73-26,315

FUENTES, Pedro Antonio, 1945-
MINIMAL BRAIN DYSFUNCTION SYNDROME:
A REEXAMINATION OF ITS AFFECTIVE AND
SOCIAL ASPECTS.

The University of Oklahoma, Ph.D., 1973
Psychology, clinical

University Microfilms, A XEROX Company, Ann Arbor, Michigan

THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

MINIMAL BRAIN DYSFUNCTION SYNDROME: A REEXAMINATION OF ITS AFFECTIVE AND SOCIAL ASPECTS

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PHILOSOPHY

BY

PEDRO ANTONIO FUENTES

Norman, Oklahoma

1972

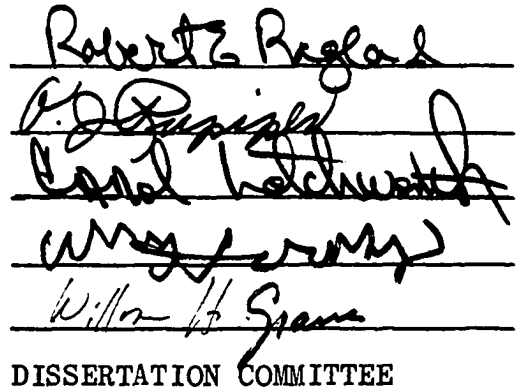
MINIMAL BRAIN DYSFUNCTION SYNDROME:

A REEXAMINATION OF ITS

AFFECTIVE AND SOCIAL

ASPECTS

APPROVED BY



DISSERTATION COMMITTEE

ACKNOWLEDGMENTS

First and foremost, I extend my deepest appreciation to Dr. Robert E. Ragland, chairman of the dissertation committee and director of my clinical internship. Although many times I was disinvolved with the formal requirements of a Ph.D. program, he trusted me and gave me ample room to breath and find my own professional stance. In addition to this, his accurate and brief comments both in this paper and as my consultant during my internship were a source of inspiration and direction for me.

The late Dr. William Hood, whose academic association with me extended back to my undergraduate years, was my professor almost to the end of this study. As a consequence of his social psychology courses, I was confronted with many disturbing questions in relation to my Spanish cultural biases. His down-to-earth, simple, Cherokee-Oklahoman approach gained my trust because it echoed the simplicity of my background, which was close to the land also. In this project he assisted me greatly in defining the limits of the study and made several comments on selection of literature which were very

useful. His death, several weeks before the completion of this paper, saddened me deeply.

Dr. William Horosz is the only member of this committee who has been with me since my undergraduate beginnings through the completion of this dissertation. The first philosophy course which I took with him shattered the Cuban-Catholic cosmology in which I operated at the time. Although I have had some problems in understanding his philosophically abstract manners of speech, I understood enough that his teachings were important forces in my making new integrations, and I trusted him enough to go through the crisis of growth because my strong Spanish-Jesuit education with its emphasis on asking ultimate questions coincided with his approach to learning.

Although the other three members of my committee were of more recent arrival due to the death of Dr. Hood, the departure of faculty members, and the new and unanticipated regulations of the Graduate College, they all made significant contributions to this paper. Dr. William H. Graves impressed me with his tolerance in allowing me the freedom to express ideas which were sometimes basically different from his. He has a deep command of words, a capacity which had not been included in my stereotype of those statistically- and research-oriented. Dr. Graves contributed largely to the verbal aspects of this study as well as with comments on the methodological aspects.

Dr. Carol Letchworth contributed her clarifications of the label "learning disabilities," which expanded my perceptions of the field considerably. She also facilitated my acquaintance with new research in the field. I am very grateful to her for her willingness to extend herself in expediting the process of the completion of this study.

Dr. Omer J. Rupiper, the fifth member of my committee and the most recent arrival, won my deep appreciation for his willingness to make an in-depth reading of this paper under great time pressure. The suggestions he made concerning structural organization of a section of this paper contributed greatly to its clarity and quality.

To my wife, Pat, go much more than the usual thanks for her cooperation and patience. She was, in spirit and energy, co-author with me in this endeavor. Since she can often be more academically disciplined and impartial than I, she "filtered" my expressions to fit the standards of a doctoral dissertation and in the process left her mark throughout this paper. In addition, she also contributed some ideas for original approaches. Thus, she actually functioned in a complementary role with me in this scholarly enterprise, and my gratitude for her assistance knows no bounds.

To the millions of children who today are termed "minimal brain dysfunction," "learning disability," and many other labels, I would like to say that the label does not matter; what counts is that there are people who care about them as people, and thus there is hope.

TABLE OF CONTENTS

MINIMAL BRAIN DYSFUNCTION SYNDROME: A REEXAMINATION OF ITS AFFECTIVE AND SOCIAL ASPECTS

CHAPTER I

INTRODUCTION

Purpose and Scope

The schools contain many children who seem destined to be educational casualties due to behavioral problems and learning disabilities. In our highly industrialized society, school failures are at a great disadvantage in the job market. Bateman (1967), in her review of developments in the field of learning disabilities, suggested that ten percent of the general school population may have learning disabilities which are identifiable. Money (1966) reported that twenty percent of primary-grade children read below their grade level. The several millions of American students who suffer from dyslexia produce massive educational problems. Werner, et al. (1968)

estimated that as many as six percent of grade school students are hyperactive. The concept of brain dysfunction as a possible causal factor of behavioral and learning problems has produced extensive multidisciplinary efforts during the last twenty years (Clements, 1966).

An individual's overall development is dependent upon biological and environmental processes. Therefore, in a child who is without intellectual deficit, deviations in behavior or performance are presented in the literature as determined by environmental or psycho-physiological factors or an interaction of both. Children with behavioral and learning problems are classified within this context. Therefore, the phrase "children with learning problems" generally does not imply any etiology in particular. The possible causal factors may be as varied as lack of experiential bases for learning, neurological dysfunctions, developmental immaturity, or emotional disturbances (Frostig, 1966). For most practical purposes, present remediation techniques follow from specific etiological theories and are generally unidimensional in that they stress treatment of neurological dysfunctions to the exclusion of counseling and psychotherapy (McCollum, 1971).

Usually the terms "minimal brain dysfunction syndrome" and "learning disabilities" are applied to a syndrome of deviations in neurologic, cognitive, behavioral, social, and emotional functions. According to Strother (1963), these characteristics include:

1. General Personality Characteristics
    a. Hyperactivity (restlessness, aimless activity, random movements)
    b. Distractibility (uncontrollably drawn to all new stimuli)
    c. Impulsiveness (spontaneous and compelling inclination to respond)
    d. Emotional instability (sudden mood changes, exaggerated and inappropriate emotional responses)
2. Specific Disabilities
    a. Perceptual disorders (errors in form discrimination, form constancy, spatial orientation)
    b. Motor disorders (lack of coordination, awkwardness, clumsiness)
    c. Language disorders (errors in processing auditory components of speech, inability to associate words with objects, confusion as to time and sequential patterns)
    d. Concept formation and reasoning disorders (inability to grasp concepts, inability to make associations between ideas, faulty judgement of relevance of associations made).

Clements (1966) extended Strother's personality characteristics into a wide spectrum of symptoms. Subsumed under the category of minimal brain dysfunction, Clements

included, among other symptoms: hyper- or hypokinesis, tics, thumb-sucking, nail-biting, head-bangning, negative responses to authority, over-dependency, shyness, inappropriate emotional responses, poor peer group relations, enuresis, encopresis, and poor frustration tolerance. The list presents many opposite characteristics. The author concluded that the extensiveness and variability of symptoms presents a problem to diagnosis which is ameliorated to some extent by the manifestations of clusters of symptoms, such as the "hyperkinetic syndrome." Clements' symptomatology overlaps and is seemingly confounded with manifestations commonly attributed to psychopathological family dynamics (Lowen, 1967; Buxbaum, 1964; Coolidge, et al., 1962) or explained by learning theories (Yates, 1970, Ullmann and Krasner, 1969; Bandura and Walters, 1963).

Because the behavioral characteristics cited by Strothers and Clements are similar to those following established cases of brain damage, such as head trauma (Blau, 1937), encephalitis (Bond, 1932), and severe cerebral palsy (Bortner and Birch, 1962), the syndrome is frequently presumed to be sufficient evidence for the existence of neurological impairments (Conners, 1965; Birch, 1964; MacKeith and Bax, 1963).

The present study will attempt to make an in-depth analysis of literature which covers the affective and social dysfunctions included in the symptomatology of the minimal brain dysfunction syndrome. The following aspects will be considered:

1. Criteria on which the affective and social dysfunctions are said to be a nosologically distinct result of neurological impairment and dysfunctions.
2. Usefulness of medical and psychometric techniques in clarifying the role of neurological impairment and dysfunction in relation to the minimal brain dysfunction syndrome.
3. Alternative theoretical approaches to the current manner in which minimal brain dysfunction symptomatology is predominantly presented in the literature.

Definition of Terminology

Clements (1966) reported thirty-eight terms to be found in the literature to describe children with cognitive, affective, and perceptual-motor deficits. The number of terms is not surprising if one takes into consideration the various disciplines -- such as neurology, psychology, psychiatry, education, optometry, speech pathology -- dealing with the problem area. Two labels currently common in the literature are "minimal brain dysfunction syndrome" and "learning disabilities."

The concept of "learning disabilities," according to Anderson, refers to

> . . . disorders of auditory language, reading, written language and arithmetic, which are related to minimal

> disturbances in neurological functioning, plus the child's conception of himself, developed in a society which perceives that he has a disability (1970b, p. 3).

It thus primarily refers to the educational end product.

The concept of "minimal brain dysfunction syndrome" is related more specifically to suspected organic bases. Clements, in the Task Force I report, stated that

> The term "minimal brain dysfunction 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 functions (1966, pp. 9-10).

The Task Force I study on minimal brain dysfunction was the first phase of a three-part investigation co-sponsored by the National Institute of Neurological Diseases and Blindness and the National Society for Crippled Children and Adults, Inc. The aims of this study were to "Define problem. Suggest nomenclature. Identify child. Delineate relationship of this problem to other handicaps. Outline diagnostic criteria (Clemments, 1966, p. 3)."

The ten outstanding characteristics of minimal brain dysfunction syndrome children, in order of frequency, are: hyperactivity, perceptual-motor impairments, emotional lability, general coordination deficits, disorders of attention (short attention span, distractability, perseveration), impulsivity, disorders of memory and thinking, specific learning disabilities (reading, arithmetic, writing, spelling), disorders of speech and hearing, electroencephalographic

irregularities, and equivocal (or "soft") neurological signs (Millman, 1970). The terms "soft" or "equivocal" neurological signs refer to:

> Equivocal plantar reflexes (uncertain direction of movement of the large toe after stimulation of the sole of the foot), hyperactive deep tendon reflexes, unusually prominent saccadic (nonrhythmically jerky) eye movements noted on following, borderline choreo-athetosis (characterized by involuntary writhing and jerky movements of the outstretched upper extremities), questionable ataxia or tremor on finger-to-nose testing, general awkwardness or clumsiness, prominent mirror movements of the upper extremities (when the child is performing a task with one arm, the other arm "mirrors" the movements), mild dystonias (abnormal, unusually "stiff," postures of the extremities) that become evident when the child walks on his heels or the sides of his feet, or other equivocal findings for age (Cantor, 1971, p. 189).

These are contrasted with "hard" signs, which are "gross motor disturbances and alterations of normal reflex patterns that have classically been correlated with damage to the central nervous system (Birch, 1964, p. 5)."

According to Masland (1968) the usefulness of the term "minimal brain dysfunction" lies in the implication that the problem originates with the child and not from environmental factors. By "usefulness" Masland means that the concept does not place undue weight on the etiologic role of parental care or social environment. Masland and others wish to reassure the parents that the problem is not related to pathological family dynamics.

Background of the Problem

Consideration of the relationship of physiology to the psyche is very old. In a molar manner, the Greeks manifested an awareness of the body as the basis of personality through their theory of the interaction of four bodily humors. Gall's system of phrenology was the theoretical basis upon which Seguin in 1842 built his system of sense training (Murphy, 1949). It was not until 1861 that neuropsychology began to emerge through the work of Broca, who showed that a lesion in the third frontal gyrus of the brain destroyed the capacity for motor speech (Murphy, 1949). Twenty years before Broca's work, however, Lordat had referred to his loss of reading ability following a brain injury (Penfield and Roberts, 1959).

The work of Goldstein during the First World War resulted in a much more complex theory of the localization of psychological functions in the brain. He presented the organism and the environment as a mutually interacting unit. His holistic philosophy was reflected in his view of the aphasias. He rejected Broca's view that the aphasias are direct results of lesions in particular areas of the brain and sustained the view that, language being one of the most important means for self-realization,

> It follows that every individual speech performance is understandable only from the aspect of its relation to the function of the total organism in its endeavor to realize itself as much as possible in the given situation (1948, p. 21).

Goldstein's view is an intermediate position in the relationship between brain lesions and concomitant psychological dysfunctions. This view is shared by Bender (1959), Birch (1964), and Eisenberg (1957), who view the behavioral syndromes associated with brain damage as determined by the individual reactions to those deficits in a particular social context.

The Brain Damage Syndrome. As early as 1947 Strauss and Lehtinen observed that in a population of mildly retarded children who were institutionalized, those who presented a history of "brain damage" or were diagnosed as such by neurological examinations (i.e., exogenously retarded) exhibited different behaviors when compared with non-brain damaged children -- endogenous retardates. The brain-injured children were described as showing erratic, uninhibited, socially unacceptable, uncoordinated, hyperactive, stereotyped behaviors; lack of fear or prudence; catastrophic emotional reactions to frustrations; and sometimes emotional shallowness and bladder and bowel difficulties (Strauss and Lehtinen, 1947).

Prior to Strauss and Lehtinen, Kahn and Cohen (1934) referred to similar behavior characteristics and labeled the syndrome "organic drivenness." Before 1920, studies on minimal brain dysfunction were rare, and they were primarily done with adult subjects (Blumberg, 1967), although the syndrome is described here and there, as in Binet and Simon's classification of "ill-balanced" children:

> The ill-balanced, who might also be called undisciplined . . . are distinguished by their unruliness, their talkativeness, their lack of attention, and sometimes their wickedness (1914, p. 179).

Strauss and Lehtinen postulated that behavioral syndromes are directly caused by general brain damage. They theorized that the destruction of tissue in the forebrain (telencephalon and diencephalon) is the direct cause of the disturbances which are characteristic of brain-injured children. Even though there are neurological sequelae to injuries below the level of the midbrain, Strauss and Lehtinen did not associate these lesions with the same personality or intellectual disorders typical of the brain-injured children. However, their position is not the same as the localization theory of Broca. The former authors' point is that the important aspect is not <u>where</u> the tissue is destroyed but <u>how much</u> tissue in the forebrain is destroyed. In contrast to Broca, they theorized that all brain lesions, wherever localized in the forebrain, produce a similar kind of disordered behavior.

Speech and specific learning deficiencies can be explained -- from the perspective of Strauss and Lehtinen -- by the old localization theory, but deviations from normal behavior which appear regardless of the forebrain area which was injured can not. The regulatory and inhibitory functions of the new brain (forebrain) are the necessary causal links to understand the deviations from normal behavior following brain injury.

> In the course of the development from animal to human being or from suckling newborn to grown child, the new brain develops more and more a softening and inhibiting power. Sudden outbursts of laughter or of crying, extreme aggressiveness in anger, or restlessness in fear become modulated and controlled by volitional forces. Remove or diminish the inhibiting effect of the new brain by a disruption of connections or by an impairment of this balancing power and the old brain acts unchecked; excessive emotional reactions and hyperactivity are the result . . . it is the disintegration of the coordinated activity of the old and new brain (Strauss and Lehtinen, 1947, p. 23).

The clinical signs of hyperactivity, emotional lability, perceptual disorders, impulsivity, distractability, and rigidity and perseveration became known as the Strauss syndrome; the concept of the "brain-damaged" child became synonymous with this syndrome.

The Central Processing Dysfunction Hypothesis. From 1947 to the present, there has been a rapid increase in the number of articles relating brain damage or dysfunction to behavioral manifestations and particularly to learning disabilities in children. Chalfant and Scheffelin (1969) published a review of research of central processing dysfunctions in children covering five thousand articles. Their research of the literature is organized according to a computer model for information processing.

> Auditory, visual, and haptic stimula (or sensory information) are transmitted to the central processing mechanism (brain) where they are analyzed, integrated, and stored. The behavioral response of the subject serves as an additional input source (feedback) for correcting or adjusting further behavioral responses (1969, p. 3).

Chalfant and Scheffelin first covered the research dealing with auditory, visual, and haptic processing problems in the analysis and synthesis of sensory information. The auditory processing mechanisms include the ear as a transducer and the cortex as an auditory processor. Auditory dysfunctions are a product of peripheral and central deafness, mental retardation, severe emotional disturbances, aphasia, or auditory imperception. The authors emphasized the difficulty in identifying the different etiological factors since the causal agents often precipitate similar hearing difficulties. Because of this problem, they recommended a diagnostic team approach which would include, in addition to the audiologist, a neurologist, a psychiatrist, and a psychologist. Even with a multidisciplinary approach, they noted that the lack of valid and reliable diagnostic tools and the absence of a standardized terminology makes fine diagnosis a difficult task.

Dysfunctions of the processing mechanisms for visual stimuli "(a) the ocular musculature as an adjustor; (b) the eye as a transducer; and (c) the cortex as a visual processor (Chalfant and Scheffelin, 1969, p. 21)." A number of cognitive tasks have been postulated which involve the analysis, integration, and synthesis of visual information. Among these tasks are spatial relationships, visual discrimination, and object recognition. Several dysfunctions would interfere with these tasks. Poor spatial orientation has been linked to impairments in the occipital cortex and to

lesions in the infero-parietal and parietal-occipital areas (Luria, 1966a). Children suffering from disorders in spatial orientation have difficulties in discriminating between left and right, in matching and copying geometric figures, and in maintaining a sense of direction; they also exhibit reversal and rotation of letters. Visual agnosia has been associated with lesions in the occipital and parietal regions of the brain. In this type of disorder, an inability to organize visual stimuli into wholes exists; therefore, object recognition is impaired.

"Haptic processing mechanisms" refers to the interrelation of cutaneous and kinesthetic information with body movement. Kinesthetic cue disturbances associated with speech interference make the acquisition of reading and writing skills difficult because children often use articulation to learn both tasks.

> Disturbances in deep muscle and joint sensation -- particularly in the upper limbs, tongue, and lips -- have been found to arise from lesions in the port-central and posterior parietal regions of the cortex of the opposite hemisphere (Chalfant and Scheffelin, 1969, p. 45).

Several cutaneous-kinesthetic remediation techniques in reading, arithmetic, and spelling are currently in use (Frostig, 1965; Kephart, 1962; Montessori, 1912). However, Chalfant and Scheffelin concluded that there is a lack of research showing the effectiveness in compensation and restoration of haptic processing dysfunctions.

The last section of Chalfant's and Scheffelin's report included specific learning disabilities -- primarily

dyslexia and dyscalculia. As in the first section, they described the symptomatic manifestations and usually linked etiology to physiological research concerning clinical cases of established brain lesions in adults. They were explicit in pointing to the present research and theoretical gaps in our knowledge of central nervous system physiology as applied to different dysfunctions. In their report, Chalfant and Scheffelin excluded consideration of the emotional and social problems usually included in the literature of the minimal brain dysfunction syndrome.

The Pragmatic Viewpoint. Many authors are pragmatically concerned with a functional diagnosis and therapy and give minimal consideration to etiology (Frostig, 1972; Kirk, McCarthy, and Kirk, 1968; Bateman, 1967; Heweth, 1964; Bond and Tinker, 1957). Their view is typified by Bateman:

> The very fact that we cannot exchange parents or repair damaged brains has led to the present day concern of many with behavioral and symptomatic . . . rather than pathological or etiological factors (1964, p. 167).

Their interest lies in the cognitive-perceptual-motor alterations as they impinge on the learning process. An example of this position is Frostig's (1964) emphasis on visual-perception problems and their contributions to learning difficulties. The Illinois Test of Psycholinguistic Abilities (Kirk, McCarthy, and Kirk, 1968) and the Frostig Developmental Tests of Visual Perception (Frostig, Lefever, and Whittlesey, 1964) are products of this orientation. These tests are geared toward subsequent remediation programs.

The Pragmatic Hypothesis. There is a classical psychodynamic-oriented view of school behavioral problems which emphasizes learning inhibitions as symptomatic manifestations of psychopathological conflicts. This tradition may have originated with Freud's paper in 1910 on the psychogenic origins of visual disturbances. Kessler (1966) classified the psychodynamic approach into four major neurotic processes. First, he considered the displacement of feelings from oral and anal functions to learning. Children with oral conflicts would have difficulty with input as a result of unconscious confusion between the intake of knowledge and food. The effects of this conflict have been represented as producing pseudo-retardation (Sperry, Stover, and Mann, 1952). Anal conflicts would likely be manifested in the output phase of learning. Kessler did not elaborate on the different techniques of treating the oral and anal conflicts.

The second classification refers to the displacement of feelings from genital functions to learning. Jarvis (1958) reported a case of reading disability in which a child was obsessed with the fear of finding penises in women; therefore, he avoided looking at anything closely and was consequently unable to read. Compulsive public masturbation has been connected with children who experience confusion about gender identity and are unable to participate in a regular classroom setting (Tarachow, 1951).

The third and fourth divisions deal with inhibitions in growing up and in competitive situations which are the

results of a lack of resolution of Oedipal conflicts. Competition might be perceived by the child as anxiety-arousing due to fear of being a rival to the father. Deep conflict between the need to be liked by peers and the school demands of competing against them may block learning in a bright child, producing pseudo-retardation (Weishopt, 1951). Kessler (1966) reported a case in which failure to learn was a result of an unconscious need for punishment. This child reacted with anger and anxiety to compliments.

Buxbaum (1964) in a psychoanalytically-oriented article dealing with the role of the parent in the etiology of learning disabilities, noticed that the psychoanalytic literature on these disorders deals primarily with intrapsychic processes. Koff (1961), in a review of the psychoanalytic literature on learning disabilities, also stressed this point.

> It seemed to me that the greatest agreement and understanding has been in the areas of the content of the fantasies of people having a disorder of learning. The nature of the conflicts, the dynamic tendencies and counter-tendencies, are most clearly described. Starting with Freud's concept of inhibition of function, the id-ego-superego conflicts are elaborated and related to instinctual development on oral, anal, phallic, and genital levels (1961, p. 9).

Buxbaum referred to two types of school problems, all-pervasive learning disorders and specific learning disabilities. The first she traced to a partial symbiotic relationship between the child and the mother which curtails the child from functioning independently of the mother and impairs his awareness of reality. Hellman (1964) has concurred with

this observation. Buxbaum considered partial symbiosis the etiologic basis for speech defects. Also, since school achievement requires some degree of independent initiative, a child functioning in this partial symbiosis would be subject to an all-pervasive learning disability. She reported that analysis offers the opportunity to understand and often to dissolve the pathological symbiosis between child and mother, therefore freeing the child of his learning disturbance.

She contrasted the etiology of generalized learning difficulties with that of specific learning problems (reading, arithmetic, etc.); this second type of problem she related to unresolved Oedipal conflicts. In one case a nine-year old boy was treated who could not read, spell, or compute arithmetic; the analysis revealed that the patient's omission of words when reading protected him from entering threatening fantasies, and making mistakes with numbers was associated with his castration anxiety and an avoidance of success. The boy's avoidance in learning was a way to give up aggressive impulses and submit to what he considered to be a dangerous father; he subsequently overcame his learning disabilities and was able to become openly and appropriately aggressive.

Buxbaum's report was based on ten years of analytic experience in a school for children with learning disorders which is part of an out-patient psychiatric facility. No mention was made of neurological examination of the children treated, nor did her case presentations deal with statistical analysis of treatment outcomes. She mentioned that close

cooperation between teacher and therapist appeared to be a significant factor in the success of treatment; another group of children to which she referred received equal treatment, but without close communication with the teacher, treatment progressed more slowly. Her descriptions of the behavioral manifestations of the children closely matched the characteristics of hyperactivity, distractability, short attention span, emotional lability, and poor school performance associated with the social and affective aspects of minimal brain dysfunction as defined by those who operated from the base of an organic etiology.

> The child whose academic learning difficulties are pervasive is disturbed not only in the area of learning but also in the area of behavior. He is unable to tolerate any pressure, for example, to sit on a chair longer than he wants; his attention span is extremely short; he is unable to tolerate any frustration, but bursts into tears or temper tantrums at the slightest difficulty. He is also unable to let anybody show him or teach him how to do something (1964, p. 425).

Psychoanalytic treatment of learning difficulties is not popular due to the time and expense involved (Coleman, 1972). It is presented here as an example of treatment based on a psychogenic etiological viewpoint which meets with apparent success. Length and/or frequency of treatment seems to be essential for success with this approach (Heinicke, 1969; Heinicke, et al., 1965). Both of these citations dealt with the same study. The subject under research was the frequency of psychotherapeutic sessions as a factor affecting the child's developmental progress. The subjects for this study were eight children, four of whom were seen

once a week and four of whom were seen four times a week. The primary reason for referral was learning disturbances; in each case this difficulty had no apparent link with organic impairment or psychotic process. The children all had at least average intelligence, but they were below average in reading, spelling, and arithmetic. Length of treatment varied from one and one-half years to two and one-half years.

Anna Freud's diagnostic profile (1969) was employed to assess the childrens' progress. Although Heinicke did not discuss this aspect, the diagnostic profile is a means by which a multiple integration of psychological and organic aspects can be presented. In order to make a diagnosis as recommended by this profile, the clinician must take into consideration the following categorizations:

> (1) that, in spite of current manifest behavior disturbances, the personality growth of the child is essentially healthy and falls within the wide range of "variations of normality;"
> (2) that existent pathological formations (symptoms) are of a transitory nature and can be classed as by-products of developmental strain;
> (3) that there is permanent drive regression to previously established fixation points which leads to conflicts of a neurotic type and gives rise to infantile neuroses and character disorders;
> (4) that there is drive regression as above plus simultaneous ego and superego regressions which lead to infantilisms, borderline, delinquent, or psychotic disturbances;
> (5) that there are primary deficiencies of an organic nature or early deprivations which distort development and structuralization and produce retarded, defective, and nontypical personalities;
> (6) that there are destructive processes at work (of organic, toxic, or psychic, known or unknown origin) which have effected, or are on the point of effecting, a disruption of mental growth (1969, p. 147).

The therapeutic orientation in Heinicke's study was that of child psychoanalysis as developed by Anna Freud (1969) who regards the success of the analysis of a child as dependent on the urge of the child to complete development. To assess the statistical significance of the differences between the children seen once a week and those seen four times weekly, an analysis of variance was carried out on the ratings of four factors: ego integration, ego flexibility, peer relationships, and self-reliance. Ego integration included ratings on such items as the child's ability to assert himself, ability to persist in a task, and realistic concern about academic progress; ego flexibility evaluated such dimensions as the capacity to express a variety of affects and nondefensive use of humor; peer relationships were rated on the bases of the degree of development from egocentricity to companionship and level of self esteem as related to body image; the fourth factor, self reliance, was defined by such items as development from wetting and soiling to autonomous bladder and bowel control. The reliability of the ratings was satisfactorily established.

There was a significant difference between the two groups of children within the dimensions of ego integration, ego flexibility, and the capacity to form peer relationships.

One year after treatment was terminated, a significant difference in achievement in reading and spelling still existed between the two treatment groups; although

improvement was noted after the initiation of therapy, there was not statistically significant difference between the groups in relation to arithmetic achievement. The children had not shown any significant intergroup differences at the beginning of treatment on any of the ratings which differentiated them after treatment.

The Neuropsychogenic Model. Anderson (1970a) introduced a theoretical framework designed to explain the interaction of psychogenic and neurological problems in the disabled child. This model resembles Goldstein's (1948) holistic position, inasmuch as the symptoms are seen within a social context. Anderson's approach is rooted in the Adlerian view of individual compensation for organic deficiencies. Out of his attempts to compensate, the child with minimal brain dysfunction could use his condition to manipulate the environment in order to remain neurotically dependent. Anderson viewed treatment for minimal brain dysfunction children as conjoining Adlerian psychotherapy with academic therapy.

Rappaport has pointed out the special incapacities of these children. He summarized the psycho-social repercussions of minimal brain dysfunction to ego development:

> Because the brain -- via the functions of the ego -- is the primary organ of adaptation, when its functions are disrupted, the child's total development is disrupted. The electrochemical and biochemical bases for brain functions do not support normal development. They do not provide the hardware that enables the child to explore, interpret, and achieve a sense of mastery in relation to his environment. In turn, the primary tools needed by the ego to function appropriately do not develop properly. Then the higher ego functions, which develop from the child's interactions with his

> environment, but are rooted in the primary tools of ego function, do not develop properly. Therefore, brain dysfunction itself robs the child of the inherent opportunity to develop effective ego functions in the usual course of growing up. His attempts at mastery result, not in success, but in frustration; not in self-esteem, but in self-derision; not in a sense of "I am one who can," but in a sense of "I am one who can not" (1969, pp. 39-40).

There is a very high incidence of emotional problems in the families of children diagnosed as having minimal brain dysfunction (Morrison and Stewart, 1971, Clements, 1966). Bender (1956) referred to the high degree of anxiety which organically impaired children manifest.

The incidence of minimal brain dysfunction among males is considerably higher than in females (Gruenberg, 1964). Bateman (1965) reported that reading retardation is seven times more common in males than in females. There is no agreement in the explanation of this sex difference. Some have suggested sex-linked susceptibility (Bateman, 1965), but according to Gurenberg (1964) and McCollum (1971), the present evidence points to social-environmental factors as accounting for the incidence of sex difference.

The Neurogenic Hypothesis. The literature of special relevance to the present paper stresses neurological damage or dysfunction underlying the affective and social aspects associated with minimal brain dysfunction syndrome. The linking of neurological impairment with behavioral and learning problems in children probably originated from an analogy with adult brain injury and aphasia and with behavior disorders of post-encephalitic children (Critchley, 1964; Bond,

1932; Sherman and Beverly, 1923). The work of these authors, in addition to the work of Strauss and Lehtinen (1947), was the major factor in the popularization of the label "brain-damaged child." The term is still popular, although in 1962 the Oxford International Study Group on Child Neurology made the decision that the term "damaged" should be discarded (Richardson, 1966). Birch, in summarizing the confusion of classification, said:

> The essential inadequacy of the term "brain damage" . . . derives from the contradiction between its singular form and the plurality of content which it seeks to embrace. As Birch and Demb, Laufer and Denhoff, and Wortis have pointed out, brain damage may vary with respect to etiology, extent, type of lesion, locus, duration of damage, rate at which damage has been sustained, time of life, and developmental stage in which the injury has occurred, and the syndromes of dysfunction that may result. In point of fact, there is not a minimally brain-damaged child but rather many varieties of brain-damaged children (1964, p. 4).

As a substitution, Clements in 1966 coined the term "minimal brain dysfunctions." This new label implies that the syndrome is presumed to be organic, but the emphasis on structural damage has been diminished by shifting the focus to faulty functioning or to an immaturity of the central nervous system. The relationship between structure and function of the developing brain is very complex. Analytical techniques are not yet available which could handle all the processes involved (Scheibel and Scheibel, 1971). However simplistic, the concept of the "brain-damaged child" remains very popular (Silver, 1971). This concept often has unfortunate connotations, as "to many physicians, the label 'organic brain injury' conveys irreversibility and hopelessness (Eisenberg, 1964)."

Pasamanick and associates (Pasamanick and Knobloch, 1960) conducted an extensive study with 500 children who had been referred to the psychological services of the Baltimore Department of Education. Forty percent were diagnosed as exhibiting the brain-damaged behavior syndrome (hyperactivity and confusion). The whole group was compared with a control group of 350 children matched for sex, race, birthplace, and educational level. The referral group presented a statistically significant excess of prematurity and pregnancy complications which led to chronic fetal anoxia such as toxemia and antepartum bleeding.

This study and others preceeding it which examined children with behavioral and neurological problems such as cerebral palsy and epilepsy led Pasamanick and Knobloch to postulate a continuum of disorders caused by perinatal brain damage, the most severe causing fetal or neonatal death, cerebral palsy, epilepsy, or mental deficiency, and then minimal, less severe central nervous system integrative difficulties such as mild cognitive, perceptual, and behavioral irregularities. This latter group they labeled the "minimally brain injured." Thus, the "minimally brain injured" label excluded the categories of mental retardation, cerebral palsy, and epilepsy. The minimally brain injured manifest disturbances which are not always evident but are rather a function of particular environmental variables -- such as socio-familial and educative demands, which for these children represent an overload.

> A dysfunction in the brain lowers the tolerance limits for processing information. A child may show symptoms of disintegration when interneurosensory and complex integrative functions are required; he then manifests poor recall, random movements, poor attention, disinhibition . . . (Johnson and Mylkebust, 1967, p. 31).

An alternative to the brain damage position is the theory that the syndrome is the manifestation of a delayed and irregular developmental pattern. The etiology of the irregular development may include chemical, genetic, metabolic, or psychological factors (Abrams, 1969; Clements, 1966). Lauretta Bender's approach to visual-motor perceptual dysfunctions is based on a "developmental lag" theory. The Bender-Gestalt Test (1938) was developed to show the level of visual-motor maturation in children. It is the age in which a particular distortion occurs which is diagnostically significant; the same distortion at an earlier age represents a normal visual-motor integration.

The developmental lag approach to behavioral, perceptual, and learning disorder is based on the progressive neurological differentiation as the organism matures. The adequacy of myelination of nerve tissue is said to be of primary importance for an adequate differentiation (Wunderlich, 1967). However, Davidson and Peters (1970) stated that the role of myelin in sensory or motor functions is a matter to be settled in future research because in many species the neural connections for these processes are formed before myelination has begun.

Delacato (1959) asserted that failure to develop one-side cerebral dominance and bilateral motor-sensory

functioning is a manifestation of neurological disorganization, thus providing an explanation of learning difficulties. With complete hemispheric dominance, motor as well as perceptual skills are correlates of the underlying neurological development. Immature neurological development results in visual-motor dysfunctions and consequently in reading difficulties. Delacato developed a therapy program based on this theory; however, Robbins (1966) found no support for Delacato's theory and therapy program in the literature.

New knowledge in neurophysiology, neurochemistry, and neuropharmacology has made possible the proposal of new views on the etiology of the minimal brain dysfunction syndrome. One present theory of brain physiology (McCleary and Moore, 1965) postulates that a vertical organization exists which involves up and down pathways between lower centers and the cortex. Vertical organization points out the role played by the subcortical systems in complex behavior (McCleary and Moore, 1965; Doty, 1961) and clarifies the functional relationship between the "old" and "new" brain which Strauss and Lehtinen posited in 1947 to explain the psychological correlates of brain damage.

> The idea that there is a horizontal organization is the older and more traditional point of view. The simplest form of this suggestion was that sensory information from the outside world arrived at the appropriate sensory area in the cortex, and nerve impulses then passed across the cortex (in so-called transcortical paths) to the motor portion of the cortex . . . the lower brain centers played only a supportive role, mediating primitive instinctual responses and simple reflex adjustments (McCleary and Moore, 1965, p. 8).

Two of the subcortical structures which have been postulated as playing a role in the minimal brain dysfunction syndrome are the limbic and reticular activating systems (Silver, 1971a). The ascending reticular activating system (Moruzzy and Magoren, 1949) operates as a modulator-activator and inhibitor in relation to the functions of the neocortex (McCleary and Moore, 1965). To avoid overloading, the ascending reticular system determines the intensity with which sensory signals enter the brain; it also appears to modulate motor output from the neocortex (Silver, 1971a). If these functions are impaired, there will be less discrimination of sensory input, resulting in an increase of non-purposeful motor activity.

Routtenberg (1968) postulated another arousal system which is interdependent with the reticular activating system for regualr functioning in synchronizing neocortical activity. This second arousal system is situated in the limbic system. The limbic system is an interconnected group of brain structures located mostly in the rhinecephalon, the most primitive portion of the cerebral hemispheres (McCleary and Moore, 1965). A dysfunction of this system seems to bring alterations in perception, affect, attention, and memory (Silver, 1971a; Routtenberg, 1968; and Grastyan, 1959).

Based on the functional interconnection between the limbic arousal system and reticular activating system in synchronizing neocortical activity, Silver (1971a)

postulated that hyperkinesis, distractability, and short attention span could be explained by a decreased performance of the ascending reticular activating system. The consequent dysfunction of the second arousal system in the limbic area could bring about perseveration, learning difficulties, inconsistent availability for learning, and emotional lability.

Several drugs are currently used as adjuncts in the treatment of minimal brain dysfunction. Dextroamphetamine and Ritalin are the most commonly employed (Conners, et al., 1967 and 1963). Tofranil is also becoming popular for controlling hyperkinesis (Huessy, 1969). The action of these three drugs seems to result in a relative increase of noradrenalin availability at cell surfaces in the central nervous system (Scharberg, et al., 1967).

Hillarp, Fuxe, and Dahlstrom (1966) mapped out some noradrenergic, dopaminergic, and seratonergic brain tracts by using a technique which produces fluorescence of neurons which contain monamines. These findings suggest that the ascending reticular activation system is predominantly noradrenergic, the limbic system more seratonergic. Therefore, Silver (1971a) postulated that the dysfunctioning of the ascending reticular activation system would be balanced by an increase of noradrenalin brought about by the administration of dextroamphetamine, Ritalin, or Tofranil. Concomitantly with the proper functioning of the ascending reticular system, the limbic system would be stabilized. The proper functioning of the two arousal systems will bring proper

synchronization of the neocortex, resulting in an improvement of the minimal brain dysfunction syndrome.

Silver's theory of the neurological basis of the minimal brain dysfunction syndrome is focused on an imbalance of noradrenalin. However, many antecendent conditions and events contribute to a given complex behavioral outcome. For example, there is evidence indicating that early malnutrition interferes with DNA synthesis and proliferation in the nervous system, and this could be a factor related to some behavioral manifestations included in this group of symptoms (Winick, 1969). In the case of a syndrome typifying children, the shifts of neuroanatomical substrata of behavior during development need to be taken into consideration (Riesen, 1971). Silver took the position that minimal brain dysfunction presents a well-defined syndrome, yet factor analytical studies have not succeeded in isolating a specific minimal brain dysfunction syndrome (Paine, Werry, and Quay, 1968; Dreger, 1964). Silver concluded that there is a need for more research to determine what relationship there might be between the minimal brain dysfunction syndrome and the metabolism of neurons before an interpretation such as his could be justified.

Despite the existence of the previous theories, there is still considerable confusion about the role and meaning of "organicity" as it relates to the syndrome. This is well exemplified by Birch.

> At least in part, the stereotype of brain damage derives from a confusion between the concept of organicity in

> dysfunction and organic brain damage. The first concept, organicity, is not a neurophysiologic but a behavioral one. It involves the idea that certain patternings of behavior and psychological functions exist as the result of damage to the cerebral hemispheres. The fact of "organicity" cannot be denied. Further, it is clear that the behaviors described under the concept almost never occur without the consequent production of "organicity" in behavior (Birch, 1964, p. 7).

Actually, as Mesinger (1965) pointed out, there is no substantial difference between the proponents of a psychogenic and a neurogenic approach in the description of the syndrome in so far as characteristics are concerned. Therefore, it is not so clear that agreement exists for Birch's statement that the behaviors in question "almost never occur in the absence of cerebral damage." Birch's use of the label "organicity" to describe a set of behaviors (psychological level of analysis) without, he says, implying by that label specific neurophysiological correlates, adds confusion to the question of etiology.

Paine (1965) reported that few, if any, abnormalities are revealed by conventional neurological signs when children with minimal brain dysfunctions are evaluated through neurological examinations. However, Silberberg and Leslie (1968) reported that behavioral manifestations of the syndrome are now usually labeled as neurological rather than psychological.

Statement of the Problem

In view of the previous discussion, it would seem to be of theoretical and practical interest to evaluate the available literature which purports to show that manifestations

of the minimal brain dysfunction syndrome originate in neurological impairment or dysfunction, to present current theoretical viewpoints of a psychosomatic orientation, and to attempt an integration of these viewpoints with minimal brain dysfunction.

Research Questions

1. Can the behavioral symptoms -- affective and social aspects -- be taken in themselves as diagnostic indices of neurological dysfunction or impairment?

2. What is the usefulness of the medical and psychometric diagnostic techniques currently in use to diagnose structural alterations or physiological dysfunctions of the central nervous system as they apply to this particular syndrome?

3. Are there alternative theoretical approaches to the current manner in which minimal brain dysfunction symptomatology is predominantly presented in the literature?

The three research questions of this study will be elaborated separately. The division is primarily for the purposes of exposition; its merit is that of convenience for the particular objectives of this study. The first two questions are being investigated for the purpose of preparing the way for discussion of the third question, which contains the most germinal and central contributions of this paper. After each research question has been discussed, some implications for future research will be presented, if pertinent.

All the studies presented will be analyzed according to methodological characteristics such as appropriate sampling, control groups, and employment of reliable and valid criteria of measurement. It is understood in advance that no study in an area such as children's behavior will be methodologically pure. This problem arises in relation to the difficulties involved in manipulating variables involving the children. Ethical principles and practical problems limit the extent to which child-rearing practices, parental personality, and environmental circumstances could be manipulated; therefore, most studies involving children are of an ex post facto nature. However, the awareness of the researcher in relation to the limitations of generalization will be a factor in evaluating the quality of the study.

The literature related to minimal brain dysfunction can be grossly divided in relation to epidemiological and psychosocial studies and a group of studies which attempt to establish diagnostic criteria through contrasting groups. The epidemiological studies deal with the history of pre- and perinatal problems, early infections, and the posterior manifestations of the syndrome. Psychosocial studies examine the family characteristics, socioeconomic variables, school environment, and the relation of these factors to the incidence of minimal brain dysfunction. There is a third group, the purpose of which is to establish diagnostic criteria through contrasting groups. All three of these groups of studies draw their samples by using behavioral criteria.

Limitations of the Study

The use of the label "minimal brain dysfunction syndrome" has become popular since its adoption in 1962 by the Oxford International Study Group of Child Neurology. The review of the literature included in this paper primarily covers the last decade. The literature has been selected in relation to the affective and social manifestations of the syndrome. Therefore, the related literature on similar adult symptoms, clear-cut brain damage, cerebral palsy, epilepsy, mental retardation, and specific aspects of learning disabilities have been eliminated except when particularly relevant to the affective and social manifestations of the syndrome in question. For further coverage of the above-mentioned related topics, see Chalfant and Scheffelin (1969), Tymchuck and Knights (1969), and Birch (1964).

A study such as this one cannot do justice to the complexity of the phenomenon in question. To exhaust the possibilities inherent in a project such as this, an interdisciplinary team and extensive financial support would be needed. The core contribution of this study, therefore, is the presentation of the minimal brain dysfunction symptomatology within the psychosomatic framework and the suggestion of new avenues of treatment which such an approach can offer.

Summary

The purpose of this study will be to analyze the available literature which covers the affective and social dysfunctions included in the minimal brain dysfunction syndrome and to present current theoretical approaches of a psychosomatic orientation which may be relevant to the behaviors in question. Particular emphasis will be placed on clarifying the role of neurological impairment and dysfunction, as it is related to the behaviors in question. The background literature on this syndrome which has been presented to this point is primarily historical and theoretical in approach rather than research-oriented.

At the present time, the predominant etiology of the syndrome is based on neurological impairment or dysfunction (McCollum, 1971; Silberberg and Leslie, 1968; Clements, 1966; Birch, 1964). Alternative theories have also been noted as they relate to a similar cluster of characteristics (Buxbaum, 1968; Mesinger, 1965; Kessler, 1961; Jarvis, 1958). It was established that the syndrome covers children of near average, average, or above average intelligence (Clements, 1966). Children suffering from gross central nervous dysfunction such as epilepsy and cerebral palsy are excluded from this group (Pasamanick and Knobloch, 1960).

CHAPTER II

BEHAVIORAL CRITERIA

Epidemiological Studies

In discussing the use of the term "minimal brain dysfunction" Reitan and Heineman (1968) commented on the fact that the term is reserved for those children in whom the non-behavioral criteria of neurological impairment is absent or at best equivocal.

> Since the term is presently used principally in instances in which the neurological evidence is equivocal, the present usage is almost tantamount to admission that the neurological evidence of brain damage is minimal. If this is true, two questions must be asked. First, what degree of validity would we expect from the identification of brain damage in those instances in which the conventional (neurological) evidence of brain damage is obscure? Obviously, we can expect little validity under these conditions if the judgement is to be based on neurological evidence. Therefore, the diagnosis of "minimal brain damage" must depend upon more evidence than that provided by the neurological findings. The second question, therefore, must inquire into the positive evidence on which the diagnosis is based. Here we find a host of behavioral manifestations cited in the literature which emphasize particularly such terms as hyperactivity, distractability, impulsivity, etc. Many children, however, demonstrate such behavior and except for indications of concomitant "slow learning," additional criteria are rarely cited that would permit differential identification of children with cerebral damage and children with emotional disorders of various types (Reitan and Heineman, 1968, p. 108).

Thus, the task at hand is to examine the evidence on which the proponents of the minimal brain dysfunction hypothesis base their rationale that the behavioral manifestations in question originate in some kind of central nervous system irregularity. A frequently cited source of possible connection between the behavioral characteristics of the minimal brain dysfunction syndrome and organicity are a series of epidemiological studies, primarily those of Pasamanick and Knobloch (1960) and Graham, et al. (1962).

Prior to the studies of Pasamanick and Graham, Heath (1946, 1944), Werner (1945), Bender (1959, 1949, 1942), Laufer and Denhoff (1957) and Windle, Hinman, and Bailey (1958), among others, reported that brain-injured children exhibited hyperactivity, restlessness, distractability, impulsivity, incoordination, and affective explosiveness. Bender (1959) in particular presented a wide and well-rounded clinical documentation of the psychiatric implications of organicity in terms of developmental lags and the severe anxiety manifested in these children. Her case sources were primarily children who had been hospitalized because of behavioral problems following such conditions as head trauma, surgery, and encephalitis.

None of these authors made any explicit generalizations implying that the presence of the particular behaviors presupposed impairment of the central nervous system when neurological criteria were not present. The origin of such a presupposition came from Gesell and Armatruda (1941), who

postulated that minimal brain damage could result from perinatal events. This was extended by the hypothesis of Pasamanick and Knobloch (1960) that there is a continuum of reproductive causality extending through varying degrees of neuropsychiatric disabilities to death, dependent upon the severity of the damage. The hypotheses of Gesell and Pasamanick imply behavioral consequences from minor, undetectable trauma.

The series of epidemiological studies by Pasamanick were mostly retrospective and took the behavior problems -- epilepsy, cerebral palsy, tics, mental retardation, behavioral disorders -- as the independent variables. In 1960 Pasamanick and Knobloch made a summary of more than fifteen years of research involving 4,000 children. The study of particular interest to this paper involved 1,151 children (reduced to 870 by missing data) who exhibited behavioral problems in school. These children were referred to the Division of Special Services of the Baltimore Department of Public Education. In order to control for teachers' biases, the control group was formed by selecting the next child in alphabetical order of the same sex and race. All the subjects were born in Baltimore.

The dependent variables were the hospital records examined on such items as abortions, still-births, previous pregnancies, length of labor, complications of delivery, neonatal course, and birth weight.

> Summarized briefly, it was found that there was approximately three times as much prematurity, as defined by birth weight, and 25 to 50 percent higher rates of complications of pregnancy, dependent upon race, in the behavior disorder cases compared to the controls. It is interesting to note that no difference was found between the cases and controls in the incidence of prolonged and difficult labor and of operative procedures at the time of delivery, such as the use of forceps, cesarian section, or breech extractions, for birth trauma (Pasamanick and Knobloch, 1960, p. 300).

In evaluating Pasamanick's studies, attention should be given to the fact that in correlational studies, both prospective and retrospective, the associations found -- as, for example, between anoxia and behavioral disorders -- could be due to the influence of variables not included in the explanation.

In a prospective study, Pasamanick and Knobloch (1960) used prematurity as the independent variable, which was a dependent variable in the previous study. This study involved 500 prematurely born children and the same number of full-term controls. The children were tested with the Gesell Developmental Examination at forty weeks of age. There was a direct relationship between birth weight (used as an index of prematurity) and serious neurological complications (i.e. "hard" neurological signs).

Drillien (1964) has called attention to the confounding effects of using birth weight as an index of prematurity. For example, low birth weight may be due to intrauterine malnutrition in a full-term baby; it may also be attributed to small genetic heritage or endowment as well as to prematurity. Even taking these factors into account, however, low birth weight is often a more reliable and easily

accessible indicator of prematurity than "guesstimates" of gestational age.

Even though there was a higher incidence of pre- and perinatal problems in the behaviorally disturbed, the same problems were present in a substantial number of the "normal" controls; for example, among the most behaviorally disturbed, forty-two percent showed natal complications as compared with occurrence of such complications in thirty-one percent of the controls. This is in agreement with the position of Birch (1964), Yates (1966), Reitan and Heineman (1968) and Luria (1963) on the nonspecific relationship between behavior disorders and possible neurological impairments.

Supporting the hypothesis of a continuum of reproductive insult, Pasamanick (1960) found that 16.3 percent of the prematures and 10 percent of the controls showed neurological deviations of a type such that compensation would occur with maturation. These deviations included maldirected reaching, increased tendon reflexes, and hypertonicity. Thus, a substantial number of the controls exhibited subtle neurological problems. Pasamanick hypothesized that this group of children would, at a later age and under strong demands (such as school settings), exhibit behavioral disorders without clear neurological signs.

A point of interest in all these studies of Pasamanick is that the behavior problems in question not only included the so-called "brain damage syndrome" behavior

but all sorts of psychopathological manifestations ranging from anti-social behavior to tics. He took the position that early trauma may be associated not only with the syndrome under discussion but also with a great variety of childhood psychopathologies ranging from autism to anti-social behavior. Other studies have also found a higher incidence of pregnancy and birth complications correlated with schizophrenia (Mednick, 1970) and with psychopathology in general (McNeill and Wiegerink, 1971).

One of the findings of Pasamanick was the relationship between low socioeconomic status and incidence of behavioral problems for both whites and blacks. Also, Pond (1961) called attention to the degree of disorganized families among the referrals. Therefore, it is unclear what role social and psychological factors play in the incidence of the disorders because of the high degree of correlation between these factors and perinatal problems.

Besides Pasamanick's studies, Graham, et al. (1963, 1962) conducted a study which, according to Yates' (1966) review of the literature on psychological deficits, was outstanding because of the comprehensiveness of discussion of the many difficult problems which arise in the area of minimal brain dysfunction research; it was also notable for the adequacy of the design, the careful description of the samples, the consideration and control of possible confounding factors, and the careful interpretation of the results.

The study involved 355 children divided into three groups -- full term normal, anoxic full term, and newborns with other complications. The findings showed that anoxic children scored significantly poorer on the cognitive tests than the controls and showed a higher incidence of positive and putative neurological signs. However, it should be noticed that among the anoxic, only 25 percent manifested these problems, as compared with 22.3 percent of the newborns with other complications and 7 percent of the normal controls.

The three groups were tested through a blind neurological and psychological examination at age three. There were very questionable differences in the personality ratings (of satisfactory reliability) between the normal controls and experimental subjects. Surprisingly, among the "positive" cases (exhibiting hard neurological signs), there was no evidence of a specific behavior syndrome (impulsivity, distractability, hyperactivity, or emotional lability).

Graham took care to control for socioeconomic status by matching parental occupations; however, no provision was made for control of family disorganization. The age at which the subjects were measured (three years) may have also been a detrimental factor; the authors suspected that all the children could have been more precisely measured at a later date, and therefore, smaller deficits probably would have been identified. On the other hand, as Pasamanick (1960) has noted, the subjects would have had more opportunity to compensate had they been measured at a later age.

Rothschild (1967), in a comprehensive review of the literature of prematurity over the previous twenty-five years, found a higher incidence of low frustration tolerance, slow motor development, language retardation, and emotional disturbance among prematures than among full-term infants. She commented on the lack of attention to incubator isolation as one of the possible causative factors and hypothesized that the extent and magnitude of these deviations would be positively correlated with the amount of time the infant had been thus isolated. Her point seems relevant in terms of the extensive literature concerning the effects of early isolation on animals and humans (Butler and Rice, 1963; Levine and Otis, 1958; Spitz, 1946).

In relation to incubator isolation, Solkoff, Joffe, and Weintraub (1967) conducted an experiment with ten premature neonates, five experimental and five control subjects. The experimental group was handled in the incubators for five minutes each hour for a ten day period; otherwise both groups received the standardized hospital care. The infants in the experimental group regained their birth weights faster than did the controls and also were more mobile. At eight months, the infants which had been handled were developing well, while only one control was developing at a normal pace. Observation of the home environments of the experimental group suggested that these children lived in a more stimulating environment than did the non-handled group; the authors speculated that changes in the infants' early physical development and behavior

as a consequence of the handling were conducive to the formation of positive maternal atmosphere.

Among the non-handled group, two children were suspected of cerebral palsy; therefore, due to the possible confounding effects of this factor, the results of this study must be considered tentative. However, some indirect support for the hypothesis of Solkoff, Joffe, and Weintraub may be seen in research into the effects of early stimulation with full-term infants (Blauvelt and McKenna, 1961).

Wortis (1964) found that, among prematures, deviant behaviors and neurological abnormalities are found more often among children having disturbed mothers and disorganized family environments. This led Wortis to speculate about an interaction between central nervous system impairment and vulnerability to poor environmental conditions. Robinson and Robinson (1965) found similar results in terms of deviant school behavior; in their study of 135 premature and 92 matched controls, when corrections were made for social class, there was no significant difference in deviant behaviors between prematures and full terms. In another study, Douglas (1956) compared 400 prematurely born with a well-matched control group. Premature children of higher socioeconomic status were less impaired than those of lower status; this again suggests an interaction between social class and the sequela of prematurity. Caputo and Mandell (1970), in referring to the interrelationship between birth weight and variables such as race and socioeconomic status, noticed that these variables

are correlated not only to prematurity but to many of the measures used to determine the consequences of prematurity.

All these studies suggest that prematurity and perinatal complications may indeed be factors in the development of later behavior problems of all sorts; however, the relationship seems to be one of interaction with psychosocial and environmental variables, first because these birth problems are highly correlated with socioeconomic and psychological variables, and second because it appears that the behavioral manifestations are to a certain extent a function of poor familial environments and stressful school situations. Thus, these children may be less resistant to poor environments as a group than children who had full term normal birth. Therefore, in order to assess the sequela of low birth weight (prematurity) and other perinatal problems, many environmental variables which interconnect with whatever central nervous system impairment may be present must be considered. There are, in addition, aspects of psychosocial and other environmental factors which influence the diagnosis of minimal brain dysfunction in older children; this will be discussed in a subsequent section.

In an epidemiological study, there are practical difficulties in applying strict controls for socioeconomic and family disorganization variables. Such controls might lower the validity of research findings due to the small samples which result from their strict application. For example, in the cases of prematurity and certain early infections, to

obtain an adequate sample from the white middle and upper classes is difficult since these problems are rare among this group. On the other hand, data from certain groups of blacks is hard to acquire, because their number in the upper socioeconomic strata in the general population is still small. Therefore, a study which would seek to control for the possible interaction of organic and psychosocial variables would require extensive funds and many years of follow-up. In a long-term longitudinal study, there is also the potential of a "low yield" as a result of frequent occurrence of missing data due to sjubjects' mobility and mortality (Caputo and Mandell, 1970). These practical and scientific considerations may conflict in the implementation of the types of studies with which we have been concerned.

After extensive research in the relationship of central nervous system impairment and subsequent behavior in childhood, Birch made the following comments:

> The consequences of brain damage in childhood have tended to be discussed as though a single syndrome characterized by hyperkinesis, distractability, perseveration, perceptual disturbances, emotional lability, atypical cognitive functioning, and disturbances in impulse control, regularly appears in development as a direct result of brain damage in children . . . . The behavioral sequela of brain damage in childhood can be most diverse, and may range from an apparent behavioral disturbance, absence of behavioral disturbance but presence of mental subnormality, to serious disorganization of social, intellectual, and interpersonal functioning, which are phenomenologically indistinguishable from the major psychoses of childhood (1964, p. 681).

The same opinion was echoed by the extensive research of Reitan and his associates in the Neuropsychology Laboratory at the Indiana University Medical Center and by the work of

Luria in the Soviet Union, as well as by Teuber (1962). All these authors stressed that in order to bring some clarification to the behavioral concomitants of brain damage, the independent variable (some sort of central nervous system lesion) should be operationally defined in relation to the extent of damage, duration of damage, locus of damage, age at time of injury, and nature of the injury (such as surgical intervention, progressive disease, etc.).

Therefore, the authors who have done the most extensive research in the area have acknowledged that a global approach to the selection of the sample -- such as "brain damage" and/or "organic behavior" -- is bound to bring problems in the reliability and validity of the results and, consequently, in the generalizability of the findings. This also applies to minimal brain dysfunction, as no set of behavioral manifestations is a valid sign of central nervous system status. All of the above authors agree that "organic" behavior (hyperkinesis, distractability, atypical cognitive functioning, emotional lability) could also be a manifestation of psychopathology. Yet, the literature is replete with studies which take a global approach to minimal brain dysfunction on the basis of certain behaviors. Further confirmational evidence for the position of these authors is found in the studies of Werry (1968), Paine (1968), Rodin (1963), Eiduson (1966), and Jenkins (1966).

Werry (1968) in an empirical analysis of the minimal brain dysfunction syndrome said:

> The tendency today has been to classify the dysfunctions on an apriori or logical basis. An alternative way would be to do this empirically, using a statistical technique such as factor analysis. . . . In order to be most meaningful, such analysis must be made on the basis of measures from all the potential areas of dysfunction. Also, the children on whom these measures are made must contain a significant proportion who fit the criteria for minimal brain dysfunction as defined by the Task Force (Werry, 1968, pp. 9-10).

The subjects of Werry's study were 103 hyperactive children who were subjected to neurological, electroencephalographic, and psychological tests as well as to inspection of hospital obstetrical records and medical histories. The number of variables was reduced to a total of 67 which were subjected to factor analysis; the factor analysis suggested that these variables might reflect ten basically unrelated dysfunctions, each of which was composed primarily of measures from one source only, such as psychologist, neurologist, etc. It was concluded that these multidisciplinary measures did not tap a single unitary dimension of "minimal brain dysfunction." Werry then suggested that any conclusions drawn about the etiological interrelationships of the various functions would be premature.

The number of variables involved in relation to the number of subjects in the study is a weakness of this particular study; this increases the likelihood of instability of relative loadings and factor placement of these items. Werry was aware of this shortcoming and justified it on the grounds of the exigencies involved in collecting such an amount of data for children in a clinical setting. In addition, no other studies of this type contradict Werry's findings (Paine,

1968). However, the validity of these findings could be further substantiated by conducting a study of this type with a larger sample size.

The results of this study were congruent with other studies, particularly those of Paine, Werry, and Quay (1968), Rodin, Lucas, and Simson (1963), and Schulman, Kaspar and Throne (1965) which will be elaborated upon in a subsequent section of this paper. In a somewhat related study, Eiduson, et al. (1966) examined the dependency of behavioral or learning syndromes established by investigators working in five different clinics on the different data collection practices of each facility; in other words, were the various syndromes in fact describing real differences in children with learning and behavior problems, or might the differences be attributed to confounding variables due to the different data collection practices?

Eiduson and his collaborators obtained fifty case records, ten for each clinic; the records selected were representative of the characteristic data collection procedures for each institution. Each of the records was transcribed into a computer-acceptable form (the Psychiatric Case History Event System). By "event" is meant everything in the case history which was recorded as having occurred to the patient or to his family. In order to determine if the clinics were collecting the same kind of information, the authors searched for those events which appeared in every one of the fifty records studied. Only two events were invariably collected by

all clinics; these were birth date of the patient and information on academic performance in school. When the sample was reduced to forty-nine instead of fifty cases, two additional events were invariant: father's vocation and school behavior of the child. When the cutoff point was further lowered to forty cases, ten additional events were reported in all cases:

> . . . appearance (patient); educational level of father; at least one event in regard to medical history (patient); personality traits (patient); relationship with siblings; religion; psychological problems other than chief complaints; interests; interview behavior (patient); and recommendations in regard to therapy (Eiduson, et al., 1966, p. 833).

The conclusion of the study was that the manner in which data are collected must be taken into consideration in evaluating clinical syndromes attributed to minimal brain dysfunction syndrome. Although mention was made of the statistical analysis of the data, the report of the study was presented in such a way that it was unclear how this analysis was made.

One finding of this study which is of special interest is the high rate of poor home environments found among most of the children; as an example, the divorce rate, even among grandparents, was much higher than in the general population. In eighteen of the fifty families, there were seven alcoholics among parents and grandparents, one addict, nine instances of mental illness serious enough to demand hospitalization, five suicidal attempts, two murder sentences, and two retardates. Care should be taken in generalizing from these findings, however, because the criteria used to select the records were not clearly established, nor was

there any indication of the socioeconomic status of the subjects.

In observing the problem from another angle, Jenkins (1966) found overlap of diagnostic characteristics between the hyperactive group (minimal brain dysfunction) and children classified as "overanxious;" he also observed these symptoms within a group labeled "socially maladjusted." He obtained these results through grouping by computer clustering the symptoms of 500 child guidance clinic referrals.

All these studies have indicated that a homogeneous category "minimal brain dysfunction" has not been established. Werry (1968) did not conclude, however, that these studies have discounted the possibility that the different dysfunctions reflect particular etiologies or neuroanatomical deficits. He suggested a study of the type done by Quay, Morse, and Cutler (1966) with the "conduct problem child." In this study children were grouped according to their estimated factor scores. For example, a "conduct problem child" was one whose estimated factor scores were high in the conduct problem dimension and low on the neurotic and immaturity dimensions (inadequacy factors). Thus, Werry suggested that the minimal brain dysfunction category be broken down into subcategories according to how they emerge from factor analytical studies; then an intensive study should be conducted with each of those subcategories in relation to etiology, response to varying kinds of treatment, long term prognosis, etc. He also suggested further studies to demonstrate the stability

of the basic dysfunction which has emerged from his study and those of Paine (1968) and Rodin (1963) and to determine valid means of measuring those dysfunctions.

Werry and Sprague (1970) and Conners, Eisenberg, and Barcai (1967) have all criticized the indirect manner in which the interpersonal aspects of the syndrome are measured in many studies. Usually a rating scale is used to assess such global and poorly defined aspects as hyperactivity and emotional explosiveness. These ratings are very much subject to the "halo" effect, primarily because the teacher who rates the children has previously referred them on the basis of the same behavior problems rated by the scale; this problem could be avoided by the use of "blind" raters and by checking for inter-rater reliability. It has also been suggested that instead of using parents and teachers to rate the children, other measuring devices could be employed: i.e., direct counts by observers (Barr and McDowell, 1963; Tizard, 1968; Hutt, et al., 1965); mechanical devices (Bell, 1968); and photoelectric (Ellis and Pryor, 1959), ballistographic (Foshee, 1968; Sprague and Toppe, 1966), cinematic (Lee and Hutt, 1964), or telemetric techniques (Davis, Sprague, and Werry, 1969; Herron and Ramsden, 1967) could be used.

Many authors have doubted the relationship between the hyperkinetic syndrome and physiological trauma (Werry, 1968; Schulman, Kaspar, and Throne, 1965; Pond, 1961); they observed that since children are diagnosed according to behavioral criteria, the evidence for a causal relationship between

the behaviors in question and cerebral dysfunction is thus based on indirect post hoc correlations. In particular, Fuisz and Fuisz (1971) and Werry (1968) contended that the present stage of research gives support to a multivariate etiology for hyperactivity.

Somewhat pertinent to Werry's point is the clinical-experimental study of brain damage and behavior conducted by Schulman, Kaspar, and Throne (1965). This study's particular importance lies in the operational definition which was made of the cluster of symptoms related to minimal brain dysfunction and the investigation into whether the four commonly presented dimensions of hyperactivity, distractability, inconsistency, and emotional lability co-vary significantly. In order to measure these behavioral dimensions, the investigators employed measuring techniques of established validity and reliability. For example, they used an actometer (the validity of which was demonstrated empirically) to rate activity levels; oxygen consumption was selected as the physiological measure against which the actometer was validated. Subjects were labeled as "brain damaged" only through hard neurological signs.

It was found that the four behavioral symptoms were not intercorrelated, nor were they related to brain damage in this particular sample. This experimental study supported the contention that no specific set of behaviors can serve as a valid indicator of neurological impairment. The study was limited by the fact that the children were institutionalized

and moderately retarded, but this factor (moderate retardation) would add weight to the hypothesis that the behaviors subsumed under the "minimal brain dysfunction syndrome" are not directly correlated with neurological damage since all the subjects in this sample had been diagnosed as presenting hard neurological signs.

In the studies which follow, it is by no means certain that homogeneous groups of children have been defined within the individual studies or that comparable groups have been assessed across different studies.

Psychosocial Aspects

The literature which links minimal brain dysfunction to different psychosocial variables is very extensive (Silver, 1971b; Grotberg, 1970; Kahn, 1969; Strickler, 1969; Jenkins, 1966; Wortis, et al., 1965 and 1963; Winner, 1962; Pasamanick, 1960; Benton, 1940). We have already referred to some of these studies; we will now review the others.

Alley, et al., (1971) studied the relationship between minimal cerebral dysfunction and socioeconomic status. An attempt was made to clarify the contradiction between the findings of Pasamanick (1961) and White and Charry (1966). The latter found a higher incidence in the upper classes, while the former found a disproportionate number of children classified as minimal brain dysfunction in the lower classes, especially among blacks. Using the hospital records, they found 255 children who, on the basis of Clements' criteria,

were diagnosed as minimal brain dysfunction; they compared this group with 93 subjects classified as normal. The social class level for all the subjects was computed by an index combining the occupation and education of the child's father. Alley concluded no difference existed in the proportion of incidence across social class levels when minimal brain dysfunction children were compared with a normal group.

Despite these results, there is a growing body of evidence showing that the prevalence of the symptomatology of minimal brain dysfunction is higher among minority groups and lower class whites (Grotgerg, 1970). The difference in the findings among White and Charry, Pasamanick, and Alley may possibly be due to sample biases; White and Charry conducted their study in suburban Westchester County in New York, while Alley's study was conducted in rural Iowa.

The contention that many of the measures used to determine the consequences of early central nervous system traumas are contaminated by cultural biases has been referred to previously. The "minimal brain dysfunction syndrome" was defined partially through the association of certain behaviors which have been discussed previously with perceptual-motor dysfunctions. No justice can be done to the topic of perceptual-motor impairments in this paper; for further treatment of this subject, refer to the review of the research by Chalfant and Scheffelin (1959). However, some observations on the relationship of cultural bias to the diagnosis of such impairments are relevant to the goals of this paper.

Deutsch (1964) found that children of low socio-economic status are inferior to their middle-class counterparts in auditory and visual perceptual skills. Relationships similar to those reported by Deutsch have been found between socioeconomic status and psycholinguistic abilities (Stepheson and Gay, 1972). It has also been shown that economically disadvantaged children are more impulsive (Kagan, 1963), have shorter attention spans (Kohlberg, 1968), are more hyperkinetic (Cazden, 1968) and, when not withdrawn, are more explosive (Klaus and Gray, 1968) than matched middle-class children. Studies such as these need to explicitly screen for hard and soft neurological signs in order to clarify in a refined manner the role played by cultural differences and deprivation.

Care must be exercised, however, in diagnosing children who manifest so-called "pure" behavioral and sensory dysfunctions (Ellenbogen and Thompson, 1972). Unless one would wish to generalize from these findings that low socioeconomic status is equivalent to minimal brain dysfunction, it would appear that an interrelationship exists between psychosocial variables and the diagnosis of minimal brain damage; the higher incidence in lower socioeconomic classes of prematurity and other perinatal problems with the concomitant possibility of minimal neurological damage has already been discussed. Another area, that of the relationship between the child diagnosed as minimally brain damaged or neurologically impaired and the norms and restrictions of the socioeconomic level will now be explored.

In a study designed to investigate the poor performance of lower class children on a test of auditory discrimination, Ellenbogen and Thompson (1972) compared a group of lower class kindergarten children with a middle class group using the Wepman Auditory Discrimination Test (Wepman, 1958) in conjunction with a similar form distorted by nonsense syllables. (The Wepman requires the child to determine whether two words are "the same" or "different" on the basis of their sound.) The results suggested that the Wepman may measure a "loaded" middle class vocabulary in addition to auditory discrimination.

This discussion of the performance of minorities in tests of perceptual dysfunction does not contradict the established validity of some of these tests. For example, the Frostig Developmental Test of Visual Perception (Frostig, 1966) and the Illinois Test of Psycholinguistics (Kirk and McCarthy, 1961) do not presume to measure organic dysfunctions; they merely measure specific deficits, regardless of the particular etiology. Psychosocial variables, however, may interact in the administration of such tests to produce marked differences in performance levels among children of differing backgrounds.

Grotberg (1969) published a review of Head Start research from 1965 to 1969 and found evidence that children attending the programs performed below middle class children on all tests, including those which are used to diagnose minimal brain dysfunction and social-emotional behavior. She

also called attention to the aspect of culture-loaded response sets; for example, John and Berney (1967) explored the effect of ethnic background in analyzing story retelling by Negro, Spanish-American, and Indian children in the Head Start Program. There were no significant differences in relation to sex, but differences did occur in relation to ethnic backgrounds. Indian children used fewer phrases; Negro and Spanish-American children took longer to complete the task.

Stodolsky and Lesser (1967) reported that children from the lower socioeconomic levels tend to have ability patterns similar to those of the cultural group to which they belong, but their scores are lower than those of children of the higher socioeconomic levels of their own culture. They also found evidence of differences in cognitive content along ethnic lines; Chinese encourage the development of spatial skills, while Jews stress verbal skills and place little emphasis on spatial skills. One might then expect a higher incidence of visual-spatial deficits among Jewish children than among Chinese children. In another study, Levinson (1959) also found that Jewish children obtained a significantly higher verbal than performance IQ on the WISC; this difference increased with age. Levinson attributed these findings to Jewish cultural values which stress verbal abilities.

Studies of "inner city" black children have shown they are unfamiliar with test situations, are intimidated by an examiner of another race, and lack motivation and interest in the test procedure. For example, Jensen (1969) found that

when black children became completely at ease with the testing surroundings and with the examiner, an average rise of eight to ten points in the IQ scores resulted. Minority group members may require as much as six or seven hours of rapport sessions with the examiner before they fully understand what they are supposed to do and are relaxed enough to work at an optimal level (Kagan, 1969). This requirement is obviously not compatible with the standardized procedures of test administration, but unless such factors are taken into account, the basis for comparison of the scores of minority group and disadvantaged children with established norms is obscured.

Other authors have suggested that the hyperkinetic syndrome is strongly related to motivational factors. In 1969 Freibergs compared a group of hyperactive children; chronic hyperactivity as the major presenting symptom, reported by both parents and the school and agreed upon by two staff psychiatrists, was the criterion for inclusion in this group. Eliminated from the study were subjects diagnosed as mentally defective, epileptic, or suffering from psychopathology, as well as those with clear-cut indications of brain damage. It is regrettable that Friebergs did not specify how it was determined whether a child fell within the psychopathological category. However, the subjects of this study clearly fulfilled the criteria of "minimal brain dysfunction" according to the directives of the Task Force I Report (Clements, 1966).

Members of the control group were children from the same school system who had no history of behavioral problems or emotional disturbance. No statistically significant differences in IQ existed between the children classified as minimal brain dysfunction and the normal sample. Studies of minimal brain dysfunction symptomatology including within the subjects cerebral palsied, epileptic, retarded, and institutionalized children are common in the literature, thus vitiating the generalizability of the results to groups not presenting such characteristics; Friebergs' study was fortunately free of these potential biases. Further discussion of the methodological details of this study would not be of significance except to note that an experimental-factorial study is a rarity in this area of research.

The results of Freibergs' study suggest the differences found between the conceptual ability of minimal brain dysfunction children and normal controls are a result of attentional and motivational variables. These findings were contrasted with Goldstein's classical symptoms of the loss of "conceptual ability" reported in adult individuals known to have suffered cerebral lesions.

Genetic Hypotheses

Coleman (1972) reported that there are no known genetic or chromosomal defects associated with minimal brain dysfunction in children; however, among the possible etiologies of this dysfunction, Clements (1966) mentioned genetic factors.

Since chromosomal abnormalities have been associated with abnormal behavior (Ferguson-Smith, 1966; Fossman and Hambert, 1963; Mozier, Scott, and Deignan, 1960), Warren (1971) made an investigation into the possible chromosomal anomalies in the etiologies of ninety-six minimal brain dysfunction (hyperkinetic) subjects (eighty-two boys and fourteen girls). No evidence of sex chromosomal aneuploidy or of other chromosome abnormality was found; it was concluded that a recognizeable chromosomal abnormality could not be cited as a major factor of the minimal brain dysfunction syndrome. However, the authors warned that the separate question of whether the incidence of sex chromosome aneuploidy in children exhibiting the minimal brain dysfunction syndrome is greater than in the general population can be definitively answered only by a much larger survey.

In an investigation of the interaction of the syndrome with familial characteristics, Silver (1970) studied eighty children referred to schools for minimal brain dysfunction children. He did not explicitly state the criteria for acceptance to these schools; however, the intake evaluation of the children included diagnostic educational testing, psychological testing, social work case studies, a psychiatric diagnostic interview, and speech and language evaluations. For a child to qualify for the full-day academic program, he had to be at least average in intelligence and show evidence in testing of minimal brain dysfunction and/or language disabilities.

The parents of the children were interviewed to determine if (1) the child was adopted; (2) if so, was this done through a private agency, state agency, relative, or other individual; (3) if the child was adopted, were the parents told of anything unusual or different about the child at the time of adoption; and (4) when did the parents first begin to suspect that their child had special problems and that he was different from other children.

Silver found that ten of the eighty children included in the study were adopted children; he compared this with the national rate of adoptions, which was on per every twenty-six live births, and also with the rate of adoption of the state in which the study was conducted (New Jersey); this rate was one adoption for every thirty-seven live births. Thus, in the random sample of eighty children, Silver should have expected to find two adopted children among the eighty; however, the rate of adoption among the children in this group was four times as high as that which would be expected in an equivalent but normal population.

In another study, Kinney, Balwin, and Mackie (1967) studied a similar population and found four times as many adopted children as would be expected, results which are similar to Silver's study; both authors commented that they could not obtain prenatal or natal histories as the adoption agencies refused to release the information, and the parents had no knowledge of it. In one of these studies, minimal brain dysfunction was exhibited in each child of a pair of fraternal

twins; also, two brothers who were not twins both presented the same syndrome. Silver speculated that the relevant variable might be inheritance of a familial pattern of nervous organization.

In a subsequent study related to this same topic, Silver (1971b) investigated the familial patterns of 556 children diagnosed as exhibiting minimal brain dysfunction who attended a special school for educational therapy. On the basis of prior school records, 372 of the children and their families were evaluated; the remaining 184 were extensively evaluated at the time of admission to the school on such items as family history of incidence of syndrome symptoms, previous history of miscarriage, Rh factor, prematurity, neonatal distress, birth order, and educational history of parents.

A stron familial pattern of incidence of the syndrome became evident in thirty to forty percent of the cases in this study. Although some of these children had a history of some type of physiological stress, Silver suggested that these difficulties were not a major contributing factor in the etiology of the syndrome, for siblings without a history of such physiological difficulties also manifested minimal brain dysfunction. He concluded that a dysfunctioning nervous system may have been inherited.

This study contained a series of methodological flaws, as the author has conceded; among these is the fact that no "normal" comparison group was established. The early history of the child and family history of incidence of the

symptoms of the disabilities were obtained through reports of the parents which were based on memory.

Silver's study focused on physiological variables; it would be of interest to conduct a study of this type in which psychosocial variables such as family disorganization, number of siblings, and hospitalization for mental problems were included in a comparison with a control group.

Silver said he was moving in the direction of questioning that the origin of the minimal brain dysfunction syndrome is always related to some kind of earlier assault on the central nervous system. In essence, he was thus questioning Clements' (1966) views on the multiplicity of etiologies for the syndrome.

> I believe that further studies would clarify the frequency of this syndrome as a familial trait and the infrequency in which it is due to central nervous system damage. As research sophistication permits further neurophysiological studies, it is probably a genetically-determined physiologic dysfunction rather than tissue damage which will be found to be the etiological factor for the neurological learning disabilities syndrome (Silver, 1971b, p. 358).

Unfortunately, Silver did not explain how he discounted the possibility of family-interrelated psychosocial factors which might be involved in the frequency with which the syndrome is found among several siblings within a family.

In another study relating familial traits and minimal brain dysfunction, Morrison (1970) associated hyperkinesis with later incidence of alcoholism; however, the question of the interrelationship between family background and the manifestations of minimal brain dysfunction again arises.

Twenty-four percent of the fathers of the children in Morrison's sample had a history of alcoholism; therefore, it is difficult to say whether alcoholism was the result of parental modeling or some physiological tendency in the adolescents in question.

The subject of genetic susceptibility to the personal and social manifestations of minimal brain dysfunction is at this point no more than a very tentative hypothesis. Despite several studies along these lines, we cannot determine to what extent the minimal brain dysfunction has been genetically or psychologically transmitted, inasmuch as the cognitive, affective, and child-rearing abnormalities of the parents could be the manifestations of genetic disorder in the parents rather than the direct cause of the problem in the child.

Heston (1966) and Karlsson (1966) used a series of adoption studies to examine this question in schizophrenia. Their method involved individuals adopted in infancy. Seen in such circumstances, the biological parents and the parents who reared the child -- the transmitters of biological heredity and the inculcators of social experience -- are seen separately, and their relative roles may be assessed. (This technique has been used also by Roe and Mittelman (1945) to evaluate similar problems with alcoholism.) Such a study in the area of minimal brain dysfunction would involve evaluating the personalities of adolescents who were born to parents presenting the symptomatology of minimal brain dysfunction;

however, they would have been raised from infancy in foster or adoptive homes with adults who did not exhibit any special psychopathological or minimal brain dysfunction syndrome problems. Comparison could then be made with a similar control group born to normal biological parents and reared under similar circumstances in foster or adoptive homes. Until studies of this kind are performed, genetic transmission as a factor in minimal brain dysfunction will remain speculation.

Even when the role of genetics becomes more clarified, the psychosocial mileau in which the child develops is inextricably related to the manifestations of the syndrome. In discussing the role of genetics in human development, the behavior geneticist Hirsch says, "High or low inheritability tells us absolutely nothing about how a given individual might have developed under conditions different from those in which he actually did develop (1970, p. 101)."

The group of studies cited which attempted to establish diagnostic criteria through contrasting groups used behavioral manifestations for the selection of subjects; however, they will be evaluated after dealing with the methodological problems associated with diagnosis in this area.

Summary

In summary, it was concluded that prematurity and perinatal complications may indeed be factors in the development of later behavior problems; however, the relationship seemed to be one of interaction with psychosocial and

environmental variables (Rothschild, 1967; Wortis, 1964; Pasamanick and Knobloch, 1960; Douglas, 1956). It was noted that the behaviors in question (hyperkinesis, distractability, atypical cognitive functioning, emotional lability) could either be manifestations of central nervous system irregularity or the result of psychopathology (Reitan and Heineman, 1968; Birch, 1964). It was also concluded that a homogeneous category, "minimal brain dysfunction," has not been established (Werry, 1968; Paine, 1968). Therefore, the selection of samples for minimal brain dysfunction syndrome research is bound to confound the generalizability of the findings.

Several authors have criticized the manner in which the interpersonal aspects of the syndrome are measured in many studies (Conners, Eisenberg, and Barcai, 1970; Werry and Sprague, 1970). The relationship of the symptomatology of minimal brain dysfunction to socioeconomic status, race, and culture was also noticed (Ellenbogen and Thompson, 1972; Birch, 1969; Cazden, 1968; Kagan, 1963; Stodolsky and Lesser, 1967). Friebergs (1969) suggested that the hyperkinetic syndrome is related to motivational variables.

It was also noted that no evidence exists in the literature associating minimal brain dysfunction with genetic or chromosomal defects (Warren, 1971). Silver (1971) observed the high incidence of psychopathology in the families of children labeled as minimal brain dysfunction and has suggested that the syndrome is either genetically or socially related to the etiology of alcoholism, hysteria, and sociopathy.

It was concluded that the behavioral symptoms of minimal brain dysfunction cannot be taken in themselves as diagnostic indices of neurological dysfunction or impairment. The present literature is rather suggestive of a multiple etiology involving in some cases suspected organicity and/or psychosocial factors.

CHAPTER III

DIAGNOSTIC CRITERIA: METHODOLOGICAL PROBLEMS

It has been concluded in a previous section that the dysfunctions attributed to neurological sources must not be based on behavioral observations which can have many different etiologies. Brain damage or dysfunction must be defined by independent observations.

> Psychometric techniques can detect anamolies in behavior only, and should be judged for their accuracy and reliability in serving that purpose. Their validity for determining the properties of the derangement in cerebral function depends on contributions from outside the province of psychology (Talland, 1963, p. 348).

The task of the psychologist is thus to make systematic observations of intact and damaged function in children who have been diagnosed by outside criteria as being impaired.

Reitan and Heineman (1968) commented on the impossibility of doing justice to the multiplicity of factors which are involved in the evaluation of the behavioral consequences of central nervous system damage. Nevertheless, they concurred with Kennedy and Ramirez (1964) in believing that a significant number of children have behavioral problems in which the role of the nervous system must be investigated.

In relation to the syndrome in question, the investigations in identifying these children have been generally unsuccessful as stated in the Phase II monograph on minimal brain dysfunction.

> Search for an assessment pocket specific to children with learning and behavior deviations due to minimal brain dysfunction has generally been unproductive. This circumstance is the result of the protean nature of the disability, as well as the variety of symptoms which manifest diversely in different children and at different ages (Paine, 1969, p. 58).

The approach of Reitan and his associates at the neuropsychology laboratory of the Indiana University Medical Center merits detailed comment. Reitan (1964) has been developing a neuropsychological test battery for children. A primary goal of the laboratory is to subdivide the "brain damage" global approach into categories which relate differentially to psychological measurements such as type, lateralization or location, and duration of brain lesions.

Since certain types of specified brain lesions are relatively rare, the accumulation of enough data requires a long time, Reitan and Heineman (1968) reported that thirteen years of intensive studies have been spent in the development of a battery for use with older children (nine to fourteen years) and eight years in collecting data for a younger group (five to eight years). In addition, a few studies were also conducted comparing the performance of children presenting central nervous system lesions with emotionally disturbed children. The test battery being developed is an adaptation of the Halstead (1947) adult battery.

Reitan's approach combines the strengths of several methods of inference in ways which complement one another to assess brain-behavior correlates. These methods are based on the implied relationship between brain lesions and behavior; they include the differential test approach (as in the Weschler), the lateralized differences in function, and tests of specific pathogenic signs of brain damage. Reitan and Heineman (1968) proposed that, once norms are established for differentiating children with definitely known central nervous system damage from normal children, the next step required would be the study of children with equivocal neurological findings.

Yates (1966) observed that when a battery of tests establishing brain damage has been validated in a selected group demonstrating lesions, variables such as locus of injury, duration, age, etc. have been controlled, suitable control groups have been used and the test has been subsequently cross-validated, the next step is to estimate the battery's predictive validity with an unselected intake group of patients. The question then will be whether the battery can predict which of the intakes will ultimately be diagnosed as brain damaged.

The following approach contrasts sharply with that of Reitan which uses nonbehavioral criteria to establish the presence of central nervous system irregularity. The type of study examined next attempts to establish diagnostic criteria through evaluation of contrasting groups which are selected

entirely on the basis of behavioral manifestations which are presumed to be representative of minimal brain dysfunction. Therefore, the validity of this group of studies in terms of establishing differential criteria for minimal brain dysfunction cannot be higher than the validity of the behaviors selected as representative of central nervous system disorders, and this validity is doubtful. Again, it is unclear whether homogeneous groups of children were defined within the individual studies or if comparable groups have been assessed across different studies. Studies such as these are legion in the literature; however, to avoid repetition only a small group has been selected for examination here.

Deutsch and Schumer pointed out, in relation to the use of "organic" behavior as a criterion for diagnosis of brain damage or dysfunction,

> . . . the circularity involved when, in the absence of evidence for a focal lesion, so-called organic behavior is utilized to suggest the presence of diffuse brain damage in children -- a behavioral rather than a neurological criterion. Such diagnoses, in turn, are sometimes used, we might add, to validate tests of brain damage, the tests "holding up," of course, in the light of the fact that the child's behavior to begin with is "deviant" (1970, p. 22).

Stevens, et al. (1967) studied twenty-six children (twenty-two boys and four girls) assigned to the minimal brain dysfunction category by clinical evaluation; these children were contrasted with twenty-six normal controls of the same age, sex, and socioeconomic status. They used Clements' (1966) criteria to classify the children within the minimal brain dysfunction category. Both this group and

the control group were administered the Weschler Intelligence Scale for Children and an oral reading test; apparently, however, no blind procedures were followed in test administration. All of the subjects were also administered a tone discrimination test, the Token Test (De Renzi and Vignolo, 1962), a gross tapping test, a motor impulsivity test (Boyle, Dykman, and Ackerman, 1965), and other tests of weight discrimination, and fine and gross motor performance.

The "scatter" of the mean subtest scores on the Weschler was greater in the minimal brain dysfunction group; this group also performed more poorly than the control group on the information, arithmetic, digit span, and coding subtests. The minimal brain dysfunction group made more errors of an impulsive type, had longer response latencies, tapped more slowly, made more errors as a function of the complexity of instructions, and had poorer tone discriminations. The authors interpreted these deficits as reflections of subtle deviant functions of the central nervous system.

This type of study does not determine reliable guides by which the presence of minimal brain dysfunction can be discerned for several reasons. In the first place, it is not clear how the children were assigned to the minimal brain dysfunction category. The results might as well be a function of interacting factors other than those presumed; for example, eight of the twenty-six minimal brain dysfunction children had lost their parents before the age of five, and the children in the control group did not present such a situation.

Intelligence quotients were not matched between the samples; as Werry and Sprague (1970) suggested, the results might have been different if these scores had been matched.

In another study, Wikler, Dixon, and Parker (1970) contrasted a group of twenty-five children, aged five to fifteen who had been referred to an outpatient clinic and who presented scholastic and behavioral problems, with a group of controls matched on the bases of sex, age, race, intelligence, and general social class; the control group had no indication from the school records of any behavioral problems. The results of the tests employed (the Weschler verbal and performance scale and the coding subtest, the Bender Gestalt, the Graham-Kendall test, and the Minnesota perceptual diagnostic test) showed a statistically significant difference between the two groups of subjects. In all of the above tests, the minimal brain dysfunction group performed more poorly than did the controls. Neurological examination revealed a greater number of soft signs and electroencephalographic abnormalities within the former group. The significance of these findings, which are fairly common in the literature, is somewhat controversial and merits some elaboration.

Tymchuck, Knights, and Hinton (1970), in a review of the behavioral significance of differing electroencephalic irregularities, mentioned that these abnormalities have been directly correlated with known seizure states, neoplasms, and mechanical injury to the brain. The degree of correlation

depends upon the age of the child, the sensitivity of the electroencephalogram interpretation (i.e., the skill and experience of the interpreter), and the amount of time between the seizure, surgery, or accident and the record of the electroencephalogram. Freeman (1967) found these results particularly unreliable in children, as the patterns are sensitive to the child's reaction to the testing situation.

In studies which examined subjects who evidenced neither neurological nor behavior disorders, false positive identifications varied from ten percent to thirty percent, depending on the study (Tymchuck and Knights, 1970; Gibb, et al., 1960; Kennard, 1960; Taterka and Katz, 1955). Kennard (1960) found that in a group of 123 experimental and 65 control children, eighty-three percent of the experimental subjects with non-organic disorders presented electroencephalographic abnormalities. Subjects with thought disorders and no signs of organicity had forty percent incidence of abnormal electroencephalogram readings; among the normal controls, twenty-three percent had abnormal electroencephalograms. Taterka and Katz (1955) found abnormal electroencephalographic readings in almost eighty-two percent of the subjects out of a group of 195 children hospitalized for emotional disturbances, as compared with positive readings for twenty-three percent of a control group of forty-four outpatients with no history or signs of organicity and/or behavioral disorders; the experimental group in this study included functional disturbances as well as organic suspects.

In essence, although such is not probable, it is possible to be epileptic, have a tumor or a cerebral lesion, and still have a reading with all the indices normal. At the same time, it is entirely possible to obtain an equivocal reading showing some degree of abnormality and still be free from neurological symptoms of any kind. Conversely, a behavior problem may be manifested without corresponding abnormality in the electroencephalogram reading. Only nineteen percent (Hughes and Park, 1968) to fifty percent (Hughes, 1968) of the children classified under the minimal brain dysfunction syndrome present such anamolies.

Laufer and Denhoff (1957), Silver (1971a), and others have postulated that the dysfunction evidenced in minimal cerebral dysfunction syndrome originates in the subcortical structures of the brain; because an electroencephalogram is basically a measure of the electrical activity of different portions of the cortex, it is not sensitive to this region.

> Electroencephalography has a place in the diagnostic evaluation of patients suspected of having minimal brain dysfunction. The EEG is, in itself, not the diagnostic tool. When properly recorded and interpreted in conjunction with the clinical history and the results of the neurological examination, the EEG can help to determine whether or not the patient has a seizure disorder, a seizure equivalent state, or organic brain disease needing more detailed investigation. It cannot, however, make the diagnosis of minimal brain dysfunction (Paine, 1969, p. 57).

In addition, no standardization of neurological examination procedures exists for the problems related to minor nervous dysfunctions. Werry (1968) concluded there has been no good neuropathological study of the brain functions

of children featuring the minimal brain dysfunction syndrome. Much significance has been attributed to soft or equivocal neurological signs. However, these are not definite indications of abnormal development; these soft signs

> . . .vary in amount with age, maturity, intelligence, and cooperation of the child. The decision that they are abnormal in any child is a subjective decision . . . . The presence of soft neurological signs may be of importance in differentiating the child with learning problems from his normal classmates, but this is unlikely. Controlled studies are needed to determine the significance, if any, of these soft neurological signs (Cantor, 1971, p. 189).

Another difficulty in accurate neurological evaluation of children lies in the fact that the child's performance constantly changes with age; age-specific standards have been applied, "but these standards are neither precise nor extensively documented. . . . (Paine, 1969, p. 57)." When variable standards, based on the child's age and examiner's experience, are applied, the subjectivity of the neurologist's evaluation becomes an important factor.

Ozer (1968) attempted to fill this gap by developing an easily administered neurological examination which is specifically oriented to measuring brain function in terms of rate of learning; this examination is based on an operant conditioning paradigm and requires only fifteen minutes to administer. He developed some measures of brain function which discriminate children with minor motor sequencing immaturity, but he has not yet established predictive validity. One set of items is similar to the face-hand test developed by Bender and associates (1951); this test requires

identification of simultaneous tactile stimuli. Other items for the ability to "tune out" irrelevant stimuli; the child must learn to focus on relevant sensory input to the exclusion of the extraneous. Ozer is interested in the rate of learning for these different tasks. The published material is not sufficiently clear to evaluate the psychometric soundness of Ozer's procedures.

A somewhat similar test is being developed by Denhoff, Hainsworth, and Siqueland (1968a). These tests Ozer and Denhoff, et al.) are geared to predict school performance, not organic pathology; thus, they are not to be considered substitutes for detailed pediatric neurological examination. However, both tests offer the potential to screen out those children who should have a more thorough neurological-pediatric examination (Denhoff, et al., 1968a). Validity has been established for Denhoff's examination. Of 355 normal children, ninety-five percent passed the test (Fuisz and Fuisz, 1971), while twenty-five children diagnosed as neurologically involved showed significantly lower scores than matched normal controls (Denhoff, Hainsworth, and Siqueland, 1968a). Further validation studies are in progress.

Towen and Prechtle (1970) have recent published a neurologic examination for the child with minor nervous dysfunctions; however, they are still in the process of establishing adequate population norms.

Of potential importance in clarifying the role of the central nervous system in the causation of the affective

and social manifestations of the minimal brain dysfunction is the work of Laufer, Denhoff, and Solomons (1957). They, with Denhoff and Chammas (1968b) have theorized that the hyperkinetic syndrome is produced as a consequence of damage to the diencephalon as the result of some kind of trauma; they support this hypothesis through the use of the Photo-Metrazol Activation Technique. This test provides a method to explore certain subcortical structures such as the diencephalon; normal adults require a certain amount of Metrazol per kilogram of body weight to evoke specific response clinically (myoclonic jerks) and in the electroencephalogram. For an elaboration of the details of the techniques involved in administering this neurophysiological test, see Gastaut (1950).

Laufer, Denhoff, and Solomons (1957) found that in a group of hyperactive children, the threshold for evocation of electroencephalogram "spike-wave" discharge (the combination of a very sharp wave of less than eighty milliseconds duration with a slow wave) and myoclonic jerks in the forearms was abnormally low in hyperactive children and was normal with non-hyperactive, emotionally disturbed controls who exhibited behavioral disorders of environmental origin. It was also noticed that the use of amphetamines raised the threshold in the hyperactive group to the level of the control group. The twenty-five hyperactive subjects were selected on the basis of whether they presented the total picture of behavior typical of the hyperkinetic impulse disorder.

The authors did not specify how the hyperkinetic syndrome may have resulted from earlier physiological trauma, particularly in the cases where no known physiological trauma had existed. Since only eleven of the twenty-five children had such a history of organicity, the etiology of the syndrome could as well have been attributed to social or interactional factors. They did state that

> The photo-Metrazol studies themselves do not give the explanation of hyperkinesis. It must be recognized that interpretations are largely speculative, as neurophysiological research concerning diencephalon and cortical relations in the human is still in such an amorphous state (Laufer, Denhoff, and Solomons, 1957, p. 45).

Furthermore, Pond (1963) concluded that the photo-Metrazol activation technique had been used in criterion groups of mixed organic, epileptic, and behavior problem children. Therefore, it cannot definitely be concluded that the technique is of differential diagnostic value.

A study which might further define the limits of the use of the photo-Metrazol technique would be one carried out to determine if a difference could be found among three groups: two groups of children exhibiting the "hyperkinetic syndrome" and a normal control group. One of the hyperkinetic groups would be chosen on the basis of clear-cut organicity (i.e., traumatic injury, tumor, congenital vascular anomaly, or other damage evidenced through hard neurological signs); the other hyperkinetic group would be selected solely on the basis of behavioral criteria and would have no suspected history of organicity (i.e., no history of trauma and no

discernible neurological anamolies).

Steward, Pitts, and Craig (1966) in an effort to discern the difference between hyperkinetic and non-hyperkinetic children followed the usual approach in selecting subjects. A group of thirty-seven first grade children referred to the guidance clinic on the basis of hyperactivity and a normal control group of similar socioeconomic level were selected.

The mothers of the children were interviewed using standardized techniques which covered such items as family, school, and developmental history; prenatal and perinatal complication; and enuresis. The differences in frequency of incidence of such factors was then compared through a chi-square test. No evidence was found for specific organic etiological factors. The hyperactive group was found to be more immature in terms of learning, coordination, speech, and feeding and sleeping habits, but these results were scarcely surprising, inasmuch as these children had been out-patients at the clinic and were referred by the school system which used corresponding criteria to support its referral action.

Kerlinger (1964) cautioned against ex post facto research such as that conducted by Stewart. Conclusions may be misleading when a study is performed without a stated hypothesis or predictions; whatever the observations, new interpretations can be construed which will "fit the facts (p. 372)." Nevertheless, studies of this kind can generate

new hypotheses which could then be tested in a more controlled fashion.

Summing up, when samples of children are selected on the basis of social and affective behavioral manifestations of the minimal brain dysfunction (hyperactivity, distractability, emotional lability, etc.), the minimal brain dysfunction sample generally shows a higher frequency of soft neurological signs, borderline electroencephalogram tracings, and poorer perceptual-motor performance when compared with a matched normal control group. The significance of these findings in terms of the integrity of the central nervous system is at best controversial. Equivocal indications of impairment are frequently cited as definitive indicators. In addition, lack of adequate control group data analysis often obscures the significance of manifestations associated with abnormal behavior; the occurrence of similar signs must be compared with non-disordered control groups. Actual distributions of results of "cerebral dysfunction children" and controls overlap considerably; when intergroup mean differences reach the .01 level of probability, "many of the 'control' subjects almost invariably perform more poorly than some of the brain damaged subjects (Reitan and Heineman, 1968, p. 98)."

Although presumably the social and affective aspects of the minimal brain dysfunction syndrome are differentiated from neurosis and character disorder by the degree to which soft neurological signs, electroencephalographic

tracings (Fuisz and Fuisz, 1971), and poorer perceptual and/or motor performance are present or absent, the frequency with which these findings differentiate between these three groups has not yet been established. Research is needed which would compare these different groups using double-blind cross-over designs on aspects such as presence of soft neurological signs and borderline electroencephalographic tracings.

While there are some tests which are probably more influenced by certain kinds of brain damage than are others, current tests which purport to detect organicity can at present be most meaningfully employed in providing a description of the specific behavioral deficit characterizing each individual. These findings can subsequently be incorporated into a therapeutic and educational program (Anastasi, 1968).

A common characteristic of all minimal brain dysfunction cases appears to be the relationship to school problems (behavioral or academic); thus, it would seem that a label such as "learning disability" is more appropriate inasmuch as it does not suggest a particular etiology.

Summary

In this chapter, the relationship of possible central nervous system dysfunction to minimal brain dysfunction has been discussed. It was noted that central nervous system dysfunctions which are attributed to neurological irregularities cannot be used as diagnostic validation of behavioral criteria since behavioral manifestations can have diverse etiologies

(Talland, 1963). The conclusion was also drawn that a global approach to minimal brain dysfunction is inadequate because of the magnitude of symptoms subsumed under such a label. Furthermore, the task of carrying out research on the behavioral correlates of brain damage proper (as based on clear-cut organicity) is in itself a titanic endeavor (Reitan and Heineman, 1968); even more difficult would be the investigation of minimal brain dysfunction, due to the absence of evidence of neurological impairment (Deutsch and Schumer, 1970).

The research reviewed in this chapter attempted to establish diagnostic criteria through the selection of minimal brain dysfunction syndrome children on the basis of behavioral criteria; therefore, the validity of all these studies is doubtful. Furthermore, presumably the differentiation of minimal brain dysfunction from psychoneurotic and character disorder children was based on the degree to which soft neurological signs, borderline electroencephalographic tracings, and poorer percpetual/motor performance were present or absent; research clearly establishing these differences seemed to be absent in the literature. It was concluded that until this type of research is performed with validated instruments differentiating between minimal brain dysfunction, neurotic, and character disorder children, current diagnostic techniques are inadequate.

CHAPTER IV

PSYCHOSOMATIC ORIENTATION

To the knowledge of this author, the symptomatology of the social and affective interpersonal sensory and motoric manifestations of the minimal brain dysfunction syndrome have not been explored within the framework of the psychosomatic viewpoint. Referring to the psychosomatic hypothesis in a different context, Ellingson (1954) explained it as follows:

> The psychosomatic hypothesis holds that as in other psychosomatic disorders, psychic influences (presumably cortical) give rise to autonomic and/or endocrine disturbances. These in turn affect cerebral functioning either directly or by altering the biochemical milieu of the brain. One result is abnormal brain waves (p. 271).

The theories of psychopathology developed by Reich (1945) along these lines were a further development of Freud's psychoanalytic theory. Reich's orientation and the related psychotherapeutic approaches of Lowen (1957) and Perls (1969) emphasized such bodily manifestations of neurosis as lack of perceptual-motor coordination, sensory insensitivity, short attention span, hyperactivity, poor muscle tone and balance,

etc. These symptoms appear to overlap with the symptomatology of minimal brain dysfunction.

Reich (1949) visualized the body as disclosing contours which betrayed the individual's dramatization of his emotions. Central to these theories is the concept of the muscular armor. This concept implies that the individual's emotional conflicts are directly correlated with the muscular system. Reich developed the means to analyze which muscular systems, through their deviant activity, made possible the observable expression of the individual's predominant emotional "set."

Reich, Lowen, Perls, and their followers considered repression to be manifested as a perceptual-motor phenomenon. In a very brief manner, their theory of repression is as follows. When needs arise in the child, sensory capacities are used as a means of orientation. Once the means of satisfying needs becomes figure in the environment (i.e., hunger satisfaction, mother's touch) the child mobilizes his motor processes, which are his means of manipulation. In the course of normal development, there is a steady progression from seeking environmental support to self support. If the environment is such that the child is chronically thwarted in satisfying his needs, he inhibits his genuine impulses by contracting the antagonistic muscles which prevent expression of his impulses. For example, in a child who is not permitted to cry, the holding back process involves contractions in the jaws and inhibitions in breathing. This eventually leads

to chronic muscular contractions which become subconscious.

> We can say that every muscular rigidity contains the history and meaning of its origin. . . . The armor, itself, is the form in which infantile experience continues to exist as a harmful agent (Reich, 1942, pp. 266-267).

Body cues (sensing and feeling) no longer are a good guide in the child's contact with the environment. The consequence is poor perceptual-motor capacities and poor contact with the environment.

Some confirmation for Reich's theory may be found in Malmo's (1970, 1957) reports on his extensive work with the electromyogram. The source of the electromyogram recording is a muscle contraction; through this apparatus, a recording is made which represents the number of muscle fibers contracting simultaneously. Through his work, Malmo has shown that subjects suffering from pathological anxiety (psychoneurotics) are slower than contrasted groups in returning to a prestimulation level of muscle contraction. The same findings have been reported by Goldstein (1965). In defining "pathological anxiety," Malmo (1957) noted that such persons were characterized by one or more of the following complaints in a persistent manner: "tension" or "strain," "restlessness," etc.

Malmo (1957) and Christiansen (1963) also found that shallow respiration is a common characteristic among schizophrenics. This is in agreement with the theory of Reich and his followers that another manifestation of psychopathology is shallow respiration due to muscular rigidities in the thoraxic region.

To this author's knowledge, no application of Reichian theory has been made to minimal brain dysfunction. There is no question, however, that some of the cases successfully treated through Gestalt therapy could have been diagnosed as minimal brain dysfunction. As an example, Rosanes-Berrett (1970) treated a six year-old hyperactive girl with reading difficulties who could not sit still for more than three minutes and was unable to concentrate on reading matter.

> Using the Gestalt approach to her experiencing aspects of a dream which she related, the therapist persuaded her to get in touch with so-called holes or alienations in her personality. After some initial anxiety, the child was able to recognize her need to keep watch against witches and ogres -- she felt like one herself. She could not allow herself to concentrate on a close point, as she might miss the danger she constantly expected. In experiencing the conflict of staying at the close point and being on guard against what might come from the distance, she realized that she could not read this way and that one eye seemed to be out in the distance while the other was at the close point. She was asked to look into the distance and become aware of being on guard and of the conflict. By constantly staying with this child, and allowing her to look into the distance and assure herself that nothing was there to hurt her, the therapist enabled her to come back and read. The hyperopia no longer appeared (Rosanes-Berrett, 1970, p. 261).

The following case involving the Gestalt therapy treatment of a female college student with extensive inability to spell, organize grammatically, and punctuate (despite a high degree of verbal understanding) was presented by Fagan (1970); the girl had previously sought extensive remedial aid and had undergone a year of psychotherapy with no resulting improvement in her condition.

The procedure employed by the therapist was based on Perls, Hefferline, and Goodman (1951); this approach

purports to increase the awareness of sensory, visceral, percpetual, affective, and motoric functions. The specific therapeutic tasks, which included such items as writing free association responses to certain words (i.e., "anger" and "play," listening to recorded tapes of orchestral selections while finger-painting, enumerating ten reasons why people should not obey rules (misspelling every word deliberately), reading poetry selections aloud, and studying a flower intensively, then finger-painting it, were selected on dynamic grounds. The therapist interpreted that the patient's failure to follow spelling, grammatical, and punctuation rules was related to her inability to express anger and rebellion openly. Fagan reasoned that if the girl could be enabled to overcome this as well as other global expressive difficulties, the more specific spelling and grammatical problems would resolve themselves.

Use of the left hand was specifically prescribed for some of the exercises. The rationale for this was based on Perls' theory of left and right splits. When muscular action is chronically held back, posture is deformed in different ways, depending upon the muscles involved. For example, if due to some kind of psychological conflict the pelvis is held rigid, Perls maintained there could then be no flexible base for movement of the upper torso, arms, and head (Perls, 1951); (i.e., in order to have proper posture, the head cannot be constrained by taut neck muscles; the chest cannot be pushed out or pulled-in back). Conflicts between head and trunk are expresed in a struggle between right and left hands

and legs, bringing lack of motor coordination and poor perceptual contact.

> When the head, for instance, is moral and "right," the person is stiff-necked -- afraid of losing his precarious balance. In this case the neck serves, not as a bridge between head and trunk, but as a barrier -- literally, a muscular bottle-neck -- between the "higher" and the "lower" functions of the personality. The shoulders, afraid to expand and work or fight are held contracted. The lower body is well "under control." Ambidextrous cooperation is lacking. One hand tends to hold down and nullify the activity of the other, and the same is true for the legs (Perls, Hefferline, and Goodman, 1951, p. 180).

Thus, a relationship would seem to exist between the phenomena discussed by Perls and the poor spatial orientation and distorted concept of body image, motor awareness, and impaired discrimination between left and right and up-down which is exhibited by children labeled minimal brain dysfunction.

The primary technique of Gestalt therapy is to focus on the on-going, present situation (the "here and now"). However, the past and the future are not considered to be inconsequential; rather, they are made relevant to the present. The therapist also attempts to help the patient regain awareness and trust in the sensory-motor systems. Laura Perls (1956) defined neurosis as a state of malcoordination of contact and support functions of the individual. Contact is made through sensory-motor activities; it "takes place against a background of organismic functions that are normally unaware and taken for granted; yet these latter provide the indispensable support for the foreground function of contact (Perls, 1956, p. 43)." Manifestations of minimal brain

dysfunction such as poor visual-motor coordination, impaired judgment of distance, distorted concept of body image, and inability to concentrate are considered in the Gestalt approach to have originated in consistent thwarting of the individual's organismic needs.

Speech problems such as mutism, stuttering, lisping, baby-talk, or abnormal voice conditions may have roots in emotional conflicts (Van Riper, 1964). Frederick Perls (1969) mentioned the treatment of a stutterer who, when he recovered the ability to express anger, ceased to stutter, and Moses (1954) presented an account of the repercussions in speech problems of different neurotic problems. He also described the subtle interplay between organic and functional speech problems.

> It is extremely difficult to draw a line between the functional and the organic. Neurotic misuse of the voice, even emotions concurrent with imaginary situations, may irritate the vocal apparatus to the point of producing organic symptoms. The opposite is also true. Organic lesions may induce psychological processes that leave scars and stimulate dysfunctions. It seems as if even the term "psychosomatic" contains too much of the old dichotomy. Psychosomatic processes do not take place in one direction only. . . . They are complex mazes (p. 5).

However, in the minimal brain dysfunction orientation, slow language development and mild speech irregularity are usually presented as prima facie evidence of some sort of central nervous system dysfunction.

The application of Gestalt therapy to the treatment of the symptomatology commonly ascribed to minimal brain dysfunction is a potentially fertile task which awaits

further investigation. The technique has been used with reported success among children having behavioral problems in school (Lederman, 1969). It has also been applied in a classroom context with children not presenting special problems without sacrificing basic content of the conventional curriculum (Brown, 1971). None of these applications has as yet been subjected to rigorous research, although successful outcomes have been reported by both Lederman and Brown.

Because of the emphasis on integration of sensory-motor perceptual functioning to affective material, it appears that Gestalt therapy is in a unique position to integrate psychotherapy with present remediation techniques for minimal brain dysfunction which stress only the sensory-motor perceptual aspects. A fruitful line of research would be the comparison of three groups of children labeled as minimal brain dysfunction; one group would receive traditional treatment, one would receive traditional treatment in conjunction with Gestalt therapy, and another would receive Gestalt therapy alone.

Another development which closely parallels the position of Reich and Perls is the technique of Structural Integration (or Rolfing) developed by Ida Rolf. Rolf maintains that certain emotional patterns as well as physical trauma can change body structure.

> An individual experiencing temporary fear, grief, or anger, all to often carries his body in an attitude which the world recognizes as the outward manifestation of that particular emotion. If he persists in this dramatization or consistently re-establishes it, thus forming what is ordinarily referred to as a "habit

> pattern," the muscular arrangement becomes set. Materially speaking, some muscles shorten and thicken, others are invaded by connective tissue, still others become immobilized by consolidation of the tissue involved. Once this has happened the physical attitude is invariable; it is involuntary; it can no longer be changed basically by taking thought or even by mental suggestion. Such setting of a physical response also establishes an emotional pattern. Since it is not possible to establish a free flow through the physical flesh, the subjective emotional tone becomes progressively more limited and tends to remain in a closely defined area (1963, p. 243).

She also believes the converse of this position to be true.

> In any attempt to create an integrated individual, an obvious starting place is his physical body, if for no other reason than to examine the old premise that a man can project only that which is within. To the medical specialist, this body, and this alone, <u>is</u> the man. To the psychiatrist, this body is less than the man; it is merely the externalized expression of personality. Neither of these specialists has accepted as real a third possibility; namely, that in some ways, as yet poorly defined, <u>the physical body is actually the personality</u>, rather than its expression is the energy unit we call man, as it exists in its material, three-dimensioned reality (1971, p. 250).

Although Perls' theory adheres to a holistic and monistic view of man (Perls, 1969), Rolf appears to be more radically monistic. This is perhaps due not to a different philosophical position but to different levels of therapeutic endeavor.

The Structural Integration technique requires at least ten sessions, each approximately one hour long. The muscular system, as a consequence of physical manipulation, is purported to be realigned in such a way that movement becomes more efficient and coordinated. The claim is that a permanent change in the muscular armor is effected which brings about physiological and psychological changes, thus reflecting Rolf's monistic point of view that a change in

the structure of the body must bring a change of function at all levels. This technique seems to be an indirect validation of Reich's muscular armor concept.

The Rolfing technique does not attempt to substitute for psychotherapy, and the patient must be ready to accept in himself the changes brought about through the physical manipulation. It appears then, that the individual can resist some changes in his muscular armor through an encoding of physiological/psychological history in the central nervous system (the "vestigal limb" experienced by amputees would be a physiological example of such). The theory is not complete, but Ida Rolf and her followers admit to these gaps and are working to present a more fully integrated theory.

> The role of the autonomic versus the central nervous system in establishing and maintaining conditions which make possible the very significant intrinsic-extrinsic myofascial balance will require further specialized study. Together, these studies will point the way toward a different understanding of the mechanism that is a human being (Rolf, 1972, p. 14).

Although the technique has been used with children under a variety of physical and emotional states, ranging from cerebral palsy to autism, no organized research has been conducted in this area. To this author's knowledge, the Rolfing approach has never been used with groups of children labeled as having minimal brain dysfunction. It would appear, however, that this technique might be of potential value in the remediation of this syndrome. Besides effecting changes at all levels, this approach is especially oriented toward alterations of posture and movement which could have a direct

impact upon such symptoms of minimal brain dysfunction as motor awkwardness, poor spatial orientation, confused perception of laterality, poor perceptual integration, etc.

The Structural Integration approach is gaining scientific support through research currently being conducted by Hunt (1972) and Silverman, et al., (1972). The published reports of this research are as yet fragmentary, which makes their evaluation difficult. Extensive use is being made in these studies of the electromyograph technique (Hunt, 1972), electroencephalogram, and other biochemical tests (Silverman, et al., 1972). Silverman's findings to date have been summarized as follows:

> Psychological and physiological effects of the Structural Integration (S.I.) technique of body organization were studied in a group of thirteen normal male subjects. Several EEG averaged evoked response procedures, an eye-movement procedure and a battery of biochemical tests were administered before, during, and after a series of ten S.I. treatments. A pattern of significant changes was found. This pattern was indicative of an increased openness and well modulated sensitivity to environmental stimulation; it was most apparent in individuals who evidenced the greatest degree of balanced muscle organization (1972, p. 15).

Further research could be carried out with minimal brain dysfunction children along the lines of that proposed for Gestalt therapy. As measuring tools, tests of perceptual-motor coordination could be used.

Biofeedback is another therapeutic technique which could possibly be applied to the treatment of the symptomatology subsumed under the minimal brain dysfunction category.

Although this author has no personal knowledge of such application,

> The unique value of feedback instrumentation is that it gives the subject an immediate indication of his progress. Through external feedback, the subject is enabled to filter out from the welter of internal existential cues, those particular ones which he must learn to manipulate (Green, 1970, p. 18).

Thus, the technique would seem to be of potential applicability to the treatment of hyperkinesis and short attention span. This approach is also rooted within the psychosomatic orientation.

> The psychophysiological principle, as we hypothesize it, affirms that "Every change in the physiological state is accompanied by an appropriate change in the mental-emotional state, conscious or unconscious, and conversely, every change in the mental-emotional state, conscious or unconscious, is accompanied by an appropriate change in the physiological state." This (principle) . . . makes possible a psychosomatic self-regulation . . . . Research using newly developed feedback techniques has demonstrated that volitional control of a number of internal states, psychological and physiological, is relatively easy to achieve (Green, 1970, p. 4).

Summary

Apparently the psychosomatic approach to the symptomatology of minimal brain dysfunction has not yet been given due consideration, although the theories of Reich (1945) and Perls (1969) emphasized bodily manifestations of neurosis which appear to overlap with the symptomatology of minimal brain dysfunction. Some research supporting these theories was commented upon in this chapter (Malmo, 1970 and 1957; Goldstein, 1965; Christiansen, 1963). It was noted that

Gestalt therapy seems to be particularly relevant to the integration of the sensory-motor and affective aspects of treatment.

Other psychosomatic perspectives which could be used in focusing on the symptomatology in question were also presented (Rolf, 1972; Green, Green, and Walters, 1970; Moses, 1954). Further research with all the theoretical and treatment modalities presented in this chapter was also proposed.

CHAPTER V

GENERAL SUMMARY AND IMPLICATIONS

Summary

The primary purpose of this study was to analyze the literature which deals with the affective and social dysfunctions included in the minimal brain dysfunction syndrome. Particular emphasis was placed on clarifying the role of neurological impairment and dysfunction, as it related to the behaviors in question. This was done with the particular goal of solidifying the ground to introduce a psychosomatic perspective to the symptomatology of the minimal brain dysfunction syndrome; this is really the core contribution of the study.

The background literature presented in the Introduction was primarily historical and theoretical in approach rather than research-oriented. The comment was made that the predominant etiology of the syndrome at this time is based on neurological impairment or dysfunction. Psychogenic theories were also noted as they related to a similar cluster of characteristics.

It was established that the syndrome applies to children of near average, average, or above average intelligence. Literature dealing with adults and children suffering from gross central nervous system dysfunction such as clear-cut brain damage, epilepsy, and cerebral palsy was excluded from consideration unless it was of particular relevance to the affective and social manifestations of the syndrome in question.

Prematurity and perinatal complications were viewed as somewhat related to the minimal brain dysfunction syndrome and to psychopathology in general. Certain studies suggested an interaction between the consequences of a traumatic perinatal period and the quality of familial and general psychosocial environmental conditions in later life.

Another approach exploring the etiology of minimal cerebral dysfunction examined the more frequent incidence of characteristic syndrome manifestations in minority group members; inherent cultural and socioeconomic biases in the diagnostic procedures and test instruments were noted. It was also concluded that the role of genetics in the etiology of this syndrome is at present little more than an interesting hypothesis.

Some of the studies reviewed concluded that symptoms subsumed under the description of minimal brain dysfunction are not a necessary cluster-type outcome of central nervous system lesions. It was noted that samples of minimal brain dysfunction children selected on the basis of

behavioral criteria, when contrasted with normal subjects, presented some equivocal neurological signs and electroencephalogram tracings and more general perceptual and/or motor and cognitive deficits as reflected in valid and reliable test instruments. The significance of these findings was not clear, inasmuch as the same signs are manifested in "normal" controls, although to a lesser degree. Such findings were not deemed adequate to differentiate between minimal brain dysfunction and childhood psychopathology in general. Promising research into the relationship of brain impairment to behavior was also commented upon.

It was concluded that at the present level of knowledge and understanding of the problem tests which purport to detect organicity in children when employed for individual diagnostic purposes are more useful in merely describing the behavioral deficits present; this information can then be incorporated into therapeutic and educational programs for the benefit of the individual child.

Some concern was expressed, however, about some psychometric instruments which are used for labeling children as "minimal brain dysfunction" amd are sensitive to biases arising from minority group status. Perhaps clinical accuracy could be improved in this area by introducing demographic data as moderator variables. The notion of moderator variables is based on the assumption that the validity of a test for a given criterion may vary among subgroups. A moderator variable enables the modification of categorical assignments

of different individuals with a given instrument.

The core contribution of this paper was the introduction of a psychosomatic orientation in viewing the behaviors in question. Gestalt therapy, because of its emphasis on integrating sensory-motor and affective aspects, was deemed particularly relevant as a theoretical and therapeutic orientation which merits further research. Similar comments were made in reference to other psychosomatic orientations such as Structural Integration and bio-feedback techniques. It was noted that no rigorous research exists in relation to the psychosomatic modalities of treatment in relation to children presenting the symptoms in question, although there were case reports of successful treatment outcomes. Further research was recommended for all the techniques discussed.

In relation to the research questions of this study, it can be concluded that the behaviors in question cannot be taken in themselves as diagnostic indices of neurological dysfunction or impairment. In addition, many of the medical and psychometric techniques currently used to identify minimal brain dysfunction children are of doubtful validity.

In conclusion, the third question posited can be answered in the affirmative: there are theoretical and therapeutic approaches of a psychosomatic orientation which have been ignored in the literature on minimal brain dysfunction. These psychosomatic approaches to treatment need to be submitted to more rigorous research.

General Implications

The subject matter of this study provokes some reflections on the problems science encounters when, after analyzing a phenomenon, a new synthesis of knowledge does not emerge.

Each researcher functions with respect to an underlying theoretical approach based upon implicit and/or explicity assumptions. In the study of human behavior the "somato-psychological" orientation stresses organic factors and relegates to a lesser role the possibility of a psychosomatic explanation. Yet "psychological" and "organic" merely refer to different perspectives of viewing phenomena. In essence, these words express different languages describing the same event (Graham, 1967). The choice of language is based on the level of analysis within which the researcher chooses to work.

Frequently, due to lack of adequate conceptual tools and measuring techniques, the two languages cannot be cross-translated; i.e., we cannot jump levels from the consequences of the Oedipal conflict to its underlying neuro-physiological correlates. Thus, within certain limits, the problem is not a matter of a theory at one level of analysis being "right" and another theory which lies within a different level being "wrong." Rather, based on given data at each specific level of analysis, the organic and the psychosocial approaches may each have some specific value in explaining the phenomena under consideration.

The choice of an "organic language," however, has more practical consequences. In the case of minimal cerebral dysfunction, the consequences of the choice of medical terminology often is used to justify attempts to solve the problem through psychoactive drugs; however, the efficacy of such treatment is still far from clearly established.

> Pharmocologicaltherapy has been evaluated primarily in patients with hyperactivity associated with learning disabilities. Subjective methods of assessment of motor activity have been employed more frequently than objective measurements, and well controlled analyses of drug effects are exceptional. An optimal design for a study of the effects of a drug on hyperactivity should be double-blind and include (1) a uniform group of patients of sufficient size to permit statistical analysis, and (2) matched controls and random allocation of patients to drug treatment or placebo (Millichap and Fowler, 1967, p. 768).

The extent of this usage of psychoactive drugs has led to the expression of some alarm (Stewart, 1971; Rogers, 1970), especially since the organic etiology of social and affective manifestations of minimal brain dysfunction (hyperactivity, emotional explosiveness, short attention span, etc.) is usually obscure and the symptoms are so loosely defined.

Another consequence of the organic orientation and therefore of the use of the label "minimal brain dysfunction" is that the symptomatology has tended to be explained in such a way that it becomes unrelated to the functioning of the child's family dynamics, the "healthiness" of the school environment for any particular child, and the quality and goals of the school system in general. This latter point is a matter of concern due to the number of "casualties" which the present system produces.

The organically-oriented may acknowledge the criticism of methodology presented in this paper while arguing that constructs such as "cerebral dysfunction" are more scientifically defensible than psychodynamic concepts such as "repression." Talland (1963), in discussing the "organic" versus "functional" (psychogenic) orientations in a context other than minimal brain dysfunction, noted that:

> The distinction between "organic" and "functional" disorders seems to imply either a theory of psychophysical interactionism or a hybrid position which admits parallelism partially or conditionally. It is improbable that many psychologists are likely to subscribe to the former, or to maintain that some behavior is accompanied by corresponding events in the brain but some is not. The distinction between "organic" and "functional," therefore, more probably reflects a decision whether one or another set of event constitutes a sufficient as well as necessary condition of a disorder. No matter which way the decision turns, which set of events is chosen, at least the final link in the chain must be some change within the patient. Any such event involves both neurological and psychological processes, and a verdict that antecedent conditions at one or the other level were of no consequence must rest on sufficient evidence. It has been demonstrated time and again that even severe injuries to the brain produce behavioral effects contingent on the patient's life history (1963, p. 347).

Current research does not warrant exclusive assumption of either position in relation to the behaviors in question. Insistence upon analysis of this phenomenon, which obviously involves both neurological and psychological processes, within one level of analysis, results in what Whitehead (1948) referred to as the fallacy of "misplaced concretion," that is, confusion of an ordering principle with reality itself. In principle it could be said that every symptom of psychopathology is an example of cerebral dysfunction, inasmuch as all behavior and experience is mediated

through the nervous system; however, because the neurophysiological and the psychological levels of analysis are not in practice directly translatable, such a position does not contribute to clarification of the phenomena in question. Furthermore, as illustrated by the works of Anderson (1970a), Rappaport (1969), and Bender (1959), there is no inherent opposition between the organic and the dynamic viewpoints.

The uncertain status of minimal brain dysfunction as a theoretical construct lies in the fact that the central nervous system impairment or irregularity is not known. One problem with this construct as defined by Clements (1966) is that the symptoms subsumed under it are so diverse as to include most behavioral disorders of children of above average, average, or near average intelligence excepting cerebral palsy, epilepsy, and extreme psychopathology such as clear-cut psychosis or autism. As proposed in the main text, research wich attacks the organic and social-psychogenic variables simultaneously is much needed.

The situation of the syndrome in question resembles a similar argument in relation to the etiology of schizophrenia. Baer (1961), in referring to this area, commented on the apparent confusion of phenotypes and genotypes; the same behavioral consequences from different causes and different behavioral consequences from the same causes.

> . . . There is sufficient evidence to suggest that organic or functional factors explain some cases and combination of both explain others, whether one speaks of anlage, triggers, or overflow. Various combinations of organic and psychogenic components will probably

> affect the potency of each component. A high degree of the psychogenic component is likely to act differently in conjunction with varying strengths of the organic component and vice versa. We may therefore expect not simply combined or mixed causation, but a more complex relationship, in which organic and psychogenic factors affect each other in producing pathology. Multiple causation suggests more than one source of pathology. The principle proposed here suggests that the several sources are not only related temporally, but are also reciprocally interactive in their effects (1961, p. 736).

It would seem that the current emphasis on the "somatopsychological" orientation to the etiology of minimal brain dysfunction is partly a reaction to the earlier dynamic position in which,

> Although lip service is often given to "constitutional factors," the overwhelming tendency has been to weave a complete causative fabric out of the fragile threads of stereotypes such as sibling rivalry, rejecting parents, repressed hostility, Oedipal conflicts, etc., much of which may well be secondary and epiphenomenal rather than primary (Clements and Peters, 1962, p. 185).

The importance of the family constellation in the development of the emotional health of the child was first recognized by the psychoanalytic approach, but this eventually became the exclusive focus of emphasis in the treatment of childhood developmental disorders (Kurlander and Colody, 1969). Misinterpretation of and dogmatic emphasis on the dynamics of the family constellation as the primary etiologic factor in learning disabilities have probably contributed greatly to the current emphasis on the organic etiology of the minimal brain dysfunction syndrome to the relative exclusion of those factors from consideration.

When presented at its best, the "family dynamics" approach can lead to genuine introspective examination by

parent and child of their respective roles. However, dogmatism, misapplication of psychoanalytic theory, and distorted popularization of the approach have contributed to a general attitude that parents are to blame for the social and emotional inadequacies of their children. Thus, parents frequently place upon themselves great responsibility for the shaping of their child's life, and a child who does not succeed contradicts the parents' desires to function successfully in this role. The subsequent guilt, feelings of failure, and occasional hostility directed against the child for not meeting the norms result in closed circuits of anxiety, guilt, and responsibility between parent and child. When this situation exists, the elicitation of these feelings can be of therapeutic value in opening them to examination and resolution, since such feelings are counterproductive for both parent and child. However, if a therapist -- out of his personal unresolved conflicts -- creates an attitude in the parents that they are to "blame" for their childrens' shortcomings, he does them a poor service and hinders further therapeutic progress. Competent psychoanalytically-oriented therapists work in helping to resolve feelings of "guilt" in both parent and child, not in creating them.

Millichap and Fowler (1967) and Conners (1967) have preferred to consider the term "minimal brain dysfunction" as a descriptive label, not a statement of a particular etiology. Masland (1969) concluded that to label a child as such was not to make any kind of diagnosis but simply served

to facilitate the practical problem of provision of services. Unfortunately, however, this label is usually reified in the literature, and children are spoken of as "having" minimal brain dysfunction.

This concept has been valuable in the sense that it has resulted in a degree of interdisciplinary cooperation in the study of childhood disorders, which is rare (Yates, 1966). Another consequence has been individualized contact, improved educational techniques, and tolerance and acceptance for children who manifest minor sensory-motor-perceptual and interpersonal problems of whatever origin. Considering present research, a multidisciplinary approach to identification as well as to treatment seems to be the most adequate choice.

REFERENCES

Abrams, A. L. Delayed and irregular maturation versus minimal brain injury: Recommendations for a change in current nomenclature. Clinical Pediatrics, 1969, 7, 344-349.

Alley, G. R., Solomons, G., and Opitz, E. Minimal cerebral dysfunction as it relates to social class. Journal of Learning Disabilities, 1971, 4:5, 246-250.

Anastasi, A. Psychological Testing. New York: MacMillan Company, 1968.

Anderson, R. P. A neuropsychogenic perspective in remediation of learning disabilities. Journal of Learning Disabilities, 1970 a, 3, 143-148.

Anderson, R. P. The child with learning disabilities and guidance. Boston: Houghton Mifflin, 1970b.

Anderson, W. The hyperkinetic child: A neurological appraisal. Neurology, 1963, 13, 968-973.

Baer, P. E. Problems in the differential diagnosis of brain damage and childhood schizophrenia. American Journal of Orthopsychiatry, 1961, 31, 728-738.

Bandura, A., and Walters, R. H. Social learning and personality development. New York: Holt Rinehart and Winston, Inc., 1963.

Barr, K. L., and McDowell Comparison of learning disabled and emotionally disturbed children in three deviant classrooms. Behavior and Exceptional Children, 1963, 39:1, 60-62.

Bateman, B. Learning disabilities -- yesterday, today, and tomorrow. In E. C. Frierson and W. B. Barbe (Eds.) Educating children with learning disabilities. New York: Appleton-Century-Crofts, 1967.

Bateman, B. Learning disabilities. Exceptional Child, 1964, 31, 167-178.

Battersby, N. S., Koieger, H. P., Pollack, M., and Bender, M. Figure-ground discrimination and the "abstract attitude in patients with cerebral neoplasms. AMA Archives of Neurology and Psychiatry, 1953, 70, 703-712.

Bax, M., and Mac Keith, R. Minimal cerebral dysfunction (Clinics in developmental medicine: Number 10). London: The National Spastics Society, 1963.

Bell, R. Adaptation of small wrist watches for mechanical recording of activity in infants and children. Journal of Experimental Child Psychology, 1968, 6, 302-305.

Bender, L. The brain and child behavior. Archives of General Psychiatry, 1961, 4, 531-547.

Bender, L. Psychiatric implications of brain damage in children. Psychiatric Quarterly, 1957, 31, 72-92.

Bender, L. The psychological problems and their management in the brain damaged child -- a summary. In Psychopathology of children with organic brain disorders. Springfield, Illinois: Charles C. Thomas, 1956.

Bender, L. Psychological problems of children with organic brain disease. American Journal of Orthopsychiatry, 1949, 19, 404-415.

Bender, L. Research monograph number three: A visual motor Gestalt test and its clinical uses. American Orthopsychiatric Association, New York, 1938.

Bender, M. B., Fink, M., and Green, M. Patterns in perception in simultaneous tactile stimuli in normal children. AMA Archives of Neurological Psychiatry, 1951, 66, 355.

Binet, A., and Simon, T. Mentally defective children. Translated by W. B. Drummond. London: Edward Arnold, 1914.

Birch, H. Research issues in child health: Some philosophic and methodological issues. In E. W. Grotberg (Ed.) Critical issues in research related to disadvantaged children. Princeton, New Jersey: Educational Testing Service, 1969.

Birch, H., Belmont, I., and Karp, E. The prolongation of inhibition in brain-damaged patients. Cortex, 1965, 1, 347-409.

Birch, H. (Ed.) Brain damage in children: The biological and social aspects. Baltimore: Williams and Wilkins, 1964a.

Birch, H., Thomas, A., and Chess, S. Behavioral development in brain-damaged children. Archives of General Psychiatry, 1964b, 11, 596-603.

Blau, A. Mental changes following head trauma in children. Archives of Neurological Psychiatry, 1937, 35, 723-769.

Blauvelt, H., and McKenna, J. Mother-neonate interaction: Capacity of the human newborn for orientation. In B. M. Foss (Ed.), Determinants of infant behavior. New York: Wiley and Sons, 1961, 3-35.

Blumberg, H. M. An investigation of the associating learning and memory span abilities of fifty brain-injured youngsters with normal intellectual potential. Temple University: Unpublished Doctoral Dissertation, 1967.

Bond, E. Postencephalitis, ordinary and extraordinary children. Journal of Pediatrics, 1932, 1, 310-314.

Bond, G. F., and Tinker, V. A. Reading difficulties: Their diagnosis and correction. New York: Appleton-Century-Crofts, 1957.

Bortner, M., and Birch, H. G. Perceptual and perceptual-motor disassociation in cerebral palsied children. Journal of Nervous and Mental Diseases, 1962, 134 103-108.

Boyle, R. H., Dykman, R. A., and Ackerman, P. T. Relationship of resting autonomic activity, motor impulsivity, and EEG tracings in children. Archives of General Psychiatry, 1965, 12, 314.

Brodie, B. B., Spector, I., and Shone, P. A. Interaction of drugs with norepinephrine in the brain. Pharmacological Reviews, June, 1959, 11, 548-564.

Brown, G. I. Human teaching for human learning: An introduction to confluent education. New York: Viking Books, 1971.

Butler, J. M., and Rice, L. N. Adience, self-actualization, and drive theory. In J. M. Wepman and R. W. Heine (Eds.) Concepts of Personality. Chicago: Aldine, 1963.

Butler, N., and Bonham, D. Perinatal mortality. Edinburgh: Livingston, Ltd., 1963.

Buxbaum, E. The parents' role in the etiology of learning disabilities. The Psychoanalytical Study of the Child, 19, 1964, 421-447.

Campbell, I. B., Douglas, V. J., and Morgenstern, G. Cognitive styles in hyperactive children and the effect of methylphenidate. Journal of Child Psychology and Psychiatry and Allied Disciplines, 1971, 12:1, 55-67.

Cantor, H. E. Role of the neurologist in management of children with learning disabilities. In J. Hartstein (Ed.) Dyslexia. St. Louis, Missouri: C.V. Mosby, 1971, 182-197.

Caputo, D. V., Edmonton, N. E., L'Abate, L., and Rondberg, S. R. Extended report: Type of brain damage and intellectual functioning in children. Paper read at Midwestern Psychological Association Meeting, Washington University School of Medicine, 1962.

Caputo, D. V., and Mandell, D. Consequences of low birth weight. Developmental Psychology, 1970, 3:3, 363-383.

Cawley, J. F. An assessment of intelligence, psycholinguistic abilities, and learning aptitudes among preschool children. Project Head Start: ED-014-325, 1966.

Cazden, C. Some implications of research on language development for preschool education. In R. D. Hess and R. M. Bear (Eds.) Early Education. Chicago: Aldine, 1968, 131-142.

Chalfant, J. C., and Scheffelin, M. A. (Eds.) Central processing dysfunctions in children: A review of research. National Institute of Neurological Diseases and Strokes. U. S. Department of Health, Education, and Welfare. Bethseda, Maryland: Urbana, Illinois: Institute for Research on Exceptional Children, 1969.

Christiansen, B. Thus speaks the body. Monograph: Institute for Social Research, Oslo, Norway, 1963.

Clawson, A. Relationship of psychological tests to cerebral disorders in children: A pilot study. Psychological Report, 1962, 10, 187-90.

Clements, S. D. Minimal brain dysfunction in children: Terminology and identification. (PHS publication #1415). Washington, D. C.: U. S. Public Health Service, 1966.

Clements, S. D., and Peters, J. E. Minimal brain dysfunctions in the school-age child: Diagnosis and treatment. Archives of General Psychiatry, 6, 1962, 185-197.

Coleman, J. C. Abnormal psychology and modern life. Glenview, Illinois: Scott, Foresman, and Co., 1972.

Conners, C. K. The syndrome of minimal brain dysfunction: Psychological aspects. Pediatric Clinics of North America, 1967, 14:4.

Conners, C. K. The effect of dexedrine on rapid discrimination and motor control of hyperkinetic children under mild stress. Journal of Nervous and Mental Diseases, 1966, 142, 429-433.

Conners, C. K., and Eisenberg, L. The effects of methylphenidate on symptomatology and learning in disturbed children. American Journal of Psychiatry, 1963, 120, 458-484.

Cravioto, J. Environmental influences. (D. C. Glass, Ed.) New York: Rockefeller University Press, 1968, 3-51.

Critchley, M. Developmental dyslexia. London: Heimermann, 1964.

Crolidge, J. C., Tessman, E., Waldfogel, S., and Willer, M. L. Patterns of aggression in school phobia. In Psychoanalytic Study of the Child, Volume XVII. New York: International Universities Press, 1962, 319-334.

Davidson, A. N., and Peters, A. Myelination. Springfield, Illinois: Charles C. Thomas, 1970.

Davis, K., Sprague, R., and Werry, J. Stereotyped behavior and activity level in severe retardates: The effect of drugs. American Journal of Mental Deficiency, 1969, 73, 721-727.

De Hersch, K. Two categories of learning difficulties in adolescence. American Journal of Orthopsychiatry, 1963, 33, 87-91.

Delacato, C. H. The treatment and prevention of reading problems: The neuropsychological approach. Springfield, Illinois: Charles C. Thomas, 1959.

Denhoff, E., and Chammas, E. Diazapan as an aid in the Photo-Metrazol Activation Test. Disorders of the Nervous System, 1968b, 29, 759-762.

Denhoff, E., Davids, A., and Hawkins, R. Effects of dextro-amphetamine on hyperkinetic children: A controlled double-blind study. Journal of Learning Disabilities, 1971, 4:9, 491-499.

Denhoff, E., Hainsworth, P. K., Siqueland, M. The measurement of psychoneurological factors contributing to learning efficiencies. Journal of Learning Disabilities, 1968, 1, 636-644.

De Renzi, E., and Vignolo, L. A. The Token Test: A sensitive test to detect receptive disturbances in aphasics. Brain, 1962, 85, 665.

Deutsch, C. P. Auditory discrimination and learning: Social factors. Merril-Palmer Quarterly, 1964, 10, 277-296.

Deutsch, C. P., and Schumer, F. Brain-damaged children. New York: Brunner/Mazel, 1970.

Doty, R. W. Conditioned reflexes forced and evoked by brain stimulation. In D. E. Sheer (Ed.) Electrical stimulation of the brain. Austin: University of Texas Press, 1961.

Douglas, J. W. B. Mental ability and school achievement of premature children at eight years of age. British Medical Journal, 1956, 1, 1210-1214.

Dreger, R. A. A progress report in a factor analytical approach to classification in child psychiatry. In R. Jenkins and J. Cole (Eds.) Research Report Number 18, American Psychiatric Association, 1964.

Drillien, C. M. The growth and development of the prematurely-born infant. Baltimore, William and Wilkins, 1964.

Eiduson, B. T., Johnson, T. C., and Rottenberg, D. Comparative studies of learning problems seen in five child clinics. American Journal of Orthopsychiatry, 1966, 36, 829-839.

Eisenberg, L. Behavioral manifestations of cerebral damage in children. In H. Birch (Ed.) Brain damage in children: Biological and social aspects. Baltimore: Williams and Wilkins, 1964.

Eisenberg, L. Psychiatric implications of brain damage in children. Psychiatric Quarterly, 1957, 31, 72.

Ellenbogen, E. M., and Thompson, G. B. A comparison of social class effects in two tests of auditory discrimination. Journal of Learning Disabilities, 1972, 5:4, 2-9-212.

Ellingson, R. J. Relationships between EEG and test intelligence: A commentary. Psychological Bulletin, 1966, 1:65, 91-98.

Ellingson, R. J. The incidence of EEG abnormality among patients with mental disorders of apparently non-organic origin: A critical review. American Journal of Psychiatry, 1954, 111, 263-275.

Ellis, A. and Pryor, R. Quantification of gross bodily activity in children with severe neuropathology. American Journal of Mental Deficiency, 1959, 63, 1034-1037.

Epstein, L., et al. Correlation of dextroamphetamine excretion and drug response in hyperkinetic children. Journal of Nervous and Mental Diseases, 1968, 146, 136-146.

Ernhart, C. B., Graham, F. K., Eichman, P. L., Marshall, J. M., and Thurton, D. Brain injury in the preschool child: Some developmental considerations; II. Comparison of brain-injured and normal children. Psychological Monographs, 1963, 77, 574.

Fagan, A. Gestalt techniques with a woman with expressive difficulties. In J. Fagan and I. L. Shepherd (Eds.) Gestalt therapy now. Palo Alto, California: Science and Behavior Books, Inc. 1970, 169-196.

Ferguson-Smith, M. A. Sex chromatin: Klinefelter's syndrome and mental deficiency. In K. L. Moore (Ed.) The sex chromatin. Philadelphia: W. B. Saunders Co., 1966, 292.

Foshee, J. Studies in activity level: I. Simple and complex task performance in defectives. American Journal of Mental Deficiency, 1958, 62, 882-886.

Fossman, H., and Hambert, G. Incidence of Klinefelter's syndrome among mental patients. Lancet, 1963, 1, 132.

Francis-Williams, J. Problems of development in children with "minimal brain damage." In M. Bax and R. Mac Keith (Eds.) Minimal cerebral dysfunction (Clinics in developmental medicine; Number 10). London: The National Spastics Society, 1963.

Freeman, R. D. Review of drug effects on learning in children. San Rafael, California: Academic Therapy Publications, 1969.

Freeman, R. D. Drug effects on learning in children: A selective review of the past thirty years. Journal of Special Education, 1966, 1, 17-44.

Freeman, R. D. Special education and electroencephalogram: Marriage of convenience. Journal of Special Education, 1967, 2, 61-73.

Freibergs, V. Concept learning in normal and hyperactive children. McGill University: Unpublished Doctoral Dissertation, 1965.

Freibergs, V., and Douglas, V. L. Concept learning in hyperactive and normal children. Journal of Abnormal Psychology, 1969, 74:3, 388-395.

Frost, B. P. An application of the method of extreme deviations to the WISC. Journal of Clinical Psychology, 1960, 16, 420-430.

Freud, A. Normality and pathology in childhod. London: The Hogarth Press and the Institute of Psychoanalysis, 1969.

Freud, S. Psychogenic visual disturbance according to psychoanalytic conceptions. Standard edition, Volume II. London: Hogarth, 1949.

Frostig, M. The relationship of diagnosis to remediation in learning problems. International Approach to Learning Disabilities of Children and Youth, 1966, 45-66.

Frostig, M., Lefever, W., and Whittlesley, J. R. Administration and scoring manual, Developmental Test of Visual Perception. Palo Alto, California: Consulting Psychologists Press, 1966.

Frostig, M., Lefever, D. W., and Whittlesey, J. R. The Marianne Frostig Developmental Test of Visual Perception. Palo Alto, California: Consulting Psychologists Press, 1964.

Fuisz, R. E., and Fuisz, R. C. The hyperactive child. Summit, New Jersey: Medcom, Inc., 1971.

Gesell, A., and Armatruda, C. Developmental diagnosis. New York: Hoeber, 1941.

Goldstein, I. B. The relationship of muscle tension and autonomic activity to psychiatric disorders. Psychosomatic Medicine, 1965, 27, 39-52.

Goldstein, K. Language and language disturbances. New York: Greve and Stratton, 1948.

Gomez, B. M. Minimal cerebral dysfunction (maximal neurologic confusion). Clinical Pediatrician, 1967, 6, 589.

Graham, F. K., Ernhart, C., Craft, M., and Berman, D. W. Brain injury in the preschool child: Some developmental consideration; I. Performance of normal children. Psychological Monographs, 1963, 77, 573.

Graham, F. K., Ernhart, C. B., Thurston, D., and Craft, M. Development three years after perinatal anoxia and other potentially damaging newborn experiences. Psychological Monographs, 1962, 76, 522.

Graham, D. T. Health, disease and the mind-body problem: Linguistic parallelism. Psychosomatic Medicine, 1967, 24, 52-71.

Graham, P., and Rutter, M. Organic brain dysfunction and child psychiatric disorder. British Medical Journal, 1968, 3, 695.

Gastaut, H. Combined photic and Metrazol activation of the brain. Electroencephalographic Clinical Neurophysiology, 1950, 2, 249.

Grastyan, E. The hippocampus and higher nervous activity. In M. A. Brazier (Ed.) The central nervous system and behavior. New York: Josiah Macey, Jr. Foundation, 1959.

Green, E. E., Green, A. M., Walters, E. D. Voluntary control of internal states: Psychological and physiological. Journal of Transpersonal Psychology, 1970, 2:1, 1-26.

Grossman, H. J. The child, the teacher, and the physician. In W. M. Cruikshank (Ed.) The teacher of brain-injured children, Syracuse University Press, 1966, 59-67.

Grotberg, E. H. Neurological aspects of learning disabilities: A case for the disadvantaged. Journal of Learning Disabilities, 1970, 3:6, 321-327.

Grotberg, E. H. Review of Research -- 1965-1969. Project Head Start: OEO Pamphlet 6108-B, 1969.

Grotberg, E. H. Role of the parent in fostering early learning. Education, 1968, 89, 35-39.

Grubler, E. R., and Gochman, S. I. Statistical observations regarding the clinical diagnosis of brain damage. Psychological Reports, 1962, 11, 221-222.

Gruenberg, E. M. Some epidemiological aspects of congenital brain damage in children. In H. C. Birch (Ed.) Brain damage in children. Baltimore: Williams and Wilkins, 1964, 118-127.

Halstead, W. C. Brain and intelligence. Chicago: University of Chicago Press, 1947.

Heath, S. R. A mental pattern found in motor deviates. Journal of Social Psychology, 1946, 41, 223-225.

Heath, S. R. Clinical significance of motor defects with military implications. American Journal of Psychology, 1944, 57, 482-499.

Hebb, D. O. Intelligence, brain function and the theory of mind. Brain, 1959, 82, 260-275.

Heinicke, C. M. Frequency of psychotherapeutic session as a factor affecting outcome: Analysis of clinical ratings and test results. Journal of Abnormal Psychology, 1969, 74:5, 553-560.

Heinicke, C. M. Frequency of psychotherapeutic session as a factor affecting the child's developmental status. Psychoanalytic Study of the Child, 1965, 20, 42-98.

Heller, M. D. A study of psychiatric patients tested on the Weschler Intelligence Scales. University of London: Unpublished Doctoral Dissertation, 1963.

Hellman, I. Some observations on mothers of children with intellectual inhibitions. Psychoanalytic Study of the Child, 1954, 9.

Herron, R., and Ramsden, R. Continuous monitoring of overt human body movement by radio telemetry: A brief review. Perceptual Motor Skills, 1967, 24, 1303-1308.

Hertzig, M. E., and Birch, H. G. Neurologic organization in psychiatrically disturbed adolescents. Archives of Psychiatry, 1968, 19, 5.

Heston, L. Psychiatric disorders in foster home reared children of schizophrenic mothers. British Journal of Psychiatry, 1966, 112, 819-825.

Hewett, F. M. The emotionally disturbed child in the classroom: A developmental strategy for educating children with maladaptive behavior. Boston: Allyn and Bacon, Inc., 1968.

Hewett, F. M. A hierarchy of educational tasks for children with learning disorders. Exceptional Child, 1964, 31, 207-214.

Hillarp, N. A., Fuxe, K., and Dahlstrom, A. Demonstration mapping of central nervous system containing dopamine, noradrenaline, and 5-hydronytryptaminl and their reactions to psychopharmaca. Pharmacological Review, 1966, 18:1, 727-741.

Hirsch, J. Behavior-genetic analysis and its biosocial consequences. Seminars in Psychiatry, 1970, 2, 89-105.

Hist, J. B. Manifestation of the brain damage syndrome in school. In Rappaport (Ed.) Childhood aphasia and brain damage: A definition. Nabreth, Pennsylvania: Livingston Publishing Company, 1964.

Huessy, H. R., and Wright, A. L. Use of imipramine in children's behavior disorders. Presented at the 122nd Annual Meeting of the American Psychiatric Association, Bal Harbor, Florida, May 7, 1969.

Hughes, J. Electroencephalography and learning. In H. Myklebust (Ed.) Progress in learning disabilities. New York: Grune and Stratton, 1968, 132.

Hughes, J., and Park, J. EEG in dyslexic children. In H. Myklebust (Ed.) Progress in learning disabilities. New York: Grune and Stratton, 1968, 132.

Hunt, H. F. Diagnostic methods: V. Psychological testing. In A. B. Baker (Ed.) Clinical Neurology. New York: Hoeber-Harper, 1955, 311-329.

Hunt, V. Tentative report of electromyographic study of Structural Integration. Ida P. Rolf Foundation for Structural Integration, 1972.

Hutt, C., Hutt, S., and Oursted, C. The behavior of children with and without upper central nervous system lesions. Behavior, 1964, 24, 246-268.

Ingram, T. Chronic brain syndromes in childhood other than cerebral palsy, epilepsy, and mental defect. In M. Bax and R. Mac Keith (Eds.) Minimal cerebral dysfunction (Clinics in developmental medicine; Number 10). London: The National Spastics Society, 1963.

Ingram, T. The neurology of cerebral palsy. Archives of Disorders of Childhood, 1966, 41, 337.

Jarvis, J. Clinical observation on the visual problems in reading disability. Psychoanalytic Study of the Child, Volume XIII, New York: International Universities Press, Inc., 1958.

Jenkins, R. L. Psychiatric syndromes in children and their relation to family background. American Journal of Orthopsychiatry, 1966, 36, 458-457.

Jensen, A. How much can we boost IQ and scholastic achievement? Harvard Educational Review, 1969, 29:1, 123.

John V., and Berney, G. Analysis of story retelling as a measure of the effects of ethnic content in stories: A study of Negro, Indian, and Spanish-American children. Project Head Start: ED-014-326, 1967.

Johnson, D., and Myklebust, H. R. Learning disabilities. New York: Grune and Stratton, 1967.

Kagan, J. Inadequate evidence and illogical conclusions. Harvard Educational Review, 1969, 39, 274-277.

Kagan, J., Moss, H. A., and Sigel, I. E. Psychological significance of styles of conceptualization. In J. C. Wright and J. Kagan (Eds.) Basic cognitive processes in children. Monographs of the Society for Research in Child Development, 1963, 28:2.

Kahn, E., and Cohen, L. H. Organic drivenness: A brain stem syndrome and an experience. New England Journal of Medicine, 1934, 210, 748-756.

Kahn, J. P. The emotional concomitants of the brain-damaged child. Journal of Learning Disabilities, 1969, 2:12, 644-651.

Karlsson, J. The biological basis of schizophrenia. Springfield, Illinois: Charles C. Thomas, 1966.

Kennard, M. Value of equivocal signs in neurologic diagnosis. Neurology, 1960, 10, 753-764.

Kennedy, C., and Ramirez, L. S. Brain damage as a cause of behavior disturbance in children: The biological and social aspects. Baltimore: Williams and Wilkins, 1964.

Kephart, N. C. The slow learner in the classroom. Columbus, Ohio: Charles E. Merrill, 1967.

Kerlinger, F. N. Foundations of behavioral research. New York: Holt, Tinehart, and Winston, 1964.

Kessler, J. W. Psychopathology of childhood. New Jersey: Prentice-Hall, 1966.

Kinney, T., Balwin, R., and Mackie, J. B. Incidence of minimal brain injury in adopted children. Child Welfare, 1967, 24-29.

Kirk, S. S., McCarthy, J. J., and Kirk, W. D. The Illinois Test of Psycholinguistics Abilities, Revised Edition. Urbana: University of Illinois, 1968.

Klatskin, E. H. Relationship of deficits in intelligence test performance of preschool children to perinatal experience. Journal of Consulting Psychology, 1964, 28, 228-233.

Klaus, R., and Gray, S. The early training project for disadvantaged children: A report after five years. Monographs of the Society for Research in Child Development, 1968, 33:4.

Knobel, M., Wolman, M. B., and Mason, E. Hyperkinesis and organicity in children. Archives of General Psychiatry, 1959, 1, 310-321.

Knobloch, H., and Pasamanick, B. The syndrome of minimal cerebral damage in infancy. Journal of the American Medical Association, 1959, 170, 1384.

Koff, R. H. A panel on learning difficulties in childhood. Reported by E. A. Anthony for the Journal of the American Psychological Association, 1961, 9.

Koffitz, E. M. The Bender Gestalt Test for Young Children. New York: Grune and Stratton, 1964.

Kohlberg, L. Montessori with the culturally disadvantaged. In R. D. Hess and R. M. Bear (Eds.) Early Education. Chicago: Aldine, 1968, 105-118.

Kong, E. Minimal cerebral palsy: The importance of its recognition. In M. Bax and R. MacKeith (Eds.) Minimal cerebral dysfunction (Clinics in developmental medicine; Number 10). London: The National Spastics Society, 1963.

Kurlander, L. S., and Colodny, D. "Pseudoneurosis" in the neurologically handicapped child. American Journal of Orthopsychiatry, 1965, 35, 733-738.

Lapouse, R., and Wouk, M. A. An epidemiological study of behavior characteristics in children. In H. Quay (Ed.) Children's behavior disorders. Princeton, New Jersey: Van Nostrand, 1968.

Laufer, M. W., Denhoff, E., and Solomons, G. Hyperkinetic impulse disorder in children's behavior problems. Journal of Psychosomatic Medicine, 1957, 19:38.

Lee, D., and Hutt, C. A play-room designed for filming children: A note. Journal of Child Psychology and Psychiatry, 1964, 5, 263-265.

Lederman, J. Anger and the rocking chair: Gestalt awareness with children. New York: McGraw-Hill, 1969.

Leonard, H. L., et al. Hazards implicit in prescribing psychoactive drugs. Science, 1970, 169, 438-441.

Levine, S., and Otis, L. S. The effects of handling before and after weaning on the resistance of albino rats to later deprivation. Canadian Journal of Psychology, 1958, 18, 103-108.

Levinson, B. M. Traditional Jewish cultural values and performance on the Weschler tests. Journal of Educational Psychology, 1959, 50, 177-181.

Lowen, A. The betrayal of the body. New York: MacMillan, 1967.

Lowen, A. Physical dynamics of character structure. New York: Grune and Stratton, 1958.

Luria, A. R. Higher cortical functions in man. New York: Basic Books, 1966.

Luria, A. R. Restoration of function after brain injury. New York: MacMillan, 1963.

McCleary, R. A., and Moore, R. Y. Subcortical mechanisms of behavior. New York: Basic Books, 1965.

McCollum, P. S. Group counseling as an adjunctive remediation technique for learning disabilities. Texas Tech University: Unpublished Doctoral Dissertation, 1971.

McDermott, F. Social class and mental illness in children: the diagnosis of organicity and mental retardation. Journal of the American Academy of Child Psychiatry, 1967, 6, 307-320.

McFarland, J., Peacock, L., and Watson, J. Mental retardation and activity level in rats and children. American Journal of Mental Deficiency, 1966, 71, 381-386.

McFie, J. An introduction to the problem of "minimal brain damage." In M. Bax and R. Mac Keith (Eds.) Minimal cerebral dysfunction (Clinics in developmental medicine; Number 10). London: The National Spastics Society, 1963.

Mac Keith, R. Defining the concept of "minimal brain damage." In M. Bax and R. Mac Keith (Eds.) Minimal cerebral dysfunction (Clinics in developmental medicine; Number 10). London: The National Spastics Society, 1963.

Mahler, M. A. A psychoanalytic evaluation of tics in psychopathology of children. Psychoanalytic Study of the Child, 1949, 279-310.

Malmo, R. B. Emotions and muscle tension: The story of Anne. Psychology Today, 1970, 3:10, 64-84.

Malmo, R. B. Anxiety and behavioral arousal. Psychological Review, 1957, 64, 276-287.

Malmo, R. B., Shagass, C., and Smith, A. A. Responsiveness in chronic schizophrenia. Journal of Personality, 1965, 19:4, 368.

Marx, M. H. Psychological theory: Contemporary readings. New York: MacMillan, 1961.

Masland, R. E. Children with minimal brain dysfunction. In Tarnapol (Ed.) Learning disabilities, Springfield: Illinois: Charles C. Thomas, 1969.

Maxwell, A. E. Discrepancies between the pattern of abilities for normal and neurotic children. Journal of Mental Science, 1961, 167, 300-307.

Medelson, W., Johnson, N., and Stewart, M. A. Hyperactive children as teenagers: A followup study. Journal of Nervous and Mental Diseases, 1971, 153:4, 273-279.

Mednick, S. A. Birth defects and schizophrenia. Psychology Today, 1971, 4, 48-58.

Mednick, S. A. Breakdown in individuals at high risk for schizophrenia: Possible predispositional perinatal factors. Mental Hygiene, 1970, 54:1, 50-63.

Mehrabian, A. An analysis of personality theories. Englewood Cliffs: Prentice-Hall, 1968.

Mesinger, J. F. Emotionally disturbed and brain injured children -- should we mix them? Exceptional Children, 1965, 32, 237-238.

Millichap, J. G., and Fowler, G. W. Treatment of "minimal brain dysfunction" syndromes. In J. G. Millichap (Ed.) The pediatric clinics of North America. Philadelphia: W. B. Saunders, 1967, 766-777.

Millman, H. L. Minimal brain dysfunction in children: Evaluation and treatment. Journal of Learning Disabilities, 1970, 3:2, 89-99.

Minde, K., Webb, G., and Sykes, D. Studies on the hyperactive child; IV. Prenatal and perinatal factors associated with hyperactivity. Developmental Medicine and Child Neurology, 1968, 10, 355-363.

Money, J. (Ed.) The disabled reader: Education of the dyslexic child. Baltimore: Johns Hopkins Press, 1966.

Montessori, M. The Montessori method. New York: Frederick A. Stokes, 1912.

Morrison, J. R., and Stewart, M. A. A family study of the hyperactive child syndrome. Biological Psychiatry, 1971, 3, 189-195.

Morrison, J. R. Hyperactivity: Links with alcoholism. Science News, 1970, 97:21, 507.

Moruzzi, C., and Magoren, H. E. Brain stem reticular formation and activation of the EEG. Electroencephalograph and Clinical Neurophysiology, 1949, 1, 455-573.

Moses, P. J. The voice of neurosis. New York: Grune and Stratton, 1954.

Mozier, H. D., Scott, L. W., and Dingman, H. T. Sexually deviant behavior and Klinefelter's syndrome. Journal of Pediatrics, 1960, 57, 479.

Mueller, R., Nylander, I., Larson, L., Widen, L., and Frankenhauser, M. Sequelae of primary aseptic meningoencephalitis: A clinical, sociomedical, electroencephalographic and psychological study. Acta Psychiatrica Scandinavica, 1958, 33, 126.

Murphy, G. Historical introduction to modern psychology (revised edition). New York: Harcourt, Brace, and World, 1949.

Ozer, M. N. The neurological evaluation of school-age children. Journal of Learning Disabilities, 1968, 1, 84-87.

Paine, R. S. Minimal brain dysfunction in children: Educational, medical, and health related services. (PHS publication #2015) Washinton, D. C.: U. S. Department of Public Health, 1969.

Paine, R. S. Neurological grand rounds: Minimal chronic brain syndromes. Clinical Procedings of Childrens' Hospital, Washington, D. C., 1966, 22, 21.

Paine, R. S. Organic neurological factors related to learning disabilities. In J. Hellmuth (Ed.) Learning Disorders. Seattle: Special Child Publications, 1965, 1-29.

Paine, R. S. Minimal chronic brain syndromes in children. Developmental Medicine and Child Neurology, 1962, 4, 21-27.

Paine, R. S., Werry, J., and Quay, H. A study of "minimal brain dysfunction." Developmental Medicine and Child Neurology, 1968, 10, 505-520.

Pasamanick, B. The epidemiology of behavior disorders of childhood. Research Publications of the Association for Research in Nervous and Mental Disorders, 1954.

Pasamanick, B., Rogers, M., and Lilienfeld, A. Programming experience and the development of behavior disorders in children. American Journal of Psychiatry, 1956, 112, 613-619.

Penfield, W., and Roberts, L. Speech and brain mechanisms. Princeton, New Jersey: Princeton University Press, 1959.

Perls, F. Ego, hunger, and agression. New York: Random House, 1964.

Perls, F., Hefferline, R., Goodman, P. Gestalt therapy. New York: Dell Publishing Company, 1951.

Perls, L. Two instances of gestalt therapy. In P. D. Pursglove (Ed.) Recognitions in gestalt therapy. New York: Harper and Row, 1968.

Pond, D. The EEG in pediatrics. In D. Hill and G. Parr (Eds.) Electroencephalography. New York: MacMillan, 1963.

Pond, D. Psychiatric aspects of epileptic and brain-damaged children. British Medical Journal, 1961, 2, 1377-1382, 1454-1459.

Pond, D. Is there a syndrome of "brain damage" in children? Cerebral Palsy Bulletin, 1960, 2, 296.

Quay, H., Morse, W., and Cutler, R. Personality patterns of pupils in special classes for the emotionally disturbed. Exceptional Children, 1966, 32, 297-301.

Rabinovitch, R. D. Reading and learning disabilities. In S. Arieta (Ed.) An american handbook of psychiatry. New York: Basic Books, 1959, 857-869.

Rappaport, S. R. Public education for children with brain dysfunction. Syracuse: Syracuse University Press, 1969.

Rappaport, J. Childhood behavior and learning problems treated with amphetamines. International Journal of Neuropsychiatry, 1965, 12, 635-642.

Reich, W. Character analysis. New York: Orgone Institute Press, 1949.

Reich, W. The discovery of the orgone. New York: Noonday Press, 1942.

Reitan, R., and Heineman, C. Interaction of neurological deficits and emotional disturbances in children with learning disorders: Methods for differential assessment. In J. Helmuth, Learning disorders, Volume 3. Seattle: Special Child Publications, 1968, 93-136.

Reitan, R. M. Relationships between neurological and psychological variables and their implications for reading instruction. In H. A. Robinson (Ed.) Meeting individual differences in reading. Chicago: University of Chicago Press, 1964, 100-110.

Richardson, S. D. Learning disabilities: An introduction. International Approach to Learning Disabilities of Children and Youth, 1966, 11-19.

Reisen, A. H. Problems in correlating behavioral and physiological development. In M. D. Sterman, D. J. McGinty, and A. M. Adriolfi (Eds.) Brain development and behavior. New York: Academic Press, 1971, 59-70.

Robbins, M. P. A study of the validity of Delacato's theory of neurological organization. Exceptional Children, 1966, 32, 517-523.

Robinson, N. M., and Robinson, H. B. A. Follow-up study of children of low birth weight and control children at school age. Pediatrics, 1965, 35, 425-433.

Rodin, E., Lucas, A., and Simson, C. A study of behavior disorders in children by means of general purpose computers. In Procedings of the conference on data acquisition and process in biology and medicine, New York: Pergamon Press, 1963, 115-124.

Rogers, J. M. Drug abuse by prescription. Psychology Today, 1971, 5:4, 16-24.

Roe, A., and Mittleman, B. Adult adjustment of foster children of alcoholic and psychotic parentage and the influence of the foster home. Memoirs, Quarterly Journal of Study of Alcoholics, Number 3, 1945.

Rolf, I. Structural integration: A contribution to the understanding of stress. Ida P. Rolf Foundation for Structural Integration, 1972.

Rolf, I. Gravity: An unexplored factor in a more human use of human beings (privately published). In S. Peterson, A catalog of the ways people grow. New York: Balla Ballantine Books, 1971.

Rolf, I. Structural integration. Systematics, 1963, 1, 1.

Rosanes-Berrett, M. B. Gestalt therapy as an adjunct treatment for some visual problems. In J. Fagan and F. S. Shepherd (Eds.) Gestalt therapy now, Palo Alto, California: Science and Behavior Books, 1970.

Rose, D., Smith, R. E., and Robles, A. Some problems in perceptual handicap of mentally retarded children. Journal of Genetic Psychology, 1964, 104, 123-133.

Rothschild, B. F. Incubator isolation as a possible contributing factor to the high incidence of emotional disturbance among prematurely born persons. Journal of Genetic Psychology, 1967, 110:2, 287-304.

Routtenberg, A. The two arousal hypotheses: reticular formation and limbic system. Psychological Review, 1968, 51-80.

Rubinstein, B. O., Fabek, M., Levitt, M., and Epstein, R. Learning impotence: A suggested diagnostic category. American Journal of Orthopsychiatry, 1959, 29, 315-324.

Sainz, A. Hyperkinetic disease of children: Diagnosis and therapy. Diseases of Nervous System, 1966, 27, 48.

Sarason, S. B. Strauss brain injured child. In Psychological problems in mental deficiency. New York: Harper Brothers, 1949.

Scharberg, S. M., Schildkraut, J. J., and Kopin, I. J. The effects of psychoactive drugs on norepinephrine - 3H metabolism in the brain. Biochemical Pharmacology, 1967, 16, 393-399.

Scheibel, M. E., and Scheibel, A. B. Selected structural functional correlations in postnatal brains. In M. B. Sternman, et al. (Eds.) Brain development and behavior. New York: Academic Press, 1971, 1-21.

Schulman, J., Kaspar, J., and Throne, F. Brain damage and behavior: A clinical experimental study. Springfield, Illinois: Charles C. Thomas, 1965.

Sherman, M., and Beverly, B. I. The factors of deterioration in children showing behavior difficulties after epidemic encephalitis. Archives of Neurological Psychiatry, 1923, 10, 329-343.

Silver, A. A. Diagnosis and prognosis of behavior disorders associated with organic brain disease in children. Journal of Internal Medicine, 1951, 38-42.

Silver, L. B. A proposed view of the etiology of the neurological learning disability syndrome. Journal of Learning Disabilities, 1971a, 4, 123-133.

Silver, L. B. Familial patterns in children with neurologically based learning disabilities. Journal of Learning Disabilities, 1971b, 4, 349-358.

Silver, L. B. Frequency of adoption in the children with the neurological learning disability syndrome. Journal of Learning Disabilities, 1970, 3:6, 306-310.

Silverman, J., Rappaport, M., Hopkins, K., Ellman, G., Hubbard, R., Belleza, T., Baldwin, T., Griffin, R., Kling, R. Stress, stimulus intensity control, and the Structural Integration technique. Ida P. Rolf Foundation for Structural Integration, 1972.

Silverman, J., Fite, M. W., Mosher, M. M. Clinical findings in reading disability children: Special cases of intellectual inhibition. American Journal of Orthopsychiatry, 1959, 29, 298-314.

Silverman, A. J., and Harris, V. W. Electroencephalography and psychometric testing in brain damaged patients. The Journal of Nervous and Mental Disease, 1954, 120, 31-39.

Sperry, B., Staver, N., and Mann, H. E. Destructive fantasies in certain learning difficulties. American Journal of Orthopsychiatry, 1952, 22, 356-366.

Sperry, B., Ulrich, D., and Staver, W. Relationship of motility to boys' learning problems. American Journal of Orthopsychiatry, 1958, 28, 640-646.

Spitz, R. A. Hospitalism: A follow-up report. The Psychoanalytic Study of the Child, Volume VII, New York: International Universities Press, 1946.

Sprague, R., and Toppe, Y. Relationship between activity level and delay reinforcement in the retarded. Journal of Experimental Child Psychology, 1966, 3, 390-397.

Solkoff, N., Joffe, S., and Weintraub, D. Effects of handling on the development of premature infants. Paper presented at the meeting of the Eastern Psychological Association Convention, Boston, 1967.

Stevens, D. A., Boydsturn, J. A., Dykman, R. A., Peters, J. E., and Sinton, D. W. Presumed minimal brain dysfunction in children: Relationship to performance on selected behavioral tests. Archives of General Psychiatry, 1967, 281-285.

Stewart, M., Pitts, F., Craig, A., and Dienerf, W. The hyperactive child syndrome. American Journal of Orthopsychiatry, 1966, 36, 861-867.

Stewart, M. A. Medical Tribune, June 7, 1972.

Stodolsky, S. S., and Lesser, G. Learning patterns of the disadvantaged. Harvard Educational Review, 1967, 37, 546-593.

Strauss, A. A., and Lehtinen, L. Psychopathology and education of the brain-injured child. New York: Grune and Stratton, 1947.

Strecker, E. Behavior problems in encephalitis. Archives of Neurology and Psychiatry, 1929, 21, 137-144.

Strother, C. L. Discovering, evaluating, programming for the neurologically handicapped child with special attention to the child with minimal brain damage. Chicago: National Society for Crippled Children and Adults, 1963.

Talland, G. A. Psychology's concern with brain damage. Journal of Nervous and Mental Diseases, 1963, 136, 344-351.

Tarachow, S. Circuses and psychoanalysis and the social sciences. New York: International Universities Press, 1951.

Taterka, J., and Katz, J. Study of correlations between electroencephalographic and psychological patterns in emotionally disturbed children. Psychosomatic Medicine, 1955, 17, 62-72.

Teuber, H. L. Effects of brain wounds implicating right or left hemisphere in man: Hemisphere differences and hemisphere interaction in vision, audition, and somesthesis. In V. B. Mountcastle (Ed.) Baltimore, Maryland: Johns Hopkins Press, 1962.

Thelander, H. E., Phelps, J. K., and Kirk, E. W. Learning disabilities associated with lesser brain damage. Journal of Pediatrics, 1958, 53, 405-409.

Tizard, B. Observations of overactive imbecile children in controlled and uncontrolled environments; I. Classroom studies. American Journal of Mental Deficiency, 1968, 32, 540-546.

Touwen, B. C., and Prechtl, H. F. The neurological examination of the child with minor nervous dysfunction (Clinics in developmental medicine; Number 38). London: William Heinemann Medical Books, Ltd., 1970.

Tymchuck, A. J., and Knights, R. M. A two-thousand item bibliography: The description, etiology, diagnosis, and treatment of children with learning disabilities or brain damage. Ottawa, Ontario: Carleton University, 1969.

Tymchuck, A. J., Knights, R. M., and Hinton, G. G. The behavioral significance of differing EEG abnormalities in children with learning and/or behavior problems. Journal of Learning Disabilities, 1969, 3:1, 547-551.

Ullmann, L., and Krasner, L. A psychological approach to abnormal behavior. Englewood Cliffs, New Jersey: Prentice Hall, 1969.

Vanderark, G. D., and Kempe, L. G. A primer of electroencephalography. Hoffmann-LaRoche, Inc., 1970.

Van Riper, C. Speech correction: Principles and methods. Englewood Cliffs, New Jersey: Prentice Hall, 1963.

Warren, R., Karduck, W., Bissorated, S., Stewart, M., and Sly, W. The hyperkinetic child syndrome. Archives of General Psychiatry, 1971, 24:2, 161-162.

Weiskopf, E. A. Intellectual malfunctioning and personality. Journal of Abnormal and Social Psychology, 1951, 46, 410-423.

Wepman, J. M. Auditory discrimination test. Chicago Language Research Associates, 1958.

Werry, J. The diagnosis, etiology, and treatment of hyperactivity in children. In J. Hellmuth (Ed.) Learning disorders. Seattle: Special Child Publications, 1970a.

Werry, J. Studies on the hyperactive child: IV. An empirical analysis of the minimal brain dysfunction syndrome. Archives of General Psychiatry, 1968, 19, 9-16.

Werry, J., and Sprague, R. Hyperactivity. In C. Costello (Ed.) Symptoms of psychopathology: A handbook. New York: John Wiley, 1970b.

Werner, H. Perceptual behavior of brain injured, mentally defective children: An experimental study by means of the Rorschach technique. Genetic Psychology Monographs, 1945, 31, 53-110.

Werner, E., Bierman, J. M., French, F. E. Reproductive and environmental casualties: A report on the ten-year follow-up of the children of the Kauai preganacy study. Pediatrics, 1968, 42, 112-127.

Wiener, G. Psychological correlates of premature birth: A review. Journal of Mental and Nervous Disease, 1962, 134, 129-144

Wigglesworth, R. Minimal cerebral palsy. Cerebral Palsy Bulletin, 1961, 3, 293.

Wikler, A., Dixon, J. F., and Parker, J. B. Brain function in problem children and controls: Psychometric, neurological, and eletroencephalographic comparisons. American Journal of Psychiatry, 1970, 127:5, 635-645.

Whitehead, A. N. Science and the modern world. New York: Mentor, 1948

Windle, W. F., Hinman, E. H., and Bailey, P. Neurological and psychological deficits of asphyxia neonatorum. Springfield, Illinois: Charles C. Thomas, 1958.

Winick, U. Malnutrition and brain development. Journal of Pediatrics, 1969, 74, 667.

Work, H. H., Haldane, J. E. Cerebral dysfunction in children. American Journal of Disabled Children, 1966, 111, 573.

Wortis, H., Fr edman, A. The contributions of social environment to the development of premature children. American Journal of Orthopsychiatry, 1965, 35, 57-68.

Wortis, H., Braine, M., Cutler, R., and Fr edman, A. Deviant behavior in a two and one-half year old premature child. Child Development, 1964, 35, 871-879.

Wortis, H. J., Bardach, R., Cutler, R., Rue, R., and Freedman, A. Child rearing practices in a low socioeconomic group: The mothers of premature infants. Pediatrics, 1963, 32:2, 298-307.

Wortis, J. A note on the concept of the "brain-injured child." American Journal of Mental Deficiency, 1956, 61, 204-206.

White, M. A., and Charry, J. (Eds.) School disorder, intelligence, and social class. New York: Teacher's College Press; Columbia University, 1966.

Wunderlich, R. C. Learning disorders. Journal of the American Physical Therapy Association, 1967, 47:8, 700-708.

Yates, A. J. Psychological deficit. In P. R. Farnsworth (Ed.) Annual Review of Psychology, 1966, 17, 111-144.

Yates, A. J., and Costell, I. C. (Eds.) Symptoms of psychopathology. New York: Wiley, 1970, 320-335.

Zrull, J., et al. A comparison of diazapan, d-amphetamine, and placebo in the treatment of hyperkinetic syndrome in children. American Journal of Psychiatry, 1964, 121, 388-389.