To the Graduate Council:

I am submitting herewith a dissertation written by Harry Stewart Shabsin entitled "Feedback Controlled Increases in P300 Visual Evoked Potential Amplitudes and Neurological Functioning and Academic Performance in Learning Disabled Children." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Joel F. Lubar, Major Professor

We have read this dissertation
and recommend its acceptance:

Accepted for the Council:

Vice Chancellor
Graduate Studies and Research

FEEDBACK CONTROLLED INCREASES IN P300 VISUAL EVOKED POTENTIAL AMPLITUDES AND NEUROLOGICAL FUNCTIONING AND ACADEMIC PERFORMANCE IN LEARNING DISABLED CHILDREN

A Dissertation

Presented for the

Doctor of Philosophy

Degree

The University of Tennessee, Knoxville

Harry Stewart Shabsin

August 1982

ACKNOWLEDGMENTS

This dissertation is dedicated to Jo Ann Murphey, a friend and colleague who gave a great deal of emotional support and encouragement, to my daughter Rhianna Elise whose birth during its progress provided a wonderful source of inspiration, and to Andrea Shabsin who put up with more than she should reasonably have expected to. It is also dedicated to my parents Edward and Dorothy Shabsin without whose assistance over the years it would not have been possible.

I would especially like to thank Dr. Joel F. Lubar, my chairman, Dr. Michael Moshell, and Dr. Schuyler W. Huck for their valuable time and advice. Two fellow graduate students, Dana DeBoskey and Corinne Bell, working on similar projects with learning disabled children also provided a great deal of assistance and support. A debt of thanks also goes to Kevin Bianchini, a graduate student in psychology, who gave many hours of his time helping collect data for this dissertation.

Finally, I would like to acknowledge Ann Lacava, Carl Wust, Wynn Brown, and Sandy Fletcher for their assistance in putting together this manuscript.

ABSTRACT

Results indicated that LD children can learn to control the amplitude of their VEPs. Analysis of variance found that the experimental children were able to significantly raise their baseline VEP amplitudes when provided with feedback. Cognitively, the effects of VEP amplitude increases in LD children were best seen in measures reflecting basic neurological functioning. Tukey's Honesty Significant Differences found LD experimental subjects to have significantly improved scores on the Halsted-Reitan Index for Children when compared to a group of normal children. VEP training appeared to affect the EEG primarily in the right parietal-occipital areas.

TABLE OF CONTENTS

CHAPTER PAGE

LIST OF TABLES

TABLE PAGE

LIST OF FIGURES

FIGURE PAGE

CHAPTER I

INTRODUCTION

From a clinical perspective the term "learning disabilities" refers to a wide variety of situations in which children and adults of normal intelligence fail to perform at their potential level in specific intellectual areas. Explanations accounting for such discrepancies have ranged over concepts based on learning theory, attentional processes, opthalmological observations, genetic disorders, emotional disturbances, perceptual processes, perceptual-motor integration, and cerebral function (Golden and Anderson, 1979). One of the earliest modern explanations for impaired intellectual performance was based on cerebral function. During the mid-nineteenth century Broca (1861) observed that linguistic difficulties were related to the dysfunctional activity in certain brain areas. Not long after, Morgan (1896) noted that dyslexia in brain-injured adults was similar to reading problems in children whom today would be considered to have a learning disability. Morgan described "word blindness," the inability to see words although lines, numbers and forms are easily recognized, in terms characteristic of a learning disability. Hinshelwood (1917) compared a number of congenitally word blind children to Monsieur C., a stroke victim, and concluded that the children may lack essential connections between visual and speech centers in the brain. Along similar lines Werner and Strauss (1939a, b, 1940) and Strauss and Werner (1942) described signs of behavioral

and visual perseveration, figure-ground confusion, inability to organize and deal with abstract figures and hyperactivity in children in terms of "exogenous" brain damage (neurological damage not due to genetic factors). These children were found to exhibit personality characteristics in which they appeared to be more erratic, impulsive, socially unacceptable, uncontrolled and uncoordinated. In later studies Strauss and Lehtinen (1947) associated these characteristics with hyperactivity.

In 1937 Orten published some of the findings from his work with children having difficulty acquiring language skills. He noted that many of these children tended to reverse letters, words, numbers and syllables and described this twisting of symbols as "strephosymbolia." Orten felt that these and other language problems were due to disturbances in cortical dominance development and proposed that these children be described in terms of specific dyslexia or specific reading disabilities since many of them exhibit reading problems. Other investigators employed different terms to describe the learning and behavioral problems seen in children we now label as learning disabled. Doll (1951) used the term "neurophrenia," Johnson (1962) "marginal children," Clements (1966) "minimal brain dysfunction" (MBD) and Chalfant and Scheffelin (1969) suggested "central processing dysfunction" to describe characteristics similar to brain injury but not always accompanied by evidence of brain injury. "Minimal brain damage" has also come into popular usage after a suggestion by Birch (1964) that brain damage implied too broad a range of pathology to properly describe learning disabled (LD) children.

In 1957 Stevens and Birch proposed using the term "Strauss syndrome" to describe the many behaviors and symptoms exhibited by children labeled as "brain injured" or "brain damaged." Their definition is based on seven specific symptoms including many of the concepts described by Strauss and Werner (1942). Finally, in 1963, at a conference on perceptually handicapped children, Kirk came up with the term "learning disabilities" and provided a definition which became the basis for the description of learning disabilities used by the National Advisory Committee on Handicapped Children (1967) and by Task Force II of the Minimal Brain Dysfunction National Project on Learning Disabilities (1969). Except for minor nuances, these two national committees have been taken as the core for descriptions of learning disabilities. As a result, five major points are almost universally present in any definition of learning disabilities (Hallahan and Kauffman, 1976, p. 20). Accordingly, the learning disabled child has: (1) academic retardation, (2) an uneven pattern of development, (3) possible central nervous system involvement (i.e. may or may not be present), (4) learning problems not due to environmental factors and (5) learning problems not due to mental retardation or emotional disturbances.

Presently the major effort in the diagnosis and remediation of learning disabilities occurs in the area of psychoeducational procedures. The field is described by Wohl (1980) as a broad spectrum of interrelated strategies incorporating information from studies in psychophysiology, perception, memory and cognition. In the area of identification and diagnosis of LD children the major tools are tests

measuring the general and specific aspects of intelligence (IQ) and the various abilities that make up IQ. Unfortunately, as Wepman, Cruickshank, Deutsch, Morenay and Strother (1975) state, there is little agreement in medicine or education (and one might add psychology) on criteria for identifying children with minimal brain dysfunction or learning disabilities. The problem arises from the vagueness of the concepts incorporated in the definitions of learning disabilities (Clements, 1966) and from the vast array of components comprising intelligence and the multiplicity of tests and techniques available to measure IQ and its various components. Clements (1966) lists 99 symptoms of MBD which he culled from the literature on learning disabilities. Wissink, Kass and Ferrell (1975) list over 35 different components for learning disabilities while Bush and Waugh (1976) list 38 separate components and over 60 different tests and measures in nine categories which have been found useful in measuring learning disabilities.

A number of classifications or categories based on theoretical and etiological considerations have been suggested to describe learning disabled children. Kephart (1960) and Barsch (1967) have both advanced the idea of inadequate perceptual-motor functions as a factor in learning disorders. Although they do not specifically list the perceptual-motor functions involved, Kephart (1960) lists posture, laterality, directionality and body image as important functions, while Barsch (1967) lists muscular strength, dynamic balance, body awareness and spatial and temporal awareness as important factors. In addition, Cratty (1968, p. 205) theorizes that figure recognition, serial memory ability and similar perceptual abilities may be enhanced

by proper motor activity while McCarthy (1974) lists body image, body orientation, body movement and haptic (touch) processes as important for teachers in dealing with learning disabilities.

Sensory integrity has also been seen as an important function for categorizing language disabilities according to deficits in the analysis of sensory information, synthesis of sensory information and the performance of symbolic operations. Kirk, McCarthy and Kirk (1968) developed the Illinois Test of Psycholinguistic abilities based on categories of auditory and visual reception, association, closure, expression, and sequencing memory. McCarthy (1974) sees cognitive, visual, auditory, memory, and intersensory and intrasensory processing as important factors in learning disabilities while Wissink, Kass and Ferrell (1975) list similar categories of sensory orientation, memory, reception, expression, and integration as important.

One of the first to describe categories for learning disabilities in language, Johnson and Myklebust (1967) included perceptual, symbolic, conceptual and imagery dysfunctions as well as receptive, expressive and inner language disorders. Frostig (1963) developed categories for learning disabilities based on visual-perceptual difficulties including problems in eye-hand coordination, figure ground discrimination, constancy, spatial relations and position in space. Similar categories were developed by Kephart (1960) in his perceptual motor scheme which included problems in form perception, figure ground discrimination and space and time discrimination. In a rather broad approach utilizing sensory, motor and neurological categories, Ayres (1972)

describes four areas from which to examine learning disabled children. Ayres' (1972) categories include generalized dysfunctions involving: 1. sensory integrative deficiencies; 2. postural and bilateral integrative dysfunctions resulting from vestibular disorders which are expressed in poor interhemisphere communication and language lateralization; 3. apraxia (intentional movement problems) resulting from poor sensory integration; and 4. problems of the left and right cerebral hemispheres which result in linguistic and other learning problems. Also utilizing a rather broad approach, Bateman (1966) discusses three major sub-categories of learning disabilities which include dyslexia (reading difficulties), verbal communication difficulties in the form of comprehension or expression of linguistic speech, and visual-motor integration difficulties.

Sensory-Motor Modalities

Over the last 25 years a number of orientations to learning disabilities have arisen based on specific theoretical considerations involving sensory, motor and cognitive systems.

Perception

Wepman (1964) has suggested the idea that children learn through a variety of perceptual modalities and that a problem may arise for some children because a particular pathway is so inefficient that it becomes an unproductive pathway for learning. In order to optimize learning, a child's strengths and weaknesses in visual, auditory and tactile modes should be assessed and the child's best modality

for learning should be utilized. Wepman (1964) lists several approaches for teaching based on such a model:

1. Teaching through the intact modality. In this approach teaching and presentations incorporating the child's best learning mode are used in providing information to be learned.

2. Strengthen the modality of deficit. Using this approach the child is given tasks which are aimed at improving the efficiency of a weak modality in order to improve it as a learning channel.

3. Combination approach. This method combines the weak and strong perceptual modalities in order to improve learning and perceptual abilities as the child integrates information.

Sensory and Perceptual Integration

In many learning tasks information received through one sensory modality must be integrated with information from other sensory modalities for efficient processing to occur. Chalfant and Scheffelin (1969) have suggested the idea of an integrative disorder to describe the breakdown of cross modality communication when two or more sensory inputs are involved in a task. They also suggest that this inability to go from one to two or more modalities efficiently may be a major factor in learning difficulties.

Another proposal for perceptual integration has been presented by Johnson and Myklebust (1967) as the "semiautonomous systems model of brain function." Briefly, this model conceptualizes the brain as being made up of semi-independent modality systems such as the visual, tactile or auditory systems. Sensory and perceptual modalities

can function in three ways as described by Johnson and Myklebust:

1. The intraneurosensory system. This system refers to learning that takes place primarily through one input pathway which functions relatively independently from other pathways.

2. The interneurosensory system. This system involves learning that occurs from the interrelated function of two or more modalities in combination. This type of brain function also includes associative processes in which one type of sensory information is converted for use by a different modality.

3. Integrative system. In this form of brain function all sensory and perceptual systems function as a unit to allow learning to take place.

It is speculated that many learning difficulties arise from problems in integration which may manifest themselves as problems in comprehension, conceptualization or nonverbal learning. One form of an integrative problem is perceptual system overloading. Perceptual overloading refers to the reception of information from one modality interfering with information coming from another modality. It is hypothesized that the child with learning disorders may have a lower tolerance for processing and integrating information from several input pathways which leads to a breakdown in perception. Johnson and Myklebust (1967) list problems from overloading as confusion, poor recall, retrogression, refusal of the task, poor attention or temper tantrums, and reading difficulties if cross modality problems are involved.

Suggestions for alleviating sensory and perceptual integrative problems typically involve the child practicing learning through two or more modalities. Frostig (1968) proposes exercises in cross modality association such as following spoken directions, describing pictures, finding certain objects in pictures, feeling objects through a curtain and drawing their shape on paper and determing whether two objects are the same or different when one is touched and the other is seen. Ayres (1968) believes that similar exercises would be helpful for improving difficulties in reading and motor execution.

Perceptual Motor Factors

Kephart (1967) has proposed a perceptual motor theory as a way of explaining learning disabilities. Kephart's theory stresses the importance of the development of normal perceptual-motor activity for the perception of stable and reliable concepts of reality. This occurs through the normal sequence of developmental motor patterns and generalizations. If stable motor patterns are not developed by age six, Kephart feels children will manifest problems when confronted with symbolic materials due to an inadequate orientation to the basic realities of the world in space and time. In order to think symbolically the child must be able to make precise observations about space and time and relate them to objects and events. A number of exercises have been suggested to improve motor patterns and generalizations. These include:

1. Balance and maintenance of posture whereby a child becomes aware of and maintains a relationship to gravity.

2. Contact whereby a child obtains information about the world by manipulating objects.

3. Locomotion whereby a child observes and learns relationships between objects in space.

4. Receipt and propulsion in which a child learns about movements of objects in space by motor activities such as catching, pulling, pushing and throwing.

In a similar approach, Cratty (1969) has emphasized movement games as a means of improving classroom learning through sensory experience. Cratty suggests how physical education activities can improve attention span, self concept, social acceptance and academic performance in a variety of ways.

Neurological Organization

A patterning theory of neurological organization has been proposed by Doman and Delacato (1966). They state that full neurological organization is attained by sequential progressions of development from lower to higher levels in the central nervous system. This progression starts in the spinal cord and medulla and ascends through the pons, midbrain and cortex and ends with the development of cerebral dominance. During this progression six functional attainments are developed. They include motor skills (mobility in walking upright), speech, reading and visual skills, writing, speech comprehension and tactile skills. Doman and Delacato maintain that the failure to pass through a particular sequential stage of development will lead to problems in mobility and communication. In order

to overcome such problems, children are given exercises designed to develop weak neurological stages which will result in the alleviation of learning difficulties. Although there is popular support for this treatment, a number of medical and health organizations have expressed concern about this form of patterning as a treatment for neurologically handicapped children.

Visual-Motor Activities

Getman (1965) has developed a model of learning based on the integration of visual and motor skills in an hierarchical fashion. In order for a child to attain proper intellectual function a number of levels or stages of development must be attained, with each higher level dependent on the successful development of lower ones. Getman terms these stages as follows:

1. The innate response system involving reflex activity such as the grasp, startle or pupil reflexes.

2. The general motor system involving locomotion and mobility activities such as crawling, walking, jumping and running.

3. The special motor system representing combination of motor skills such as eye-hand coordination and voice-gesture relationships.

4. The ocular motor system in which eye movements must be developed and coordinated with specific tasks in order for successful classroom performance to occur. Special eye skills include fixation, pursuit, and rotation.

5. The speech motor system which involves the integration of speech motor and auditory processing systems based on efficient visual and ocular systems.

6. The visualization system whereby the ability to visually recall an image in the absence of the original sensory input is developed. Tactile, auditory, haptic, and olfactory senses are also seen as contributing to this system.

7. Vision or perception resulting from the experiences, skills and systems of lower levels.

8. Cognition based also on proper development of lower levels in which abstract and symbolic thought occurs.

Although Getman's theory is similar to other motor theories in its elaboration of motor development in the lower stages of development, it differs in the higher levels in its emphasis on the visual sensory system. Getman suggests that without proper visual integration with the motor systems proper learning cannot occur. This system has been criticized for being too reliant on the visual system and for oversimplifying the development of learning (Myers and Hammil, 1969).

Biomedical Factors

A number of reports have also been presented describing medical and environmental problems as factors in learning disabilities. Hallahan and Cruickshank (1973) have suggested that early malnutrition may be related to attention and perceptual problems. That improper nutrition can lead to learning impairment is supported by a number of studies. Winnik and Rosso (1975) report on a number of animal and human studies which found that prenatal and postnatal dietary deficiencies can lead to a decrease in the number of brain cells produced. For instance, pyridoxine (B_6) has been used as an illustration of the effects of vitamin deficiencies on brain

function (Coursin, 1968). Serendipitous pyridoxine deficiencies in human infants have been found to be the cause of convulsive seizures, behavioral disorders, hyperirritability and performance decrements. Animal studies have shown B_6 deficiencies to be a factor in impairing conditioned reflex responses and in increasing learning time.

The effects of malnutrition on a child's interactions with and subsequent learning about his environment and surroundings appears to be as much a factor in intellectual development as are the physiological problems that arise from early malnutrition (Levitsky, 1976). Laboratory animals raised in conditions of normal and deficient diets and normal and deficient environments show the greatest intellectual impairment in the diet deficient-environment deficient condition. Other biochemical factors reported to be involved in learning disabilities include hypoglycemia (Dunn, 1973), allergic reactions (Havvard, 1973), and food additives (Feingold, 1975).

Test Procedures

A central issue in the amelioration of learning disabilities is one of diagnosis or the ability to correctly label those youngsters "at risk" from becoming underachievers or school failures (Mercer, Algozzine and Trifiletti, 1979; Adelman, 1978). It is only necessary to look at the varying estimates of LD prevalence, which range from 1% to 30% of the preschool and school population (Adelman, 1979a; Belmont, 1980), to appreciate the difficulty in diagnostic assessment. The major difficulty lies in the heterogeneity of symptoms and the overlap of characteristics and correlates of learning disabilities

with other categories such as "emotionally disturbed," "disadvantaged," "educably mentally retarded" and "behavioral problems" (Adelman, 1978; Wepman, Cruickshank, Deutsch, Morency, and Strother, 1975). Other factors leading to diagnostic difficulties arise from problems of methodology (Adelman, 1979b; Gallagher and Bradley, 1972; Hanna, Dyck, and Holden, 1979; Mercer et al., 1979), lack of specific criteria for cutoff scores and labeling and construction of normal-abnormal standards (Adelman, 1978; Strupp and Hadley, 1977), inadequacies in the measurement and conceptualization of the particular processes with which diagnosticians have been concerned and fluctuations in motivation (Cronback, 1975; Salvia and Ysseldyke, 1978; Torgensen, 1979), poor validity or inconsistent findings of IQ measures to predict school performance, especially for borderline cases (Ables, Aug, and Loof, 1971; Helper, 1980; Keogh and Becker, 1973; Smith and Marx, 1972; Waksman, 1978; Wohl, 1980), and disagreement over interpretation and observation of less clear "soft signs" (Wepman et al., 1975). These considerations can lead to a lack of standardization and specificity which can result in children with the same behaviors and symptoms being seen as problems in one situation or school and not another (Adelman, 1978; Strupp and Hadley, 1977).

In a discussion of the differential diagnosis of learning disabilities the preceding considerations have led many authors to conclude that, at present, relationships between single specific preschool tests, test patterns, questionnaires, rating scales and physiological, psychological and social measures are too low to predict later school achievement or to be certain as diagnostic of any given underlying condition (Keogh and Becker, 1973; Wohl, 1980). Similar concern has

been expressed by Adelman (1978), Wepman et al., (1975) and Torgesen (1979). Ackerman, Peters and Dykman (1971) found no difference on the Wechsler Intelligence Scale for Children (WISC) between mildly reading disabled children and a control group. They also found no correlation between visual motor integration problems seen in neurological examination and WISC subtest scores. Hutcherson (1971) found similar Wechsler Intelligence Scale for Children Revised (WISC-R) verbal, performance and subscale scores for mentally retarded (MR) and LD groups. Similar reports of poor diagnostic profile differentiation between above average, MR good and poor readers, and between perceptual, reading and other groups and control groups have been reported by Chapman and Wendell (1972), Hirshoren and Kavale (1976), Miller and Brecht (1978), Miller, Staneburner and Brecht (1978), Romanauskas and Burrow (1973) and Stevenson (1979). Coles (1978) in a review of validation studies focusing on the 10 most often recommended tests and evaluations for learning disabilities concluded that there is an inadequate empirical base for claiming that such procedures can validly diagnose learning disabilities. Tests reviewed by Coles included the Illinois Test of Psycholinguistic Abilities (ITPA), the Bender Visual-Motor Gestalt, the Frostig Developmental Test of Visual Perception, the Wepman Auditory Discrimination Test, the Lincoln-Oseretsky Motor Development Scale, the Grahm-Kendall Memory for Designs Test, the Purdue Perceptual-Motor Survey, the WISC, a neurological evaluation and an electroencephalogram.

In contrast, numerous examples can be found in reviewing the literature on measures such as Coles (1978) lists which show the tests

to be helpful in differentiating learning disabled children from other educationally handicapped and normal children (e.g., Edmunsen, 1973; Belmont, 1980; Golden and Anderson, 1979; Hallahan and Kauffman, 1976; Helper, 1980; Mercer et al., 1979; Sanders, 1979; Schain, 1977; Wohl, 1980). Using the WISC, Sobotka, Black, Hill, and Porter (1977) found verbal and performance scores to distinguish between 24 dyslexic boys and 24 controls at ages 7, 9, 11 and 13 years. The WISC-R has been found to differentiate between groups of learning disabled and reading disabled children, between educable mentally retarded (EMR) and LD children, between learning disordered hyperactive, EMR and LD groups, and between emotionally disturbed and LD children (Feeler, 1975; Keogh et al., 1973; Simonds, 1974; Wetter, Keogh, McGinty, and Donlon, 1972). In a study of 114 10-13 year-olds, Simonds (1974) found the Wide Range Achievement Test (WRAT) to be useful for screening for emotional and learning problems and Koppitz (1970) notes numerous studies which provide evidence that the Bender Gestalt Test can be used to distinguish between children with and without brain damage and to distinguish between those that do or do not have reading problems. Wagonseller (1973) reports the WISC, WRAT, Behavioral Stimulus Differential (Ideal) Test Score and the Quay and Peterson's Behavior Problem Checklist all capable of differentiating elementary LD, institutionalized, and non-institutionalized emotionally disturbed children.

The question of diagnosing and predicting learning disabilities from other problems becomes not one of finding tests but rather one of how to increase the efficiency and effectiveness of such procedures. Suggestions for psychologists, educators and other professionals fall

into two general categories: utilization of patterns of subtest scores, especially WISC and WISC-R scores, and use of multiple tests and procedures incorporating information from educational, medical and psychological areas.

Test Patterns--WISC and WISC-R

The importance of subtest patterns is pointed out in a discussion of WISC-R patterns by Banas and Wills (1977). They suggest that while intelligence quotients such as full scale scores on the WISC-R may mask differences in performance, a knowledgeable analysis of subtest patterns can help to identify weak and strong learning avenues in children. In a study of 67 LD, 19 slow learners, 60 controls and 71 gifted children, Gordon (1978) reports that the LDs scored lower on five out of six verbal subtests given when compared to the control group. Only coding of the performance subtests was found to be lower. Anderson, Kaufman, and Kaufman (1976) studied 41 6-15 1/2 year-old learning disabled children and found the LD children did better on the performance than the verbal subtests, being particularly deficient in information, similarities and vocabulary. Similar profiles have been reported by Smith (1978), Wallbrown, Vance, and Blaha (1979) and Greer (1972). Ekwall (1966) reports findings similar to Gordon's for a group of poor readers who showed decreased verbal and increased performance WISC subtest scores with coding being the best performance indicator.

Different patterns of subtest scores have been proposed by Witkin, Dykes, Faterson, Goodenough, and Karp (1962) as delineating three relative independent skill areas for the WISC. These areas were identified as verbal-comprehension composed of the information, vocabulary and comprehension subtests; analytic-field-approach

composed of object assembly, block design and picture completion subtests; and attention-concentration composed of arithmetic, digit span and coding. Kaufman (1975) reports that the WISC-R can be divided into three skill areas very similar to those described by Witkin et al. (1962). In a study of children at 11 age levels between 6 1/2 and 16 1/2 years, Kaufman found the freedom from distractibility (identical to Witkin et al.'s attention-concentration factor) to be useful for diagnosing reading and learning disabilities. Stevenson (1980) found decreased digit span, arithmetic, coding and information scores on a group of 55 clinic referrals. She found that this LD group had the lowest WISC-R factor score on the attention-concentration component of Witkin et al.'s three subtest categories. Smith (1977) Vance and Singer (1979), and Bannatyne (1974) all found reading or learning disabled groups to score lower on WISC-R subtest of arithmetic, coding, digit span and mazes, while Tabachnik (1979) reports greater performance but not verbal subtest scatter when LD children are compared to normals.

Multiple Tests and Procedures

A number of authors have suggested that multi-test procedures provide more utility due to their ability to provide a greater overall analysis of factors involved in the genesis of learning disabilities. Because, as Koppitz (1973) points out, "there is no one-to-one relationship between any single symptom or characteristic of a child and his social adjustment or progress in school," the broader area covered in using several measures allows for a more specific pinpointing of a child's problems which in turn allows for more accuracy in predicing future performance. For instance, Mercer

et al. (1979) lists the overall hit rate for 30 studies using single or multiple test procedures to identify children who are at risk of future school problems. Although there are methodological considerations for not doing so, if one were to average the results from the single and multiple tests reported by Mercer et al., the multiple procedures average almost 10% better at identifying children who will have school problems.

By using a large number of tests to evaluate language and perceptual and motor abilities, deHirsh, Jansky, and Langford (1966) and Jansky and deHirsch (1973) were able to predict between 76% and 91% of those children tested in kindergarten who would have trouble with or fail reading by the end of grade two. DeHirsch et al. (1966) developed the "Predictive Index" out of the 10 tests which were of greatest value in identifying children as being at risk of future failure. The 10 best identifiers were pencil use, Bender Gestalt Test, Wepman Auditory Discrimination Test, number of words used in a story, categories, Horst Reversals Test, Gates Word Matching subtests, Word Recognition I and II, and Word Reproduction.

In 1972, Eaves, Kendall, and Chrichton used a "Modified Predictive Index" (addition of a Draw a Person and Name Printing to Predictive Index), a neurological examination consisting of the Beery Test of Visual-Motor Integration, Weschler Preschool and Primary Scale of Intelligence, Illinois Test of Psycholinguistic Abilities, Kephart Motor Survey and a behavioral rating scale to identify kindergarten children in terms of learning problems. Children were initially identified by the Modified Predictive Index (MPI). Twenty-five children considered to

have failed the MPI (score of 0 or 1 out of 10) were matched with 25 controls and then classified as MBD, immature or normal based on the neurological and psychological exams. The MPI was found to have a 92% correspondence with the neurological and psychological exams in classifying the children into these three groups. Of 196 variables looked at by Eaves et al. (1972) a small number distinguished the three groups perfectly. These variables are given in rank order as : visual motor coordination on the Kindergarten Development Record given in June (i.e. teacher's assessment of the child after one year in school), the Horst Reversals Test administered in October, neurological assessment of short attention span and temper tantrums, speech quality as assessed by kindergarten teacher, visual association scale score on ITPA, Wepman Auditory Discrimination Test from the October MPI, and the number of words in a story from the June MPI. The MBD group scored lowest on all but the last variable on which the immature group scored lowest.

In a follow-up of these children at the end of grade two, Eaves, Kendall, and Crichton (1974) report that a combination of the MPI, neurological, psychological and kindergarten teacher evaluation could successfully predict up to 95% of the children as to grade two teacher's estimate of readiness for grade three from a group of only 14 variables. Conners (1973) found that children referred for evaluation of treatment of behavior disorders, learning disorders or combinations of both could be separated into six distinct groups based on a number of psychological and neurological measures. The identification of problems based on these tests ranged from a group suspected of diffuse

brain damage to a group thought to be normally functioning but whose problems stemmed from anxiety and impulsiveness generated by parental factors. Finally, Alley, Deshler, and Warner (1979), using Bayesian methods, report increasing predictability and specificity as more tests and measures are utilized.

In conclusion, in looking through the literature on learning disabilities, one is struck initially by what appears to be a consensus on the difficulty or inability to diagnose learning disabilities using present test procedures. However, as one proceeds through at times contradictory and conflicting studies, a pattern does emerge. What is being said is not that we cannot differentiate LD children from others but rather that the use of a variety of strategies and procedures is needed in order to improve our present abilities. For the most part, these strategies suggest looking at particular patterns of results rather than general or overall scores in order to better outline learning disabilities from other forms of learning problems as well as distinguishing among the different forms of learning disabilities themselves. Such patterns can occur not only within a single measure but among a number of different measures as well. In part, if one were looking for the best predictor, it would have to be in the use of a wide variety of tests cutting across disciplines. It seems clear that as one includes measures from neurological, psychological, environmental, and teacher assessment, the ability to pinpoint specific types of learning disabilities improves. Any psychologist, educator or M.D. would improve diagnostic reliability by utilizing such procedures. Additional suggestions for improving the recognition of

learning disabilities include the use of national rather than local data for normative and derived scores (Hanna et al., 1979), the use of short-term measures and frequent testing (Keogh and Becker, 1973), and the use of task and situational contexts (Torgesen, 1979). Koppitz (1977) proposes the need to explore six interrelated yet distinct areas in order to achieve a more meaningful diagnosis. These areas are listed as inner control including concentration, attention span, frustration level, hyper-/hypo-activity, and perseveration among others; intersensory integration including perceptual-motor activity, short- and long-term memory, and intermodality sensory integration; reasoning; emotional adjustment; social adjustment; and developmental and social background.

Cortical Electrophysiology

From a behavioral point of view the term learning disabilities refers to a number of situations in which intellectual potential and academic performance do not conform. A variety of theories have been proposed to explain such discrepancies including several based on brain function. Psychological findings in individuals with known brain damage by investigators such as Werner and Strauss (1939) and Strauss and Lehtinen (1947), and investigations by a number of individuals (Kirk, 1967; Haring and Bateman, 1969; McCarthy and McCarthy, 1969) as well as task forces (Bateman and Schiefelbusch, 1969; Clements, 1966) have established one possible etiology for learning disabiltiies in the functional and physiological integrity of brain activity.

One means of looking at brain activity is by recording the electroencephalogram (EEG) or evoked potential (EP) under various conditions. Although not all investigators (Burnett and Struve, 1974; Dubey, 1976) consider the electrical activity of the brain to be a reliable indicator of learning disabilities, a number of studies have obtained positive results in identifying abnormalities in the EEG of learning disabled children. Daryn (1960) reported a 40% rate of abnormalities, Muehl, Knott, and Beuton (1965) a 63% rate and Capute, Niedermeyer, and Richardson (1968) a 50% incidence. Overall studies such as these suggest a 60% incidence of EEG abnormalities in LD children compared to approximately a 20% rate in control (normal) children. Penn (1966) concluded that 70-75% of reading disabled children have EEG abnormalities compared to a 5-10% incidence in control populations. Similar results have been reported by Kennard, Rabinowitch, and Wexler (1952).

EEG abnormalities can generally be separated into two groups, questionable and definite (Schain, 1977). Questionable abnormalities include:

6 and 14 per second positive spikes

Occipital or posterior temporal slowing

Nonfocal sporadic sharp waves

Excessive slowing or amplitude

Mild diffuse dysrhythmias

Definite abnormalities include:

Paroxysmal spike-wave discharges

Paroxysmal polyspike complexes

Repetitive focal spiking or slowing

Amplitude asymmetries greater than 50%

Marked diffuse dysrhythmias

Most of the EEG abnormalities reported for learning disabled children fall under the questionable category.

Neurological Factors

A frequently used measure with learning disabled children is the neurological examination (Benton, 1974; Goff, 1979). The purpose of the examination is to test the integrity of the central nervous system (CNS). A complete neurological investigation consists of tests of cranial nerve function; tests of reflexes primarily involving arms, hands and legs; tests of cerebellar function; tests of sensory perception; and tests of general cerebral function including emotional responsiveness, memory, social skills, verbal and spatial abilities, intelligence and right-left discriminations.

Like the EEG, neurological abnormalities can be divided into two categories encompassing "hard" and "soft" neurological signs. These indicators are, for the most part, differentiated by the extent and intensity of the abnormal performance displayed by the learning disabled children. In an examination of 90 minimally brain dysfunctional (MBD) labeled school children and 15 normal school children, Hertzig, Bortner, and Birch (1969) found a 29% incidence of hard signs and a 90% incidence of soft signs. Overall, 94% of the MBD group had some neurological impairment. Hard signs were mostly motor deficits when comparing one side of the body to the other. Soft signs most frequently

included speech, balance and coordination problems and, to a lesser extent, disorders in double simultaneous tactile stimulation, muscle tone, and overflow movements. Peters, Romine, and Dykman (1975) list the following neurological signs as differentiating LD and control children:

Holding arms out straight	Foot tapping
Rotation of the head	Finger to nose--eyes closed
Copying finger movements	Bilateral coordinated movements
Abnormal motor movements	Reflexes
Hopping on one foot	Right-left confusion
Standing on one foot	Eye movements
Walking a straight line	Two-point tactile discrimination
Reading, spelling and writing problems	

Similar findings have been reported by Clements and Peters (1962), Ingram (1973), Penn (1966) and Wikler, Dixon, and Parker (1970).

The occurrence of neurological soft signs has been found in a number of studies to coincide and overlap with the occurrence of EEG abnormalities in MBD children. Because of the underlying relationship between these two factors, the observation of positive neurological signs can give added weight to the probability that EEG abnormalities seen in learning disabled populations represent a homogeneous diagnostic criterion rather than an extension of the variance of the EEG as seen in normal children. In a series of studies by Stevens, Sachdev, and Milstein (1968) and Stevens and Milstein (1970), clumsiness and hyperactivity, among other signs, were found to be more frequently

associated with children displaying EEG abnormalities in a group of behaviorally disordered children. Capute et al. (1968) found a 50% incidence of EEG abnormality in MBD children diagnosed by symptomology and soft neurological signs, while Gerson, Barnes, Mannino, Fanning, and Burns (1972) found twice the incidence of neurological abnormalities in a group of LD children displaying EEG abnormalities as compared to those without EEG abnormalities. Satterfield (1973) in a study of 88 6-9 year-old MBD children, reports that children with an abnormal EEG or four or more soft neurological signs responded better to treatment with methylphenidate than children without such abnormalities.

Electroencephalogram

In reviewing the literature concerning electroencephalographic differences reported between learning disabled and normal children, two main divisions can be established. The first of these involves the EEG and is primarily concerned with frequency distribution of the electrical activity of the brain. The second involves the evoked or average evoked potential (EP). Findings concerning the EP center mainly around amplitude and latency shifts seen among the various peaks. Common to both these types of brain activity are findings of topographical or spatial differences between normal and LD children. Diagnostically the above findings have generally been divided into three broad categories that can best be labeled as LD-MBD, dyslexia and hyperactivity. These categories should probably best be thought of as encompassing generic rather than specific forms of the problem subsumed under each heading due primarily to differences encountered

between studies in terms of selection criteria, testing materials, and severity of the disability. For instance, Satterfield, Cantwell, Lesser, and Podosin (1973) had an IQ criterion of $\geq$ 80 and independent diagnosis of MBD by two psychiatrists while Hughes (1971) used an IQ criterion of $\geq$ 90 and classified children as MBD by a score of $\geq$ 90 on Myklebust's (Myklebust and Boshes, 1970) learning quotient score. In general, however, it can be stated that most studies involve children with approximately normal IQ's and exhibiting some form of learning deficiency.

Six and 14 Hz positive spikes. A pattern of activity involving 6 and 14/sec positive spikes has also been primarily associated with problems in reading and is seen most frequently during sleep recordings. Muehl et al. (1965) found a 59% incidence of positive spikes in a group of dyslexic children. Similar findings are reported by Hughes and Park (1968) who found a 44% incidence in a dyslexic group of children. Stevens et al. (1968) found a 36% incidence in a group of behavior disordered children also displaying math problems and Roberts (1966) reports bilateral occipital or parietal spikes in children with visual perceptual problems.

Although positive spikes have been found with a number of children diagnosed as LD, their diagnostic value has recently been questioned. Capute et al. (1968), while finding a 24% incidence of 6 and 14/sec positive spikes in a group of 106 MBD children, question its significance because of the relatively high incidence of this activity in normal children. In the study conducted by Stevens et al. (1962) one control group was found to have a 36% incidence of positive

spikes and Hughes (1971) found a 16% and 14.3% incidence in two LD control groups. Also, Gerson et al. (1977) report positive spikes being negatively correlated with a history of brain damage or with impulsivity. It would appear that positive spikes may be of limited value, especially if they are the only signs of EEG abnormalities.

3-4/sec slow waves. By far the most frequent EEG abnormality associated with learning disabilities is the occurrence of slow waves, particularly in the posterior-temporal region. Aird and Gastaut (1959) report slow waves in subjects selected from the general population from 5 to 25 years old with a maximal occurrence at age 10. This activity was observed in the occipital, parietal and posterior temporal areas, was of a synchronous polyrhythmic random nature, blocked with eye opening, accentuated with hyperventilation, was rarely seen in bursts and showed a right hemisphere dominance in 50% of the subjects. Aird and Gastaut terms these waves "slow posterior waves of youth" and concluded these waves were not associated with EEG abnormalities or symptoms other than immaturity. These authors did, however, consider larger bursts or continuous slow waves of a rhythmical or sinusoidal nature to be abnormal. Sorel, Leotard, Delaite, Rucquoy-Ponsar, Basecqz, and de Biolley (1965) found a 13% incidence of posterior slow waves in a group of 14-20 year-old normals, of whom 35% showed problems of scholastic adaptation (i.e., school related problems).

In the context of learning disabilities, Pavy and Metcalfe (1965) found slow waves associated with poor visual motor performance and speech and language problems. Hughes and Myklebust (1968) found

an 18.8% incidence of posterior slowing as the sole abnormality in a group of 154 learning disabled children. Hughes and Park (1968) found a similar incidence in a smaller group of dyslexics. However, if occipital slowing and positive spikes were included, the number of children showing abnormalities increased to 33%. Slow waves have also been associated with hyperactivity and behavioral learning problems (Cohn and Nardini, 1958; Wikler et al., 1968). Using sophisticated statistical and computerized techniques, John (1977) found parieto-occipital slowing to be the most common abnormality in a group of 50 children considered at risk for minimal brain dysfunction underlying their learning disorders. Of this group, 46 out of the 50 children showed a significant excess of slow (delta) activity in at least one brain region. Lairy (1965) reports slow occipital waves as characterizing a group of children with language problems and Fenelon, Holland, and Johnson (1972) report increased occipital frequency occurring in a group of reading disabled children showing a favorable response to drug therapy.

EEG frequency patterns. With the notable exception of John (1977), most of the studies mentioned so far have utilized "traditional" test procedures for recording the EEG. For the most part this encompasses recording the background EEG while subjects are awake with eyes open or closed, asleep, and/or during hyperventilation. While the value of EEG in MBD children is generally accepted, several authors (Burnett and Struve, 1974; Dyment, Lattin, and Herbertson, 1971) have expressed concern for its use as a diagnostic tool because of the somewhat nonspecific nature of the findings. However, Burnett and his

associates (1974) conclude that new advances in EEG technology and new procedures will probably make the EEG recording more important as a diagnostic tool.

During the last decade many researchers have developed new procedures for examining the electrical activity of the brain in learning disabled children. These techniques primarily involve looking at a number of frequency bands simultaneously while having subjects perform different tasks. Findings have been most notable for children labeled LD and dyslexic. In a single case design Piggot (1972) looked at the EEG of an 11 year-old girl with EEG abnormalities and mild dyscalculia under conditions of rest, key press, arithmetic, learning and easy reading. Results showed significantly greater EEG discharge during the arithmetic and learning tasks than during any of the other conditions.

In a study involving 24 9-10 year-old boys labeled as having specific reading disabilities (SRD) and 24 matched controls, Mechelse, van Gemund, Nije, Burg, and Laurs (1975) found significant increases in delta and theta activity in the SRD group and significant increases in 9.5 to 12.4 Hz activity in the controls. Data were collected using auto, cross and phase spectral and coherence measures in an eyes closed condition. Similar frequency findings are reported for a group of 8-11 year-old learning disabled boys by Douglas (1978). Sklar, Hanley, and Simmons (1972) report quite interesting results obtained by spectral analysis on a group of 13 dyslexic and 12 matched controls. EEG data were collected during eyes closed rest, eyes open attention, performing mental arithmetic, reading word lists and reading text. The most

prominent differences occurred in the parieto-occipital region during the eyes closed condition. On the average, dyslexics had more energy in the 3-7 Hz bands while the normals had more energy in the 9-14 Hz range. During reading the 16-32 Hz band differences were reversed with the normals showing more energy than the dyslexics. During reading, coherence patterns between electrode positions were the best discriminator between the two groups. Dyslexics displayed more coherence within the same hemisphere while normals showed more coherence between hemispheres. Overall, reading test, eyes open, and eyes closed conditions were the best discriminators between dyslexics and normals. Differences in hemispheric patterns have also been reported by Rebert, Wexler, and Sproul (1978). In a comparison between a group of dyslexic and nondyslexic children they found that the dyslexic group showed a reversal of theta asymmetry from an eyes closed to an eyes open condition which was most prominent at parietal placements. Finally, in a study comparing 20 age and IQ matched LD and normal males, Johnson (1977) obtained EEG spectrals during eyes open resting, verbal-visual task, verbal auditory task, and tactile-kinesthetic task conditions. She found the most significant differences occurred between the EEG's of the two groups in the 12.75-17.5 (beta) frequency range with LD's having more slow activity and less beta activity. Provocatively, in a 20 month follow-up testing, Johnson (1977) found that children who improved on the performance tasks showed a decrease in slow frequency activity and an increase in the incidence of beta activity between the two testing periods.

whole page

Alpha activity. Recently a number of studies have appeared which indicate that alpha (broadly defined as 8-13.5 Hz) activity may serve as a useful indicator differentiating MBD-LD children from normal children. Alpha deficits have been found in children exhibiting attention and concentration problems, dyslexia, and decreased academic performance. In groups of 8-11 year-old normals, dyslexics, and attention problem children, Martinius and Hoovey (1972) found that occipital alpha activity and synchrony between right and left occipital alpha pairs could reliably distinguish between the normal controls and the clinical groups. Examining 8, 9 and 10 year-old dyslexics and normal children, Colon, Notermans, de Weerd, and Kap (1979) found increased alpha in normal 8 year-olds, increased mu in 9 year-old normals and decreased theta activity in the 10 year-olds. In a study involving 11 year-old children with above average, average and poor abilities to concentrate, Grünewald-Zuberbier, Grünewald, and Rasche (1978) found significant differences between groups. Compared to children with low concentration abilities, children with average and especially above average abilities showed stronger central occipital alpha reduction to a warning stimulus, more occipital alpha reduction to an imperative stimulus and stronger alpha attenuation to a discriminative versus a simple reaction task. Ludlam (1979) found increased alpha attenuation with reading improvements in two patients with visual and reading problems who had originally shown an inability to suppress alpha. This decrease occurred during and after a program of visual training in which the two patients participated. Fuller (1976) reports similar findings of a lack of alpha attenuation in a group of LD children. Compared to a group of normals, the LD group

showed considerably less alpha attenuation during mental arithmetic and immediate memory than did the normals. Fuller interprets these results as indicative of attentional deficits in the LD group. Duffy, Denkla, Bartels, and Sandini (1980) found alpha differences between a group of 9-11 year-old dyslexics and normals in the left medial frontal, midtemporal and anterior lateral frontal regions and bilaterally in the medial frontal area for a variety of tasks. In general, the dyslexics had an increase in alpha activity compared to the normals. Duffy et al. interpret this as suggesting relative cortical inactivity in the dyslexic children.

In a group of children with homogeneous verbal, motor and perceptual test scores compared to a group of children with gross discrepancies in these measures and severe scholastic and extrascholastic difficulties, Lairy, Rémond, Rieger, and Lesèure (1969) found a greater average alpha asymmetry in the control group and poorer spatial organization and a greater spread about the mean for alpha activity in the group with learning difficulties. Shabsin (1980), in a comparison of children diagnosed as LD and normal, found the LD group to have greatly reduced alpha activity compared to the controls in an eyes open resting condition and to have less discrepancy in alpha activity between resting and performance conditions.

Only two studies were found which looked specifically at alpha activity in hyperactive children. In one (Grünewald-Zuberbier et al., 1975), it is difficult to assess the results because no normal control group was utilized. Grünewald et al. compared 11 hyperactive and 11 nonhyperactive (mean age 12.2 years) children with behavior

problems. They found in stimulation-free periods that the hyperactive group showed more alpha and beta amplitude, more alpha waves and less beta activity in comparison to the nonhyperactive group. The other study (Montagu, 1975) looked at alpha propagation time using spectral analysis and phase alignment procedures in a group of 10 hyperactive and 10 matched controls. No significant differences were found for these two groups on the alpha index used. The best differentiation was obtained by use of an EEG coherence function. It is interesting to note, however, that Montagu does report intrahemispheric activity up to 8 Hz as being significantly higher in LD children. He also reports LD children as having reduced 10 Hz activity in the right central-parietal and occipital areas. Since neither of these studies clearly address the issue of alpha activity and hyperactivity (although Montagu utilized a control group, other factors besides alpha propagation time may be relevant), it would appear that a clarification of this form of minimal brain dysfunction and alpha activity must await further research.

Hemispherical asymmetries. A large number of studies have shown that the cerebral hemispheres in normal humans are not functionally symmetrical for different tasks and that these differences can be observed by the use of EEG recordings (Butler and Glass, 1974; Doyle, Ornstein and Galin, 1974; Ehrlichman and Wiener, 1980; Galin and Ornstein, 1972; Osborn and Gale, 1976). Generally, the left hemisphere has been found to be dominant for verbal and quantitative tasks and the right to be dominant for spatial and nonverbal activities. A lack of these typical asymmetry patterns has been seen in the EEG of several

groups of LD and dyslexic children. These studies tend to indicate either a deficit in left hemisphere dominance or a right hemisphere dominance for tasks normally processed by the left hemisphere.

Using a dichotic listening procedure with language disordered, articulation disordered and normal children, Pettit and Helms (1979) found the language disordered but not the articulation or control groups to show a lack of left hemisphere dominance. Similar findings have been reported for LD children by Witelson (1962) and dyslexics by Zurif and Carson (1970). Duffy et al. (1980) found significant difference in the EEG activity of dyslexics and normals in bifrontal as well as left temporal and left parietal regions which are areas normally involved in reading and speech. Shabsin (1980) reports typical normal patterns of alpha blocking and beta activity during verbal and spatial tasks for a group of controls. An LD group, however, showed this pattern only in the right hemisphere regardless of task. Cunningham (1978) reports similar findings in which the right hemisphere appeared to be doing an inordinate amount of processing to the decrement of verbal tasks in a group of LD children.

Evoked Potentials

Besides the EEG, the averaged evoked response (AER) has also been shown to be an indicator of brain function differentiating LD-MBD and normal children. The AER can basically be thought of as the brain's immediate or short term response to a stimulus. Generally, the most salient features of the AER are the amplitude and latency of the various peaks representing the electrical activity of the cortex or

underlying structures. Figures 1 and 2 show typical evoked potentials recorded from the cortex and the brain stem. While the AER may be evoked by a variety of stimulus modes, the majority of the studies employing LD children utilize visual and auditory stimulation to investigate brain function. Collapsing across the 19 electrode positions of the 10/20 system, John (1977) reports significant latency differences between samples of LD and normal children for grids of different spatial density, patterns, letters, and random and regular flashes and clicks. On the whole, studies incorporating AER data have dealt with three subpopulations of learning disabilities generally termed MBD-LD, dyslexia and MBD-hyperkinetic children treated with medication.

MBD-LD. Buchsbaum and Wender (1973) recorded visual evoked potentials (VEP) from 24 MBD and 24 matched controls for different intensity light flashes. The MBD children exhibited significantly greater N140-P200 amplitudes and shorter latencies for peaks at P100 and P200. The MBD group also showed a greater increase in N140-P200 amplitude with increasing stimulus intensities than did the normals. In comparing the AERs of the two groups, Buchsbaum and Wender noted that the MBD children showed AER characteristics of relatively younger normal children. Ahn (1978) found significant latency differences between normal and underachieving children using a number of visual stimulus conditions taken from the neurometric battery intended to challenge sensory, perceptual and cognitive processes. Verbal underachievers showed left hemisphere C3 and P3 differences at 290-380 milliseconds (msec) and C3 differences at 410-480 msec. Arithmetic

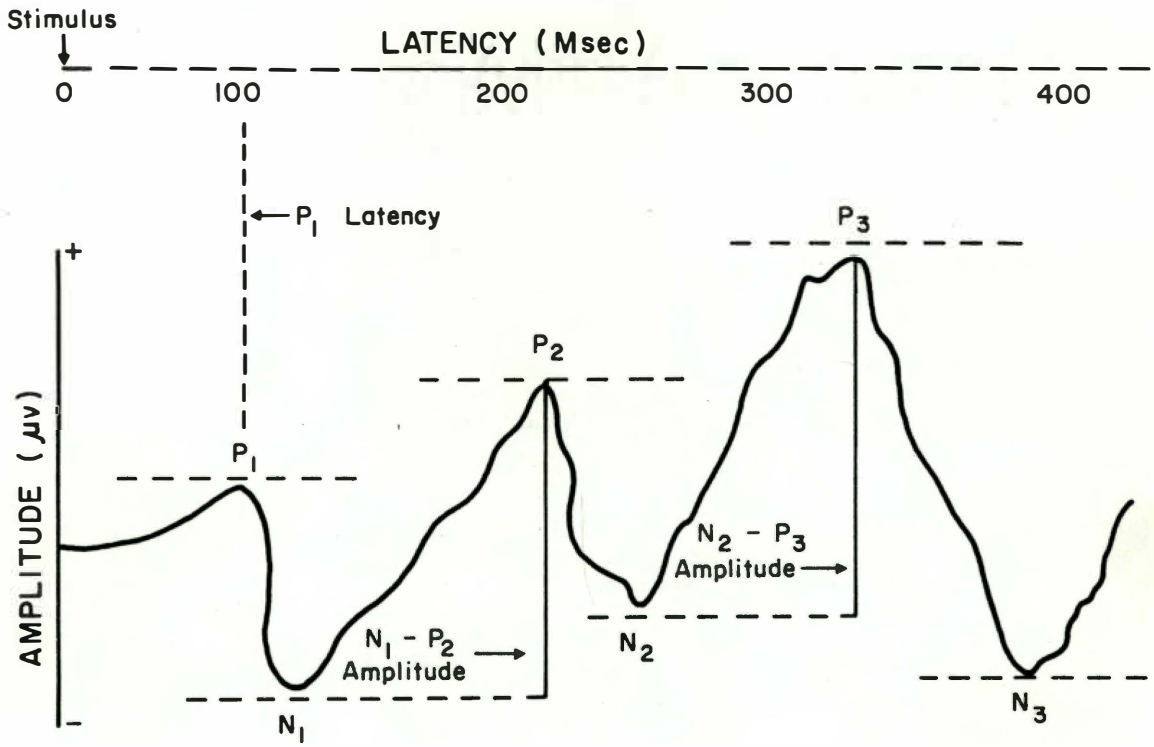


Figure 1. Typical human cortical evoked potential to repeated presentation of an auditory or visual stimulus. Amplitude and latency measurements for various peaks are shown.

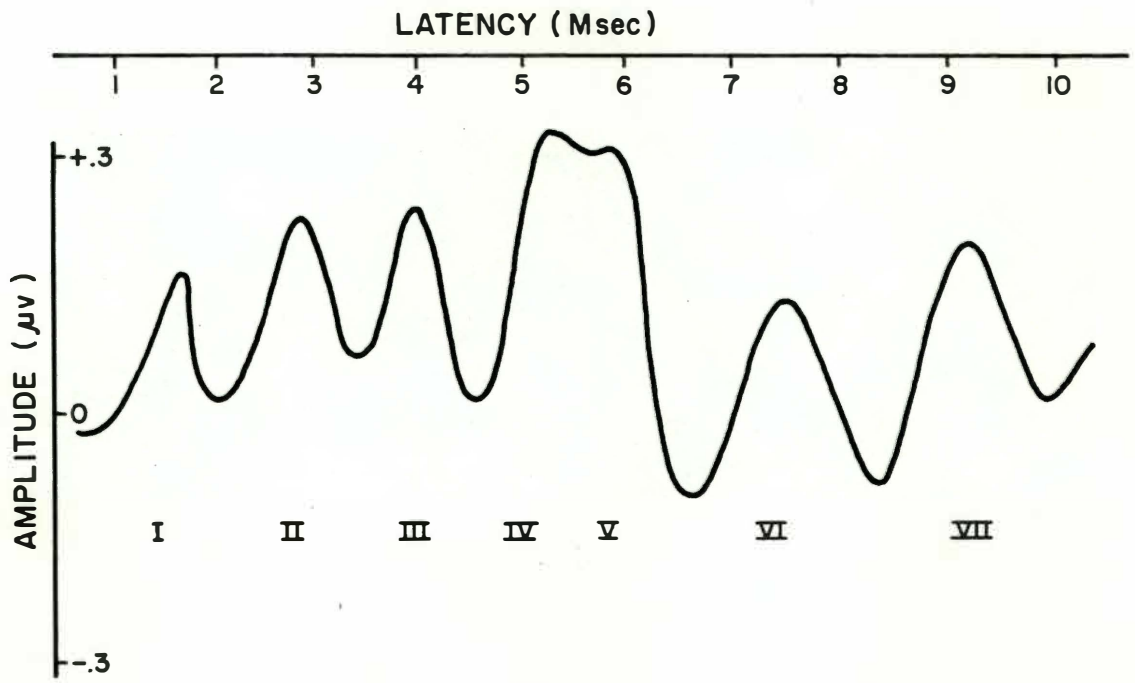


Figure 2. Typical human auditory brain stem response to click stimuli. Positive peaks labeled I-VII represent different structures in the auditory pathway. In ascending order these structures are the 8th cranial nerve, the cochlear nuclei, the superior olivary complex, the lateral lemniscus, the inferior colliculus, the medial geniculate, and the auditory radiations.

underachievers showed right hemisphere differences (C4 and P4) at 290-380 msec and combination verbal and math underachievers showed latency changes for left hemisphere positions C3, P3, O1 and T5 at 230-260 msec.

Shields (1973), using light flashes, words, designs and pictures as stimuli, found longer latencies (P1, N1, P2, N2, P3) and greater amplitude (P1, P3) for 10 LD (visual perception) compared with 10 matched controls. Grünewald, Grünewald-Zuberbier, and Netz (1978) report latency and amplitude differences in children with different abilities to concentrate for electrode positions CZ, OZ and FZ. Compared to average and good concentraters, children with poor concentration abilities showed longer P250 latencies and smaller P250 and P330 amplitudes. A standard CNV (contingent negative variation) paradigm was used and S2 stimuli were circles and squares. In a comparison of bright and dull children, Rhodes, Dustman, and Beck (1969) found increased VER excursions attributed mainly to amplitude over central scalp location (100-250 msec) for the bright children. Conners (1970) found significant negative correlations between achievement (reading and spelling) and amplitude of the N200 component of the left parietal (P3) VEP to light flashes in a group of 27 third and fourth grade LD children. Conners also found that a group of low verbal-high performance LD children had higher amplitude of P140 at P3, P4 and O2 and shorter latencies of N200 at O1 and O2 when compared to a high verbal-low performance group.

Using clicks as stimuli, Satterfield (1973) found the auditory evoked potential (AEP) to be significantly different in a group of

31 6-9 year-old MBD boys and 21 age matched controls. AEP's of the MBD group showed decreased amplitudes and increased latencies for the N1 and P2 components. Satterfield suggests that these findings in conjunction with EEG data mentioned previously indicate a delayed maturation of the CNS in some MBD children. In a study comparing nine males diagnosed as hyperactive and nine matched controls, Zambelli, Stamm, Maitinsky, and Loiselle (1977) compared the AEP elicited by tone pips for relevant and nonrelevant signals. A significant amplitude increase in the N1-P2 peak for signals presented to the attended ear versus the nonattended ear was found for the controls but not for the MBD group. Similar findings are reported by Prichep, Sutton, and Hakerem (1976) for single and paired click stimuli under conditions of certainty and uncertainty. In this study MBD children differed less in their AEP's between the two conditions than did the control group. MBD children had a smaller P200 and larger N250 than normals and displayed a larger rather than smaller P3 component during the certain condition. This last finding suggests that the MBD children are not responding the same as the normals to the attentional demands of the situation. Finally, Sohmer and Student (1978) report normal threshold but larger latency for auditory nerve and brain stem evoked potentials to click stimuli in a group of 16 3-11 year-old MBD children compared to 18 4-10 year-old normal children.

Dyslexia. A number of investigators have reported differences in the AER of poor readers compared to normals. While most of the recent reports concern amplitude and latency changes, several reports

also look at latency differences between hemispheres. Ertl and Douglass (1970) measured VEP latency to light flashes from both cerebral hemispheres using a Neural Efficiency Analyzer (NEA). Latency was defined as the average time following the stimulus presentation to the second and third baseline crossings (zero voltage point) of the AER wave form. A group of nine reading disabled subjects were found to exhibit significantly above average right-to-left latency differences. Walsh (Ertl, 1975) is reported to have correctly identified 95 MBD subjects out of a group of 100 MBD and 100 normal subjects using NEA latency scores. Absolute hemispheric difference scores of 10 msec or more were used to differentiate MBD's from normals. No information on type of disability or ages of subjects was reported. In a study of 64 LD, 13 gifted and 13 normal children utilizing NEA absolute differences scores, Evans, Martin, and Hatchette (1976) found no significant hemispheric latency differences between the three groups of children nor between subgroups of the LD group. At present the question of hemispheric latency asymmetries and learning disabilities remains to be settled.

Conners (1970) compared 10 good and 10 matched poor readers for VEP activity to flash stimulation over occipital, parietal and central vertex cortical areas. A significant negative correlation was found between reading ability and the N200 component of the left parietal (P3) VEP. Similar left parietal findings were also noted in five out of six members of a family group suffering from reading disabilities, indicating a possible genetic dysfunction. The sixth member, the mother, had normal reading abilities. Conners' results

were confirmed in a study comparing nine 9-10 year-old reading disabled children to nine controls matched on age and IQ and nine controls matched on reading level and IQ conducted by Preston, Guthrie, and Childs (1974). Employing light flashes and the word "cat" as stimuli, the amplitude of the left parietal (P3) N180 component of the VEP to the light flashes was found to be significantly lower in the dyslexic group than for the controls. All three groups produced a greater negative response to the word stimulus. These investigators speculate that since words require more processing than light flashes, the amplitude of the left parietal area may be reflective of cortical information processing functions and that the decreased amplitude seen in the reading disabled group may be indicative of a neurological deficit. These authors do caution, however, that attentional differences between the three groups could not be ruled out in this study.

Left parietal and occipital (P3, O1) differences have also been reported for early components of the VEP between reading disabled and normal children by Symann-Louett, Gascon, Matsumiya, and Lombrosso (1977). Using three- and four-letter words, animal pictures or body part presentations, the dyslexic group displayed more late waves than controls although this difference was not statistically significant. Recording from CZ and OZ, Muso and Harter (1978) also report increased latencies, P300 (270-435 msec) in two 7-12 year-old dyslexic groups (auditory and visual perceptual difficulties) when compared to normals. Unlike Prichep et al. (1976) and Zambelli et al. (1977), however, they report that dyslexic and controls all showed P300 amplitude increases

to relevant vs. irrelevant stimuli using a standard CNV S1-S2 paradigm. Utilizing dim flashes embedded in a train of faster flashes, Sobotka and May (1977) found significantly greater amplitudes to unattended stimuli in both hemispheres for a group of 24 7, 9, 11 and 13 year-old dyslexics compared to a group of 24 matched controls. These differences occurred in the parietal areas for components P1-N1 and N1-P2 and in occipital areas for components N1-P2. AER differences between normal and MBD-LD children to relevant and irrelevant stimuli may represent the AER equivalent of the EEG changes associated with attentional deficits mentioned previously. A number of studies have shown the AER to be sensitive to attentional factors (see Hillyard, Picton, and Regan (1978) for a review) and Musso and Harter's (1978) failure to find differences between MBD and normal children along a relevant-irrelevant dichotomy may reflect procedural rather than underlying attentional mechanisms.

Medication studies. A number of studies have been conducted comparing the AER of MBD children before and after drug therapy. Generally, results show a "normalization" of the AER as a result of medication although several studies have been reported (Shagass, Ornitz, Sutton, and Teuting, 1978) in which no change or contradictory findings were obtained. Satterfield et al. (1972) using click stimuli compared 6-9 year-old good and poor hyperkinetic responders to methylphenidate hydrochloride (Ritalin) to each other as well as to a group of matched controls. During the pretreatment portion of this study, good responders showed a greater AEP amplitude at P60, N120, P180 and N280 than did poor responders. When put on medication best responders decreased AEP amplitudes to stimulation while poor

responders increased amplitudes. Pre-drug amplitudes of good responders and control were quite similar.

In a comparison of 24 MBD and 24 matched controls, Buchsbaum and Wender (1973) found amphetamine responders in a pre-drug condition to have above normal P200 latency increases with increasing stimulus intensities as well as faster rates of amplitude increases. Decreased N140 and P200 latency and rate of amplitude increase with increasing stimulus intensity was found for drug responders while nonresponders showed the opposite response. Conners (1972) found decreased VEP latencies at P180 and N240 to light flashes in amphetamine- and Ritalin-treated children with behavioral and/or academic problems. A placebo group from the same population did not show these changes.

Evoked Potentials, Attention and Learning Disabilities

It has been speculated that attentional deficits are the cause of many problems seen in learning disabilities (Rosenthal and Allen, 1978). During the last decade a number of theoretical approaches to information processing and attentional capabilities in humans have proposed that sensory inputs are first analyzed and processed through several stages before selection of behavioral or cognitive responses are made (Broadbent, 1970, 1971; Erdely, 1974; Kahneman, 1973; Treisman, 1969). The location at which selective attention occurs in this process has been subject to considerable debate. Disagreements have centered on what level in the central nervous system (CNS) filtering of information occurs and on what type of filtering is occurring. Generally, two theoretical models have developed involving either a serial processing approach or a multiprocessing model of information processing.

The serial model or single channel approach advocates an early stage in the sensory information processing channels as the location of the information reducing processor. This processor has been variously labeled a filter attenuator or input selector (Broadbent, 1971; Treisman, 1969). These selectors either completely or partially block out information to higher CNS centers from sensory channels competing with the attended channel. This filtering is based mainly on the physical attributes of various stimuli. The attenuation of nonattended stimuli is thought to be quite rapid, occurring very early in the information processing sequence and is analogous to the peripheral gating mechanism described by Adrian (1954), Broadbent (1958), Hernandez-Peon (1960) and Lindsley (1961).

The multi-processing approach uses a parallel processing model in which sensory information from all channels is allowed to reach higher CNS centers where decisions are made based on the relevance of the various stimuli being received (Deutsch and Deutsch, 1963; Moray, 1975; Norman, 1968; Schiffran and Grantham, 1974). In this model filtering occurs because of the information and attentional limitations of higher cognitive and memory functions. This type of information processing shows up later in the EP (> 100 msec) and may vary considerably based on the number and types of inputs and the decisions to be made.

The enumeration of the various considerations possible in describing a theoretical model for selective attention in humans helps to explain the differences in the view held by researchers in this

field. For instance, Näätänen (1975) states the goal for research in this area is "[the psychological and behavioral phenomenon of] selective attention based on selective filtering or blocking of sensory impulses" (p. 283). Parallel processing forms of selective attention are not considered valid by Näätänen. Studies relating selective attention to the later portions of the EP are thus recategorized by definition in that the changes are occurring too late to be related to attention. For Näätänen the maximum latency deriving from the activation of selective filters would be in the range of 20-40 msec. Thus, while admitting the general high quality of studies such as those by Hillyard, Hink, Schwent, and Picton (1973), Näätänen is able to criticize comments that the N2 component found occurring at 60-70 msec is analogous to Broadbent's (1970) stimulus set (selective filtering). Näätänen, citing his own work (1967) and that of Wilkinson and Lee (1972), feels that these early components are the result of "the non-specific reactiveness of the organism and the physical properties of the stimulus, but not its relevance or meaningfulness" (p. 294). The implication here is that higher cortical areas, i.e., a brief precategorical sensory store or perception, are being activated with attentional modulation following.

Researchers such as Callaway (1975), on the other hand, display a much more global view of attentional effects on the EP. Callaway's views concern the broader cognitive aspects of attention and the attentional components of such psychological constructs as arousal, expectancy, interest, uncertainty and informational context. Theoretically, Callaway appears to accept both the serial and parallel models of attention as indicated by his agreement with Hillyard et al.'s

(1973) conclusion that changes they found in early (N90) components of the EP represent stimulus set while later (P300) changes represent response set (higher cognitive processing) aspects of attention. In addition, Callaway takes a much more lenient view of the physiological representation of attention in the EP, allowing up to 200 msec for the simpler relevant modality recognition functions of attention and 200-400 msec when more complex cognitive processes are involved. Citing Picton and Hillyard's (1974) research on auditory EPs as supportive evidence, he discusses the probability that EP components before 75 msec do not show the effects of attention. In addition, Callaway (1975) cites several studies (Donald and Goff, 1971; Roth, Rothbart, Darley, Tinklenberg, and Kopell, 1974; Tueting, Sutton, and Zubin, 1970) showing the cognitive independence or disassociation of the P300 and CNV in the EP under various situations, thus indicating his belief that the later components of the EP can be related to attentional factors rather than to a post stimulus positivity resulting from the resolution of the CNV.

In order to assess the validity of the positions expressed by Callaway (1975) and Näätänen (1975), it is necessary to determine whether (a) the early and late changes seen in the EP can be dis-associated from each other, (b) short latency changes occur in the EP with manipulations of attention and the time at which these changes take place and (c) the later (P3) components of the EP can be separated from attentional factors related to prestimulus CNV activity.

Early, Middle and Late Latency Components

A number of investigators have recorded far field (e.g., brain stem, lemniscal pathways) EP's in order to test peripheral gating mechanisms (Debecker and Desmedt, 1968; Picton, Hillyard, Galambos, and Schieff, 1971; Picton and Hillyard, 1974; Velasco, Velasco, Machado, and Olvera, 1973). A study conducted by Woods and Hillyard (1978) is typical of many of the procedures assessing attention in the auditory pathway. Using dichotic listening procedures subjects were presented with verbal and click stimuli simultaneously to each ear and were instructed to attend to one or the other channel. Attend sides were counterbalanced and maintenance of attention was tested by questionnaire. Although subjects reported an attenuation of the unattended signal, brain stem evoked responses were not found to change significantly in latency or amplitude as a function of attention. In addition, midlatency (10-30 msec) responses were reported as not being systematically related to attention.

Using similar procedures, Hink and Hillyard (1976) report that middle wave correlates of selective attention (i.e., N2, 80-120 msec; P2, 160-200 msec) were enhanced by attention to relevant signals. Recording somatosensory evoked potentials (SEP) from depth electrodes in patients with Parkinson's desease, Velasco and Velasco (1975) report no changes in the early components localized to somatosensory thalamic areas. There were, however, marked changes in later EP components recorded from thalamic, reticular formation and cortical areas. Recording simultaneously from brain stem and cortical areas, Picton and Hillyard (1974) found no significant changes in brain

stem responses when click stimuli were attended to, but did find substantial enhancement in the N1-P2 components. Disassociation of N1 and P3 components has also been shown to occur between relevant and irrelevant targets (increased P3) of the same dimensions and channel (Courchesne, Hillyard, and Galambos, 1975; Picton and Hillyard, 1974) and when subjects select a target occurring between two different sensory channels (Hink, Hillyard, and Benson, 1978; Schwent, Snyder, and Hillyard, 1976). These findings suggest a bimodal distribution of selective attention in the CNS analogous to Broadbent's (1970) stimulus and response sets with the earliest effects occurring at latencies approximated by N1-P2 components of the evoked potential.

N1-P2 Components and Attention

A great deal of research has gone into the effort to identify mid-latency (80-200 msec) aspects of the EP with attention, as indicated by reviews such as Näätänen's (1975). In a number of studies not suffering from differential preparatory states of nonselective arousal or arousal due to general activation of non-specific portions of the nervous system, research has indicated little or no change in the N1-P2 components of the EP when attention was shifted between auditory channels (Smith, Donchin, Cohen, and Star, 1970; Wilkinson and Ashby, 1974), between auditory and visual channels (Hartley, 1970; Näätänen, 1967) and within auditory channels when attention was shifted between auditory and visual channels (Schechter and Buchsbaum, 1973). This last study did find up to a 53% increase in the P100-N140 component when attention was shifted to visual channels. However, this finding

is open to criticism since eye blinks and eye position were not monitored. Needless to say, no such criticism applies to the auditory channels in this study.

In discussing these findings it is necessary to consider the information rates presented to the subjects. Many of the studies failing to find N1-P2 changes related to attention used long inter-stimulus intervals (ISI's). The minimum time between presentations in the studies just discussed was 1 sec. Recently several authors have suggested that low information rates resulting from long ISI's or too easy a task may result in little or no activation of selective attention (Hartley, 1979; Schwent, Hillyard, and Galambos, 1976a; Shiffrin and Grantham, 1974). If such is the case, shortening ISI's or increasing task difficulty should result in positive indications of selective attention.

In a series of experiments utilizing auditory channels, Schwent and his associates (Schwent and Hillyard, 1975; Schwent et al., 1976a, 1976b) manipulated stimulus presentations in order to test the effects of information loading on the EP. They found that as the ISI decreased, or as the number of channels to be monitored increased, the amplitude of the N1 component of the EP was greatly enhanced as attention was directed towards relevant stimuli. Similar findings of N1 enhancement have also been reported for somatosensory and visual channels by Desmedt and Debecker (1979); Harter and Previc (1978); Hillyard and Picton (1979); and Hink, Van Voorhis, Hillyard, and Smith (1977). It appears that under conditions of high information load during which

stimulus classes are separated by physical cues which allow for simple and rapid analysis, the N1 component of the EP can be considered to be a sign of selective attention (Hillyard, Picton, and Regan, 1978, p. 288).

Late Components and the CNV

Prior to Näätänen's (1975) paper, opinion was divided on the relationship of the CNV and the post imperative or second stimulus late (P3) components of the EP. This division centered on whether the P3 components were the result of CNV rebound (Karlin, 1970; Wilkinson and Ashby, 1974) or whether they represented different aspects of attention (Friedman, Hakerem, Sutton, and Fleiss, 1973; Lombroso, 1969; Teuting and Sutton, 1973). Since that time a number of reports, with the exception of Wilkinson (1978) and Näätänen (Näätänen and Miche, 1979; Näätänen, Gaillard, and Montysalo, 1978) have expressed the opinion that CNV waves and later EP events represent different aspects of selective attention. Donchin, Teuting, Ritter, Kutes, and Heffely (1975), for instance, report that CNV and P300 distributions over the scalp differ, indicating that the CNV and P300 are generated over different populations of neurons. However, the spread of CNV activity to P300 generator cells may still be a factor in P300 activity.

In a study designed to assess the relationship between P300 and CNV, Desmedt and Debecker (1979) presented equiprobable sequences of auditory and somatosensory stimuli over ISI's varying at random between 1 and 15 seconds. This uncertainty of target intervals

resulted in a large P350 wave in the absence of any CNV to target stimuli. P300 was missing from nonrelevant trials. The authors stressed that no CNV or slow potential shift occurred either before or after the stimuli, indicating that the CNV and P300 react differentially to experimental variables and that the P300 can occur in the absence of any prestimulus CNV. Recent evidence for the dissociation of CNV and P300 waves has also arisen from a number of other sources (Chapman, McCrary, Bragdon, and Chapman, 1979; Donchin et al., 1975; Leifer, Otton, Hart, and Huff, 1976; Peters, Billinger, and Knott, 1977; Poon, Thompson, and Marsh, 1976; Ruchkin and Sutton, 1979) under a variety of experimental conditions. At present it would appear that the majority of the evidence points to the CNV and P300 components of the EP as being differential indications of cognitive process and that the EP can be an important tool in the exploration of cognitive process in humans.

EP and Specific Learning Disabilities (SLD)

The relationship between attentional aspects of the EP and SLD's arises from reports of "attentional deficits" in children labeled as learning disabled and particularly as hyperactive (Conners, Rothschild, Eisenberg, Schwartz, and Robinson, 1969; Douglas, 1972; Dykman, Ackerman, Clements, and Peters, 1971; Halliday, Rosenthal, Naylor, and Calloway, 1976; Naylor and Callaway, 1976; Satterfield, 1973). Several researchers have looked at attentional mechanisms in MBD children using AER responses to drug treatment. Klorman, Salzman, Pass, Borgstedt, and Dainer (1979) compared 18 normal and 18 hyperkinetic boys in a drug-

placebo counterbalanced design. Children were tested during a passive phase in which they were presented the letters X, B, S, D, W or T and during an active phase in which they were required to press a button each time an X appeared. No differences between the experimentals and controls were found during the passive phase. During the active phase the placebo group showed a decrease in amplitude of the late positive component (200-550 msec) when compared to normals. Treatment with methylphenidate produced an increase in amplitude of the late positive component of the hyperactive children. Halliday et al. (1976) found hyperkinetic children responding to methylphenidate to have smaller CZ VEP (N145-P190) amplitudes to light flashes before treatment when compared to nonresponders under conditions of attention. Under the same conditions these amplitudes increased when responders were given methylphenidate. Prichep, Sutton, and Hakerem (1976) found the P186 AEP amplitude to clicks to be increased and the N250 amplitude to be decreased in a group of hyperkinetic children when given methylphenidate. These changes produced amplitudes more like those seen in control groups. In addition, AEP differences between conditions of uncertainty and certainty became greater when children went from a placebo to a drug situation. Both Prichep et al. (1976) and Halliday et al. (1976) feel that such differences represent attentional differences between MBD and normal children and that methylphenidate has a normalizing effect on attentional responses.

In an interesting study utilizing dichotic listening tasks in 12-14 year-old hyperactives and age-matched controls, Loiselle, Stamm, Maitinsky, and Whipple (1980) found significant enhancement for the

N100 amplitude of the auditory EP for controls but not for the clinical group. P300 latencies and amplitudes were also found to be significantly greater for the control children but not the hyperactives and P300 but not N100 amplitudes were significantly correlated with correct detection of relevant stimuli. The authors suggest these findings indicate severe selective attention dysfunctions for the clinical group involving both stimulus and response sets. It appears that disturbances of selective attention may be an underlying factor in disorders associated with SLD's and that the EP can supply valuable information about attentive capabilities as well as assess some of those abilities more sensitively than currently available behavioral tests (Hillyard et al., 1978).

In summary, there are a number of different electrical patterns obtained from the cortex which indicate differences in brain function between different populations of MBD children and normal children. In the EEG these patterns are represented by changes in frequency response to various stimuli, hemispherical asymmetries, abundances of delta and theta activity and inappropriate activity in the higher alpha and beta ranges. Evoked potential changes, on the other hand, are shown by amplitude and latency differences. In general, MBD children seem to have lower amplitudes and longer latencies to stimulus presentations. However, a wide variety of various AER differences have been noted as differentiating MBD from normal children. At present it is difficult to come up with definitive descriptions of electrical

patterns representing specific learning disabilities. A number of factors such as age of subjects (see Shagass, Ornitz, Sutton, and Teuting, 1978), group compositions, procedural differences and topographical locations must be given more careful consideration and standardization before a more precise understanding of brain function and learning disabilities can be achieved. It does appear, however, that MBD and normal children do not process information in the same manner and that these differences may be reflected in the electrical patterns of brain activity recorded from these children.

Cortical Self Control

Perhaps the earliest report on the ability of an individual to voluntarily control electrocortical activity was presented by Kamiya (1962). In 1968 Kamiya reported a series of studies involving alpha enhancement and discrimination. Utilizing auditory feedback, subjects were able to voluntarily increase or decrease levels of cortical alpha activity at their discretion. In addition, Kamiya (1968) noted that between 80 and 90% of the subjects could discriminate alpha and non-alpha activity in the absence of feedback. In a second study, Kamiya (1968) found that subjects were also able to shift their dominant alpha frequency by 10 to 12% from its pretraining location when provided with feedback.

Epilepsy

Since Kamiya's studies (1962, 1968), a growing amount of data has been accumulating on the self control of brain electrophysiology for use in behavior modification and therapeutic treatment. In 1972

Sterman and Friar reported a reduction in seizure activity following sensorimotor (SMR) EEG feedback training in humans. Sterman (1973) reported on four cases in which epileptic patients were given biofeedback training to increase SMR activity in the central rolandic area of the brain. Inhibit circuitry was used which denied patients feedback for slow, epileptiform EEG activity as well as a lack in production of SMR rhythms. Sterman reports all four patients reduced seizure frequency and increased seizure-free periods. In addition, significant changes in EEG activity occurred with reductions in slow wave and spike activity and increases in SMR densities.

Similar decreases in seizures have been reported by Finley, Smith, and Etherton (1975) and Lubar and Bahler (1976). In a double blind crossover study designed to determine the effectiveness of operant conditioning of the EEG as an anticonvulsant, Lubar, Shabsin, Natelson, Holder, Whitsett, Pamplin, and Krulikowski (1981) found supression of low (3-8 Hz) activity as well as enhancement of 12-15 Hz rhythms effective in reducing epileptic seizures. A noncontingent and reversal phase were also employed in this study. Following a baseline period, the eight patients involved in this study were given feedback having no relation to their actual EEG patterns in order to assess any placebo effects of participating in the study. A therapeutic training phase followed next after which a reversal phase occurred in which patients were given feedback for activity exactly opposite to that in the preceding phase. This was followed by a final therapeutic training phase. No systematic change in seizure activity was observed during the noncontingent portion of the study. During the reversal portion of the study a number of patients dramatically increased their seizure

rates. Overall, these authors report a 35% seizure reduction among the eight patients as a result of changing EEG patterns. Interestingly, the patient who showed the greatest increase in 12-15 Hz activity also showed the greatest improvement in seizure reduction.

Kuhlman and Kaplan (1979) have described two approaches to explaining how EEG feedback can reduce seizures. The first, the neural exercise model, involves long-term changes in EEG functioning having anticonvulsant properties obtained through exercise. The second, the voluntary control model, is based on learned discrimination of EEG states which can be employed in a phasic manner to abort seizures but does not involve any permanent EEG alterations. These authors suggest that most of the evidence so far favors the neural exercise model. Sterman (1973) has proposed that long-term EEG training for increases in SMR activity leads to neural reorganization and selective overstimulation of inhibitory motor pathways, resulting in increased seizure thresholds. Based on conditioning studies of single cortical cells in monkeys, Wyler, Fetz, and Ward (1974) proposed that seizure generation required a critical number of neurons discharging in synchrony and suggest that EEG training might be useful in desynchronizing seizure prone neurons before they triggered an epileptic event.

Hyperactivity

In a series of reports (Lubar and Shouse, 1976, 1977; Shouse and Lubar, 1979), EEG training has been described as helpful for some of the problems associated with hyperactive children. In an investigation involving 12 normal children and 12 hyperactive children,

Lubar and Shouse provided SMR biofeedback training to four of the hyperkinetic children distinguished by the most pronounced classroom misconduct combined with the greatest deficiencies in SMR and general physiological arousal levels as reflected by auditory evoked potential, galvanic skin response and electromyographic recordings. These children were classified as low arousal hyperkinetics (Satterfield et al., 1972) and treatment was given over 160 training sessions involving two 5 minute baseline periods preceding and following two 15 minute SMR feedback trials. The design was an A B A C one in which A represents Ritalin plus SMR, B represents Ritalin plus SMR reversal training and C represents no drug plus SMR training. The authors report that eight of 13 behavioral measures of overactivity and distractability taken in the classroom improved as SMR acquisition occurred in three of the four children. The child that failed to exhibit any behavioral changes also failed to show any SMR enhancement. Lubar and Shouse (1977) found SMR training to be more effective for the overactive problems of hyperactivity than for attentional deficits and suggest that EEG biofeedback is an effective technique for dealing with hyperkinesis, especially since they found the combined effects of SMR plus Ritalin resulted in improvement beyond drugs alone.

Learning Disabilities

Recently, several authors have reported on the effects of EEG biofeedback with learning disabled children. In a small study utilizing four LD boys with WISC-R verbal and performance IQ discrepancies of 20 points or greater, verbal IQ low, Carter and Russell (1981a) found that alternating production of alpha (8-13 Hz) and beta

(13-28 Hz) activity in the left hemisphere using EEG biofeedback resulted in a 12 point gain in verbal IQ and a decrease in verbal-performance IQ differences. In a follow-up study, Carter and Russell (1981b) chose 20 LD boys again with 20 point or greater WISC-R verbal-performance discrepancies. Subjects were separated into two equal groups, one with verbal IQ higher than performance IQ and the other with performance IQ higher than verbal IQ. Subjects from each group were then randomly placed in either right hemisphere or left hemisphere training groups. Training consisted of two 30-minute sessions per week for eight weeks. Results after training indicated significant increases in verbal scores for verbal low left hemisphere trained but not right hemisphere trained subjects. The opposite results were found for performance low subjects. For this group, only right hemisphere training significantly increased performance IQ scores. Carter and Russell suggest that in normal children each hemisphere shifts from a resting alpha state to a more activated beta state as needed for varying tasks. They state that children having difficulty making this shift will have problems doing certain activities and suggest that EEG biofeedback enables children to reorganize and control specific brain patterns and learn to produce the mental state associated with that pattern. They assume that inefficient learning is a result of a significant verbal-performance IQ discrepancy and that if that difference can be decreased, learning efficiency in children will be increased.

Lubar (1980) has reported on a number of cases treated using EEG biofeedback in conjunction with a number of remediation tasks.

Children were trained to increase 12-15 Hz rhythms while simultaneously decreasing 4-8 Hz activity. Fourier analysis done at varying intervals showed systematic changes occurring in the EEG over the training period associated with the feedback protocol. Besides the changes seen in the EEG, Lubar also reports concomitant improvements in academic performance by children involved in the training. Additional support for the use of EEG biofeedback comes from a study by Schwartz, Davidson, and Pugash (1976). They found that normal subjects trained with biofeedback to produce asymmetrical hemispheric activation of alpha (8-13 Hz) reported verbal or visual cognitions as alternate on-off patterns of hemispherical alpha activation were produced. The authors state that these two cognitive modes are the ones most often associated with either left or right hemispherical EEG activation.

Mechanism

The exact neural mechanism by which EEG modification might alter behavior or change performance characteristics has not been elucidated even though a number of theories have been proposed. In 1950 Lashley used the term neural engram to describe patterns of electrical and cellular activity in the brain where experience and information are stored. Hebb (1949) proposed that individual experiences are coded in the brain in patterns of nerve cells called cell assemblies. Through experience a number of these cell assemblies could become organized into large neural patterns called phase sequences. Essentially, both Lashley's (1950) and Hebb's (1949) proposals suggested that the cellular connections of the brain could be altered by experience.

Lynch and Wells (1978) offer some possible explanations as to how experience and learning may affect brain activity. These can be summarized as follows:

1. Cortical growth. Studies employing enriched environments have found animals to show thicker and heavier cerebral cortexes. This effect has also been found to be reversible if the environments are impoverished.

2. Dendritic growth. Restriction of sensory input has been shown to affect the number of dendritic spines. Dendritic size, shape and configuration are affected in both adult and juvenile animals. Stimulation studies have also been found to change dendritic structure. It appears that mature dendrites are not immutable but instead depend on external input for their maintenance and retain considerable capacity of growth.

3. Axonal plasticity. Most studies utilizing degenerative techniques have shown that synaptic terminals can form new connections and that it is also possible that an axon may generate new terminals. In addition, data indicate that new growth in both immature and mature animals is accompanied by the development of operative circuitry. Although there is not much evidence, there is some indication that a fair amount of axonal turnover occurs under normal conditions (Burgess, 1973). Sotelo and Palay (1971) report that degenerating endings are frequently found in normal brains and are often accompanied by what appear to be axonal growth cones. They view degeneration and sprouting of axon terminals as part of the regular activity of the brain. Lynch and Wells (1978) speculated that this might be

the mechanism through which the brain responds to the behavioral activities of the organism and allows new cortical patterns to appear.

Rationale for this Study

Evoked potentials are rapidly becoming an accepted technique for investigating and diagnosing psychophysiological problems in a number of areas (Lubar and Deering, 1981; Shagass et al., 1978). Among other problems, alterations in brain function associated with learning disabilities are reflected in evoked potentials recorded from a number of cortical locations (Duffy et al., 1980). Based on these observations, a reciprocal relationship might exist in which alterations of evoked potentials might be reflected in changes in behavior.

Several studies have indicated that the human cortical evoked potential is receptive to voluntary control. Rosenfeld, Rudell, and Fox (1969) reported that subjects could modify the N200 peak of their auditory evoked potential when provided with feedback. In a more elegant design, Roger and Galand (1981) found that subjects were able to control polarity and amplitude voluntarily in nine different segments of their visual evoked potential. Both of these studies controlled for peripheral mediation and the authors suggest that changes in wave forms were the result of variations in size or populations of neuronal pools. Subjects reported using various cognitive strategies such as imagery or vigilance to produce changes in their evoked potentials.

A provocative report on the relationship between changes in the evoked potential and behavior comes from Lubar and Shouse (1977).

In a study designed to assess the effects of EEG training on hyperactive boys, they found spontaneous changes in the auditory evoked potentials of subjects who acquired the EEG task and whose behavior improved. This finding in conjunction with the number of neural correlates of learning disabilities reflected in the evoked potential leads to speculation as to what effect changes in the evoked potentials of learning disabled children might have on their academic difficulties. This study was planned in order to help assess this question. Specifically, this study addresses three questions:

1. Can children learn to control components of their visual evoked potential? No studies are known to date which have addressed this problem in children.

2. If learning disabled children can be taught to alter their evoked potentials, will such changes have any effect on academic performance as measured on a number of neurological and psychoeducational assessments?

3. Will alterations in evoked potentials have any effect on the spontaneous EEG recorded from a number of locations on the scalp under different conditions?

If certain components of the evoked potential can be manipulated in a specific fashion such that changes seen in the EP correlate with improvements in academic performance, then EEG biofeedback may provide a suitable avenue for some LD children to improve their learning skills.

CHAPTER II

METHODS

Subjects

Initially, participants in this study included 14 learning disabled and 10 normal male children between the ages of eight and 12 years. Prior to the beginning of the study, the LD children were randomly divided into a treatment group consisting of six children and a control group consisting of eight children. Of these children three from the control group were dropped from the study for failing to participate in the post testing session and one from the treatment group was dropped because of geographical relocation. This resulted in a total of 10 LD children participating in the study. The mean age of the children was 10 years 7 months for the treatment group, 10 years 6 months for the control group and 10 years 8 months for the normal group.

The learning disabled children selected to participate in the study were chosen according to the following criteria at the time of the study:

1. Belonged to lower middle class to upper middle class families in terms of socioeconomic status.

2. Were currently diagnosed as learning disabled as a result of psychoeducational assessment by their school psychologist.

3. Were currently participating in remedial resource programs at their schools.

4. Were not receiving therapy, counseling or any other form of specialized services outside the school system.

5. Showed no indications of any history of seizures, hyperkinesis, brain injury, articulation disorders or other severe handicap.

6. Were not on any medication regime prescribed for the conditions listed in Item 5 above.

7. Obtained evaluations on the Weschler Intelligence Scale for Children-Revised (WISC-R) not more than two standard deviations below the mean for full scale score.

8. Scored within or bordered on the LD category (between 18 and 43 points) on the Selz and Reitan (1979) scoring system for the Halstead-Reitan Neuropsychological Battery. Because the Selz and Reitan scoring system is relatively new, it is reproduced in Table 1.

9. Parent willingness to provide transportation for their child and to continue to participate in the study if their child was selected to participate in the LD control group. Parents were informed at this time that their child would be provided with the same type of treatment that the experimental group received if he was placed in the control group. All control children of parents so desiring it were provided with such treatment at the end of the study.

10. Children were residing in the greater Knoxville, Tennessee area and there were no plans for the family to move out of the area during the next year.

The normal children participating in the study were selected on the following criteria:

Table 1. Selz and Reitan Scoring System.

Test		0	1	2	3
Level of Performance					
1.	Category-errors	34 or less	35-55	56-74	75+
2.	Tactual Performance Test (TPT) - total time	less than 6'	6-9.9'	10.13.9'	14'+
3.	TPT-memory	6, 5	4	3	2, 1, 0
4.	TPT-localization	3+	2	1	0
5.	Trails A-time	15" or less	16-25"	26-35"	36"+
6.	Trails B-time	39" or less	40-55"	56-70"	71"+
7.	Speech-errors	10 or less	11-15	16-20	21+
8.	Rhythm-correct	25+	21-24	16-20	15 or less
9.	VIQ	90+	80-89	70-79	69 or less
10.	PIQ	90+	80-89	70-79	69 or less
11.	FSIQ	90+	80-89	70-79	69 or less
12.	Tapping, preferred hand - # taps	34+	30-33	26-29	25 or less
13.	Tapping, non-preferred hand - # taps	30+	27-29	23-26	22 or less

Pattern: Extreme scatter on the subtest scores on the Wechsler scale is abnormal. The following conversion measures the degree of scatter: (largest subscale score - smallest subscale score) ÷ mean of scores.

14.	Pattern IQ	.99 or less	1.00-1.40	1.41-1.75	1.76+

Right-Left Differences: The following tests compare performance of the right and left hands. Ratios for #15-17 are derived from this formula: 1 - (nonpreferred hand ÷ preferred hand). The scores in #18 and 19 are derived from the conversion formula presented in Table 2.

Table 1 (Continued).

Test		0	1	2	3
15.	Tapping	.04-.16	.03-(-.15) .17-.30	-.16-(0.25) .31-.40	-.26 or less .41+
16.	Grip	0.0-.20	-.01-(-.06) .21-.26	-.07-(-.12) .27-.32	-.13 or less .33+
17.	TPT	.11-.49	.10-(-.05) .50-.65	-.06-(-0.20) .66-.80	-.21 or less .81+
18.	Name writing - preferred hand (converted score- see Table 2)	10, 8	6, 4	2	0
19.	Name writing- difference (converted score- see Table 2)	6-10	4	2	1
20.	Tactile finger recognition. Right hand errors Left hand errors	0, 1	2	3	4+
21.	Finger-tip number writing. Right hand errors Left hand errors	0-2	3-4	5-6	7+

Pathognomonic Signs: For these tests, normal performance consists of perfect performance. Allowance was made for the fact that even normal children tend to make more errors than adults.

22.	Imperception-errors	0	1	2	3+
23.	Tactile Finger recognition-errors	0-3	4-5	6-8	9+
24.	Finger-tip number writing-errors	0-7	8-10	11-14	15+
25.	Tactile form recognition-errors	0	1	2	3+

Table 1 (Continued).

Test	0	1	2	3
	Aphasia Battery		Score for Deviant Performance	
26.	Constructional Dyspraxia		2	
27.	Dysnomia		3	
28.	Spelling Dyspraxia		1	
29.	Dysgraphia		2	
30.	Dyslexia		2	
31.	Central Dysarthria		2	
32.	Dyscalculia		2	
33.	Right-Left Confusion		1	
34.	Auditory Verbal Dysgnosia		3	
35.	Visual Number Dysgnosia		3	
36.	Visual Letter Dysgnosia		3	
37.	Body Dysgnosia		3	

Source: M. Selz and R. M. Reitan. Rules for neuropsychological diagnosis: Classification of brain function in older children. Journal of Consulting and Clinical Psychology, 1979, 47(2), 258-264.

1. Were not experiencing any academic or behavior problems in their school settings.

2. Were not on any medication for and had not experienced any history of seizures, hyperkinesis, learning disability, brain injury, articulation disorder or other severe handicap.

3. Scored within the normal intelligence range on the WISC-R full scale score.

4. Had scores on the Selz and Reitan scoring system between 0 and 19.

5. Had parents willing to bring them to both the pretest and posttest evaluative sessions.

6. Their families had no plans for relocating during the following year.

Procedures

Psychoeducational and Neuropsychological Measures

Prior to treatment and again following treatment, all subjects were administered the following tests utilizing standard testing procedures: Wechsler Intelligence Scale for Children-Revised (WISC-R), Wide Range Achievement Test (WRAT) (word recognition, spelling and arithmetic), Spache Diagnostic Reading Scales (oral and silent reading levels), Bender Gestalt Designs and the Halstead-Reitan Neuropsychological Battery. All tests were administered by one of two individuals competent in testing procedures. These testers had no other connection with the study and were unaware of which of the three groups a child being tested belonged to. Changes in test scores between pretest and

posttest conditions were used as one of two dependent variables to measure treatment effects.

Electroencephalographic Measures

The second dependent measure utilized to assess treatment effects consisted of electroencephalograms (EEG) recorded from each of 16 locations on the scalp prior to and again following the training program. Silver disk electrodes (Grass Instrument Co. No. E5SH electrodes) were applied in eight bipolar derivations using electrode paste (Grass Instrument Co. EC2 Electrode Cream). Electrodes were positioned according to the International 10-20 System and consisted of the following bipolar pairs: F3-F7, F4-F8, C3-T3, C4-T4, O1-P3, O2-P4, F7-T5, F8-T6. In addition to the EEG, electromyographic (EMG) activity was recorded from the right frontal region using two of the 16 EEG electrodes in that region. All electrode impedances were below 10 thousand (K) ohms and typically below 5 K ohms.

The EEG was recorded in a sound attenuated, electronically shielded room while the subject sat in a comfortable lounge chair. Each subject was asked to perform three 5-minute tasks. During this time the EEG data were recorded on magnetic tape using a Teac R-7 FM 4-channel tape recorder for off line analysis at a later time. Data were recorded from one hemisphere at a time after which the three tasks were repeated and data from the opposite hemisphere were recorded. EEG data were amplified by two Grass Instruments Co. P5H11 preamplifiers and six preamplifiers built to specifications by a student in the Electrical Engineering Department at The University of Tennessee, Knoxville. The two Grass preamplifiers had half

amplitude filter settings at 1 and 100 Hz; 60 Hz notch filters were used. The remaining six preamplifiers had high pass filters set at .5 Hz and low pass cutoffs set at 30 Hz. Because of the 30 Hz low pass filtering, no 60 Hz notch filters were used.

The activities the children performed while their EEG was being recorded included a baseline task, a reading task and a figure drawing task. For the baseline situation each subject was instructed to remain as relaxed as possible with his eyes open. The reading task consisted of the subjects reading silently at the instructional level from graded Reader's Digest books. During the drawing task each child was asked to copy designs from Berry's Developmental Test of Visual-Motor Integration. The children were instructed to remain as still as possible for each of the situations. In order to facilitate relaxation, the subjects were provided with a red light which was activated whenever right frontal EMG levels exceeded 12 microvolts (uv) of amplitude as measured peak to peak.

Off line analysis of the EEG data was performed by a Digital Equipment Corporation PDP 11-04 computer with 32 K words of memory. Data were fed to the computer from FM tape, digitized and subjected to Fast Fourier Transformations (FFT). The FFT analysis produced tables of percent and power values of the EEG, broken down into 4 Hz frequency bands between 0 and 28 Hz. This resulted in seven frequency bands each for percent values and power values. In addition, a special 6-10 Hz band was also analyzed for both power and percent tables and a total power band was analyzed. Power values were in relative pico watts of amplitude and percent values represented a

specific frequency band's portion of the total power. In addition to tables, graphical representations of Fourier transformed EEG were also obtained on a Houston Instruments DP-1 Digital Plotter.

Treatment

Treatment for the experimental group consisted of two visual evoked potential (VEP) training sessions per week for a total of 30 complete sessions. Sessions in which the subjects were sleepy, exhibited excessive movement or failed to attend to the task were not counted as completed sessions. Each subject was monitored by a trained observer and verbal reports were obtained from each child at the end of a session. Subjects were considered sleepy if their eyes remained closed for four consecutive VEP trials and were scored as inattentive to the task if they were observed to be looking away from the VEP target for more than four consecutive trials. Subjects' self report of sleepiness or uninvolvement in the task was also used in scoring the subjects' attentional state. Subjects were considered to be exhibiting excessive movement if any portion of a session could not be completed in 15 minutes or if they had to be cautioned by the observer more than twice about remaining still. Children were cautioned if they were moving for two consecutive VEP trials.

The VEP was recorded using two Grass Instrument Co. No. E5SH silver electrodes placed at International 10-20 System locations F_z and C_z. Electrodes were placed on the scalp using Grass Instrument Co. EC-2 Electrode Cream in monopolar configuration referenced to

right ear lobe. The VEP was recorded from subjects seated in a comfortable lounge chair located in an electrically shielded, sound attentuated room. Visual stimuli appeared on a Tektronix 935 oscilloscope screen placed directly in front of the subjects. The generation and presentation of the VEP stimuli as well as the collection and analysis of the data were controlled on line by a Digital Equipment Corporation PDP 11-04 mini computer. Input to the computer was provided by two Grass P5H11 Preamplifiers. Ambient room temperatures were maintained throughout the study and average room lighting was maintained at 10 footcandles. Sixty-five decibels of white noise was provided throughout the procedure. The intensity of the VEP stimuli was maintained at plus four dial units from the lowest setting on the display oscilloscope. Light levels were obtained using a General Electric light meter, type 214 and noise levels were measured using a Realistic Corporation sound level meter.

In addition to VEP data, electromyographic (EMG) and electro-ocular (EOG) information was also collected and analyzed by the PDP 11-04 computer. EMG activity was obtained from the C_z scalp electrode and EOG activity was collected from a pair of electrodes placed 1 inch above and to the side of the right eye and 1 inch below and to the side of the left eye. The computer was set to record more than 10 microvolts (uv) of EMG activity and 60 uv of EOG activity. EMG and EOG levels were monitored in order to help control for muscle and eye movement contamination of the VEP during baseline and training sessions and to help prevent trivial mediation of the VEP by these sources during training sessions.

Prior to the beginning of the treatment period for the experimental group, each child was given individual instruction on the procedure to be used. During this instruction the experimenter sat in the room with the child for two practice sessions of 50 trials each. During the first session the experimenter explained the procedure to the child, answered his questions and guided him through the 50 stimulus presentations. Guidance consisted of instruction and comments concerning body movement and position, muscle activity, relaxation and eye movements. During the second training session the child received guidance from the experimenter only when needed to elucidate proper procedures. Following these two training sessions, each child participated in eight baseline sessions in which no feedback was provided. The first four baseline sessions were counted as practice and the last four sessions were used to obtain baseline values from each child.

The procedure for the collection of the VEP was as follows. Once the children were seated in front of the display screen, they were instructed to maintain a relaxed bodily state, to face the screen in front of them and told that the procedure would begin momentarily. The experimenter or observer then left the room and the children were observed through a one-way glass. Once the procedure began, a small cross of luminance and brightness equal to the stimulus appeared on the screen. The cross was to inform the subject that a stimulus was about to appear and to provide a fixation point. The cross remained on the screen for a minimum of 3.25 seconds after which time a

stimulus appeared. The stimulus consisted of a random presentation of .50 inch letters of the alphabet. EEG data were collected for 750 milliseconds after the appearance of the stimulus.

The procedure outlined above was identical for both baseline and treatment sessions. Following the presentation of the stimulus during baseline, the screen remained blank for 5 seconds at which time the sequence of cross and stimulus was repeated. During training sessions a series of zero to eight stars appeared on the screen in a bottom left to top right diagonal following the disappearance of the stimulus. Stars represented feedback to the subjects for specific changes in VEP activity. The presentation of feedback was followed by a 5 second blanking of the screen after which the sequence was repeated. This sequence of presentations is illustrated in Figure 3.

The feedback provided to each child was based on the difference between the amplitude of the P300 peak recorded during the first baseline condition of each session and the highest amplitude recorded from the feedback session within a ±30 msec latency range of the P300 baseline latency. After the first baseline of each training session session the standard deviation of the P300 peak was calculated and used to determine the amount of feedback provided during the training conditions. Subjects received one feedback star for each .10 to .15 of a standard deviation increase in the trained P300 amplitude over the first baseline P300 amplitude. Subjects were paid one cent for each five stars they obtained. In order to help avoid large fluctuations in the amount of feedback provided during training a running weighted average was used to produce feedback for each trial. Feedback for increases in P300 amplitude was thus based on the calculated average

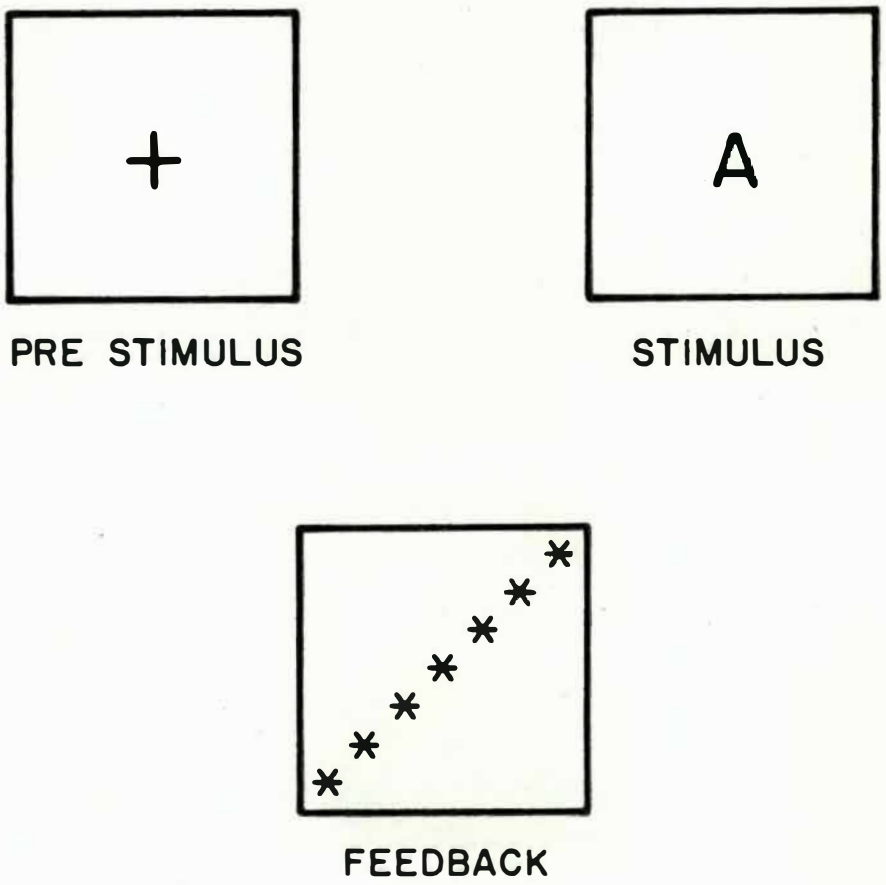


Figure 3. Display sequence for stimulus presentation and feedback.

of any particular trial plus the two preceding trials. No feedback was provided for the first three trials of any training session.

Baseline sessions consisted of trials of 75, 50 and 35 presentations with rest periods in between. Treatment sessions consisted of a 50-trial baseline portion, followed by two 40-trial feedback trainings, followed by a 35-trial baseline portion, with rest periods in between the different portions of the session. Sessions lasted an average of 45 minutes. During all trials for both baseline and training sessions, eye movements and muscle tension levels were monitored by the computer. If EEG artifact from these sources was detected prior to a stimulus presentation, the stimulus was delayed until 3 seconds of artifact-free EEG had occurred. If EMG or EOG artifact occurred during a stimulus presentation, that particular trial was dropped from the average for that portion of a session. Thus, the sessions were self-paced with each portion of a session ranging from 7 to 15 minutes. In addition, the number of trials in an averaged evoked potential was not always the number presented. Averages were typically within two trials of the number of presentations for any portion of a session. Four baseline sessions were administered to each child following the last training session. Postbaseline sessions were identical to prebaseline sessions. Electrode impedance for all sessions was 10 K ohms or less. No treatment was provided for either the LD control group or the normal comparison group although the LD control group, like the LD treatment group, continued to receive resource training in the schools.

CHAPTER III

RESULTS

Neuropsychological Data

Halstead-Reitan Neuropsychological Battery: Selz and Reitan Score

Table 2 shows the mean Selz and Reitan scores for both pretesting and posttesting situations for the treatment, LD control and normal comparison groups. These figures represent the range of neuropsychological impairment with a score of 0-19 defined as normal, 20-35 as learning disabled and a score greater than 35 as brain damaged. Table 2 indicates that all three groups showed improvement in the neurological impairment index with the greatest gain being shown by the treatment group followed by the LD control group. The normal comparison group showed the smallest change of the three groups.

Table 2. Mean Selz and Reitan scores for treatment, LD control and normal comparison groups.

	Treatment (n=5)	LD Control (n=5)	Normal Comparison (n=10)
Pretest	28.4	31.00	11.3
Posttest	17.2	23.20	10.1

In order to assess the significance of these changes, Tukey's Honestly Significant Difference (HSD) was applied in a planned pairwise comparison fashion. Table 3 shows the mean change score differences on the Selz and Reitan Index for the three groups. For this and all other comparisons using Tukey's HSD in this section the .05 level of significance for a two-tailed test with 3,17 degrees of freedom equals 3.68. The n" value for comparison of the two LD groups equals 5. The n" value for all other comparisons equals 6.66. Tukey's HSD found no significant difference at the .05 level between the two LD groups nor between the LD control and normal comparison groups. However, differences between the treatment group and the normal comparison group were found to be significant at the .025 level.

Table 3. Mean change scores for Tukey pairwise comparisons on Selz and Reitan evaluation for treatment, LD control and normal comparison groups. *equals .025 level of significance

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	-10.60*	-7.80	-1.4	
δ	5.72	5.12	6.45	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

Bender Gestalt Designs

Table 4 shows the mean error scores obtained on the Bender Gestalt test. Errors for the control and comparison groups remained essentially constant between testings while errors decreased for the treatment group. The Koppitz scoring system (Koppitz, 1963) was used for evaluating subjects' production of the Bender Gestalt designs. Mean change scores for the three groups are shown in Table 5. Using Tukey's HSD, no significant differences were found between the three groups.

Wechsler Intelligence Scale for Children-Revised (WISC-R)

The mean intelligence quotients (IQ) scores for the WISC-R full scale, verbal IQ and performance IQ for all three groups can be seen in Table 6. This table shows that verbal IQ decreased 3 points between testings for the treatment group and increased 5.2 points for the LD control group and 1.3 points for the normal comparison group. Performance IQ increased for all three groups with the largest changes being seen in the LD control group, followed by the treatment and comparison groups. Full scale IQ scores also increased most for the control group but were followed by smaller changes in the comparison and treatment groups in respective order.

Tables 7, 8 and 9 show the mean change scores for the Tukey HSD test for planned comparisons for the verbal IQ, performance IQ and full scale portions of the WISC-R. The results from the statistical analysis reveal no significant differences for changes in the verbal IQ score between the three groups. Comparisons of changes

Table 4. Means pre and post Bender Gestalt errors for treatment, LD control and normal comparison groups.

	Treatment (n=5)	LD Control (n=5)	Normal Comparison (n=10)
Pretest	3.20	4.40	2.10
Posttest	2.20	4.40	2.00

Table 5. Mean change scores for Tukey pairwise comparisons on the Bender Gestalt for treatment, LD control and normal comparison groups.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	-1.0	0	-.10	
δ	1.22	1.22	1.10	
n	5	5	10	
df	4	4	9	Σdf=17

Table 6. IQ scores on the WISC-R for treatment, LD control and normal comparison groups.

	Pretest	Posttest
	Treatment (n=5)	
Verbal IQ	95.60	92.60
Performance IQ	105.40	114.40
Full scale IQ	99.60	102.80
	LD Control (n=5)	
Verbal IQ	90.80	96.00
Performance IQ	100.60	112.80
Full scale IQ	95.00	103.80
	Normal Comparison (n=10)	
Verbal IQ	117.80	119.10
Performance IQ	114.50	120.20
Full scale IQ	118.10	122.20

Table 7. Mean change scores for Tukey pairwise comparison of WISC-R verbal IQ score.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	-3.0	5.20	1.30	
δ	6.52	7.40	6.58	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

Table 8. Mean change scores for Tukey pairwise comparison of WISC-R performance IQ score.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	9.0	12.2	5.70	
δ	10.29	10.55	7.51	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

Table 9. Mean change scores for Tukey pairwise comparison for WISC-R full scale IQ score.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	3.20	8.80	4.10	
δ	7.04	8.11	6.05	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

obtained between testings for both performance IQ and full scale IQ also show there to be no significant differences between the treatment, control and comparison groups.

Psychoeducational Data

Spache Diagnostic Reading Scales

The Spache Diagnostic Reading Scale was used to obtain a measure of reading comprehension gains occurring over the testing interval. Because scores are reported as grade level equivalents rather than raw scores, Kadson (1977) has indicated that the results may only be properly analyzed using procedures appropriate for less than interval level data. Change scores were thus ranked as suggested by Inman and Conover (1971) and Tukey's HSD was performed on the ranked scores for each group.

Table 10 shows the mean grade equivalent scores obtained by each group for both the oral and silent portions of the Spache. The oral or instructional score represents the highest grade level that a child can read to the tester with 85% comprehension. The silent or independent score represents the highest level the child can read alone with 60% comprehension. This table shows both LD groups having increased scores on the oral and silent portions of the Spache while the comparison group posted small decreases in grade equivalents between tests. Increases were slightly higher for the treatment group than for the control group for the oral comprehension score and were higher in the control group as compared to the treatment group for the silent scores. Tables 11 and 12 show the mean

Table 10. Mean grade equivalence scores on the Spache Diagnostic Reading Scales for treatment, LD control and normal comparison subjects.

	Pretest	Posttest
	Treatment (n=5)	
Oral	3.34	4.12
Silent	3.72	3.78
	LD Control (n=5)	
Oral	3.6	4.28
Silent	4.42	5.08
	Normal Comparison (n=10)	
Oral	7.9	7.7
Silent	8.1	7.9

Table 11. Mean change scores for Tukey pairwise comparison of Spache oral reading grade equivalents in ranks for treatment, LD control and normal comparison groups.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	14.4	12.5	7.55	
δ	2.30	7.0	5.13	
n	5	5	10	
df	4	4	9	Σdf=17

Table 12. Mean change scores for Tukey pairwise comparison of Spache silent reading grade equivalents in ranks for treatment, LD control and normal comparison groups.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	10.5	14.6	8.45	
δ	5.17	4.32	5.17	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

scores for the treatment, LD control and normal comparison groups used in the Tukey HSD for both oral and silent comprehension. None of the comparisons between the three groups for either the silent or oral reading levels were found to be significant.

Wide Range Achievement Test (WRAT)

All three portions of the WRAT were administered in order to assess academic gains made by the subjects over the training period. Table 13 indicates mean raw scores for the reading (word recognition), spelling and arithmetic profiles for the treatment, LD control and normal comparison groups. As can be seen in Table 13, all three groups showed improvements for all sections of the WRAT from pretest to posttest intervals. Changes in reading scores were quite close for all three groups with the greatest change being shown by the comparison group followed by the treatment and LD control groups in

Table 13. Mean raw scores on the Wide Range Achievement Test for treatment, LD control and normal comparison groups.

	Pretest	Posttest
	Treatment (n=5)	
Reading	50.60	54.00
Spelling	31.60	35.40
Arithmetic	28.80	29.80
	LD Control (n=5)	
Reading	51.20	54.40
Spelling	33.40	34.80
Arithmetic	28.80	30.20
	Normal Comparison (n=10)	
Reading	77.50	81.40
Spelling	51.30	53.00
Arithmetic	38.50	40.20

that order. The treatment group showed the largest gains in spelling followed by smaller gains in the comparison group which was slightly larger than the LD control group. Changes in arithmetic scores were greatest for the normal comparison group followed by the LD control group which showed greater change than the treatment group.

Tables 14, 15 and 16 show the mean change scores for the reading, spelling and arithmetic portions of the WRAT used in the Tukey HSD planned comparisons. No significant difference for any of the three sections of the WRAT was found for either the treatment, LD control or the normal comparison groups.

Table 14. Mean change scores for Tukey pairwise comparison of WRAT word recognition raw scores.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	3.40	3.20	3.90	
δ	7.54	1.48	2.55	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

Table 15. Mean change scores for Tukey pairwise comparison of WRAT spelling raw scores.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	3.8	1.40	1.70	
δ	4.09	2.61	1.77	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

Table 16. Mean change scores for Tukey pairwise comparison of WRAT arithmetic raw scores.

	Treatment	LD Control	Normal Comparison	
$\bar{x}$	1.0	1.40	1.90	
δ	1.22	1.14	1.66	
n	5	5	10	
df	4	4	9	$\Sigma df=17$

EEG Data

Spectral Analysis

Spectral analysis was performed using a Fast Fourier Transform (FFT) on the EEG of the treatment, LD control and normal comparison groups for both pretest and posttest conditions. This analysis yielded values for the EEG in terms of power in pico watts for nine frequency bands. These bands ranged from 0-28 Hz divided into four Hz bands plus a 6-10 Hz band plus a total power band. The percentage of the total power contained in each of the four Hz bands was also calculated for each of the three groups. This resulted in 17 EEG values obtained from each of eight locations per subject from which data were recorded or 136 values per subject for each of the testing conditions.

EEG of Normal Subjects Compared to LD Subjects

Because the treatment group and the LD control group were chosen at random from the same initial population, these two groups were combined and compared to the normal comparison group for any difference occurring before training. Figure 4 represents the differences between these two groups in terms of power and percentage power during baseline, reading and drawing conditions. The symbols B, R and D stand for the baseline, reading and drawing conditions, respectively, and the plus (+) sign indicates that the LD subjects had greater scores than the normal subjects. The minus (-) sign indicates lower scores for the LD subjects when compared to normal children. An analysis of variance was performed on the EEG scores and only differences significant at the .05 level or less are shown.

In terms of EEG power, Figure 4 indicates that the LD children exhibited more power in both frontal locations for the low frequencies (0-4 Hz) and for the higher frequencies (16-24 Hz). LD children show increased power in the right frontal area for reading in the low frequencies and for all tasks in the higher frequencies. The left derivation shows greater frontal activity for drawing in the lower frequencies and for baseline in the higher activity band.

Percentage values from Figure 4 show that LD subjects displayed more fast activity in the left central and parietal locations before treatment when compared to normal subjects during a drawing task. There is also some indication that LD children show more intermediate (12-16 Hz) activity during a baseline condition in the left frontal

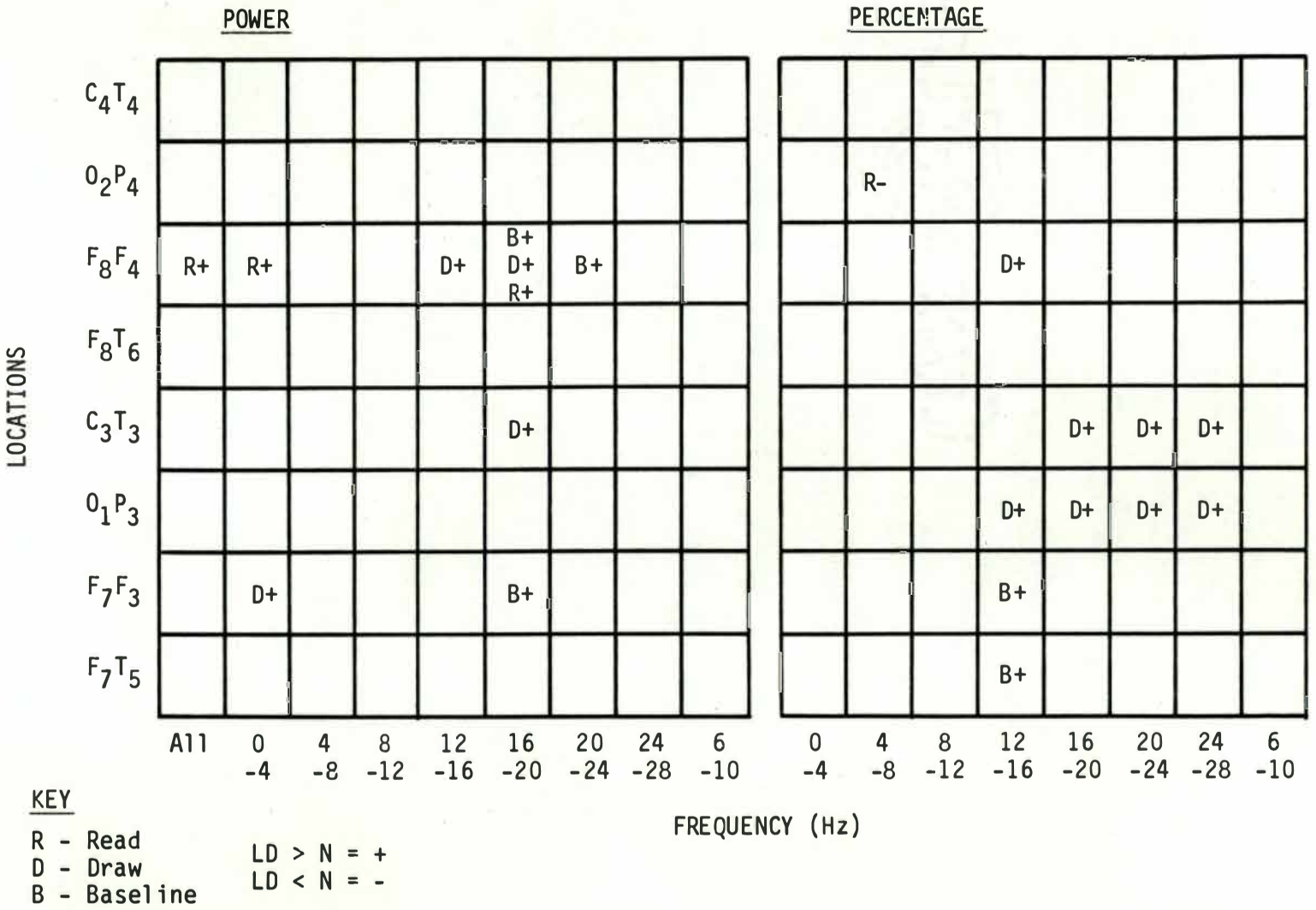
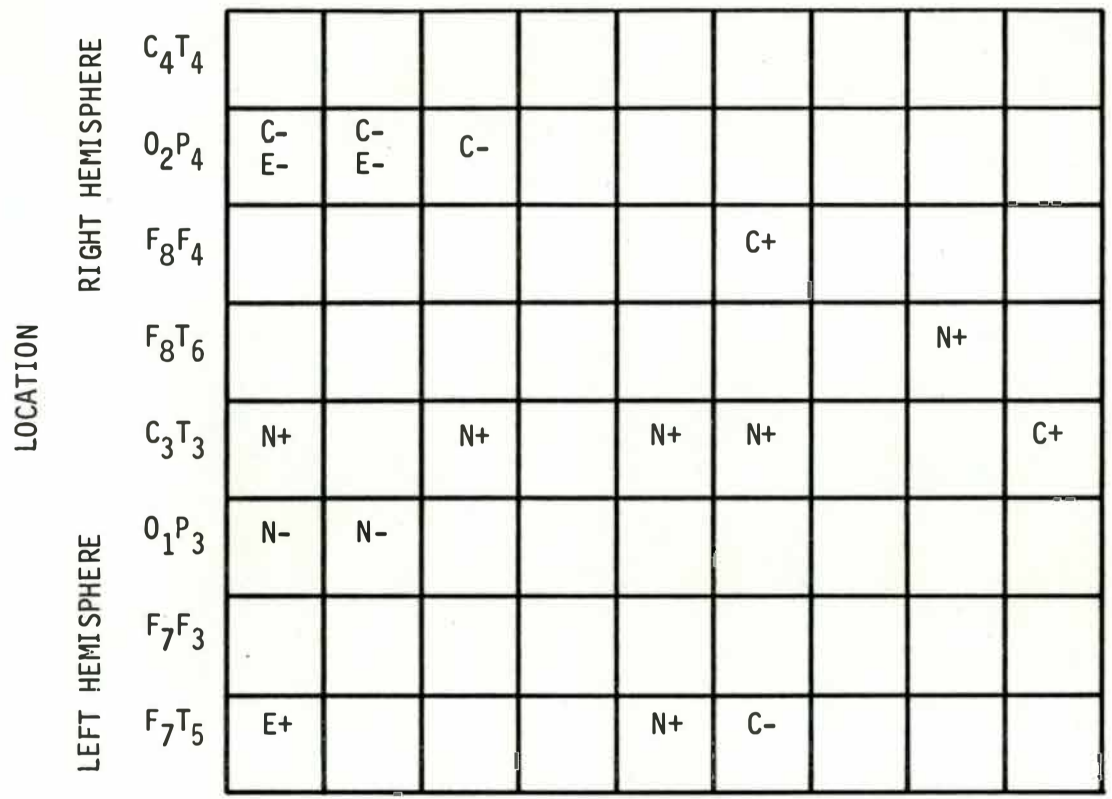


Figure 4. EEG comparison for eight locations between 0-28Hz for LD and normal children.

and temporal derivations. The only area where LD children showed any reduction in percent power occurred in the lower frequencies of the right occipital and parietal areas.

Changes in Spectral Power Over Treatment

In order to assess the effects of treatment on the EEG during the baseline, reading and drawing conditions, a one-way analysis of variance (ANOVA) was performed on the change scores between pretest and posttest spectral values for the treatment, LD control and normal comparison groups. Tests were conducted for each of the frequency bands and for each cortical location. Change scores have been reported to provide a more powerful procedure (Gailford and Fruchter, 1973) than t tests on sets of data and were thus utilized. Significant differences occurring during the ANOVA were then subjected to t tests in a post hoc fashion in order to determine in what manner the control, comparison and treatment groups differed from each other. Figures 5 through 10 represent these differences during the baseline, reading and drawing tasks for both power and percent spectral values. The letters C, N and E stand for control, normal and experimental groups and the plus (+) sign indicates that posttest scores were greater than pretest scores while the minus (-) sign indicates that pretest scores were greater than posttest scores. An alpha level of .05 was used for all tests.



Key

C = Control

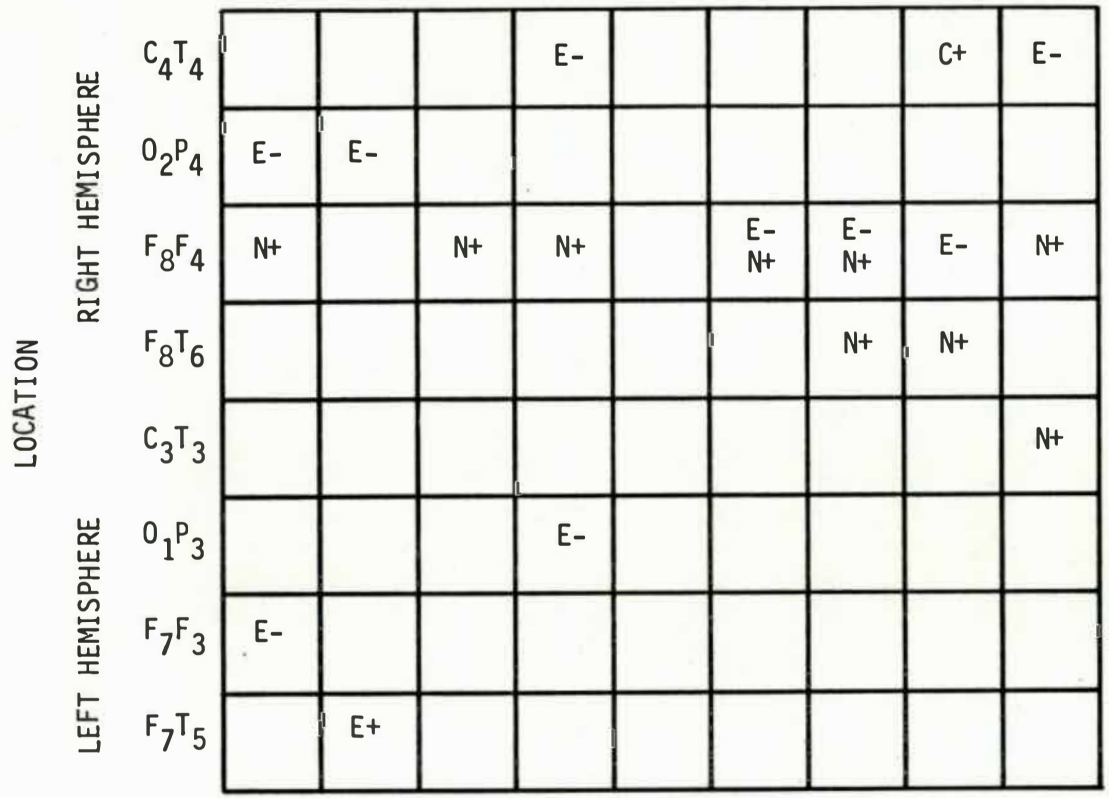
E = Experimental

N = Normal

\+ = Post > Pre

\- = Post < Pre

Figure 5. Directional comparisons for pre and post training scores for baseline power.



KEY

C = Control

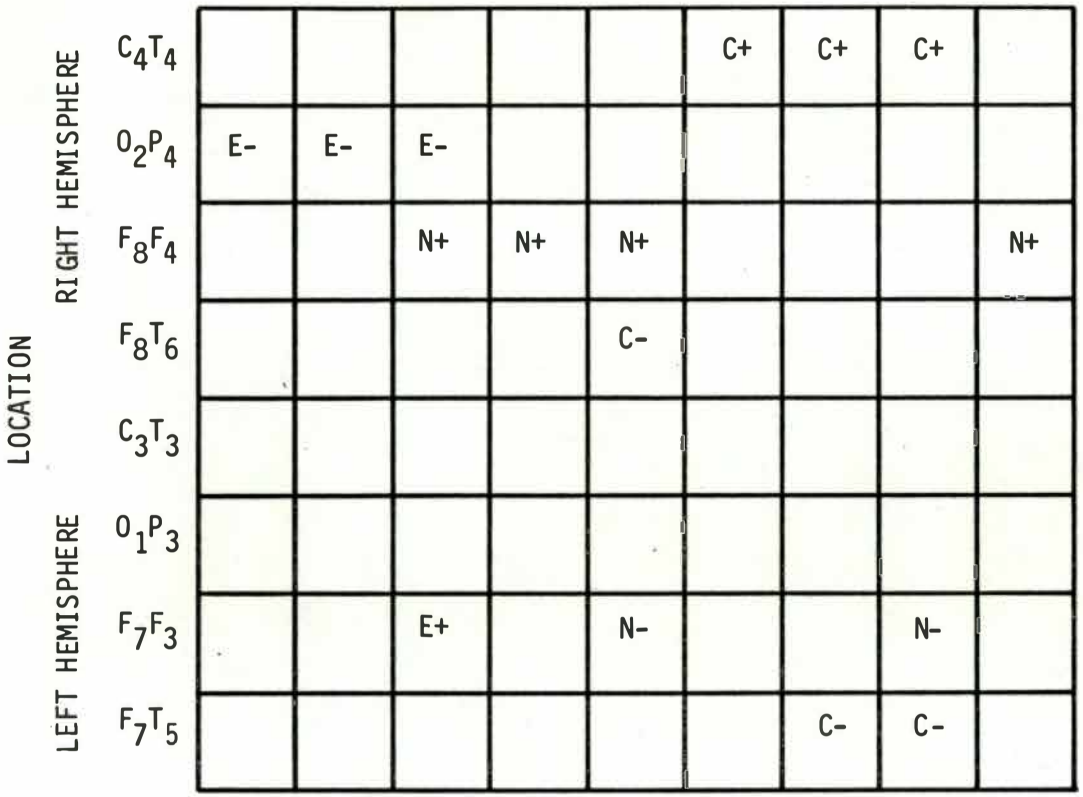
E = Experimental

N = Normal

\+ = Post > Pre

\- = Post < Pre

Figure 6. Directional comparisons for pre and post training scores for reading power.



KEY

C = Control

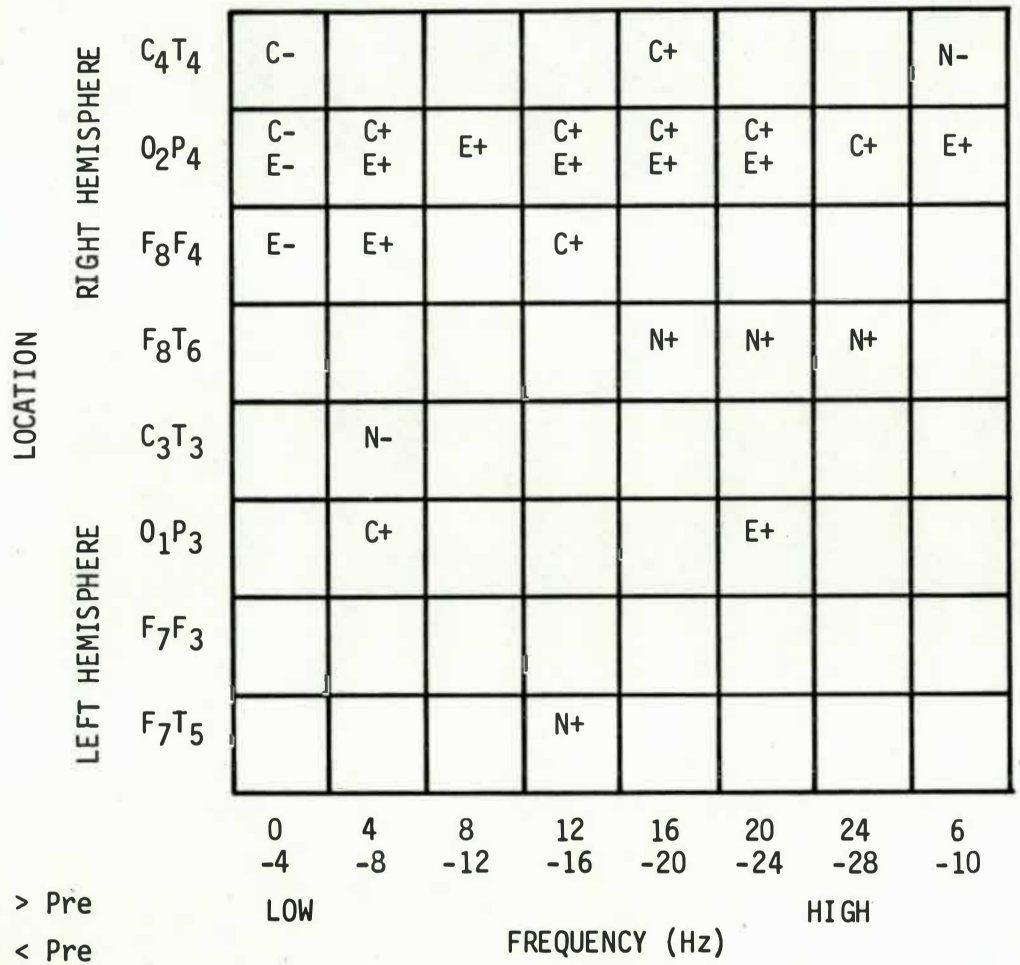
E = Experimental

N = Normal

\+ = Post > Pre

\- = Post < Pre

Figure 7. Directional comparisons for pre and post training scores for drawing power.



KEY

C = Control

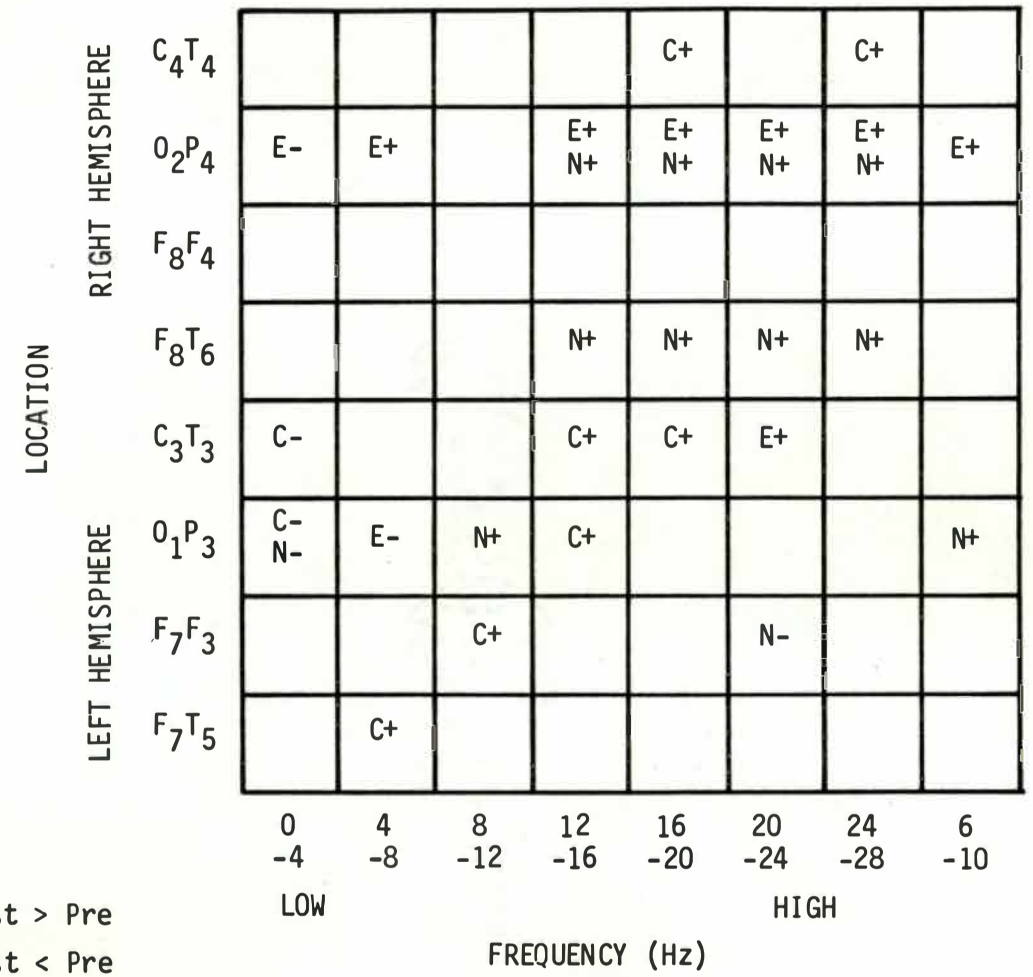
E = Experimental

N = Normal

\+ = Post > Pre

\- = Post < Pre

Figure 8. Directional comparisons for pre and post training scores for baseline percentage.



KEY

C = Control

E = Experimental

N = Normal

\+ = Post > Pre

\- = Post < Pre

Figure 9. Directional comparisons for pre and post training scores for reading percentage.

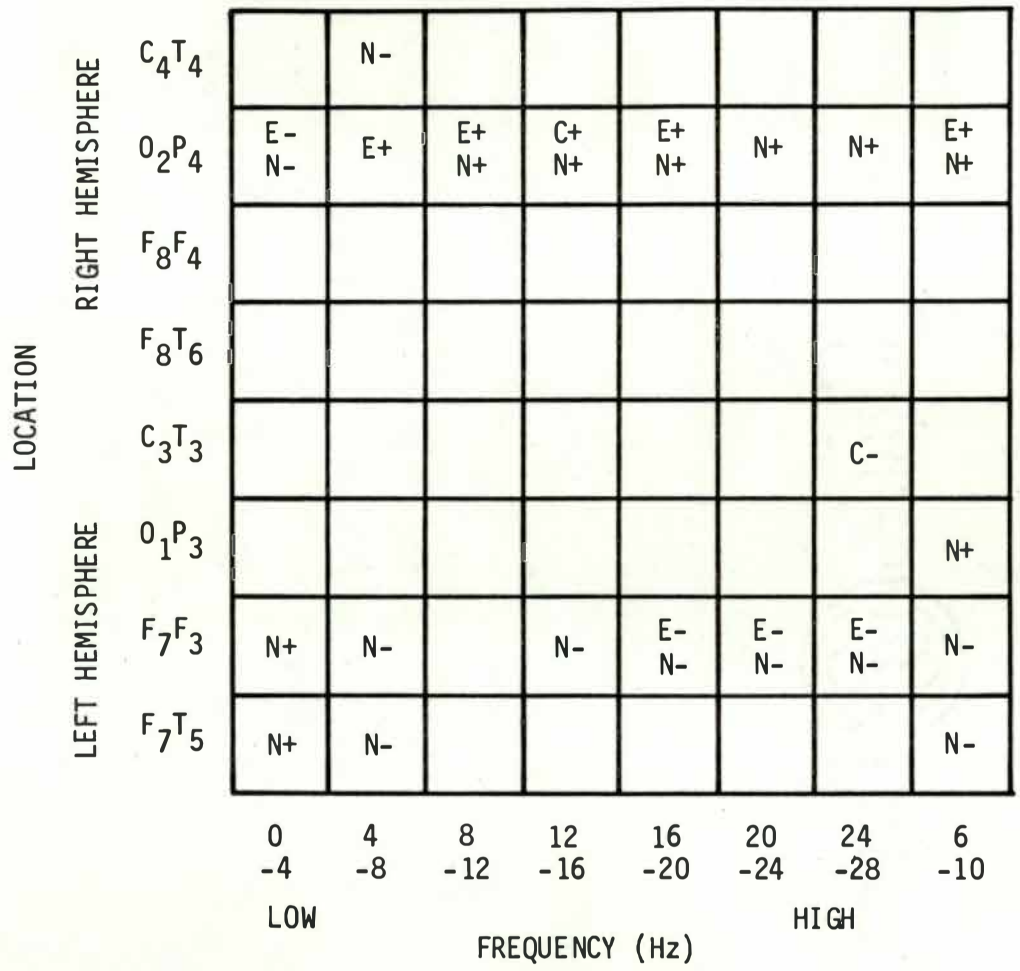


Figure 10. Directional comparisons for pre and post training scores for drawing percentage.

Spectral Power During Baseline

Figure 5 depicts the significant differences found between the treatment, LD control and normal comparison groups for spectral power during baseline conditions. For the right hemisphere both the controls and the experimentals showed significant decreases in power in the occipital and parietal regions. In the left hemisphere, most of the changes were shown by the normal comparison group. These changes occurred primarily in the central and occipital areas and are primarily reflected as increases in power in the lower and intermediate frequencies for the central locations and decreases in power in the occipital areas in the lower frequencies.

Spectral Power During Reading

Significant changes in spectral power during the reading condition are shown in Figure 5. This figure indicates that the treatment group had decreases in spectral power in both frontal areas. These decreases occurred in the higher frequencies of the right hemisphere and in the total power band of the left hemisphere. The experimental group also showed decreases in power in the occipital-parietal region, primarily in the lower frequencies. Contrary to the decreases in power shown by the experimental group, the normal group showed increased right frontal power in the lower intermediate and higher frequency bands.

Spectral Power During Drawing

Figure 7 represents the change in EEG power which occurred during the drawing condition for the normal, control and experimental groups. For right hemisphere derivations, the experimental group decreased the amount of power in their EEG in the lower frequencies in the occipital-parietal region. The control group increased their power values in the higher frequencies of the right central region and the normals increased right frontal lower and intermediate frequencies. Few changes occurred in the left hemisphere although there is an indication of decreased temporal power for the LD control group in the higher frequencies.

Percentage Power Changes During Baseline

Figure 8 represents the changes occurring between EEG recordings for the normal, treatment and comparison groups for percent power. This figure indicates that the control and treatment groups both decreased power percentages in the very low frequencies but showed increases in all other frequencies in the right occipital-parietal area. Increases were also shown in the right temporal area in the higher frequencies by the normal comparison group. Little change of significance was found for left hemisphere locations for any of the groups.

Percentage Power Change During Reading

Reading changes expressed as power percentages for the normal, control and comparison groups are illustrated in Figure 9. Right

hemisphere changes occurred primarily in the frontal and occipital-parietal regions. Right occipital-parietal areas showed decreases in the very low frequencies by the experimental group and increases in the intermediate and higher frequencies by both the experimental and normal groups. The right frontal area also showed increases in the intermediate and higher frequencies by the normal comparison group. Left hemisphere changes occurred primarily in the central and parietal-occipital areas. Posteriorly, the control, normal and experimental groups all decreased low frequency percentages while the controls and normals increased intermediate frequencies.

Percentage Power Changes During Drawing

Drawing changes expressed as percentages can be seen in Figure 10 for the normal comparison, LD control and treatment groups. Differences are found primarily in right occipital-parietal and left frontal and temporal regions. In the right hemisphere both the experimental and normal groups decreased power percentages in the very low frequencies and increased percentages in the intermediate and higher frequencies. In the left hemisphere the normal comparison group increased very low frequencies in both frontal and temporal regions and decreased percentage power in the next higher frequency band. Both the normal comparison and the experimental group decreased power percentages in the higher frequencies in the left frontal region.

Summary

In order to obtain a better overall picture of the changes that may have occurred as a result of treatment Figure 11 summarizes

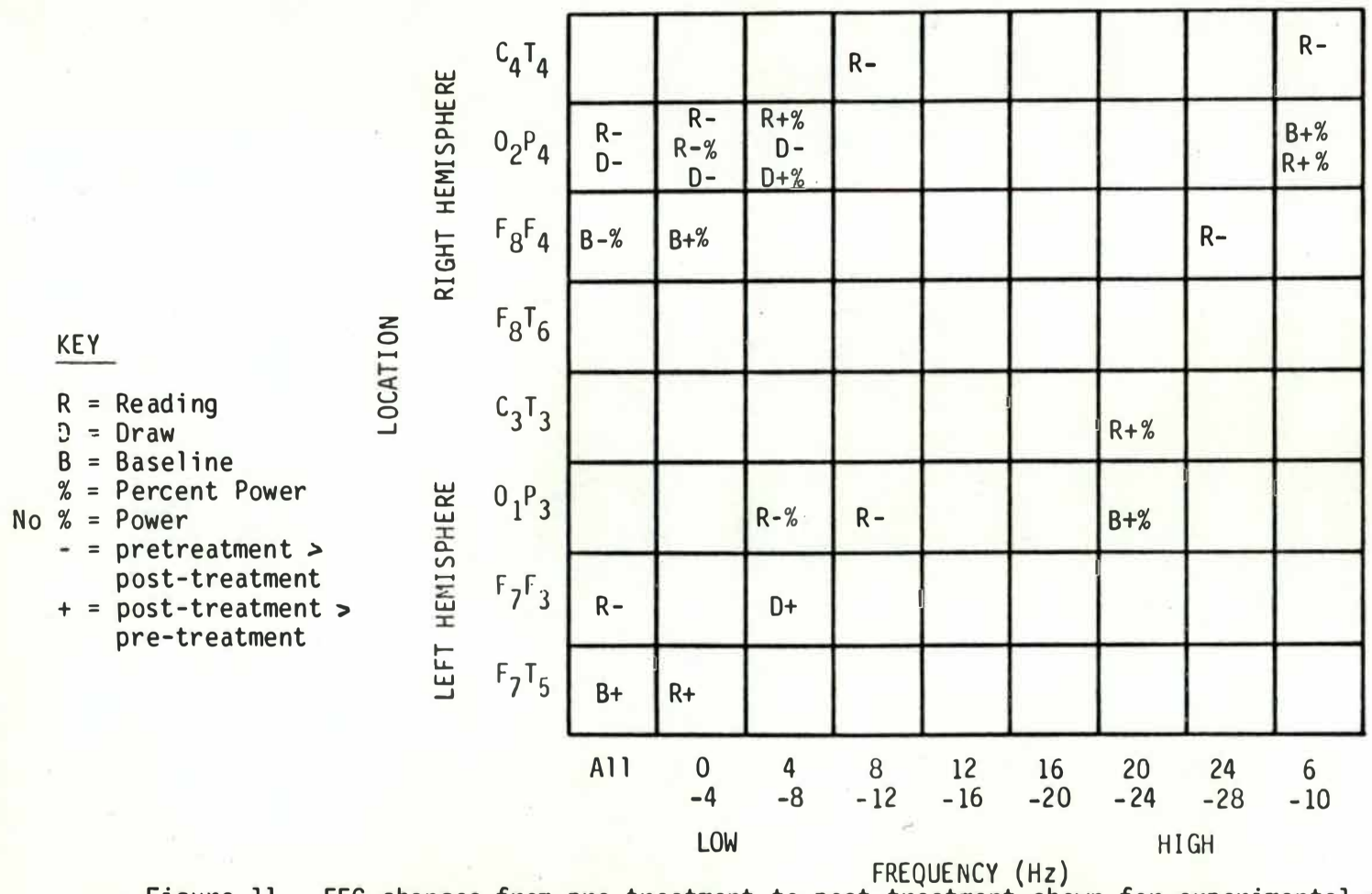


Figure 11. EEG changes from pre treatment to post treatment shown for experimental group only.

Figures 5-10 for locations and frequencies in which only the experimental group showed a change. As can be seen most of the differences occurred in the right occipital-parietal area. In general these changes indicate reductions in slower frequency activity and increases in higher frequency activity. Except for the right central-temporal region in which power for reading was decreased in the 6-12 Hz range no other systematic changes appear to have developed as a result of training.

Evoked Potential Data

P300 amplitudes and latencies recorded from Cz during the baseline and experimental portions of the study were subjected to several one and two way repeated measures analyses of variance. In addition a Pearson product-moment correlation coefficient for amplitudes and latencies versus days was also obtained. P300 peaks during baselines were defined as the major positive deflection originating between 300 and 500 msec after the stimulus presentation. In the case of dual peaks the one of greatest amplitude was chosen. For feedback trials the P300 peak was defined as the major (only) positive peak or as the largest positive peak (100% greater than other positive peaks) originating between 300 and 500 msec after the stimulus presentation. In the case of dual positive peaks of approximately equal value P300 was defined as the peak closest to the latency of the baseline P300 peak for that session if the peaks showed less than about 20-25% differences in amplitude or as the peak of greatest amplitude if their differences were greater than 25%. Small

fluctuations in polarity were not counted as peaks if they occurred on either the negative or positive slope of a P300 wave. It was not necessary for a peak to fall inside the latency range for which feedback was provided to be counted as a P300 peak.

P300 Amplitude

Table 17 shows the mean microvolt (uv) amplitudes from all five subjects for baseline sessions of 75, 50, and 35 trials for both the pre and post treatment conditions as well as the collapsed mean for these conditions. A one way repeated measures analysis of variance performed on the two baseline periods indicates no significant differences between the different averages used in obtaining the EPs for either the pre or post treatment conditions. Averages were then grouped and a two way repeated measures analysis of variance performed on the amplitudes of the five subjects for pre treatment baselines versus post treatment baselines. No significant difference was found for P300 amplitudes obtained from these two conditions even though post treatment baseline showed a rather large (1.65 uv) decrease in amplitude. This lack of significance was more than likely due to very large variances occurring during the first baseline period.

A repeated measures two way analysis of variance was next conducted on the training sessions consisting of an initial baseline condition, two feedback conditions, and a final baseline condition.

Table 17. Mean baseline amplitude in uv's for trials consisting of 75, 50, and 35 averages, and collapsed across trials.

	Pre Treatment	Post Treatment
75 averages	6.95	4.40
50 averages	6.62	5.19
35 averages	5.92	4.95
Group mean	6.50	4.85

Figure 12 shows the mean uv value for P300 amplitudes averaged across all subjects and all sessions for each condition involved in the experimental portion of the study and Table 18 shows these same conditions broken down into six session blocks. The analysis of variance indicated that the four conditions are significantly different ($F = 4.24$, $p < .03$) and that the various blocks constituting sessions are also significantly different ($F = 5.03$, $p < .02$). In order to assess these differences a Newman-Keuls post-hoc test was performed for both between condition scores and within condition scores. Figure 13 shows the within condition scores (blocks by days) collapsed across subjects and conditions.

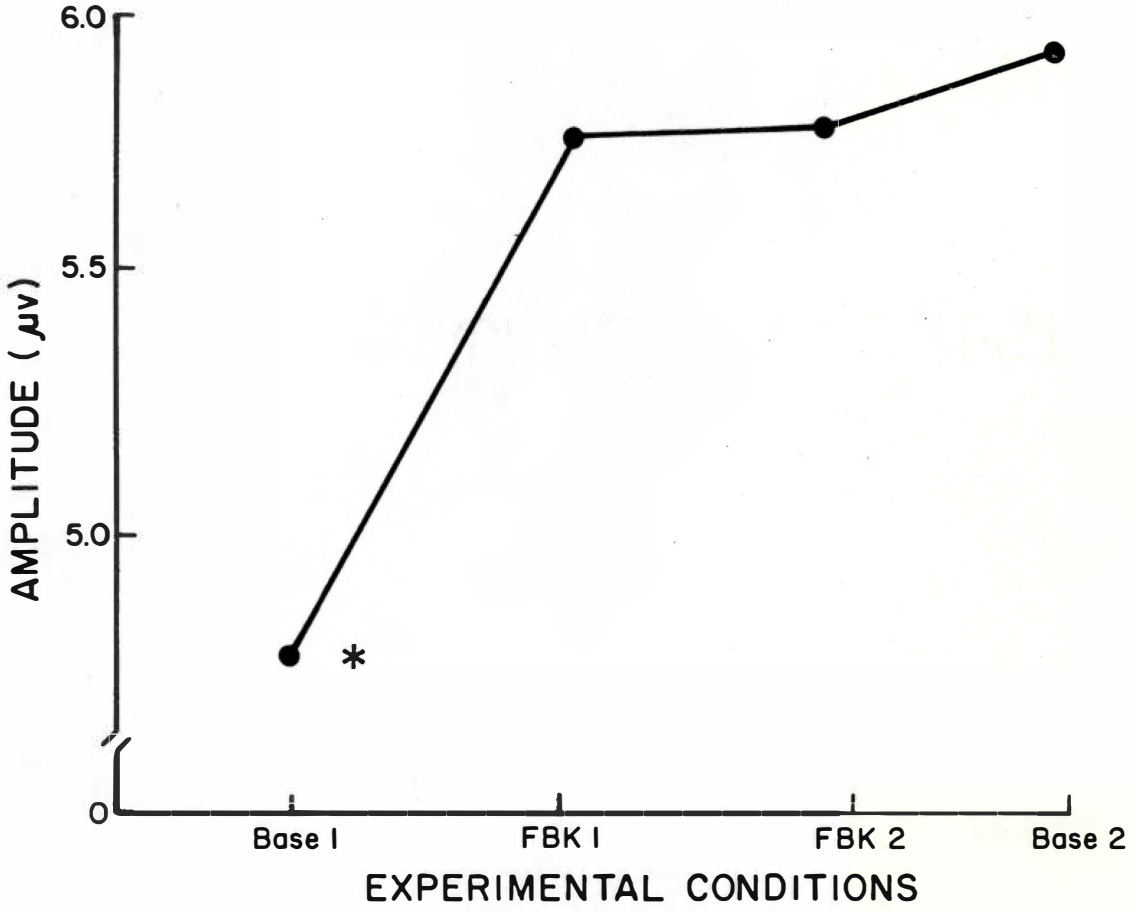


Figure 12. Mean P300 amplitude value for all subjects for each experimental condition. * indicates significance at the .05 level. Base = Baseline, FBK = Feedback.

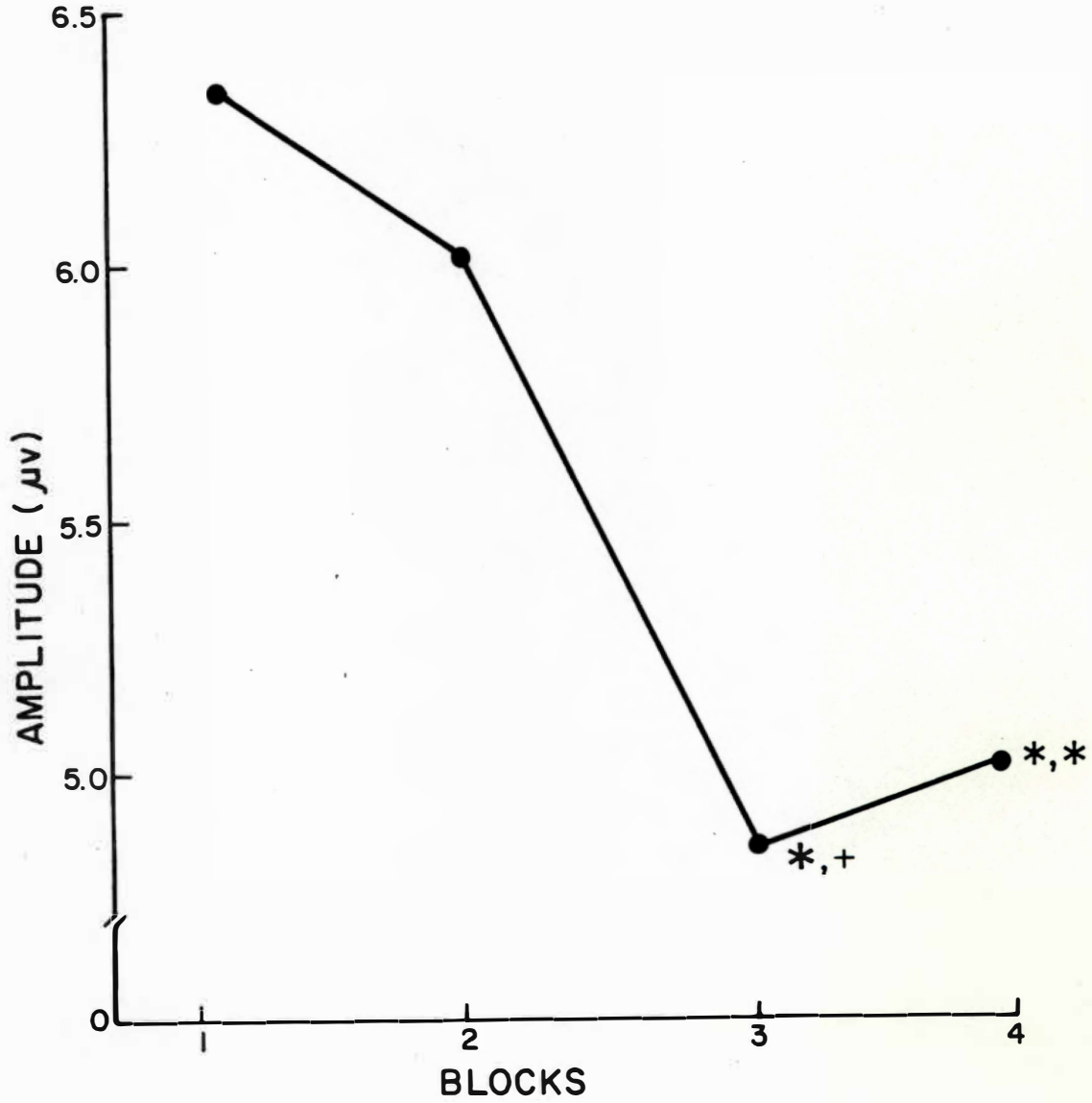


Figure 13. Mean P300 amplitude for all subjects and conditions for each block of experimental procedure. * indicates .05 level of significance. + indicates significance level between .1 and .05.

Table 18. Mean P300 uv amplitude averages for all subjects for treatment conditions and six session blocks.

Blocks	Baseline 1	Feedback 1	Feedback 2	Baseline 2
1	5.75	6.79	6.04	6.80
2	5.11	6.26	6.45	6.49
3	4.24	5.14	5.11	4.94
4	4.06	4.89	5.60	5.54

The data shown in Figure 12 indicates that during training the experimental children were able to significantly raise the amplitude of the P300 component of their evoked potentials over baseline levels when provided with feedback. These increases were also maintained during a final baseline following training. No significant differences were found between the two feedback and final baseline conditions. Although the subjects were able to increase their initial baseline level of activity throughout the study Figure 13 shows that the amplitude of this activity was decreasing as the training sessions progressed. Figure 13 also illustrates that when training sessions are broken down into six session blocks from the beginning to the end of training that block three is significantly different from block one at the .05 level. In addition, block three approaches being significantly different from block two ($.10 > \alpha > .05$). Block four is significantly lower than both block one and two. No difference was found between blocks one and two or between blocks three and four. Although it may seem as if block three should

also be significantly different from block two at the .05 level, block three did not quite reach significance because the Newman-Keuls test ranks the scores and takes into consideration the distance between each block. The greater the distance the larger the score needed for significance. Thus block three was actually further away from blocks one and two than was block four and needed a greater score for the same level of significance than did block four.

Table 19 shows the results of a Pearson product-moment correlation in terms of the correlation coefficients and significance levels for amplitude versus days for the four training conditions. As can be seen all conditions were negatively correlated with amplitudes decreasing as days (training sessions) increased. All but the third condition were significantly correlated with conditions one and two being highly significant. Figure 14 shows the relationship between amplitude and sessions for all conditions employed in the study.

Table 19. Pearson product-moment correlation coefficients for experimental conditions by sessions (days). n=24

	Baseline 1	Feedback 1	Feedback 2	Baseline 2
Days	-0.769	-0.806	-0.394	-0.474
Significance level	0.0001	0.0001	0.056	0.019

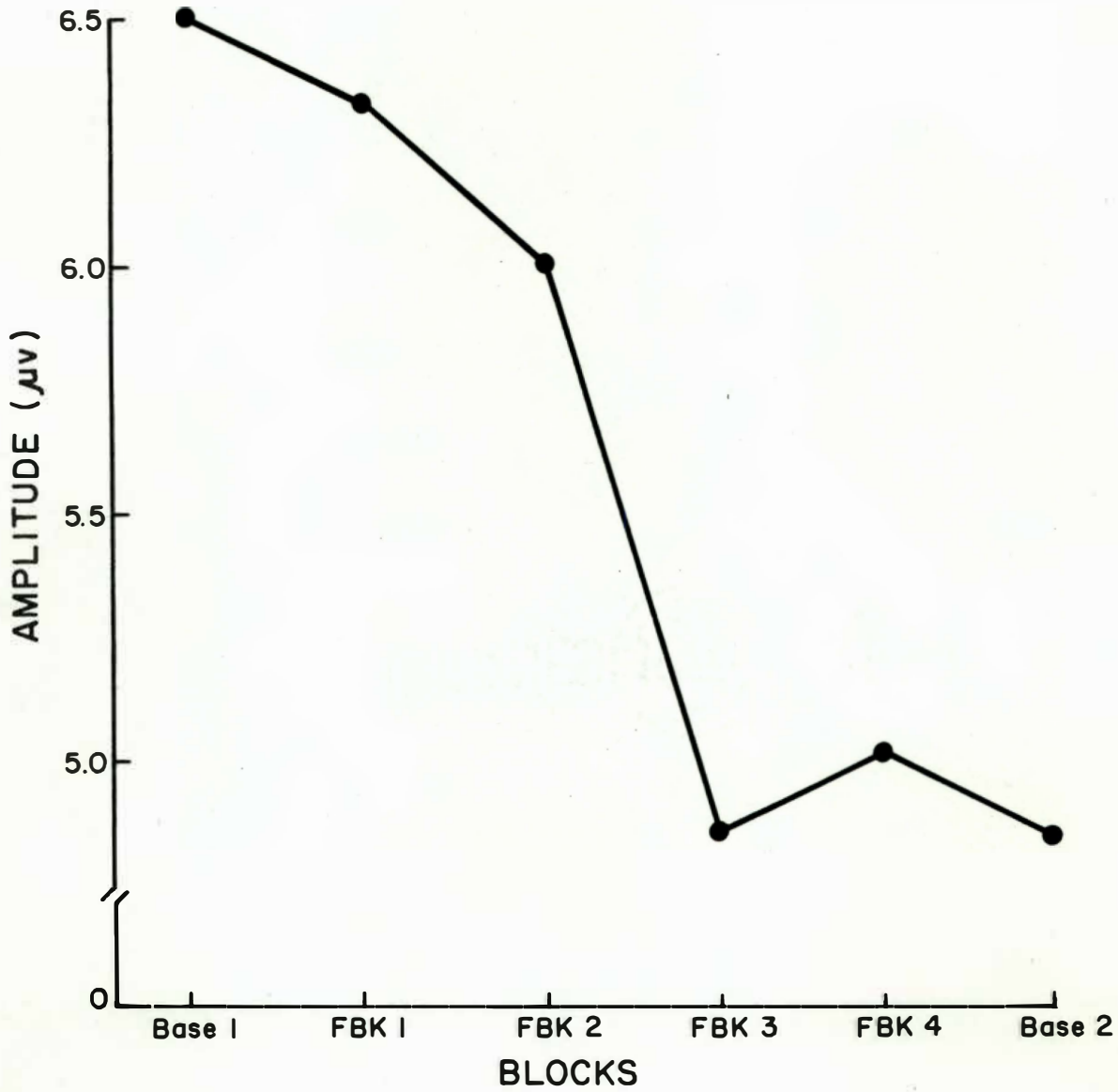


Figure 14. Mean P300 amplitude for pre and post treatment blocks and four experimental blocks. Base = Baseline; FBK = Feedback.

P300 Latency

A one way repeated measures analysis was again used to assess any differences between the three conditions (75, 50, and 35 trial averages) of both pre and post baseline latencies of the P300 peak for all five children participating in the study. No difference was found between the three conditions for either the pre treatment or post treatment. The three different averages were then grouped and a two way repeated measures analysis of variance performed to compare pre treatment latencies to post treatment latencies. No significant differences were found for the latencies of the baseline periods preceding and following the training sessions. Table 20 gives the mean latency values in milliseconds (msec) for all five subjects for each of the different averages as well as the overall mean for each group. As can be seen there is virtually no difference between pre treatment baseline latencies and post treatment latencies.

Table 20. Mean baseline P300 latencies in msec for trials of 75, 50, and 35 averages and for groups.

	Pre Treatment	Post Treatment
75 averages	462	457
50 averages	472	462
35 averages	460	445
Group mean	465	455

The four conditions (baseline 1, feedback 1, feedback 2, baseline 2) of the experimental portion of the study were also subjected to a two way repeated measures analysis of variance. Table 21 shows the latencies of the P300 peak in six session blocks for the four treatment conditions. Figures 15 and 16 show the mean latency scores for the four conditions and for the four blocks. The analysis of variance indicates no significant latency changes occurred between any of the experimental conditions or between any of the blocks (days) of the experimental period. Figure 15 shows that P300 latencies increased slightly from the first baseline condition to the first feedback condition and then decreased to the end of training. Figure 16 shows that latencies of the P300 peak increased as the experimental procedure progessed. It should be noted that the latency changes shown in Figures 15 and 16 are quite small and represent total changes of less than 2%.

Figure 21. Mean P300 latency in msec averages for treatment conditions and blocks.

Blocks	Baseline 1	Feedback 1	Feedback 2	Baseline 2
1	455	451	439	443
2	453	456	458	450
3	453	459	462	450
4	471	475	455	453

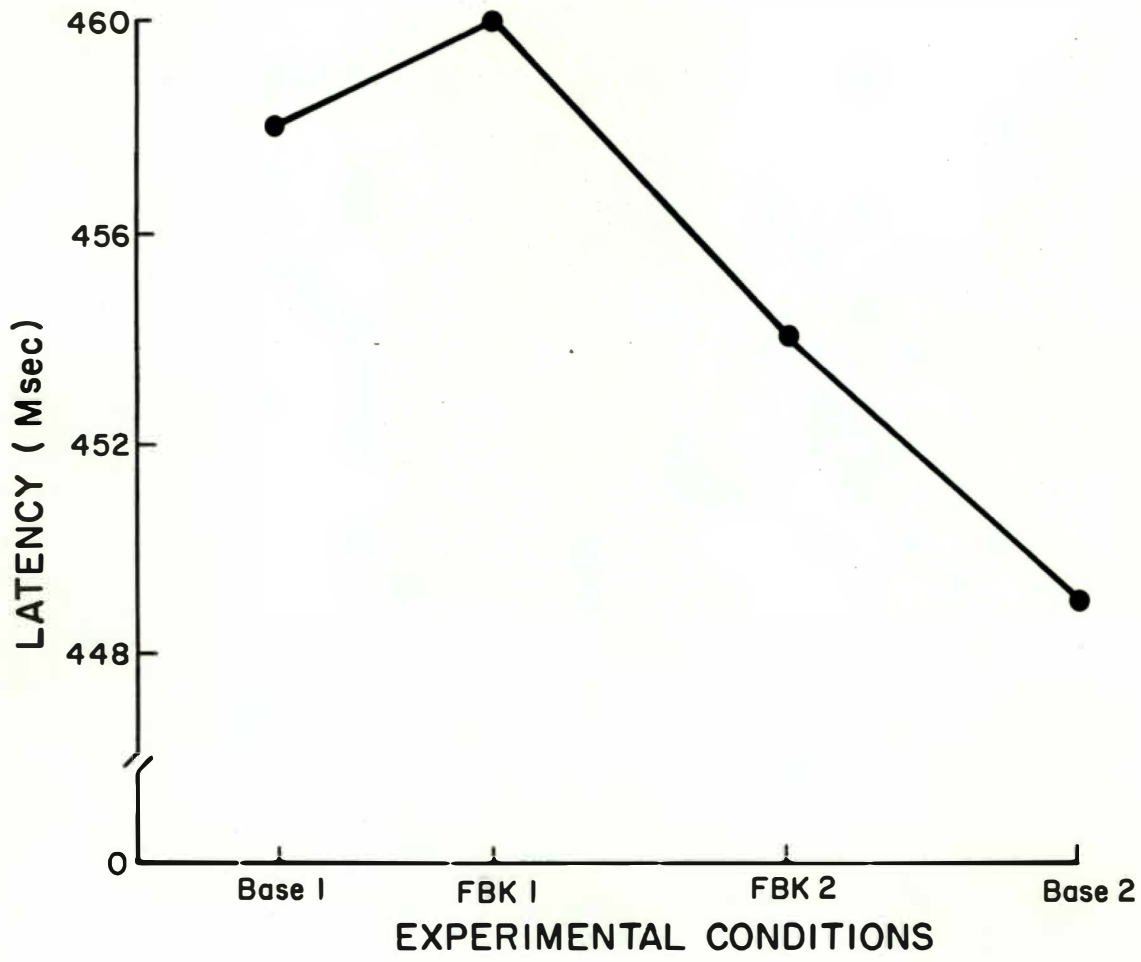


Figure 15. Mean P300 latencies for all subjects for each experimental condition. Base = Baseline; FBK = Feedback.

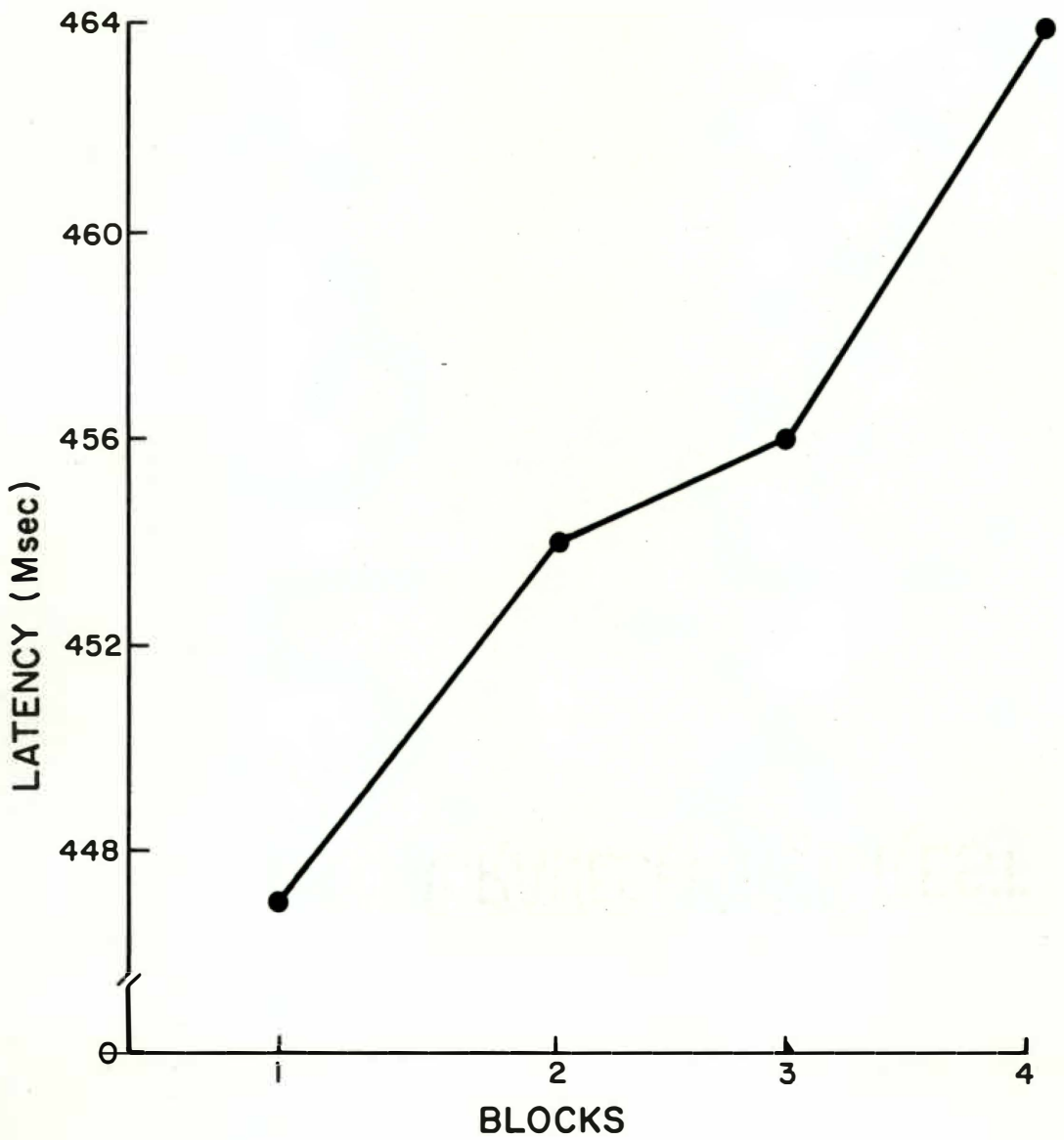


Figure 16. Mean P300 latency for all subjects and conditions for each block of experimental procedure.

Table 22 shows the results of a Pearson product-moment correlation conducted for each of the experimental conditions versus training days. The first line of the table gives the correlation coefficient and the second line the significance level. As can be seen no relationship was found to exist between the experimental conditions and the progression of the training. Figure 17 shows the mean latency for each of the baseline periods and the four, six session blocks of the experimental period. It can be seen that latencies dropped between the first baseline period and the first block of the experimental period. Latencies then increased until the end of training to almost pre training levels and then decreased during the second baseline. Again it should be noted that these changes are very small and represent less than a 2% difference as measured from greatest to smallest values.

Table 22. Pearson product-moment correlation coefficients for experimental conditions by sessions (days). n=24

	Baseline 1	Feedback 1	Feedback 2	Baseline 2
Days	0.167	0.253	0.243	0.029
Significance level	0.436	0.233	0.252	0.891

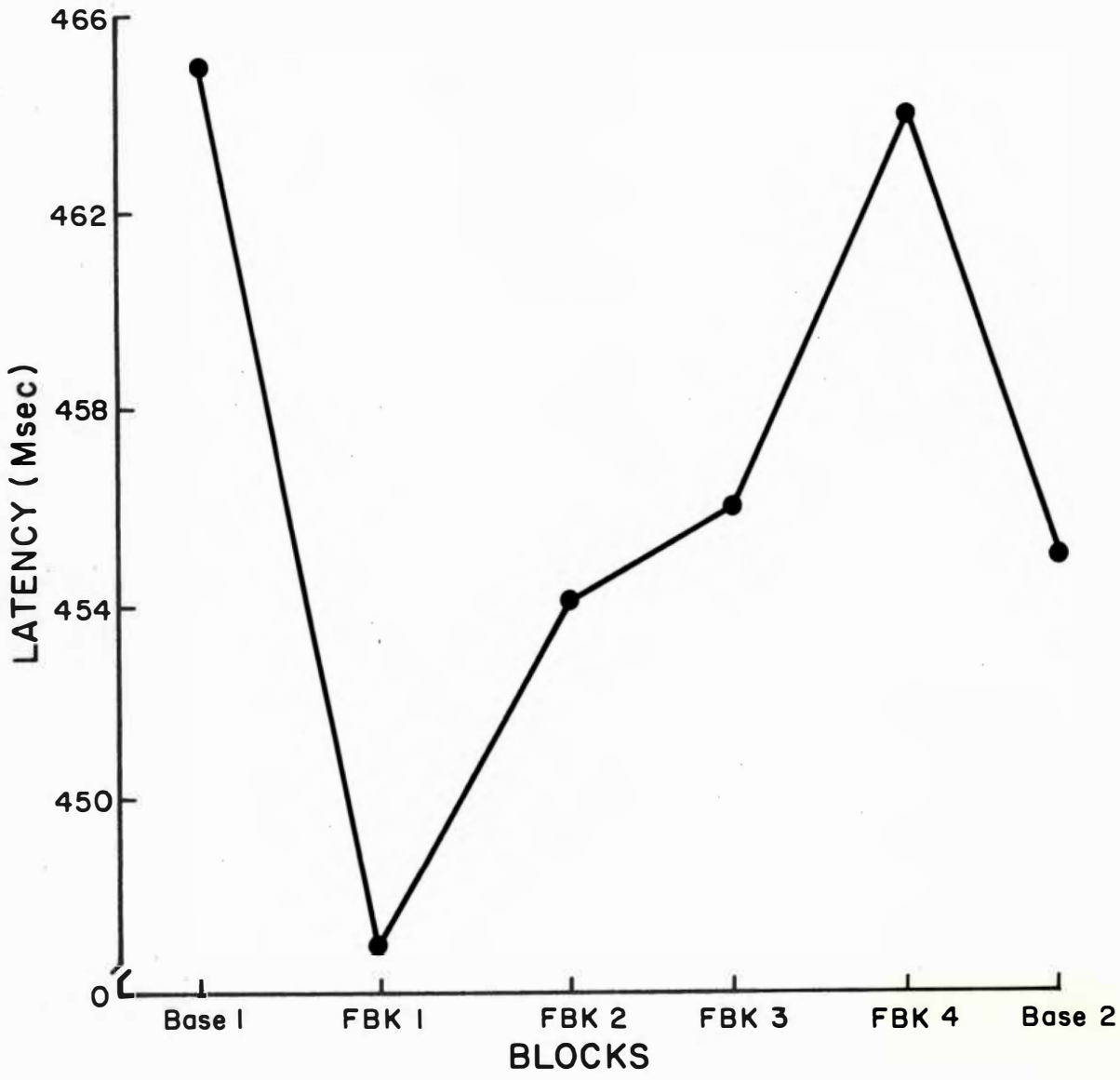


Figure 17. Mean latency for pre and post treatment blocks and four experimental blocks. Base = Baseline; FBK = Feedback.

CHAPTER IV

DISCUSSION

Neuropsychological and Psychoeducational Tests

Short of tutoring children on the specific answers it would be highly unrealistic to expect any one particular procedure to produce positive, let alone statistical, results on a number of tests designed to measure different aspects of neurological integrity, intellectual functioning, and academic skills. Rather, it might be more productive to rate expectations of changes in test scores according to their closeness to the treatment employed, in this case visual evoked potential (VEP) training. Since the VEP is considered to be a basic indicator of neurological and sensory functioning one might expect to find the most improvement in tests which measure these kinds of factors. Of the 10 measures reported in this study the two most closely related to the treatment would be the Halstead-Reitan Battery which measures basic neurological integrity and the Bender-Gestalt Designs which measures sensory-perceptual integrity and eye-hand integration. The other tests employed, the full scale, performance, and verbal scores of the Weschler Intelligence Scale for Children-Revised (WISC-R), the oral and silent reading levels of the Spache Diagnostic Reading Scales, and the reading, spelling and arithmetic scores of the Wide Range Achievement Test (WRAT), all measure much higher levels of integrated cortical and intellectual functioning.

In looking at the tests used as dependent measures one finds that the two tests, the Halstead-Reitan Battery and the Bender Gestalt Designs, which measure basic cortical function are the ones on which the treatment group showed the greatest gain over the learning disabled control (LDC) and normal comparison (NC) groups.

In examining the results from the Halstead-Reitan and the Bender Gestalt scores (Table 3, p. 79 and Table 5, p. 81) only the difference between the NC group and the treatment group is significant at the .05 level for the Halstead Reitan Battery. It can, however, be noted that the differences are fairly large between the LDC group and the treatment group, even though these differences are not significant, for both the Halstead-Reitan and the Bender-Gestalt. This is especially true when compared to the changes seen in the NC group which received no remediation at all. Thus it appears that treatment effected a significant change over a group receiving no treatment (NC group) for the Halstead-Reitan, thereby ruling out test-retest savings or tester differences as a cause for score changes. However, it cannot be said that the NC group acted as a completely adequate control for a no treatment group. Because the NC group represents a different population than the treatment or LDC groups maturational factors may be acting differentially for the LD children and the normal children. Nevertheless, since handicapped children are required by law to be given treatment, a normal group of children may be the only alternative to a nontreatment LD group. In addition, the literature suggests that maturational differences between LD and normal children are ones of immature intellectual or academic

performance rather than ones of pace. Thus the NC group could be seen as an adequate control for improvements due to the passage of time between testings. In this fashion the improvement seen between the treatment group and the NC group can thus be attributed to the effects of the treatment. The results of the Halstead-Reitan Impairment Index for Children, then, suggest that VEP training statistically improves neurological functioning over a nontreatment group but not over a group receiving remediation in the schools. Results also indicate that school remediation is not statistically more effective than no treatment. It appears that combining VEP training with school remediation provides the best improvement in neurological function as measured by the Halstead-Reitan Battery. It should be noted, however, that the use of an NC group matched for IQ might be desirable although it would still not overcome the problem of having the NC group belong to a separate population, since these children would still not possess the assumed neurological impairment underlying a learning disability.

From a statistical standpoint VEP training had no other effect than improving performance on the Halstead-Reitan index. However, several factors need to be considered when looking at the test results. It should be pointed out that very small group sizes were employed thus making it difficult to obtain significant results even with fairly large differences between groups. While statistical significance is desirable, the implications of a study may not have to depend entirely on such findings. One has to ask, had the three groups employed had group sizes of 100 instead of 5 and 10 and had statistical significance been found for the same results, if the effects of

treatment would have been any more meaningful. The question really being asked by this study involves the ability of the treatment to help LD children. Taken in this context, it may be illuminating to look at the improvement shown by the treatment group over the other two groups irrespective of statistics.

Of the two measures most closely associated with the treatment, the treatment group showed large improvements over the NC group and sizable ones over the LDC group. For the Halstead-Reitan the treatment group showed more than a sevenfold improvement over the NC group and improved neurological functioning by 36% over the LDC group receiving remediation in the school. These changes were even larger for the Bender-Gestalt tests. The treatment group on this measure showed a tenfold increase over the NC group and an even greater improvement over the LDC group which showed absolutely no improvement at all. On the only other two measures (Table 11, p. 85 and Table 15, p. 88) out of the ten employed in which the treatment group improved over both the LDC and NC groups scores were much more evenly distributed. For the Spache oral reading the treatment group improved over the NC group by less than onefold and the treatment group improved over the LDC group by less than 16%. For the spelling scores on the WRAT the treatment group improved 2.7 times over the LDC group but only slightly more than twice over the NC group. On all other measures the treatment group improved less than the LDC or NC groups. It thus appears on measures closely associated with the training procedure that VEP training can produce significant or large increases in performance, especially when combined with academic remediation. It is of interest

to note that post-treatment scores would result in reclassifying the treatment group but not the LD control group from their initial category of LD to normal based on Halstead-Reitan Index for children.

In considering the clinical implications of this study it is important to have an understanding of the meaningfulness of the statistical findings concerning the changes seen in the Reitan scores observed between the treatment group and the no treatment group. At first glance it might appear that a large number of tests were conducted since there were 10 separate test scores each involving 3 comparisons. This would result in 30 tests and make it very difficult to obtain a significant result on any of the comparisons because of the necessity of protecting from type 1 errors with an extremely low alpha level. However, since Tukey's (HSD) already uses an adjusted alpha level for the number of comparisons made for each test, the number of tests conducted is immediately reduced from 30 to 10. This in itself, however, would not alleviate the problem since 10 tests would also require a fairly high level of significance to be meaningful. If, in fact, the 10 tests conducted on the psychometric data had been from the same data set one would not be justified in finding significant differences in any of the groups because of experimenterwise error rates (Meyers, 1979). However, in considering levels of probability one must obtain to achieve significance Meyers (1979) suggests that the most important factor is whether tests are done on the same source of variance (error rate experimenterwise) or on different sources of variance (error rate familywise). The importance of this distinction

for the present study is derived from the fact that each of the 10 psychometric tests involved a different data set and hence a different source of variance or family. Since only one test was conducted per family no adjusted alpha rates were necessitated. In effect, this results in the reported difference between groups on the psychometric tests being valid at the levels indicated. Specifically, in this case, one can have a reasonable amount of confidence that the difference found on the Reitan scores are quite meaningful at the .025 level of significance.

EEG Scores Prior to Treatment

A number of findings in the literature on the EEG's of learning disabled and normal children indicate that LD children display more slow activity than normals or less fast activity or a combination of these factors during various conditions. Figure 4 (p. 91) illustrates EEG differences found prior to any training for 18 LD children and 10 normal children between the ages of 7 and 11 years that participated in the initial portion of this study. In terms of power across frequency bands the LD children displayed more low frequency activity in the frontal regions than did the normal children during reading and drawing tasks. These findings concur with reports from similar studies comparing LD and normal children (Sklar et al., 1972; Douglas, 1978; Johnson, 1977). No such findings are shown for percentage power as shown in Figure 4. It would appear that the EEG power pattern displayed in the frontal areas by the LD children in this study replicates earlier reports of slowing when these children are compared to normals.

In addition to increases in low frequency activity Figure 4 (p. 91) also shows the LD subjects displayed an increase in middle frequency activity in the alpha or low beta range for both power and percent power data. A number of authors have suggested (Fuller, 1976; Grünewald-Zuberbier et al., 1978; Duffy et al., 1980) that LD children display increased alpha activity or lack of alpha blocking during a variety of tasks. The increases in 12-16 Hz activity shown in percent percentage power and 16-20 Hz activity for power may be a reflection of this increased alpha activity found in LD children, especially for the reading and drawing conditions. Although this activity is of a slightly higher frequency than the traditional alpha range (8-13 Hz), Dumermuth, Gasser, Hecker, Herdan, and Lange (1976) suggest that harmonics of the alpha rhythm can show up as beta activity in children with MBD syndrome. The increases in drawing shown for frequencies greater than 16 Hz in the percentage portion of Figure 4 are felt to be an artifact of muscle movements which are inevitable during any drawing task. Although the children were instructed to move as little as possible and were provided with feedback for EMG activity, increases in EMG levels were observed in the EEG tracings during drawing for many of the children. The attribution of these higher frequencies to muscle origin is further supported by their occurrence in all the frequency bands examined at a level greater than 16 Hz.

EEG Scores After Treatment

Figures 5-10 (pp. 93-98) show the differences observed in the EEG's of the LDC, treatment, and NC groups collected after treatment

from the EEG's of these groups collected prior to treatment. As can be seen, significant changes occurred at a number of locations and a number of frequencies for all three groups. It is perhaps not necessary to point out here that changes due solely to the training should occur in only the experimental group. Thus locations or frequencies in which a change in the power or percentage power of the experimental group was accompanied by a change in either the LDC or NC group must be attributed to nonspecific factors associated with the training but not to the specific training itself. Figure 11 (p. 102) shows that relatively few changes were shown by the experimental group only.

Taken by location, only one area of the brain seems to show any pattern of change after training. Fully 48% (11/23) of the changes occurred in the right occipital-parietal region. No other location showed more than two significant changes for any particular condition between pre treatment and post treatment EEG's. In general, right occipital parietal areas showed power decreases in the total power band and the 0-4 Hz band for both reading and drawing and in the 4-8 Hz band for drawing. Percentage change decreased for reading in the 0-4 Hz band and increased for drawing in the 4-8 Hz band and for reading in the 4-10 Hz band. Baseline increased in the 6-12 Hz bands. These changes are consistent with reports in the literature (i.e., Johnson, 1977; Sklar et al., 1976) which indicate desirable changes in behavior are accompanied by decreases in lower frequency activity and increases in faster activity. In addition, the increases in baseline alpha activity are supported as being beneficial and germane by reports of LD children displaying less alpha activity than normals during baseline

conditions (i.e., Colon et al., 1979). The only other area which showed any pattern of change was the right central-temporal region where (6-12 Hz) power during reading decreased. The remainder of the changes were scattered rather randomly across the other cortical locations with no changes at all occurring in the right frontal-temporal region. Overall then, it appears that VEP training had the most effect on the EEG in the right occipital-parietal and central-temporal regions.

In interpreting these results, however, a note of caution needs to be expressed. The EEG values for pre and post treatment differences were originally subjected to a six way analysis of variance. However, these results became extremely difficult to interpret because a number of the higher order interactions, including the six way interaction, turned out to be significant. Therefore, t tests were performed on each of the pre-post pairings to assess treatment effects. This resulted in a total of 408 tests. However, since each cortical location and each task can be considered to be a separate source of variance the number of tests conducted in a familywise fashion for each EEG location for each task is actually 17. Even so, this would result in a fairly high level of significance ($< .003$) being needed for actual significance at the .05 level. Therefore, the .05 level of significance was reported for each t test performed without consideration for the number of tests conducted per family.

When using an overall significance level of .05 one expects to obtain one significant result out of twenty by chance. In interpreting Table 11 (p. 85) one would thus expect to obtain approximately one

letter (regardless of whether it represents power or percent power) for each of the three tasks for each cortical location by chance. Taking this into consideration it would appear that the only change occurring at more than the chance level was for reading and drawing in the right occipital parietal area. All other locations appear to be operating at chance or only slightly higher levels. The fact that some locations showed patterns of change associated with reported improvements in learning disabilities perhaps serves to ameliorate this situation somewhat. However, the reported changes in the EEG found over treatment should more than likely only be interpreted as weak results perhaps indicative of a general trend at best.

Evoked Potential Training

A review of the evoked potential literature indicates that LD children differ from normal children in both latencies and amplitudes to a number of different types of stimuli. For instance, John (1977) found latency differences to patterns, letters, flashes and clicks. Of the articles reviewed earlier on learning disabilities and evoked potentials 15 reported specific differences in both amplitude and latency for LD children when compared to normals. These differences occurred for a variety of stimuli and fell into three general categories: 1) decreased amplitude and increased latency; 2) increased amplitude and decreased latency; and 3) increased amplitude and increased latency. These differences were found for peaks as early as P1 and as late as P3 or beyond. Sixty-seven percent of the studies reviewed

reported LD children to have decreased amplitudes and increased latencies. These findings occurred to simple visual and auditory stimuli as well as to more complex stimuli. Twenty percent of the studies reviewed reported increased amplitudes and increased latencies in LD children for both simple and complex stimuli and 13% or two studies reported increased amplitudes and decreased latencies to light flash presentations. No studies found LD children to have higher latencies and higher amplitudes than normal children. Because of the lack of standardization in the procedures used, the definitions of LD populations used, and the stimuli used to evoke cortical potentials, it is presently impossible to specify any particular set of parameters by which the evoked potential might represent neurological factors inherent in learning disabilities. In general, however, it can be said that the majority of findings report LD children as having increased latencies and decreased amplitudes to stimuli.

The design of the present study was based on the premise that most LD children decrease amplitudes to a variety of stimuli and that if they could learn to increase these amplitudes a concomitant improvement in academic performance might result. In order to attribute any changes in the dependent test measures to the treatment, it is first necessary to show that the subjects were able to alter the amplitude of their VEP's. Figure 12 (p. 106) shows the mean changes from the first baseline of the experimental portion of the study through the feedback portions and second baseline. As can be seen, the children did learn to significantly increase their VEP amplitudes for all trials following the initial baseline recordings. It would thus appear that VEP

biofeedback training can enable LD children to significantly increase the amplitude of their cortical evoked responses.

Although the experimental children were successfully able to increase the P300 component of their evoked potentials from the beginning to the end of each session Figure 13 (p. 107) shows that the children were also significantly decreasing the overall amplitude of their VEP's from the beginning to the end of the training period. The lack of any findings indicative of interaction effects derived from the two way analysis of variance of the data in Table 18 (p. 108) plus the results of the Pearson product-moment correlations in Table 19 (p. 109) indicate that within this general decline of amplitudes across time the children were always able to raise the amplitude of their VEP's when provided with feedback.

The fact that VEP amplitudes were increasing within sessions while decreasing across sessions lends itself to speculation about the effects of repeated exposure over time to the same stimulus set. A number of reports have indicated that some or all of the components of the event related potential (ERP) decline in amplitude as stimuli are successively and monotonously repeated (Regan, 1972, p. 134). At present it is unclear as to whether this decline represents adaptation effects involving the refractiveness of ERP generators (Ritter, Vaughan and Costa, 1968) or is due to habituation of cortical and neural activity (Fruhstorfer, Soveri, and Jarvilehto, 1970). For the most part these decreases occur within the first few repetitions (Fruhstorfer et al., 1970) and do not appear to involve psychological variables such as attention (Ritter et al., 1968; Barker and Gallaway,

1979). While such effects have been observed for the initial trials constituting a single ERP, no studies are known which address the issue of habituation or adaptation of the average evoked potential collected at regular intervals over a long period of time in humans. Yet it appears that some form of habituation or adaptation was occurring in the evoked potentials recorded from the beginning to the end of the study as shown in Figure 14 (p. 110). These decreases appear to have begun with the first baseline period and to have continued through the last baseline period. This suggests that the reductions seen in VEP amplitude may be independent of the experimental treatment. In any case, this phenomenon of continued reductions in VEP amplitude over time to a repeated stimulus set seems to warrant further research as it may offer a new means for examining neurological integrity and function.

Even though the subjects were able to significantly change VEP amplitudes no such findings were found for latencies. Since a number of authors (i.e., Satterfield, 1973) have indicated that both amplitudes and latencies differ in LD children it would seem reasonable to expect that latency changes might accompany changes in amplitude. The question as to whether latencies co-vary with amplitudes during amplitude training or whether they can be disassociated cannot be answered, however, by this study. Because of the design of the study latencies were not free to vary but were held rather constant about initial baseline levels. The subjects received maximum reward for increasing amplitudes within a fixed latency range after the beginning of each session. Thus, the only time latencies were free to change was during the first baseline

period of each training session. While it is possible to say that latencies between sessions did not change it cannot be determined how latencies might have changed within sessions had they been able to vary freely with changes in amplitude. Additional studies on amplitude and latency training are needed before this relationship between amplitude and latency can be understood better.

In considering the increases seen in the evoked potential amplitudes for the experimental group, one might legitimately speculate about the size of these increases in terms of their ability to be clinically significant in spite of statistical significance. One way to assess this question might be to measure the changes in the children's ability to increase their VEP amplitudes over time. One could do this by taking the initial value obtained from each of the blocks for baseline one in Table 18 (p. 108) and comparing it to the highest value obtained over the next three conditions for each block in Table 18. If children are only able to raise their VEP amplitudes a fixed amount, which does not change from the first block of time to the last block of time, then it may be that VEP amplitude changes only have limited clinical significance due to a ceiling effect restricting the amount of change that can occur in cortical activity. On the other hand, if increases in VEP amplitude changes are not fixed, but increase over time, then changes in VEP moderated cortical activity would not be seen as restricted, giving more clinical relevance to VEP feedback procedures.

Table 18 indicates that VEP amplitude increases are not fixed but vary systematically over time. By comparing first baseline values to feedback or second baseline values one finds a general increase

in the amount of increase seen over time. Specifically, children increased VEP amplitudes by 1.05 uv in block one, 1.37 uv in block two, .90 uv in block three, and 1.54 uv in block four. Although block three showed a decrease in amplitude increase, the general trend indicates increasing VEP amplitude differences over time. In addition, because initial baseline amplitudes were decreasing over sessions, the percentage of change seen in the increase in VEP amplitudes is greater than absolute uv values might indicate. If one were to look at percentage values, increases in VEP amplitude from block one through block four would be 18.3%, 26.8%, 21.09%, and 37.9%. Overall, the experimental children learned to increase their absolute initial baseline uv levels by 46.6% during the final sessions when compared to increases seen in initial sessions. It would thus appear that the effects of feedback on modulating VEP amplitudes were cumulative in that subjects became better at increasing their initial baseline amplitudes over sessions and that they were not limited in the amount of change they could produce. However, the absolute increase uv values needed for clinical significance cannot be determined from this study and assessments of this nature must await future investigations.

Conclusion

In conclusion this study suggests a number of directions for future research. First, since any changes in cerebral activity elicited by VEP training appears to be reflected best in basic measures of sensation, perception, and neurological functioning additional tests

in these areas might be added in future investigations. In addition, it appears that the increases seen between the two feedback periods and the initial baseline are approximately equal. This leads to the possibility that the training sessions could be shortened without affecting the efficiency of the training. With regard to the EEG results one has to be somewhat curious as to all the significant changes which occurred in the normal comparison and LD control groups. It would appear that there are nonspecific factors operating which certainly deserve investigation. Finally, the appearance of what may be a novel event perhaps offers the most impetus for future research. The finding of significant reductions in the VEP recorded across a number of days may be common only to LD children or may differ between normal and disadvantaged groups of both children and adults. It may also differ under different conditions and tasks. Indeed, one can think of any number of ways in which it might be possible to use ERP's over time as a tool for investigating brain functioning and cortical activity. In summary then, it would appear that LD children can learn to alter their VEP's and that this may be reflected in measures of basic cerebral activity.

BIBLIOGRAPHY

BIBLIOGRAPHY

Ables, B. S., Aug, R. G., and Loof, D. H. Problems in diagnosis of dyslexia: a case study. Journal of Learning Disabilities, 1971, 4, 409-417.

Ackerman, P. T., Peters, J. E., and Dykman, R. A. Children with specific learning disabilities: WISC profiles. Journal of Learning Disabilities, 1971, 4, 150-166.

Adelman, H. S. Diagnostic classification of LD: research and ethical perspectives as related to practice. Learning Disability Quarterly, 1979, 2, 5-15 (a).

Adelman, H. S. Diagnostic classification of LD: a practical necessity and a procedural problem. Learning Disabilities Quarterly, 1979, 2, 56-62 (b).

Adelman, H. S. Predicting psycho-educational problems in childhood. Behavioral Disorders, 1978, 3, 148-159.

Adelman, H. S. Learning problems: Part I. An interactional view of causality. Academic Therapy, 1970-71, 6, 117-123.

Admunson, M. S. A preliminary screening program to identify functional strengths and weaknesses in pre school children. ERIC Document #071743, 1973.

Adrian, E. D. The physiological basis of attention. In J. F. Delafresnaye (Ed.), Brain mechanisms and consciousness. Oxford: Blackwell, 1954.

Ahn, H. Electroencepholographic evoked potential comparisons of normal children and children with differential modes of underachievement. Dissertation Abstracts International, 1978, 38 (7-B), 3453.

Aird, R., and Gastaut, Y. Occipital and posterior slow rhythms. Electroencephalography and Clinical Neurophysiology, 1959, 11, 637-656.

Alley, G. R., Deshler, D. D., and Warner, M. M. Identification of learning disabled adolescents: a bayesian approach. Learning Disability Quarterly, 1979, 2, 76-83.

Anderson, M., Kaufman, A. S., and Kaufman, N. L. Use of the WISC-R with a learning disabled population: some diagnostic implications. Psychology in the Schools, 1976, 13, 381-386.

Ayres, A. J. Reading--a product of sensory integrative processes. In H. K. Smith (Ed.), Perception and reading. Newark: International Reading Association, 1968.

Ayres, J. A. Sensory integration and learning disorders. Los Angeles: Western Psychological Service, 1972.

Banas, N., and Wills, I. H. Prescriptions from WISC-R patterns. Academic Therapy, 1979, 13, 224-246.

Bannatyne, A. Diagnosis: a note on recategorization of the WISC scaled scores. Journal of Learning Disabilities, 1974, 7, 272-273.

Barber, C., and Galloway, N. R. Adaptation effects in the transient visual evoked potential. In D. Lehmann and E. Callaway (Eds.), Human evoked potentials: applications and problems. New York: Plenum Press, 1979.

Barsch, R. H. Achieving perceptual motor efficiency. Seattle: Special Child Publications, 1967.

Bateman, B. Three approaches to diagnosis and educational planning for children with learning disabilities. Academic Therapy, 1966-67, 2, 215-222.

Bateman, B. L., and Schiefelbusch, R. L. Section II. Minimal brain dysfunction. National Project on Learning Disabilities in Children, Phase Two. Public Health Publication No. 2015, p. 7, 1969.

Belmont, L. Epidemiology. In H. E. Rie and E. D. Rie (Eds.), Handbook of minimal brain dysfunction: a critical review. New York: John Wiley and Sons, 1974.

Benton, A. L. Clinical neuropsychology of childhood: an overview. In R. M. Reitan and L. A. Davison (Eds.), Clinical neuropsychology: current status and applications. New York: John Wiley and Sons, 1974.

Birch, H. G. The problem of "brain damage" in children. In H. G. Birch (Ed.), Brain damage in children: the biological and social aspects. Baltimore: Williams and Wilkins, 1964.

Birch, H., and Gussow, J. Disadvantaged children: health, nutrition, and school failure. New York: Grune and Stratton, 1970.

Broadbent, D. E. Perception and communication. Oxford: Permagon Press, 1958.

Broadbent, D. E. Stimulus set and response set. Two kinds of selective attention. In D. I. Mostofsky (Ed.), Contemporary theory and analysis. New York: Appleton-Century-Crofts, 1970.

Broadbent, D. E. Decision and stress. New York: Academic Press, 1971.

Broca, P. Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie (perte de la parole). Bulletin de la Société Anatomique de Paris (2nd Series), 1861, 6, 330-357.

Buchsbaum, M., and Wender, P. Average evoked responses in normal and minimally brain dysfunctioned children treated with amphetamine. Archives of General Psychiatry, 1973, 29, 764-770.

Burgess, P. R. Specific regeneration of cutaneous fibers in the cat. Journal of Neurophysiology, 1973, 36, 101-114.

Bush, W. J., and Waugh, K. W. Diagnosis of learning disabilities. Columbus: Charles Merrill, 1976.

Butler, S. R., and Glass, A. Asymmetries in the electroencephalogram associated with cerebral dominance. Electroencephalography and Clinical Neurophysiology, 1974, 36, 481-491.

Burnett, L. L., and Struve, F. A. The value of EEG in minimal brain dysfunction. Journal of Clinical Psychology, 1974, 30, 489-495.

Callaway, E. Brain electrical potentials and individual differences. New York: Grune and Stratton, 1975.

Capute, A. J., Niedermeyer, E. F., and Richardson, F. The electroencephalogram in children with minimal cerebral dysfunction. Pediatrics, 1968, 41, 1104-1114.

Carter, J. L., and Russell, A. L. Changes in verbal-performance I.Q. discrepancy scores of the left hemisphere EEG frequency control training: a pilot report. Americal Journal of Clinical Biofeedback, 1981, in press.

Carter, J. L., and Russell, H. L. EEG alpha and beta training for learning disabled boys with intellectual verbal-performance I.Q. discrepancies (unpublished manuscript), 1981b.

Chalfant, J., and Scheffelin, M. Central processing dysfunction in children: a review of research. National Institute of Neurological Disease and Stroke, Monograph No. 9. Washington, D.C.: Department of Health, Education and Welfare, 1969.

Chapman, L., and Wedell, K. Perceptual motor abilities and reversal errors in children's handwriting. Journal of Learning Disabilities, 1972, 5, 321-325.

Chapman, R. M., McCrary, J. W., Bragdon, H. R., and Chapman, J. A. Latent components of event-related potentials functionally related to information processing. In J. E. Desmedt (Ed.), Progress in clinical neurophysiology (Vol. VI). Bassel: Karger, 1979.

Clements, S. D. Minimal brain dysfunction in children--terminology and identification (NINDB Monograph No. 3). Washington, D.C.: U.S. Public Health Service, 1966.

Clements, S. D. Minimal brain dysfunction in children--terminology and identification. U.S. Public Health Service Publication No. 1415. Washington, D.C., 1969.

Clements, S. D., and Peters, J. E. Minimal brain dysfunction in the school age child. Archives of General Psychiatry, 1962, 6, 185.

Cohn, R., and Nardini, J. The correlation of bilateral occipital slow activity in the human EEG with certain disorders of behavior. American Journal of Psychiatry, 1958, 115, 44-54.

Coles, G. S. The learning disabilities test battery: empirical and social issues. Harvard Educational Review, 1978, 48, 313-340.

Colon, E. J., Notermans, S. L., de Weerd, J. P., and Kap, J. The discriminating role of EEG spectra in dyslexic children. Journal of Neurology, 1979, 221, 257-262.

Conners, C. K. Cortical visual evoked responses in children with learning disorders. Psychophysiology, 1970, 7, 418-428.

Conners, C. K. Stimulant drugs and cortical evoked responses in learning and behavior disorders in children. In W. L. Smith (Ed.), Drugs, development and cerebral function. Springfield: Charles Thomas, 1972.

Conners, C. K. Psychological assessment of children with minimal brain dysfunction. Annals of New York Academy of Sciences, 1973, 205, 283-302.

Conners, C. K., Rothschild, O., Eisenberg, L., Schwartz, L. S., and Robinson, E. Dextroamphetamine sulfate in children with learning disorders. Archives of General Psychiatry, 1969, 21, 182-190.

Courchesne, E., Hillyard, S. A., and Galambos, R. Stimulus novelty, task relevance and the visual evoked potential in man. Electroencephalography and Clinical Neurophysiology, 1975, 39, 131-143.

Coursin, D. B. Vitamin difficiencies and developing mental capacity. In N. Scrimshaw and J. Gordon (Eds.), Malnutrition, learning, and behavior. Cambridge: M.I.T. Press, 1968.

Cratty, B. J. Psychology and physical activity. Englewood Cliffs: Prentice Hall, 1968.

Cratty, B. Perceptual-motor behavior and educational processes. Springfield: Charles C. Thomas, 1969.

Cronbach, L. J. Five decades of public controversy over mental testing. American Psychologist, 1975, 307, 1-14

Cunningham, M. D. The effects of bilateral EEG biofeedback on verbal, visual-spatial, and creative skills in learning disabled male adolescents. Dissertation Abstracts International, 1978, 39 (1-B), 373.

Daryn, E. Problems of children with "diffuse brain damage." Archives of General Psychiatry, 1960, 4, 299.

Debecker, J., and Desmedt, J. E. Rate of intermodality switching disclosed by sensory evoked potentials averaged during signal detection task. Journal of Physiology, 1966, 185, 52-53.

de Hirsch, K., Jansky, J., and Langford, W. S. Predicting reading failure. New York: Harper and Row, 1966.

Delacato, C. H. Neurological organization and reading. Springfield: Charles C. Thomas, 1966.

Desmedt, J. E., and Debecker, J. Wave form and neural mechanisms on the decision P350 elicited without pre-stimulus CNV or readiness potential in random sequences of near-threshold auditory clicks and finger stimuli. Electroencephalography and Clinical Neurophysiology, 1979, 47, 648-670.

Deutsch, J. A., and Deutsch, D. Attention: some theoretical considerations. Psychological Review, 1963, 70, 80-90.

Doll, E. Neurophrenia. American Journal of Psychiatry, 1951, 108, 50-53.

Donchin, E., Tueting, P., Ritter, W., Kutos, M., and Heffley, E. On the independence of the CNV and P300 components of the human average evoked potential. Electroencephalography and Clinical Neurophysiology, 1975, 38, 449-462.

Douglas, M. V. Behavioral and electrophysiological correlates of visual motor performance in normal and learning disabled boys. Dissertation Abstracts International, 1978, 39 (4-A), 2125.

Douglas, V. I. Stop, look and listen: the problem of sustained attention and impulse control in hyperactive and normal children. Canadian Journal of Behavioral Science, 1972, 4, 259-281.

Doyle, J. C., Ornstein, S., and Galin, D. Lateral specialization of cognitive mode: II. EEG frequency analysis. Psychophysiology, 1974, 11, 567-578.

Dubey, R. Organic factors in hyperkinesis: a critical review. American Journal of Orthopsychiatry, 1976, 46(2), 353-366.

Duffy, F. H., Denkla, M. B., Bartels, P. H., and Sandini, G. Dyslexia: regional differences in brain electrical activity by topographical mapping. Annals of Neurology, 1980, 7, 412-420.

Dumermuth, G., Gasser, T., Hecker, A., Herdan, M., and Lange, B. Exploration of EEG components in the beta frequency range. P. Kellaway and I. Petersen (Eds.), Quantitative analytic studies in epilepsy. New York: Raven Press, 1976.

Dykman, R. A., Ackerman, P. T., Clements, S. D., and Peters, J. E. Specific learning disabilities: an attentional deficit syndrome. In H. R. Myklebust (Ed.), Progress in learning disabilities (Vol. 2). New York: Grune and Stratton, 1971.

Dyment, P. G., Lattin, J. E., and Herbertson, L. M. The value of the electroencephalogram in evaluating children with minimal cerebral dysfunction. Journal of School Health, 1971, 41, 9-11.

Eaves, L. C., Kendall, D. C., and Crichton, M. B. The early detection of minimal brain dysfunction. Journal of Learning Disabilities, 1972, 5, 5-13.

Eaves, L. C., Kendall, D. C., and Crichton, M. B. The early identification of learning disabilities: a follow-up study. Journal of Learning Disabilities, 1974, 7, 42-48.

Ehrlichman, H., and Wiener, M. S. EEG asymmetry during covert mental activity. Psychophysiology, 1980, 17, 228-235.

Ekwall, E. E. The use of WISC subtest profiles in the diagnosis of reading difficulties. Dissertation Abstracts International, 1966, 27, 950-951.

Erdely, M. H. A new look at the new look: perpetual defense and vigilance. Psychological Review, 1974, 81, 1-25.

Ertl, J. Research Newsletter No. 1. Neural Models Limited, Toronto, 1975.

Ertl, J., and Douglass, V. Evoked potential and dyslexia. Neural Models Limited, Toronto, 1970.

Evans, J. R., Martin, D., and Hatchette, R. Neural efficiency analyzer scores of reading disabled, normally reading, and academically gifted children. Perceptual Motor Skills, 1976, 43, 1248-1250.

Feeler, T. J. A comparison of Wechsler Intelligence Scale for Children scores of specific learning disabled and reading disabled students. Dissertation Abstracts International, 1975, 36A, 1432-1433.

Feingold, B. F. Why your child is hyperactive. New York: Random House, 1975.

Fenelon, B., Holland, J. T., and Johnson, C. Spatial organization of the EEG in children with reading disabilities: a study using nitrazepan. Cortex, 1972, 8, 444-464.

Finley, W. W., Smith, H. A., and Etherton, M. D. Reduction of seizure and normalization of the EEG in a severe epileptic following sensorimotor biofeedback training: preliminary study. Biological Psychology, 1975, 2, 189-203.

Ford, J. M., Roth, T. W., Dirks, S. J., and Kopell, B. S. Evoked potential correlates of signal recognition between and within modalities. Science, 1973, 181, 465-466.

Friedman, D., Hakerem, G., Sutton, S., and Fleiss, J. L. Effect of stimulus uncertainty on the pupillary dilation response and the vertex evoked potential. Electroencephalography and Clinical Neurophysiology, 1973, 34, 475-484.

Frostig, M. A developmental test of visual perception of evaluating normal and neurologically handicapped children. Perceptual and Motor Skills, 1963, 12, 383-394.

Frostig, M. Education for children with learning disabilities. In H. Myklebust (Ed.), Progress in learning disabilities. New York: Grune and Stratton, 1968.

Fruhstorfer, H., Soveri, P., and Järvilehto, T. Short term habituation of the auditory evoked response in man. Electroencephalography and Clinical Neurophysiology, 1970, 28, 153-161.

Fuller, P. W. Computer estimated alpha attenuation during problem solving in children with learning disabilities. Electroencephalography and Clinical Neurophysiology, 1977, 42, 149-156.

Galin, D., and Ornstein, R. Lateral specialization of cognitive mode: an EEG study. Psychophysiology, 1972, 9, 412-419.

Gallagher, J. J., and Bradley, R. H. Early identification of developmental difficulties. In I. J. Gorden (Ed.), Early childhood education. Chicago: University of Chicago Press, 1972.

Gerson, I. M., Barnes, T. C., Mannino, A., Fanning, T. M., and Burns, J. J. EEG of children with various learning problems. Diseases of the Nervous System, 1977, 33, 170-177.

Getman, G. N. The visuomotor complex in the acquisition of learning skills. In J. Hellmuth (Ed.), Learning disorders, Vol. I. Seattle: Special Child Publications, 1965.

Goff, J. R. A neurological approach to the learning disabled child. In M. I. Gottlieb, P. W. Zinkus, and L. J. Bradford (Eds.), Current issues in developmental pediatrics: the learning disabled child. New York: Grune and Stratton, 1979.

Golden, C. J., and Anderson, S. Learning disabilities and brain dysfunction: an introduction for educators and parents. Springfield: Charles Thomas, 1979.

Gordon, R. L. WISC-R patterns in diagnosis and remediation of learning disabled children. Dissertation Abstracts International, 1978, 38A, 4684.

Greer, F. S. A study of the Wechsler Intelligence Scale for Children in a reading clinic population. Ann Arbor: University Microfilms #72-34,077, 1972.

Grünewald, G., Grünewald-Zuberbier, E., and Netz, J. Late components of average evoked potentials in children with different abilities to concentrate. Electroencephalography and Clinical Neurophysiology, 1978, 44, 617-625.

Grünewald-Zuberbier, E., Grünewald, G., Rasche, A., and Freiburg, U. Hyperactive behavior and EEG arousal reactions in children. Electroencephalography and Clinical Neurophysiology, 1975, 38, 149-159.

Grünewald-Zuberbier, E., Grünewald, G., Rasche, A., and Netz, J. Contingent negative variation and alpha attenuation responses in children with different abilities to concentrate. Electroencephalography and Clinical Neurophysiology, 1978, 44, 37-47.

Guilford, J. P., and Fruchter, B. Fundamental statistics in psychology and education. New York: McGraw-Hill, 1973.

Hallahan, D. P., and Cruickshank, W. M. Psychoeducational foundations of learning disabilities. Englewood Cliffs: Prentice Hall, 1973.

Hallahan, D. P., and Kauffman, J. M. Introduction to learning disabilities. Englewood Cliffs: Prentice Hall, 1976.

Halliday, R., Rosenthal, J. H., Naylor, H., and Calloway, E. Averaged evoked potential predicters of clinical improvement in hyperactive children treated with methylphenidate: an initial study and replication. Psychophysiology, 1976, 13, 429-440.

Hanna, G. S., Dyck, N. J., and Holen, M. C. Objective analysis of achievement-aptitude discrepancies in LD classification. Learning Disabilities Quarterly, 1979, 2, 32-38.

Harter, M. R., and Previc, F. H. Size-specific information channels and selective attention: visual evoked potential and behavioral measures. Electroencephalography and Clinical Neurophysiology, 1978, 45, 628-640.

Hartley, L. P. The effect of stimulus relevance on the cortical evoked potentials. Quarterly Journal of Experimental Psychology, 1970, 22, 531-546.

Haring, N. G., and Bateman, B. D. Section I. Minimal brain dysfunction. National project on learning disabilities in children. Phase two. Public Health Publication No. 2015, pp. 1-4, 1969.

Harvard, J. School problems and allergies. Journal of Learning Disabilities, 1973, 6, 492-494.

Hebb, D. O. The organization of behavior. New York: John Wiley and Sons, 1949.

Helper, M. M. Follow-up of children with minimal brain dysfunctions: outcomes and predictors. In H. E. Rie and E. D. Rie (Eds.), Handbook of minimal brain dysfunction: a critical review. New York: John Wiley and Sons, 1980.

Hernandez-Peon, R. Reticular mechanisms of sensory control. In W. A. Rosenblith (Ed.), Sensory communication. New York: John Wiley and Sons, 1961.

Hertzig, M. F., Bortner, M., and Birch, H. G. Neurological findings in children educationally designated as "brain damaged." American Journal of Orthopsychiatry, 1969, 39, 437.

Hillyard, S. A., Hink, R. F., Schwent, V. L., and Picton, T. W. Electrical signs of selective attention in the human brain. Science, 1973, 182, 177-180.

Hillyard, S. A., and Picton, T. W. Event related brain potentials and selective information processing in man. In J. Desmedt (Ed.), Progress in clinical neurophysiology (Vol. 6). Bassel: Karger, 1979.

Hillyard, S. A., Picton, T. W., and Regan, D. Sensation, perception and attention: analysis using ERP's. In E. Callaway, P. Tueting, and S. H. Koslow (Eds.), Event related brain potentials in man. New York: Academic Press, 1978.

Hink, R. F., and Hillyard, S. A. Auditory evoked potentials during selective listening to dichotic speed messages. Perceptual Psychophysiology, 1976, 20, 236-242.

Hink, R. F., Hillyard, S. A., and Benson, P. J. Event related brain potentials and selective attention to acoustic and phonetic cues. Biological Psychology, 1978, 6, 1-16.

Hink, R. F., Van Voorhis, S. T., Hillyard, S. A., and Smith, T. S. The division of attention and the human auditory evoked potentials. Neuropsychologia, 1977, 15, 597-605.

Hinshelwood, J. Congenital word blindness. London: H. K. Lewis, 1917.

Hirshoren, A., and Kavale, K. Profile analysis of WISC-R: a continuing malpractice. Exceptional Child, 1976, 23, 83-87.

Hughes, J. R. Electroencephalography and learning disabilities. In H. R. Myklebust (Ed.), Progress in learning disabilities (Vol. II). New York: Grune and Stratton, 1971.

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

Hughes, J. R., and Park, G. E. The EEG in dyslexia. In P. Kellaway and I. Peterson (Eds.), Clinical electroencephalography in children. New York: Grune and Stratton, 1968.

Hutcherson, R. R. Differentiating MR and LD groups by WISC-R profile analysis. Educational Resources Information Center, Document #ED 140154, 1971.

Ingram, T. T. Soft signs. Developmental Medicine and Child Neurology, 1973, 15, 527.

Inman, R. L., and Conover, W. J. A modern approach to statistics. New York: John Wiley and Sons, 1983.

Jansky, J., and de Hirsch, K. Preventing reading failure: prediction, diagnosis and intervention. New York: Harper and Row, 1973.

John, E. R. Functional neuroscience (Vol. II). New York: John Wiley and Sons, 1977.

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

Johnson, P. A. Longitudinal analysis of EEG behavior in learning disabled children. Dissertation Abstracts International, 1977, 28 (4-B), 1942.

Johnson, S. The marginal child: workshop proceedings. State University of New York at Plattsburgh, Plattsburgh, 1962.

Kahneman, D. Attention and effort. Englewood Cliffs: Prentice Hall, 1973.

Kamiya, J. Conditioned discrimination of the EEG alpha rhythm in humans. Paper presented at the Western Psychological Association. San Francisco, 1962.

Kamiya, J. Conscious control of brain waves. Psychology Today, 1968, 1, 57-60.

Karlin, L. Cognition, preparation and sensory evoked potentials. Psychological Bulletin, 1970, 73, 122-136.

Kaufman, A. S. Factor analysis of the WISC-R at 11 age levels between 6 1/2 and 16 1/2 years. Journal of Consulting and Clinical Psychologists, 1975, 43, 135-147.

Kennard, M., Rabinowitch, R. D., and Wexler, D. The abnormal electroencephalogram as related to learning disability in children. Canadian Medical Association Journal, 1952, 67, 330.

Keogh, B. K., and Becker, L. D. Early detection of learning problems: questions, cautions, and guidelines. Exceptional Children, 1973, 40, 5-10.

Keogh, B. K., Wetter, J., McGinty, A., and Donlon, G. Functional analysis of WISC performance of learning-disordered, hyperactive, and mentally retarded boys. Psychology in the Schools, 1973, 10, 178-181.

Kephart, N. C. The slow learner in the classroom. Columbus: Charles Merrill, 1960.

Kephart, N. C. Perceptual-motor aspects of learning disabilities. In E. Frierson and N. Barbe (Eds.), Educating children with learning disabilities. New York: Appleton-Century-Crofts, 1967.

Kirk, S. A. Behavioral diagnosis and remediation of learning disabilities. Proceedings of the Annual Meeting Conference on Exploration into Problems of the Perceptually Handicapped Child, Evanston, Ill. (Vol. 1), pp. 1-7, 1963.

Kirk, S. The ITPA: its origins and implications. In J. Hellmuth (Ed.), Learning disorders (Vol. 3). Seattle: Special Child Publications, 1967.

Kirk, S. A., McCarthy, J. J., and Kirk, W. D. Examiner's manual, Illinois Test of Psychological Abilities. Urbana: University Press, 1968.

Klorman, R., Salzman, L. F., Pass, H. L., Borgstedt, D. D., and Dainer, K. B. Effects of methylphenidate on hyperactive children's evoked responses during passive and active attention. Psychophysiology, 1979, 16, 23-29.

Koppitz, E. M. The Bender Gestalt Test for young children. New York: Grune and Stratton, 1963.

Koppitz, E. M. Brain damage, reading disability and the Bender Gestalt Test. Journal of Learning Disabilities, 1970, 3, 6-10.

Koppitz, E. M. Strategies for diagnosis and identification of children with behavior and learning problems. Behavioral Disorders, 1977, 2, 136-140.

Kuhlman, W. N., and Kaplan, B. T. Clinical applications of EEG feedback training. In R. J. Gatchel and K. P. Price (Eds.), Clinical applications of biofeedback: appraisal and status. New York: Pergamon Press, 1979.

Lairy, G. C., Rémond, A., Rieger, H., and Lesèure, N. The alpha average. 3 clinical applications in children. Electroencephalography and Clinical Neurophysiology, 1969, 26, 453-467.

Lairy, G. C. EEG of children with behavioral disorders. Proceedings of 2nd advanced course in electroencephalography and clinical neurophysiology. Vienna: Salzburg Academy Medical Publishing Department, 1965.

Lashley, K. S. In search of the engram. Symposia of the Society for Experimental Biology, 1950, 4, 454-482.

Leifer, L. T., Otto, D. A., Hart, S. C., and Huff, E. M. Slow potential correlates of predictive behavior during a complex learning task. In W. C. McCallum and J. R. Knott (Eds.), The responsive brain. Bristol: John Wright, 1976.

Levitsky, L. D. Ill-nourished brains. Natural History, 1976, 85, 8-16.

Lindsley, D. B. The reticular activating system and perceptual integration. In D. E. Sheer (Ed.), Electrical stimulation of the brain. Austin: University of Texas Press, 1961.

Loiselle, D. L., Stamm, J. S., Maitinsky, S., and Whipple, S. C. Evoked potential and behavioral signs of attentive dysfunctions in hyperactive boys. Psychophysiology, 1980, 17, 193-201.

Lombroso, C. T. The CNV in tasks requiring choice. In C. R. Evans and T. B. Mullholland (Eds.), Attention in neurophysiology. London: William Clowes, 1969.

Lubar, J. F., and Bahler, W. W. Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. Biofeedback and Self Regulation, 1976, 1, 77-104.

Lubar, J. F., and Deering, W. M. Behavioral approaches to neurology. New York: Academic Press, 1981.

Lubar, J. F., Shabsin, H. S., Natelson, S. E., Holder, G. S., Whitsett, S. F., Pamplin, W. E., and Krulikowski, D. I. EEG operant conditioning in intractable epileptics. Archives of Neurology, 1981, 38, 700-704.

Lubar, J. F., and Shouse, M. N. Use of biofeedback in the treatment of seizures, disorders and hyperactivity. In B. B. Lahey and A. A. Kazdin (Eds.), Advances in clinical child psychology, Vol. 1. New York: Plenum Press, 1977.

Lubar, J. F., and Shouse, M. N. EEG behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): a preliminary report. Biofeedback and Self Regulation, 1976, 1, 293-301.

Lubar, J. O. Management of specific learning disabilities and hyperkinesis in children using biofeedback and behavioral techniques. Paper presented at the 11th annual meeting of the Biofeedback Society of America, 1980.

Ludlum, W. M. Visual training, the alpha activation cycle and training. Journal of the American Optometrical Association, 1979, 50, 111-115.

Lynch, G., and Wells, J. Neuroanatomical plasticity and behavioral adaptability. In T. J. Teyler (Ed.), Brain and learning, Dordrecht: Reidel, 1978.

Martinius, J. W., and Hoovey, Z. B. Bilateral synchrony of occipital alpha waves, occulomotor activity and "attention" in children. Electroencephalography and Clinical Neurophysiology, 1972, 32, 349-356.

McCarthy, J. J., and McCarthy, J. F. Learning disabilities. Boston: Allyn and Bacon, 1969.

McCarthy, J. M. Personal communication, 1974, cited in Bush, W. J. and Waugh, K. W., Diagnosing learning disabilities. Columbus: Charles Merrill, 1976.

Mechelse, K., van Gemund, J. J., Nije, J. D., Burg, L. V. D., and Laurs, J. Visual and quantitative analysis of EEGs of normal school children with specific reading disabilities (SRD). Electroencephalography and Clinical Neurophysiology, 1975, 39, 106-107.

Mercer, C. D., Alqozzine, B., and Trifilletti, J. Early identification, an analysis of the research. Learning Disability Quarterly, 1979, 2, 12-24.

Miller, M., and Brecht, R. D. WISC subtest patterns in two learning disabilities. Diagnostique, 1978, 2, 1072.

Miller, M., Stoneburner, R. L., and Brecht, R. D. WISC subtest pattern as discriminators of perceptual disabilities. Journal of Learning Disabilities, 1978, 11, 449-452.

Minimal brain dysfunction national project on learning disabilities in children. Task Force II. U.S. Public Health Service Publication No. 2015, 1969.

Montagu, J. D. The hyperactive child: a behavioral electrodermal and EEG investigation. Developmental Medicine and Child Neurology, 1975, 17, 299-305.

Moray, N. A data base for theories of selective listening. In P. M. A. Rabbit and S. Dornic (Eds.), Attention and performance (Vol. V). New York: Academic Press, 1975.

Morgan, W. P. A case of congenital word blindness. British Medical Journal, 1896, 2, 1612.

Muehl, S., Knott, J. R., and Benton, A. L. EEG abnormality and psychological test performance in reading disability. Cortex, 1965, 1, 434.

Muso, M. F., and Harter, M. R. Contingent negative variation, evoked potential and physiological measures of selective attention in children with learning disabilities. In D. A. Otto (Ed.), Multidisciplinary perspectives in event related brain potential research. Washington, D.C.: U.S. Environmental Protection Agency, 1978, 300-302.

Myers, J. L. Fundamentals of experimental design, third edition. Boston: Allyn and Bacon, 1979.

Myers, P., and Hammill, D. Methods for learning disorders. New York: John Wiley and Sons, 1969.

Myklebust, H. R., and Boshes, B. Minimal brain damage in children. Washington, D.C.: U.S. Government Printing Office, 1970.

Näätänen, R. Selective attention and evoked potentials in humans--a critical review. Biological Psychology, 1975, 2, 237-307.

Näätänen, R. Selective attention and evoked potentials. Annales Academie Scientiarum Fennicae, 1967, B151, 1, 1-26.

Näätänen, R., Gaillard, A. W. K., and Montysalo, S. The N1 effect of selective attention reinterpreted. Acta Psychologia, 1978, 42, 313-329.

Näätänen, R., and Miche, P. T. Different variants of endogeneous negative brain potentials in performance situations: a review and classification. In D. Lehmann and E. Callaway (Eds.), Human evoked potentials. New York: Plenum Press, 1979.

National Advisory Committee on Handicapped Children. Conference sponsored by Bureau of Education for the Handicapped, U.S. Office of Education, Washington, D.C., 1967.

Norman, D. A. Toward a theory of memory and attention. Psychological Review, 1968, 75, 522-536.

Orton, S. T. Reading, writing and speech problems in children. New York: Norton, 1937.

Osborne, K., and Gale, A. Belated EEG differentiation of stimuli. Biological Psychology, 1976, 4, 185-196.

Pavy, R., and Metcalfe, J. The abnormal EEG in childhood communication and behavior abnormalities. Electroencephalography and Clinical Neurophysiology, 1965, 19, 414.

Penn, J. M. Reading disabilities: a neurological deficit? Exceptional Children, 1966, 33, 243.

Peters, J. E., Romine, J. S., and Dykman, R. A. A special neurological examination of children with learning disabilities. Developmental Medicine and Child Neurology, 1975, 17, 63.

Peters, J. F., Billinger, T. W., and Knott, J. R. Event related potentials of brain (CNV and P300) in a paired associate learning paradigm. Psychophysiology, 1977, 14, 579-585.

Pettet, J. M., and Helms, S. B. Hemispheric language dominance of language-disordered, articulation-disordered, and normal children. Journal of Learning Disabilities, 1979, 12, 12-17.

Piggot, L. R., Hyman, L., Kempler, H. L., Perez-Borja, C., and Cutler, C. Stressful school work: an agent of EEG deterioration. Journal of Learning Disabilities, 1972, 5, 61-67.

Picton, T. W., and Hillyard, S. A. Human auditory evoked potentials. II. Effects of attention. Electroencephalography and Clinical Neurophysiology, 1974, 36, 191-200.

Picton, T. W., Hillyard, S. A., Galambos, R., and Schiff, M. Human auditory attention: a central or peripheral process? Science, 1971, 171, 351-353.

Poon, L. W., Thompson, L. W., and Marsh, G. R. Average evoked potential changes as a function of processing complexity. Psychophysiology, 1976, 13, 43-49.

Preston, M. S., Guthrie, J. T., and Childs, B. Visual evoked responses (VER's) in normal and disabled readers. Psychophysiology, 1974, 11, 452-457.

Prichep, L. S., Sutton, S., and Hakerem, G. Evoked potentials in hyperkinetics and normal children under certainty and uncertainty: a placebo and methylphenidate study. Psychophysiology, 1976, 13, 419-428.

Ramanauskas, S., and Burrow, W. H. WISC profiles: above average and MR good and poor readers. Mental Retardation, 1973, 11, 12-14.

Rebert, C. S., Wexler, B. N., and Sproul, A. EEG asymmetry in educationally handicapped children. Electroencephalography and Clinical Neurophysiology, 1978, 45, 436-442.

Rhodes, L. E., Dustman, R. E., and Beck, E. C. The visual evoked response: a comparison of bright and dull children. Electroencephalography and Clinical Neurophysiology, 1969, 27, 364-372.

Ritter, W., Vaughan, H. C., and Costa, L. D. Orienting and habituation to auditory stimuli: a study of short term changes in average evoked responses. Electroencephalography and Clinical Neurophysiology, 1968, 25, 550-556.

Roberts, A. C. The aphasic child. Springfield: Charles Thomas, 1966.

Roger, M., and Galand, G. Operant conditioning of visual evoked potentials in man. Psychophysiology, 1981, 18, 477-482.

Rosenfeld, J. P., Rudell, A. P., and Fox, S. S. Operant control of neural events in humans. Science, 1969, 165, 821-823.

Roth, W. T., Rothbart, R., Darley, C. F., Tinklenberg, J. R., and Kopell, B. S. A model of CNV resolution in a memory retrieval task. Paper presented at the 14th annual meeting of the Society for Psychophysiological Research, 1974.

Ruchkin, D. S., and Sutton, S. CNV and P300 relationships for emitted and for evoked cerebral potentials. In J. S. Desmedt (Ed.), Progress in clinical neurophysiology (Vol. VI). Bassel: Karger, 1979.

Salvia, J., and Yssldyke, J. E. Assessment in special and remedial education. Boston: Houghton Mifflin, 1978.

Sanders, M. J. Psychological evaluation of the learning disabled child. In M. I. Gottlieb, P. W. Zinkus, and L. J. Bradford (Eds.), Current issues in developmental pediatrics: the learning disabled child. New York: Grune and Stratton, 1979.

Satterfield, J. H. EEG issues in children with minimal brain dysfunction. In S. Walzer and P. H. Wolff (Eds.), Minimal brain dysfunction in children. New York: Grune and Stratton, 1973.

Satterfield, J. H. EEG issues in children with minimal brain dysfunction. Seminars in Psychology, 1973, 5, 35-46.

Satterfield, J., Cantwell, D. P., Lesser, L. I., and Podosin, R. L. Physiological studies of the hyperkinetic child. American Journal of Psychiatry, 1972, 128, 102-108.

Satterfield, J. H., Lesser, L. I., Saul, R. E., and Cantwell, D. P. EEG aspects in the diagnosis and treatment of minimal brain dysfunction. Annals of the New York Academy of Science, 1973, 205, 274-282.

Schain, R. J. Neurology of childhood learning disorders. Baltimore: Williams and Wilkins, 1977.

Schechter, G., and Buchsbaum, M. The effects of attention, stimulus intensity and individual differences on the average evoked response. Psychophysiology, 1973, 10, 392-400.

Schwartz, G. E., Davidson, R. J., and Pugash, E. Voluntary control of patterns of EEG parietal asymmetry: cognitive concomitants. Psychophysiology, 1976, 13, 498-504.

Schwent, V. L., and Hillyard, S. A. Auditory evoked potentials and multichannel selective attention. Electroencephalography and Clinical Neurophysiology, 1975, 38, 131-138.

Schwent, V. L., Hillyard, S. A., and Galambos, R. Selective attention and the auditory vertex potential. I. Effects of stimulus delivery rate. Electroencephalography and Clinical Neurophysiology, 1976a, 40, 604-614.

Schwent, V. L., Hillyard, S. A., and Galombos, R. Selective attention and the auditory evoked potential. II. Effects of signal intensity and masking noise. Electroencephalography and Clinical Neurophysiology, 1976b, 40, 615-622.

Schwent, V. L., Snyder, E., and Hillyard, S. A. Auditory evoked potentials during multichannel selective listening: role of pitch and localization cues. Journal of Experimental Psychology: Human Perception and Performance, 1976, 2, 313-325.

Shabsin, H. S. Electroencephalographic differences in learning disabled and non learning disabled children. Paper presented at the 11th annual meeting of the Biofeedback Society of America, Colorado Sprrings, 1980.

Shagass, C., Ornitz, E. M., Sutton, S., and Teuting, P. Event-related potentials in psychopathology. In E. Calloway, P. Teuting, and S. H. Koslow (Eds.), Event-related brain potentials in man. New York: Academic Press, 1978.

Shields, D. T. Brain responses to stimuli in disorders of information processing. Journal of Learning Disabilities, 1973, 6, 501-505.

Shiffran, R. M., and Grantham, D. W. Can attention be allocated to sensory modalities? Perceptual Psychophysiology, 1974, 15, 460-474.

Shouse, M. N., and Lubar, J. F. Operant conditioning of EEG rhythms and ritalin in the treatment of hyperkenesis. Biofeedback and Self Regulation, 1979, 4, 299-312.

Simonds, J. F. Relationship between children's learning disorders and emotional disorders at a mental health clinic. Journal of Clinical Psychology, 1974, 30, 450-458.

Sklar, B., Hanley, J., and Simmons, W. W. An EEG experiment aimed toward identifying dyslexic children. Nature, 1972, 240, 414-416.

Smith, D. B. D., Donchin, E., Cohen, L., and Starr, A. Auditory average evoked potentials in man during binural listening. Electroencephalography and Clinical Neurophysiology, 1970, 28, 146-152.

Smith, M. D. Stability of WISC-R subtest profiles for learning disabled children. Psychology in the Schools, 1978, 15, 4-7.

Smith, M. D., Coleman, J. M., Dokecki, P. R., and Davis, E. E. Recategorized WISC-R scores of learning disabled children. Journal of Learning Disabilities, 1977, 10, 437, 443.

Smith, P. A., and Marx, R. W. Some cautions on the use of the Frostig Test: a factor analysis study. Journal of Learning Disabilities, 1972, 5, 357-362.

Sobotka, K. R., Black, W. F., Hill, S. D., and Porter, R. J. Some psychological correlates of developmental dyslexia. Journal of Learning Disabilities, 1977, 10, 363-369.

Sobotka, K. R., and May, J. G. Visual evoked potentials and reaction time in normal and dyslexic children. Psychophysiology, 1977, 14, 18-14.

Sohmer, H., and Student, M. Auditory nerve and brain-stem evoked responses in normal, autistic, minimal brain dysfunction and psychomotor retarded children. Electroencephalography and Clinical Neurophysiology, 1978, 44, 380-388.

Sorel, L., Lebrun, P., Leotard, G., Delaite, F., Rucquoy-Ponsar, M., Basecqz, G., and de Biolley, D. Records of a group of 200 normal subjects aged 14-20 years. Electroencephalography and Clinical Neurophysiology, 1965, 18, 633.

Sotelo, C., and Palay, S. L. Altered axons and axon terminals in the lateral vestibular nucleus of the rat: possible example of axon remodeling. Laboratory Investigation, 1971, 14, 211-453.

Sterman, M. B. Neurophysiologic and clinical studies of sensori EEG biofeedback training: some effects on epilepsy. In L. Birk (Ed.), Seminars in Psychiatry, 1973, 5, 507-526.

Sterman, M. G., and Friar, L. Suppression of seizures in an epileptic following sensorimotor EEG feedback training. Electroencephalography and Clinical Neurophysiology, 1972, 33, 89-95.

Stevens, G., and Birch, J. A proposal for clarification of the terminology used to describe brain injured children. Exceptional Children, 1957, 23, 341-349.

Stevens, J. R., and Milstein, V. Severe psychiatric disorders of childhood: electroencephalogram and clinical correlates. American Journal of Diseases in Children, 1970, 120, 182-192.

Stevens, J. R., Sachdev, K., and Milstein, V. Behavior disorders of childhood and the EEG. Archives of Neurology, 1968, 18, 160.

Stevenson, L. P. WISC-R analysis: implications for diagnosis and intervention. Journal of Learning Disabilities, 1980, 13, 346-349.

Stevenson, L. P. WISC-R analysis: implications for diagnosis and educational intervention of LD children. Paper presented at the annual international convention, The Council for Exceptional Children (57th, Dallas, Texas, April 22-27, 1979, session T-31).

Strauss, A. A., and Lehtinen, L. E. Psychopathology and education of the brain injured child (2 vols.). New York: Grune and Stratton, 1947.

Strauss, A. A., and Werner, H. Disorders of conceptual thinking in the brain injured child. Journal of Mental and Nervous Diseases, 1942, 96, 153-172.

Strauss, A. A., and Lehtinen, L. E. Psychopathological education of the brain injured child. New York: Grune and Stratton, 1942.

Strupp, H. H., and Hadley, S. M. A tripartite model of mental health and therapeutic outcomes with special reference to negative effects in psychotherapy. American Psychologist, 1977, 32, 187-196.

Symann-Louett, N., Gascon, G. G., Matsumiya, Y., and Lombrosso, C. T. Wave form differences in visual evoked responses between normal and reading disabled children. Neurology, 1977, 27, 156-159.

Tabachnik, B. G. Test scatter on the WISC-R. Journal of Learning Disabilities, 1979, 12, 60-62.

Torgesen, J. K. What shall we do with psychological processes? Journal of Learning Disabilities, 1979, 12, 16-23.

Treisman, A. M. Strategies and models of selective attention. Psychological Review, 1969, 76, 282-299.

Tueting, P., and Sutton, S. The relationship between pre-stimulus negative shifts and post-stimulus components of the average evoked potential. In S. Kornblum (Ed.), Attention and performance (Vol. IV). New York: Academic Press, 1973.

Tueting, P., Sutton, S., and Zubin, J. Quantitative evoked potential correlates of the probability of events. Psychophysiology, 1970, 7, 385-394.

Vance, H. B., and Singer, M. G. Recategorization of the WISC-R subtest scaled scores for learning disabled children. Journal of Learning Disabilities, 1979, 12, 487-491.

Van Voorhis, S. T., and Hillyard, S. A. Visual evoked potentials and selective attention to points in space. Perceptual Psychophysiology, 1977, 22, 54-62.

Velasco, M., and Velasco, F. Differential effect of task relevance on early and late components of cortical and subcortical somatic evoked potentials in man. Electroencephalography and Clinical Neurophysiology, 1975, 39, 353-364.

Velasco, M., Velasco, F., Machado, J., and Olvera, A. Effects of novelty, habituation, attention and distraction on the amplitude of the various components of the somatic evoked responses. International Journal of Neuroscience, 1973, 5, 101-111

Wagonseller, B. R. Learning disability and emotional disturbance: factors relating to differential diagnosis. Exceptional Children, 1973, 40, 205-206.

Waksman, S. Psychometric phrenology revisited: comments on neuropsychological testing. Journal of Consulting and Clinical Psychology, 1978, 46, 1489-1490.

Wallbrown, F. H., Vance, H., and Blaha, J. Developing remedial hypotheses from ability profiles. Journal of Learning Disabilities, 1979, 12, 557-561.

Weiss, G. MBD: critical diagnostic issues. In H. E. Rie and E. D. Rie (Eds.), Handbook of minimal brain dysfunction: a critical review. New York: John Wiley and Sons, 1980.

Wepman, J. The perceptual basis for learning. In H. A. Robinson (Ed.), Meeting individual differences in reading. Chicago: University of Chicago Press, 1964.

Wepman, J. M., Cruickshank, W. M., Deutsch, C. P., Morency, A., and Strother, C. R. Learning disabilities. In N. Hobbs (Ed.), Issues in the classification of children. San Francisco: Jossey-Bass, 1975.

Werner, H., and Strauss, A. A. Types of visuo-motor activity in their relation to low and high performance ages. Proceedings of the American Association of Mental Deficiency, 1939, 44, 163-168.

Werner, H., and Strauss, A. A. Causal factors in low performance. Journal of Mental Deficiency, 1940, 45, 213-218.

Werner, H., and Strauss, A. A. Problems and methods of functional analysis in mentally deficient children. Journal of Abnormal and Social Psychology, 1939, 34, 37-62 (a).

Werner, H., and Strauss, A. A. Types of visuo-motor activity in their relation to low and high performance ages. Proceedings of the American Association of Mental Deficiency, 1939, 44, 163-168 (b).

Wetter, J., Keogh, B. K., McGinty, A., and Donlon, G. Functional analysis of intelligence test performance of children with learning problems. Proceedings of the Annual Convention of the American Psychological Association, 1972, 7, 551-552.

Wikler, A. W., Dixon, J. F., and Parker, J. B. Brain function in problem children and controls: psychometric, neurological and electroencephalographic comparisons. American Journal of Psychiatry, 1970, 127, 634-645.

Wilkinson, R. T., and Ashby, S. M. Selective attention, contingent negative variation and the evoked potential. Biological Psychology, 1974, 1, 167-179.

Wilkinson, R. T. Relationship between CNV, its resolution and the evoked response. In W. C. McCallum and J. R. Knott (Eds.), The responsive brain. Bristol: John Wright, 1976.

Wilkinson, R. T., and Lee, M. V. Auditory evoked potentials and selective attention. Electroencephalography and Clinical Neurophysiology, 1972, 33, 411-418.

Winik, M., and Rosso, F. Malnutrition and the central nervous system. In J. Prescot, M. Read, and D. Coursin (Eds.), Brain function and malnutrition. New York: John Wiley and Sons, 1975.

Wissink, J. E., Kass, C. E., and Ferrell, W. E. A bayesian approach to the identification of children with learning disabilities. Journal of Learning Disabilities, 1975, 8, 58-66.

Witelson, S. Perception of auditory stimuli in children with learning problems. Unpublished master's project, McGill University, 1962.

Witkin, H. A., Dyke, R., Faterson, H., Goodenough, D., and Karp, S. Psychological differentiation. New York: John Wiley and Sons, 1962.

Wohl, T. H. Psychological and psychoeducational assessment techniques. In H. E. Rie and E. D. Rie (Eds.), Handbook of minimal brain dysfunction: a critical review. New York: John Wiley and Sons, 1980.

Woods, D. L., and Hillyard, S. A. Attention at the cocktail party: brainstem evoked responses reveal no peripheral gating. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related brain potential research. Washington, D.C.: U.S.E.P.A., 1978.

Wyler, A. R., Fetz, E. E., and Word, A. A. Effects of operantly conditioned epileptic unit activity on seizure frequencies and electrophysiology of neocortical experimental foci. Experimental Neurology, 1974, 44, 113-125.

Zambelli, A. J., Stamm, J. S., Maitinsky, S., and Loiselle, D. L. Auditory evoked potentials and selective attention in formerly hyperactive adolescent boys. American Journal of Psychiatry, 1977, 34, 742-747.

Zurif, E. B., and Carson, G. Dyslexia in relation to cerebral dominance and temporal analysis. Neuropsychologia, 1970, 8, 351-361.

VITA

Harry Stewart Shabsin was born in Chicago, Illinois on February 21, 1948. Through the good graces of his parents he has lived on both the east and west coasts of the United States as well as in Europe and the Asian Rim. After graduating from Redlands High School in Redlands, California, he revisited Southeast Asia courtesy of the United States Army. Upon discharge from the Army, Mr. Shabsin attended the University of Indiana, Bloomington, for its pastoral setting and excellent liberal arts program. After graduating from U.I. with a B.A. degree Mr. Shabsin continued his education at The University of Tennessee, Knoxville, working in the neuropsychology laboratory of Dr. Joel Lubar. Upon completing his doctoral degree Mr. Shabsin plans to pursue post-doctoral training in Baltimore at the Johns Hopkins School of Medicine.