2. The Ritalin treated children will be less impaired in neuropsychological functioning than control children on tests which are sensitive to the effects of right cerebral hemisphere dysfunction in adults, i.e., impaired spatial-relations abilities and impaired motor skills on the left side of the body.

3. The Ritalin treated children will be less impaired in neuropsychological functioning than control children on tests which are sensitive to the effects of left hemisphere brain dysfunction in adults, i.e., damage which results in impaired verbal-language skills and motor skill deficits on the right side of the body.

4. The Ritalin treated children will be less impaired in neuropsychological functioning than control children in their perceptual, perceptual-motor and motor performance.

5. The Ritalin treated children will be less impaired in neuropsychological functioning than control children on those tests and items of the battery measuring attention and concentration.

6. Ritalin treated children will be less impaired in neuropsychological functioning than control children on tests and measures comprising the impairment index. They will also have a lower percent of their performance in the impaired range than the control children.

Subjects

Thirty children, all between the ages of eight years, nine months and twelve years, four months and classified as LD-MBD by their respective schools and physicians were used as subjects in this study. Fifteen of the children were taking Ritalin as a physiological treatment of the problem behaviors associated with the LD-MBD syndrome. The experimental group consisted of two girls and thirteen boys between the ages of nine years, seven months and twelve years, four months. The control group consisted of fifteen children, three girls and twelve boys, who were also diagnosed LD-MBD but who

were not receiving Ritalin therapy at the time of the testing. The ages of the control children ranged from eight years, nine months to eleven years, four months.

No effort was made to match the groups on age, sex, IQ, handedness, or grade placement. It was assumed that no significant difference would exist on these variables. This assumption was tested and only sex and IQ measures met the above assumption. The presence of three left-handed children in the control group and one in the experimental group made the experimental group significantly more likelv to be right-handed ($p \leq .10$). The experimental group had an average age of 10.7 years, the controls were younger with an average age of 10.1 years ($p \leq .05$). The mean grade placement was 4.5 for the controls and 5.1 for the experimental subjects ($p \leq .10$). Of these differences, age probably is the most important, but the trends for handedness and grade placement suggested that matching for all three of these variables might have provided a better study.

All of the subjects were selected from the public school system of two Kansas cities. All children were living in their natural home environments and they were considered to be in good physical health. The LD-MBD diagnosis was determined by each child's physician and the school psychologist. All children were receiving special assistance, either from a learning disabilities teacher in a self-contained classroom or from a learning disabilities teacher in a resource classroom. All children were classified as having average or potentially average intelligence with an IQ of 80 or above on the WISC (Wechsler, 1949).

In all cases Ritalin had been prescribed by the child's physician. The attending physician determined the appropriate therapeutic dosage. The drug treated children were receiving from 15 to 40 milligrams of Ritalin daily. The neuropsychological testing was administered without any change in the previous drug regime. No subjects were dropped from the study.

Instrument

The Halstead-Reitan Neuropsychological Test Battery for older children was used to assess neuropsychological functioning (see the literature review and Appendix B). Quantified raw scores were obtained for the performance of each child on each item or test in the battery.

For each child the performance measures or scores on twelve tests of the battery were converted to standardized ratings of impairment called impairment indices (see Appendix D for raw score conversions to impairment indices for each of the 12 tests). The impairment indices represent the degree of departure of the score or performance on a particular test from that of a population of non-brain damaged or impaired children.

Five levels of performance have been established for various ranges of performance. A performance on a test is given a rating of "0" if the score on the test is better than that found in the average non-brain impaired child. A rating of "1" represents the average performance of the same non-brain impaired population. A rating of "2" reflects mild impairment or mild decrement of performance in relation to the normative population. A rating of "3" represents

moderate impairment, and a rating of "4" severe impairment relative to the performance of non-brain impaired children for that particular test. The ratings on the 12 tests are combined to establish an average impairment index for each child. The average impairment index can theoretically vary in each case from 0.00 to 4.00. The average impairment index is a global measure of the degree of impairment. Generally an average impairment index of 1.50 and above is considered the cut-off score between non-brain damaged and brain damaged status (Russell, Neuringer & Goldstein, 1970). Another global measure of impairment is the percent of the performances on the twelve tests that were found to fall in the impaired range.

Procedure

Except for the WISC and the Wide Range Achievement Test, none of the children had had any prior experience with the tests of the Halstead-Reitan Neuropsychological Test Battery. The neuropsychological testing of these children took place at either the Fort Hays State College Psychology Clinic or in the office of a Wichita clinical psychologist. The testing was divorced from the public school environment to minimize contaminating influences, ensure privacy, and to better control the testing environment. The number of control subjects tested at each facility was approximately equal to the number of experimental subjects tested at the same facility. All evaluations were conducted by an experienced Halstead-Reitan Examiner.

After approval was obtained from the research committees of the two public school systems which participated in the study, letters explaining the study (Appendix E) were sent to parents of the LD-MBD

children. One of the parents, usually the mother, accompanied the child to the testing site. At that time, the parent signed a consent form and a release of information form (Appendix F). The parent was interviewed separately from the child to obtain drug dosage information and to explain the purpose of the study and testing. A follow-up appointment for the parent to discuss the findings from the evaluation was also arranged. All testing of the children was started in the morning and, in all cases, it was concluded the same day with a one hour lunch break. Test administration took from 3½ to 7 hours. The average was 5 hours and there was no difference in the time it took the Experimental and Control groups to complete the testing.

Statistical Analyses

A series of step-wise linear discriminant function analyses were computed to examine the differences among the groups on the hypothesized relationships. Specific scores from the data collected with the Halstead-Reitan Neuropsychological Test Battery were used to form a composite which best discriminated between the Ritalin and non-Ritalin groups. The BMD-P6M computer program was used to calculate the analyses and the computations were executed at the Oklahoma State University Computer Center.

The discriminant function analysis is used to determine which variables contribute the most information about group membership (Cooley & Lohnes, 1971, p. 243f; Overall & Klett, 1972, p. 280f). That is, it is used to find the subset of variables which maximize group differences. The discriminant function analysis utilizes a

weighting system which maximizes the variance between groups while minimizing the variance within groups. There are two basic assumptions in the use of the discriminant function analysis; first, that misclassification costs are equal and that the prior probabilities of each population are equal. In the calculation of this statistical procedure, an analysis of variance is first run. Such an analysis gives us initial F value in order to determine the first entry into the function. This initial F value determines the significance of a particular variable in the separation of the groups without reference to any other variable. After the first step is completed, a second series of analyses determine new F values. The new F values are a measure of the contribution of the new variable given the condition that the previously entered variable will be used. Thus, the second set of F values indicates the amount of additional variation which can be accounted for by the addition of the second variable. All F values calculated after the first step are conditional values which indicate the significance of a variable after part of the variance has been accounted for by variables added to the equation previously. At each step new F values are calculated to determine the part of the remaining variance which can be accounted for by each remaining variable. The factor with the highest F value is entered at each step. Provisions are also made to delete variables if their contribution to the total variation falls below a pre-established level. This, too, is checked at each step. The processes continue until no significant reduction in variance is made by the introduction of a new variable.

The step-wise linear discriminant function analysis is a special case of the linear discriminant function analysis which allows the examination of each step in the establishment of the subset of variables. The usefulness of the subset of variables established by this analysis is also dependent upon the percentage of cases correctly classified. When the percentage is high, group differences are considered to exist, and are considered to be measured by the variables named.

The major hypothesis was evaluated by use of a step-wise discriminant function analyses for the variables which had an initial F value significant at the .10 level (see Appendix C for a list of variables). When very highly correlated items were encountered, only one was used. This step-wise discriminant function analysis was accomplished with F values for inclusion and deletion set at the .10 level of significance ($2.88 \leq F$).

The first minor hypothesis, which dealt with clinically selected tests, was evaluated using a step-wise discriminant function analysis (see Appendix C for a list of variables). The F value for inclusion and deletion was set at the .10 level of significance (2.88 F).

The remaining minor hypotheses, involving logical subsets described in Chapter III, were also evaluated using the step-wise discriminant function analyses (see Appendix C for a list of the variables). The F value for inclusion was set at .001 and the F for deletion was set at .0005. These very low F values were employed so all of the remaining variables would be included in the discriminant function. These step-wise function analyses were done

primarily to determine the extent of correct classification or hit rates resulting from discriminant function, including all the remaining variables.

Factor analytic techniques were employed in a second phase of the data analysis. This phase of the analysis began with the computation of a product-moment correlation matrix giving the intercorrelations of 49 variables. The product-moment correlation matrix included all variables except for the Impairment Indices and variables with no variation in scores or identical scores for each group on the variables. These correlations are given in Appendix G.

In the application of factor analytic procedures, the correlation matrix was searched to find a highly correlated cluster of Halstead-Reitan variables. In two cases where variables were very highly correlated with each other (the Percent in the Impaired Range and Average Impairment Index variables, and the WRAT spelling and reading variables), one of each pair of highly correlated variables was chosen for the cluster to be factor analyzed. The cluster selected on this basis was made up of four variables: Percent in the Impaired Range, Speech Sounds Perception Test errors, Tapping Speed dominant hand, and WRAT spelling.

In order to determine if there was one underlying factor in the Halstead-Reitan Neuropsychological Test Battery which could differentiate between the Ritalin therapy group and the non-Ritalin therapy group, a centroid component was extracted from the cluster of four variables (Cooley and Lohnes, 1971, p. 138f, and Overall and Klett, 1972, p. 94f). The centroid component for the four variable cluster is the single component of the four variables which extracts

nearly maximal variance from the group. This component or "factor" was used to compute the component loadings for all variables and component scores for all subjects.

Since the component scores represent an approximately maximum amount of the systematic variance in the Halstead-Reitan variables, these scores were used to test the hypothesis of no mean differences between the Ritalin and non-Ritalin therapy groups. That is, a test of differences was done on the factor loadings for the two groups. The test employed was an F test.

CHAPTER IV

RESULTS

The results of the discriminant function analyses and the factor analysis are presented separately. Two approaches were used in the examination of the results from the discriminant function analyses. First, the initial F values generated for the first step of the step-wise discriminant function analyses were examined. These initial F values were developed to determine which variables, when considered separately, significantly differentiated the groups being compared. Thus, these initial F values were the result of a series of one-way analyses of variance. These initial F values were examined in conjunction with each original hypothesis in turn.

Second, the intermediate and end results of some of the step-wise discriminant function analyses were examined. Since the subset of variables for each discriminant function analysis was derived from one of the original hypotheses, the intermediate and end results of each discriminant function analysis were examined in conjunction with the appropriate original hypothesis.

Initial F Values

The major hypothesis was weakly supported. The hypothesis stated that the Ritalin treated children would be less impaired on the measures taken by the Halstead-Reitan Neuropsychological Test

Battery. Twelve of the approximately 60 (12 indices and 49 raw score variables) significantly differentiated the two groups. Two variables significantly differentiated the groups at the. .05 level. They were Percent in the Impaired Range and Perceptual Disorders Impairment Index. Ten variables reached the .10 level of significance. The ten variables which differentiated the groups at the .10 level were WISC Arithmetic; Average Impairment rating; Speech Sounds Perception Test; Trail Making Part B, time; Tapping Speed right hand; Trail Making Part B, Impairment Index; WRAT Spelling, WRAT Arithmetic; Fingertip Number Writing, left hand; and Finger Agnosia left hand. All of these differences were in the direction hypothesized. The Ritalin treated group showed less impairment in functioning than the non-Ritalin treated group. The means and standard deviations for the experimental and control groups, and the F values for the differences between groups, are presented in Appendix H.

Minor hypothesis (1) was weakly supported. The hypothesis stated that clinically selected tests would be used to differentiate the groups, and that the Ritalin treated children would show less impairment than the non-Ritalin treated children on the tests. One measure, Percent in the Impaired Range, significantly differentiated the groups at the .05 level. Six of the ten tests showed a difference significant at the .10 level. They were: WISC Arithmetic; Speech Sounds Perception Test; Finger Agnosia, left hand; Fingertip Number Writing, left hand; WRAT Reading and WRAT Arithmetic. Only the WISC Digit Symbol, mixed eye-hand Dominance, and Tactual Performance Test total time were not significant at the .10 level

or better. All significant differences were in the direction hypothesized.

Minor hypothesis (2) was not supported. The hypothesis stated that the Ritalin treated children would be less impaired on tests sensitive to spatial-relations deficits or right hemisphere dysfunction than non-Ritalin treated children. Only three of the ten tests showed a difference which was significant at the .10 level. No tests in this group reached the .05 level of significance. The experimental group was slightly superior to the controls in performance on the WRAT Arithmetic; Finger Agnosia, left hand; and Fingertip Number Writing, left hand ($p < .10$). No differences were found in Tactual Performance Test time, left hand; Grooved Pegboard time, left hand; Tactual Performance Test Location; Tapping Speed, left hand; Greek Cross scores; WISC Performance IQ, and Trail Making Test Part A.

Minor hypothesis (3) was not supported. The hypothesis stated that performance on tests sensitive to verbal-language deficits or left hemisphere functioning would differentiate the groups in favor of the Ritalin treated children. Only five of the eleven measures differentiated the experimentals from the controls (in the favor of the former) at the .10 level of significance. No tests in this group reached the .05 level of significance. The Ritalin treated children as a group had fewer Speech Sounds Perception errors; they performed faster on Trail Making Test Part B; Tapping Speed, right hand was better; and they were better on WRAT Reading and WRAT Spelling. There was no difference between the groups on the other

left hemisphere measures: WISC Verbal IQ, "H" Words; Tactual Performance Test time, right hand; Aphasia Screening Test scores, and Grooved Pegboard time, right hand.

Minor hypothesis (4) was not supported. The hypothesis stated that the Ritalin treated children would perform better than the non-Ritalin treated children on measures of perceptual, perceptual-motor, and motor functioning. Only Finger Agnosia errors, left hand; Fingertip Number Writing errors, left hand; and Tapping Speed, right hand, reached the .10 level of significance. No significant differences were found in the Suppressions, right hand; Suppressions, left hand; Suppressions, right face; Suppressions, left face; Tapping Speed, left hand; Finger Agnosia, right hand; and Fingertip Number Writing, right hand.

Minor hypothesis (5) was not supported. The hypothesis stated that the Ritalin treated children would be less impaired on tests measuring attentional capacity and concentration than the non-Ritalin treated children. The only significant difference between the groups on these measures was the WISC Arithmetic. Performance of the groups did not differ significantly on the following tests measuring attentional capacity and concentration: Halstead Category Test, WISC Digit Span, WISC Digit Symbol, Seashore Rhythm Test, or total errors for Finger Agnosia, Fingertip Number Writing or Suppressions for face and hands.

Minor hypothesis (6) was weakly supported. The hypothesis stated that the Ritalin treated children would show less impairment in functioning as measured with the fourteen indexed measures of

impairment than the non-Ritalin treated children. The Ritalin treated children had a lower Percent in the Impaired Range ($p < .05$) and a lower Average Impairment Rating ($p < .10$). These two measures are the summary of the total scores on the Impairment Indices. The Ritalin treated children also did better on the Perceptual Disorders Index ($p < .05$) and Trail Making Test, Part B Index ($p < .10$).

Discriminant Functions

The major hypothesis was examined with the step-wise discriminant function analysis by using ten of the best discriminator variables as determined by the F tests. Several variables were deleted because they were extremely highly correlated with other and better predictors. The variables included were: WISC Arithmetic; Percent in the Impaired Range; Speech Sounds Perception Test errors; Trail Making Test, Part B time; Trail Making Test, Part B; Impairment Index; Tapping Speed, right hand; WRAT Arithmetic; Perceptual Disorders; Impairment Index; Finger Agnosia, left hand; and Fingertip Number Writing, left hand. For this analysis an F value equal to 2.88 ($p < .10$) was required for entry into the step-wise discriminant function analysis. Only one variable had the required F value. The variable entered was Perceptual Disorders, Impairment Index. The correct assignment of cases was five control cases and thirteen experimental cases for a 60% accurate assignment.

In the initial examination of the minor hypothesis by the step-wise discriminant function analyses, several of the analyses were found to contain too few significant variables to warrant further data processing. The hypotheses eliminated were hypothesis (2)

items sensitive to spatial-relations deficits or right hemisphere functioning, hypothesis (4) perceptual, perceptual-motor and motor tasks, hypothesis (5) items measuring attentional and concentration abilities. The other three minor hypotheses were analyzed using only those variables which had an initial F value which was significant at the .10 level or better (F = 2.88).

Minor hypothesis (1) was examined, the subset of predictor variables for the ten best discriminating tests were determined using the step-wise discriminant function analysis. The F value of 2.88 to enter was required. The variables included were: WISC Arithmetic and Digit Span; eye-hand mixed Dominance; Percent in the Impaired Range; Tactual Performance Test time, both hands; Speech Sounds Perception; Finger Agnosia, left hand; Fingertip Number Writing, left hand; and WRAT Spelling and WRAT Arithmetic. The percent in the Impaired Range was entered into the function on the first step. After step one no variables attained the required F value. The hit rate for correct predictions was 10 control subjects and 11 experimental subjects or 70% correct group placement using only the Percent of scores in the Impaired Range. The division was made at the 50% in the Impaired Range line. All subjects with 50% or fewer scores in the impaired range were classified as Experimental subjects and all above as Control subjects. There was a 30% overlap of the two groups. The result was a significant separation of the groups ($p \leq .05$) which was in favor of the Ritalin treated children.

Minor hypothesis (3) which predicted that the experimentals would perform better than the controls on tests sensitive to left

hemisphere functioning was examined. The step-wise discriminant function analysis evaluated this hypothesis using only Speech Sounds Perception errors, Trail Making Test Part B, and Tapping Speed, right hand. All these variables were included and they were entered in the order listed. Nine control cases were correctly classified , 6 misclassified, and 12 experimental subjects were correctly placed and 3 misclassified. The total misclassification was 30% but the number of misclassifications was greater for the control group. Forty percent of the controls were misclassified and 20% of the experimental group subjects.

TABLE I

STEP-WISE DISCRIMINANT FUNCTION ANALYSIS FOR TESTS SENSITIVE TO LEFT HEMISPHERE DYSFUNCTION

Variables	F Values, Initial	F Values, at Inclusion
Speech Sounds Errors	4.16	4.16
Trails B	3.07	1.57
Tapping Speed, Dominance	3.27	0.31

Minor hypothesis (6) that impairment indices would separate the two groups was subjected to the step-wise discriminant function analysis. The variables used were Perceptual Disorders, Trail

Making Test Part B, and Percent of scores in the Impaired Range. The variables were entered in the order listed. The F value for entry was 2.88. This subset of predictor variables produced a correct classification in nine control cases and eleven experimental cases. This selection of variables resulted in a 67.8% correct group classification.

TABLE II

STEP-WISE DISCRIMINANT FUNCTION ANALYSIS
FOR ITEMS FROM THE IMPAIRMENT INDEX

Variables	F Value, Initial	F Value, at Inclusion
Perceptual Disorders	5.65	5.65
Trails B	3.18	1.52
Percent in Inpaired Range	5.39	0.26

Factor Analysis

The centroid component loadings obtained from the factor analytic techniques described in Chapter III are given in Table III. Each of these loadings is the product moment correlation of one of the original Halstead-Reitan variables with the component score. The highest loadings, of course, are for the four variables in the

TABLE III

LOADING FACTORS

Variable	Loading	Variable	Loading
WISC Information	-60	TPT Memory	-48
WISC Comprehension	-29	TPT Location	-52
WISC Arithmetic	-58	Speech Sounds Perception	84
WISC Similarities	-43	Rhythm Errors	48
WISC Digit Span	-14	Trails A Seconds	51
WISC Vocabulary	-61	Trails B Seconds	48
WISC Digit Symbol	-50	Trails B Errors	46
WISC Picture Completion	-34	Finger Tapping, right	-84
WISC Block Design	-57	Finger Tapping, left	-60
WISC Picture Arrangement	-22	Hand Suppression, right	-22
WISC Object Assembly	-46	Hand Suppression, left	-60
WISC Verbal IQ	-68	Face Suppression, right	13
WISC Performance IQ	-62	Face Suppression, left	8
WISC Full Scale IQ	-71	Finger Agnosia, right	30
"H" Words	-72	Finger Agnosia, left	44
Mixed-Dominance, eye-hand	24	Fingertip Writing, right	24
Age	-39	Fingertip Writing, left	46
% Impaired Scores	87	Aphasia	24
Average Rating	87	Greek Crosses	31
Grade	56	Pegboard time, right	49
Category errors	16	Pegboard time, left	40
TPT time, right	11	WRAT Reading	-86
TPT, left	36	WRAT Spelling	-85
TPT time, both	1	WRAT Arithmetic	-76
TPT time, total	34		

TABLE IV

FACTOR SCORES IN STANDARD SCORE FORM

Controls		Experimentals	
1.	1.653	16.	.716
2.	.199	17.	-2.145
3.	1.186	18.	.208
4.	1.099	19.	.243
5.	-.198	20.	-1.871
6.	.680	21.	1.231
7.	.431	22.	-.425
8.	.232	23.	-1.802
9.	-.296	24.	-1.522
10.	.191	25.	-.213
11.	2.031	26.	1.041
12.	-.833	27.	-.059
13.	-.277	28.	-.985
14.	-.302	29.	-.514
15.	.453	30.	-.152

cluster which was used to extract the component (and those variables which were excluded from the cluster because of their extremely high correlations with the variables wtihin the cluster). Other high loadings are for WRAT Arithmetic, "H" words, and WISC Full Scale IQ. Thirty loadings are .40 or greater with the lowest of these being for Peg Board time for the left hand.

The centroid component scores are given in Table IV in standard score form. For the sake of convenience only, control and experimental subjects are aligned in the rows of this table. If these scores were used to classify the subjects with a cutting score of zero, the result would be ten correct classifications for each group or 67% correct placement.

TABLE V

ANALYSIS OF VARIANCE TABLE
FACTOR LOADING
SCORES

	Sum of Squares	df	Mean Square	F Ratio	Critical F (5% Level)
Between Groups	5.206	1	5.206	4.89	4.20
Within Groups	29.252	28	1.045		
Total	34.458	29			

The results of the F test used to test the hypothesis of no mean difference in the component scores from the two groups are shown in Table V. In this case the value for F is ($p \leq .05$). Thus, the hypothesis of no mean difference is rejected.

CHAPTER V

DISCUSSION

The LD-MBD child has long been considered to be experiencing learning difficulties due to mild brain damage or dysfunction (Clements, 1968, Werry, 1968, Wender, 1962 and others). The present study supported the theories of the previously mentioned investigators in their hypothesis of the existence of some type of neuropsychological deficit in the LD-MBD child. The large majority of the children in the present study, both experimental and control, showed a pattern and a level of performance unlike the neurologically normal child. The neurologically intact child has an average on the Impairment Index which is very close to 1.00 with few test performances, if any, in the impaired range (2 or higher) on the Halstead-Reitan Neuropsychological Test Battery. Clinicians using the Halstead-Reitan Neuropsychological Test Battery with a level of performance approach generally accept an average of 1.50 on the Impairment Index as the cut-off point between a non-impaired and an impaired level of performance (Russell, Neuringer, and Goldstein, 1970). Twenty-two of the 30 children in the study had an Average Impairment Index at or above 1.50. The Average Impairment Index mean for the combined groups was 1.64. This was not an exceptionally high Average Impairment Index but it was at a level at which one would expect the child to be encountering mild adaptive difficulties.

Similar to the Average Impairment Index, the absolute number of test performances in the impaired range is taken as a global indicator of degree of impairment. Four or fewer scores in the impaired range (0% to 33%) is not neurologically significant in most cases but at the other extreme 9 to 12 (67% to 100%) scores in the impaired range strongly suggests cerebral pathology. The average percent of scores in the impaired range was 51.5% for the combined groups. This finding places the LD-MBD children in this study mid-way between neurologically normal and neurologically damaged children. The performance shown by the LD-MBD children in the present study is in essential agreement with the findings of Klonoff and Low (1974) and Rourke and Boll (1973). Thus the hypothesis that the LD-MBD syndrome is associated with a "minimal brain damage or dysfunction" which interferes with the child's performance in many subtle ways has been supported by this research.

Many investigators have found that Ritalin produces behavior changes in LD-MBD children which appear to make them more tractable to the learning process (Eisenberg, Conners and Sharpe, 1965; Lytton, 1958; Cromwell, 1963; and others). These authors observed gross changes in behavior of a positive nature rather than specific changes in neuropsychological functioning of the LD-MBD children they studied. The present research did not examine the exact mechanism of the dysfunction or the location of the dysfunction or cerebral abnormality, but various aspects of the data do indicate areas in which Ritalin positively influenced neuropsychological functioning. On twelve performance measures the drug treated children were superior to the controls at a significance level of .10 or better. Two of these

twelve differences reached the .05 level of significance or better. The number and magnitude of the differences found were clearly not overwhelming, but the trend is quite strong and all differences were in the predicted direction, that is, superior functioning of the drug treated children. The data suggest a slight reduction of neuropsychological deficits in the drug treated children as well as the gross behavior changes noted by others.

The experimental and control groups could be differentiated by using one measure, the Percent in the Impaired Range. The Percent in the Impaired Range is a measure taken from the number of the twelve major measures which showed at least a mild degree of impairment in adaptation. An analysis of the data using the discriminant function analysis showed that 70% of the subjects could be correctly classified for group membership with that one variable, Percent in the Impaired Range. This variable was found to be the best single predictor of group membership and the number of correct classifications was not improved by the introduction of other variables, even in combination. Ritalin appeared to have a general facilitative effect on a wide range of neuropsychological abilities. The present data will not allow more specific statements as to the exact changes produced. Further studies with larger sample sizes or a more homogeneous LD-MBD population might, however, increase the magnitude of the differences found in the present study and allow more specified designation of areas of improvement associated with Ritalin treatment.

The general nature of the improvement in neuropsychological functioning found in the present study was underscored by the factor analysis of the data. This factor analysis of the level of

performance scores on 49 variables further supported the hypothesis that Ritalin therapy was related to performance differences in favor of the experimental children. A central component score was extracted from the intercorrelation of the test performance of all children in the study on the above mentioned variables. This component score was derived from four variables. These variables were the four most highly correlated non-overlapping scores from the battery of tests: Percent in the Impaired Range, Speech Sounds Perception Test errors, Tapping Speed with the right hand, and WRAT Spelling. The central component score can be said to represent a common factor for all of the tests and one might call it a "cerebral efficiency" index. This factor contains the largest share of the variance with regard to the test performance differences of the two groups. The cerebral efficiency factor appears to be a common element in the performance of the children on a wide range of tasks assessing a wide variety of abilities.

The cerebral efficiency index generated correlations of .40 or more with 30 of the 49 Halstead-Reitan variables (Appendix G). By itself the cerebral efficiency index was found to significantly differentiate between the experimental and control groups ($F = 4.89$, $p \leq .05$). An examination of the initial F values from the step-wise discriminant function analysis had indicated that there was some central factor which was influenced by the experimental treatment, and which was very highly correlated with Percent in the Impaired Index among others. Moreover, the failure of the step-wise discriminant function analyses to include more than one variable, when

the F value for inclusion was set at .10 level of significance, was itself due to the high intercorrelation of the first entered variable with the remaining ones.

The literature referred to earlier, as well as the results of the present study, suggests that Ritalin does have a beneficial effect on LD-MBD children but the mechanism or site of its action is not clear. Fredericks (1974) believed that the RAS played a major role in the LD-MBD child's problems and he further believed that amphetamine type drugs have specific effects on the RAS. He hypothesized that the drug's effect on the RAS would reduce the child's attentional weaknesses, distractibility, over-activity, irritability, impulsiveness, low frustration and impaired learning. If Frederick's theory of RAS functioning and LD were correct, specific attentional deficits and distractability would be improved by Ritalin therapy. The data from the present study showed deficits of attention and concentration in both groups of these LD-MBD children which were in agreement with the theory of RAS involvement. However, the Ritalin treated children were no less impaired on these abilities than the non-treated children in spite of other significant differences in favor of the Ritalin treated group. Furthermore, the present study showed areas of specific deficits in functioning which cannot be accounted for by the RAS theory of LD-MBD. The present study suggests an involvement of the cerebral cortex as one of the sites of the problem.

Deficits which are associated with known cerebral lesions were found in the test performances of the LD-MBD children in the present study. The means for the combined groups were in the impaired

range (above 1.50 in the Impairment Index) on the following tests: Tactual Performance Tests time, Tactual Performance Test location, Tapping Speed right hand, Spatial Relations, Perceptual Disorders, Aphasia Screening Test and Category Test. Deficits on the Category Test have been found to be associated with diffuse, generalized, or significant localized lesions in both children (Reed, Reitan & Kl ve, 1966, among others) and adults (Reitan, 1955, and others). Deficits on the remaining six tests named have been found to be associated with localized or generalized lesions which involve specific areas of the cortex. No deficits have been found due to subcortical lesions on any of the named tests other than Tapping Speed right hand and Tactual Performance Test time. Tapping Speed right hand and Tactual Performance Test time are dependent, in part, on motor speed and coordination which can be impaired by subcortical lesions as well as cortical lesions.

The experimental or Ritalin treated children were significantly superior to the control group on the following tests: Speech Sounds Perception errors, Seashore Rhythm Test, Trail Making Test Part B, Tapping Speed right hand and Perceptual Disorders. Performance on each of the five tests named above is known to be dependent at least in part on cortical integrity. The data indicates that the cortex is involved in both the areas of significant deficits in neuro-psycological functioning and in differences in neuropsychological functioning related to Ritalin therapy. The data does not exclude attentional deficits dependent upon subcortical dysfunction.

Attentional deficits have been seen by many authors as primary primary to the LD-MBD syndrome, but the data from the present study

suggests that the problem is much more complex than that. Studies by Segundo, Arana-Inignty, and French (1955) and Segundo, Nagnet, and Buser (1955) indicated a possible mechanism for cortical involvement. The above studies demonstrated the intimate relationship between the cortex and the RAS. These investigations showed that the cortex has many areas which function to arouse the organism, through feedback loops between the cortex and the RAS. Thus, it may be that if the RAS is involved in the LD-MBD syndrome it is through involvement of the cortex. Abnormal or impaired cortical structures may disrupt subcortical control mechanisms, which lead to adaptive problems for the individual which are partially remediated by Ritalin. But deficits are not limited to attentional and concentration deficits, instead a wide range of neuropsychological functions appear to be involved. These neuropsychological functions depend upon cortical integrity, not subcortical structural integrity, and the present study suggests that Ritalin therapy modifies the degree of deficit in many of the neuropsychological functions. Therefore, the RAS theory of LD-MBD dysfunction appears to be too limited and perhaps more adequate theories which include the functioning of the cortex can be developed as more facts and data are collected.

Of additional interest in the present study was the fact that the two groups of children were significantly different in their academic skills, on reading and spelling (after correction for grade placement) as measured by the WRAT. In reading, the Ritalin treated children scored .53 years (10%) above grade placement while the non-treated control children scored .34 years (8%) below grade placement. In spelling, the Ritalin treated group scored .57

years (11%) below grade placement while the non-treated group scored 1.19 years (26%) below grade placement. The differences were not found for arithmetic. The Ritalin group scored 1.05 years (20%) below grade placement, which was not significantly different from the 1.02 years (22%) below grade placement for the control group.

To reiterate, the experimentally treated children were significantly better on both reading and spelling, but not on arithmetic. Reading and spelling are skills which require adequate functioning in specific areas of the left hemisphere. Arithmetic skills which did not differentiate the groups have been found in factor analysis of the WISC scales to loan on attentional factors (van Hagen, & Kaufman, A.S., 1975). The reader will recall that the experimental children were also superior to the controls on the Speech Sounds Perception Test and Finger Tapping right hand, both of which are again like reading and spelling mediated by the left cerebral hemisphere . The following must be taken very tentatively but Ritalin may have a more facilitating neuro-psychological effect on the cerebral hemispheres--and their functions--than the subcortical structures.

Methodological problems common to many studies in this field were found in the present study and these may have reduced the magnitude of the performance differences between the two groups. First and most important was the lack of rigor in the diagnosis of cases. The prime requisite for such cases should be a significant degree of academic retardation but a number of experimental and control children did not meet this criteria according to WRAT scores. Only seven of

the control subjects and eight of the experimental subjects were one-half or more years behind their grade level in reading; three of the control and four of the experimental children were not 10% or more below their grade level in any of the three academic areas. The children without academic retardation appear to have been diagnosed LD-MBD for one of two reasons. The children may never have had actual learning difficulties but may have been diagnosed for reasons other than impaired school performance, or the children who presently show no academic deficit may have experienced those deficits earlier in their school careers and have since remedied them. The last possibility again shows the need for longitudinal studies in this area. However, both cross-sectional and longitudinal studies in LD-MBD should be conducted with very rigorous diagnostic classification.

Precision in diagnosis is important for both research purposes and clinical practice. A single diagnostic designation for such a broad syndrome, with the particular clinical phenotype varying from case to case, may indeed reflect a clinical entity but the syndrome and clinical experience suggest the need to derive major clinical subtypes to better classify the phenotypic variance. For example, a few of the children in the present study were not academically retarded, yet they probably had other characteristics of the syndrome. To continue along this line, a number of LD-MBD children are hyperactive but others are not. A better classification system would hopefully lead to better prediction about drug response in specific cases or prediction of benefit from particular remedial measures.

One needs to know what types of LD-MBD children will respond favorably to Ritalin and which will not. The same holds true for predicting response to non-drug remedial training for LD-MBD children .

The findings of the present study are consistent with the generally held belief that Ritalin is effective in the treatment of some LD-MBD children. The drug, however, is typically used in a shotgun approach, helping many but not benefiting others. There is a need to learn the factors involved in the good and poor responses to the drug. The greatest need, however, is to discover more about the relevant variables and factors involved in the LD-MBD syndrome. Increased knowledge of the process and the factors involved will hold more potential benefit for these disabled children than anything else, including chemical treatment.

CHAPTER VI

SUMMARY

This study was designed to assess the effects of Ritalin therapy on the neuropsychological functioning of LD-MBD children. Empirically, Ritalin seems to have a beneficial effect on the behavior and learning receptivity of many of these children, but the neurophysiological data for these positive drug effects have been lacking. The literature suggested that there was some type of neuropsychological deficit, probably of the RAS, in these children which was altered by the drug. The implication from the literature was that the neurophysiological effects of the drug alter the basic neuropsychological functioning in LD-MBD children. To test the general hypothesis of drug induced positive changes in neuropsychological status, 15 non-drug treated LD-MBD children and 15 Ritalin treated LD-MBD children were compared in their test performance on the Halstead-Reitan Neuropsychological test battery for older children.

The theory that LD-MBD children were experiencing a neuropsychological deficit was supported. The LD-MBD children as a group were more impaired in neuropsychological functioning than normal children. They were also less impaired in their performance than children with documented brain damage.

The theory that LD-MBD dysfunction is due to attentional deficits which resulted from RAS inadequacies was not supported. No differences

were found between the two groups on attentional deficits. However, higher neuropsychological functions dependent upon cortical integrity were found to be more impaired in the LD-MBD children than non-LD-MBD children.

The clinical observation that Ritalin improved neuropsychological functioning was supported by the research data. There were significant differences between the Ritalin treated and non-treated LD-MBD children. The Ritalin treated LD-MBD children were found to be less impaired in test performance than the non-treated children in all cases where there were significant differences.

The present study cannot be considered definitive because of small number of subjects and subject heterogeneity, but the data is suggestive. Future studies utilizing larger sample size, more homogeneous groups or longitudinal studies with each child as his own control should further clarify the etiology of LD-MBD and the effect and mechanism of treatment with stimulant drugs such as Ritalin.

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APPENDIX A

DEFINITIONS OF TERMS USED TO DENOTE LD-MBD LIKE DISORDERS

Definitions of Terms Used to Denote
LD-MBD Like Disorders

Bateman, B. An educator's view of a diagnostic approach to learning disorders. Learning Disorders, Volume I. Seattle, Washington: Seattle Sequin School, 1965 (p. 220).

"Children who have learning disorders are those who manifest an educationally significant discrepancy between their estimated intellectual potential and the actual level of performance related to basic disorders in the learning processes, which may or may not be accompanied by demonstrable central nervous system dysfunction, and which are not secondary to generalized disturbance or sensory loss."

Clements, S. D., Project Director. Task Force I: Minimal Brain Dysfunction in Children, National Institute of Neurological Diseases and Blindness, Monograph No. 3, U.S. Department of Health, Education, and Welfare, 1966 (pp. 9-10).

"The term 'minimal brain dysfunction syndrome' refers in this paper to children of near average, average, or above average general intelligence with certain learning or behavioral disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system. These deviations may manifest themselves by various combinations of impairment in perception, conceptualization, language, memory, and control of attention, impulse, or motor function."

"Similar symptoms may or may not complicate the problems of children with cerebral palsy, epilepsy, mental retardation, blindness or deafness."

"These aberrations may arise from genetic variations, biochemical irregularities, perinatal brain insults or other illness or injuries sustained during the years which are critical for the development and maturation of the central nervous system, or from unknown causes."

Kass, Corrine. Conference on Learning Disabilities. Lawrence, Kansas: November, 1966.

"A child with learning disabilities is one with significant intradevelopmental discrepancies in central-motor, central-perceptual, or central-cognitive processes which lead to failure in behavioral reactions in language, reading, writing, spelling, arithmetic, and/or content subjects."

"The hyperactive behavior pattern which has been described is customarily the combination of developmental hyperactivity and associated major features: inattentiveness, a learning or perceptual-cognitive disability, a conduct problem, and immaturity. However, each of the major features of HA may occur without HA and, in fact, HA may occur without any of the major features of the pattern." (p. 10).

Wender, M.D. Minimal Brain Dysfunction in Children. New York: Wiley-Interscience, a Division of John Wiley & Sons, Inc., 1971 (p. 12).

"MBD children manifest dysfunction in the following areas: motor activity and coordination; attention and cognitive function; impulse control; interpersonal relations, particularly dependence-independence and responsiveness to social influence; and emotionality."

First Annual Report, National Advisory Committee on Handicapped Children, January 31, 1968. "Special Education for Handicapped Children, Toward Fulfillment of the Nation's Commitment..."

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

Learning Disabilities Division Formulational Meeting, National Council on Exceptional Children (C.E.C.), St. Louis, Missouri, April 1967.

"A child with learning disabilities is one with adequate mental abilities, sensory processes and emotional stability who has a limited number of specific deficits in perceptive, intergrative, or expressive processes which severely impair learning efficiency. This includes children who have central nervous system dysfunction which is expressed primarily in impaired learning efficiency."

Kirk, S. A. Educating Exceptional Children. Boston: Houghton Millin Co., 1962 (p. 261).

"A learning disability refers to a retardation, disorder, or delayed development in one or more of the processes of speech, language, reading, spelling, writing, or arithmetic resulting from a possible cerebral dysfunction and/or emotional or behavioral disturbance and not from mental retardation, sensory deprivation, or cultural or instructional factors."

Kirk, S. A. The Diagnosis and Remediation of Psycholinguistic Abilities. Institute for Research on Exceptional Children, University of Illinois, 1966 (pp. 1-2).

"A learning disability refers to a specific retardation or disorder in one or more of the processes of speech, language, perception, behavior, reading, spelling, or arithmetic."

Mylkebust, H. R. Psychoneurological learning disorders in children. In S.A. Kirk and W. Becker (Eds.), Conference on Children With Minimal Brain Impairment. Urbana, Illinois: University of Illinois, 1963 (p. 27).

"...we use the term 'psychoneurological learning disorders' to include deficits in learning, at any age, which are caused by deviations in the central nervous system and which are not due to mental deficiency, sensory impairment, or psychogenicity. The etiology might be disease and accident, or it might be developmental."

Safer, D. J. and Allen, R. P. Hyperactive Children: Diagnosis and Management. Baltimore: University Park Press, 1976.

"A certain amount of the confusion regarding hyperactivity stems directly from problems inherent in the terminology. In diagnoses, two terms are commonly used, often interchangeably. The first diagnostic term is "hyperactivity" itself. It is used synonomously with the more sophisticated medical label, hyperkinetic behavior pattern. Hyperactivity (HA) is simply defined as a long term childhood pattern characterized by excessive restlessness and inattentiveness. It is a developmental disorder which begins in early to mid-childhood (ages 2-6), and begins to fade during adolescence. During childhood, the pattern is consistent year after year, i.e., it is not observed for one year but absent the next two."

"The second diagnostic term that pertains to hyperactivity is 'minimal brain dysfunction.' It is known commonly by its initials, MBD. MBD is generally based on a learning or perceptual impairment, usually associated with hyperactivity and inattentiveness. A behavioral difficulty is sometimes added as a diagnostic feature of MBD." (pp. 6-7)

APPENDIX B

HALSTEAD-REITAN NEUROPSYCHOLOGICAL TEST BATTERY

The Halstead Category Test

The Halstead Category Test has been modified by Reitan for both older and younger children. This experiment used the modification for older children (Reitan and Heineman, 1963).

The Category Test is a complex test used to measure the subject's ability to identify the concept or principle that governs a series of numerical figures or geometric materials. A projection apparatus is used for the presentation of the one-hundred-and-seventy stimulus figures on a milk glass screen. There is an answer panel which consists of four levers numbered from one to four. This panel is attached to the above mentioned apparatus which is placed before the subject.

In the instructions the subject is told that he should depress one of the four buttons for each of the pictures which appear on the screen. The depression of any of these levers will cause either a bell or a buzzer to sound. The sound emitted depends on whether the lever selected is the "right" one, signified by a chime, or a "wrong" response signified by a buzzer. There is only one correct response for each stimulus displayed and only one response is allowed for each item. The subject is told that the test consists of a number of groups of pictures and that each group will have a single principle running through the entire group from the beginning to the end. His task is to determine that principle. The Examiner announces the end and the beginning of each group. The Examiner also points out that the principle in the new group may be the same or it may be different from that of the preceding group and it is the subject's task to try to determine the correct response based on that principle.

On the first item of any group a subject's only option is to guess, but as he progresses through the group the sound of the bell or the buzzer with each response indicates whether his guess was correct or incorrect. In this way, the subject may test one possible hypothesis after another until the correct one is discovered, which leads to consistent positive reinforcement by the bell. The subject is never told the principle for any group, regardless of the difficulty he encounters; even severely brain damaged children have no difficulty learning the correct principle in the first two groups. The first series requires only the matching of Arabic numerals with the Roman numeral which is shown on the screen. In the second series the subject must learn to respond with the lever which has a number corresponding to the number of items appearing on the screen, regardless of the content. The concept in the third group of items is based on the principle of uniqueness; four figures appear in each item and the subject must learn to depress the lever corresponding to the figure which is most different from the others. Although this group begins rather simply, it progresses to items in which one figure may differ from the other in three or more respects. The rest of the figures differ from each other in only two respects. There are two additional principles or concepts which are used in the next three series but the last series is a summary or review of the preceding ones, and it is the subject's task to remember the previously learned concepts.

The total number of errors made on all subtests of the Category Test provides the score on this test.

The Tactual Performance Test

The Tactual Performance Test contributes the next three items to the Halstead Impairment Index and it is a modification of the Sequin-Goddard formboard. In this modification the subject is never allowed to see the formboard or the blocks. He is blindfolded before, during and immediately after performing the task and the board is removed from his sight before the blindfold is removed. His task is to fit the blocks into the proper locations on the board, first using only the tactile sense of his preferred hand. The time it takes the subject to perform this task is recorded. After this trial, he is instructed to perform the same task using his non-preferred hand, following which he performs the task a third time using both hands. The time required to complete the task is recorded for each trial. Only the sume of the time for the three trials is used to determine the Total Time which provides another part of the Halstead Impairment Index; however, in analyzing the data the relative time on each trial is noted to provide a comparison of the efficiency of the two sides of the body.

After the third trial, the board is removed from the subject's view before the blindfold is removed. After the blindfold is removed the subject is asked to draw a diagram of the board representing the blocks in their proper location. From this drawing two other components of the Halstead Impairment Index are obtained: the Memory component, which is based on the number of block shapes correctly reproduced in the drawing, and the Location component, which is based on the number of blocks which are correctly located. The formboard is also a measure of several complex abilities. Components which are

tapped are tactile form discrimination, kinesthetic feedback, coordination in the movement of the upper extremities, manual dexterity, and visualization of spatial configurations, both in terms of their shape and in terms of their relative position.

The Sound Perception Test

This test consists of sixty spoken nonsense words. An audio-tape of the presentation is played to the subject who must listed to the spoken nonsense syllable, then underline the correct alternative from three possibilities typed on the test form. This test of course requires the maintenance of attention throughout the sixty items, which constitute the test, but it also requires the ability to perceive the spoken stimulus sound through the auditory channel, the ability to perceive the visual form correctly, and to analyze them into the sound which they represent. The subject must then relate the sound perceptions to the correct configuration of letters on the test form. This task is a measure of audio-visual association, and it requires auditory and visual discriminatory abilities.

The Rhythm Test

The Rhythm Test is a subtest taken from the Seashore Test of Musical Talent. The task requires the subject to differentiate the similarity or difference between members of thirty paired rhythmic beats. The subject records his judgment with an "S" if he perceives the pair to be identical and a "D" if the pair is perceived to be different. The number of errors is recorded and this score is converted into another component of the Halstead Impairment Index. This

task requires sustained attention, alertness, the ability to perceive different rhythmic sequences, and the ability to attend to temporal sequencing and to remember that sequence.

Finger Tapping Speed Test

This task assesses the average number of taps which the subject can make in five consecutive ten-second trials and is a measure of motor speed. During these trials the hand is held in a constant position and no movement is allowed in the hand or in the arm. Only the index finger is allowed to move in depressing the key. The trials are performed in five or more consecutive ten-second trials which are separated by fifteen-second rest intervals. The subject is encouraged to tap as rapidly as he can. First the dominant hand is evaluated, and then the test is repeated. The number of taps with the non-dominant hand is obtained in the repetition.

The tapping speed for the dominant hand is used to determine the Halstead Impairment Index for Tapping.

Trail Making Test (Trails A & B)

This task was obtained from the Trail Making Test for adults which was modified by Reitan for older children. The Trail Making Test consists of both Part A and Part B, with only the score from Part B contributing to the Halstead Impairment Index. Both Parts A and B are used in comparing the relative efficiency of the two hemispheres. In this test the ability of the subject to make rapid searches for specific spatial designs, to identify and to follow

visual patterns which have a conventional meaning is assessed. That is, the subject is required to connect circles which contain specific symbols with a pencil line as rapidly as possible.

In Trails A, the subject is to connect numbers from one through thirteen and his only requirement is to perform this task by connecting them in numerical sequence as rapidly as he can. Trails B is more complex and consists of a like number of circles which contain numbers (one through eight) and letters (A through G). On Trails B, the subject is required to connect the circles but now he must alternate between numbers and letters as he proceeds. The subject goes from number '1' to the letter A, the number '2' to the letter B, etc. The number of seconds required to perform each task, as well as the number of errors made on each part of the Trails test, is recorded and scored.

Digit Symbol

Digit Symbol is a measure obtained by comparing the subject's performance on the Coding (Digit Symbol) subtest of the WISC with the mean of his performance on the Block Design, Picture Arrangement and Object Assembly subtests of the WISC. The score on Digit Symbol is another component of the Average Impairment Index.

Halstead Aphasia Screening Test

This test is a survey of possible aphasias and dyspraxias. The subject is asked to name common objects and shapes, to spell, and to identify individual numbers and letters. He also must read, enunciate,

and understand the spoken word and be able to restate the meaning of what he hears. He is also asked to write, to calculate, to identify body parts, to draw a familiar object from a model, and to differentiate right and left.

A single score from this test also contributes to the Halstead Impairment Index; however, each item is examined to determine the particular type of aphasia or dysphaxia present.

Spatial Relations

No specific test measures this ability. Spatial relations abilities are estimated from the performance of the subject in the drawing of a geometric figure and the drawing of a key from the Aphasia Test combined with the score on the Block Design subtest of the WISC. The spatial relations abilities contribute to the Halstead Impairment Index.

Perceptual Disorders

The Perceptual Disorders test includes measures taken from a battery of measures to assess the tactile, auditory and visual sensoriums. The assessment of perceptual disorders requires careful training and attention to detail for the examiner. He must also be able to elicit the full cooperation of the subject.

Tactile, Auditory and Visual Imperception

This procedure attempts to assess the accuracy of the subject's perception of bilateral simultaneous sensory stimulation. The subject's ability to perceive unilateral stimulation must first be

assessed before bilateral stimulation acuity can be determined. This procedure is used for the tactile, visual, and auditory modalities in separate tests. For tactile functioning, the subject is told to close his eyes; each hand is then touched lightly in random order to determine that the subject can respond accurately. Without notifying the subject that the procedure has changed, simultaneous touching of both hands is interspersed with unilateral stimulation to assess bilateral stimulation perception. The subject may consistently miss one hand, or completely suppress perception of simultaneous touching, or he may suppress perception of each hand about equally. The number of suppressions is recorded. This procedure is repeated for each cheek and the contralateral hand. The number of suppressions is again recorded.

Auditory perception of bilateral stimulation is made using a light but quick rubbing together of the fingers beside each of the subject's ears, using the procedure outlined above.

Visual perception of the simultaneous stimulation is tested using slight movements of the Examiner's fingers at the edge of the subject's visual field. The visual field must be mapped before this procedure is attempted. If irregularities are found, adjustments in the placement of the Examiner's hands are made and the perception is tested for upper, middle and lower visual fields and the number of suppressions is recorded for each area.

Tactile Finger Recognition

This test is an assessment of finger agnosia.

The subject is asked to place both hands on the table before him

with his fingers spread and his palms down. The fingers are usually numbered so that the subject and the Examiner can communicate accurately. Generally the thumb is named "one" and the other fingers are named consecutively toward the fifth finger. Then with his eyes closed, the subject is asked to name the finger which the Examiner has gently touched. Each finger is touched four times in random order, first for the right hand and then the left. The number of errors made is recorded for each hand.

Fingertip Number Writing

This procedure requires the subject to report the number written on his fingertips for each hand while he has his eyes closed. A total of four trials is given for each finger and the score is recorded as the number of errors in twenty trials for each hand.

Average Impairment Index

The raw scores from the preceding ten tests or procedures are converted to the Halstead Index. Then the Indices are averaged to obtain the Average Impairment Index which gives an overall estimate of the severity of the impairment present.

Supplementary Test

The following procedures are used to help determine lateralization and status of any lesion, i.e., hemisphere involvement, acute, progressive, statis, level of intellectual functioning and the like.

The Wechsler Intelligence Scale for Children

The Wechsler Intelligence Scale for Children (WISC) (Wechsler, 1949) is the most widely used individually administered intelligence test given to children of school age. This test consists of six subtests which are heavily loaded in verbak skills: Information, Comprehension, Arithmetic, Similarities, Vocabulary, and Digit Span and six tests which assess abilities which are more dependent upon spatial motor skills. These latter subtests are Picture Completion, Picture Arrangement, Block Design, Object Assembly, Coding and Mazes. The Mazes subtest is not used in the Halstead-Reitan battery and is not given to the children in this study.

The WISC is administered as designated in the WISC Test Manual.

Lateral Dominance

This is an assessment of the "handedness", "footedness" and "eyedness" of the subject. The "handedness" is assessed through observation and recording of the hand used in seven different tasks combined with the time taken for the subject to write his name and an unpracticed word and the relative grip strength for each hand. "Footedness" is determined by observing the foot used on various tasks. "Eyedness" is determined by counting the number of times the subject sights with each eye using the A-B-C Visition test.

Wide Range Achievement Test

The WRAT is administered as the testing manual suggests (Jastak & Jastak, 1969) to measure roughly academic achievement in Reading,

Spelling, and Arithmetic. This is used to determine the concordance between measured intelligence and level of achievement.

Motor Steadiness

The Purdue Peg Board is used to determine motor steadiness under a speed pressure condition. The time taken to complete the task of placing non-circular pegs into corresponding holes is measured for first the dominant, then the non-dominant hand and the number of errors (dropped pegs) is also recorded.

APPENDIX C

LISTING OF TEST MEASURES STATISTICALLY ANALYZED FOR EACH HYPOTHESIS

Listing of Test Measures Statistically Analyzed for Each Hypothesis

Major Hypothesis:

Twelve variables with F significant at $P < .10$ level:

- Percent in the Impaired Range
- Perceptual Disorders Impairment Index
- WISC Arithmetic
- Average Impairment Rating
- Speech Sounds Perception List
- Trail Making Part B, Time
- Tapping Speed, Right Hand
- Trail Making Part B - Impairment Index
- WRAT Spelling
- WRAT Arithmetic
- Fingertip Number Writing, Left Hand
- Finger Agnosia, Left Hand

Minor Hypothesis 1:

Ten clinically selected tests:

- Percent in the Impaired Range
- WISC Arithmetic
- Speech Sounds Perception Test
- WRAT Reading
- WRAT Arithmetic
- Fingertip Number Writing, Left Hand
- Finger Agnosia, Left Hand
- WISC Digit Symbol
- Mixed Eye-Hand Dominance
- Tactual Performance Test, Total Time

Minor Hypothesis 2:

Tests measuring right hemisphere or spatial-relations deficits:

- WRAT Arithmetic
- Fingertip Number Writing, Left Hand
- Tactual Performance Test, Time Left Hand
- Grooved Pegboard, Time Left Hand
- Tactual Performance Test Location
- Tapping Speed, Left Hand
- Greek Cross Scores
- WISC Performance IQ
- Trail Making Test Part A

Minor Hypothesis 3:

Tests measuring left hemisphere or verbal-language deficits:

Speech Sounds Perception Test
Trail Making Test Part B
Tapping Speed, Right Hand
WRAT Reading
WRAT Spelling
WISC Verbal IQ
"H" Words
Tactual Performance Test, Time Right Hand
Aphasia Screening Test Scores
Grooved Pegboard, Time Right Hand

Minor Hypothesis 4:

Tests measuring perceptual, perceptual-motor and motor functioning:

Fingertip Number Writing, Left Hand
Finger Agnosia, Left Hand
Tapping Speed, Right Hand
Suppressions, Right Hand
Suppressions, Left Hand
Suppressions, Left Face
Suppressions, Right Face
Tapping Speed, Left Hand
Finger Agnosia, Right Hand
Fingertip Number Writing, Right Hand

Minor Hypothesis 5:

Tests measuring attentional capacity and concentration:

WISC Arithmetic
WISC Digit Span
WISC Digit Symbol
Halstead Category Test
Seashore Rhythm Test
Finger Agnosia, Total Errors
Fingertip Number Writing, Total Errors
Suppressions, Face, Total Errors
Suppressions, Hands, Total Errors

Minor Hypothesis 6:

The indexed measures of impairment:

Percent in the Impaired Range
Average Impairment Rating
Halstead Category Impairment Index

Tactual Performance Test, Time Impairment Index
Tactual Performance Test, Memory Impairment Index
Tactual Performance Test, Location Impairment Index
Speech Sounds Perception Test, Impairment Index
Seashore Rhythm Test, Impairment Index
Trail Making Test Part B, Impairment Index
Tapping Speed Dominant Hand, Impairment Index
Digit Symbol Rating, Impairment Index
Aphasia Screening Test, Impairment Index
Perceptual Disorders, Impairment Index
Spatial Relations, Impairment Index

APPENDIX D

CALCULATION OF IMPAIRMENT INDICES FOR HALSTEAD-REITAN NEUROPSYCHOLOGICAL TEST BATTERY FOR OLDER CHILDREN

Calculation of Impairment Indices for
Halstead-Reitan Neuropsychological
Test Battery for Older Children

Index	0	1	2	3	4
Category	≤ 20	21-42	43-65	66-89	≥ 90
Tact. Performance	≤ 8.0	8.1-17.0	17.1-23.0	23.1-29.0	≥29.1
Tact. Perf. Memory	10-9-8	7-6-5	4-3	2-1	0
Tact. Perf. Loc.	10-6	5-4	3-2	1	0
Speech Percept.	≤ 4	5-16	17-28	29-36	≥ 37
Rhythm	≤ 2	3-8	9-12	13-14	≥ 15
Trails B	≤25	77-149		150-185	≥186
Tapping					
Male 12-15	≥48	43-47	34-42	26-33	≤25
Male 9-11	≥44	39-43	30-38	22-29	≤21
Female 12-15	≥44	39-43	30-38	22-29	≤21
Female 9-11	≥ 40	35-39	26-34	18-25	≤17

Digit Symbol Index

Compute: $\frac{\text{Average Picture Arrangement + Picture Completion + Block Design}}{3}$

Index	0	1	2	3	4
Avg. DS $\geq$ 12	DS $\geq$ (AV-1)	DS $\geq$ (AV-1)			
DS = 9-11		DS = 9-11			
DS = 8 or 7		DS $\geq$ (Av-1)	DS $\geq$ (Av-1)		
DS = 5 or 6			DS $\geq$ (Av-1)	DS $\geq$ (Av-1)	
DS = 3 or 4				DS $\geq$ (Av-1)	DS $\geq$ (Av-1)
DS $\leq$ 2					DS = 2

APPENDIX E

LETTER TO PARENTS

Dear Parent,

I am conducting a dissertation study to investigate the way the drug Ritalin changes learning in the Learning Disabled child. We have evidence which shows that Ritalin helps many L-D children reduce their activity level, but we do not yet know how Ritalin changes the child's ability to learn. This is what my study will investigate.

I need pupils who are nine, ten, or eleven years old and who are now taking a prescribed dosage of Ritalin to participate in this study. I will be giving the children a series of psychological tests, which many find fun to perform, in order to see how Ritalin changes their functioning on these tests. These tests will be given at my husband's office, 700 North Topeka.

I will do a clinical evaluation of the test data and give the parents full results, and I will also send my findings to the child's teacher so that they may gain knowledge about your child. Many parents and teachers have found this data useful and we hope that it will help you and your child's teacher to understand your child better, as well as increase our knowledge of Learning Disabilities.

If you are interested in having your child participate in this study or if you have any further questions regarding it, please call me at 262-1411.

Thank you.

(signed) Courtney L. Ruthven

Courtney L. Ruthven, M.A.
Doctoral Psychology Intern

APPENDIX F

CONSENT FORMS AND RELEASES

Consent for Participation in Research Activity

I, _____, hereby authorize Courtney L. Ruthven, M.A., to administer the Halstead-Reitan Neuropsychological Battery to_____ for Dissertation Research use.

Signature_____

Date _____

Study Subject No._____

Dissertation Research
Statistical Data

Date_____

Referring Physician:_____

Name:_____ Parents Name:_____

Birthdate:_____ Address:_____

Grade Level:_____ Phone: _____

Comments:

Research release signed _____

Medical release signed _____

Date of testing _____

Summary sent to
Physician _____

Consent for Release of Information

I hereby authorize Courtney L. Ruthven to release information and findings concerning the Halstead-Reitan Neuropsychological Battery testing for dissertation research given to:

_____ Birthdate_____

To: _____

_____

_____

_____

_____

_____ _____

Date Signature of parent or guardian

APPENDIX G

CORRELATION MATRIX FOR 49 HALSTEAD-REITAN NEUROPSYCHOLOGICAL TEST BATTERY ITEMS

CORRELATION MATRIX FOR 49 HALSTEAD-REITAN

NEUROPSYCHOLOGICAL TEST BATTERY ITEMS

Var.* No.	1	2	3	4	5	6	7	8	9
1	1.00								
2	.31	1.00							
3	.50	.15	1.00						
4	.41	.37	.11	1.00					
5	.14	.08	.25	.02	1.00				
6	.49	.62	.50	.53	.07	1.00			
7	.09	.19	.02	.54	-.03	.48	1.00		
8	.33	.32	.32	.27	.40	.49	.30	1.00	
9	.32	.23	.51	.34	.39	.43	.33	.57	1.00
10	.30	.29	.20	.24	.30	.37	.11	.18	.20
11	.35	.42	.30	.53	.29	.45	.43	.47	.62
12	.72	.68	.66	.61	.34	.85	.33	.51	.55

*See Appendix H for definition of Variables.

Var. No.	1	2	3	4	5	6	7	8	9
13	.43	.40	.44	.56	.39	.63	.61	.70	.80
14	.62	.57	.59	.64	.38	.80	.52	.66	.74
15	.50	.17	.47	.28	-.02	.33	.39	.22	.32
16	.03	.00	.01	.00	.00	.00	.01	.00	.05
17	-.11	-.36	.09	-.28	.07	-.21	-.07	- .18	.17
18	-.53	-.23	-.62	-.41	-.18	-.63	-.51	- .51	-.66
19	-.57	-.27	-.55	-.50	-.15	-.66	-.56	- .52	-.59
20	-.02	-.20	.36	-.12	.10	.02	.13	- .02	.37
21	-.27	-.10	.04	-.52	-.12	-.31	-.48	- .31	-.20
22	-.02	-.01	.13	-.36	.10	-.02	-.44	- .25	-.00
23	.16	-.26	-.18	-.16	-.06	-.28	-.30	- .52	-.37
24	-.11	-.17	.12	-.29	.22	-.13	-.24	- .38	-.07
25	-.15	-.25	-.04	-.34	-.06	-.23	-.50	- .52	-.36
26	.41	.30	.24	.18	.17	.30	.15	.39	.13
27	.34	.35	.29	.15	.50	.34	.19	.59	.33

Var. No.	1	2	3	4	5	6	7	8	9
28	-.60	-.20	-.46	-.19	-.04	-.44	-.26	-.18	-.26
29	-.27	-.11	-.29	-.14	-.32	-.22	-.06	-.27	-.50
30	-.18	-.16	-.25	-.44	.23	-.43	-.52	-.03	-.12
31	-.12	.16	-.54	-.11	.20	-.40	-.45	-.14	-.46
32	-.26	.08	-.18	-.07	.49	-.22	-.72	-.05	-.00
33	.25	.28	.33	.43	.10	.51	.52	.17	.49
34	.28	.30	.33	.24	.19	.48	.27	.23	.32
35	-.82	.09	-.19	.48	-.71	.52	.80	.09	-.92
36	-.85	-.17	-.73	.36	-.75	-.08	.88	-.83	-.27
37	.50	-.50	-.50	-.87	.50	-.50	-.24	.87	-.50
38	-.90	-.73	-.87	-.69	-.73	-.87	.72	-.50	-.94
39	-.57	-.34	-.33	-.17	-.01	-.50	.13	-.22	-.24
40	-.43	-.04	-.11	-.34	.27	-.52	-.16	-.03	.01
41	-.22	-.21	-.11	-.25	.18	-.26	-.09	-.04	-.07
42	-.17	-.02	-.02	-.25	.31	-.41	-.14	-.07	.09

Var. No.	1	2	3	4	5	6	7	8	9
43	-.48	.09	-.17	-.01	-.29	-.14	.33	-.01	-.07
44	-.09	-.15	-.12	-.13	-.02	-.24	-.15	.01	-.17
45	.03	-.14	-.34	-.11	.02	-.26	-.37	-.12	-.36
46	.04	-.15	-.37	-.08	.07	-.28	-.27	-.14	-.29
47	.67	.15	.60	.34	.17	.54	.42	.34	.52
48	.65	.29	.56	.42	.17	.50	.42	.32	.52
49	.31	.01	.52	.19	.27	.33	.38	.16	.53

Var. No.	10	11	12	13	14	15	16	17	18
10	1.00								
11	.41	1.00							
12	.41	.57	1.00						
13	.49	.87	.70	1.00					
14	.48	.79	.91	.93	1.00				
15	-.03	.19	.45	.33	.43	1.00			
16	.02	.09	.07	.06	.06	.07	1.00		
17	-.09	-.02	-.22	-.04	-.13	.22	.09	1.00	
18	-.30	-.55	-.67	-.75	-.77	-.60	.11	-.25	1.00
19	-.34	-.42	-.70	-.70	-.76	-.61	.00	-.17	.94
20	-.03	.13	.02	.19	.11	.43	.03	.88	-.45
21	-.34	-.26	-.31	-.44	-.40	-.03	.07	.23	.27
22	.09	-.22	-.03	-.24	-.16	-.07	.06	.19	.16
23	-.31	-.23	-.28	-.46	-.41	-.09	.05	.05	.48
24	-.29	-.19	-.09	-.27	-.21	-.06	.09	.49	.12

Var. No.	10	11	12	13	14	15	16	17	18
25	-.27	-.35	-.26	-.54	-.44	-.29	.01	.18	.45
26	.05	.07	.39	.22	.32	.30	.07	.07	-.46
27	.19	.31	.45	.43	.46	.27	.07	-.04	-.48
28	-.17	-.25	-.50	-.34	-.46	-.68	.07	-.38	.65
29	-.09	-.19	-.33	-.32	-.36	-.29	.01	-.07	.34
30	-.10	-.28	-.34	-.33	-.37	-.44	.07	-.06	.49
31	.07	-.18	-.25	-.38	-.35	-.44	.04	-.26	.60
32	.28	-.25	-.08	-.33	-.25	-.37	.05	-.19	.61
33	.28	.35	.49	.50	.54	.47	.09	.42	-.67
34	.36	.25	.45	.38	.45	.33	.04	.20	-.39
35	-.18	.27	-.56	.16	-.24	.62	.03	.31	-.21
36	-.85	.33	-.50	-.43	-.52	-.28	.06	-.17	.62
37	.33	-.50	-.28	-.06	-.10	.63	.00	.77	-.16
38	-.98	-.50	-.87	-.97	-.92	.06	.07	-.37	.78
39	-.50	-.13	-.55	-.28	-.44	-.05	.08	.10	.42

Var. No.	10	11	12	13	14	15	16	17	18
40	.02	.14	-.37	-.03	-.23	-.17	.09	-.03	.40
41	-.07	-.28	-.25	-.19	-.24	-.02	.09	.09	.41
42	.05	.10	-.19	.01	-.09	-.11	.09	-.03	.32
43	-.29	.06	-.27	.00	-.13	-.04	.00	-.12	.19
44	-.30	-.33	-.20	-.26	-.25	-.19	.00	-.30	.26
45	-.19	-.40	-.23	-.43	-.36	-.30	.03	-.39	.51
46	-.17	-.31	-.23	-.36	-.32	-.31	.02	-.25	.46
47	.15	.31	.63	.51	.63	.72	.09	.26	-.67
48	-.00	.40	.66	.51	.64	.70	.01	.26	-.64
49	.11	.25	.42	.44	.47	.56	.07	.52	-.62

Var. No.	19	20	21	22	23	24	25	26	27
19	1.00								
20	-.39	1.00							
21	.39	.17	1.00						
22	.23	.13	.00	1.00					
23	.54	-.10	.26	.30	1.00				
24	.16	.41	.29	.32	.58	1.00			
25	.54	.03	.25	.67	.85	.66	1.00		
26	-.55	.18	-.04	-.22	-.23	.02	-.28	1.00	
27	-.53	.16	.11	-.39	-.42	-.11	-.51	.80	1.00
28	.63	-.53	-.01	.07	.25	-.05	.19	-.37	-.32
29	.29	-.02	.20	-.36	.12	.08	-.04	-.02	.25
30	.47	-.20	.20	.04	-.08	.03	-.00	-.09	.25
31	.51	-.46	.16	-.08	.12	-.06	.04	.04	.20
32	.52	-.30	.61	.14	.02	.13	.11	-.34	.17
33	-.71	.51	-.12	-.09	-.26	.06	-.27	.43	.19

Var. No.	19	20	21	22	23	24	25	26	27
34	-.48	.28	-.17	.16	-.20	.01	-.12	.32	.29
35	-.01	.44	.65	-.70	-.56	-.07	-.60	-.82	.76
36	.76	-.17	.61	-.36	.54	.65	.30	-.70	-.66
37	-.30	.23	.19	-.68	-.09	-.06	-.08	-.50	.03
38	.60	-.98	.90	-.08	-.06	.13	-.84	.00	.05
39	.47	-.02	.30	-.42	.09	.05	-.05	-.42	-.45
40	.52	-.02	.22	.23	.21	-.14	.15	-.38	-.13
41	.40	.03	.12	.13	.07	.15	.21	-.18	-.19
42	.45	-.05	.36	-.07	.14	-.06	.01	-.19	-.09
43	.23	-.07	-.02	-.15	.10	-.12	-.02	-.38	-.45
44	.25	-.35	-.23	.05	.08	-.16	.07	-.05	-.34
45	.45	-.54	-.01	.01	.18	-.12	.13	-.14	-.17
46	.40	-.45	-.06	-.00	.24	-.05	.14	-.13	-.32
47	-.73	.45	-.08	-.10	-.27	.01	-.27	.35	.34

Var. No.	19	20	21	22	23	24	25	26	27
48	-.68	.43	-.15	-.09	-.24	.06	-.23	.36	.26
49	-.65	.68	.01	-.04	-.25	.23	-.21	.35	.25

Var. No.	28	29	30	31	32	33	34	35	36
28	1.00								
29	.23	1.00							
30	.43	.20	1.00						
31	.29	.22	.55	1.00					
32	.59	.13	.53	.59	1.00				
33	-.57	-.46	-.52	-.34	-.37	1.00			
34	-.48	-.36	-.34	-.08	-.16	.72	1.00		
35	-.09	.81	-.19	-.38	.07	.09	-.22	1.00	
36	.79	.18	-.37	-.38	-.47	.10	-.54	.54	1.00
37	-.50	.63	.97	.72	.00	-.50	-.28	.50	-.50
38	.92	.00	.92	-.26	.07	-.09	-.19	.03	.13
39	.36	.09	.10	.03	.23	-.18	-.44	.48	.77
40	.38	.09	.18	.21	.20	-.32	-.19	-.40	.36
41	.16	-.11	.23	.17	.25	-.07	.08	-.08	.24
42	.17	.01	.17	.27	.36	-.16	-.18	-.47	.25

Var. No.	28	29	30	31	32	33	34	35	36
43	.19	-.09	-.25	-.10	-.33	-.01	-.09	.50	.90
44	.23	-.37	.19	.05	-.31	-.31	-.41	-.85	.06
45	.34	-.03	.58	.47	.35	-.55	-.46	-.51	-.35
46	.32	-.09	.54	.44	.30	-.41	-.43	-.68	-.08
47	-.74	-.32	-.33	-.47	-.26	.62	.53	-.20	-.68
48	-.65	-.35	-.28	-.40	-.35	.60	.46	-.00	-.60
49	-.56	-.25	-.38	-.53	-.17	.71	.47	.29	-.13

Var. No.	37	38	39	40	41	42	43	44	45
37	1.00								
38	.12	1.00							
39	.28	.21	1.00						
40	-.50	.94	.32	1.00					
41	.31	.58	.48	.28	1.00				
42	.11	.11	.46	.70	.52	1.00			
43	-.12	.00	.38	.48	.11	.23	1.00		
44	-.14	-.50	.23	.06	.10	.07	.23	1.00	
45	.79	.19	.18	-.01	.08	.06	-.13	.61	1.00
46	.37	.18	.38	.01	.05	.12	-.11	.59	.93
47	.76	-.62	-.29	-.34	-.12	-.16	-.24	-.32	-.30
48	.50	-.52	-.25	-.29	-.17	-.17	-.13	-.25	-.29
49	.76	-.89	-.01	-.23	-.00	-.03	-.26	-.37	-.55

Var. No.	46	47	48	49
46	1.00			
47	-.21	1.00		
48	-.18	.91	1.00	
49	-.43	.78	.71	1.00

APPENDIX H

TABLE OF MEANS, STANDARD DEVIATIONS AND F VALUES FOR VARIABLES

TABLE OF MEANS, STANDARD DEVIATIONS
AND F VALUES FOR VARIABLES

		Control		Experimental		
No.	Variables	Mean	Standard Deviation	Mean	Standard Deviation	F Value
1	WISC Information	9.53	2.70	9.93	2.87	0.16
2	WISC Comprehension	9.40	3.25	9.27	2.34	0.02
3	WISC Arithmetic	7.80	1.61	9.47	3.42	2.92*
4	WISC Similarities	11.27	2.55	11.20	2.14	0.01
5	WISC Digit Span	8.80	2.01	8.67	2.29	0.03
6	WISC Vocabulary	9.20	3.12	10.87	2.80	2.37
7	WISC Digit Symbol	9.00	2.80	9.80	3.43	0.49
8	WISC Pic. Completion	10.60	2.41	11.07	3.08	0.21
9	WISC Block Design	9.80	2.65	10.87	3.66	0.84
10	WISC Pic. Arrangement	11.20	2.42	10.33	2.32	1.00
11	WISC Object Assembly	11.93	2.87	11.87	3.38	0.00

No.	Variables	Control Mean	Control Standard Deviation	Experimental Mean	Experimental Standard Deviation	F Value
12	WISC Verbal IQ	95.67	9.95	99.60	12.22	0.93
13	WISC Performance IQ	103.67	11.89	106.00	15.97	0.20
14	WISC Full Scale IQ	99.13	10.84	103.00	14.35	0.69
15	"H" Words	7.67	4.20	9.00	4.69	0.67
16	Mixed eye-hand dominance	0.40	0.51	0.47	0.52	0.13
17	Age	10.09	0.77	10.71	0.90	4.24**
18	% in Impaired Range	60.07	17.88	42.87	22.51	5.37**
19	Avg. Impair. Index	1.78	0.36	1.49	0.50	3.32*
20	Grade	4.50	0.85	5.15	1.03	3.58*
21	Category errors	60.73	22.88	65.33	23.31	0.75
22	TPT, time rt. hand	8.73	1.94	8.29	1.53	0.53
23	TPT, time left hand	6.66	2.29	5.75	2.33	1.17

No.	Variables	Control Mean	Control Standard Deviation	Experimental Mean	Experimental Standard Deviation	F Value
24	TPT, time both	3.57	1.21	4.58	3.42	1.15
25	TPT, time total	18.99	4.02	17.92	5.96	0.33
26	TPT Location	3.33	3.11	3.93	2.94	0.22
27	TPT Memory	6.53	1.68	6.80	1.42	0.30
28	Speech Perception	13.20	7.53	8.47	4.91	4.16**
29	Rhythm Errors	9.13	4.42	7.47	4.52	1.04
30	Trails A time	22.27	11.34	21.73	8.80	0.02
31	Trails B time	64.67	27.28	48.20	24.07	3.07*
32	Trails B Errors	1.03	1.07	.46	1.15	0.87
33	Finger Tap rt.	31.07	3.43	34.27	5.93	3.27*
34	Finger Tap left	29.93	3.51	30.07	5.35	0.01
35	Suppress	0.33	0.90	0.47	1.25	0.11

No.	Variables	Control Mean	Control Standard Deviation	Experimental Mean	Experimental Standard Deviation	F Value
36	Suppression hand left	0.53	1.13	0.33	1.05	0.25
37	Suppression face rt.	0.07	0.26	0.27	0.70	0.23
38	Suppression face left	0.07	0.26	0.20	0.56	0.43
39	Finger Agnosia rt.	2.67	2.13	2.60	2.90	0.01
40	Finger Agnosia left	3.87	3.27	2.07	1.49	3.77*
41	Fingertip # Wrt. rt.	8.00	3.84	7.00	4.64	0.41
42	Fingertip # Wrt. left	7.27	2.87	5.53	2.85	2.76
43	Aphasia	4.67	3.42	4.47	3.40	0.03
44	Crosses	3.87	1.25	3.93	1.67	0.02
45	Peg-Board rt.	76.60	9.51	65.87	26.57	0.01
46	Peg-Board left	86.20	21.24	87.47	55.34	0.01

No.	Variables	Control Mean	Control Standard Deviation	Experimental Mean	Experimental Standard Deviation	F Value
47	WRAT Reading	4.16	2.18	5.59	2.43	2.86
48	WRAT Spelling	3.33	1.29	4.61	2.26	3.68*
49	WRAT Arithmetic	3.48	0.74	4.09	1.13	3.03*
Impairment Indices						
	Category	2.13	0.92	2.33	0.49	0.56
	TPT Time	1.87	0.74	1.67	1.05	0.36
	TPT Memory	0.87	0.63	0.60	0.51	1.60
	TPT Location	1.93	1.62	1.67	1.50	0.22
	Speech Perception	1.26	0.59	0.93	0.59	2.36
	Rhythm	1.87	1.13	1.27	1.03	2.31
	Tapping Speed	2.33	0.62	1.93	0.80	2.35
	Trails B	1.33	0.49	1.00	0.53	3.18*
	Digit Symbol	1.40	1.18	1.27	1.22	0.09

No.	Variables	Control Mean	Control Standard Deviation	Experimental Mean	Experimental Standard Deviation	F Value
	Aphasia	1.87	0.64	1.67	0.49	0.92
	Spatial Relations	2.07	0.59	1.93	0.96	0.21
	Perceptual Dis.	2.40	0.83	1.73	0.70	5.64**

VITA

Courtney Lewis Ruthven

Candidate for the Degree of

Doctor of Philosophy

Thesis: THE EFFECTS OF METHYLPHENIDATE (RITALIN) ON THE NEUROPSYCHOLOGICAL STATUS OF LEARNING DISABLED-MINIMAL BRAIN DYSFUNCTION CHILDREN

Major Field: Psychology

Biographical:

Personal Data: Born in Hixon, Tennessee, May 16, 1936, the daughter of Joseph Darwin Lewis and Harriet Courtney Lewis. Married July 16, 1955, to Leslie Ruthven. Mother of three children, Harriet Elizabeth, Leslie Darwin, and Laura Anne.

Education: Graduated from Red Bank High School, Red Bank, Tennessee, in May, 1954; received Bachelor of Science degree with a major in Chemistry, minors in Mathematics and Physics from the University of Tennessee, March, 1963; received Master of Arts degree in Psychology from Wichita State University, June, 1972; completed requirements for the Doctor of Philosophy degree at Oklahoma State University in December, 1977.

Professional Experience: Served as a graduate assistant in the Department of Psychology, Wichita State University, 1969-70; clinical psychology graduate assistant, Wichita Child Guidance Center, Wichita, Kansas, 1969-70; psychological examiner, Dr. Leslie Ruthven, Wichita, Kansas, 1970-74; Reitan examiner and intake coordinator, 1970-74, E.S. Edgerton Foundation, Wichita, Kansas; graduate teaching assistant, Department of Educational Psychology, Oklahoma State University, 1973-74; psychology internship at the Kansas Elks Training Center and Dr. Leslie Ruthven, Wichita, Kansas, 1974-76; staff psychologist at Kansas Elks Training Center, Wichita, Kansas, 1977.