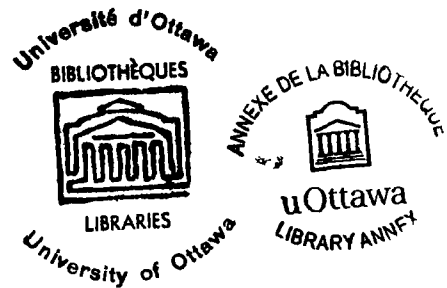


SKELETAL AND CENTRAL NERVOUS
SYSTEM MATURATION IN
THE MBD CHILD

by Lydia Makrides

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Studies as partial fulfillment of the
requirements for the degree of Master
of Science in Kinanthropology

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

1. Introduction.

Minimal Brain Dysfunction (MBD) or Learning Disabilities represent but two of the terms used to describe the child who exhibits various motor, behavioural and learning problems. The clinical phenomena to which such terms have been applied represent vaguely defined and imperfectly understood conditions which range from the markedly abnormal to subtle variations (Clemmens and Kenny, 1972)

MBD constitutes the single most common cause of chronic behavioural problems in the paediatric age group. It has been reported that about 10% of school-age children have learning difficulties which require special services outside the regular classroom (Abdou, 1970; Minskoff, 1970; Silver, 1971; Denhoff, 1972; Nissen, 1973). Other estimates range from 5% to 30% (Schain, 1970; Frischkecht, 1973; Becker, 1974). Colin, 1973, reported that 126,000 children in South African provincial schools were exhibiting various learning problems due to MBD. Recent UNESCO and WHO studies revealed a similarly high incidence of MBD in Great Britain, the Scandinavian countries and other Western European countries (Becker, 1974).

The high incidence of MBD in boys remains an enigmatic phenomenon. The ratio of boys to girls reported varies from four to one, to eight to one (Schain, 1973); Colin, 1973; Abdou, 1970).

There exists a general state of confusion in the literature regarding MBD. The primary reason stems from the fact that MBD is not a single condition but a multiple one. In other words, there is not one minimally brain dysfunctioned child, but many varieties of minimally brain dysfunctioned children. Adding to the problem is the fact that too many professionals with diverse backgrounds and interests

converge on the same area. This is not undesirable, provided that they all agree on the terminology. McCarthy (1973), stated:

None of us, regardless of professional orientation, knows precisely what he is dealing with! In the field of learning disabilities we are all groping together against a conglomerate elusive disability about which few generalizations can be made (p. 362).

Clemmens (1972), stated:

Atypical children have already taught us many things; they remind us constantly how much there is yet to be learned (p. 313).

The common concept prevailing throughout the literature is that of an involvement of the central nervous system. Denhoff (1972), reported that a large number of learning problems belong within the category of neuro-developmental disorder and that school failure is only one of the signs. The question to be investigated in the present study is whether the MBD child is in fact physically and neurologically ready to perform the tasks expected of him. In other words, is the MBD child's skeletal and neurologic age "on a par" with his chronologic age?

In the following two sections the literature pertaining to the classification and etiology of MBD is presented.

2. Classification

Educators have long been aware of children who experience difficulties in school or are functioning below their expected achievement level. These children were described as mentally retarded, autistic, emotionally disturbed, behaviourally maladjusted or aphasic (Lerner, 1971).

Strauss (1947), was the first to create a new category of exceptional children, known as brain injured children. It was theorized that such an impairment was due to an injury in the pre-natal stage, during birth or at some point after birth. The concept of brain injury however, met with criticism mainly due to the lack of neuropathological correlates between minimal clinical signs and minimal brain pathology (Cohn, 1964). Another objection often cited in the literature is that brain injury or brain damage is too general a term, of no diagnostic value and too alarming for the parents.

Stevens and Birch (1957), suggested the term Strauss Syndrome. The new label focussed on a collection of behavioural characteristics as originally introduced by Strauss (1947).

Brain damage seemed the most popular term used until 1966, when the Committee for Task Force I of the U.S. National Health and Welfare Department was established (Clements, 1966). The aims of this project were to define the problem, suggest nomenclature, identify the child and delineate the relationship of this problem to other handicaps. A total of 38 terms were used to describe the same problem (appendix A). The term proposed by the committee was minimal brain dysfunction, which is generally the term used by the medical specialists. Educators still prefer to use terms such as learning disabilities, hyperactivity or specific learning disabilities.

It is evident throughout the literature, that MBD refers to a host of signs and that one MBD child is not like another. Clements (1966), found many signs attributed to MBD (Appendix B). It was therefore suggested that the MBD child may exhibit minor signs in varying degree and combinations. These were listed as follows: (Clements, 1966).

1. Impairment of fine movement or coordination;
2. Electroencephalographic abnormalities without actual seizures which may be associated with fluctuations in behaviour or intellectual function;
3. Deviations in attention, activity level and impulse control;
4. Certain perceptual, intellectual and memory deficits;
5. Nonperipheral impairment of vision, hearing, haptics and speech.

Silver (1971) and Wender (1973) proposed essentially the same characteristics as the ones suggested by Clements (1966).

Schain (1970), concluded that speech disorders and hyperkinetic syndromes were the most common features in children who have learning problems. Page-E1 (1973), stated however that:

Children with Learning Disorders exhibit diverse characteristics and there is no consistent behavioural pattern which identifies the group in question. (p.599)

It is reported in the literature that so called soft neurologic signs are found in a large number of MBD children (Wender, 1972; Kohler, 1973; Gensener, 1973; McKeith, 1973; Thomas, 1973). It was stated that approximately half of all children with MBD have soft neurologic signs (Wender, 1973).

Hart, Rennick, Klinge and Schwartz (1974), compared 129 children experiencing school problems with 30 normal children, in order to determine if underachieving children were neurologically identifiable. It was concluded that the frequency of soft neurologic findings was greater in underachievers. Positive neurologic findings, however, were also found in the group of normal children. Thirty-five per cent of the underachieving group and six per cent of the control group exhibited soft neurologic signs. Based on these findings, the authors hypothesized that not all soft signs are indicative of neurologic impairment.

This hypothesis is also supported by other authors. Svoboda (1974), suggested that soft signs are manifestations of developmental delays, and that these immature signs would be normal in a younger child. Barlow (1974), also questioned the validity of soft signs and stated that..."there are no 'soft' signs only 'soft' neurologists." (p.605).

Perhaps the strongest opposition to the soft signs debate comes from Cohn (1964; 1974); Ingram, (1973); and MacKeith (1973). Cohn (1964), cautioned that soft signs cannot be equated with MBD, on the basis of three major considerations:

1. It is only by definition that certain clinical findings are described as "minimal".
2. It has not been demonstrated neuropathologically that minimal clinical signs are in fact related to MBD.
3. The ensemble of clinical signs is directly dependent on the variety of tests used by the neurologist and on the basic philosophy that underlies his thinking (p.p. 179-180).

In summary, it is evident that the question of classification of MBD children is far from being resolved. This is not surprising, if one considers the fact that there is no agreement on basic matters such as the terminology.

In this study, Clements's (1966), classification will be adopted, as it is the one generally accepted in the literature. Briefly, it consists of:

1. Impairment of fine movement or coordination.
2. Borderline EEG abnormalities.
3. Deviations in attention, activity level and impulse control.
4. Specific perceptual and intellectual deficits.
5. Nonperipheral impairments of vision, hearing, haptics and speech.

3. Etiology

The group of children described as having MBD exhibit deviations from the normal, which stem from various causes (Masland, 1974). The most common factors of MBD are presented in this section.

Clements (1966), reported a multiple causality, such as genetic variations, biochemical irregularities, perinatal brain insults, or other injuries and illnesses sustained during the years, which are critical for the development and maturation of the central nervous system.

The question of a delayed maturation of the central nervous system as a cause of MBD, has been raised by several researchers (Peters, 1974; McCarthy, 1973; Denhoff, 1973; Ingram, 1973; Svoboda, 1974; Satterfield, Lesser, Saul and Cantwell, 1970; Buchsbaum, Wender and Bethesda, 1973; Ames, 1968; Bateman, 1964). Some authors have attempted to make a fine distinction between developmental abnormalities and developmental delays. The point in question was whether there was an abnormality in the developing brain, or a lag in maturation (Critchley, 1968; Kinsbourne and Warrington, 1963; Paine, 1968; Wepman, 1963). No conclusive evidence seems to be available at the present time.

Ellingson (1967), questioned the assumption that a first grader has reached a stage of neurologic development that is comparable to his chronologic age. Ames (1968), reported that in most cases where difficulty in school was evident, the child's behaviour age was below his chronologic age; and thus, below the level of maturity required for successful school performance.

Other authors attempted to relate MBD with organic brain pathology. Towbin (1971), examined the relationship between delayed acquisition of language, clumsiness and hyperactivity with pathologic changes observed in brain autopsies of adults and neonatal cases.

It was concluded that the foetus and the newborn incur cerebral hypoxic lesions which result in MBD. Towbin's conclusions however, were highly criticized on the grounds that he attempted to relate cases with extensive brain lesions who displayed grossly disturbed language and behaviour to MBD. Cohn (1971), questioned whether these subjects were in fact diagnosed as having MBD.

Wender (1971), theorized that MBD may be the result of an abnormality in the metabolism of monoamines, which may affect the brain's level of arousal and reinforcement systems, thus producing hyperactivity and short attention span.

Kittler (1970), suggested that allergy may cause the findings observed in MBD. EEG changes and performance on psychometric tests were observed after subjects were placed on restricted diets. An overall improvement was observed, which led the author to suggest that the reason why some children's learning and behaviour is improved while taking amphetamines or tranquilizers, may be due to the fact that these drugs contain some antihistamine properties.

The role of psychologic factors in the etiology of MBD is an ambiguous one. Psychologic problems and learning difficulties are commonly found in association (Nissen, 1973; Walzer and Richmond, 1973; Bauer, 1974; Abdou, 1970). The question remains however, whether the psychologic disorders are a consequence of school failure or a cause of it. More consideration is given to the possibility that the psychologic disorders for many children are a consequence of school failure.

In summary, the possible causes of MBD reported in the literature were presented. Briefly these were:

1. Developmental delay of the maturation of the central nervous system;
2. Organic pathology of the brain;
3. Biochemical factors;
4. Psychological disorders.

The theory of a developmental delay of the central nervous system as a cause of MBD, has been primarily examined through indirect measures, i.e., behavioural evaluations (Ames, 1968), motor performance tests, questionnaires, and visual and auditory tests (Kinsbourne, 1973).

The present study is therefore undertaken in order to provide objective data on the neurologic as well as skeletal developmental levels of MBD children.

4. Rationale for the Study.

4.1. Myelination of the Central Nervous System and Neural Function.

MBD refers to a deviation or an impairment in some of the functions of the brain. It is well known that myelination is closely related to function of the central nervous system. According to Breckenridge and Murphy (1969), myelination is necessary for fibres to conduct nervous impulses sufficiently well to allow muscles to make delicate and precise movements. The mechanism by which myelin is formed is not completely understood, nor is the stimulus which leads to its formation.

A fibre system or a region of the central nervous system may begin to myelinate either early or late in foetal life or even only after birth, and it may attain the relative term of myelination either rapidly or slowly (Yakovlev and Lecours, 1964). Myelogenesis is an important parameter of regional maturation and one may reasonably assume that the development of myelin reflects the position of a fibre system in the hierarchy of functional organization of the developing nervous system. Different regions or fibre systems of the central nervous system mature at different times. Anokhin (1964), called this phenomenon heterochrony of development. An example of heterochronic maturation is that of the functional system of sucking. It was reported by Anokhin (1964), that in the newborn, a marked disproportion existed in the degree of maturation between the separate fibres of the facial nerve. It was found that the nerve fibres supplying the orbicularis oris myelinate and establish contact with the muscle fibres much before the rest of the nerve fibres supplying the other facial muscles.

Yakovlev and Lecours (1964), studied the myelination of regions and fibre systems in the brain stem and forebrain. The majority of the material consisted of cerebra ranging in age between the fourth foetal month and thirty years of age, with a few older

specimens. It was concluded that, in general, the fibres of the median thalamus, hypothalamus and the reticular formation of the brain stem continue to myelinate until at least the third decade of life.

Yakovlev (1962), reported that myelination of the cerebral cortex follows a developmental sequence from birth on, which parallels the successive phases of the organization of behavioural patterns from infancy to puberty. It was stated that myelination of the cerebral cortex continues throughout life to old age. The cerebral cortex thus exhibits the longest cycle of myelination (Yakovlev and Lecours, 1964).

The phylogenetic rate of development of the various parts of the central nervous system is different. In addition to this, ontogenetic differences exist (Breckenridge and Murphy, 1969). Kinsbourne (1973), reported that the findings associated with MBD indicate a delay in some aspect of neurologic maturation as a result of slowed development of cerebral function. Denckla (1974), suggested that delayed neurophysiologic connections between the two hemispheres of the brain may constitute one of the reasons why MBD children resemble younger, normal children.

The theory of a delayed maturation of the central nervous system in the MBD child gained support through the work of Satterfield, Lesser, Saul and Cantwell (1973). The study examined the auditory evoked cortical responses, clinical Electroencephalograms (EEG) and neurologic examinations of 31 MBD children and 21 controls. It was concluded that the most common clinical EEG abnormality found was an excessive amount of slow wave activity. This abnormality, like the increased latency of the evoked response, was reported to be consistent with delayed maturation of the nervous system, which was in turn consistent with the clinically observed immature behaviour of the MBD children.

Buchsbaum, Wender and Bethesda (1973), studied the visual and auditory average evoked responses in 24 MBD children and 24 age-

and-sex matched controls. It was concluded that MBD children showed average evoked responses characteristic of relatively younger normal children and that the data supported the concept of a neurophysiologic maturational lag in MBD children.

Kinsbourne (1973), stated that the findings upon which the diagnosis of MBD is made are abnormal only with reference to the child's age. It was also reported that all indicators of MBD represent the normal state of affairs in younger children.

Wikler, Dixon and Parker (1970), compared the EEG tracings of a group of children with scholastic-behavioural problems, with a group of normal controls. The findings indicated that the mean total slow percent activity (a single index of slow frequency brain waves), was significantly greater in the experimental group. Specifically, greater incidence of diffuse, rhythmic slow activity (4.0-7.9 Hz) and greater incidence of abnormal discharges were observed in the experimental group.

A physiologic retardation in bone age was reported by Oettinger (1974,1975). X-rays of the left wrist and hand were obtained from 53 MBD children for the determination of bone age, together with physical, neurologic and laboratory examinations. Two-thirds of the children in the MBD group were found to fall below the mean of the standard norms. Bone age for the MBD group was found to be significantly retarded compared with the standard group's norms.

The present study will investigate the skeletal and central nervous system maturation of MBD children in an effort to provide evidence either supporting or disproving the theory of a neuro-physiologic maturational lag in these children. If the data indicate that the MBD children are skeletally and neurologically behind their normal controls, several implications arise.

These are:

- 1) Further research will have to be undertaken to determine the cause of such physiologic delay.

2) The question of whether it is in fact a delay, or a more permanent impairment in the normal development of the central nervous system, will also have to be investigated.

3) A child's skeletal and neurologic age will have to be considered by the various professionals involved in child welfare and education.

In summary, direct evidence on the neurophysiologic status of MBD children is scarce. It is hypothesized that children diagnosed as having MBD may exhibit a maturational lag involving the skeletal and central nervous systems. Specifically, these children may not be physiologically ready to perform the tasks expected of them. It is therefore suggested that chronologic age alone may be too unreliable a criterion for placing a child in school or indeed for judging a child's abilities.

4.2. EEG as a Measure of the Developing Brain.

The EEG consists of continuous rhythmic oscillations of a spontaneous or autonomous character (Lindsley and Wicks, 1974). This rhythmic activity is probably maintained and regulated by sub-cortical pacemakers, namely nuclei in the thalamus, which provide the activating mechanism for the cerebral cortex (Lindsley and Wicker, 1974). The literature pertaining to the use of EEG as an indicator of central nervous system maturation is presented in this section.

During the first years of electroencephalography, much attention was focused towards the developmental EEG changes in childhood (Bernhard and Meyerson, 1968; Dreyfus-Brisac, 1966). Among the first papers published examining the relation between age and the EEG was by Berger in 1931 (Berger, 1969). Berger's observation that the alpha activity is slower in children than in adults, stimulated much interest in the progressive increase of the alpha frequency during childhood (Corbin, and Bickford, 1955; Brazier, 1972).

Lindsley (1936), studied the ontogeny of the brain's rhythms and established that although the EEG is present at birth, the alpha frequency recorded over the occipital areas is lower than in the adult EEG.

The frequency of the adult alpha activity lies between 8-13 Hz (Gibbs and Gibbs, 1964), and is the most dominant rhythmic activity recorded over the occipital region of the head. Based on extensive studies of normal children of different ages, Henry (1944), and Lindsley (1939), formulated a developmental curve showing the increase in alpha frequency during childhood to adolescence. Attempts were also made to find the mathematical expression for the alpha frequency as a function of age (Bernhard and Meyerson, 1968). It has also been shown that the developmental curve of the alpha frequency in man and monkey follow a strikingly similar pattern (Lindsley and Wicke, 1974; Bernhard and Meyerson, 1968).

Dreyfus-Brisac and Minkowski (1968), studied the EEGs of 96 newborns with low birth weight in order to determine the gestational age of these infants as indicated by the neurologic age obtained through the EEG. It was concluded that the EEG assessment of gestational age was correct in 76% of the cases and errors of more than 2 weeks were made in only 4% of the cases.

Lindsley and Wicke (1974), reported that the alpha rhythm seems to mark the beginning of functional activity in the cortex as well as integration with subcortical structures. This theory gained support from studies performed on anencephalic infants, who survived for 2 months. These subjects manifested the same reflex development as the normal infants of the same age, which suggests that the normal infant of 2 months has a cortex which is yet not functionally integrated with the rest of the brain, and only becomes so at the age of 3 to 4 months, when regular alpha activity occurs.

The tables published by Lindsley (1939), and Henry (1944), showed that beyond the age of 5 years, no occipital alpha waves with a frequency of less than 7 Hz can be expected, and at the age of 9 years no such waves with a frequency of less than 8 Hz can be expected in normal children. A mean frequency of 10 Hz was found by Lindsley (1939), at the age of 11 to 12 years and by Henry (1944), at 13 to 14 years.

The increase of the alpha frequency may not be continuous in the individual case, and sudden shifts may occur, which may be transitory or may persist for several years (Henry, 1944). The range of alpha frequency is wide among children (Straus, Ostow and Greenstein, 1952). Lindsley showed a variation of 6.0 to 9.2 Hz at the age of 4 years. Individual variabilities of 1.5 to 2.0 Hz have been reported in children by Henry (1944). Lindsley and Wicke (1974), reporting on the EEG of 4 adult women recorded at the same time for 32 days, found that under normal conditions the alpha frequency remains constant from day to day. It was stated that the standard deviation of the alpha frequency for any subject did not exceed 0.47 Hz.

Pond (1963), defined maturation as the diminution of the amplitude and spread of the slowest components of the EEG, with corresponding increases in the amplitude of the alpha frequencies.

The progressive changes of the EEG as a function of age have also been studied using auditory, visual or photic average evoked responses. Dustman and Beck (1968), studied the Visually Evoked Responses (VER) of 215 normal subjects between the ages of 1 month and 81 years. It was reported that the VER changed markedly with age. Ellingson (1960), found that the cortical evoked responses in the sensory areas of infant animals differ from those in mature animals of the same species. Similar developmental changes were also shown between the species studied (cat and rabbit). In an investigation involving the VERs of 693 infants, Ellingson (1960), concluded that developmental changes occur with increasing age.

In summary, the EEG has long been used as an indicator of the progressive maturational changes occurring in the developing brain. The primary use of EEG in MBD has been the investigation of clinical abnormalities, such as paroxysmal spikes or irregular rhythms. The EEG has met with criticism however as a diagnostic tool, due to two main reasons: the EEG does not always show abnormal tracings in MBD children; and some normal children may exhibit certain EEG abnormalities (Capute, Niedermeyer and Richardson, 1968; Page-El and Grossman, 1973; Hart, Rennick, Klinge and Schwartz, 1974).

Data are lacking in MBD children, with respect to the use of EEG as an indicator of neurologic maturation. The present study is undertaken in order to provide such data.

4.3. Skeletal age as a Measure of Maturation.

Children with MBD are often described as immature by teachers, parents and other professionals concerned with their developmental status (Ames, 1964; Kinsbourne, 1973; Oettinger, 1974). As the present study is undertaken in order to investigate the developmental status of MBD children, an assessment of skeletal maturation is a necessary parameter.

The radiographic study of the hand and wrist is one of the most useful procedures for determining the physical maturity of children (Greulich and Pyle, 1970; Pozsonyi and Zarfas, 1963; Tanner, 1961). There is considerable regularity in the order in which the carpals and epiphyses begin to ossify and in the order in which the epiphyses eventually fuse with the shafts. A single radiograph of a child's hand and wrist provides the following information: (Greulich and Pyle, 1970).

1. It offers an objective measure of the progress which the child has made towards attaining physical maturity;

2. It reveals imbalances in skeletal development and affords a comparison of a child's developmental status with that of others of the same age and sex.

Skeletal maturity is closely related to the development of the reproductive system (Tanner 1961; Greulich and Pyle, 1970). There is however, no conclusive evidence to date, with respect to the extent that skeletal maturity may correlate with measures of central nervous system maturation. Tanner (1961), suggested that the brain may share to a small extent in the general factor of skeletal maturation during the growing period, but conclusive evidence is as yet lacking.

Although a number of studies have been reported investigating the skeletal status of atypical children (Mosher and Yeates, 1973; Gothberg and Dayton, 1958; Pozsonyi and Zarfas, 1963), the only work available in the area of MBD is that of Oettinger (1974, 1975). A significant retardation in bone age was reported by Oettinger (1974;1975) in the MBD group as compared to the standard norms of Greulich and Pyle (1970).

It is hoped that this study will add to the present knowledge in the area of MBD, by investigating both the skeletal and neurologic maturation of these children as well as the relationship, if any, between these two maturational indicators.

5. Statement of the Problem.

The questions to be investigated in the present study are as follows:

1. Is the skeletal age of a group of MBD children significantly different from that of a group of normal children?
2. Is the alpha frequency of MBD children significantly different from that of a group of normal children?
3. Is the percentage of theta activity of MBD children significantly different from that of a group of normal children?

Two additional questions will also be explored. These are as follows:

1. Is there a significant positive relationship between skeletal age and alpha frequency?
2. Is there a significant negative relationship between skeletal age and percentage of theta activity?

6. Hypotheses.

It is hypothesized that:

1. The mean skeletal age of MBD children is significantly below that of a group of normal children.
2. The mean alpha frequency of MBD children is significantly below that of a group of normal children.
3. The mean percentage of theta activity of MBD children is significantly greater than that of a group of normal children.

7. Definition of Terms.

1. MBD refers to children of average or above average general intelligence, who experience certain learning or behavioural disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system (Clements, 1966).

2. Minimal dysfunctions of the central nervous system refer to borderline or subclinical deviations from the normal in perception, language, memory, conceptualization, control of attention, impulse or motor function (Clements, 1966).

3. Normal children are a group of children who were never diagnosed as having MBD.

4. Central nervous system maturation refers to the attained level of development of the central nervous system, as measured by the alpha frequency and the percentage of theta activity in the resting EEG.

5. Theta activity refers to the slow wave component of the EEG consisting of waves with a frequency of 4-7.9 Hz.

6. Alpha frequency refers to brain waves with a frequency of 8-13 Hz.

7. Skeletal age refers to the attained level of skeletal development of a child as indicated by a radiogram of the left wrist and hand.

8. Resting EEG refers to the EEG recording obtained with the subject sitting comfortable in an armchair, eyes closed, forearms supported and feet flat on the floor.

9. The criteria for the determination of hypoglycemia are as follows:

a) If the blood sugar level falls below 50mg% in any one of the seven readings taken during a five-hour glucose tolerance test.

b) If the blood sugar level falls faster than 60mg% per hour.

10. During a five-hour glucose tolerance test, a fasting blood sugar sample is obtained, followed by 100 mg of glucose drink. Blood samples are then obtained, every 30 minutes during the first hour, and every hour from then on. Thus seven readings of a subject's blood sugar level are obtained.

8. Limitations of the Study.

1. The power of the statistical tests was limited by the sample size.

2. Two channels of activity were recorded which do not necessarily represent total EEG activity.

CHAPTER II

MATERIALS AND METHODS

In the following section the subjects, apparatus and equipment, procedures and the method of data analysis are described.

1. Subjects.

Twenty male and female children diagnosed as having MBD using Clements (1966) criteria, between the ages of seven years and nine months and twelve years and six months, constituted the experimental group. These children were selected from patients under the care of two medical doctors. The control group consisted of 20 male and female children between the ages of eight years and six months and twelve years and five months. The controls were selected from children attending the summer day camp at the University of Ottawa, Ottawa, Ontario. The groups were equated on mean chronologic age and male-female ratio. This mean age was nine years and nine months and the male-female ratio was three to one.

Due to the fact that MBD is a general term referring to a heterogeneous group of children, certain criteria had to be met by each MBD subject. These were as follows:

1. Clinical diagnosis of MBD by a medical doctor.
2. No gross neurological signs such as seizures, paresis of a limb or spasticity.
3. No hypoglycemia as indicated by a five-hour glucose tolerance test.
4. No medications for at least five days prior to testing.

A typical MBD subject was of average or above average general intelligence as reflected by results of the Weschler Intelligence Scale for Children, which was part of the subject's medical record. He may have exhibited hyperactive behaviour, perceptual or cognitive anomalies, language difficulties oral and/or written and learning problems at school.

2. Apparatus and Equipment.

The apparatus and equipment used are described in this section.

2.1. EEG Equipment.

A Nihon Kohden 13 channel electroencephalograph, model ME-135D was used to obtain a two-channel recording of the right and left occipital and temporal lobes of the brain.

A Nihon Kohden EEG frequency analyzer, model MAF-5 was connected to the electroencephalograph, which provided a histogram of the delta, theta, alpha, β_1 , and β_2 frequency bands elicited in the raw EEG. Each histogram so produced, consisted of the integrated value of the amplitude, frequency and time that each frequency band occupied in the subject's raw EEG.

2.2. X-Ray Equipment.

A picker X-ray machine was used for the radiograms of the left wrist and hand.

3. Testing Procedures.

Two different sets of data were obtained from both groups. These consisted of a resting EEG and a radiogram of the left wrist and hand. Prior to testing, a consent form was signed by the parents of all children involved.

3.1 Resting EEG

The alpha frequency and the percentage of theta activity were the variables investigated in the EEG recording of each subject.

3.1.i. EEG Testing Procedure.

Prior to each recording, the electroencephalograph and frequency analyzer were calibrated. By pressing the buttons on the analyzer marked with the frequency bands α (alpha) and θ (theta), a histogram of the centre frequency of the band concerned was generated. The centre frequency of the alpha band was 10.2 Hz and of the theta band 5.65 Hz (Nihon Kohden frequency analyzer manual). The amplitude of the calibrated unit was 50 μ V, the time of each epoch was set at 5 secs., and the paper speed of the electroencephalograph was 1.5 cm/sec. This procedure was repeated at the end of each EEG recording.

One subject was tested at a time, while the next subject was allowed to watch the testing through a one-way mirror. This was done in order to reassure a child and to familiarize him with the testing situation.

Each subject was seated in an armchair with eyes closed, arms supported and feet flat on the floor. A brief explanation and necessary instructions were given to each child prior to electrode placement (Appendix C)

Once the hair was parted, two disc electrodes were placed over the occipital bones. An earlobe electrode was also placed on each ear. The electrode positions were O_1A_1 and O_2A_2 (international system).

The resistance of the skin was tested by an Ohm meter, such that maximum resistance was 5,000 Ohms. The occipital electrodes were held in position by a head band. Beckman Offner paste was used for better electrode contact with the scalp.

A one minute EEG tracing was obtained, prior to the test recording, at a paper speed of 1.5 cm/sec., in order to allow a subject to get comfortable and relaxed in the testing situation. A two-minute resting EEG was then obtained at a paper speed of 3 cm/sec.

3.1.ii EEG Evaluation.

All artifact-free epochs of the two minute EEG tracing from both channels were evaluated by visual-manual measurement. The mean number of epochs examined per record was 23.6. The method of evaluation of the two variables, i.e. alpha frequency and percentage of theta activity, is presented in the following section.

3.1.ii(a) Alpha Frequency.

The distance occupied by at least three or more successive alpha waves in each epoch was measured manually and the peaks were recorded. The distance was then divided by the paper speed of the electroencephalograph (3 cm/sec.), in order to convert distance into time. The number of peaks was then divided by the time, thus for each epoch the frequency of the alpha waves per second was obtained.

This procedure was repeated for each epoch from recordings of the right and left channels. Upon the completion of each record, the mean alpha frequency was calculated for the right and left hemispheres.

3.1.ii(b) Percentage of Theta Activity.

The height of the histograms of each artifact-free epoch

was measured manually. The total height of all right-sided histograms was then obtained and the percentage of theta activity for each epoch was calculated by dividing the height of the theta histogram by the total height of all histograms on the right side. This procedure was repeated for the recording of the left channel. Upon the completion of the record for each side, the mean percentage of theta activity for both sides was calculated.

3.2. Radiogram of Left Wrist and Hand.

Skeletal age was the variable investigated in the radiogram of each subject.

3.2.i. Testing Procedure.

A radiogram of the left wrist and hand was obtained at the Radiology Department of the Riverside Hospital, Ottawa, Ontario.

3.2.ii Radiogram Evaluation

All bones of the left wrist and hand, when present, were evaluated by two radiologists of the Riverside Hospital, and a University professor, department of Kinanthropology, University of Ottawa. The evaluations were made according to the standards of Greulich and Pyle (1971)

4. Data Analysis.

The methods of data analysis of the hypotheses under investigation are described in this section.

4.1. Hypotheses One, Two and Three.

1. The mean skeletal age of MBD children is significantly below that of a group of normal children.
2. The mean alpha frequency of MBD children is significantly below that of a group of normal children.
3. The mean percentage of theta activity of MBD children is significantly greater than that of a group of normal children.

The Student t-test for independent groups was used to determine if there were significant differences between the two groups in hypotheses one, two and three (Downie and Heath, 1965,1974)

4.2. Exploratory Questions One and Two

4. There is a positive relationship between skeletal age and alpha frequency.
5. There is a negative relationship between skeletal age and percentage of theta activity.

The Pearson product-moment correlation coefficient was used to determine the relationship in exploratory questions one and two (Downie and Heath,1965;Cooper, 1974).

CHAPTER III

RESULTS

The results pertaining to the hypotheses under investigation are presented in this section.

1. Hypothesis One.

The mean skeletal age of MBD children is significantly below that of a group of normal children.

The Student t-test for independent groups was used to determine if there are significant differences in the mean skeletal age of the MBD and control groups (Downie and Heath, 1965; Cooper, 1974).

Reliability of the X-ray interpretations was tested statistically by Pearson product-moment correlation coefficient (Downie and Heath, 1965; Cooper, 1974). The degree of inter-rater reliability was very high ($r = 0.90$). The results of the third reader¹ were used in the statistical analysis pertaining to the present study, as it was felt that his method of interpretation was more valid² compared to the clinically-oriented method of interpretation of the two radiologists³.

1. University professor, skilled in the interpretation of skeletal age radiograms, Department of Kinanthropology, University of Ottawa, Ottawa, Ontario.

2. Interpretation of skeletal age films by assigning a skeletal age to each bone in the wrist and hand, and subsequently calculating the mean skeletal age for each subject.

3. Interpretation of skeletal age films without assigning a skeletal age to each bone in the wrist and hand, but rather matching an X-ray film to a standard film (Greulich and Pyle, 1970), and subsequently using clinical judgement in assigning the skeletal age.

Table I presents the results of the Student t-test between the skeletal age of the MBD and control groups. No significant difference was found between the two groups.

TABLE I

Student t-test Between Skeletal Age of MBD and Control Groups.

Group	N	Mean Chrono- logical Age (in Months)	Mean Skeletal Age (in Months)	S.D.	t
MBD	20	119.7	116.7	17.94	-0.38
Control	20	119.3	114.4	19.53	

Critical value of $t = 2.02$ $\alpha = 0.05$, $ndf = 38$.

2. Hypothesis Two.

The mean alpha frequency of MBD children is significantly below that of a group of normal children.

The Student t-test for independent groups was used to determine if there are significant differences in the mean alpha frequency of the MBD and control groups.

Table II presents the results of the t-test between the mean alpha frequency of MBD and control subjects. A significant difference was found between the two groups, with the MBD children found to exhibit a lower mean alpha frequency than their control counterparts.

TABLE II
Student t-test Between Alpha Frequency of MBD and Control Groups.

Group	N	Mean Alpha Frequency (Hz)	S.D.	t
MBD	20	8.96	0.44	-6.15
Control	20	9.94	0.56	

Critical value of $t = 2.02$ $\alpha = 0.05$, $ndf = 38$.

3. Hypothesis Three.

The mean percentage of theta activity of MBD children is significantly greater than that of a group of normal children.

The Student t-test was used to determine if there are significant differences between the mean percentage of theta activity of the MBD and control groups.

Table III presents the results of the t-test between the percentage of theta activity in MBD and control groups. No significant difference was found between the two groups.

TABLE III

Student t-test Between Percentage of Theta in MBD and Control Groups.

Group	N	Mean Percent Theta	S.D.	t
MBD	20	28.01	5.36	-1.93
Control	20	24.83	5.06	

Critical value of $t = 2.02$, $\alpha = 0.05$, $ndf = 38$.

4. Exploratory Question One.

Is there a significant positive relationship between skeletal age and alpha frequency?

The Pearson product-moment correlation coefficient was used to determine if there is a significant relationship between skeletal age and alpha frequency (Downie and Heath, 1965; Cooper, 1974). Results indicated that there is no significant relationship between skeletal age and alpha frequency ($r = 0.12$, critical value of $r = 0.44$).

5. Exploratory Question Two.

Is there a significant negative relationship between skeletal age and percentage of theta activity? The Pearson product-moment correlation coefficient was used to determine if there is a significant relationship between skeletal age and percentage of theta activity (Downie and Heath, 1965; Cooper, 1974). Results indicated that there is no significant relationship between skeletal age and percentage of theta activity ($r = -0.40$, critical value of $r = 0.44$).

CHAPTER IV

DISCUSSION

The discussion of the results pertaining to the hypotheses under investigation, is presented in this section.

1. Hypothesis One.

The mean skeletal age of MBD children is significantly below that of a group of normal children.

The results of this study do not support the above hypothesis (Table 1). The only study relevant to this hypothesis, reported in the literature, is that by Oettinger (1974), who found significant retardation in bone age in a group of MBD children as compared to the standard norms of Greulich and Pyle (1964).

There are two main reasons why the results of this study may differ from Oettinger's findings. These are:

1. The interpretation of the skeletal age radiograms.
2. The difference in experimental design.

1.1. The Interpretation of the Skeletal Age Radiograms.

Oettinger (1974), reported that three physicians, two radiologists and an endocrinologist, read the films independently, and that inter-rater reliability was high ($r = 0.87$).

In the present study, three readers interpreted the x-ray films, two radiologists and a University professor skilled in reading skeletal age radiograms. The interrater reliability was high ($r = 0.90$). However, it was found that the two physicians tended to read the X-ray films on a lower scale than the non-physician reader.

It is therefore suggested that a difference in training, as well as a difference in the clinical approach to X-ray interpretation versus the research approach, may account for the different results obtained in the present study as compared to Oettinger's results.

1.2. The Difference in Experimental Design.

Contrary to the present study, Oettinger (1974), did not include a control group. The skeletal age of the MBD children used in Oettinger's study were compared to the standard norms of Greulich and Pyle (1964.)

Examination of the results obtained in the present study (Table I), indicated that the mean skeletal age of the control group was 4.8 months below their chronologic age. In fact, both the experimental and control groups were found to have a lower mean skeletal age as compared to the standard norms of Greulich and Pyle (1970). The mean chronologic age (both groups) was 119.5 months and the mean skeletal age (both groups) was found to be 115.5 months. Two questions therefore arise. These are:

1. Would there still be significant differences if a control group was used in Oettinger's study?

2. What are the implications, if any, in using the standards of Greulich and Pyle (1970) on populations other than the ones on which the standards were based?

In summary, the findings of the present study indicate that hypothesis one cannot be supported. The mean skeletal age of MBD children is not significantly below that of a group of normal children. The main reasons why these results differ from Oettinger's (1974) findings may be due to a difference in the method of skeletal age interpretation, and / or the lack of a control group in Oettinger's study.

2. Hypotheses Two and Three.

The results of hypotheses two and three are discussed together as they are both measures of central nervous system development.

2. The mean alpha frequency of MBD children is significantly below that of a group of normal children.

3. The mean percentage of theta activity of MBD children is significantly greater than that of a group of normal children.

The results of this study indicate that hypothesis two is supported. The mean alpha frequency of MBD children was found to be 8.96 Hz, while the mean alpha frequency of normal children was found to be 9.94 Hz (Table II). Therefore MBD children were found to have a significantly lower mean alpha frequency than normal controls. No other study has been reported comparing the alpha frequency of MBD and normal children.

The data pertaining to hypothesis three indicate that this hypothesis cannot be supported. The mean percentage of theta activity of MBD children was found to be 28.01% of the total EEG activity, while the mean percentage of the controls was found to be 24.83% of the total EEG activity (Table III). The fact remains however, that MBD children exhibited a greater percentage of theta activity than the controls, although the difference was not statistically significant ($t = 1.93$, critical value of $t = 2.02$, Table III). This may be due to the fact that delta⁴ activity, which constitutes the second component of slow wave activity in the EEG, was not included in the measurements.

Wikler, Dixon and Parker (1970), reported a significant difference between the experimental and control subjects in mean total slow percent (TS%), a single index of slow wave activity. This difference however, was due to the excessive amount of slow wave activity in the nonhyperactive experimental subgroup. Total slow

4. Brain waves with a frequency below 4Hz

percent activity of the hyperactive experimental subgroup was slightly greater than that of the controls, but the difference was not statistically significant. These findings therefore indicate that differences existed between hyperactive and nonhyperactive children. This distinction was not made in the selection of the sample for the present study. Examination of the childrens' medical records revealed that at least 12 out of the 20 MBD children were considered to be hyperactive.

A further point emerging from Wikler, Dixon and Parker's (1970) findings, was the fact that the hyperactive experimental subgroup was younger than the nonhyperactive experimental subgroup. Since slow wave activity in the EEG tends to decrease with age, the finding that the nonhyperactive, older children exhibited significantly more TS% than the hyperactive younger children, led the authors to suggest that some factor other than age differentiated the hyperactive from the non-hyperactive children with respect to TS%. The authors concluded that two distinct syndromes emerged from their findings. These were listed as follows: (Wikler, Dixon and Parker, 1970).

1. A syndrome characterized by hyperactivity, perceptual-motor deficits, soft neurologic signs and non-age-dependent slow wave activity.

2. A syndrome characterized by soft neurologic signs, and age-dependent slow wave activity, without hyperactivity or perceptual-motor deficits.

The authors based their conclusions as regards age or non-age-dependent slow wave activity on the correlation values obtained between the mean TS% and the mean chronologic age for each experimental subgroup. (Hyperactive subgroup: $r = -0.12$, nonhyperactive: $r = -0.87$, Wikler, Dixon and Parker, p.642) It can therefore be inferred from these findings, that the tendency for the slow wave activity of non-hyperactive children to decrease with age is greater than in hyperactive children. Conclusive evidence to this theory however, must

await the outcome of longitudinal studies on hyperactive and non-hyperactive MBD children.

The property of the central nervous system which determines its ability to learn is an independent dimension of higher neurologic activity. It is well known that the MBD child does not learn in the way that the normal child learns, despite adequate intelligence and adequate physical development, as indicated by the skeletal age data (Table I.) The findings pertaining to the two neurologic maturational indicators, namely alpha frequency and theta activity in the EEG, suggest that the MBD child is neurologically different from the normal child.

There is general agreement in the literature that the MBD child shows more slow wave activity than the normal child (Satterfield, Lesser, Saul and Cantwell, 1973; Buchsbaum, Wender and Bethesda, 1973; Wikler, Dixon and Parker, 1970; Nebylitsyn, 1972). However data on the alpha frequency of these children are lacking. As this study has shown, the MBD child exhibits a slower alpha frequency than the normal child (Table II). This finding is not unexpected if one considers the fact that as the frequency of the brain's electrical activity increases with age, more alpha will be generated and less theta or delta frequencies (Berger, 1969; Bernhard and Meyerson, 1968; Dreyfus-Brisac, 1966; Lindsley, 1936; 1939; Henry 1944). The relationship between the alpha and other frequencies in the EEG has been investigated by Nebilytsin (1972), who found that the frequency of the alpha activity correlated negatively with the frequencies of all other rhythms.

The questions pertaining to the relationship, if any, between skeletal age and measures of central nervous system maturation, are discussed in the following section.

2.1. Exploratory Question One.

1. Is there a significant positive relationship between skeletal age and alpha frequency?

The results indicate that a low positive correlation exists between skeletal age and alpha frequency for all subjects ($N = 40$, $r = 0.12$). When the skeletal age and alpha frequency was correlated for each group separately, results indicated a low positive correlation for the MBD group ($N = 20$, $r = 0.18$) and a somewhat higher correlation for the control group ($N=20$, $r = 0.38$).

The overall low positive correlations found, seem to suggest that the skeletal and central nervous systems do not necessarily mature together. The positive sign of the correlation indicates that as the skeletal system matures the alpha frequency also increases. The correlation values for each group (MBD: $r= 0.18$, control: $r = 0.38$) also suggest that there is more tendency for the normal children's alpha frequency to increase as they develop physically than there is within the MBD children.

However, the generally low positive correlation obtained between skeletal age and alpha frequency ($r = 0.18$) appears to indicate that the two systems mature at different times and possibly at different rates. Since no significant difference was found between the mean skeletal age of the MBD and control groups and a significant difference was found between the mean alpha frequency of the two groups, it follows that an adequately developing skeleton does not necessarily suggest an adequately developing central nervous system.

The conclusive answer to this question however, must await the outcome of further research on a homogeneous, larger sample of normal children.

2.2. Exploratory Question Two.

2. Is there a significant negative relationship between skeletal age and percentage of theta activity?

The results indicate that a negative correlation exists between skeletal age and percentage of theta activity for all subjects ($N = 40$, $r = -0.40$). When the skeletal age and percentage of theta activity was correlated for each group separately, similar values were obtained (MBD: $r = -0.51$, control: $r = -.50$). Although the overall correlation value did not meet statistical significance, these findings seem to suggest that theta activity correlates more with skeletal age than does alpha frequency. This theory, if indeed true, further suggests that different factors may govern the development of different subsystems within the central nervous system. It is possible that different factors may operate in both the reduction of slow wave activity and the increase in alpha frequency of the brain's bioelectrical activity. This concept is in accordance with Anokhin's (1964) theory of the heterochronic maturation of the central nervous system discussed earlier (Chapter I, p.12).

In summary, the findings of the present study suggest the following:

1. The skeletal age of the MBD child is not different from the skeletal age of the normal child.
2. The MBD child is neurologically different from the normal child, in that he exhibits a slower alpha frequency and a greater percentage of theta activity.
3. The maturation of the skeletal and central nervous system may be governed by different factors. In addition, the two systems may mature at different times and possibly at different rates.
4. Theta activity in the EEG may correlate more with skeletal age than does alpha frequency. This finding further supports the theory of heterochronic maturation of the central nervous system (Anokhin, 1964; Yakovlev, 1962; Yakovlev and Lecours, 1964).

CHAPTER V

SUMMARY AND CONCLUSIONS

The present study was undertaken in order to investigate the theory of a neurophysiologic maturational lag in MBD children. Three hypotheses were investigated. These are as follows:

1. The mean skeletal age of MBD children is significantly below that of a group of normal children.
2. The mean alpha frequency of MBD children is significantly below that of a group of normal children.
3. The mean percentage of theta activity of MBD children is significantly greater than that of a group of normal children.

Twenty male and female children diagnosed as having MBD, between the ages of seven and nine months, and 12 years and six months and 20 normal children between the ages of eight years and six months and 11 years and four months took part in this study. The groups were equated on mean age and male-female ratio. The mean age for both groups was nine years and nine months, and the male-female ratio was three to one.

In order to test the theory of a neurophysiologic maturational lag in MBD children, the following data were obtained from each child:

1. A radiogram of the left wrist and hand for the determination of skeletal age.
2. A resting EEG for the calculation of the alpha frequency and the percentage of theta activity in each child's EEG.

The student's t-test for independent groups was used to determine if significant differences existed between the two groups (Downie and Heath, 1965; Cooper, 1974).

It was found that:

1. No significant difference existed between the mean skeletal age of MBD and normal children (Table I). The mean skeletal age of the MBD group was 116.7 months, and the mean skeletal age of the control group was 114.4 months.

2. A significant difference existed between the mean alpha frequency of MBD and normal children (Table II). The mean alpha frequency of the MBD group was 8.96 Hz, while the mean alpha frequency of the control group was 9.94 Hz.

3. No significant difference existed between the mean percentage of theta activity of MBD and normal children (Table III). The mean percentage of theta activity of the MBD group was 28.01%, while the mean percentage of theta activity of the control group was 24.83%.

As a result of this investigation, and within the limitations of the present study, the following conclusions are drawn:

1. The physical maturation of the MBD child as determined by a skeletal age radiogram is not significantly different than that of the control children.

2. The MBD child is neurologically different from the normal child, in that he exhibits a significantly lower alpha frequency as indicated by a resting EEG.

3. The possibility of central nervous system immaturity should be considered by professionals involved in the education and social planning of the child with MBD. Longitudinal studies are indeed needed in order to determine whether the MBD child suffers from a neurodevelopmental delay or a more permanent neural abnormality.

In summary, the findings of the present investigation support the theory of a neurodevelopmental maturational lag in MBD children. Since learning is primarily a function of the central nervous system, it comes as no surprise to find these children neurologically different from normal children. Until such time, that more will be known about the factors influencing the normal development and maturation of the central nervous system, the MBD child will remain an enigma of medical, educational, and psychological scientific research.

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APPENDIX A

List of Terms Used to Describe MBD

(Clements, 1966, p.9)

Group 1 - Organic Aspects.

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

Group II - Segment or Consequence

Hyperkinetic Behaviour Syndrome
Character Impulse Disorder
Hyperkinetic Impulse Disorder
Aggressive Behaviour Disorder
Psychoneurological Learning Disorders
Hyperkinetic Syndrome
Dyslexia
Hyperexcitability Syndrome
Perceptual Cripple
Primary Reading Retardation
Specific Reading Disability
Clumsy Child Syndrome
Hypokinetic Syndrome
Perceptually Handicapped
Aphasoid Syndrome
Learning Disabilities
Conceptually Handicapped
Attention Disorder
Interjacent Child

APPENDIX B

List of Symptoms Attributed to Children With
MBD (Clements, 1966, p.11).

A. Test Performance Indicators

1. Spotty or patchy intellectual deficits.
Achievement low in some areas; high in other.
2. Below mental age level on drawing tests (man, house, etc.).
3. Geometric figure drawings poor for age and measured intelligence.
4. Poor performance on block design and marble board tests.
5. Poor showing on group tests (intelligence and achievement) and daily classroom examinations which require reading.
6. Characteristic subtest patterns on the Wechler Intelligence Scale for Children, including "scatter" within both Verbal and Performance Scales.

B. Impairments of Perception and Concept-Formation

1. Impaired discrimination of size.
2. Impaired discrimination of right-left and up-down.
3. Impaired tactile discriminations.
4. Poor spatial relations.
5. Impaired orientation in time.
6. Distorted concept of body image.
7. Impaired judgement of distance.
8. Impaired discrimination of figure - ground
9. Impaired discrimination of part - whole.
10. Frequent perceptual reversals in reading and in writing letters and numbers.
11. Poor perceptual integration. Child cannot fuse sensory impressions into meaningful entities.

C. Specific Neurologic Indicators.

1. Few if any, apparent gross abnormalities.
2. Many "soft", equivocal, or borderline findings.
3. Reflex assymetry frequent.
4. Frequency of mild visual or hearing impairments.
5. Strabismus.
6. Nystagmus.
7. High incidence of left, and mixed laterality
and confused perception of laterality.
8. Hyperkinesis.
9. Hypokinesis.
10. General Awkwardness.
11. Poor fine visual - motor co-ordination.

D. Disorders of Speech and Communication.

1. Impaired discrimination of auditory stimuli.
2. Various categories of aphasia.
3. Slow language development.
4. Frequent mild hearing loss.
5. Frequent mild speech irregularities.

E. Disorders of Motor Function.

1. Frequent athetoid, choreiform, tremulus, or
rigid movements of hands.
2. Frequent delayed motor milestones.
3. General clumsiness or awkwardness.
4. Frequent tics and grimaces.
5. Poor fine or gross visual - motor co-ordination.
6. Hyperactivity.
7. Hypoactivity.

F. Academic Achievement and Adjustment.

1. Reading disabilities.
2. Arithmetic disabilities.

3. Spelling disabilities.
4. Poor printing, writing or drawing ability.
5. Variability in performance from day to day or even hour to hour.
6. Poor ability to organize work.
7. Slowness in finishing work.
8. Frequent confusion about instructions, yet success with verbal tasks.

G. Disorders of Thinking Processes.

1. Poor ability for abstract reasoning.
2. Thinking generally concrete.
3. Difficulties in concept - formation.
4. Thinking frequently disorganized.
5. Poor short - term and long - term memory.
6. Thinking sometimes autistic.
7. Frequent thought perseveration.

H. Physical Characteristics.

1. Excessive drooling in the young child.
2. Thumb - sucking, nail - biting, head - banging, and teeth - grinding in the young child.
3. Food habits often peculiar.
4. Slow to toilet train.
5. Easy fatigability.
6. High frequency of enuresis.
7. Encopresis.

I. Emotional Characteristics.

1. Impulsive.
2. Explosive.
3. Poor emotional and impulse control.
4. Low tolerance for frustration.
5. Reckless and uninhibited; impulsive then remorseful.

J. Sleep Characteristics

1. Body or head rocking before falling asleep.
2. Irregular sleep patterns.
3. Excessive movement during sleep.
4. Sleep abnormally light or deep.
5. Resistance to naps and early bedtime, e.g.,
seems to require less sleep than average child.

K. Relationship Capacities.

1. Peer group relationships generally poor.
2. Overexcitable in normal play with other children.
3. Better adjustment when playmates limited to one or two.
4. Frequently poor judgement in social and interpersonal situations.
5. Socially bold and aggressive.
6. Inappropriate, unselective, and often excessive displays of affection.
7. Easy acceptance of others alternating with withdrawal and shyness.
8. Excessive need to touch, cling, and hold on to others.

L. Variations of Physical Development

1. Frequent lags in developmental milestones, e.g.,
motor language, etc.
2. Generalized maturational lag during early school years.
3. Physically immature.
4. Physical development normal or advanced for age.

M. Characteristics of Social Behaviour.

1. Social competence below average for age and
measured intelligence.
2. Behaviour often inappropriate for situation.
3. Possibly negative and aggressive to authority.
4. Possibly antisocial behaviour.

N. Variations of Personality.

1. Overly gullible and easily led by peers and older youngsters.
2. Frequent rage reactions and tantrums when caressed.
3. Very sensitive to others.
4. Excessive variation in mood and responsiveness from day to day and even hour to hour.
5. Poor adjustment to environmental changes.
6. Sweet and even tempered, co-operative and friendly (most commonly the so-called hypokinetic child).

O. Disorders of Attention and Concentration.

1. Short attention span for age.
2. Overly distractible for age.
3. Impaired concentration ability.
4. Motor or verbal perseveration.
5. Impaired ability to make decisions, particularly from many choices.

APPENDIX C

Explanation and Instructions Given to SubjectsPrior to EEG Recording.

"I will put these electrodes on your head (electrodes shown), so that your brain waves will be recorded on this paper (electroencephalograph shown). It only takes a few minutes so I would like you to sit back and relax. Close your eyes, think of something nice and keep as still as possible."

ABSTRACT

Twenty male and female children diagnosed as having minimal brain dysfunction (MBD), and 20 normal children, had a radiogram of the left wrist and hand taken for the determination of skeletal age. A resting electroencephalogram (EEG) was also obtained from all subjects, for the determination of alpha frequency (8-13 Hz) and percentage of theta activity (4-7.9 Hz). Both groups were equated on mean age (9 years and 9 months), and male-female ratio (3 to 1). No significant difference was found between the mean skeletal age of the two groups. However, a significant difference was found in mean alpha frequency, i.e. the MBD group exhibited a lower mean alpha frequency than the control group. In addition, the MBD children exhibited a greater mean percentage of theta activity than the normal children, although the difference was not statistically significant. These findings support the theory of a neurodevelopmental maturational lag in MBD children.