



## SPECIAL REPORT

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CHANGES OVER EIGHT MONTHS IN THE  
ELECTROENCEPHALOGRAMS OF NORMAL  
CHILDREN AND CHILDREN WITH MINIMAL  
CEREBRAL DYSFUNCTION

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CHANGES OVER EIGHT MONTHS IN THE ELECTROENCEPHALOGRAMS  
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B.D. MURDOCH

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## SUMMARY

EEGs of 35 children at a centre for remedial education and 35 age- and sex-matched normal controls at a private school were recorded on two eight-month separated occasions during which the minimal cerebral dysfunction (MCD) group received specialised remedial instruction and the control group normal classroom schooling. The EEGs of each group were compared both before and after the eight-month period, and in relation to the differences in changes that had taken place during the period. The results of previous investigations showing that children with MCD have a significantly higher incidence of EEG abnormalities of a slow wave type, were confirmed in the initial group comparison. Further, the MCD group differed from the control group on a number of measures related to alpha activity, and showed a significantly greater subcortical excitability. Right posterior EEG dysfunctions were implicated in a significantly higher proportion of the MCD group, and appeared to be related specifically to reading disability. Differences between control and experimental groups were reduced after the eight-month period between EEGs, suggesting that a process of normalization, related to either maturational changes, to the remedial instruction received, or both, was operative in the MCD children.

## OPSOMMING

EEGs van 35 kinders by 'n sentrum vir leergestremdes is vergelyk met dié van 35 geslag- en ouderdom-gepaardes wat 'n private skool bygewoon het (kontrole groep) op twee geleenthede met 'n tussen-periode van agt maande. Gedurende dié periode het die leer-gestremdes gespesialiseerde remediale en die kontrole groep normale klaskamer-onderrig ontvang. Die resultate van vorige ondersoeke wat gewys het dat leer-gestremdes 'n hoër voorkoms van EEG-afwykings wat stadige golwe betrek in vergelyking met normale kontrole, is ondersteun. Die leer-gestremde groep het ook van die kontrole groep ten opsigte van talle alfa-variante verskil en het ook 'n beduidend hoër mate van subkortikale opwekkendheid getoon. Die leer-gestremde groep het 'n hoër voorkoms van afwykings wat die regter agter-kortikale gebiede betrek, gehad, veral diegene met lees-moeilikhede. EEG-verskille tussen die groepe is verminder na die agt maande tussen opnames, en dit is moontlik dat die ontwikkelingsproses en/of die gespesialiseerde onderrig wat die leergestremdes ontvang het, daartoe bygedra het.

## INTRODUCTION

Aetiological, symptomatological, terminological, psychometric and remedial aspects of minimal cerebral dysfunction (MCD) are beyond the scope of this discussion and have besides been adequately considered by Clements<sup>1)</sup> (1966) and Johnson and Myklebust<sup>2)</sup> (1967), amongst others. This introduction will be concerned with attitudes governing and experimental approaches towards, utilization of the electroencephalogram (EEG) in the investigation of minimal cerebral dysfunction. The definition of minimal cerebral dysfunction formulated by representatives of the National Society for Crippled Children and Adults, and the National Institute of Neurological Diseases and Blindness of the U.S. Public Health Service would appear to have gained at least a measure of general acceptance, was applied in the present study and is as follows :

"The term 'minimal brain dysfunction syndrome' refers  
 ..... to children of near average, average or above  
 average general intelligence with certain learning or  
 behavioural 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, conceptualisation, language,  
 memory, and control of attention, impulse, or motor  
 function." (Clements<sup>3)</sup>, 1966, pp. 9 and 10)

Since use of the term 'minimal cerebral dysfunction' implies recognition of at least a functional cortical disturbance if not of an underlying organicity, use of the EEG would appear to have direct diagnostic significance. However most investigations have failed to reveal an EEG pattern specifically related to the MCD symptom constellation and the individual case presenting such a constellation

may have a normal EEG. The following authors have commented on the EEG in MCD : Clements and Peters<sup>4)</sup> (1962) and Paine<sup>5)</sup> (1965) suggest that the EEG of a child with MCD is borderline abnormal or abnormal, while Wilson<sup>6)</sup> (1970) states that, on examination, the EEG of such a child 'is found to be normal' (p. 45). Gaddes<sup>7)</sup> (1968) considers that the MCD child 'may have a normal EEG, or at most a dysrhythmia grade I (minimal)' (p. 49). Clements<sup>8)</sup> (1966) suggests that 'electroencephalographic abnormalities without actual seizures' (p. 10) characterise the child with MCD. Critchley<sup>9)</sup> (1966) proposes that the mild, non-specific and often asymmetrical dysrhythmias not uncommonly associated with MCD suggest a cerebral immaturity. Aron<sup>10)</sup> (1972) describes non-specific slowing, asymmetry or sharp waves in the EEGs of children with MCD, but feels that 'more frequently the EEG will be normal' (p. 149). Paine<sup>11)</sup> (1968), on the other hand, states that EEGs with minor abnormalities characterise MCD. The relationship between MCD and the EEG may, therefore, justifiably be described as less than distinct and as detracting from the diagnostic validity of the EEG in this context (Freeman<sup>12)</sup>, 1967; Di Leo<sup>13)</sup>, 1970). However, utilisation of the EEG as a research tool in the study of MCD would appear to require little justification (Paine<sup>14)</sup>, 1968) and relevant studies are considered below.

Paine<sup>15)</sup> (1962) recorded the EEGs of 17 children of a group of 41, 29 of whom had IQs greater than 80, test not specified. (It is not stated how many of the 17 had IQs less than 80, thus not displaying the 'near average, average or above average general intelligence' of Clements's<sup>16)</sup> (1966) definition.) The most characteristic presenting problems were poor school work or hyperactivity. Six of the 17 children displayed abnormalities in 'voltages or rates' and five cases spike or spike-and-wave discharges, although only three of these had had clinical seizures. A total of 15 of the 17 cases had abnormal or borderline abnormal EEGs.

Paine, Werry and Quay<sup>17)</sup> (1968) applied sophisticated statistical techniques in their analysis of the association between historical, neurological, electroencephalographic, psychometric and behavioural variables in MCD. Their subjects numbered 83 and all had IQs greater than 80 on either the Wechsler Intelligence Scale for Children (WISC) or Stanford-Binet Scale. They were referred for hyperactivity and non-achievement at school and were included in the study if they displayed any one of the following : abnormal neurological signs, an abnormal EEG, or psychometric patterns of the type seen in organic encephalopathies plus either excessive clumsiness or an abnormal EEG. Forty-one percent of the group had abnormal EEGs (degree of abnormality is not specified), the most frequent findings being irregular slowing and irregularity of background activity (each appearing in 70% of the abnormal group). Focal abnormalities were seen in 40% of those with abnormal EEGs, and 'seizure discharges' in one-third. The factor 'abnormal EEG' was correlated positively with a high neurotic score, with difficulties in abstraction, an irregular spread of scores on the WISC subtests and with impaired WISC performance IQ, but surprisingly not with any neurological-historical factors. Paine et al<sup>18)</sup> conclude that their results suggest that : 'minimal cerebral dysfunction is not a homogenous diagnostic entity, but rather a way of describing a variety of unrelated minor dysfunctions, some neurological, some behavioural and some cognitive, which may put a child in difficulties with his social and familial environment' (1968, p. 516).

Perhaps the most frequently quoted contribution to the literature on the EEG in MCD is that of Capute, Niedermeyer and Richardson<sup>19)</sup> (1968). Their group consisted of 106 children aged 2,3 to 16,3 years attending the JohnsHopkins Diagnostic and Evaluation Centre for Handicapped Children selected at random according to the following criteria : the presence of 'soft' neurological signs (use of this term is, incidently, criticised by Critchley<sup>20)</sup> (1966) ), symptomatology (hyperkinesis and learning disorders were most common), a significant degree of variability of intelligence subtest scores and a full-scale IQ equivalent score greater than 79. The Stanford-Binet and Hiskey

scales were used for children aged two to five years and the WISC and Stanford-Binet for those five years and older. A control group of 33 'physically and mentally healthy' children aged three to 15 years was used for comparative purposes. Fifty-three (50%) of the MCD group had abnormal EEGs (45 slightly to moderately and eight markedly abnormal). The most common, mild abnormality was excessive bilateral posterior slowing. Non-specific abnormalities were seen in 24 of the MCD group, and paroxysmal disturbances in 14. The incidence of focal abnormalities was low, only nine of the total of 53 showing these features. Twenty-eight children in the control group had normal, and five (15%) slightly abnormal EEGs. The difference in incidence of control and MCD abnormalities was statistically significant ( $p < 0,01$ ). It was postulated that the EEG abnormalities in the MCD group indicate a cerebral dysfunction which may have its basis in morphology or altered physiology, and further that the MCD cases who presented normal EEGs may have had similar dysfunctions, the electrical activity of which was masked by that of other structures.

Mišurec and Vrzal<sup>21)</sup> (1969) recorded EEGs from 43 children with MCD aged six to 14 years. Their criteria was the 'association of hyperkinesia, specific learning, perceptual motor and general motor deficits, impulsiveness and emotional lability'. This group was compared with 29 children 'with a partial symptomatology of this syndrome' and 109 age-matched controls. Abnormal EEGs were obtained from 65,2% of the MCD group, 31,1% of the group with the partial syndrome and 10,1% of the controls. Group differences reached statistical significance.

Quitkin and Klein's<sup>22)</sup> (1969) study approached the question of MCD and the EEG from a novel angle. They divided 105 adolescent and young adult (aged under 25 years) voluntary psychiatric patients into four groups on the basis of present symptomatology and clinical psychiatric history: a group with no 'soft' behavioural signs; a borderline group; an impulsive-destructive group; and a socially awkward-withdrawn group. The latter group exhibited egocentricity,

rigidity, lack of perseverance and difficulty in comprehending complex but routine verbal instructions, amongst other symptoms, and it was proposed by the authors that a definite organicity underlies this complex. The impulsive-destructive group, presenting largely emotionally unstable character disorders, was considered to present a functional, but not organic, dysfunction. Broadly speaking, the former group approximates to an MCD sample with a predominantly learning disability, the latter to one with a hyperkinetic behaviour disorder. Subjects in all four groups had Wechsler Adult Intelligence Scale (WAIS) full-scale scores greater than 90. EEGs were recorded from 102 subjects. Seventy-six percent of the 'no soft signs' group, 56% of the borderline group, 53% of the impulsive-destructive group and 50% of the socially awkward-withdrawn group had normal EEGs. 'Probably abnormal' and 'definitely abnormal' EEGs were present in 24% of the 'no soft signs' group, 44% of the borderline group, 47% of the impulsive-destructive group and 50% of the socially awkward-withdrawn patients. This was regarded as supporting the hypothesis that patients in the socially awkward-withdrawn and impulsive-destructive groups have different types of neurologic dysfunction, each producing a relatively specific clinical syndrome.

Kenny and Clemmens<sup>23)</sup> (1971) studied the EEGs of 88 children referred by outside sources with a provisional diagnosis of MCD, of whom only 14 were finally diagnosed as exhibiting the MCD syndrome in terms of a definition very similar to that of Clements<sup>24)</sup> (1966). Seven of the 14 had abnormal EEGs. No further details relating specifically to this group were supplied. This study is reported as an example of the hazards inherent in relying on a diagnosis of MCD by a number of different sources (in this case teachers, principals, social workers, psychologists and medical sources), and confirms the previous observations of the present author (Murdoch<sup>25)</sup>, 1971) in this respect.

The studies presented up to this point have considered MCD mainly in children displaying behavioural hyperkinesis, sensory-motor impairment and learning disorders in general. However, a number of investigations have been concerned with the relationship between the EEG and specific learning disabilities, for example, in the fields of reading, spelling and arithmetic function. Some of these will be considered.

Tuller and Eames<sup>26)</sup> (1966) studied the EEGs of seven children aged eight to nine years who rated below the 25th percentile of the Detroit Reading Test II, Form B, in comparison with local school norms. No IQ scores were given, but the children were 'free from symptoms or signs of mental retardation and of normal dexterity and alertness'. All subjects had abnormal EEGs in comparison with normative data for the age group, and although no consistent EEG abnormality characterised the group as a whole, the parieto-post-temporal areas were involved in all cases. The authors suggest that their results indicate that investigation utilizing the bipolar EEG recording technique instead, as is common practice in the USA, of the monopolar technique, would allow more precise localization of the area involved in reading. Abnormalities in this area would then differentiate poor readers and allow early remedial measures to be applied. They speculate that Brodmann area 39 is most likely to be involved in the reading process. This is located roughly at the intersection of the mid-temporal gyrus and angular gyrus (Herrick<sup>27)</sup>, 1924) in the parieto-post-temporal region.

Ingram<sup>28)</sup> (1970) selected 82 children aged seven years or more, all showing a greater than two-year retardation in reading-age (measured on the Schonell Graded Word Test) in relation to chronological age. None were below average intelligence on the Stanford-Binet Intelligence scale. Subjects were further divided into two groups : the 'specifics' - 62 children who showed specific

reading and spelling disability only; and the 'generals' - 20 children who displayed a general learning disability reflected in reading, spelling and arithmetic failure. Comparison of 'generals' and 'specifics' in terms of history, neurological status and EEGs suggested that the latter group had less evidence of brain damage or dysfunction than the 'generals', with a statistically significant difference in incidence of normal EEGs of 66% in the 'specifics' and only 16% in the 'generals'.

In contrast to the results of Tuller and Eames<sup>29)</sup> (1966) and to a lesser extent, those of Ingram<sup>30)</sup> (1970), Hartlage and Green<sup>31)</sup> (1971) found no difference in reading, spelling and arithmetic scores on the Wide Range Achievement Test for children separated into four groups, one with normal EEGs (N = 30), one with left hemisphere abnormalities (N = 13), a third with EEG abnormalities involving the right hemisphere (N = 14) and a fourth with diffuse EEG abnormalities (N = 57). The children ranged in age from six to 16 years, but it is not stated whether all had IQs greater than 80. The authors suggest that their negative results may be attributed to the inability of the EEG to detect circumscribed areas of dysfunction, and may partly be attributed to probable use of the monopolar recording technique.

A study which does not fall readily into either of the two main groups considered (i.e. those relating EEG variables to MCD in general, and those considering the relationship between the EEG and reading disability in particular) but which has special relevance for this investigation, that of Behrens<sup>32)</sup> (1963), will be discussed separately. This is the only one to this author's knowledge, which specifically considers change in EEG and psychological correlates of MCD during the period of remedial education.

Behrens<sup>33)</sup> (1963) recorded the EEGs of 30 of 41 children in his study, who had been selected according to the following criteria : all were aged between 5,6 and 14,8 years when the EEG was first recorded and

between 6,3 and 15,8 years on the second occasion of EEG recording. All demonstrated average or better intelligence (i.e.  $IQ > 90$ ) on either the verbal or performance scale of the WISC; and all subjects exhibited learning disorders which were not due primarily to emotional disturbance (this variable was rated independently by three trained observers). All subjects were selected from the files of the Institute for Language Disorders, Northwestern University, and displayed evidence of a neurogenic learning disorder. The length of time between examinations ranged from six to 24 months, and the period of remedial education during this time ranged from 20 to 300 hours. A tendency toward normalization of the EEG was noted when first and second EEGs were compared, the number of abnormal EEGs decreasing from 24 (80%) to 20 (66,7%). When individual EEGs were compared on a test-retest basis, 55,6% showed some improvement, 18,5% showed no change (that is, first and second EEGs had similar abnormalities), and 25,9% showed some deterioration. (These incidences are based on the cases remaining when the three normal EEGs on both occasions of testing were excluded.) More detailed analysis of EEG changes from first to second recordings showed that the latter tended to be less severely abnormal and their abnormalities more localized in the occipital areas than during the first EEG. On both occasions, the most common abnormality was bilateral 4-6 Hz activity. No relationship was found between type of EEG abnormality, pre- and perinatal history, or history of illness. Comparison of EEG trends with test-retest performance on the psychometric measures revealed the following trends : subjects with normal EEGs on both testing occasions performed at a superior level on the cognitive measures, but were inferior on the oral reading tests and measures of social maturity. The group showing EEG improvement maintained a consistent, moderate trend of improvement on most of the measures. The group showing deterioration in EEGs displayed, in several instances, the highest positive change on some measures, but their level of performance on both testing occasions was inferior to those of subjects in the other categories.

It is clear, then, that many of the opinions expressed by authors such as Wilson<sup>34)</sup> (1970) and Aron<sup>35)</sup> (1972), regarding the EEG and MCD, and quoted previously, depart from the findings of experimental investigations in this field, although many of the latter are themselves not free from deficiencies. Apart from obvious inadequacies in numbers of subjects with MCD (for example, in the studies of Paine<sup>36)</sup>, 1962; Kenny and Clemmens<sup>37)</sup>, 1971; Tuller and Eames<sup>38)</sup>, 1966), in number of control subjects (Capute, Niedermeyer and Richardson<sup>39)</sup>, 1968), or of definition of the MCD syndrome (Paine<sup>40)</sup>, 1962; Mišurec and Vrzal<sup>41)</sup>, 1969), most experimental approaches to MCD have failed to employ a control group.

Although some (e.g. Tuller and Eames<sup>42)</sup>, 1966) have compared EEG results obtained from MCD children with norms previously published, this is no substitute for comparison of EEG results obtained for control and experimental groups under standardized conditions in terms of the same criteria. Tuller and Eames<sup>43)</sup> (1966) have furthermore pointed out the need for additional studies of MCD using the bipolar technique of recording, and this appears to be confirmed by the results of Hartlage and Green<sup>44)</sup> (1971). Moreover the results of Behrens<sup>45)</sup> (1963) indicated that the EEG may reflect changes in electrocortical organisation during the period of remedial education applied to children with MCD. Finally, a previous study by this author (Murdoch<sup>46)</sup>, 1971) suggested the desirability of further investigation of the MCD syndrome in which a definition of this complex was applied uniformly and not, as in that study, by a number of outside sources (see also Kenny and Clemmens<sup>47)</sup>, 1971).

## AIMS

These considerations led to the initiation of an investigation with the following aims :

1. To compare the EEGs of children with MCD as defined by Clements<sup>48)</sup> (1966) with those of an age- and sex-matched control group with no behavioural manifestations of this syndrome.
2. To compare changes in the EEGs of children with MCD occurring during a period of remedial education with those taking place in a control group receiving normal schooling.

## SUBJECTS

The experimental group consisted of 35 children (23 males and 12 females) attending a centre for remedial education. All had been referred to the centre because of poor performance or specific learning problems at either nursery or primary school, (11 showed specific reading, writing or spelling problems, or combinations of these), and had been assessed as necessitating remedial education by teachers, occupational therapists and psychologists at the centre. All had full-scale IQs greater than 80 with the exception of one whose Verbal IQ could not be validly ascertained as he, an Italian-speaking immigrant child, could only understand and speak English with difficulty. His performance IQ, however, was 87, and for this reason and for the fact that his language problem possibly reflected a particularly potent learning disability (he had learned very little English in spite of spending four years at English-medium schools) he was included in the experimental group. The mean full-scale IQ for the group was  $101,559 \pm 11,978$  (excluding the child mentioned above) with a range of 80 to 126. Thirty-two subjects were tested on the South African

Individual Scale, and three on the Merrill-Palmer Scale. Subjects ranged in age from 6 years 7 months to 13 years (mean 9,703 years, S.D. 21,02 months) on the first occasion of EEG recording, and 7 years 4 months to 13 years 7 months (mean 10,412 years, S.D. 20,93 months) on the second occasion. Thirty-four of the subjects were right-handed, with the handedness of one male not ascertained.

The majority of subjects in the experimental group had been at the centre for between one and three years ( $N = 24$ ), with a few attending for only two months ( $N = 8$ ) and fewer for longer than three years ( $N = 3$ ), at the time the first EEG was recorded. Details of pre- and perinatal, and subsequent developmental histories were obtained from the centre for children in the experimental group where these were available. Prenatal histories were normal in 17 cases, perinatal in 12 and subsequent histories in four cases. Three children were reported to have had positive prenatal histories (a maternal fall at five months; and two 'difficult' pregnancies, one complicated by maternal hypertension, the other by numerous threatened miscarriages managed by hormone treatment). Perinatal histories were positive in 12 children (births three to five weeks premature, labours of excessive duration, cord strangulation, cyanosis in the child, foetal cardiac distress and post-natal incubation). A total of 23 children were reported to have had post-natal histories which were positive. Ten of these involved relatively serious illnesses or accidents (chorea, meningitis, hepatitis, pneumonia, rheumatic fever, dysentery and stemetol poisoning) and 13 the common childhood diseases. Details of pre- and perinatal and subsequent developmental histories were not available for the remaining experimental subjects.

The control group comprised 23 males and 12 females attending a private (i.e. non-state financed) primary school. All were of normal intelligence (although IQs were not measured for this project). None reported any difficulties in the classroom situation which might have suggested a learning disability. Control subjects were sex- and age-matched with the children in the experimental group. They ranged in age from 6 years 8 months to 12 years 11 months (mean 9,805 years,

S.D. 21,554 months) on the first occasion of EEG testing, and 7 years 4 months to 13 years 4 months (mean 10,471 years, S.D. 21,404 months) on the second occasion. Control and experimental group age differences did not approach the 5% confidence level for either the first ( $t = 0,045$ ;  $df\ 68$ ) or second ( $t = 0,141$ ;  $df\ 68$ ) occasion of EEG recording. Thirty-three control subjects were right-handed, and one (a male) left-handed. The handedness of the remaining case was not determined.

Control subjects were presumably not aware of, and were not asked for, details of pre- and perinatal histories, although one did mention spontaneously that her's had been a breech delivery. Eleven reported cuts, bumps and bruises involving the head, sometimes necessitating stitches. A further case reported trauma to the head leading to loss of consciousness. Three children in addition mentioned relatively serious illnesses (pneumonia, rheumatic fever and smallpox). The incidence of the common infectious diseases of childhood was not determined.

## PROCEDURE

### 1. EEG Recording

EEGs were recorded from children at the centre for remedial education on two occasions, separated by a period of eight months, and one year later from the control subjects at the private school, again on two eight-month-separated occasions. Recordings, employing the bipolar technique, were made on a portable Galileo E8b 8-channel electroencephalograph with five different bipolar montages of electrodes placed according to the ten-twenty system (Jasper<sup>49</sup>), 1958). Successful hyperventilation for three minutes was obtained in all cases, but photic stimulation

could not be applied to ten MCD subjects on the first, and five on the second occasion of testing because of technical difficulties. All control subjects received photic stimulation on both occasions of testing.

## 2. EEG Analysis

All recordings were analyzed by the same electroencephalographer by eye with the aid of a millimetre rule. Unfortunately, it proved impossible to wait until EEGs had been recorded from both groups for 'blind' analysis, as the recordings extended over a period of nearly two years when temporal allowance for school schedules had been made. Therefore the EEGs were analysed in two groups, firstly those from the experimental, and subsequently those from the control group. The electroencephalographer was thus aware of the group designation of each EEG and although he attempted as objective an analysis as possible, the possibility of unconscious bias in the interpretations cannot be excluded.

Control and experimental groups were compared, and group changes assessed, in terms of the following variables :

Alpha activity : mean alpha frequency (Hz), amplitude ( $\mu$ V) and index (rated on a five-point scale with zero indicating the absence of alpha activity and four continuous alpha activity); alpha organisation (a combined rating of frequency amplitude and incidence on a four point scale, zero indicating the very low amplitude and/or very low incidence and three a relatively unmodulated consistent rhythm, usually monorhythmic); the number of alpha components (that is, the number of frequency components within the alpha range of 8-13 Hz which were present); and the alpha frequency range (the difference between the highest

and lowest frequencies present in the 8-13 Hz band). Degree of abnormality of the resting EEG and during hyperventilation, degree of response to the latter and time taken to recover from its effects, were also rated.

In addition, the presence or absence of alphoid (that is, activity within the alpha frequency band but occurring in cortical areas not usually associated with alpha activity), theta (4-7 Hz), delta ( $\leq 4$  Hz), beta ( $> 13$  Hz), focal (activity occurring unilaterally in a specific cortical area), paroxysmal (sudden changes in ongoing activity greater than 50% of the mean preceding amplitude), sharp wave and spike (that is, sharp-tipped activity faster than 8 Hz), and spike-and-wave (sharp-tipped activity faster than 13 Hz associated with slow wave activity usually occurring at 3 Hz) activity were likewise assessed for each subject on each occasion of EEG recording. Next, the presence or absence of a response to the activation procedures of photic stimulation and hyperventilation was recorded for each subject for each variable apart from those relating to alpha activity, together with the presence or absence of harmonics and/or subharmonics during photic stimulation. If the EEG was abnormal, location of the dominant abnormality in terms of left or right hemisphere, frontal (i.e. anterior to the mid-line) posterior, bilateral or diffuse, was determined.

After the EEGs had been analysed as set out above, they were rated independently in terms of improvement, deterioration or 'no change' during the eight month interval between EEGs by two experienced electroencephalographers. A seven-point scale was employed, ranging from -3 (indicating marked deterioration) through zero (no change) to +3 (indicating marked improvement). The procedure was as follows. The two EEG recordings for each subject were bound in blank covers, with the birth date of the subject

and a serial number followed by 1 or 2. EEGs obtained on the first and second occasion of testing were randomly assigned the latter digits. This procedure ensured that the two electroencephalographers were unaware of the group designation of each subject, and further did not know which of the two EEGs had been recorded first. They were required to consistently rate EEG No. 2 as 'better' or 'worse' than, or no different from, No. 1 in terms of the scale previously detailed. After this had been done, ratings were corrected for the actual temporal sequence of recording employed (for example, if EEG No. 2 had been given a rating of -2 in relation to recording No. 1 and had in fact been recorded before No. 1, the rating was corrected to +2. This would mean that the EEG which had been recorded after the interval of eight months was the one which showed improvement. If the sequence of recording in fact corresponded to the numbers assigned then, of course, no correction was necessary). The ratings of the two electroencephalographers were then given group identifications and the group ratings compared on the two occasions of EEG recording. This was done as a check on the reliability of rating a variable as ephemeral as EEG improvement or deterioration.

### 3. Other Indices of Change

The teachers of children in the experimental (MCD) group were asked to fill in a questionnaire requiring details of changes in the child's behaviour, personality and emotional characteristics and scholastic performance, with possible causes of any changes observed, during the eight-month interval between EEG recordings. Teachers were also asked to rate the child's overall improvement or deterioration on an eleven point scale from -5 (marked deterioration or regression) through zero (no change) to +5 (marked improvement).

#### 4. Statistical Treatment

Measurements of alpha activity, degree of abnormality of the resting EEG and that during hyperventilation, and degree of response to, and time taken to recover from, hyperventilation were rated on continuous scales. The t test (Guilford<sup>50</sup>), 1956, p. 220) was therefore used to compare differences in experimental and control group means, both before and after the eight month period had elapsed. Differences between control and experimental groups in respect of changes over the eight month period were compared using the t test on difference scores. All other measurements were on discrete scales and group differences in incidence of each variable were compared on each occasion of EEG recording using chi-square in contingency table form (Guilford<sup>51</sup>), 1956, p. 230). Changes within groups in incidence of these variables over the eight month period were compared using McNemar's test for change in a 2 x 2 table (Harvard University<sup>52</sup>), 1955, p. xxxviii). Computations relating to the continuous scale variables were made by means of an IBM 360 computer, and those relating to variables scored discretely on a Hewlett-Packard 9100B desk computer. Group differences were considered significant if at the 5% level or less.

1. First EEG Recording

Tables 1, 2, 3 and 4 present the results of the analysis of data obtained during the first EEG recording for experimental and control groups.

TABLE 1

Incidence of normal and abnormal EEGs  
in control and experimental groups

	MCD	CONTROL	TOTAL
Normal and Mildly Abnormal	7	29	36
Moderately Abnormal and Severely Abnormal	28	6	34
TOTAL	35	35	70

$$\chi^2 = 27,68; p < 0,01$$

TABLE 2

Alpha activity, hyperventilation response and  
recovery, and abnormalities during rest and  
hyperventilation for MCD (N = 35) and control (N = 35) groups

Variable	MCD Group		Control Group		t
	Mean	S.D.	Mean	S.D.	
Mean Alpha Frequency (Hz)	9,407	1,198	9,507	0,905	0,388
Mean Alpha Amplitude ( $\mu$ V)	38,771	12,697	41,371	11,757	0,876
Alpha Index (0-4)	2,343	0,954	2,829	0,878	2,184*
Alpha Organisation (0-3)	1,286	0,436	1,657	0,444	3,484**
Alpha Frequency Range (0-5)	0,786	0,768	1,643	1,060	3,820**
Number of Alpha Components	1,943	0,860	2,571	0,728	3,252**
Response to Hyperventilation (0-3)	1,457	0,498	1,143	0,682	2,170*
Hyperventilation Recovery Time (seconds)	17,571	8,732	17,514	22,538	0,014
Rest Abnormality (0-3)	1,600	0,504	0,471	0,597	8,421**
Hyperventilation Abnormality (0-3)	1,271	0,453	0,657	0,773	4,001**

\*p &lt; 0,05

\*\*p &lt; 0,01

TABLE 3

Incidence of EEG variables during rest, photic stimulation  
and hyperventilation for control and experimental groups

CONDITION	VARIABLE	MCD GROUP		CONTROL GROUP		CHI-SQUARE
		Present	Absent	Present	Absent	
Rest	Alphoid	1	34	3	32	1,061
	Beta	5	30	5	30	0
	Theta	32	3	22	13	8,102**
	Delta	32	3	22	13	8,102**
	Focal	14	21	6	29	4,480*
	Paroxysmal	7	28	1	34	5,081*
	Sharp wave & spike	19	16	7	28	8,811**
	Spike & wave	1	34	0	35	1,015
	Asymmetry & Asynchrony	6	29	0	35	6,563*
Augmentation by photic stimulation <sup>1</sup>	Beta	1	24	0	35	1,424
	<7 Hz activity	9	16	5	30	3,844*
	Focal	4	21	1	34	3,298
	Paroxysmal	4	21	0	35	6,000*
	Sharp wave & spike	2	23	1	34	0,812
	Spike & wave	2	23	0	35	2,897
Augmentation by hyperventilation	Beta	1	34	7	28	5,081*
	<7 Hz activity	34	1	28	7	5,081*
	Focal	11	24	9	26	0,280
	Paroxysmal	10	25	6	29	1,296
	Sharp wave & spike	10	25	7	28	0,699
	Spike & wave	1	34	1	34	0
Photic stimulation <sup>1</sup>	Harmonics	0	25	2	33	1,478
	Subharmonics	6	19	1	34	6,326*
	Asymmetry & Asynchrony	4	21	3	32	0,781

\*p &lt; 0,05

\*\*p &lt; 0,01

<sup>1</sup>Technical problems prevented the administration of photic stimulation to 10 MCD subjects.

TABLE 4

Location of predominant abnormality  
in MCD and control groups

LOCATION	MCD	CONTROL
Right frontal	1	0
Right Posterior	15	3
Left Frontal	3	3
Left Posterior	3	6
Diffuse or bilateral	13	7
Total all abnormal	35	19

$\chi^2 = 7,739$ ; not significant

## 2. Second EEG Recording

Tables 5, 6, 7 and 8 represent the results obtained after analysis of data obtained on the second occasion of EEG recording eight months after the first, for experimental and control groups.

TABLE 5

Incidence of normal and abnormal EEGs in control and MCD groups

	MCD	CONTROL	TOTAL
Normal and Mildly Abnormal	10	31	41
Moderately Abnormal and Severely Abnormal	25	4	29
TOTAL	35	35	70

$$\chi^2 = 25,963; p < 0,01$$

TABLE 6

Alpha activity, hyperventilation response and recovery and abnormalities during rest and hyperventilation for MCD (N = 35) and control (N = 35) groups

VARIABLE	MCD Group		Control Group		t
	Mean	S.D.	Mean	S.D.	
Mean Alpha Frequency (Hz)	9,329	1,043	9,386	0,809	0,252
Mean Alpha Amplitude ( $\mu$ V)	38,857	9,862	41,429	12,454	0,944
Alpha Index (0-4)	2,400	0,868	2,857	0,833	2,215*
Alpha Organisation (0-3)	1,300	0,657	1,700	0,496	2,834**
Alpha Frequency Range (0-5)	0,771	0,647	1,229	0,669	2,863**
Number of Alpha Components	1,971	0,736	2,543	0,805	3,054**
Response to Hyperventilation (0-3)	1,414	0,579	1,229	0,614	1,284
Hyperventilation Recovery Time (secs)	15,943	10,068	16,286	11,361	0,132
Rest Abnormality (0-3)	1,214	0,613	0,429	0,536	5,626**
Hyperventilation Abnormality (0-3)	1,143	0,515	0,657	0,607	3,558**

\*p < 0,05

\*\*p < 0,01

TABLE 7

Incidence of EEG variables during rest, photic stimulation and hyperventilation for control and experimental groups

CONDITION	VARIABLE	MCD Group		Control Group		Chi-Square
		Present	Absent	Present	Absent	
Rest	Alphoid	2	33	0	35	2,059
	Beta	4	31	5	30	0,128
	Theta	29	6	25	10	1,296
	Delta	24	11	11	24	9,657**
	Focal	17	18	9	26	3,916*
	Paroxysmal	5	30	6	29	0,108
	Sharp wave & spike	18	17	9	26	4,884*
	Spike & wave	1	34	0	35	1,015
	Asymmetry & Asynchrony	3	32	0	35	3,134
	Beta	0	30	0	35	-
Augmentation by photic stimulation	< 7 Hz activity	2	28	2	33	0,025
	Focal	1	29	1	34	0,012
	Paroxysmal	1	29	1	34	0,012
	Sharp wave & spike	2	28	4	31	0,437
	Spike & wave	0	30	0	35	-
	Beta	1	34	3	32	1,061
Augmentation by hyperventilation	< 7 Hz activity	33	2	28	7	3,188
	Focal	13	22	10	25	0,583
	Paroxysmal	8	27	6	29	0,357
	Sharp wave & spike	18	17	13	22	1,448
	Spike & wave	1	34	0	35	1,015
Photic stimulation <sup>1</sup>	Harmonics	4	26	7	28	0,511
	Subharmonics	2	28	9	26	4,169*
	Asymmetry & Asynchrony	0	30	0	35	-

\*p < 0,05      \*\*p < 0,01

<sup>1</sup> Technical difficulties prevented the administration of photic stimulation to 5 MCD subjects.

TABLE 8

Location of predominant abnormality in MCD and control groups

LOCATION	MCD	CONTROL
Right Frontal	1	7
Right Posterior	18	3
Left Frontal	1	0
Left Posterior	3	5
Diffuse or bilateral	8	5
Total all abnormals	31	20

$$\chi^2 = 15,768; p < 0,01$$

### 3. Changes from first to second EEG recordings

Tables 9 and 10 show changes in EEG variables from first to second recordings for MCD and control groups.

TABLE 9

Differences in alpha and hyperventilation  
scores obtained on two eight-month separated  
occasions by MCD (N = 35) and control (N = 35) groups

VARIABLE	MCD GROUP		CONTROL GROUP		t
	Mean dif.	S.D. of diff.	Mean dif.	S.D. of dif.	
Mean Alpha Frequency (Hz)	-0,786	7,387	-1,214	9,380	0,209
Mean Alpha Amp. ( $\mu$ V)	0,086	12,427	0,057	13,086	0,009
Alpha Index (0-4)	0,057	1,170	0,029	1,028	0,107
Alpha Organisation (0-3)	0,014	0,722	0,043	0,498	0,190
Alpha Freq. Range (0-5)	-0,014	0,914	-0,414	1,045	1,680*
No. of Alpha Components	0,029	1,055	-0,029	0,941	0,236
Resp. to Hyp. (0-3)	-0,043	0,625	0,086	0,858	0,706
Hyp. Recovery Time (secs)	-1,629	13,111	-1,229	25,686	0,081
Rest Abnormality (0-3)	-0,385	0,599	-0,043	0,750	2,083*
Hyp. Abnormality (0-3)	-0,129	0,590	0	0,828	0,738

\* $p < 0,05$

In this table, a minus sign before a value indicates a decrease from first to second occasion of EEG testing.

TABLE 10

Changes in incidence of EEG variables during rest,  
photic stimulation and hyperventilation for  
experimental (MCD) and control groups.

CONDITION	VARIABLE	MCD Group			Control Group		
		A/P <sup>1</sup>	P/A <sup>2</sup>	p <sup>3</sup>	A/P <sup>1</sup>	P/A <sup>2</sup>	p <sup>3</sup>
Rest	Alphoid	2	1		0	3	
	Beta	1	2		1	1	
	Theta	0	3		5	2	
	Delta	1	9	<0,02	0	11	<0,01
	Focal	9	6		6	3	
	Paroxysmal	2	4		5	0	<0,05
	Sharp wave & spike	6	7		7	5	
	Spike and Wave	0	0		0	0	
	Asymmetry & Asynchrony	1	4		0	0	
Augmentation by photic stimulation	Beta	0	0		0	0	
	> 7Hz activity	0	7	<0,01	2	5	
	Focal	1	3		0	0	
	Paroxysmal	1	3		1	0	
	Sharp wave & spike	1	1		4	1	
	Spike and wave	0	1		0	0	
Augmentation by hyper- ventilation	Beta	1	1		1	5	
	> 7Hz activity	1	2		4	4	
	Focal	6	4		5	4	
	Paroxysmal	4	6		3	3	
	Sharp wave & spike	10	2	<0,05	6	3	
	Spike and wave	0	0		0	1	
Photic Stimulation	Harmonics	3	0		7	2	
	Subharmonics	0	5	<0,05	9	1	<0,02
	Asymmetry & Asynchrony	0	3		0	3	

<sup>1</sup>Indicates that the variable was not measured on the first occasion of EEG recording but was on the second.

<sup>2</sup>Indicates that the variable was measured during the first EEG recording but not on the second.

<sup>3</sup>From Harvard<sup>53</sup>), 1955, Table 6, p. 401.

#### 4. Coefficients of Correlation

Table 11 shows the inter-rater agreement for EEG and teacher ratings of change over the eight-month period between recordings.

TABLE 11

Inter-electroencephalographer and teacher-electroencephalographer agreement on ratings of change over an eight-month period

Group	Rating of Change	Correlation Coefficient	p	Common Variance
Experimental (N = 35)	EEGer A vs EEGer B	+0,432	<0,01	18,66%
	EEGer A vs Teacher	-0,315	NS	9,92%
	EEGer B vs Teacher	-0,115	NS	1,32%
Control (N = 35)	EEGer A vs EEGer B	+0,488	<0,01	23,81%

## DISCUSSION

### 1. First EEG Recording

MCD and control groups differed very significantly ( $p < 0,01$ ) in incidence of normal and questionable EEGs and those which were definitely and severely abnormal (Table 1), and MCD subjects had EEGs which were significantly more abnormal on average during rest than those in the control group (Table 2). While only 20% of the MCD group had EEGs which were normal or questionably so, 82,3% of the control group showed EEGs which fell into this category. Further, the average rest abnormality of MCD subjects was 1,6 and that of control subjects 0,47, both rated on a 0-3 point scale. These figures would appear to refute the claims of those such as Wilson<sup>54)</sup> (1970) and Aron<sup>55)</sup> (1972) who suggest that a normal EEG is characteristic of the child with MCD, and to support the experimental findings of investigators such as Behrens<sup>56)</sup> (1963), Capute et al.<sup>57)</sup> (1968), Mišurec and Vrzal<sup>58)</sup> (1969), Kenny and Clemmens<sup>59)</sup> (1971) and Tuller and Eames<sup>60)</sup>

(1966) who report abnormal EEGs in 80%, 53%, 65,2%, 50% and 100% respectively, of their subjects with MCD or reading disability. The differences in incidence of EEG abnormality, and the differences between them and the present results, may be attributed to differences in definition of MCD, to interpretative criteria applied to the EEGs and to age differences in the samples used, and should not be assigned, in the present author's view, much significance due to the oft-reported (e.g. Vizioli<sup>61</sup>), 1953; Torres and Blaw<sup>62</sup>), 1968; Fish<sup>63</sup>), 1971) difficulties in analyzing the EEGs of children, and in particular the dubious significance of slow activity in children's EEGs (Henry<sup>64</sup>), 1944; Petersén and Eeg-Olofsson<sup>65</sup>), 1971). These factors are also likely to be involved in the difference between the control group abnormality incidence obtained of 17,7% in this sample aged 6 years 7 months to 13 years, and Petersén and Eeg-Olofsson's<sup>66</sup>) (1971) incidence of 13,5% of 'slight' and 'moderate' increases in low frequency activity (these authors choose not to use the term 'abnormal' in categorising the EEGs of their subjects who were strictly selected for normality) in their group aged one to 15 years.

A more detailed comparison of the EEGs of MCD and control groups, which does not seem to have appeared previously in the literature, reveals interesting and statistically significant differences in respect of measures of alpha activity (Table 2). The control group showed a significantly higher index of alpha activity, a more organised alpha, a greater alpha frequency range and a greater number of frequency components in the 8-13 Hz band than the MCD group. Although it may be hazardous to speculate on the relationship between the picture presented of a less organised, less variable EEG, with a more restricted frequency range, reduced number of alpha components and lesser overall amount of alpha activity, and learning disability, it is of interest to point out that Walter as early as 1953 (Walter<sup>67</sup>), 1953), suggested a greater variation in the EEG

frequency spectrum among his 'more brilliant colleagues' than among his 'duller patients'. This observation was not subsequently upheld by Ellingson et al.<sup>68)</sup> (1957) in a group aged 19 to 40 years, but the present finding suggests that further investigation in a younger group may prove worthwhile.

The finding of a significantly greater, and more abnormal, response to hyperventilation in the MCD in comparison with the control group (Table 2) suggests that the EEGs of subjects with MCD may reflect a higher subcortical excitability than those of normal controls of the same age. This would appear to be confirmed by the significantly higher incidence of augmentation of activity slower than 7 Hz and of paroxysmal activity, the former by both photic stimulation and hyperventilation, the latter by photic stimulation alone, and by the significantly greater incidence of subharmonics during photic stimulation in the MCD in comparison with the control group (Table 3).

In common with the results of a number of previous investigators (Behrens<sup>69)</sup>, 1963; Paine et al.<sup>70)</sup>, 1968; Capute et al.<sup>71)</sup>, 1968) the most frequent EEG abnormality in the MCD group was slow wave activity, in this case both theta (4-7 Hz) and delta (<4Hz) activity during rest (Table 3). The incidence of these variables in the MCD group was significantly higher than in the control subjects. However, activity slower than 7 Hz occurred in 22 of the control subjects, and this probably represents the effects of a delayed electrocortical maturation related to the mean age (9,703 years) of this sample. This relatively high incidence of slow activity in a normal group is due to the inclusion of all subjects who showed more than very slight amounts of this activity in this category.

The following significant differences between MCD and control groups also emerged (Table 3). The former had a higher incidence of paroxysmal, sharp wave, spike and focal activity as well as asymmetry and asynchrony during rest, the latter a higher degree of beta augmentation during hyperventilation. The paroxysmal and sharp wave and spike activity in the MCD group suggests that latent epileptogenic disturbances may accompany at least some of the learning disabilities in this group. Similar EEG findings in respect of an MCD group have been reported by Paine<sup>72)</sup> (1962), Paine *et al.*<sup>73)</sup> (1968) and Capute *et al.*<sup>74)</sup> (1968). The higher incidence of resting asymmetry and asynchrony in the MCD group may provide further evidence of a less organised EEG pattern associated with this syndrome (already suggested by the findings relating to alpha activity). The higher incidence of focal EEG disturbances (indicated by the technique of phase reversal) in the experimental group suggested that further analysis of the location of the principal EEG abnormality in control and MCD groups might prove worthwhile. This was accordingly undertaken, and the results appear in Table 4. All cases displaying any degree of abnormality (including those of borderline significance) were included in this analysis. Although the overall trend of location of abnormalities for the groups did not differ significantly ( $\chi^2 = 7,739$ ;  $df\ 4$ ), a comparison of groups in respect of incidence of right posterior abnormalities reveals a significantly higher frequency in the MCD group ( $\chi^2 = 4,06$ ;  $p < 0,05$ ). As 11 of the 15 children with abnormalities in the right posterior quadrant were referred to the centre for remedial education specifically for reading difficulties, or had a reading age more than one year retarded in relation to chronological age, this may provide evidence for Tuller and Eames's<sup>75)</sup> (1966) contention that parieto-post-temporal dysfunction (in this case specifically in the right hemisphere) is associated with reading disability. Further support for this finding has been obtained in 16 children, referred for reading

disabilities, whose EEGs were analysed by the present author. Seven of the 16 had right posterior EEG dysfunctions, (Humphriss and Murdoch<sup>76</sup>), 1972).

The meaning of the difference between groups in incidence of augmentation of beta activity during hyperventilation is not clear. There may exist an association between this phenomenon and anxiety in the subjects displaying it (Walter<sup>77</sup>), 1963), and lack of the latter in the MCD subjects, but confirmation of this would require further investigation.

## 2. Second EEG Recording

Tables 5, 6 and 7 indicate that whereas significant group differences in incidence of EEG abnormality, in variables relating to alpha activity and hyperventilation response and in incidence of EEG variables during rest, photic stimulation and hyperventilation still exist, they are generally reduced, both in number and in size of difference, on the second occasion of EEG recording. Further no 'new' variables are added to the number of those that differentiate significantly between control and MCD groups on the second occasion of testing. This suggests that the MCD group tends to approximate more closely to the normal control group in terms of EEG variables over the period between EEG tests. This agrees with the conclusion arrived at by Behrens<sup>78</sup>) (1963), whose words may be applied directly to the present study : 'the changes in the EEG characteristics encompassed by this study indicate an apparent trend towards normalization' (p. 176).

It is interesting that while most EEG differences between MCD and control groups were reduced, those relating to location of EEG abnormality appeared to have been intensified (Table 8). Although the total number of subjects in the MCD group

displaying any EEG abnormality had declined from 35 to 31, the number of children whose abnormality was located principally in the right posterior quadrant had increased from fifteen to eighteen. The difference between MCD and control groups in incidence of right posterior abnormalities on the second occasion of testing is very significant ( $\chi^2 = 9,308$ ;  $p < 0,01$ ). While it is perhaps unrealistic to expect the EEG to reflect improvements related to remedial education methods, as were applied to the MCD group in the eight-month interval between EEG recordings, it is equally unrealistic to expect remedial techniques to increase the incidence of dysfunction in the area, which, according to Tuller and Eames<sup>79)</sup> (1966), is associated with the reading process in which most of the group showed deficiencies. This finding, then, would appear to adduce further evidence in support of Behrens's<sup>80)</sup> (1963) conclusion that 'no specific trend of relations between behavioural characteristics evidenced by psychological measurements and electroencephalographic pathologies can be observed at this time in children with neurogenic learning disorders' (p. 178).

### 3. Differences between first and second EEG recordings

The difference scores relating to alpha activity and hyperventilation variables show few significant group differences (Table 9). The MCD group did, however, show a significantly smaller drop in alpha frequency range and in rest abnormality than the control group. The former trend may be regarded, speculatively, as a maturational tendency to a monorhythmic adult alpha rhythm, the latter as adducing to the normalization process already commented upon. This evidence of EEG normalization lies in the significantly greater number of individuals in both groups in whom delta activity disappeared over the eight-month period than in those in whom it appeared (Table 10). The decline in number of individuals in whom photic stimulation augmented  $>7\text{Hz}$  activity is evidence in the same direction. The augmentation of sharp wave and spike activity by hyperventilation in a significantly greater number of MCD

individuals than those who showed the disappearance of this response, and the appearance of paroxysmal activity during rest in significantly more individuals than those in whom it disappeared in the control group must be regarded as against this trend, but are hardly typical of the general weight of evidence for both groups, and probably represent only minor deviations of the type described by Henry<sup>81)</sup> (1944). The significance of the conflicting experimental and control group trends in relation to changes in incidence of subharmonics during photic stimulation is not, at this stage, clear.

#### 4. Coefficients of Correlation

The low agreement between two independent raters of change in MCD and control EEGs over an eight-month period, and the associated common variances of only 18,7% and 23,8% respectively (Table 11) provide some support for the views of Freeman<sup>82)</sup> (1967) who expressed reservations regarding the reliability of the EEG, particularly in the field of remedial education. However, analysis of the EEGs of children is generally considered a difficult task, as has been pointed out previously, and, as the rating of EEG change is probably not the least difficult aspect of this task, the low inter-rater agreement is at least partly understandable. The very low, and negative, agreement between ratings of EEG change and teachers' ratings of behavioural, emotional and personality change over the eight-month period tends to support Behrens's<sup>83)</sup> (1963) previously quoted observations in this regard.

## 5. General

The tendencies towards normalization of the EEG apparent in both the present investigation and that of Behrens<sup>84)</sup> (1963) may be regarded as evidence for a functional rather than organic basis for the learning disabilities of at least a proportion of the MCD group. As such, the normal process of maturation, either singly or in association with the techniques of remedial education applied in the eight months between EEG recordings, might be expected to account for EEG normalization. However, a functional disturbance per se should not be considered to account for all learning disabilities, as Drake<sup>85)</sup> (1968) has produced post mortem evidence of bilateral anatomical anomalies of the parietal lobe in a child with marked reading and mild arithmetic disabilities. As Paine<sup>86)</sup> (1965) has in addition pointed out, such changes might be found more frequently in children with learning disabilities if more brains were available for examination, and their apparent absence may reflect the limitations of present neuropathological methods. It would appear, therefore, that the distinction between 'functional' and 'organic' may be somewhat artificial in the present context in spite of the evidence of Quitkin and Klein<sup>87)</sup> (1969) to the contrary, derived from an older sample.

It is of interest to point out that both control and experimental groups showed a decrease in mean alpha frequency from first to second occasion of EEG recording (Table 9) whereas an increase would have been expected in view of the results of Lindsley<sup>88)</sup> (1939), Petersén and Eeg-Olofsson<sup>89)</sup> (1971) and numerous others. This may suggest that an interval of eight months is not sufficient to reflect the operation of the maturational process in children of the age of these in this sample, and this should be a factor to be considered in further investigations of this nature. Errors of measurement and temporary deviations from ongoing maturational processes are, however, possible alternative explanations.

## CONCLUSIONS

1. This study confirms the results obtained previously by other investigators in respect of the association of a high incidence of EEG abnormality in children with MCD.
2. In addition, the MCD subjects differed significantly from the control group on numerous measures of alpha activity, and showed a significantly greater subcortical excitability than the control subjects.
3. Most marked EEG abnormality in the MCD group comprised theta and delta activity.
4. Right posterior dysfunctions were implicated in significantly more MCD subjects, particularly those with prior evidence of reading disability.
5. EEG differences between control and MCD groups were reduced after an eight-month interval during which the former received general classroom and the latter specialized remedial instruction, suggesting that a process of normalization was operative in the MCD group.
6. Behrens's<sup>90)</sup> (1963) observation of a low agreement between behavioural and EEG indices of change is upheld by the present results.

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