An Empirical Analysis of Brain Damage and Minimal Brain Dysfunction in School Age Children.

Gregory Blaine Sisk
Louisiana State University and Agricultural & Mechanical College

Follow this and additional works at: https://digitalcommons.lsu.edu/gradschool_disstheses

Recommended Citation
https://digitalcommons.lsu.edu/gradschool_disstheses/3499
INFORMATION TO USERS

This was produced from a copy of a document sent to us for microfilming. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the material submitted.

The following explanation of techniques is provided to help you understand markings or notations which may appear on this reproduction.

1. The sign or “target” for pages apparently lacking from the document photographed is “Missing Page(s)”. If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting through an image and duplicating adjacent pages to assure you of complete continuity.

2. When an image on the film is obliterated with a round black mark it is an indication that the film inspector noticed either blurred copy because of movement during exposure, or duplicate copy. Unless we meant to delete copyrighted materials that should not have been filmed, you will find a good image of the page in the adjacent frame.

3. When a map, drawing or chart, etc., is part of the material being photographed the photographer has followed a definite method in “sectioning” the material. It is customary to begin filming at the upper left hand corner of a large sheet and to continue from left to right in equal sections with small overlaps. If necessary, sectioning is continued again—beginning below the first row and continuing on until complete.

4. For any illustrations that cannot be reproduced satisfactorily by xerography, photographic prints can be purchased at additional cost and tipped into your xerographic copy. Requests can be made to our Dissertations Customer Services Department.

5. Some pages in any document may have indistinct print. In all cases we have filmed the best available copy.
SISK, GREGORY BLAINE

AN EMPIRICAL ANALYSIS OF BRAIN DAMAGE AND MINIMAL BRAIN DYSFUNCTION IN SCHOOL AGE CHILDREN

The Louisiana State University and Agricultural and Mechanical Col. PH.D. 1980

University Microfilms International 300 N. Zeeb Road, Ann Arbor, MI 48106 18 Bedford Row, London WC1R 4EJ, England
AN EMPIRICAL ANALYSIS OF BRAIN DAMAGE AND
MINIMAL BRAIN DYSFUNCTION IN
SCHOOL AGE CHILDREN

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by

Gregory Blaine Sisk
B.A., University of Missouri, 1974
M.A., Louisiana State University, 1976
May 1980
ACKNOWLEDGEMENTS

The author is indebted to a number of persons whose efforts made this research possible. Special thanks are due to Ralph Mason Dreger, under whose leadership this dissertation was completed. His masterful knowledge of personality and assessment and his willingness to discuss the conduct of scientific investigation enabled the author to translate vague ideas into a workable research project. His warmth and encouragement are deeply appreciated and will always be remembered. Similarly, the author is much indebted to the other members of the dissertation committee, Arthur Riopelle, Arthur Rosenkrantz, William Waters, and Donald Williamson. Their guidance and suggestions helped to place the results of this study in proper perspective. Also, thanks go to Paula Egel, Jenny Barth, Betty Carlock, Martha Pope, and William Flynn for providing subjects for this research. Finally, but of equal importance, is Mary Mevers, whose proficient typing skills helped to place this manuscript in its final form.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>TABLE OF CONTENTS</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>TITLE PAGE</td>
<td>i</td>
</tr>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>ii</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>v</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>vi</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>vii</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>DESCRIPTION OF THE PROBLEM GROUP</td>
<td>3</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>3</td>
</tr>
<tr>
<td>Symptomatology</td>
<td>4</td>
</tr>
<tr>
<td>Etiology</td>
<td>7</td>
</tr>
<tr>
<td>Prevalence</td>
<td>9</td>
</tr>
<tr>
<td>Prognosis</td>
<td>10</td>
</tr>
<tr>
<td>Summary</td>
<td>11</td>
</tr>
<tr>
<td>HISTORICAL REVIEW</td>
<td>12</td>
</tr>
<tr>
<td>Brain Damage Model</td>
<td>12</td>
</tr>
<tr>
<td>Prospective vs. Retrospective Studies</td>
<td>16</td>
</tr>
<tr>
<td>Non-Brain Damage Model</td>
<td>23</td>
</tr>
<tr>
<td>Logical vs. Empirical Classification</td>
<td>36</td>
</tr>
<tr>
<td>Empirical Approaches to MBD</td>
<td>39</td>
</tr>
<tr>
<td>Current Status</td>
<td>40</td>
</tr>
<tr>
<td>RATIONALE FOR THE PRESENT INVESTIGATION</td>
<td>42</td>
</tr>
<tr>
<td>Cluster Analysis</td>
<td>43</td>
</tr>
<tr>
<td>Section</td>
<td>Page</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>A Research Strategy</td>
<td>44</td>
</tr>
<tr>
<td>Specific Goals</td>
<td>46</td>
</tr>
<tr>
<td>Summary of Hypotheses</td>
<td>51</td>
</tr>
<tr>
<td>METHODOLOGY</td>
<td>52</td>
</tr>
<tr>
<td>Subjects</td>
<td>52</td>
</tr>
<tr>
<td>Assessment Techniques</td>
<td>54</td>
</tr>
<tr>
<td>Procedures</td>
<td>70</td>
</tr>
<tr>
<td>RESULTS</td>
<td>76</td>
</tr>
<tr>
<td>DISCUSSION</td>
<td>90</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>107</td>
</tr>
<tr>
<td>APPENDIX A</td>
<td>118</td>
</tr>
<tr>
<td>APPENDIX B</td>
<td>120</td>
</tr>
<tr>
<td>APPENDIX C</td>
<td>122</td>
</tr>
<tr>
<td>APPENDIX D</td>
<td>125</td>
</tr>
<tr>
<td>VITA</td>
<td>138</td>
</tr>
</tbody>
</table>
# LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Description of Brain Damaged Subjects</td>
<td>128</td>
</tr>
<tr>
<td>2.</td>
<td>Descriptive Statistics and Tests of Significance for</td>
<td>129</td>
</tr>
<tr>
<td></td>
<td>each Group on Identifying Data</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Reduced Factor Structure Matrix for Rotated Factors</td>
<td>130</td>
</tr>
<tr>
<td></td>
<td>Factor Scores for Each Group</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Means and Standard Deviations of Factor Scores for</td>
<td>133</td>
</tr>
<tr>
<td></td>
<td>Each Cluster</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Cluster Membership by Original Group Classification</td>
<td>134</td>
</tr>
</tbody>
</table>
LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Scree Test for Fifteen Factors</td>
<td>135</td>
</tr>
<tr>
<td>2.</td>
<td>Graph of Mean Factor Scores by Group</td>
<td>136</td>
</tr>
<tr>
<td>3.</td>
<td>Graph of Mean Factor Scores by Cluster</td>
<td>137</td>
</tr>
</tbody>
</table>
ABSTRACT

Minimal brain dysfunction (MBD) in children is described in terms of diagnosis, symptomatology, etiology, prevalence, and prognosis. The MBD area is reviewed with reference to brain damage (BD), non-BD, and empirical models of classification. A rationale is developed for empirically derived categories of MBD using (a) a comprehensive assessment battery, (b) the administration of this battery to BD, MBD, and normal children, (c) a factor analysis of the children's scores on the test variables, and (d) a cluster analysis of all the children based on the similarity of their factor score profiles. In addition, a canonical correlation between early life-history data and the factor scores in each cluster is used to determine the presence of any early high-risk signs that could predict a child's subsequent behavior.

In the actual investigation, 90 Ss ranging from 8 to 12 years of age were divided into three groups on the basis of a priori criteria, Group I consisting of 11 children with verified BD, Group II consisting of 55 children with learning and/or behavior problems indicative of MBD, and Group III consisting of 24 children who are progressing normally through school with no history of neurological impairment.

Administration of the assessment techniques yielded 36 scoring variables, which were intercorrelated and submitted to a principal components analysis. The majority of the total variance was accounted for by six factors, which are discussed in terms of the test variables comprising them.

A multivariate analysis of variance determined that the overall
pattern of factor scores differs from one group to the others. Univariate analyses of variance were used to compare differences among the three groups on each factor. The MBD group differed the most from the other groups, while the BD and normal groups were more similar. MBD children were characterized by their social, learning, and motor problems, while BD children were described in terms of their deficits in learning and motor areas. In terms of profile similarities, MBD children contrasted the most with normal children.

The results of the cluster analysis yielded five meaningful clusters. MBD classified children showed the least similarity of factor profiles, while the normal group showed the greatest similarity. Differences among cluster profiles were not significant. Also, the canonical correlation failed to show any systematic relationship between factor scores for each cluster and early life-history variables.

The findings led to hypotheses concerning the behaviors observed and reported in MBD, as well as to considerations for future research. A unitary view of MBD behavior is contraindicated. Treatment implications for reclassified MBD children are also discussed.
INTRODUCTION

In recent years clinics and schools have experienced what appears to be an alarming increase in the number of children with learning and/or behavior problems assumed to be indicative of neurological dysfunction. The increase may be genuine, but more likely it is a reflection of the growing sensitivity among professionals regarding the importance of identifying such children early and referring them to diagnostic and remedial services so that corrective measures may be initiated. Early detection of this population is hampered because the research literature suggests that children with known and suspected brain dysfunction constitute a very heterogeneous assortment of several relatively distinct groups. Since no adequate classification scheme exists, there is a need for an empirically derived grouping of these children into meaningful clusters for diagnostic and prescriptive purposes. Also, there is clearly a need for reliable and valid indicators in very young children who have a high risk of developing learning or behavior problems. Such an approach may not only enhance the diagnosis of these children, but also suggest possible etiologies associated with their condition.

The purpose of the present study is twofold. The first phase concerns the identification of the symptoms, syndromes, and clusters that characterize children with known and suspected brain dysfunction. It involves the evaluation of brain damaged children, children with learning and/or behavior problems indicative of minimal brain dysfunction, and normal children on a variety of behavioral, educational, and
neurological measures. The second phase concerns the identification of high-risk signs predictive of the syndromes and clusters of children derived from the preceding analysis. This second phase employs a retrospective analysis to determine the correlation between the children and early life-history data.
DESCRIPTION OF THE PROBLEM GROUP

The incidence of learning and behavior problems among elementary school children is a widespread and pervasive phenomenon. Since many of the deficits are thought to reflect some sort of minor central nervous system disturbance, a substantial number of professionals in the area label the condition "minimal brain dysfunction" (MBD). Brain dysfunction in these cases is said to be minimal when it cannot be detected by any unequivocal techniques, although it is assumed to exist. As a result, there has been a tendency to oversimplify and overgeneralize the concept, disregarding the traditional diagnostic landmarks within the field of brain dysfunction. Several recent reviews discuss the lack of criteria in diagnosing known and suspected brain dysfunction (Cantwell, 1975; Conners, 1973; Chalafant, 1976; Denckla, 1972; De La Cruz, Fox, Roberts, & Tarjan, 1973; Gross & Wilson, 1974; Kass, 1977; Reitan & Boll, 1973; Ross & Ross, 1976; Rourke, 1975; Schrag & Divoky, 1975; Wender, 1971; among others). In delineating some of the problems involved in the identification of MBD and associated conditions, the major viewpoints concerning the diagnosis, symptomatology, etiology, prevalence, and prognosis will be presented.

Diagnosis

MBD has become an umbrella concept covering sundry other terms, e.g., brain damage (BD) behavior syndrome, minimal brain damage, minimal cerebral dysfunction, brain-injured, hyperkinetic behavior syndrome, developmentally delayed, learning disabled. Clements (1966)
listed 38 different terms that more or less describe the conditions grouped under the MBD diagnosis. Selection of a term for a given diagnosis is not unlike a projective device because every professional person interprets the condition in terms of his or her own background, orientation, interests, or school needs. Wender & Eisenberg (1975) offer the following account of this semantic fluidity:

Few clinical problems incite such disputation as the concept of MBD . . . . There are those who deny its existence and others who see the syndrome in the majority of troublesome children. The confusion stems from an interaction of the following factors: the differing viewpoints of the professionals who encounter its manifestations; the variability of its manifestations in different settings; the variability in the syndrome itself; and the variable meanings inferred from the diagnosis by professionals and parents when it is encountered (p. 130).

**Symptomatology**

Based on a series of studies distinguishing "brain-injured" from non-brain-injured mentally retarded children, Strauss and his associates (Strauss & Lehtinen, 1948; Strauss & Kephart, 1955) characterized a cluster of interrelated behaviors later known as the "Strauss syndrome" (Stevens & Birch, 1957). Strauss reported that the major symptoms of brain-injured children are hyperactivity, or hyperkinesis, distractibility, and impulsivity. Strauss inferred that all children exhibiting this pattern of behavior are BD. Other symptoms were later added to Strauss' syndrome, such as poor organization of behavior, awkward motor performance, and inappropriate behavior with even mild provocation (Stevens & Birch, 1957).

A behaviorally similar description from a different perspective was offered by Laufer & Denhoff (1957), who described several developmental anomalies as "syndromes of cerebral dysfunction." When the
syndrome under consideration involved disorders in the areas of motility, impulse, and attention, they termed it "hyperkinetic impulse disorder," which includes, "... involuntary and constant overactivity, short attention span and poor powers of concentration, impulsivity or the inability to delay gratification, and anhedonia ... compounded by problems in the visuomotor and concentration areas." The World Health Organization and the American Psychiatric Association both recognize hyperactivity as the cardinal symptom of MBD, the former referring to the "hyperkinetic syndrome" while the latter prefers the term "hyperkinetic reaction of childhood." O'Malley & Eisenberg (1973) define hyperactivity more in terms of excitability. This excitability is manifested in temper tantrums, fights over trivial matters, low frustration tolerance, and exaggerated activity in stimulating situations, such as classrooms or other large groups.

Systematic reviews of the literature by government sponsored task forces in England (Bax & MacKeith, 1963) and the United States (Clements, 1966) addressed the terminology and identification of MBD. Clements arrived at the following definition:

The MBD syndrome refers ... 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 (p. 9-10).

Clements (1966) also surveyed many of the writers in the field and identified the ten most frequently cited symptoms of the MBD syndrome according to their lists (in order of occurrence): a) hyperactivity, b) perceptual motor impairment, c) emotional lability, d) general
coordination defects, e) disorders of attention, f) impulsivity, g) disorders of memory and thinking, h) specific learning disabilities, i) disorders of speech and hearing, and j) equivocal electroencephalographic irregularities and neurological signs.

Because many children with MBD are not identified until they enter school, the National Advisory Committee on Handicapped Children convened in 1968 to develop a definition that classroom teachers would be able to use to identify children with a "learning disability" (LD), a term introduced by Kirk (1963) which is presumably associated with a "biological defect." The Committee gave this formal definition of LD:

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 language. 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 due primarily to visual, hearing, or motor handicaps, to mental retardation, emotional disturbance, or to environmental disadvantage (p. 14).

A final perspective on symptomatology of MBD concerns more direct pathophysiological indicators of central nervous system dysfunction. A characteristic feature of such children is the prevalence of equivocal, or "soft," neurological signs, e.g., poor fine motor coordination, graphesthesia, impaired visual motor coordination, poor balance, diadochokinesia, clumsiness, strabismus, choreiform movements, and poor speech (Adams, Kocsis, & Estes, 1974; Dargassies, 1977; Denckla, 1972; Kennard, 1966; Paine, Werry, & Quay, 1968; Prechtle & Stemmer, 1962; Satterfield, Lesser, Saul, & Cantwell, 1973; Werry, 1968). The following frequencies of equivocal signs in MBD children have been reported: a)
extraocular muscle dysfunction (strabismus), 44%; b) tremor, 42%; c) athetoid movements, 32%; d) diadochokinesia, 29%; e) Babinski sign, 20%; (Kennard, 1966).

The preceding discussion of symptomatology demonstrates that everything from fist fights to EEG irregularities is being used as criterion for diagnosis of MBD and its associated conditions. The extreme diversity of signs and symptoms of MBD points out why a classification based on sound research is needed. For shorthand purposes only, the term "MBD" will be used to cover the melange of diverse behaviors found in the children in the present study until we can begin to bring order out of chaos. MBD will not be used to refer to actual structural damage, rather the term "BD" will be used to describe children with demonstrable brain insult. When discussing another study, the term used for the population in that study will be reported.

Etiology

For some time, controversy has existed concerning the etiology of MBD. Hundreds of publications later, there is still no resolution of this issue, which, along with symptomatology, is intricately involved in the diagnosis of the condition. The confusion is evident in a task force discussion of the underlying causes (Clements, 1966).

These aberrations may arise from genetic variations, biochemical irregularities, perinatal brain insults, or other illnesses or injuries sustained during the years which are critical for the development and maturation of the central nervous system, or from unknown causes (p. 10).

Generally, causative factors may be grouped as either a) genetic-familial factors, or b) non-genetic biological factors.

Proceeding from a genetic-familial approach are a number of
studies suggesting that MBD children are born to families with an incidence of psychiatric illness (epilepsy, alcoholism, sociopathy, suicide, and hysteria) significantly higher than that found in the general population (Cantwell, 1972; Satterfield, Cantwell, & Satterfield, 1974; Stewart, Pitts, Craig, & Dieruf, 1966; Wender, 1971).

These findings suggest two explanations. First, there is evidence to support the notion that genetic transmission is involved in MBD. Willerman (1973) asked the mothers of 93 sets of twins to complete a questionnaire concerning their MBD children. The correlation of scores was significantly higher for monozygotic twins \(r = .92\) than for dyzygotic twins \(r = .60\). This may, however, reflect mothers' well-known tendency to see and treat monozygotic twins alike. Second, the prevalence of familial pathology may reflect the contributory effects of poor parenting in the genesis of MBD. Children predisposed to learning and behavioral problems because of constitutional factors have been found to react negatively to familial and environmental stresses that exceed their tolerance (Bell, 1968; Bettleheim, 1973; Thomas, Chess, & Birch, 1970), or deprive them of experiences (Cole & Bruner, 1972; Hewitt, 1973; Schultz & Aurbach, 1972; Wender, 1971).

Others investigating the etiology of MBD proceed by identifying non-genetically based factors. Children with BD due to such neurological insults as encephalitis, head trauma, or lead encephalopathy, often show MBD behavior either with or without classical neurological signs or gross mental deficiency (Benton, 1973; Strauss & Kephart, 1955; Wender, 1971; Wender & Eisenberg, 1975). Other writers point to reproductive pathology as a causative factor in MBD. Anoxia, prematurity,
precipitous labor, and a host of other pre-, peri-, and postnatal anomalies have been associated with MBD (Dargassies, 1971; Cantwell, 1975; Knobloch & Pasamanick, 1966; Quay & Werry, 1972; Towbin, 1971; Wender, 1971; Wender & Eisenberg, 1975). Wender (1971) has suggested that MBD is due to an irregularity of brain monoamine metabolism.

Although some alteration in neurochemistry may be present, it is not known what the causal sequence is (as with serotonin in schizophrenia) or, that the alteration is what Wender suggests.

**Prevalence**

Paine (1968) reported that MBD is the "most common neurological disorder among children." Despite its pervasiveness, epidemiological data for MBD are relatively scarce. Surveys of U.S. children in large metropolitan schools revealed a prevalence rate of 28% of the total school population (Eisenberg, 1966). More conservative estimates range from four percent of the elementary school population in St. Louis (Stewart, et al., 1966) to 15% in Montgomery County, Maryland (Wender, 1971). Wender also summarized the above findings with data from Holland (15%) and Vermont (10%). Based on neurological signs, teacher's reports of hyperactivity and attention span, and academic performance, an incidence rate of approximately 10% was found for MBD in the elementary school population. Also, boys were much more likely to be affected than girls, with a sex ratio of 4-10 boys to one girl (using hyperactivity as the criterion). Finally, generally higher prevalence rates have been found for non-white children and children from lower socio-economic class families (Willerman, 1973).
Prognosis

For many years, MBD was believed to be a time-limited condition which disappeared as the child approached puberty (Eisenberg, 1966; Laufer & Denhoff, 1957; Wender, 1971). A follow-up study, however, of an MBD group re-examined at age 14, five years after the initial diagnosis, disclosed that such children perform more poorly in school, report more personality problems, and remain more active and impulsive than a parallel group of controls (Dykman, Peters, & Ackerman, 1973). Dykman, et al., found that children with less evidence of neurological soft signs initially were significantly less retarded academically at follow-up. Mendelson, Johnson, & Stewart (1971) interviewed the mothers of teenage children diagnosed as hyperactive six years earlier. Not only did over 75% of the sample continue to demonstrate cardinal symptoms of MBD, but also nearly 60% had had some contact with the police as a result of antisocial behavior. However, Mendelson, et al., did not report the percent of normal children who have had contact with the police (even without normal controls, 60% would appear to be high). Generally, it appears that poor adolescent adjustment in MBD children is related to initially higher ratings on target symptoms, positive neurological signs, lower socioeconomic status and unfavorable family situations.

Some writers propose that MBD persists into adulthood where it may be seen in impulse disorders, e.g., alcoholism and drug addiction (Wood, Reimhess, Wender, & Johnson, 1976). It has also been suggested that MBD children with primary perceptual deficits develop secondary emotional problems that are an adult subgroup of schizophrenia (Bellak, 1976).
Summary

The preceding description of the MBD syndrome discloses some of the complexities involved in identifying the disorder. The lack of convergence in the field is also confounded by evidence which questions the very basis of the syndrome. For instance, MBD is thought to reflect some minor neurological impairment because children with actual BD frequently display a pattern of behavior identical to MBD. Yet, there are numerous studies reporting a discrepancy between BD and behavioral sequelae. A severely physically handicapped child with cerebral palsy may show laudable learning and behavior, while another BD child may exhibit normal motor functioning and be profoundly retarded (Wender & Eisenberg, 1975). As a result, there is considerable disagreement concerning which behaviors are to be included in the syndrome, or whether MBD is actually a single distinct syndrome. At one extreme are those who suggest that all children incur minor congenital brain lesions (Towbin, 1971), while at the other extreme are those who deny the existence of the syndrome, referring to it as a "myth" which serves to relieve parents and society of the guilt they would otherwise experience for poor child rearing (Schrag & Divoky, 1975). Evidence supporting the myth of MBD comes from studies reporting insignificant differences in objectively measured hyperactivity between groups of supposedly hyperactive children and normal children (Cantwell, 1975). Also, Kennard (1966) found surprisingly high percentages of neurological signs in a population of normal schoolchildren (5-17%). In fact, approximately 70% of his normal group displayed some neurological signs.
HISTORICAL REVIEW

Tracing the history of our present state of confusion regarding the behavioral sequelae of brain damage and MBD in particular is not an easy task. Since the 1900's, conceptualizations of brain-behavior relationships have shifted in accordance with the prevailing Zeitgeist. Briefly reviewing the development of the concept of MBD, its associated conditions, and our attempts to grapple with it reveals some of the more salient features of the disorder. At the risk of oversimplification, approaches to brain damage and suspected brain damage can be grouped according to the underlying assumptions of brain status; thus, there are brain-damaged and non-brain-damaged models.

Brain Damage Model

Specific effects. In the tradition of Titchenerian structuralism, the pioneer investigators of brain-behavior relationships attempted to unravel the "morphological" components from the tangle of behaviors associated with BD. Many of the first references to behavioral sequelae secondary to BD emerged from the analysis of the neural substrates of aphasia in adults. Broca, Wernicke, Exner, and others stimulated research in this area when they demonstrated, by autopsy, that patients with speech, hearing, and writing difficulties had localized lesions in certain cortical areas. Afterwards, specific centers of the brain were posited for virtually every aspect of behavior. These "localizationists" implied that the particular type of disability observed in BD patients was due to the specific effect of a localized lesion. For instance,
higher mental functions were assigned to the frontal lobes (Tilney, 1930). Halstead (1947) followed by developing a battery of tests to assess the intellectual deficits produced by damage to these "organs of civilization."

The study of brain-behavior relationships in children did not attract the attention in this era that studies of the adult population were receiving. When the issue of BD in children was considered, writers generally invoked explanations that were based on the adult model. Consequently, studies of BD children proposed a link between alexia and parietal lobe lesions, auditory aphasia and temporal lobe lesions, dementia and frontal lobe lesions, etc. (Orton, 1937).

**Critical review.** Although the early structural analyses of brain-behavior relationships did much to establish a causal link between certain disorders and organic factors, the narrowness of the approach has been attacked. Subsequent research showed that frontal lobe damage either did not produce a long-lasting deterioration of higher mental processes, or produced less psychometric deficit than damage posterior to the Rolandic fissure (Haynes & Sells, 1963). Faced with the complexity of brain functioning and the limited methods available to study it, these localizationists found it difficult to establish a simple one-to-one structural relationship between BD and the variety of subsequent disorders.

**Nonspecific effects.** The *Zeitgeist* of functionalism after the turn of the century led investigators of brain-behavior relationships to emphasize the operation and purpose of conscious phenomena. An "organismic" approach was adopted which stressed the unitary functioning
of the brain as a whole, rather than the specificity of functioning proposed by earlier theorists. Gestalt psychology was stressing the importance of the functional integrity of the whole organism in response to a total situation. Brain damage was thought to have pervasive, non-specific effects which disrupted the functional organization in many diverse aspects of the organism's behavior. Consequently, Head (1926) described language impairment in aphasic adults as one manifestation of a more general loss of "symbolic formulation and expression." Proposing a hierarchical organization of mental processes in the brain, Jackson (1932) explained that BD resulted in an extensive "dissolution" of function, so that aphasic symptoms were general "disorders in propositionizing."

The organismic approach to brain-behavior relationships culminated in the classic work of Goldstein (1939). An explicit example of the unitary viewpoint of brain functioning can be found in his description of a group of BD soldiers with a slight injury to the visual cortex. Such patients did not lose a specific portion of their visual experience, rather, "the entire visual field was recast and a completely new way of perceiving the world was forced upon the patients." In accordance with the notion that BD has extensive nonspecific influences, Goldstein suggested that all BD patients exhibit a similar qualitative syndrome. These BD patients performed concrete tasks effectively, but were unable to function on tasks requiring more abstract thinking. Besides disturbances in cognition, Goldstein emphasized the defensive reactions ("catastrophic" behavior, e.g., meticulosity) described in MBD children today. He proposed that the consummation effect of BD was a
qualitative loss of "abstract attitude."

Organismic theory developed from adult studies of BD, so when cases of childhood BD were addressed, the adult model was applied directly and the search was on for a qualitative sign of BD in the behavioral sequelae of children. As early as 1902, an English pediatrician named Still reported "defects of moral control" in children with lesions, diseases, or other conditions what resulted in BD. Studies of the behavioral sequelae of children who recovered from the 1918 encephalitis epidemic described catastrophic personality changes in the absence of dementia (Ebaugh, 1923; Hohman, 1922). The low self-esteem and attention-seeking activities they reported are similar to contemporary descriptions of MBD children. Kahn and Cohen (1939) examined these postencephalitic children thoroughly and described a persistent syndrome they termed "organic driveness," which included hyperkinesis, distractibility, and short attention-span.

Critical review. The unitary view of BD, and particularly organismic theory, has been seriously challenged (Haynes & Sells, 1963; Hebert, 1964; Yates, 1954). Yates criticized procedures used in many studies wherein groups of BD patients with heterogeneous pathologies are indiscriminately accepted as homogeneous in their behavior. Haynes and Sells cited research showing differential test performances between right and left, or anterior and posterior, cerebral lesions. They argued that there is no justification for a single, unitary behavioral sequela to BD. Hebert addressed the definitional problems with ambiguous terms such as "propositionizing" and "abstract attitude." Whereas localizationists were challenged for being too specific, organismic theorists
have been criticized for their vagueness.

**Prospective vs. Retrospective Studies.** It should be noted that studies demonstrating a causal link between certain behaviors and brain dysfunction generally proceed in one of two directions: a) prospective studies look for behavior sequelae to demonstrable brain dysfunction; while, b) retrospective studies look for demonstrable evidence of brain dysfunction after observing behaviors similar to the behavior sequelae of brain dysfunction. With the exception of the early work of Broca and his colleagues, the preceding discussion in the Historical Review involved only prospective studies. Once prospective studies had established behavior sequelae to BD, investigators reasoned that individuals who exhibited similar behaviors were also BD, even though existing methodology could not detect any physical evidence. Some key research provided the foundation for accepting the logic of such presumptive evidence.

In his study of BD children with "defects in moral control," Still (1902) also described behavioral homologues in children who had normal intelligence and no history of BD or severe physical illness. His descriptions are not unlike those of MBD children today, i.e., "ungovernable, restless, excitable, impulsive, and disobedient." Still concluded these children were suffering from an organically based disorder possibly related to genetic factors or environmental stresses.

Orton (1937) proposed that children with language disturbances in the absence of detectable BD had failed to establish "unilateral brain superiority." Orton arrived at this decision because he felt there was "no other reason than neurological impairment for these children to
do so poorly in school," since they were "children of superior intelligence . . . from educated families."

Orton can be credited with the formal development of the concept of presumptive evidence in the diagnosis of brain status. His work contained, in seminal form, all the assumptions on which contemporary MBD theory was founded; such as, "the medical diagnosis . . . the pathological syndrome, the idea of equal rates of achievement in reading, spelling, etc., and the faith in the power of IQ tests to assess innate intellectual capacity" (Schrag & Divoky, 1975).

Orton's early proposals were echoed tentatively in Strauss' first book (Strauss & Lehtinen, 1948). By the time his second book was published, Strauss made an explicit statement regarding the validity of presumptive evidence in the diagnosis of brain dysfunction.

We select a group of individuals who behave in a certain fashion. The vast majority of these individuals display definite signs of brain-injury. About the few remaining, we do not know one way or the other. It would seem that we are justified in assuming that the factor which is causative in the vast majority is causative in the few remaining, especially in view of the fact that the common neurological examination is known not to be infallible . . . such a line of reasoning may be open to the criticism that it is circular in nature . . . if this reasoning is circular it may yet lead us to our goal without undue error (Strauss & Kephart, 1955, p. 121).

Critical review. Strauss' successors have discovered some evidence in support of his speculations. Prospective and retrospective studies have demonstrated previously undetected minor lesions throughout the cortex using neuroradiological techniques (Pasamanick & Knoblich, 1966; Towbin, 1971). With regard to the diagnosis of MBD, however, the number of retrospective or prospective studies using such sophisticated techniques is quite limited when compared to those that
infer brain status on the basis of "Straussian logic."

Assuming analogous neural substrates for similar behavior patterns on the basis of observation served not only to "explain" the etiology of a curious group of children, but also to set a pattern of interpretation followed by many practitioners today. In their expedience to account for BD-like behavior in non-BD children, Orton, Strauss, and others committed several logical fallacies. First, it does not follow that BD produces the same effects in all individuals, as the previous review has shown. Second, even if certain effects always follow specific causes, one cannot logically argue that the mere fact of following proves the causes—*post hoc, ergo propter hoc*. Third, studies which verify congenital lesions or abnormalities in MBD school children do not prove that the observed behavior is caused by the defect. What follows an event is not necessarily caused by it; on the other hand, what follows may be directly caused by it. Apparently, congenital abnormalities may be a sufficient, though not a necessary condition for subsequent learning and/or behavior problems.

The nature of retrospective experimental designs also entails certain methodological problems. The reliability and validity of the behavioral data employed in these studies is frequently suspect, especially when the data depend on parents' reports (Wender, 1971). In addition, many studies reporting presumptive evidence of brain status use correlational statistics to determine the relationship between observed behavior and early life-history data, and a significant coefficient does not in itself give any adequate evidence for cause-effect relations. Perhaps a less dogmatic approach for the interpretation of retrospective
correlations is to refer to the significant life-history variables as indicators of children with a "high-risk" of developing problems. Later research could explore the etiological significance of these high-risk factors.

In summary, retrospective studies attempt to argue backwards, so to speak, from effect to possible cause. This is not a simple problem since the hypothesized cause presumably occurred in the past and the investigator has no direct control over it. The best he can do is to try to evaluate its presence in some way. This situation is analogous to the scientific problems in astronomy, for the universe comes to us presently and we predict backward to the etiology of the stars. Astronomers certainly cannot manipulate interplanetary bodies. And, the science of brain-behavior relationships is in a similar position in this regard because our value system would not condone the types of experiments needed to fully understand the neural substrates of a child's disorders, i.e., lesion studies.

"Minimal" Brain Damage. While retrospective studies provided a theoretical justification for inferring BD from behavior, in order to arrive at our present concept of MBD writers still needed to establish the validity of "minimal" amounts of brain damage and disability. Unable to identify structural damage in children exhibiting BD-like behavior, practitioners continued to infer the presence of BD, but presumed it was minimal when existing methods were not sufficiently sensitive to detect it.

Antecedents to the concept of minimal amounts of BD associated with minimal disorders can be found in Tredgold's (1908) account of
"mentally deficient" children. He believed these children (who were not mentally retarded) had actually suffered some slight BD at birth, most likely due to anoxia, which went unnoticed until its effects became apparent with the demands of early education. The concept of minor, relatively undetectable BD gained support with the popularity of the term "minimal cerebral palsy" (Doll, Phelps & Melchen, 1932). Doll and his associates explained that the less differentiated neural structure of the child's central nervous system resulted in more diffuse kinds of impairment with minor insults.

A theoretical framework for inferring minimal BD was not available, however, until Ingalls & Gordon (1947) introduced the idea of a "biological gradient of disease." The theory proposed that the attenuated BD-like symptoms in non-BD children were actually linked to less traumatic instances of gross structural BD. Like Tredgold, Ingalls and Gordon suggested that minor BD usually occurred at birth and was not manifested until later stages of development. Subsequent theorists modified the gradient notion somewhat and substituted the more specific terminology, "Continuum of reproductive pathology" (Knobloch & Pasamanick, 1966), which views the effect of BD according to its extent; when severe, death or cerebral palsy occur; when minimal, mild "integrative" difficulties occur such as perceptual-motor or cognitive disorders. Later research by Towbin (1971) and others verifies this concept. He considers that the congenital lesions are irreversible, are capable of producing varying degrees of later disability, and possibly create long-lasting effects into adulthood. Interestingly, Towbin reports that most of these lesions are due to minimal acute hypoxia, which is what
Tredgold proposed over 60 years ago:

Critical review. The concept of minimal degrees of BD underlying minimal learning and behavior disorders has met severe criticism because "the psychological problems are anything but minimal . . . injury either exists or it does not . . . and the concept of 'minimal' distorts the picture" (Cruickshank, 1971). It should be noted that "minimal" as a descriptive adjective does not necessarily increase the accuracy or add to the validity or utility of terms such as BD. In light of Towbin's neuroradiological findings however, one cannot dispute the existence of minimal BD per se; only that it does not always account for problems a child might have that resemble BD symptoms. Nevertheless, the concept remained overextended and was often freely inferred purely on the basis of certain behavioral symptoms or obstetrical complications. The logic of such an inference is just as circular as presuming gross BD and is subject to the same criticisms mentioned in the previous section. Of course, minimal BD can be interpreted as a proposition that the behavioral constellation in MBD children is ultimately related to a neural substrate. Then the search for the neural substrate of MBD would be analogous to the search for the neural substrates of psychoses, neuroses, or any other forms of psychopathology.

Benton (1973) criticizes the concept of minimal as being a product of confused thinking in the sense that it directly translates behavior into a hypothetical state of the brain. He suggests that it would be fruitful to assume that major, rather than minimal, BD is responsible for the learning and behavioral problems of MBD children. Benton discusses animal studies which demonstrate the failure of
unilateral lesions of the motor cortex in infants to produce the severe
cortical motor impairment seen in older animals. Corroborating
evidence for the dispensability of large amounts of brain tissue in
infancy is furnished by the results of prospective studies of children
with unilateral hemispherectomies. Not only do such operations rarely
harm the child, they also lead to the alleviation of seizures associated
with intractable epilepsy. In many cases these children show a rise in
tested intelligence following the operation. Benton's position seems
to be an eloquent blend of the viewpoints expressed by Goldstein and
Cruickshank.

Cerebral lesions in children must either be quite extensive or
have specific disorganizing functional properties in order to
produce important behavioral abnormalities. It follows that
if the behavioral deviations defining MBD are to be ascribed to
brain damage or dysfunction, then that damage or dysfunction
can hardly be considered 'minimal' in character. Nor is there
evidence that it is actually less extensive than the cerebral
alterations underlying mental deficiency or cerebral palsy;
the differences may well be qualitative in nature (p. 30).

In a factor analytic study of BD, suspected MBD, and normal chil-
dren, Crinella (1973) partially confirms the notion that many NBD
labelled children are, in fact, more similar to those with the BD diag-
nosis. His neuropsychological data suggest that many MBD children are
actually BD or "afflicted with agenesis of particular cortical or sub-
cortical areas." Crinella reports behavioral communality on factors
related to learning disabilities, such as spatial disorientation, visual-
sequential confusion, and tactual imperception. Some aggressive MBD
children were also very much like BD children with septal-thalamic
insults.

Similarly, Reitan (Reitan & Boll, 1973) has reported the results
of BD, MBD, and normal children on an extensive battery of psychological tests. He differentiates the three groups on the basis of their scores on the following functions: motor speed, coordination, tactile imperception, academic achievement, visual-spatial relations, and concept formation. His overall judgement regarding the adequacy of each subject's brain functions is that MBD children are more similar to normal controls than to BD children. The typical profile for the MBD child appears to be quite different from the pattern of behavior that accompanies his sample of BD children. Although the MBD profile was closer to the normal profile, Reitan did find definite areas of deficit that distinguish them from controls. Interestingly, both Reitan and Crinella suggest that the MBD group is not homogeneous with respect to symptom patterns.

Based on parent responses to a behavior checklist, Fitch (1976) proposes that BD children more closely resemble normals than MBD children. Generally, the MBD children are viewed in a quite negative light by parents, while BD children are described by characteristics directly related to their condition, i.e., lack of coordination, speech difficulties, etc. Although it is questionable at this point, the current trend seems to be to interpret such studies as indications that MBD does not represent an intermediate point on a continuum of "organicity"; thus, it does not appear that MBD behavior can be considered a form of "minimal" BD behavior patterns.

Non-Brain Damage Model

Attempts to link structural brain pathology, whether gross or minimal, to a particular behavior pattern did not prove entirely
successful. Uncomfortable with the etiological implications of the term, "minimal" BD, writers began using more neutral terms (MBD, developmental delay, LD, etc.) which did not precisely specify the origin of the disorder, but remained biologically based. Many of these writers accepted the notion that learning and/or behavior problems are due to a multiplicity of non-structural pathologies, such as biochemical alterations or maturational lags. In addition, another group of investigators advocating a non-biologically based approach suggested that MBD behavior is fashioned by a host of psychogenic factors.

**Minimal brain "dysfunction."** The original proclamation adopting the term, "MBD", came from the International Study Group on Child Neurology which convened at Oxford University in 1962 to discuss the definition and diagnosis of minimal BD. The conference recommended substituting "dysfunction" for "damage" when diagnosing the condition because the consensus among the delegates was that BD should not be inferred from behavioral signs alone (Bax & MacKeith, 1963). A similar conference held in the U.S. in 1966 was sponsored by the U.S. Public Health Service and the National Easter Seal Society. Reviewing nearly one hundred symptoms related to minimal BD, perceptual handicaps, and LD, this task force not only concluded that "MBD" is the appropriate diagnosis, but also supported the etiological notion of non-structural pathology (Clements, 1966).

With the advent of increasingly refined techniques for measuring the properties of the brain, certain previously undetected dysfunctions have emerged to explain the abnormalities in children collectively diagnosed as MBD. Many studies report an increased incidence of abnormal
electrical recordings of skin conductance, auditory evoked responses, and various brain wave patterns in MBD, hyperactive, and LD children (Cantwell, 1975; Satterfield, et al., 1973; 1974; Wender, 1971). Interpreting these findings, Satterfield, et al., (1974) suggest that the hyperactivity associated with these disorders is the result of a lowered level of central nervous system arousal and inhibition. The essence of the theory is that the children have a "lack of inner controls over motor output and sensory input, resulting in behavioral and learning problems."

Another line of research that demonstrated a therapeutic response to stimulant medication in children with learning and behavior problems has been considered positive proof of a non-structural, biological base to the disorder. Bradley (1937) is often credited with discovering the therapeutic effect of amphetamines for MBD; and, like some of the most significant findings in medicine, his discovery was serendipitous. Bradley noted a remarkable remission of symptoms in otherwise unmanageable children after he gave them stimulants in an attempt to reduce their complaints of headaches. These children also improved in their classroom performance, referring to their medication as "arithmetic pills." Research supporting the positive effects of amphetamines or other stimulants on various learning and behavior problems continues to proliferate (Cantwell, 1975; Conners, 1973; Satterfield, et al., 1974; Wender, 1971). The effects are sometimes so dramatic that the children's condition could be whimsically referred to as "hypoamphetaminemania."

In accordance with the regulatory effect of so-called "paradoxical medication" on MBD symptoms, Wender (1971) suggests a biochemically based
explanation of the MBD syndrome. He proposes that amphetamines activate the child's inhibitory centers in the brain, thereby enabling more efficient processing of information. He suggests that the condition is the result of an abnormality in the metabolism of brain monoamines (specifically, norepinephrine and dopamine). Wender links this notion to what he calls the two "primary" symptoms of all MBD children: a) an apparent increase in arousal, accompanied by an increased activity level and a decreased ability to concentrate, focus attention, or inhibit response to the irrelevant; and b) a diminished capacity for positive and negative affect, accompanied by a decreased sensitivity to positive and negative reinforcement. Although Wender uses a great deal of anecdotal evidence to support his views, the theory represents one of the more viable approaches for understanding MBD today.

Developmental approach. The idea that developmental immaturity can account for MBD is, of course, not a new one. As early as 1930, Ewing was challenging the validity of presumptive diagnoses of BD, in favor of a more developmental orientation to childhood problems. He found that one group of purportedly BD children with auditory aphasia were unable to understand spoken language because of a high frequency hearing loss, not due to BD. Ewing suggested the term "linguistically retarded" instead of the then popular "congenital aphasia," thus, avoiding specific etiological debates. Orton (1937) also anticipated many later theorists when he reported that developmental lags result in a failure to establish efficient brain functioning.

Conventional theories which account for the relationship between development and neurological functioning elaborate on the concept of
MBD from a non-BD basis, too. Here the emphasis is on maturation as a biological process, or an inherent tendency to modify patterns of behavior and functioning. Clearly, the frequent finding that MBD symptoms disappear at puberty lends credibility to a developmentally based understanding of the disorder.

Gesell and Amatruda (1945) propose a maturational theory of child development in which the child's growth proceeds according to a time schedule. According to this theory, perseverations, impulsivity, and low frustration-tolerance are characteristic of the "terrible twos"; therefore, such behavior in older children would reflect delayed development. Furthermore, since these behaviors are presumably characteristic of the neurological development in a two year-old central nervous system, the MBD child would be considered neurologically delayed.

Bender (1969) describes a similar theory regarding "neurodevelopmental lag." Susceptibility to delays is thought to be determined by genetic factors, which, in turn, are triggered by biological crises. A biological crisis precludes neurological development beyond a given age level in certain areas, such as perceptual motor skills. In short, a child's learning and behavior problems are considered the expression of a "neurodevelopmental lag associated with constitutional biological defects."

Taking into account more of the variance in the diagnosis of developmental delays, Piaget (Hewitt, 1973) states that it is not the child's immature neural development as described by Gesell and Bender, but the distorted interactions the child has with his environment as a result of his immature neural development, which accounts for the
observed deficiencies. Experiences at different stages of cognitive
development in the form of assimilations and accommodations are consid­
ered necessary for normal development. Behavioral and learning
deficiencies would be seen from the perspective of a failure to profit
from, and subsequently master, experiences with the environment.

Psychoeducational approach. Although many professionals working
with a psychoeducational framework still regard LD as a manifestation
of central nervous system dysfunction, the recent trend is to disregard
such "naive physiologizing" in favor of a more descriptive approach to
the child's behavior. Since the term LD includes, and is often synony­
mous with, the population of children labelled MBD, it is only fitting
that the literature in the field of education is as overwhelmingly con­
fusing as that in the field of MBD. Several excellent reviews of the
development of the LD field are available and the reader is referred to
these (Cruickshank & Hallahan, 1975; Lerner, 1971; Mann, Goodman, &
Wiederholt, 1978).

The principal innovators in the educational field included Orton,
Strauss, Wepman, Kirk, and Cruickshank. These professionals charac­
terized the LD child as deficient in one or more of the "basic psycho­
logical processes or abilities" relevant to the acquisition of academic
skills. Basic-processes that contribute to differences in educational
performance include cognitive abilities, perceptual processes, psycho­
linguistic abilities, and attentional processes (Cruickshank & Hallahan,
1975; Salvia & Ysseldyke, 1978). These early explanations consider LD
to be the result of "unspecified" brain dysfunction. Dysfunctioning, in
this sense, is still biologically based and involves a deficit in the
processing capacity of the brain which precludes the efficient performance of certain neural activities.

The criterion for determining which children are LD on the basis of process-ability tests was originally offered by Bateman (1965). He said LD children manifest: "... an educationally significant discrepancy between their estimated intellectual potential and actual level of performance related to basic disorders in the learning process (p. 220)."

In the wave of the behaviorist movement in psychology and education, a new approach has surfaced in opposition to the basic-process model. Users of this new model are not concerned with the causes of learning problems or the methods a child adopts to process information. Identification of the problem and the ability to manipulate the behaviors involved is sufficient; there is no need to look further for underlying explanations or processes. The "task-analysis" model (Mann, et al., 1978; Salvia & Ysseldyke, 1978) emphasizes the identification of specific skill development weaknesses; such as, long division, fractions, letter identification, etc. A child is then remediated in these areas without further diagnosis.

Critical review. Evidence that basic-process or ability deficits are the basis of LD has been described as "at best incomplete and hypothetical" (Mann, et al., 1978). One problem is the question of what constitutes an "educationally significant discrepancy" between observed and expected abilities at a given age level. There is increasing evidence that the hypothetical constructs used to explain LD are misleading and irrelevant for a meaningful understanding of the disorder (Salvia & Clark, 1973; Ysseldyke & Salvia, 1974). Because of its
simplicity, the task-analysis model has gained popularity with the classroom teacher.

**Psychosocial approaches.** Many psychologists and psychiatrists view MBD with regard to the psychological implications of parenting and the community, generally irrespective of biological models. Even though MBD characteristics are repeatedly found in child psychiatric populations, there is a relative paucity of thoughtful studies addressing the personality dynamics of MBD children. Psychogenic factors involve the interactions between child and mother; and how this interaction accounts for the MBD syndrome, or behaviors similar to it. A more ambiguous source of effects stem from deprived early environments. These "sociogenic factors" usually involve the interactions between the child and his impoverished community, which is thought to produce certain deficits characteristic of MBD.

**Psychogenic factors.** Some writers suggest a behavioral pattern in some children similar to MBD behavior except that it is usually found among emotionally disturbed children. Chess (1960) describes an over-active, inattentive, and impulsive group of children whose condition originates from what she calls "neurotic hyperactivity." These children have no history of brain trauma or hyperactivity in early childhood. She explains that the hyperactivity manifested at school-age is the child's attempt to cope with neurotic conflicts. Later writers have elaborated on the nature of various neurotic conflicts in such children.

Malmquist (1971) considers hyperactivity to be a "depressive equivalent," or "masked depression," because of the occurrence of certain key depressive symptoms in hyperactive children, e.g., morbidity and low
self-esteem. He suggests that the same factors which lead to classical affective disturbances in adults lead to the display of hyperactivity in childhood. The rationale is that children develop hyperactivity in situations where depression would be expected, such as object loss. Malmquist concludes that the hyperactivity serves to defend the child against such depression.

In their classic study, Thomas, Chess, Birch, and Hertzig (1970) describe primary reaction patterns in children indicative of a particular temperament similar to the behavior problems in the MBD child. Children with this difficult temperament pattern are characterized by high activity, irregularity, non-adaptability, high intensity, and negative mood; and they are identifiable in the first year of life. This pattern continues into the early school years, where the child's behavior is usually labelled as hyperkinetic or MBD. The variance in behavior and adaptation at the school level, however, is presumably related to differential practices in child management by parents.

It is important to recognize the bidirectionally of effects in the mother-child dyad. Once a pattern of abnormality is established in the child, both members of the dyad contribute to the problem through an interactive process that maintains, and even exacerbates, the child's disorder. Battle & Lacy (1972) report that mothers of hyperactive male children are critical of their babies from early infancy and show a lack of affection for them during the preschool years. The mothers continue to be disapproving of them throughout the elementary school period; that is, "by pressuring them to be independent, using severe penalties for disobedience, and assessing their son's intelligence as lower than
the mothers of boys with only a moderate level of hyperactivity.

Similar findings have been reported by Fitch (1976) with an MBD population, and by Conners (1970) with a hyperkinetic population.

When parents view their child in such a negative light, there is an increased potential for the child to react negatively with a further deterioration in behavior. In his diathesis-stress model of hyperactive MBD children, Bettelheim (1973) discusses a constitutional predisposition to hyperactivity which becomes manifest only if environmental pressures exceed an intolerable level. For instance, an impatient mother may react to a restless or cranky infant with resentment. A chaotic relationship ensues in which the child fights back with restlessness and resistance when unable to cope with the mother's demands for quiet behavior. Increasing maternal disapproval leads to a deterioration in the child's behavior, such as lowered self-esteem and inability to learn (LD). In sum, Bettelheim agrees with Malmquist and views the disordered behavior, especially hyperactivity, as the child's defense against a rejecting environment. Bettelheim advocates more warmth, acceptance, and flexibility from the mother as a prophylaxis.

In order for parental pressures to account for some of the deficits in MBD, one must first assume that the child is sensitive to environmental pressures. Evidence in support of the disruptive effects of stress is provided by Conners' (1976) experiment involving the performance of hyperkinetic MBD children on an information processing task. As more information was added to the task, the Ss showed more sensitivity to the disruptive effects of a noxious stimulus (noise); both the number of errors and the length of search time on the task increased directly
with stress (demand + noise). Although these results are suggestive, they do not unequivocally prove that MBD children react to maternal pressure with a deterioration in behavior. One would expect normal children to respond in the same manner in Conners' study.

Obviously, the child's awareness of his problems constitutes an integral part of his behavior pattern. The results of studies that have been conducted, and the interpretations given by writers, consistently suggest that MBD is accompanied by low self-esteem or self-concept (Bettelheim, 1973; Thomas, et al., 1970; Malmquist, 1971; Fitch, 1976; Wender, 1971). The psychoanalytic viewpoint sheds some light on the nature of the MBD child's poor self image. Anthony (1973) proposes a psychodynamic model of MBD that emphasizes the child's unconscious awareness of body image, the effects of any deficiency in it, and his evolving concept of "self." The suggestion of body damage or deficiency in these MBD diagnosed children is considered prominent, and may be linked to feelings of inferiority and frustration, to anxieties in unfamiliar environments, to shame on recognizing their differences, and to guilt with regard to their destructiveness and impulsiveness. Anthony advocates exploring the child's own self-image in order to fully understand the dynamics involved in the observed behavior patterns of MBD.

Sociogenic factors. Wender (1971) recognizes a "privation-produced" form of MBD in which an early deviant environment precipitates abnormal patterns of behavior. Descriptions of this group include weak objective relationships and "vacillating excessive affection," usually referred to as "primary affect hunger." These symptoms are believed to be the direct result of an impoverished background, e.g., inconsistent
affiliations, segregation, lack of adequate care, etc. However, such conditions may also be present in otherwise "normal" environments; for example, one should recall the mothers in the Battle and Lacy (1972) study who showed a lack of affection for their hyperactive children from early infancy.

Research with children implies that deviant early environments may produce repercussions we subsequently label MBD. These may be similar to the incompetencies noted in institutional and disadvantaged children. Goldfarb (1943) described language disturbances and abstraction (cognitive) difficulties in institutionalized youngsters very much like those detected in MBD. Bernstein (1962) hypothesized a "restricted code" operating in the symbolic, and especially the linguistic, environment of lower-class, disadvantaged children. Their use of less explicit, restricted language codes engages them in less "verbal planning."

Assuming that the time a speaker sponds in hesitation is an index of his verbal planning, Bernstein further hypothesized lower-class children would pause less during and between phrases. When this was experimentally verified, Bernstein concluded that restricted codes inhibit cognitive processing (and underlie poor academic performance).

Generally, Wender's (1971) privation-produced form of MBD is subsumed in the more popular, "cumulative deprivation hypothesis." Briefly, the hypothesis assumes that a community under conditions of poverty is a disorganized community, and this disorganization eventually expresses itself in various forms of static deficits. For example, Mischel (1966) offers an intuitive social learning theory explanation for the impulsivity often seen in disadvantaged children. The uncertainty experienced
by youngsters in a deviant community is sufficiently increased, so that a child learns to react quickly to potentially reinforcing situations. Consequently, he has difficulty delaying reinforcement and may be labelled as impulsive when this learned pattern is observed in later behavior at school. Other deficits resulting from a poor early environment are presumably expressed at school in his lowered test scores and academic performance. These children are often said to be deficient in basic psychological processes or abilities on the basis of their poor test performance. Recent reviews of the psychometric literature, however, reject such deficiencies in children from impoverished backgrounds. Some writers propose that low test performance is not analogous to low competence (Cole & Bruner, 1972; Schultz & Aurbach, 1972).

Critical review. The preceding discussion of non-biologically based influences on learning and behavior problems is not meant to minimize the fact that there may be biological bases to MBD, but rather to emphasize that neurological abnormalities alone are not a necessary condition to account for MBD disorders. Indeed, if reports of psychogenetically determined MBD behavior patterns are correct, and they appear to be, then neurological abnormality may not always be a necessary condition for MBD.

The importance of social class or environmental background for the development of MBD has not been realized; research by Wender (1971) and Werry (1968) suggest it is a significant aspect of the disorder, while Conners (1970) reports it has no effect. In a prospective study, Willerman (1973) found that social class produced only small differences in intellectual and motor development when measured at 8 months and again
at four years in children without evidence of neurological abnormality. However, among children with neurological abnormalities, the differences in intellectual and motor development between higher and lower class were far greater at four years than they had been at 8 months. Willerman implies that child rearing practices can compensate for biological impairment in higher social class children. As the primary agent of socialization, the mother is in a position to prevent some of the learning and behavioral impairments which might otherwise occur in MBD. Actually, it is unclear whether child rearing practices enhance development in the higher social classes, or whether lower social class parents are so ineffective with their "handicapped" child that they impair his/her development. Certainly, the studies of bidirectionality in the mother-child dyad, along with the writings of Bettelheim, suggest that either or both effects could be occurring. Finally, very little is known regarding paternal influences because of the prejudicial notion that fathers play a very small role in early child rearing (Wender, 1971).

**Logical vs. Empirical Classification of MBD**

*A priori* approaches. The vague, overlapping, and often untestable conceptualizations of MBD to this point may be related to previous attempts to classify the disorder on an *a priori* or logical basis. Of the hundreds of studies which suggest a "logical" classification of MBD children, the following are representative of the type generally proposed. For instance, Wender (1971; Wender & Eisenberg, 1975) advocates a Bleulerian, or unitary, model of MBD, which includes four "subsyndromes": classical hyperactive, neurotic, psychopathic, and specific learning disabilities. On the other hand, Laufer & Denhoff
(1957) represent an a priori, pluralistic approach to classification in their list of "syndromes of cerebral dysfunction." The particular type of syndrome manifested is thought to depend upon the area of dysfunctioning involved. Thus, hyperkinetic impulse disorder involves motility, impulse, and attention problems; specific LD involves perception, association, retention, abstraction, and expression processes; dysphasias involve communication difficulties; cerebral palsy involves neuromotor impairment; etc. The syndromes described by Denckla (1972) are representative of the type of logical "splitting" writers are currently suggesting. She divides the ten most frequent symptoms of MBD (Clements, 1966) into three separate syndromes: specific language disabilities, specific visuo-spatial disabilities (also the Gerstmann syndrome), and the "dyscontrol syndrome."

**Dimensional approaches.** Clearly, there is a need to classify more accurately the nature of the MBD syndrome, or syndromes, in order to improve diagnosis, treatment, and research into etiology. An alternative to a priori classification is to use a behaviorally data-based approach. A technique must be used which accounts for the naturally-occurring relations among the behavioral data elements. Factor analysis appears to be such a technique. O'Leary (1972) has summarized the usefulness of such an approach:

Factor analytic approaches to assessment . . . have aided greatly in reducing a myriad of deviant behaviors to a small number of relatively reliable and consistent dimensions. For those concerned with arriving at a conceptual schema that organizes deviant child behavior in some meaningful way, the multivariate approach should provide some closure . . . . As yet, however, . . . the utility of the dimensional approach requires much additional research (p. 247).

In one of the earliest attempts to ascertain the principal
dimensions underlying childhood behavior disorders in general, Hewitt & Jenkins (1946) listed behavioral traits from the case histories of 500 children referred to a child guidance clinic for some behavior problem. An "eyeball" analysis of the pattern of interrelationships among the behavior traits revealed which ones occurred together, or clustered, into syndromes of deviant behavior. The three primary syndromes identified were labeled, "unsocialized aggressive, socialized delinquent, and overinhibited."

Although Hewitt & Jenkins used a very unsophisticated methodology by today's standards, their findings were generally supported by later research. Using the more formal technique of factor analysis, Peterson (1961) identified what have proven to be two stable and pervasive dimensions of childhood psychopathology. A child with a "conduct problem" has a tendency to express impulses against society; while a "personality problem" implies low self-esteem, social withdrawal, and dysphoric mood. Other less prominent dimensions were also identified, such as "immaturity" and "socialized delinquency."

The first attempts to apply the dimensional approach to MBD, or its associated conditions, sought to identify the underlying dimensions of a unitary MBD syndrome. Results failed to support this notion, however. Conners (1970) factor analyzed parent symptom ratings of 120 neurotic, 133 hyperkinetic, and 365 normal children. The factor scores discriminated between patients and normals and between neurotic and hyperkinetic groups but, the same basic factors appeared in all groups. The factors were termed "aggressive conduct disorder, anxious-inhibited, antisocial, enuresis, psychosomatics, and anxious-immature."
The first two factors appear highly congruent with conduct and personality problems, respectively, identified by Peterson (1961). Also, this study supports Peterson in another way; there was a remarkable and unpredicted lack of qualitative differences between normal and hyperkinetic, or even neurotic, children. Instead, the children differed in terms of severity of symptomatology.

Paine, Werry, & Quay (1968) factor analyzed the scores of 83 children with suspected "minimal cerebral dysfunction" on neurological examinations, EEGs, psychological tests, and behavior ratings. The seven factors, or symptoms, extracted were labeled "perceptual deficits, motor incoordination, abnormal paranatal history, abnormal EEG, later birth order, abnormal prenatal history, and abnormal reflexes." Since most of the symptoms were comprised individually of uncorrelated measures coming from one particular source of information, i.e., neurologist or birth history or psychologist or parents, the authors concluded, "minimal cerebral dysfunction is not a homogeneous diagnostic entity, but rather a way of describing a variety of unrelated minor dysfunctions, some neurological, some behavioral and some cognitive" (p. 516).

In a similar empirical analysis of 103 hyperactive MBD children, Werry (1968) attempted to delineate more clearly the syndrome by extending the range of variables on which the factor analysis was performed. In addition to the type of data collected in the Paine, et al., study, Werry included variables from psychiatric examinations, mothers' history, and obstetrical records--67 variables in all, compared to 33 in the other study. The nine factors extracted were "motor
incoordination, impaired drawing ability, dysgnosia-dyspraxia, psychopathology-poor environment, immaturity, electrophysiological instability, subcortical neurological impairment, impaired cognitive performance, and abnormal paranatal status." Werry concluded, "These multidisciplinary measures do not tap a single unitary dimension . . . but rather a series of dimensions each, or any combination of which, may be impaired in MBD" (p. 15).

Current Status

The search for a single underlying syndrome in MBD children, like the search for a single unitary sign of BD, has not proven altogether fruitful. In response to this problem, a conference was held in 1973, sponsored by the National Institute of Child Health and Human Development, the National Institute of Neurological Diseases and Stroke, the CIBA Pharmaceutical Company, and the New York Academy of Sciences (De La Cruz, Fox, Roberts, & Tarjan, 1973). They concluded that the problems subsumed by the terms MBD or LD do not constitute a single disease and the labels should not be used as diagnostic terms. A major emphasis of the conference was the efforts to determine specific entities, or definable subgroups, within the larger group of individuals who have certain common deviations of behavior and methods of learning. For example, they applauded the work of Prechtl identifying a particular subgroup of hyperactive MBD children who exhibit a characteristic "choreiform syndrome" (Prechtl & Stemmer, 1962). The general tone of this conference is reflected in the following excerpt from the proceedings:

We call the several features of MBD a 'syndrome,' but there is in fact no evidence that this congeries of diverse abnormalities--motor
defects; impulsivity, distractibility, and hyperactivity; perceptual-motor disabilities; specific failure in language development; personality deviation; and conduct disorder—warrant the designation either in the sense that the separate elements occur together or in the sense that they arise from a common underlying functional abnormality. The question is more than academic in nature because its answer will determine the direction of investigative work designed to develop the most effective modes of treatment for them. 'Syndrome analysis' through objective assessment methods . . . would thus seem to be a necessary step if further understanding of MBD is to be achieved. Such analysis should include consideration not only of behavioral characteristics but also of infra-behavioral data such as the so-called 'soft' neurological signs. . . . A possible outcome of this type of analysis may be the formulation of more limited syndromes with distinctive background characteristics and distinctive implications for treatment (Strother, p. 33).
RATIONALE FOR THE PRESENT INVESTIGATION

Today neither a causative agent nor a behavioral pattern is available to authenticate the original concept of the MBD syndrome as a distinctive diagnostic entity. As we have seen, some theorists maintain that there is enough behavioral and etiological communality for the symptoms to be classified into a single syndrome, e.g., Strauss, Wender, Satterfield, the American Psychiatric Association, the World Health Organization, etc. However, there is still no general agreement as to the symptoms defining the disorder or the labels attached to it; so that the various terms used today are frequently interchangeable. This situation has led one writer to comment that the problems of classification remain, "a thicket of thorny problems."

Despite their heuristic value, a priori classification schemes are often confusing and unworkable, while dimensional approaches using factor analysis propose an empirically derived method of describing MBD. Finally, attempts to identify a "typical" MBD syndrome are begging the question; actually the task should be to specify behaviorally similar clusters within the large group of individuals having certain learning and behavior problems. Therefore, there must be two stages for the completion of the task: (a) factor analysis of a comprehensive set of variables regarded relevant to "MBD," and (b) cluster analysis of individuals on the basis of their profiles on the resulting factorial dimensions.
Cluster Analysis

In general, cluster analysis techniques seek out profiles or patterns of scores on various measures which are typical of different clusters or groups of individuals. Such a technique is appropriate for the present study because current research suggests the presence of specific entities within the larger group of MBD children (Strother, 1973). Since it is also likely that the individual comprising a specific entity differ in some respects from one another, it is reasonable to assume the existence of a stochastic distribution of characteristics. It follows that the characteristics of different entities will have a different stochastic distribution. The overall population of measures on MBD children may be viewed as a stochastic distribution which is a mixture of several component distributions. Clustering techniques will identify and describe the distribution for each specific entity using a sample drawn from the overall population of MBD.

One method of deriving these specific entities is to intercorrelate the test scores from a sample of MBD children and extract the underlying dimensions by factor analysis. Then, the investigator could begin the cumbersome task of continuously ordering all individuals on the same underlying dimensions (factors). This is known as the R-technique.

Another method of cluster analysis, though not without criticism, is to modify the usual correlation method that is performed on test scores. In the inverse factor analysis, or Q-technique, individuals and test scores are interchanged with respect to normal factor analysis, so that the intercorrelations become correlations among individuals.
rather than test scores. These correlations are called "similarity coefficients" because they measure the relationship among individuals. The usual methods of factor analysis are applied to this similarity matrix of intercorrelations. Individuals are then assigned to clusters on the basis of their factor loadings on the extracted entities, or syndromes. Thus, a child with a high loading on a certain syndrome will be placed in the same cluster as other children having high loadings on that syndrome.

However, the Q-technique transforms each individual's scores to a scale with a common mean and standard deviation. These ipsative scores would not be appropriate for a highly heterogeneous population such as BD and MBD children because individual differences are removed with ipsative scaling. The Q-technique is appropriate only when the similarity of an individual's profile shape is important. This is because the Pearson r does not account for the profile level. The r is equal to 1.00 whenever two profiles are parallel, irrespective of how far apart they are. Because the present investigation is interested in shape as well as level, the coefficient of profile similarity ($r_p$) was used (Cattell, 1949). With this coefficient, the $r_p$ is equal to 1.00, or unity, when two profiles are perfectly alike; and $r_p$ is negative one when differences are as great as they can be. The $r_p$ has a known expected distribution so that significance tests are possible (Horn, 1961).

A Research Strategy

Dreger (1964) has suggested a particularly sophisticated research strategy using psychological tests to sort suspected 'MBD' children into
relatively homogeneous patterns of response, rather than attempting to "diagnose" different "disease" conditions. This approach allows the investigator to formulate hypotheses regarding the variable, or variables, leading to a behaviorally homogeneous pattern after the patterns are identified empirically. Such a strategy is the opposite of one that starts with a sample of putatively BD or MBD children and proceeds to look for some common symptoms of disturbed functioning.

Conners (1973) factor analyzed scores from 267 MBD diagnosed children referred for learning and/or behavior disorders. Included in the 14 test variables were measures of general intelligence, achievement, performance, and visuo-motor and perceptual processes. A cluster analysis of the factors (general IQ, achievement, rote learning, attentiveness, and impulse control) yielded five groups which were characterized in terms of the mean group profile across all factors. Conners then demonstrated the ways these groups differed on motor development, parent-teacher ratings, response to medication, and asymmetry of cortical evoked response.

Conners' study suggests a viable research strategy for the empirical identification of behaviorally homogeneous subgroups of MBD children. Some of the curious shortcomings and rather dubious procedures in his research propose questions for investigation in the present study. For instance, Conners partialled out age from the test scores and failed to include several other variables in his factor analysis which have been salient in some past studies; such as, sex, social status, neurological development, behavior ratings, and personality traits. His qualitative interpretation of planning, foresight, and impulsivity
characteristics on the basis of a performance test is questionable. Also, each of the achievement scores (reading, spelling, arithmetic) is based on a small number of items. Thus, the scores are of uncertain reliability and validity. Although the post hoc comparisons of clusters examined some useful parameters, the study disregarded historical, and conceivably etiological, variables. Finally, Conners failed to include contrast or criterion groups of any kind, which severely limits the interpretation of his results.

Specific Goals

The purpose of the present study is to refine the categorization of certain learning and/or behavioral problems in elementary school children by: (a) empirically deriving behaviorally homogeneous clusters on the basis of individuals' similarity profiles (factor score patterns), and, (b) relating them to early events predictive of later syndromes or group membership. This procedure was expected to generate hypotheses concerning the principal differences among the obtained behavior patterns, or profiles.

The general strategy that was used to investigate the behavioral communality in children with suspected MBD involves the following steps:

(a) Development of a comprehensive assessment battery that samples variables (symptoms) in behavioral, educational, and neurodevelopmental domains.

(b) Administration of this battery to school children with known BD, children referred for learning and/or behavior problems indicative of MBD, and normal children.
(c) Factor (syndrome) analysis of the children's scores on each variable, followed by statistical comparisons of the factor scores across groups.

(d) Cluster (type) analysis of the children based on the similarity of their factor score profiles, followed by statistical comparisons of the children's factor scores across clusters.

(e) Canonical correlation between early life-history data (from each child's mother and medical record and each cluster or factor, followed by statistical tests to determine early life-history variables that are significantly predictive.

Besides the suspected MBD group, two other groups of school children were investigated. A normal control group was included to account for normal variation in the population sampled. Also, a BD contrast group was included for comparison with other groups, as well as the testing of several hypotheses related to MBD and BD.

Werry (1968) demonstrated how the MBD syndrome could be delineated more clearly by extending the range of variables for factor analysis. The present study employed an assessment technique that samples a variety of behavioral expressions reflecting central and peripheral nervous system functioning. The present study uses measures of intelligence, visuomotor and visuomemory abilities, achievement, neurodevelopmental functioning, personality traits, and both teacher and parent ratings of each child's behavior. Also, age, sex, race, and socioeconomic status were included as variables.

In contrast to the general achievement test used in the Conners
(1973) study, separate tests of "skill-development" in each relevant academic subject (reading, spelling, math) were administered. Evidence suggests that the skill-development tests are more descriptive of educational deficits than general achievement tests (Mann, et al., 1978; Salvia & Clark, 1973). Since previous studies of MBD tended to overlook the importance of the child's apprehension of his own experience, this investigation included two self-report inventories that furnish objective measures of several personality traits. Additional information regarding personality traits was obtained from two behavior checklists with the teacher and parent as respondents. In general, the various sampling domains were included in hopes that the extra coverage would yield more accurate descriptions of behavior patterns.

Because many different areas of deviation are possible with BD or MBD, several factors were hypothesized to result from the factor analysis. A factor that has consistently appeared in previous studies, motor incoordination or neurological impairment (Crinella, 1973; Fitch, 1976; Paine, et al., 1968; Werry, 1968), was expected in the present study. Visual-motor dysfunction, impaired drawing ability, or poor eye-hand coordination (Crinella, 1973; Paine, et al., 1968; Werry, 1968) was another anticipated factor. Perceptual deficits or visual-sequential confusion, a factor distinct from visual-motor dysfunction in some studies (Crinella, 1973; Paine, et al., 1968), was also expected. A kind of "organic driveness" which includes hyperactivity, poor impulse control, and distractibility (Conners, 1970; 1973) was hypothesized to result from the factor analysis. Aggressiveness, antisocial behavior, or a conduct problem at home and school (Conners, 1970; Fitch, 1976;
Peterson, 1961) was another anticipated factor. An anxious-inhibited, fearful, or psychoid factor was also expected (Conners, 1970; Fitch, 1976; Peterson, 1961; Werry, 1968).

The three groups, problem, normal, and BD, were hypothesized to differ significantly in their mean factor scores, even though some overlap of individuals was expected within factor score rankings among the groups (Crinella, 1973). Individuals between and within each group were expected to differ in terms of the severity of symptomatology they manifest, as described in previous investigations using a dimensional approach (Conners, 1970; Paine, et al., 1968; Reitan & Boll, 1973; Werry, 1968). Some factors were expected to be better discriminators of the groups than others, e.g., BD children may score consistently higher on a factor like "motor impairment" (Fitch, 1976). Furthermore, it was predicted that the association between minimal amounts of BD and MBD found by Crinella (1973) would not be supported; instead, the majority of children in the MBD group should appear as dissimilar in their factor scores to normal as to BD children (Fitch, 1976). A major focus of this study, however, was not to establish the presence or absence of brain insult or central nervous system deviation.

It was hypothesized that more than one cluster would be extracted from the factor score matrix (Conners, 1973); and these clusters of individuals were describe, interpreted, and operationally-defined in detail. It was hypothesized that the largest cluster of individuals would be composed of children originally belonging to the normal control group, because these individuals were expected to show the most overall behavioral communality as a group (especially on factors heavily
weighted on variables from the educational and neurodevelopmental domains). On the other hand, the BD group was expected to have the least similarity of profiles (Crinella, 1973; Reitan & Boll, 1973), due to the great number of potentially mutable functions associated with cerebral insults, while the MBD group was expected to split into a few relatively distinct clusters of behaviorally similar individuals (Strother, 1973). Some overlap of individuals across groups during assignment of children to clusters was expected.

Assuming the existence of clusters similar to those hypothesized, they then would be submitted to a canonical correlation to determine their relationship to early life-history variables, which previous research has implicated as possible predictors or potential causative events of later disorders (Dargassies, 1971; Cantwell, 1975; Knobloch & Pasamanick, 1966; Quay & Werry, 1975; Towbin, 1971; Wender, 1971; Wender & Eisenberg, 1975). These include medical histories during gestation, delivery, infancy, and early childhood.

The life-history variables were hypothesized to covary with the empirically derived categories obtained in the preceding analyses. If certain variables were significantly related to the syndrome clusters, they then would be considered reliable indicators of high risk for the individuals in this study. They would need further investigation before they could be considered applicable to the population at large. Significant correlations would suggest other variables for cross-validation, in addition to revealing certain events for possible etiological investigation. However, it was hypothesized that only the most behaviorally homogeneous clusters would pose any real chance of correlating
significantly with the life-history variables.

Summary of Hypotheses

(a) Several factors will result from the factor analysis; such as, motor incoordination, visual-motor dysfunction, perceptual deficits, organic driveness, aggressiveness, anxiousness, age, socioeconomic status, sex, and race.

(b) The BD, MBD, and normal groups will differ significantly in their mean factor scores, although some overlap of individuals will occur on some factors.

(c) Some factors will be better discriminators of BD, MBD, and normal groups than others.

(d) More than one cluster will be extracted from the factor score matrix.

(e) The largest cluster of individuals will consist of normal children.

(f) The BD group will have the least similarity of cluster profiles.

(g) The MBD group will split into a few relatively distinct clusters.

(h) Only the most behaviorally similar clusters will correlate significantly with early life-history variables.
METHODOLOGY

Subjects

The 90 Ss in this study were divided into three groups, (I) a BD contrast group (n=11), (II) an MBD problem group (n=55), and (III) a normal control group (n=24). All groups consisted of children of both sexes and mixed racial and ethnic backgrounds, with chronological ages ranging from eight through twelve years. An attempt was made to distribute age, sex, race, and socioeconomic status variables evenly across all groups. All Ss resided and attended schools in the Baton Rouge, Louisiana area. Characteristics of the sample are presented in Table B. Each participating mother, or mother substitute, was asked to sign a consent form (Appendix A) which explains the study, the measures taken to insure confidentiality, and the provisions for withdrawal. All Ss were generally healthy during data collection and all had an IQ of at least 70, no history of severe familial mental defect, and no profound sensorimotor deficits which could preclude their participation on the tasks in the study.

Group I. In order to be included in this group of BD children, each S satisfied three criteria: (a) had received a documented medical diagnosis of unequivocal BD; (b) had a history of at least one event which could feasibly result in BD; (c) manifested at the time of data collection learning difficulties, motor impairment, or other behavioral symptoms of central nervous system disorder severe enough to cause his/her parents to seek professional help. To avoid problems related to post-traumatic encephalopathy, however, no Ss were included in this
study sooner than six months following the occurrence of the BD. The search for BD subjects conforming to the above requirements proved quite difficult. Many of the referrals were mentally retarded, or were too severely impaired physically, to complete the tasks involved in the assessment procedures. Rather than subject the sample to extreme variance, mentally retarded and severely handicapped children were not included. Thus, subjects were assigned to Group I on the basis of dependent screening of referral source statements so that the final composition of the group conformed to all of the requirements. BD subjects in the present study were referrals to the Baton Rouge Cerebral Palsy Center, Earl K. Long Hospital, or Baton Rouge General Hospital. Descriptions of the diagnoses for each BD child are presented in Table 1.

Group II. At the time of data collection, each S in this group manifested behaviors that would generally be considered indicative of MBD, according to the definition presented earlier by Clements (1966). All Group II Ss were referrals from the local school systems for evaluation of learning and/or behavior problems in the classroom. Assignment to this group was also made on the basis of dependent screening of referral source statements. In particular, children were selected whenever they presented problems that research literature has linked with minor deviations in central nervous system functioning. Excluded from this group were children with cerebral insult, severe emotional problems, or children who are on medication. All Ss in Group II were referrals from public, private, or parochial schools to a diagnostic team at the Baton Rouge Cerebral Palsy Center (this center provides services to children with other than cerebral palsy problems).
Group III. Children in this group were non-randomly selected from elementary schools on the basis of the following criteria: (a) at least average academic performance for chronological age; (b) absence of disturbing behavior traits; (c) no history of neurological impairment, disease, or insult. All Ss in this group were obtained following an interview with the child, his parents, and the teacher. An attempt was made to match the demographic variables of this group with those of the problem group. Group III Ss were obtained from public and parochial schools. Characteristics of Group III are presented in Table 2.

Assessment Techniques

Wechsler Intelligence Scale for Children-Revised. All 12 subtests of the WISC-R were administered to Ss in one session according to the standardized procedures for administration and scoring noted in the manual (Wechsler, 1974). The standardization sample for the WISC-R includes 200 children in each of eleven age groups ranging from 6 1/2 to 16 1/2 years. Stratification along age, sex, race, region, occupation of head of household, and urban-rural variables is arranged in accordance with the 1970 Census. The WISC-R yields quite satisfactory measures of internal consistency, split-half reliability coefficients ranging from .95 to .96 for the full scale. The standard error of measurement varies with age and subtest, but ranges from 1.02 to 1.84 across the levels (expressed in scaled-score units) that were involved in the present study. Likewise, stability coefficients over a 3 to 5 week period for the normative group comparable to this study's sample range from .72 to .86.
Littell (1960) noted a dearth of content validity for the test that still exists. On the other hand, much research attests to its predictive validity (see Anastasi, 1975). Some researchers suggest that relative intellectual retardation on the WISC-R is frequently the case not only for BD, but also for MBD (Benton, 1973; Reitan & Boll, 1973; Reitan & Davison, 1974). In contrast, Conners (1973) points out that MBD is not characterized by any single WISC-R pattern of subtest performance. Studies using various WISC-R test patterns as parameters (Verbal-Performance discrepancies, subtest scatter, individual subtest scores, etc.) generally fail to differentiate BD children from normals (Anastasi, 1975; Herbert, 1964; Yates, 1954). Moreover, physiological variables, such as EEG abnormality or site of lesion, are generally not predictable from WISC-R performance. Corroborating research shows that Verbal-Performance discrepancies and scatter patterns do not consistently distinguish MBD youngsters from normals (Paine, et al., 1968), or from dyslexic or emotionally disturbed children (Hartlage, 1970). Finally, with regard to concurrent validity, Wechsler (1974) reports an overall correlation of .73 with the Stanford-Binet Form L-M.

Considerable support has accumulated for a recategorization of the WISC-R proposed by Bannatyne (1968; 1974) as a practical device which "reorganizes the subtest scores into a more useful and statistically valid format than Wechsler's own grouping of Verbal and Performance" (p. 273, 1974; author's italics). The categories have been validated by several studies with various populations (LD, genetic dyslexia, reading disabled, MBD, etc.). The three primary factors proposed by Bannatyne are: (a) conceptual ability, which represents abilities closely related
to general command of language (subtests in this category are comprehension, similarities, and vocabulary); (b) spatial ability, which requires the ability to manipulate objects in three dimensional space either directly or symbolically (picture completion, block design, object assembly); (c) sequence ability, requiring short-term memory storage of sequences of auditory and visual stimuli (digit span, coding, arithmetic). Factor analytic studies reveal that the factor structure of the WISC-R corresponds closely to these categories (Kaufman, 1975).

Research has consistently indicated a clear and statistically reliable tendency for the spatial score to receive the highest relative ranking, the conceptual score intermediate ranking, and the sequential score lowest relative ranking among groups of heterogeneous LD children (Bannatyne, 1968; 1974; Rugel, 1974; Smith, et al., 1977).

Furthermore, Dykman, et al., (1973) have found that Bannatyne's constructs satisfactorily differentiate MBD children from normal controls. As a result, Bannatyne's recategorization was also used in the present study to yield three variables for intelligence from each S. Scores for each variable were calculated by finding the mean scaled score of all subtests comprising that factor, as recommended by Bannatyne (1974). If such categories do reflect "true" constructs of the WISC-R, they should be more meaningful than Wechsler's IQ Scales in the classification of the sample in the current study.

Benton Visual Retention Test. Each S was asked to copy 10 designs in Administration A of the BVRT (Benton, 1974). This administration involves a 10-second exposure followed by immediate sequential reproduction of the 10 designs on Form C and is considered to assess visuoconstructive and visuomemory ability. Although the BVRT yields two
types of scores (a total number correct and an error score indicating type of error), the present study used the more objective and reliable total number correct score.

Benton reports that retest reliability with children's total number correct scores is .85. Interscorer reliability for total scores is reported to be .95. Normative data for Administration A is based on the performance of over 600 adults and children, although the precise breakdown of subjects is not given. Selection criteria for Iowa school children used in this sample, however, appear quite restrictive.

Concurrent validity of the BVRT with the WISC-R has not been reported. In general, Benton states that validity coefficients between scores on the test and scores on standard intelligence tests are approximately .70. Reviewing several studies attesting to the predictive validity of the BVRT, Benton reports significant positive associations between impaired test performance and neurological signs, EEG abnormality, and pathological radiographs in children. The BVRT also seems to be useful in discriminating between BD and "psychogenic emotional disturbance in children" (Benton, 1974). On the other hand, the BVRT appears to be less sensitive to deficits related to specific LD. For example, Symmes and Rapoport (1972) found that the performance of dyslexic children was unremarkable, and they suggested that, "the association of immaturity in visual-motor function that is frequently related to reading difficulty appears only in populations heavily biased in the direction of attendant neurological signs."

Children's Personality Questionnaire. As mentioned earlier, Anthony (1973) advocates exploring the child's perception of his behavior
in order to understand the dynamics underlying certain patterns of behavior in MBD children. Researchers are becoming increasingly alert to this source of information, as Ross and Ross (1975) note:

Information regarding a child's problems is typically obtained from parents and teachers rather than from the child himself. The approach of allowing the child to provide information relevant to his own behavior is a potentially valuable one that has received little attention in the assessment . . . of his problems (p. 274).

Form A of the CPQ (Porter & Cattell, 1975) was administered to each S according to standardized procedures; eight of the 14 personality factors it assesses were used as variables in the present study (see Table 3 for a listing of these). The eight variables used were chosen a priori and were based on a literature review of proposed personality traits believed to be characteristic of Ss in Groups I and II of this study (Anthony, 1973; Bettelheim, 1973; Cantwell, 1975; Chess, 1960; Clements, 1966; Malmquist, 1971; O'Malley & Eisenberg, 1973; Strauss & Kephart, 1955; Thomas, et al., 1970; Wender, 1971; among others).

Porter and Cattell (1975) point out some of the difficulties involved in relating scores and norms for a population about which only a minimal degree of knowledge is available. The primary consideration for the use of the CPQ in the present study is the identification and comparison of Ss within and between groups, as well as how these children contrast with the "typical" population. Therefore, the standardization sample and scores to be used in this study include n-stens (normalized stens) based on the normative sample of 2,982 boys aged 8 through 13 years (demographic characteristics of this sample are not reported).
One-week test-retest reliability coefficients for Form A range from .28 to .75 for the factors used in this study. The authors report that internal consistency for the CPQ computed with the Kuder-Richardson Formula 21 is .49 to .89 for these factors.

Criterion-related validity of the CPQ is currently being collected regarding juvenile delinquency, school achievement, and mental retardation (Porter & Cattell, 1975). The multi-purpose nature of the CPQ and the many different dimensions of personality it measures presuppose an underlying theoretical structure which influences measures of validity. In this light, construct validity for the CPQ refers to the adequacy of the test as a measure of each personality construct it samples. Coefficients for this type of "concept" validity range from .38 to .78 for the nine factors mentioned above (this reflects the mean correlation of several groups of items with the factor they are purported to measure). Recent introduction of new data for the CPQ, however, renders such coefficients tentative at this time. Concurrent validity has been demonstrated using the CPQ to predict WISC IQ groups (Kirkendall & Ismail, 1970). Superior children in this study were characterized as more outgoing, warm-hearted, emotionally stable, calm, gay, forthright, and natural than average or low intelligence groups.

**Missouri Children's Picture Series.** An easily administered and scored card-sorting personality test for children, the MCPS, was presented to each S using the procedures for individual administration outlined in the manual (Sines, Pauker, & Sines, 1963). The test procedure consists of having the child sort 200 pictures into two stacks, "looks like fun," and "does not look like fun." The MCPS is
standardized on 3,877 children across 12 age ranges from kindergarten through the eleventh grade, using group testing procedures. The authors fail to report demographic data regarding the standardization sample.

Three of the eight scales from the MCPS were selected for study in a manner identical to the judgements made for the CPQ variables. An attempt was made to reduce redundancy in the measures of personality traits for this study by selecting MCPS variables not covered by the CPQ. Split-half reliabilities for the MCPS, using Spearman-Brown correlations, range from .33 to .73 for boys on the scales used in the present study. Ten day test-retest reliabilities range from .45 to .77, and, from .39 to .65 over a six month period for these scales. The authors note that scores for boys are consistently less stable, particularly with clinic boys.

Construct validity has been provided for the aggression, inhibition, and activity level scales that were used in the present study. Owen and Sines (1970) studied 42 pairs of like-sexed twins and reported significant heritabilities for these three scales. Concurrent validity also exists for these scales and WISC IQs (Baker, 1968). This study concluded that the highly significant correlations do not necessarily suggest that the scales are dependent to any large extent on tested intelligence. Institutional records have provided criterion related validity for high and low scoring boys on the aggression and inhibition scales (Sines, 1966). "Cerebral dysfunctioning" is reported to be the most prominent characteristic noted in institutional boys whose highest score is aggression. Sines suggests that boys scoring high on inhibition fall into one of two subsets: (a) shy, or not outgoing types with
average intelligence; or (b) rebellious, or acting-out types with evidence of "minimal neurological involvement."

Children's Behavioral Classification Project. The CBCP instrument (Dreger & Dreger, 1962; Dreger, 1977) is a 277-item questionnaire which samples a variety of school-age behaviors. It was administered to all Ss' parents, usually the mother, who was asked to indicate whether that behavior has been observed in the six months preceding the child's evaluation. Preliminary standardization and analysis of the CBCP instrument has yielded 30 factors from a group of 1,203 subjects from Florida and Louisiana, ranging in age from 6 to 13, and chosen to represent the usual proportions of urban-rural, sex, race, social class, and clinic-nonclinic children. Additional standardizations and analyses are currently underway prior to publication of the instrument. For the present study, scores for each S were compared to a normative subgroup of this larger sample (341 of the most representative subjects). Standard scores were computed from the raw factor scores and then were "normalized" on a 5-point scale.

Interrater reliability has not yet been firmly established. Mother-father correlation coefficients were found to be .40 for clinic children and .42 for controls (Gilkey, 1972). This study found parent-teacher and especially father-teacher, coefficients even lower. However, Dreger (1977) reports a correlation of .76 between parents of clinic children on a reduced number of factors. It should be noted that the same class of respondents should be used when making comparisons.

High internal Consistency reliability estimates of .94 are reported for the CBCP using the alpha coefficient; but, a more accurate
estimate would be the average correlation of .64 which is reported for individual item consistency (Dreger, 1977). Finally, test-retest reliability over a one month period is reported to be .79.

Using cluster analysis, Gay (1974) identified 8 groups that were distinguishable on the basis of the CBCP factors. These clusters of clinic and non-clinic children are not unlike the descriptions of children offered by other dimensional approaches to classification (Peterson, 1961; Quay & Werry, 1972). Costelloe (1973) also reports satisfactory criterion-related validity for the CBCP using a sample of visually-handicapped and normal children. With a reduced comparison of factors, Glanville (1974) found highly significant discrimination among psychotic, educable mentally retarded, and normal children. Finally, the study mentioned previously, in which Fitch (1976) described BD children as more similar to normal children than to MBD children, was based on CBCP reports of their mothers. MBD children were seen in a quite negative light, while BD children were characterized more by their motor clumsiness and incapacity. In light of these findings, the present study used only those 14 variables found by Fitch to be significantly discriminatory of BD, MBD, and normal Ss (see Table 3 for a listing of these variables). Reliability was also expected to increase by using only the S's mother as respondent.

Rationale for Tests of Skill Development. Although most investigations in this area use some broad-range achievement test to assess the child's educational level, the present study assessed the child's academic skills in each individual area of school curriculum with a separate test, as recommended by the "task-analysis" writers (Bijou, 1971;
Hammill, 1978; Ysseldyke & Salvia, 1974; among others). Instead of the Wide Range Achievement Test (WRAT), Metropolitan Achievement Test (MAT), etc., the present approach used a reading, arithmetic, and spelling test which offers normative assessment in addition to its criterion referenced purposes. Salvia and Ysseldyke (1978) review some of the criticisms leveled at the WRAT, MAT, and other such achievement tests they generally regard as inadequately normed, questionably validated, and poorly suited for describing the child's academic skills. Using separate tests to measure several aspects of each S's performance in terms of the school curriculum was thought to be a more accurate way of assessing academic achievement. Furthermore, the greater breadth of items on which each of the tests is based should provide a more inclusive sample of the S's functioning in each area.

Woodcock Reading Mastery Test. Each S was administered all five subtests (letter identification, word identification, word attack, word comprehension, and passage comprehension) included in Form A of the WRMT (Woodcock, 1973). The test yields grade, age, percentile, standard, and "mastery" scores in each area; but the author states that the total score from all tests combined provides the most reliable index of reading skills for normative purposes. This score was used as the "reading" variable in the present study. Since a grade equivalent is a readily understandable unit, each S's score was expressed as an overall grade level.

The WRMT is standardized on 5,252 students from kindergarten through grade 12 and is representative of demographic data in the 1970 U.S. population. Split-half reliabilities for the total reading score on Form A range from .98 to .99 for the age-grade levels in the present
study. Test-retest reliabilities over a one week period range from .83 to .87 for this sample. Subtest intercorrelations are highly variant (-.04 to .92), but are not relevant to the present study which used the more reliable total score. Construct validity using the multi-trait-multimethod matrix indicates very high convergent and discriminant validity. However, this is based on alternate forms of the WRMT and is probably not a sufficient divergence in method. Actually, what this "validity" data represents is good alternate-form reliability. Needless to say, the WRMT has been employed in a number of studies with reading disabled, LD, etc., children which demonstrate its criterion-related validity (see Woodcock, 1973, for a review of its usefulness).

**KeyMath Diagnostic Arithmetic Test.** All 14 subtests of the KDAT (Connolly, Nachtman, & Prichett, 1971) were administered to each S to assess math skill development. Despite the fact that the test provides scores and grade equivalents for each subtest and for three areas of "math skills" (content, operations, applications), only the total score (expressed in grade equivalents) was used in the present study in order to afford optimum reliability for the "math" variable.

The KDAT is normed on 1,222 children from kindergarten through seventh grade in 42 different schools from 8 states. Norm-referenced data are weighted to conform to the demographic proportions regarding race and community size. Internal consistency reliability coefficients, computed with the Spearman-Brown formula and reported in terms of total grade level scores, range from .95 to .97 for the grade levels used in the present study. No test-retest reliability is reported. Standard errors of measurement are reported by subtests and scores, revealing
Content of the KDAT has been selected by its authors on the basis of the combined item pools of their coordinated doctoral dissertations, which involved a total of 1400 youngsters in 20 states. In contrast to other arithmetic tests included in broad-range achievement batteries, e.g., the California Arithmetic Test in the California Achievement Test (CAT), Tinney (1975) reports that the KeyMath samples much more of the contemporary elementary school curricula. The open-ended format used for the items reduces the influence of guessing and enhances reliability. Also it does not require the child to read. The face validity of the KDAT is believed to maximize student interest and contribute to the validity of individual scores. Concurrent validity is also provided by Tinney, which suggests a significant positive relationship between the KDAT and the California Arithmetic Test with an LD population. The authors of the KDAT report a significant positive relationship between 28 normal fifth-grader's scores on the KDAT and the arithmetic subtest of the Iowa Test of Basic Skills (r = .69). Also, one of the test authors reports concurrent validity with assorted IQ measures in a population of 45 educable mentally retarded adolescents, coefficients averaging .59 overall. In short, the KDAT has shown respectable content, criterion, and construct validity.

Test of Written Spelling. The TWS (Larsen & Hammill, 1976) was administered to each S according to the "dictation" format described in the manual. The 60 item test consists of 25 unpredictable and 35 predictable words, on the basis of known spelling rules, and yields three types of scores; spelling ages, grade equivalents, and spelling
quotients. To be consistent with other achievement tests in the battery, the grade equivalents were used in this study for the "spelling" variable. The TWS is normed on 4,544 children in grades 1 to 8 from schools in 22 states. Demographic characteristics closely resemble those of the U.S. population in 1974.

Internal consistency measures computed with the Kuder-Richardson formula yield reliabilities from .85 to .91 for the grade levels used in the present study. Concurrent validity for 63 fourth-grade children in Austin, Texas is reported between the TWS and the Durrell Analysis of Reading Difficulty (r=.90), the WRAT (r=.84), the CAT (r=.80), and the SRA Achievement Series (r=.69); the latter coefficients were computed with the spelling subtest of the battery. Such high coefficients may indicate that the TWS is not actually different from the tests above; in fact, both the Durrell and the WRAT are also dictation format spelling tests. However, the greater number of items on which the score is based renders it preferable to these other measures. The general popularity of the Durrell and the other tests probably accounts for the lack of studies using the TWS.

**Teacher Rating Scale.** Although educational skills were assessed across the three variables above, additional behavior ratings in the school setting were needed to get a more complete picture of the child in the academic domain. Therefore, each S's current teacher was asked to complete a brief forced-choice questionnaire concerning the child's classroom behavior (see Appendix B for a list of items). The questionnaire is one used by the Baton Rouge Cerebral Palsy Center routinely for school referrals, and has been derived from various sources. It employs
several significant items in the array of such scales available for this purpose (for a discussion of these, see Lerner, 1971).

Each item was scored on a 3-point scale according to presence and severity of disturbing behavior, i.e., whether the behavior occurs "rarely, sometimes, or often." Next, the scores across all items were summed to obtain a single numerical score, which reflects, according to the teacher's reports, increasing amounts of disturbing classroom behavior with increasing value. Since the reliability and validity of this measure are not known, the rating was restricted to a single variable, "teacher's report."

Neurological Examination. Each S underwent a standard neurological examination conducted by a qualified pediatric neurologist on the staff at Louisiana State University Medical Center in Baton Rouge. Granting the questionable reliability and validity that these reportedly subjective examinations incur, the present study will adopt a similar strategy to the one discussed above regarding the teacher's report.

A review of the literature in this domain suggests that objectively defined techniques are lacking (Adams, et al., 1975; Denckla, 1972; Kennard, 1966; Pond, 1961; Yates, 1954). Adams, et al., proposed norms for a uniform examination of seven specific neurological signs; but research only supported two in his comparison of fourth-grade LD children. However, Adams did demonstrate that it is feasible to introduce objectivity into the process. To insure uniformity of procedure in the collection and interpretation of neurological status in the present study, a usable measure was developed for the practitioner. The 10 items listed in Appendix C were based on an extended review of the
literature, and are believed to cover most of the areas of neurological functioning and development in the Ss in the present study. Items were scored in a manner identical to the one described above.

Life-History Variables. Obstetrical, medical, and some behavioral data were needed for each S in the final phase of the investigation. A concise, unambiguous measure of possible causative agents and/or events thought to underlie central nervous system impairment was needed. The variables were supported by studies demonstrating a link between the agents or events and subsequent BD, MBD, learning and behavior disorders, or other conditions consistent with the Ss in this study. Also, the significant agents/events were readily observable and commonly understood by practitioners in order that they could rely on the indicators to predict high-risk children.

Previous research by Paine, et al., (1968) used quite stringent criteria in data collection and found various pre-, peri-, and post-natal abnormalities in children later diagnosed as MBD. Although Paine's investigation was retrospective in nature, causal data was based on documented medical evidence. Some of the more significant events were prenatal hemorrhages, low birth weight, prolonged labor, and trauma. These events are not unlike those reported as significant in a similarly designed study of hyperactive MBD children, e.g., maternal age at birth, previous miscarriages, birth weight, head injury, colic, hours of labor, complicated or abnormal delivery, infant distress, and use of resuscitation (Werry, 1968).

More recently, Dargassies (1977) has reported several types of impairment following premature, low-weight, and complicated births. She
has periodically checked the development of such babies in a 16-year prospective investigation. Many of these children have certain vegetative, intellectual, motor, and affective disturbances that appear to overlap considerably with the population under investigation in the present study. Approximately 18% of the 286 children with high-risk histories demonstrated some type of psychic and intellectual impairment, while almost 13% were more severely disturbed (psychotic, educable mentally retarded, etc.). Among children with impairments, the following historical events were significant: threats of, or previous, miscarriage; toxemia; precipitous labor; premature rupture of the membranes; infant distress; hemorrhages during gestation; apnoea; cyanosis; "prolonged grunting"; and neonatal anemia.

The present study used hospital records, whenever possible, concerning each S's medical and obstetrical history. As shown in Appendix D, 51 different agents and/or events were coded on one of four general variables regarding; pregnancy, delivery, infancy, and early childhood.

Life-history variables were scored and interpreted in a manner similar to that described earlier for the neurological and teacher's report measures: except that the items were not scored on a 3-point scale; rather, a simple 1 or 0 was recorded for each item. Then, all item-scores were summed to obtain a single score for each variable, as described above in the neurological and teacher's report variables. Higher scores indicate more abnormality.

**Socioeconomic Status.** The McGuire-White Index of Social Status (McGuire & White, 1955) was used in the present study. The parent established as head of household was ranked on each of three scales:
(a) occupation; (b) source of income; (c) educational attainment. On each scale, values were assigned ranging from seven (lowest status) to one (highest status). The three scales were averaged together to yield one variable for each S.

Procedures

The assessment techniques were administered by the author over a two-to-three week period for each subject. The assessment required about four hours for each subject and was presented in the order and according to the procedures discussed above. To avoid fatigue effects subjects were assessed in three or four separate sessions with at least one day between sessions. Medical information and teacher ratings were obtained by mail.

Scorer reliability. Two separate Pearson r coefficients were calculated to determine the reliability of information obtained on the neurological examination and on the early life-history variables. On the neurological examination, a subsample of fourteen MBD (6) and normal (8) children were scored for neurological signs by the author. These scores were correlated with those obtained by the pediatric neurologist. On the early life-history variables, the sum of the mother's scorings for her child on the pregnancy, delivery, infancy, and early childhood variables were correlated with the sum of the variable scores obtained from the medical history furnished by the child's physician. Twenty-five sets of scores were obtained for this subsample, which included BD (2), MBD (14), and normal (9) children. Each correlation coefficient was tested for significance by comparing it with the conventional tabled value for a Pearson r with an alpha set at .01. The null
hypothesis in each case was that the scores do not vary systematically.

**Descriptive data.** Five separate univariate ANOVAs were carried out for descriptive data (age, race, sex, socioeconomic status, and Wechsler Full-scale IQ) to determine the presence of any significant group differences. The null hypothesis in each case was that there are no differences among the groups on the identifying variable. Obtained F-values greater than the expected value for an alpha level of .01 was the criterion for calling the null hypothesis into question. For significant ANOVAs, the Duncan Multiple Range Test was employed for all possible pairwise comparisons between groups. The null hypothesis for each post-ANOVA test was that no significant difference exists between groups on that variable when alpha equals .01.

**Factor analysis.** All 36 scores were put on cards, intercorrelated with a Pearson r, and factor analyzed using programs supplied by SAS—Statistical Analysis Systems (Barr, Goodknight, Soll, & Helwig, 1976). The procedures used in the present factor analysis are described in detail elsewhere (Gorsuch, 1974; Harris, 1975) unless otherwise indicated. Unities were inserted in the diagonals and the correlation matrix was subjected to a principal components analysis. Cattell's Scree Test (1966) was used to determine the number of factors extracted. This procedure is believed to have yielded the minimum number of factors necessary to account for the maximum amount of variance in the matrix.

Although several techniques are available for rotation to simple structure, they generally yield the same results. For example, Gorsuch (1974) calculated independent varimax promax, biquartimin, maxplane, and other rotations on the same data and found only one
significant difference—varimax and promax were 5 to 30 times faster. Consequently, the present study initially used varimax rotations to obtain a structure with each variable loaded on as few factors as possible. This was followed by oblique rotations with promax.

An attempt was made to minimize the number of factor loadings by non-significant variables. The factor structure matrix was inspected and factor structure weights which failed to exceed twice the conventional tabled value for significant Pearson r coefficients were not considered salient and were assigned a value of .00. Thus, interpretation of a factor was made on the basis of significant variable loadings only.

In order to compare group and cluster performance across factors, a factor score matrix was computed with a simple matrix algebra technique. To obtain the standard factor scores, the standard score matrix was algebraically multiplied by the factor structure matrix. The resulting standard factor scores were used in all subsequent analyses.

**Analysis of variance.** A separate univariate ANOVA was calculated for each factor and tested for significance to determine whether the groups can be distinguished on the basis of their factor scores. The null hypothesis in each case was that the BD, MBD, and normal groups do not differ significantly on that factor. If the obtained F-value was greater than the expected value for an alpha of .01, the null hypothesis was rejected.

A multivariate analysis of variance was performed on all groups of subjects to determine whether the overall pattern of factor scores differs from one group to the other. The MANOVA tested the null
hypothesis that there are no overall differences in standard factor score patterns among the three groups. The null hypothesis was called into question only after determining that the overall F in the MANOVA met the appropriate criterion for significance established by the Wilks lambda statistic when alpha equals .01.

Significant F-values in the univariate ANOVAs fail to specify the way in which the factor score contributed to that significance. All possible pairwise comparisons of the mean factor scores between groups were computed with the Duncan Multiple Range Test. These comparisons reveal which factors differentiated each group, as well as which group contrasts the most with other groups. The Duncan was chosen because it is a more powerful post-Anova procedure than the t-test. The null hypothesis in each comparison was that no difference exists between group means on each factor when alpha is set at .01.

Group profile similarity. The relationship between group profiles was tested for significance in a procedure using Cattell's $r_p$ coefficient (1949). The mean factor score profiles for each group were intercorrelated with the $r_p$ using Horn's (1961) tabled value for significant $r_p$ coefficients as the critical value (alpha equals .01). The null hypothesis was that the three profiles of group means do not differ significantly.

Cluster analysis. Coefficients of profile similarity, $r_p$, were computed for all pairs of individuals across the standard factor scores. The intercorrelations for each pair of subjects yields an $r_p$ matrix, which was subjected to a hierarchical cluster analysis in the SAS package. The particular hierarchical analysis used by SAS is an
agglomerative method described in detail by Johnson (1967). Briefly, clustering begins by treating every subject as a separate entity at the bottom level (weak cluster). Then, the two entities with the smallest d (distance) are selected to form a new cluster, or entity, at the next higher level. Using the minimum distance of two members of a cluster with all other entities for recalculation of a new inter-entity distance matrix, the clustering procedure continues at the next higher level until all entities are grouped into a single entity (strong cluster). The clusters at each level were inspected to determine the most meaningful aggregation.

Cluster profile similarity. To determine the relationships between cluster profiles, all mean standard factor scores were intercorrelated using the $r_p$ coefficient. Differences between mean cluster profiles were then tested for significance, accepting the tabled value for $r_p$ coefficients (Horn, 1961) as the minimum criterion when alpha is set at .01. The null hypothesis for each comparison between clusters was that the mean factor score profiles do not differ significantly.

Canonical analysis. For each cluster of individuals, a CANONA was conducted in order to determine the extent to which various constellations of early life-history variables are "predictive" of factor scores. The early life-history variables (pregnancy, delivery, infancy, and early childhood) were not used in previous analyses. Canonical correlation is basically the value of the maximum possible Pearson r between two sets of variables. The particular CANONA employed in the present study is based on the "general linear model" in the SAS package, which is described in detail by Harris (1975).
Coefficients of canonical correlation ($R_c$) were computed for each cluster. The CANONA package in SAS yields sets of canonical variates equal to the number of variables in the smaller group. Thus, four sets of canonical variates were derived for each cluster. Chi-square tests for overall significance were conducted for each $R_c$ in each cluster. The null hypothesis in each case was that no significant relationship exists between individuals' factor scores and their scores on the four life-history variables when alpha equals .01. For significant chi-square tests, the corresponding canonical variates were tested for significance to determine how the factor scores vary with the life-history variables in that cluster. Individual canonical variates were tested for significance at the .01 level, using critical values from conventional tables for the Pearson $r$. The null hypothesis for each of these comparisons was that factor scores do not vary systematically with scores of the life-history variables.
RESULTS

Identifying Data

As shown in Table 2, the BD, MBD, and normal groups do not differ significantly with respect to age, race, sex, or socioeconomic status ($p > .01$). However, there are IQ differences and the null hypothesis of no significant differences is rejected ($p < .01$). The Duncan post-ANOVA test reveals that the normal group has the highest IQ, followed by the MBD group ($p < .01$). The BD group has the lowest IQ ($p < .01$). The IQs for the MBD and normal groups fall within the average range of intelligence while the IQ of the BD group lies within the low average range.

Scorer Reliability

The coefficient of scorer reliability for the neurological examination is .84, which suggests significant agreement between the two examiners. The scorer reliability on life history variables is .88, which is also highly significant. The degree of agreement between the mother's report and the child's medical history suggests that the mother can give reliable retrospective information concerning her child.

Factor Analysis

The scree of the distribution (see Figure 1) occurs at an eigenvalue of 1.7 and yields six factors which account for approximately 60% of the total variance. A reduced factor structure matrix containing only salient variable weights is presented in Table 3. Group means and standard deviations across factors are presented in Table 4. Also, the
term "his" in subsequent discussions will be used to refer to both sexes unless otherwise indicated.

**Factor 1.** As shown in the factor structure matrix in Table 3, the first factor extracted, which accounts for the greatest amount of the total variance (22%), has heavy loadings on variables from the CBCP instrument. Significant loadings from the CBCP instrument include: Immature, neurasthenic, paranoic reactions; Disobedient, sullen, hyperactive aggressiveness; Verbal psychoid reactions; Intellectual and scholastic retardation vs. alert socialized achievement; Anti-social aggression; Negativism vs. peer aggressive obedience to authority; Temper tantrums; Appreciative, concerned, obedient social orientation vs. unappreciative, aggressive disobedience; Fearful desurgent seclusiveness vs. sociableness; Self-derogating school phobia; and Clumsiness and visual problems. The Teacher's report is the only non-CBCP instrument variable that has a significant loading. In subsequent discussions, Factor 1 is called "Anxious-aggressive social behavior vs. sociableness."

A child with a low score on this factor tends to be labelled by his parents and teacher as immature, disobedient, and unappreciative. The lower scorer is negativistic and displays a great deal of "foot-dragging" behavior, i.e., loses things, does not respond to questions, etc. Such a child tends to engage in anti-social behavior such as lying, stealing, and damaging property. Low scorers have temper tantrums, argue, tease, and pick on others. Yet, they blame others and complain that others are picking on them. The low scoring child is socially inappropriate and he is often seclusive and plays alone or with younger children. He has a fear of and hatred towards school. At school,
individuals with low factor scores are slow to begin classroom tasks, clumsy, and forget what they are trying to say. Conversely, a child with a high score on this factor is viewed by others as obedient, sociable, and unaggressive. Such a child is alert, uses words easily, and performs well in school. The high scoring child plays fair with others and others ask him to play with them.

Factor 2. The second factor extracted, accounting for 12% of the total variance, resembles a learning behavior problem or poor school performance. The highest loadings occur on the spelling, reading, and arithmetic achievement tests (TWS, WRMT, and KMDT, respectively). Two of the WISC-R factors, sequential and conceptual abilities, as well as the BVRT visuomemory variable, have significant loadings. The Neurological examination, the Teacher's report, and one variable from the CBCP instrument (Intellectual and scholastic retardation vs. alert socialized achievement) also have loadings on this factor. Factor 2 is similar to Factor 1 in that a low score indicates the problem behavior. In subsequent discussions, Factor 2 is called "Learning behavior problems vs. achievement."

A low factor score indicates a child who is well below his expected grade level in spelling, reading and arithmetic. In addition to these specific content disabilities, the low scoring child shows deficits in basic processes or abilities, such as sequential organization, conceptual thinking, and visual memory. The low scoring child's disabilities are pervasive and are found in variable loadings derived from several different sources. Neurological signs are seen by the neurologist, poor classroom performance is observed by the teacher, and
non-achievement is reported by the mother. On the other hand, high scores on this factor represent good achievement test scores, as well as effective basic processing skills and an alert, motivated approach to school.

Factor 3. Nine percent of the variance is accounted for by the third factor extracted, which loads with items that measure a dimension of emotional lability.

Six of the seven significant variable weights are CPQ items, such as, Phlegmatic vs. excitable, Relaxed vs. tense, Affected by feelings vs. emotionally stable, Uncontrolled vs. controlled, Expedient vs. conscientious, and Shy vs. venturesome. The Teacher's report is the only non-CPQ variable with a significant loading on this factor. In subsequent discussions, Factor 3 is called "Emotional stability vs. emotional lability."

Individuals scoring high on this factor are impatient and undependable. They often overreact on slight provocation and disregard rules and bypass obligations. High scorers seem to have weak ego and superego strength. For example, a child with a high score on this factor might be in trouble with school authorities, not through delinquent intent, but through carelessness and neglect. This factor also seems to relate to symptomatic behavior generally explained in terms of undischarged drive or nervous tension. High scorers have a low frustration tolerance that may give way to displays of temper and irritability. Such a child is more easily intimidated and does not cope effectively, nor interest freely, with others. Conversely, the low scorer is characterized as calm and conscientious. They are deliberate and are in strong
control of their behavior. The low score on this dimension apparently reflects the extent to which the child has incorporated the values of the adult world. The child with a low score presents a kind of relaxed composure that makes for easy interaction with others and a generally untroubled approach to life.

**Factor 4.** The fourth factor extracted (accounting for 67% of the total variance) has loadings on several different variables that seem to measure perceptual-motor incoordination and associated behaviors. The Neurological examination has the highest weight, followed by the CBCP instrument variables, Anxious有机ism and Clumsiness and visual problems. The BVRT, measuring visuomemory and visuoconstructive abilities, has a high loading also. Finally, two other CBCP instrument variables, Verbal psychoid reactions and Fearful desurgent seclusiveness vs. sociableness, load on this factor. In subsequent discussions, Factor 4 is called "Relaxed motor coordination vs. anxious motor incoordination."

The child who scores high on Factor 4 tends to display neurological "soft" signs, such as fine-motor incoordination, synkinesis, and disturbances of balance and directionality. High scorers do not draw well from memory and they have trouble concentrating. Such a child stumbles and falls easily and is generally not in control of his muscles and senses. The loading on seclusiveness suggests that high scorers tend to play alone. As with Factor 2, this factor is identifiable from different referral sources—the child, the mother, and the neurologist. The low scoring child tends to be coordinated, with good visual memory and the ability to concentrate. Also, the low scorer gets along well
with others.

**Factor 5.** The fifth factor extracted accounts for five percent of the total variance and appears to measure the child's activity level. The highest loading is on the MCPS variable, activity level, followed by another loading on the same test, Aggression. The CPQ variable, Uncontrolled vs. controlled, also has a high loading on this factor. This factor is structured so that the higher the score the less activity is reported. In subsequent discussions, **Factor 5** is called "Uncontrolled overactivity vs. normal activity."

Individuals who score low on this factor report a preference for more active behavior that frequently involves physical movement outdoors, e.g., playground games, sports, etc. In addition, much of this overactivity is of the aggressive type, e.g., competitive acts, fights, outbursts, etc. Inspection of the MCPS cards for the Aggression scale reveals that most of the aggressive items also involve physical activity. Evidence suggests that the low scorer is careless of, or unable to conform to, environmental restraints and sometimes engages in active behaviors in situations where it is not appropriate. High scorers, on the other hand, tend to be controlled and prefer indoor games. However, they do participate in some organized physical activities outdoors.

**Factor 6.** The sixth factor extracted, which accounts for 5% of the total variance, has high loadings on items that reflect inhibition, avoidance, and withdrawal. The MCPS variable, Inhibition, and the CPQ variable, Affected by feelings vs. emotionally stable, have the highest loadings on **Factor 6.** Two variables from the CBCP instrument,
Self-derogating school phobia and Displaced aggressiveness vs. direct aggressiveness, also load on this factor. In subsequent discussions, Factor 6 is called "Spontaneous approach vs. inhibition/avoidance."

The child with a high score on Factor 6 is shy, inhibits many of his impulses, and avoids social participation. High scorers may be rejected by their peers and may fear or hate school for the social demands it makes on them. The child is moody and easily upset and seeks, through withdrawal, to avoid social threat and overstimulation. The child is emotionally unstable and may occasionally be subject to a loss of emotional control resulting in outbursts and acting-out behaviors. These children do not express anger well and they tend to displace their aggressiveness, e.g., drawing pictures on the walls, being too obedient, etc. In contrast, low scorers are uninhibited and spontaneous. A low score indicates an individual who faces reality, enjoys school, and expresses his feelings directly.

Analyses of Variance

A MANOVA was performed to determine whether the overall pattern of factor scores differs from one group to the others. The Wilks lambda statistic is apparently highly significant ($\lambda = .0001$). No widely agreed-upon distribution of lambda exists (Overall & Klett, 1972). However, approximations of F based on Hotelling-Lawley and Pillai'd Trace were significant at the .0001 level. Therefore the results suggest that the factor score patterns clearly differentiate among at least two of the groups in the present study.

As shown in Table 4, three of the six univariate Fs met the minimum criterion at the .01 level or beyond—Factors 1, 2, and 4.
Factors 3, 5, and 6 did not differentiate the three groups at the a priori level of significance (ps > .01).

Group differences were calculated with Duncan's Multiple Range Test and the results are presented in Table 4. On the social behavior factor, the MBD group has significantly more social problems than either the BD or the normal groups (ps < .01), but there is no difference between the BD and normal groups (p > .01). Each group differed significantly on Factor 2, Learning behavior problems, with the normal group having the best learning behavior, followed by the MBD group, and then the BD group (ps < .01). On Factor 4, the BD group has the most anxious motor incoordination, the MBD group moderate anxious motor incoordination, and the normal group has relaxed motor coordination (ps < .01). Duncans were not performed on the other factors because significant F-values were not obtained in the univariate ANOVA.

As shown in Table 4, the MBD group differed from normal and BD subjects on all three Duncan comparisons, while the normal and BD groups differed on only two of the comparisons. Thus, the greatest contrast is between the MBD and normal groups and the fewest differences are found between the normal and BD subjects. In other words, based on their factor scores, the normal and BD subjects are more similar to each other than either group is to the MBD children.

The profiles of group means across each factor are presented graphically in Figure 2. Compared to other groups, factors (polar descriptions) significant to the BD group are:

Factor 2: Learning behavior problem

Factor 4: Anxious motor incoordination

Factors significant to MBD subjects are:
Factor 1: Anxious-aggressive social behavior
Factor 2: Learning behavior problem
Factor 4: Anxious motor incoordination

Factors significant to normal subjects are:
Factor 2: Achievement
Factor 4: Relaxed motor coordination

Group Profile Similarity

The relationship between group profiles was tested in a procedure using Cattell's (1949) coefficient of profile similarity ($r_p$). The $r_p$ coefficients are listed in tabular form in Figure 2. As shown, the results confirm that the MBD and normal subjects differ significantly at the .01 level ($r_p=-.35$). The profiles of the MBD group were not related to the BD group in any systematic way ($r_p=-.03$), nor were the profiles of the BD group and normal group significantly related ($r_p=-.26$), indicating that performances across the six factors did not differ with respect to the level and shape of their profile configuration ($p < .01$).

Cluster Analysis

The means and standard deviations of the five clusters are presented in Table 5. The composition of each cluster by original group classification is presented in Table 6. The results of the cluster analysis yielded hierarchical clusters. Five clusters were selected for subsequent analyses because the aggregation represents the most meaningful groupings.\(^1\) Proceeding from the single (strong) cluster, \(^1\)The method of cluster extraction is described in the Discussion section.
the second aggregation yielded the division between Clusters B and C in the present study. At the third level, Cluster A divided from Cluster B. In the next aggregation, Cluster E split-off from Cluster C. Cluster D appeared at the fifth level. There was little change through eight clusters, with one or two MBD subjects forming separate clusters at each of the levels. Slightly larger divisions in Cluster C and Cluster D account for groupings at the ninth and tenth levels, respectively.

Cluster A consists of three girls and one boy (all are white) with a mean IQ of 92 and a mean age of 9.3 years. Three of the children were originally members of the BD group. The BD conditions associated with these subjects are a malignant ganglioma of the left cerebellar hemisphere, cerebral palsy with left spastic hemiplegia and cranial nerve impairment, and hydrocephalus secondary to spina bifida. The other child in Cluster A was originally classified in the MBD group, but has a history of two separate head injuries from falls. Compared to other clusters, factors (polar descriptions) characteristic of Cluster A are

Factor 4: Anxious motor incoordination
Factor 2: Learning behavior problem
Factor 1: Anxious-aggressive social behavior

The most distinctive factor of Cluster A is the severe perceptual-motor impairment, which includes BVRT scores much below that expected for age-level and many neurological signs. Relative to the other clusters, Cluster A is not markedly different with the exception of their high scores on Factor 4. However, they do display some social and learning behavior problems.
Cluster B consists of 18 children, 16 of whom were originally classified MBD and two who were in the BD group. The BD conditions are encephalitis and minimal cerebral palsy. The mean age is 9.6 years and the average IQ is 94. Four of the children in this cluster are Black, which is the highest proportion in any of the clusters. Four of the members are girls. Cluster B has the lowest mean socioeconomic level of any cluster. Compared to other clusters, factors (polar descriptions) characteristic of Cluster B are:

Factor 1: Anxious-aggressive social behavior
Factor 2: Learning behavior problem
Factor 3: Emotional lability
Factor 6: Inhibition/avoidance

Behaviorally, these children have the most extreme scores of any cluster on the above factors and they represent the most problematic group.

Cluster B subjects display moderate anxious motor incoordination, but Factor 4 does not seem to distinguish them relative to the other clusters.

Cluster C contains 15 subjects, which is mostly MBD diagnosed children but includes two BD and one normal subject. The average IQ is 100 and the mean age is 10.1 years, which is the oldest of all the clusters. Only one girl is included in Cluster C and only two members are Black. One BD child has an ependymoma of the fourth ventricle and the other has congenital hydrocephalus (corrected by surgery). Compared to other clusters, factors (polar descriptions) characteristic of Cluster C are:

Factor 1: Anxious-aggressive social behavior
Factor 3: Emotional stability
Factor 5: Uncontrolled overactivity
Factor 6: Inhibition/avoidance

Relative to the other clusters, Cluster C individuals show a strong preference for activity. However, they seem to inhibit many of their impulses and they present acceptable social behavior. Moreover, these subjects show low anxiety and they are emotionally stable. Although they demonstrate learning and anxious motor problems, their scores on these factors do not distinguish them from other clusters.

Cluster D consists of 18 individuals, two of whom were originally classified as normal (MBD=16). They are the youngest cluster, with a mean age of 9.0 years. Compared to previous clusters, they have a higher IQ (104) and a greater proportion of girls (5). Cluster D has the highest socioeconomic status of any cluster. All subjects are white. Compared to other clusters, factors (polar descriptions) characteristic of Cluster D are:

Factor 1: Anxious-aggressive social behavior
Factor 3: Emotional lability
Factor 5: Normal activity
Factor 6: Spontaneous approach

Children in this cluster are not clearly distinguishable from those in previous clusters with respect to learning behavior problems and motor incoordination. They have moderate difficulties in both of these areas. However, they are uninhibited and less active than previous clusters. Cluster D children are often moody and anxious. Generally, they present problems similar to Cluster B, though less severe.

Cluster E consists of 17 subjects, with a mean age of 9.5 years
and a mean IQ of 105, which is the highest IQ among the clusters. With the exception of one MBD classified subject, all of these children were originally assigned to the normal group. This cluster has the largest female composition (5) and contains three Black children. Compared to other clusters, factors (polar descriptions) characteristic of Cluster E are:

- Factor 1: Sociableness
- Factor 2: Achievement
- Factor 3: Emotional stability
- Factor 4: Relaxed motor coordination
- Factor 5: Normal activity
- Factor 6: Spontaneous approach

In all respects, this cluster represents a normal group of children. These children have no social or learning behavior problems, they are emotionally calm, and they are well-coordinated. They do not attack or withdraw from their environment. Instead, they tend to be deliberate, conscientious, and non-aggressive. These children seem to have a positive view of themselves and evoke favorable responses from others.

Cluster Profile Similarity

The profiles of cluster means across each factor, as well as the $r_p$ coefficients between clusters, are presented in Figure 3. The results indicate that the level and shape of the cluster profiles do not differ across the six factors at the a priori level of significance ($ps > .01$).

Canonical Analysis

Four separate CANONAs were calculated for Clusters B-E to
determine the relationship between individual's factor scores and their scores on the four early life-history variables. Cluster A was not included because the sample size is too small. The results yielded four sets of canonical variates for each cluster. None of the chi-square tests for overall significance that were conducted for each variate in each cluster were significant at the .01 level. For the present clusters, there appears to be no systematic relationship between individuals' factor scores and their scores on the four life-history variables.
DISCUSSION

In connection with the present study, the results of the factor analysis suggest that the multivariate approach effectively reduced the large number of variables to a smaller number of meaningful dimensions. The factors that emerged have significant loadings on variables from behavioral, educational, and neurological domains. Thirty of the thirty-six variables load onto factors in the present study. It should be noted that the names assigned to factors represent only an intuitive guess as to the nature of the underlying symptom and are inferred from the items loading on that factor.

Factor Analysis

The behaviors described by Factor 1, Anxious-aggressive social behavior vs. sociableness, appear to be similar to dimensions identified in previous studies. Conners (1970) extracted a factor called "aggressive conduct disorder" which has loadings on parent behavior ratings of their hyperactive MBD child. As in the current study, the factor in Conners' study loaded on items such as "childish or immature, overasserts self, restless, temper tantrums, problems making friends, lying, and problems in school." Factor 1, however, loads on variables that describe a personality problem also. There is an anxious component to the child's conduct.

Conduct disorders and personality problems represent two dimensions that have been found in the behavior of a wide range of childhood psychopathology (Peterson, 1961; Quay & Werry, 1979). Based on parent
behavior ratings, a conduct disorder involves generally disturbing behavior and poor social relationships with adults and peers as well as both verbal and physical aggressiveness. A personality problem is defined as low self-esteem, social withdrawal, and dysphoric mood. The behaviors characteristic of the MBD children on **Factor 1** appear to correspond to both conduct and personality problems.

It should be noted that, of the fourteen factors from the CBCP instrument that distinguished MBD children in Fitch's (1976) research, ten of them load onto **Factor 1**, which supports his notion that MBD children tend to be viewed in a negative light by their parents. Moreover, BD children do not differ from normal children in the parent's assessment of their social behavior. For the MBD child, the possibility that negative parental response can exacerbate symptoms has been discussed (Battle & Lacy, 1972; Bettelheim, 1973) and, based on the present results, these effects demand further investigation.

Previous studies using factor analysis on an MBD population generally have not included separate tests of school performance. Conners (1973) extracted a factor he named "achievement" which was loaded with the spelling, reading, and arithmetic subtests of a wide-range achievement test. However, **Factor 2** in the present study, Learning behavior problems vs. achievement, is unique in that it not only loads onto three separate tests of school achievement, but also loads onto other variables that provide a more thorough profile of learning behavior. **Factor 2** includes parent's rating of achievement, the teacher's rating of classroom performance, the neurologist's evaluation of central nervous system functioning, and certain assumed
underlying factors of intelligence, e.g., WISC-R sequential and con­ceptual abilities (Bannatyne, 1968; 1974). The test data also confirm previous findings which show that children with learning problems obtain lower scores on the sequential factor (Bannatyne, 1968; 1974; Rugel, 1974; Smith, et al., 1977). It is tempting to suggest that children who score high on Factor 2 have a learning disability based on poor sequential organization and conceptual thinking which disrupts their performance in reading, spelling, and arithmetic and leads to poor motivation and disruptive classroom behavior.

Factor 3 in the present study is unique in that it describes the child's subjective sense of anxiety and emotional stability. Similar factors in previous studies of MBD include "anxious impulsivity" (Conners, 1970) and "psychopathology" (Werry, 1968), but these factors were derived from parent ratings in the former and psychiatric inter­views in the latter. Although one variable loading on Factor 3, Emotional stability vs. emotional lability, is the Teacher's report, the other six weights are from the CPQ and reflect the child's self­report of his emotional state. Interestingly, for both groups and clusters, children who score high on Factor 3 also have scores on Factor 1 which indicate anxious-aggressive social behavior problems. Since Factor 1 is derived from information obtained from parents and teachers, the data suggest agreement between other's perceptions and the child's own perception of his emotional stability. This sort of validation has not been reported in previous studies of MBD children.

In a study of a general child clinic population, Harris, Drummond, & Schultz (1977) identified six CPQ factors that were significant to
what they called a "conduct disorder." These are the same six CPQ variables that load onto Factor 3 in the present study. Harris, et al., also found that three of these six identified a "personality problem." A description of conduct disorders and personality problems has already been given. Again, it seems that MBD children display behaviors characteristics of both conduct and personality problems. It would be interesting to compare a group of MBD children with a conduct disorder group and a personality problem group to investigate the similarities and differences. It may be that, because the MBD child is anxious and unstable, he elicits negative feedback from his environment.

The variety of variables which load on Factor 4 are intuitively named Anxious motor incoordination vs. relaxed motor coordination. Actually the loadings on this factor contain many of the same items found to load on similar factors in previous studies. Some of these other factors were called "perceptual deficits and motor incoordination" (Werry, 1968), "motor incoordination" (Paine, et al., 1968), and "poor eye-hand ability" (Crinella, 1973). All of these factors, like the one in the present study, have loadings on neurological signs and paper-and-pencil tests of visual-motor functioning. However, Factor 4 in this study also includes specific behavioral correlates of motor incoordination. For example, children who score high on this factor do not concentrate well and they often play alone. The high loading on seclusiveness suggests that these children withdraw from others, which could be due to a poor self-image connected with their incoordination. Anthony (1973) contends that MBD children have an impaired body
image that results in a poor self-image. Of course, it is also possible that children with motor problems play alone because they are physically restricted in their ability to participate in many of the games of their peers.

Factor 5, Uncontrolled overactivity vs. normal activity, is unique in the same sense as Factor 3; that is, both give evidence which suggests that the child's subjective experience agrees with what others are reporting about him. Children who report high scores on Factor 5 through their performance on the MCPS and the CPQ also receive poor ratings from parents and teachers on Factor 1 with respect to their social behavior. Excessive activity has traditionally been the hallmark of MBD and is often found in factor analytic studies (Conners, 1970; Werry, 1968; among others). Such studies, however, are usually based on parent ratings and not the child's point of view. Observational studies of MBD children have not consistently shown that they have increased levels of activity (Cantwell, 1975). It would be interesting to observe MBD children who have rated themselves on activity to see if there is a relationship between their assessment and their actual behavior.

The type of activity reported by high scorers on Factor 5 seems to be an indiscriminate preference for physical movement. The child is not reporting purposeful activity or activity directed towards a goal. Indications are the child engages in aggressive activities, not out of anger, but because aggression permits an additional outlet for his activity. Since this factor also loads on a variable in which children describe themselves as careless or uncontrolled, they seem to recognize
that their behavior is disruptive, but they do not have the ability to
control their drive. In many ways the type of activity described by
Factor 5 is similar to the "organic driveness" characteristic of some
postencephalic children (Kahn & Cohen, 1933).

Factor 6, Spontaneous approach vs. Inhibition/avoidance, is
similar to the "anxious-inhibited" factor Conners (1970) found in a
study of hyperactive MBD children. The factor in Conners' study loaded
onto variables from a parent rating scale, as did half of the loadings
for Factor 6 in the present study. Again, the current factor includes
loadings derived from the child's report which tends to corroborate
his parent's report. All of the behaviors described by the variable
weights represent avoidance rather than approach reactions, which is
consistent with the inhibition factor frequently found in general child
clinic populations (Peterson, 1961; Quay & Werry, 1979). Such children
tend to withdraw rather than attack, or they isolate themselves rather
than participate. The children not only avoid social situations but
also inhibit the expression of impulses.

MBD subjects in the present study have the highest relative scores
on both uncontrolled overactivity and inhibition/avoidance behavior.
Although this may appear to be inconsistent, it suggests an interesting
possibility for speculation. It may be that MBD children view them-
selves as potentially much more disruptive and overactive than they are,
and that, from their perspective, they are avoiding many unacceptable
impulses.

Group Comparisons

Comparisons of groups across each factor reveal that MBD children
differ the most from the others and that BD children are identified primarily by their motor and learning problems. In fact, BD children are similar to normal children in their social and emotional behavior. Among group profiles, the MBD pattern is inversely related to the normal pattern, suggesting that the greatest contrast is between MBD and normal children. Also, the mean group scores on Factor 3 suggest a pattern that is not contradictory to the above results. The obtained probability (.03) in the univariate ANOVA does not meet the \textit{a priori} criterion; post-ANOVA testing suggested that the MBD group has more emotional lability than either the BD or normal groups (p < .05). Again, the BD and normal groups do not differ.

These tendencies, along with the obtained results that are significant at the \textit{a priori} level, are consistent with the results reported by Fitch (1976). Thus, the present study rejects the notion of a BD behavior model of MBD, wherein MBD behavior represents a "minor" form of BD behavior. It does not appear tenable to assume that the social and emotional behavior of MBD children represents an intermediate point on a continuum of "organicity." On the basis of behavior, MBD seems to be a distinct entity, as suggested by Wender (1971), and not a lesser form of BD as suggested by Ingalls and Gorden (1937) and Knobloch and Pasamanick (1966). Rather, it would seem that, if a continuum for social and emotional behavior exists, then MBD and normal conditions represent the end-points, with BD behavior between the two (though probably closer to the normal end).

\textbf{Cluster Analysis}

Are the various clusters, or "types" of children grouped together
by the cluster analysis meaningful classifications of psychological functioning? Do these groupings of children reflect behavioral communality? Do the groupings vary with respect to early life history variables and, perhaps, with respect to etiologies? These are the kinds of questions that the cluster analysis was meant to answer.

In Cluster A the number of BD individuals and the extent of their motor impairment (almost four S.D.s above other clusters) suggests the presence of an organic syndrome. Crinella (1973) performed a cluster analysis of BD, MBD, and normal children based on their scores on sixteen factors. One cluster he identified had a normal IQ but a striking number of visual-motor and coordination problems. These BD and MBD children also tended to be irritable, confused, and not well-adapted socially. This cluster in Crinella's study seems similar to Cluster A in the present study. Generalizations made from the pattern of scores in Cluster A should be interpreted with caution because they are based on a very small sample (n=4).

However, the data suggest that some BD children exhibit a set of related behaviors which are associated with severe motor impairment. Interestingly, one of the children in this cluster was originally diagnosed as MBD, which would lead one to suspect that there are some MBD children who are actually BD but have been misclassified. Although Cluster A children demonstrate problems in the social and learning behavior areas also, relative to their anxious motor incoordination, these problems are not outstanding. It may be that the social and learning problems they exhibit are secondary to, or a consequence of, their motor impairment. Thus, their clumsiness and incoordination lead
to poor school performance and restrict their social development.

Cluster B presents the most psychopathology relative to the other clusters. Crinella (1973) described a similar cluster in his study which he termed "Irascible." These children had school behavior problems, emotional outbursts, especially of an aggressive nature, and hyperactivity and restlessness. Conners (1973) also described a cluster of MBD diagnosed children with very poor impulse control and very poor learning and achievement. The clusters in Conners' study, however, were not based on factors from objective self-report inventories. In the present study, Cluster B subjects report a great deal of subjective anxiety and emotional instability. Strong tendencies toward avoidance and inhibition are also reported, which may explain the absence of overactive behavior. Racial and environmental influences are also operating in this syndrome, as Blacks account for a higher proportion of the members of this cluster than any other, and Cluster B has the lowest socioeconomic status of any cluster. Granting that this is the most problematic cluster, this finding supports the notion that Black, lower-class MBD children have more deficits than their white middle-class counterparts (Willerman, 1973; Wender, 1971). Perhaps Wender's (1971) "privation-produced" form of MBD deserves further consideration.

Cluster C in the current study seems to have much in common with clusters identified in previous studies (Crinella, 1973; Conners, 1973; Reitan & Boll, 1973). Crinella described a group of MBD and BD children with average IQs and moderate scores on perceptual-motor factors who, nevertheless, exhibited severe learning difficulties. There was an absence of emotional problems in these children. They also demonstrated
appropriate social behavior. Conners' cluster was low in achievement and learning but good in impulse control. Conners described this cluster further on the basis of parent rating scales evaluated after the formation of the clusters. These children were not rated as anxious and they behaved in a mature fashion for their age. Although Reitan and Boll defined their clusters a priori (academic vs. behavior problems) they were able to establish an empirical basis for their groupings with a large number of psychometric tests. MBD children with academic problems were more impaired than behavior problem individuals on measures of intelligence, achievement, and sensory-motor functioning.

As in the above clusters, Cluster C in the present study is characterized by emotional stability and good social behavior. Yet, Cluster C children have many learning problems. Although these children report that they are overactive, they also score high on the inhibition/avoidance factor. Since these children are older than children in other clusters, it is tempting to suggest that they have attained some adjustment to their problems with activity and learning, so that they are able to inhibit impulses that would tend to get them into trouble. They are not viewed as a social behavior problem. At any rate, these children subjectively feel that they have attained some adjustment to their environment and others perceive that they are sociable and obedient.

The behaviors characteristic of Cluster D have much in common with those described in Cluster B. That is, these children are emotionally labile and they have both social and learning behavior problems.
Accordingly, they are similar to groups of children in studies by Crinella (1973), Conners (1973), Peterson (1961), and Quay and Werry (1979). **Cluster D** individuals differ from **Cluster B** subjects in that they demonstrate spontaneous approach behaviors and they have normal levels of activity. In fact, **Cluster D** children show so much approach behavior that they seem almost disinhibited, which may contribute to their poor social behavior. Generally though, they seem less disturbed than **Cluster B** individuals across all factors.

**Cluster D** is also the youngest cluster and comes from the highest socioeconomic status family. The age difference for this cluster suggests that there may be developmental effects operating in the MBD child's behavior. One can speculate that **Cluster D** children might develop a profile similar to **Cluster B** if their behavior continues to deteriorate as they get older. On the other hand, it is possible that **Cluster D** children might tend to be more like **Cluster C** if they develop more control and higher levels of inhibition and avoidance behavior as they get older. Regarding socioeconomic status, Willerman (1973) proposed that the child-rearing practices of higher socioeconomic status mothers could compensate for impairment in MBD children. Whatever the reason, the current finding that white, higher social class children have less impairment than Black, lower class children is consistent with other research (Bernstein, 1962; Mischel, 1966; Wender, 1972; Willerman, 1973). For **Clusters B** and **D** in the present study, the results indicate that environmental and racial effects contribute to the pattern of behaviors in these children. Further research is needed, however, to test these hypotheses.
As pointed out in the Results section, Cluster E represents a normal group of children. All but three of the normal children in the sample are classified in this cluster. Although the present study suggests there is not much overlap between MBD diagnosed children and normal children (only one child in 18 is MBD in Cluster E), the necessity of a control group seems inherent in good research design. Not only does the normal group in the present study serve as a check on the clustering technique, it provides a basis for comparisons and generalizations among problem groups.

Cluster Comparisons

With respect to the original group classifications, MED children show the least overall similarity of factor profiles. An MBD child is present in every cluster obtained. The BD and normal groups have individuals placed in three of the five clusters. Thus, the cluster analysis confirms the notion that MBD children constitute a heterogeneous assortment (Cantwell, 1975; Conners, 1973; De La Cruz, et al., 1973; among others). In fact, contrary to what was predicted, MBD children show less similarity of factor profiles than the heterogeneous BD group. As predicted, normal children show the greatest similarity of factor profiles and the most behavioral communality of any of the original group classifications. Finally, it should be pointed out that sex of the subjects does not appear to be an important variable for cluster membership. Girls were present in every cluster and the proportion did not vary much from cluster to cluster.
Canonical Analysis

The results of the canonical analysis fail to indicate that certain early life history events can predict cluster membership. The data do not suggest any possible etiologies for the obtained clusters. Perhaps it would have been more fruitful to examine cluster relationships with respect to motor development, response to drug therapy, or cortical evoked response as suggested by Conners (1973). Conners found significant differences on the above variables in post-hoc comparisons of his clusters.

Although the clusters did not vary systematically with the life history variables, there may have been a relationship with the original group classifications. The BD group has the highest relative means on each of the four variables. Also, the MBD group has more signs on each of the four variables than the normal group has. Such tendencies deserve exploration in future research. The notion that MBD classified children have a greater incidence of pre-, peri-, and post-natal insults (Dargassies, 1977; Paine, et al., 1968; Werry, 1968) is not automatically refuted by the present findings. Rather, the various "subtypes" of MBD do not appear to differ with respect to their early life history.

Limitations of the Study

Differences between groups are not attributable to differences in age, race, sex, or socioeconomic status. However, the normal group has a higher IQ than the MBD group, and the BD group has the lowest IQ. Inspection of group means on factors reveals that this pattern is followed on only two of the factors, Factors 2 and 4. BD subjects obtained
the highest scores on Learning behavior problems and Anxious motor incoordination. One would expect such a finding, but it is not clear whether a low IQ accounts for the factor scores or whether learning and motor problems account for low IQ test scores. In the actual search for BD subjects, it was clear that this pattern was virtually unavoidable with the requirements for selection specified earlier.

Although the univariate ANOVA for age across factors was not significant at the a priori level, it could be argued that the obtained alpha of .05 in some way accounts for differences between groups. However, the results of a Duncan post-ANOVA test revealed that there were no differences between groups with respect to age (p > .05). It might be argued that the obtained dimensions are "instrument factors" (Cattell, 1977) because some of the factors have most of their weights on one specific test. Factor 1 and Factor 3 have heavy loadings on particular tests (the CBCP instrument for the former and the CPQ for the latter). Instrument factors have been criticized by Cattell because they may represent biases in measurement arising from the structure of the instrument, the way it is scored, etc. However, in the present study, instrument variables do not appear to distort the factor, e.g., high scores on the CBCP Disobedient variable load on Factor 1 to indicate more social behavior problems, while a high score on the CBCP Appreciative variable indicates more agreeable social behavior and, accordingly, has an inverse loading on Factor 1. In addition, it should be borne in mind that no factor in the present study has all its loadings from a single instrument. Therefore, stigmatizing the results as instrument factors does not seem justifiable.
Also, the results of the present study might be limited by developmental differences between the groups. The BD children were all diagnosed at an early age and had been receiving special treatment from significant others for a long time. The MBD group, on the other hand, was generally being evaluated for the first time and had not had the privilege of special treatment. Parents and teachers had probably already adjusted to the BD child's problems, while the MBD child was in the process of being unable to adjust to his environment (which was the reason he was referred). The MBD child was possibly receiving negative feedback and may have been having difficulty in his interactions with others. Such events could have adversely affected his scores, accounting for his emotional instability and poor parent and teacher ratings.

It would be very difficult to design a study that would overcome these developmental differences. The present results show that there are no clear signs that identify MBD early in life, while BD children, as a rule, are identified before they enter school (Wender, 1972). Finding a sufficient number of MBD and BD children for study who had matched developmental histories would be a formidable task.

Another problem involved with classification which is confronted by many researchers is that of the clustering technique used. Presently, numerous clustering techniques exist, ranging from hand analyses to computerized programs. The difficulty with most of these techniques is that there is no precise mechanical way to go about determining the number of clusters to be extracted. Current procedures rely heavily on a "cut-and-fit" approach, wherein the researcher
examines the clusterings and selects the most meaningful aggregation. Fortunately, Tzeng and May (1979) are working on a rotational technique for hierarchical clusters that will enable researchers to arrive at meaningful groupings in an empirical manner.

Concluding Remarks

The results of the present study show that the MBD classification does in fact describe a group of children who share some behavioral communality. If the MBD diagnosis can differentiate them from BD and normal children, then why proceed to regroup the MBD children further? The cluster analysis reveals that this group of MBD individuals segregates into subtypes that have profiles different from the original MBD group. This finding implies that different treatment strategies are necessary to deal with the child's problems. For example, Cluster C subjects have few problems with their social or emotional behavior, but they have many learning problems. Educational remediation would seem to be the primary focus for dealing with these children. Cluster B children not only have learning problems, but also emotional lability and social maladjustment. These children would need a more diverse approach in treatment. Their learning skills could be developed by educational remediation. They could be taught more effective ways of coping in behavior therapy. Group therapy could be employed to enhance their social skills. Additional research might show that there is a differential response to medication among cluster types. For example, Conners (1973) found a cluster which is similar to Cluster B in the present study which showed a decrease in anti-social behavior with stimulant medication. However, children with behaviors similar to
Cluster C in the present study showed a minimal response to medication. In sum, it seems reasonable to proceed with classification schemes beyond the MBD categorization as long as the new categories provide meaningful groupings which have implications for treatment.

If one accepts the maxim proposed by Conners (1970), Strother (1973, and Werry (1968) that a diagnostic term should be a descriptive label and a prescription for action and not a statement of etiology, then the dysfunctions displayed by MBD children should be described in behavioral and experiential, rather than neurological terms. The present study shows that these children can be grouped effectively on the basis of their behaviors. This seems to be the most appropriate course of action until a biological model can be shown to be of primary relevance in the planning of treatment strategies for individual children.
REFERENCES


McGuire, E. & White, M. Index of Social Status Characteristics. "The measurement of social status." Research paper in human development, No. 3 (Revised), 1955, Department of Educational Psychology, U. of Texas, Austin, Texas.


Mann, L. "Perceptually training revisited. The training of nothing at all." Rehabilitation Literature, 1971, 32, 322-327.


Thompson, R., Arabie, G., & Sisk, G. "Localization of the 'incline plane discrimination memory system' in the white rat." Physiological Psychology, 1976, 4, 311-324.


CONSENT FORM

The present study requires the mother and child to answer some ques­
tions about that child's history and behavior. The child's physician
and teacher will be asked some questions concerning that child's
health and schoolwork. This information is being collected in an
effort to better classify the behaviors of children. No discomforts,
risks, or direct benefits are involved in the study. Information
collected will be kept confidential. Any questions concerning the
procedures will be answered by the project director.

I, the undersigned, understand the above explanations, have no
unanswered questions about the procedure at the present time, and
give consent to my voluntary participation in the research project
entitled, "An Empirical Analysis of School Age Children with Brain
Damage and Minimal Brain Dysfunction," to be done by Mr. Gregory Sisk.
I understand that answers to inquiries that I have concerning the
procedure of this activity will be given at any time. I understand
that I am free to withdraw my consent and to discontinue participa­
tion in this activity at any time.

__________________________________  ______________________________
Date                                                   Signature of subject or legally
authorized representative

__________________________________  ______________________________
Location                                               Signature of project director
APPENDIX B
### TEACHER RATING SCALE

1) **Makes good use of time in classroom**  
   - Often  
   - Sometimes  
   - Rarely

2) **Works independently**  
   - Often  
   - Sometimes  
   - Rarely

3) **Requires extra help**  
   - Often  
   - Sometimes  
   - Rarely

4) **Upset by change in routine**  
   - Often  
   - Sometimes  
   - Rarely

5) **Is distractible (attends to small noises and movements)**  
   - Often  
   - Sometimes  
   - Rarely

6) **Confused by groups other than his own**  
   - Often  
   - Sometimes  
   - Rarely

7) **Slow to begin classroom tasks**  
   - Often  
   - Sometimes  
   - Rarely

8) **Clumsy**  
   - Often  
   - Sometimes  
   - Rarely

9) **Quality of work varies a great deal**  
   - Often  
   - Sometimes  
   - Rarely

10) **Follows oral directions**  
    - Often  
    - Sometimes  
    - Rarely

11) **Follows written directions**  
    - Often  
    - Sometimes  
    - Rarely

12) **Academic performance in general**  
    - Low  
    - Average  
    - High
1) **Fine-motor incoordination.** Subject was asked to imitate examiner by: a) tapping each finger on thumb successively for each hand; b) extending tongue and moving it up, down, right, and left; c) drawing a triangle if age was less than nine, or drawing a Greek cross if age was over nine. Subject was given a score of zero (0) if all items were done correctly. Subject was given a score of one (1) if one item was missed. Subject was given a score of two (2) if two or more items were missed. 

2) **Gross-motor incoordination.** Subject was asked to imitate the examiner by: a) tandem walking forward; b) tandem walking backward; c) skipping for ten feet. Subject was given a score of zero (0) if all items were done correctly. Subject was given a score of one (1) if one item was missed. Subject was given a score of two (2) if two or more items were missed. 

3) **Balance.** Subject was asked to imitate the examiner by lifting one foot (subject's choice) and placing it against the opposite knee while keeping both arms at his side. Subject was given a score of zero (0) if during a 15-second period, the elevated foot did not touch the floor, the arms were not used, the floor foot was not shifted, or torso gyrations were not used to maintain balance. Subject was given a score of one (1) if he did any of the above during the 15-seconds. Subject was given a score of two (2) if he could not place one foot against the opposite knee. 

4) **Directionality.** Subject was asked to: a) identify his right and left hand and foot; b) identify the examiner's right and left hand and foot when examiner was facing subject; c) identify the examiner's right and left hand and foot when the examiner has his back to the subject. Subject was given a score of zero (0) if all items were done correctly. Subject was given a score of one (1) if one item was missed. Subject was given a score of two (2) if two or more items were missed. 

5) **Synkinesis.** Subject was asked to imitate the examiner by: a) extending and wagging tongue up, down, right, and left; b) tapping index finger to thumb on right hand and then left at the rate of one tap per second for 15-seconds. Subject was given a score of zero (0) if both items were done correctly and if jaw movements did not accompany tongue-wagging and if thumb or other finger movement did not accompany finger-tapping. Subject was given a score of one (1) if he displayed accompanying movements on either item. Subject was given a score of two (2) if he displayed accompanying movements on both items.
NEUROLOGICAL EXAMINATION (cont.)

6) Babinski sign. Subject was asked to remove shoes and socks and lie down. Examiner used fingertips to lightly stimulate sole of each foot. Subject was given a score of zero (0) if there was a flexion of the toes instead of an extension. Subject was given a score of one (1) if there was a dorsal extension of the great toe and a spreading apart of the toes on one foot. Subject was given a score of two (2) if the above sign appeared on both feet.

7) Strabismus. Subject was asked to look at examiner and to follow examiner's thumb with his eyes as the examiner moved his thumb. Examiner stood approximately three feet away from subject and moved his thumb up, down, right, and left. Subject was given a score of zero (0) if both eyes were on proper axis and if eye-tracking was done correctly. Subject was given a score of one (1) if eyes were on proper axis but eye-tracking was done incorrectly. Subject was given a score of two (2) if either eye was not on its proper axis.

8) Motor-impersistence. Subject was given a plastic tube, "telescope," and asked to look thru it. Subject was given a ball and asked to throw it to the examiner. Subject was asked to stand approximately five feet away from ball on the floor. Subject was then asked to walk up to ball and kick it with his foot. Subject was given a score of zero (0) if eye, hand, and foot preference were all unilateral. Subject was given a score of one (1) if he showed mixed preference on any item. Subject was given a score of two (2) if he could not look through tube or throw or kick the ball.

9) Graphesthesia. Subject was told that numbers would be "drawn" on his hand. Subject was asked to close his eyes while examiner used eraser tip of a pencil to draw single digit numbers on the palm of the dominant hand. Three trials were given with different numbers given on each trial. Subject was given a score of zero (0) if he identified the number correctly on each trial. Subject was given a score of one (1) if one number was identified incorrectly. Subject was given a score of two (2) if two or more numbers were identified incorrectly.

10) Other signs. If other signs were noted during the examination, e.g., choreoathetoid movements, gait disturbances, speech or hearing impairment, inability to follow directions, etc., this category was scored. Subject was given a score of one (1) if sign was present. Subject was given a score of two (2) if sign was present and pronounced.
LIFE-HISTORY DATA

PREGNANCY

Maternal age at time of birth-score if age is less than 17
Previous miscarriages-score for each previous miscarriage
Previous premature births-score for each previous premature birth
Previous abortions-score for each previous abortion
Disease related to infection, e.g., polio, rubella, influenza,
    smallpox, cowpox, chickenpox, common measles, etc.
Hemorrhages-score for each event of hemorrhaging
Threatened miscarriage
Toxemia
Anemia
X-rays
High blood pressure
Medication-does not include vitamins
Emotional problems-received or sought consultation during pregnancy
Duration of pregnancy-score if premature by at least two weeks

DELIVERY

Birthweight-score if weight less than six pounds
Complicated delivery
Long labor-score if labor was longer than nine hours
Anesthesia-does not include spinal blocks
Premature rupture of membranes
Precipitous labor
Emergency cesarean section
Abnormal presentation
Forcep delivery
Cord around neck
Fetal distress syndrome

INFANCY

Resuscitation
Jaundice
Cyanosis
Apnoeic spells
Anaemia
Convulsions
Incubator
Feeding problems
Poor cry
Hematoma
Intracranial hemorrhage/elevated CSF fluid
Neoplastic processes
LIFE-HISTORY DATA

EARLY CHILDHOOD

Traumatic head injury—score for each incident where x-rays were taken and/or stitches were received.

Encephalitis
Hydrocephalus
Cerebrovascular disturbances
Meningitis
Anoxia
Seizures—score once if seizure occurred at any time
Operations—score for each operation
Hospitalizations—score for each hospitalization
Abscessed ears
Fever over 105° F
Colic
Diseases, e.g., measles, rubella, mumps, whooping cough, diphtheria, asthma, etc.—score once if any of the above diseases have occurred
### TABLE 1
DESCRIPTION OF BRAIN DAMAGED SUBJECTS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>103</td>
<td>Head trauma; hematoma on left posterior occipital lobe</td>
</tr>
<tr>
<td>104</td>
<td>Head trauma; posttraumatic brain syndrome; bilateral subdural hematoma</td>
</tr>
<tr>
<td>105</td>
<td>Cerebral palsy; spastic quadriplegia; focal epilepsy, left midtemporal lobe</td>
</tr>
<tr>
<td>106</td>
<td>Cerebral palsy; spastic paraparesis; focal epilepsy, left frontal lobe</td>
</tr>
<tr>
<td>107</td>
<td>Malignant glioma of left cerebellar hemisphere</td>
</tr>
<tr>
<td>108</td>
<td>Ependymonia of the fourth ventricle</td>
</tr>
<tr>
<td>109</td>
<td>Congenital hydrocephalus</td>
</tr>
<tr>
<td>110</td>
<td>Minimal cerebral palsy; left hemiparesis</td>
</tr>
<tr>
<td>111</td>
<td>Spina bifida; hydrocephalus</td>
</tr>
<tr>
<td>112</td>
<td>Cerebral palsy; left spastic hemiplegia and cranial nerve impairment</td>
</tr>
<tr>
<td>113</td>
<td>Postinfectious encephalitis; hydrocephalus</td>
</tr>
</tbody>
</table>
# TABLE 2

DESRIPTIVE STATISTICS AND TESTS OF SIGNIFICANCE
FOR EACH GROUP ON IDENTIFYING DATA

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group N=90</th>
<th>F-value</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BD n=11</td>
<td>MBD n=55</td>
<td>Normal n=24</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>10.4</td>
<td>9.4</td>
<td>10.0</td>
</tr>
<tr>
<td>S.D.</td>
<td>1.8</td>
<td>1.4</td>
<td>1.3</td>
</tr>
<tr>
<td>SES&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>3.3</td>
<td>3.4</td>
<td>3.7</td>
</tr>
<tr>
<td>S.D.</td>
<td>1.2</td>
<td>1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>IQ&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>80.4</td>
<td>100.9</td>
<td>107.0</td>
</tr>
<tr>
<td>S.D.</td>
<td>13.7</td>
<td>10.8</td>
<td>9.7</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>82%</td>
<td>89%</td>
<td>87%</td>
</tr>
<tr>
<td>Black</td>
<td>18%</td>
<td>11%</td>
<td>13%</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>64%</td>
<td>76%</td>
<td>63%</td>
</tr>
<tr>
<td>Female</td>
<td>36%</td>
<td>24%</td>
<td>37%</td>
</tr>
</tbody>
</table>

<sup>a</sup>Multiple comparisons were not calculated for nonsignificant F-values (p ≥ .01).
<sup>b</sup>Scores were derived from the McGuire-White Index (1955) and range from 1-7, with low scores indicating higher socioeconomic status.
<sup>c</sup>Full Scale IQ obtained from Wechsler Intelligence Scale for Children-Revised (1974).
<sup>d</sup>Percentages were rounded-off.
<table>
<thead>
<tr>
<th>Variable Description</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>h²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>61</td>
</tr>
<tr>
<td>2. SES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>51</td>
</tr>
<tr>
<td>3. WISC-R Conceptual factor</td>
<td>46</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>71</td>
</tr>
<tr>
<td>4. WISC-R Spatial factor</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>76</td>
</tr>
<tr>
<td>5. WISC-R Sequential factor</td>
<td>85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>81</td>
</tr>
<tr>
<td>6. BVRT Visuomemory ability</td>
<td>65</td>
<td></td>
<td></td>
<td>-62</td>
<td></td>
<td></td>
<td>79</td>
</tr>
<tr>
<td>7. MCPS Aggression</td>
<td></td>
<td>-59</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>69</td>
</tr>
<tr>
<td>8. MCPS Inhibition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>79</td>
<td></td>
<td>76</td>
</tr>
<tr>
<td>9. MCPS Activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-72</td>
<td></td>
<td>68</td>
</tr>
<tr>
<td>10. CPQ Reserved vs. warmhearted</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>71</td>
</tr>
<tr>
<td>11. CPQ Affected by feelings vs. emotionally stable</td>
<td>-69</td>
<td></td>
<td></td>
<td></td>
<td>-41</td>
<td></td>
<td>71</td>
</tr>
<tr>
<td>12. CPQ Phlegmatic vs. excitable</td>
<td></td>
<td>82</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>77</td>
</tr>
<tr>
<td>13. CPQ Obedient vs. assertive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>85</td>
</tr>
<tr>
<td>14. CPQ Expedient vs. conscientious</td>
<td></td>
<td>-59</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>64</td>
</tr>
<tr>
<td>15. CPQ Shy vs. venturesome</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-58</td>
<td></td>
<td>75</td>
</tr>
<tr>
<td>16. CPQ Uncontrolled vs. controlled</td>
<td></td>
<td>-61</td>
<td></td>
<td></td>
<td>-43</td>
<td></td>
<td>78</td>
</tr>
<tr>
<td>17. CPQ Relaxed vs. tense</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>79</td>
<td>71</td>
</tr>
<tr>
<td>18. CBCP Appreciative, concerned, obedient, social orientation vs. unappreciative, aggressive disobedience</td>
<td>65</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>72</td>
</tr>
</tbody>
</table>

Matrix contains only salient variable weights, i.e., loadings exceed twice the critical value of a Pearson r at the .05 level (r ≥ .37).

Decimal points have been omitted.
<table>
<thead>
<tr>
<th>Variable Description</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
<th>Factor 4</th>
<th>Factor 5</th>
<th>Factor 6</th>
<th>$h^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>19. CBCP Intellectual and scholastic retardation vs. alert socialized achievement</td>
<td>-79</td>
<td>-49</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>86</td>
</tr>
<tr>
<td>20. CBCP self-derogating school phobia</td>
<td>-63</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>37</td>
</tr>
<tr>
<td>21. CBCP Disobedient, sullen, hyperactive aggressiveness</td>
<td></td>
<td>-79</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>86</td>
</tr>
<tr>
<td>22. CBCP Anti-social aggressiveness</td>
<td>-79</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>79</td>
</tr>
<tr>
<td>23. CBCP Negativism vs. peer-aggressive obedience to authority</td>
<td></td>
<td>-77</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>71</td>
</tr>
<tr>
<td>24. CBCP Temper tantrums</td>
<td></td>
<td></td>
<td></td>
<td>-77</td>
<td></td>
<td></td>
<td>70</td>
</tr>
<tr>
<td>25. CBCP Phobic, negativistic, finicky eating vs. positive eating</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-69</td>
<td>56</td>
</tr>
<tr>
<td>26. CBCP Immature, neurasthenic paranoia reactions</td>
<td></td>
<td></td>
<td></td>
<td>-86</td>
<td></td>
<td></td>
<td>76</td>
</tr>
<tr>
<td>27. CBCP Fearful desurgent seclusiveness vs. sociableness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-47</td>
<td></td>
<td>63</td>
</tr>
<tr>
<td>28. CBCP Verbal psychoid reactions</td>
<td>-80</td>
<td></td>
<td></td>
<td>41</td>
<td></td>
<td></td>
<td>79</td>
</tr>
<tr>
<td>29. CBCP Anxious organicism</td>
<td></td>
<td></td>
<td></td>
<td>72</td>
<td></td>
<td></td>
<td>59</td>
</tr>
<tr>
<td>30. CBCP Clumsiness and visual problems</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-47</td>
<td></td>
<td>73</td>
</tr>
<tr>
<td>31. CBCP Displaced aggressiveness vs. direct aggressiveness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-57</td>
<td>37</td>
</tr>
<tr>
<td>32. WRMT (Woodcock) Reading</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-69</td>
<td>37</td>
</tr>
<tr>
<td>33. KMDT (KeyMath) Arithmetic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>39</td>
<td>78</td>
</tr>
<tr>
<td>34. TWS Spelling</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>81</td>
</tr>
<tr>
<td>35. Teacher's report</td>
<td>-57</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>39</td>
<td>81</td>
</tr>
<tr>
<td>36. Neurological examination</td>
<td></td>
<td>-51</td>
<td></td>
<td></td>
<td></td>
<td>74</td>
<td>78</td>
</tr>
</tbody>
</table>
TABLE 4
MEANS, STANDARD DEVIATIONS, AND SIGNIFICANCE TESTS ON FACTOR SCORES FOR EACH GROUP

<table>
<thead>
<tr>
<th>Factor</th>
<th>Group</th>
<th>F-value</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BD</td>
<td>MBD</td>
<td>Normal</td>
</tr>
<tr>
<td>Factor 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>.42</td>
<td>- .46</td>
<td>.95</td>
</tr>
<tr>
<td>S.D.</td>
<td>.76</td>
<td>.89</td>
<td>.42</td>
</tr>
<tr>
<td>Factor 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-1.08</td>
<td>- .33</td>
<td>1.20</td>
</tr>
<tr>
<td>S.D.</td>
<td>.76</td>
<td>.66</td>
<td>.60</td>
</tr>
<tr>
<td>Factor 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>- .41</td>
<td>.23</td>
<td>- .41</td>
</tr>
<tr>
<td>S.D.</td>
<td>.70</td>
<td>.98</td>
<td>1.02</td>
</tr>
<tr>
<td>Factor 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>1.90</td>
<td>- .02</td>
<td>- .65</td>
</tr>
<tr>
<td>S.D.</td>
<td>1.43</td>
<td>.73</td>
<td>.29</td>
</tr>
<tr>
<td>Factor 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>- .02</td>
<td>- .16</td>
<td>.39</td>
</tr>
<tr>
<td>S.D.</td>
<td>.45</td>
<td>1.09</td>
<td>.83</td>
</tr>
<tr>
<td>Factor 6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>- .15</td>
<td>.13</td>
<td>- .26</td>
</tr>
<tr>
<td>S.D.</td>
<td>1.12</td>
<td>1.11</td>
<td>.55</td>
</tr>
</tbody>
</table>

*Multiple comparisons were not calculated for nonsignificant F-values (α = .01).
<table>
<thead>
<tr>
<th>Factor</th>
<th>Cluster</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A n=4</td>
<td>B n=18</td>
<td>C n=15</td>
<td>D n=18</td>
<td>E n=17</td>
</tr>
<tr>
<td>Factor 1</td>
<td>Mean</td>
<td>- .40</td>
<td>- .69</td>
<td>.19</td>
<td>- .32</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>1.24</td>
<td>1.05</td>
<td>.56</td>
<td>.83</td>
<td>.41</td>
</tr>
<tr>
<td>Factor 2</td>
<td>Mean</td>
<td>- .45</td>
<td>- .56</td>
<td>- .39</td>
<td>- .17</td>
<td>1.22</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>.73</td>
<td>.84</td>
<td>.60</td>
<td>.84</td>
<td>.60</td>
</tr>
<tr>
<td>Factor 3</td>
<td>Mean</td>
<td>.13</td>
<td>.69</td>
<td>- .57</td>
<td>.44</td>
<td>- .73</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>1.09</td>
<td>.80</td>
<td>.77</td>
<td>.80</td>
<td>.83</td>
</tr>
<tr>
<td>Factor 4</td>
<td>Mean</td>
<td>3.25</td>
<td>- .07</td>
<td>.11</td>
<td>- .20</td>
<td>- .57</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>.43</td>
<td>.63</td>
<td>.58</td>
<td>.69</td>
<td>.36</td>
</tr>
<tr>
<td>Factor 5</td>
<td>Mean</td>
<td>- .41</td>
<td>- .16</td>
<td>- .78</td>
<td>.44</td>
<td>.48</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>.94</td>
<td>.78</td>
<td>.87</td>
<td>.93</td>
<td>.97</td>
</tr>
<tr>
<td>Factor 6</td>
<td>Mean</td>
<td>- .25</td>
<td>.49</td>
<td>.35</td>
<td>- .63</td>
<td>- .11</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>1.56</td>
<td>1.21</td>
<td>.75</td>
<td>.78</td>
<td>.67</td>
</tr>
<tr>
<td>Group</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>BD</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>MBD</td>
<td>1</td>
<td>16</td>
<td>12</td>
<td>16</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>16</td>
<td></td>
</tr>
</tbody>
</table>

\[ N = \]

\[ 4 \quad 18 \quad 15 \quad 18 \quad 17 \]
FIGURE 1
SCREE TEST FOR FIFTEEN FACTORS

Eigenvalue vs. Factor Number
FIGURE 2

GRAPH OF MEAN FACTOR SCORES BY GROUP

<table>
<thead>
<tr>
<th>Group</th>
<th>BD</th>
<th>MBD</th>
<th>Norm</th>
</tr>
</thead>
<tbody>
<tr>
<td>BD</td>
<td>1.00</td>
<td>-0.93</td>
<td>-0.26</td>
</tr>
<tr>
<td>MBD</td>
<td>1.00</td>
<td>-0.35</td>
<td></td>
</tr>
<tr>
<td>Norm</td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
</tbody>
</table>
FIGURE 3

GRAPH OF MEAN FACTOR SCORES BY CLUSTER

<table>
<thead>
<tr>
<th>Cluster A</th>
<th>Cluster B</th>
<th>Cluster C</th>
<th>Cluster D</th>
<th>Cluster E</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Cluster A" /></td>
<td><img src="image2" alt="Cluster B" /></td>
<td><img src="image3" alt="Cluster C" /></td>
<td><img src="image4" alt="Cluster D" /></td>
<td><img src="image5" alt="Cluster E" /></td>
</tr>
</tbody>
</table>

**Coefficient of Profile**

<table>
<thead>
<tr>
<th>Similarity Matrix</th>
<th>Cluster B</th>
<th>Cluster C</th>
<th>Cluster D</th>
<th>Cluster E</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.00</td>
<td>0.11</td>
<td>-0.10</td>
<td>-0.28</td>
</tr>
<tr>
<td>B</td>
<td>1.00</td>
<td>-0.08</td>
<td>0.05</td>
<td>-0.35</td>
</tr>
<tr>
<td>C</td>
<td>1.00</td>
<td>-0.37</td>
<td>-0.08</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>1.00</td>
<td>-0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Factor Number**

1 2 3 4 5 6

**Factor Number**

Mean Factor Score

1.00 0.11 -0.10 -0.28

1.00 -0.08 0.05 -0.35

1.00 -0.37 -0.08

1.00 -0.15

1.00
NAME: Gregory Blaine Sisk

ADDRESS: 1330 Jim Taylor Ave., #3
          Baton Rouge, Louisiana, 70808
          Telephone: 504-766-6602

BIRTHDATE: August 22, 1953; Poplar Bluff, Missouri

EDUCATION:
1971, Graduated from public high school, Poplar Bluff, Missouri
1974, B.A., cum laude, University of Missouri, Columbia, Missouri
1976, M.A., Louisiana State University, Baton Rouge, Louisiana
1980, Ph.D., Louisiana State University (Major: Clinical Psychology
         Minor: Behavioral Neurology  APA Approved)

HONORS AND
ORGANIZATIONS:
University of Missouri: Curator's Scholar, 1971-74; Dean's List,
          1971-74; Phi Eta Sigma, 1972; Ollie B. Reed Scholar, 1974; Psi Chi, 
          1973-74; General Honors Certificate, 1974; Psychology Honors Program,
          1974; Phi Beta Kappa, 1974.

Louisiana State University: Dean's List, 1974-80; Phi Kappa Phi, 1976;
          Sigma Xi, 1976; APA Student Member, 1977; Louisiana Competent Authority
          for Special Education, 1977 (Lic. # 000102); NIMH Training Fellow, 1979.

TRAINING EXPERIENCE:
1974-77 Teaching and Research Assistant. Dept. of Psychology, LSU.
1976-77 Practicum. LSU Counseling and Mental Health Service.
1978-79 Internship. Western Missouri Mental Health Center, Kansas City,
          Missouri (APA Approved).
1978 Workshop. Marriage and Family Therapy II, The Menninger Clinic,
          Kansas City, Kansas.

WORK EXPERIENCE:
1975-77 Associate Faculty Member. School of Social Welfare, LSU.
1979-80 **Clinical Assistant.** The Psychology Group, Baton Rouge, La.

1980-Present **Clinical Psychologist.** Western Missouri Mental Health Center, Kansas City, Mo.

**PUBLICATIONS:**


**PAPERS:**


EXAMINATION AND THESIS REPORT

Candidate: Gregory Blaine Sisk

Major Field: Psychology

Title of Thesis: An Empirical Analysis of Brain Damage and Minimal Brain Dysfunction in School Age Children

Approved:

[Signature]
Major Professor and Chairman

[Signature]
Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

February 27, 1980