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OUTCOME MEASURES OF NEUROFEEDBACK TRAINING FOR ADHD

BY

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled: OUTCOME MEASURES OF NEUROFEEDBACK TRAINING FOR ADHD, submitted by MELLISA M. LAYMAN in partial fulfilment of the requirements for the degree of MASTER OF EDUCATION in COUNSELLING PSYCHOLOGY.

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ABSTRACT

This study examined three outcome measures of neurofeedback training for ADHD/ADD children to determine if success could be seen on any of the measures. The outcome measures used were training data collected at training sessions, QEEG assessments before and after neurofeedback training, and pre and post-training parental behavioral ratings. While training data did not indicate success, QEEG data and behavioral ratings did. Success was evident from QEEG outcome measures in terms of significantly reduced theta magnitudes in two conditions, eyes closed and eyes open, for the sites FZ, CZ, C3, and C4. Reduced theta magnitude was also evident in the reading condition, although the decrease was only significant at CZ. Variability in theta magnitude between subjects, as measured by standard deviations, was also reduced after training, although not significantly so. Neither the beta1 or beta2 frequency bands showed significant increase in magnitude after neurofeedback training. Success was also evident on a behavior ratings scale in which parents rated their children before and after neurofeedback training. Statistically significant improvement was seen on a scale measuring attention. Thus two of the three outcome measures indicated success following neurofeedback training.

TABLE OF CONTENTS

CHAPTER FIVE

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by inattention, impulsivity, and hyperactivity (Sattler, 1992). Estimates of the prevalence of this disorder range from 1% to 20% of the childhood population depending upon the criteria used (Ross & Ross, 1982). Rutter (1984) reported that the prevalence of ADHD in the pediatric population was as high as 10%. In a study of over 14,000 students in a public school district in Nebraska, only 136 (.96%) of students were found to be diagnosed as ADHD (Reid, Maag, Vas, Wright, 1994). The DSM-IV suggested that 3-5% of the childhood population has ADHD and that the male to female ratio ranges from 4:1 to 9:1 (American Psychiatric Association, 1994). A recent prevalence study of ADHD in a Western Canadian public school system estimated that 6.4% of the children were identified as ADHD (Francis, 1993); and a male to female ratio of 4:1 was reported in this study.

By the time most ADHD children are six years of age and entering school, they display a degree of inattention, impulsivity and hyperactivity which clearly exceeds that of normally-functioning children (Barkley, 1989). ADHD children have particular difficulty in the school environment because this environment requires sustained attention and self-regulatory abilities which greatly taxes these children. This can lead to academic underachievement and low self-esteem (Sattler, 1992). Other difficulties often seen with ADHD children are mood shifts, a low tolerance for frustration, temper outbursts and social problems (Goldstein & Goldstein, 1989).

Clearly, these difficulties pose problems for ADHD children as well as for parents and educators of these children.

According to Lubar et al. (1995), ADHD is a pervasive, life-long disorder which can not be cured but can be managed. Similarly, Barkley (1989) has suggested that only a small percentage of ADHD children will "outgrow" their symptoms. Follow-up studies of ADHD children have demonstrated that a significant number of these children as adolescents continue to have difficulties in academic achievement, behavioral inhibition and attention (Fisher et al., 1990 cited in Schwean et al., 1993). A 15 year follow-up study of ADHD teenagers found that as adults they continued to have difficulties in home and community adjustment, and in academic achievement (Weiss, Hechtman, Milroy, & Perman, 1985).

A number of etiologies for ADHD have been proposed over the years, including brain damage (Barkley, 1989), food additives (Finegold, 1975), elevated blood lead levels (Goldstein & Goldstein, 1989), maternal alcohol consumption and cigarette smoking (Denson, Nanson & McWatters, 1975; Streissguth et al., 1984). More recently, this disorder has been described as a developmental disability (Barkley, 1989), a behavioral syndrome (Sattler, 1992), a neurobehavioral disorder (Schwean, Parkinson & Lee, 1993), and a neological disorder (Lubar, Swartwood, Swartwood & O'Donnell, 1995). Today most researchers agree that ADHD is an organic neurological dysfunction, the severity of which can be exacerbated by environmental factors (Barkley, 1989; Lubar, et al., 1995; Schwean et al., 1993). Lubar et al. (1995) have argued that the primary symptoms of ADHD of inattention,

impulsivity and hyperactivity, are actually secondary outcomes resulting from an underlying neurological disorder.

Treatment approaches for ADHD have utilized have included dietary restrictions in which additives, colorings and refined sugar are removed from the diet, psycho-pharmacological treatments, including stimulants such as methylphenidate (Ritalin) and tricyclic antidepressants, behavior and cognitive-behavioral therapies, and family systems therapy (Barkley, 1989). Another treatment modality for ADHD children has arisen, primarily as the result of the work of Joel Lubar who developed a technique of neurofeedback training, a specific type of biofeedback linked to the electrical activity of the brain (Lubar, 1991). The aim of this training is to teach ADHD/ADD children to alter their brain wave patterns to make them more like the patterns of non-ADHD/ADD children. Neurofeedback training is discussed in more detail in the next chapter. The University of Alberta is the first research site in Canada to provide neurofeedback training for ADD/ADHD. Over one hundred children have received neurofeedback training through the Cognitive Re-Regulation Program at the University of Alberta. The present study examined data collected from a sample of participants in this program. It is important that neurofeedback training be evaluated to determine if ADHD children can successfully be taught to alter their brain wave patterns to become more similar to the brain wave patterns of non-ADHD children the educational and social implications could be significant.

CHAPTER TWO

LITERATURE REVIEW

This chapter will begin with a review of the changes in definitional changes of ADHD from the early 1900s to the present. This is followed by brief discussions of the comorbidity of ADHD with other conditions as well as genetic/familial influences in ADHD to provide a broader context for understanding ADHD. The role of behavioral assessment in ADHD is then discussed, followed by discussion of the neurological bases of ADHD. Quantitative EEG (QEEG) research is then outlined, leading to a discussion of neurofeedback training for ADHD. Finally, conclusions from the literature are presented and the purpose of the study and research questions for this study are outlined.

The History of the Conceptualization of ADHD

The disorder now referred to as "Attention Deficit-Hyperactivity Disorder" (ADHD) has undergone many definitional changes within this century, and some of the diagnostic definitions of ADHD have been quite controversial (Lahey, 1994; Reid, Maag, & Vasa, 1993). These diagnostic definitions, from the concept of a deficiency in "volitional inhibition" to the most recent definition of this disorder in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) will be reviewed.

Early History

One of the first detailed accounts of hyperactivity appeared in the medical

literature in the early 1900s. George Still, an English pediatrician, published a series of lectures that he had given to the Royal College of Physicians in England (Still, 1902). Still described children that he had encountered in his practice who were extremely active, defiant, and lacked self-control. He argued that these children experienced deficiencies in the "volitional inhibition" of behavior and that these deficiencies were the result of biological factors, such as central nervous system damage, rather than environmental factors, such as child rearing.

In the 1920s, researchers examining cases of encephalitis in children after the flu epidemic of 1918 noted that hyperactivity and inattention, as well as anti-social behavior and emotional problems, were typical behavioral sequelae (Bond & Partridge, 1926; Hohman, 1922; Kennedy, 1924; Stryker, 1925).

Minimal Brain Damage/Dysfunction

The next influential researchers were Strauss and Lehtinen who observed that inattention, hyperactivity and impulsivity were characteristic of brain-injured mentally retarded children but not of non-brain injured mentally retarded children. Strauss and Lehtinen (1947) concluded that inattention, hyperactivity and impulsivity were the result of brain damage, and the term "minimal brain damage" was applied to children exhibiting these symptoms. The minimal brain damage symdrome also came to be referred to as the "Strauss Syndrome" (Lubar, 1991).

According to Barkley (1989), over time researchers began to focus less upon brain damage as a cause of hyperactivity. Subsequently, there was a change in terminology from "minimal brain damage" to "minimal brain dysfunction."

Specifically, this terminology shift occurred in 1962 at a conference held by the Oxford International Study Group on Child Neurology which focused upon definitions and diagnosis in child neurology. Because of the lack of specific neurological signs associated with minimal brain damage, it was determined that "dysfunction" was a better term than "damage" (Mann, 1990).

Hyperkinesis

By 1968, neurological damage was no longer included in the diagnostic terminology of ADHD (Barkley, 1989), and the second edition of the DSM referred to the disorder as "Hyperkinetic Reaction of Childhood" (American Psychiatric Association, 1968). However, brain damage was still seen as the major cause of the disorder until the mid 1970s when studies began to emphasis that inattention and deficits in impulse control appeared to be central in children with this disorder (Barkley, 1989).

Attention Deficit Disorder/Attention Deficit-Hyperactivity Disorder

In the third edition of the DSM, the American Psychiatric Association (1980) relabelled hyperkinesis as "Attention Deficit Disorder" with two subtypes: with hyperactivity (ADD/H) or without hyperactivity (ADD/WO). This was a reflection of a shift in thinking which relegated hyperactivity to a somewhat secondary role. However, hyperactivity re-emerged as a central feature of this disorder in 1987 when it was relabelled in DSM III-R as "Attention-Deficit/Hyperactivity Disorder" and the subtype of "without hyperactivity" was removed.

Current Definitions

Most recently, the fourth edition of the DSM (American Psychiatric Association, 1994) referred to "Attention-Deficit/Hyperactivity Disorder" with three subtypes: ADHD, predominately inattentive type; ADHD, predominately hyperactive-impulsive type; and ADHD, combined type. For a diagnosis of ADHD to be made, six or more symptoms of inattention and/or six or more symptoms of hyperactivity-impulsivity must have persisted for a least six months and at a degree inconsistent with a child's developmental level. Additionally, some symptoms must have been present before seven years of age, the symptoms must be present in two or more of a child's environments (i.e. school and home), and must be present to a degree which significantly impairs a child's academic functioning (see Appendix A for DSM-IV diagnostic criteria for ADHD).

The three subtypes of ADHD emerged from DSM-IV field trials (Lahey et al., 1994). The predictive validity of the proposed DSM-IV criteria of disruptive behavior disorders, including ADHD, conduct disorder, and oppositional defiant disorder, was tested in field trails with 440 clinic-referred youth (Frick et al., 1994). In the field trials, the DSM-IV criteria identified more girls and preschool children with ADHD than DSM-III-R criteria had identified (Lahey et al., 1994).

The DSM-IV criteria for ADHD have been positively received by many researchers and the two dimensional approach emphasizing both inattention and hyperactivity-impulsivity has been praised as an improvement over DSM-III-R criteria which implied a unitary dimension of hyperactivity (Erk, 1995; Lahey et al., 1994;

McBurnett, Lahey & Pfiffner, 1993; Sabatino & Vance, 1994). However, McBurnett, Lahey & Pfiffner (1993) have cautioned that because the accuracy and reliability of DSM diagnoses are associated with the degree of psychological training and education, many issues may be raised about the accuracy of DSM IV diagnoses in educational settings. Erk (1995) stressed that school counsellors should be very familiar with the DSM IV criteria for ADHD to facilitate communication with other professionals.

Comorbidity of ADHD and Other Psychiatric Conditions

Critics of the DSM taxonomy approach to ADHD argue that the disorder should not be conceptualized as a psychiatric condition which can be differentiated from other conditions through differential diagnosis because DSM criteria for ADHD, despite revisions over the years, represents an "ill-defined constellation of behaviors" (Reid et al., 1993, p.200). Mann (1990) has noted that while impulsivity is a central feature of ADHD, it is also found in many childhood psychiatric disorders. Similarly, Cadoret & Stewart (1991) and Sabatino & Vance (1994) have noted the comordidity of ADHD with other psychiatric conditions.

A meta-analysis of psychiatric and psychological literature of empirical studies examining the comorbidity of ADHD with other disorders indicated considerable comorbidity: ADHD and conduct disorder were found to occur together in 30-50% of cases; ADHD and oppositional defiant disorder occurred together in at least 35% of cases; and ADHD and anxiety disorders were found to occur together in approximately 25% of cases (Biederman, Newcorn & Sprich, 1991). Wide ranges

(10-92% of cases) were found for the comorbidity of ADHD and learning disabilities and also for ADHD and mood disorders, ranging from 15-75% of cases. The researchers argued that ADHD is a heterogeneous disorder with varied comorbidity which does not appear to be random. Thus they suggested that subgroups of ADHD children might be delineated based upon the comorbidity of the disorder with other disorders and the subgroups may have differing responses to pharmacological interventions (Biederman et al., 1991). In a twin study of the comorbidity of ADHD and reading disabilities, Gilger, Pennington, & DeFries (1992) concluded that while ADHD and reading disabilities are largely genetically independent, in some cases they may occur together because of a shared genetic etiology.

Genetic and Familial Influences in ADHD

A meta-analysis of some 150 studies examining the role of genetics in childhood psychiatric disorders discovered that both twin and adoption studies provide definitive evidence of a genetic component in ADHD (Lombroso, Pauls & Leckman (1994). It has been stressed that while some evidence indicates that ADHD is a familial disorder, it is important to understand that "familial" and "genetic" are not synonymous because many disorders are found in families because of environmental (i.e dysfunctional family) and not genetic factors (Biederman et al., 1995).

Biederman et al. (1995) studied 140 ADHD and 120 control children to determine their "environmental adversity" (parental psychopathology and parental conflict). They found that parental conflict, diminished family cohesion, and parental

psychiatric illness existed significantly more frequently in the families of ADHD children. The researchers concluded that both psychosocial and genetic risk factors are important in ADHD. Similarly, Cadoret & Stewart (1991) concluded that ADHD is a disorder with a variety of associated behaviors and suggested that each of these behaviors is influenced by different genetic and environmental factors.

Behavioral Assessment of ADHD

Childhood psychiatric disorders are typically assessed with two types of approaches. The first type of approach is the categorical medically-based approach in which psychopathologies are seen as distinct syndromes and a diagnosis is made based upon pre-determined symptoms (i.e. DSM criteria). The second type of approach is dimensional and psychometrically-based in which psychopathologies are seen as a quantitative deviation from the norm (i.e. behavioral assessment, intelligence tests) (Biederman et al., 1993).

Many studies have demonstrated the validity and reliability of the Child Behavior Checklist (CBCL) developed by Achenbach (1991) with clinical and non-clinical populations (Biederman et al., 1993). Convergent validity of the CBCL with the Teacher's Report Form (TRF), also developed by Achenbach, has been found by researchers (Emerson, Crowley, & Merrell, 1994; Lee, Elliott, & Barbour, 1994). The cross-cultural validity of the CBCL has also been supported by research (DeGroot, Koot, & Verhulst, 1994; Fombonne, 1992; Hellinckx, Grietiens, & Verhulst, 1994).

The CBCL is often used in combination with other instruments to diagnose

ADHD (Drotar, Stein, & Perrin, 1995). Biederman et al. (1993) found excellent convergence between the CBCL Attention Problems scales with the diagnosis of ADHD in both non-comorbid and comorbid (i.e. child also meets criteria for another disorder) cases of ADHD. The researchers suggested that the CBCL is a useful screening instrument for ADHD. Similarly, McConaughy, Mattison, & Peterson (1994) found the CBCL Attention Problems scale was a significant predictor of behavior problems in children. Caution has been advised, however, by several researchers when using the CBCL because of possible subjectivity by the rater (Drotar, Stein, & Perrin, 1995; Reid, Maag, & Vasa, 1993). Nonetheless, Drotar et al. stressed that the development and continue refinement of the CBCL is a "major advance" in the assessment of children's behavior.

Neurological Bases of ADHD

Three main neurological theories of ADHD exist in the literature. These are: (1) ADHD is the result of dysfunction within a specific part of the brain, particularly the right hemisphere or the frontal lobe; (2) ADHD is the result of a neurochemical imbalance, such as inadequate neurotransmitter levels; (3) ADHD is the result of abnormal brain wave activity, such as excessive slow wave activity. This section provides an overview of these three neurological theories of ADHD, with emphasis upon the third theory, abnormal brain wave activity, which is the basis of neurofeedback training.

Right Hemisphere and Frontal Lobe Dysfunction

Right hemisphere dysfunction has been implicated in Attention Deficit Disorder (ADD) and ADHD while, conversely, left hemisphere damage has been implicated in learning disabilities by some researchers (Cunningham & Murphy, 1981). Voeller (1986) examined fifteen children with right hemisphere damage and found that all but one met the DSM criteria for ADD. In a second study with ADD children, Voeller & Heilman (cited in Branch, Cohen, & Hynd, 1995) found that ADD children exhibited subtle left-sided neurological signs indicative of right hemisphere dysfunction. Branch, Cohen, & Hynd (1995) examined children with right and left hemisphere dysfunction and found that right hemisphere dysfunction was more frequently associated with difficulties in attention and overactivity. Branch et al. found limited support for Voeller & Heilman's conclusion that right hemisphere dysfunction is directly implicated in ADD.

It has been argued that a number of different brain systems, and not simply right hemisphere dysfunction, may be involved in ADHD and that different systems may be involved in children with and without hyperactivity (Matazow & Hynd, 1992 cited in Riccio, Hynd, Cohen, & Gonzalez, 1993). Frontal lobe dysfunction has also been implicated in ADD (Chelune, Ferguson, Koon, & Dickey, 1986; Hynd, Semrud-Clikeman, Lorys, Norey, & Elipulos, 1990; Mattes, 1980). Kozoil & Stout (1993) found that ADHD children performed significantly poorer on a verbal fluency measure, a frontal lobe task, compared to controls and hypothesized that this supported the theory of frontal lobe involvement in ADHD.

Neurochemical Basis of ADHD

Some researchers have argued that specific neurotransmitters are involved in ADHD. Catecholamines, specifically dopamine and norepinephrine, which affect attention and inhibition, have been implicated in ADHD (Riccio et al., 1993; Voeller, 1991). For example, in a study with ADD adolescents and controls it was concluded that catecholamine differences existed between the ADD and control subjects with ADD subjects having lower catecholamine levels (Garfield, Brown, Klee, Braden, Beauchesne, & Shapiro, 1986).

In a review of the literature on the role of the neurotransmitter serotonin in behavior disorders of childhood, including ADHD, Zubieta & Alessi (1993) concluded that substantial evidence exists implicating serotonin in the modulation of activity, aggressio and learning. Zubieta & Alessi suggested that further subclassifications of behavior disorders could be made on the basis of biochemical and neuroendocrine measures. In a study in which ADD children received a placebo or one of two types of amino acids (tyrosine or tryptophan) thought to be the nutrient precursors of serotonin, dopamine, and norepinephrine, no differences between groups were apparent on teacher rating scales of the children's behavior, although significant differences were apparent on parent rating scales of behavior between the placebo and tryptophan groups (Nemzer, Arnold, Votolato, & McDonnell, 1986). It has also been suggested that monoamine oxidase (MAO), which metabolizes neurotransmitters such as serotonin, dopamine, norepinephrine, may be implicated in ADHD because MAO inhibitors, which are a type of antidepressant, have been shown

to help ADHD children (Weiss, 1991).

Other researchers have argued that the "dopamine hypothesis" of ADHD has not been supported by research but that there is some support for a relationship between ADHD and norepinephrine abnormalities (Sood, Wood, Ellis, Burns, & Singh, 1993). However, Sood et al. suggested that while some studies have found support for the hypothesized relationship between ADHD and norepinephrine functioning, other studies have failed to find support for this relationship. The researchers thus concluded that no single neuro-transmitter system can be implicated in ADHD but rather several systems seem to be involved.

It has been noted that a very large body of literature exists on the effects of stimulant medication on children with ADHD and ADD (Swanson et al., 1993). Hutchens (1987) has suggested that the use of stimulants such as methylphenidate (Ritalin) for behavioral control of children is one of the most controversial issues in the pharmacological field. In a major review of some 400 studies in this area, Swanson et al. (1993) concluded that, overall, the research indicated that temporary management of overactivity, inattention and impulsivity as well as temporary improvement in aggression, compliance and academic productivity was achieved when ADHD/ADD children were administered stimulant medication. However, improvement in academic achievement, reading skills and social skills was not found, and the researchers concluded that less improvement was found on learning and achievement measures in comparison to improvement in behavior and attention measures. Rigger and Bowers (1993) found that ADHD children who received

stimulant medication still had significant differences with normal controls on cognitive measures and visual-motor measures and suggested that ADHD children may have persisting neuropsychological difficulty even after taking stimulant medication.

Halperin, Gittelman, Katz, & Struve (1986) studied eighty hyperactive children who were given either methylphenidate or a placebo and found that drug responsiveness could not be predicted by either EEG measures or neurological soft signs. However, the authors stressed that their findings should not be interpreted as indicating that hyperactive children do not have a neurological dysfunction, but rather that treatment outcome can not be predicted from neurological measures. According to Halperin et al., the behavioral disturbances of hyperactive children and neurological abnormalities are part of a constellation of symptoms of an underlying neuro-physiological disturbance. Conversely, Bawden (1993) has stressed that EEG measures, specifically neurometric measures which were developed by John & Prichep (discussed further later in this chapter), can very successfully be used to predict the response of ADHD children to methylphenidate.

Abnormal Brain Wave Activity

Excessive Slowing and Underarousal

Over the past 50 years, many researchers have found EEG abnormalities in hyperactive children. As early as 1938, Jasper & Solomon reported that the electroencephalograms (EEGs) of behavior problem children were abnormal in comparison to a control group of normal children. The predominant EEG abnormality was excessive slow waves. Similarly, Werry, Weiss and Douglas (1964)

found evidence of excessive slow waves in hyperactive children. Specific regions of the brain have also been found to produce excessive slow waves in the posterior (Capute, Niedermeger & Richardson, 1968) and occipital areas (Hughes & Myklebust, 1971) in children with minimal brain dysfunction. Behavior disordered children were found to have temporal slowing which was significantly associated to a family history of nervous and mental disorders (Stevens, Sachdev & Milstein, 1968).

Wikler, Pixon & Parker (1970) delineated two subgroups of children with behavior problems, those with and those without hyperactivity, and found that both groups had excessive slow wave activity. Satterfield et al. found that hyperkinetic children who had more slow wave activity responded better to methylphenidate than did hyperkinetic children who did not have excessive slow wave activity (Satterfield, Cantwell, Lesser & Podosin, 1972). Shouse & Lubar (1978) investigated the paradoxical calming effect of stimulant medication on hyperkinetic children and found that those children who responded most favorably to stimulants were physiologically underaroused, or hypoactive, on EEG and galvanic skin response (GRS) measures. This finding, according to Shouse & Lubar was consistent with the arousal hypothesis postulated by such researchers as Satterfield et al. (1972).

Alpha Attenuation

In 1933, Hans Berger, who developed the EEG and discovered the alpha rhythm of 8-13 Hertz (Hz), suggested that alpha rhythms are blocked by thalamic inhibition during attention. Berger discovered that alpha rhythms were disrupted by visual stimulus, although it was later found that a visual stimulus is not necessary for

alpha activity to be disrupted (Mulholland, 1972). This blocking of alpha activity is referred to as the alpha attenuation response. Some researchers have argued that hyperactive and learning disabled children may have a deficit in attention because of a lowered alpha attenuation response (Fuller, 1978; Grunewald-Zuberbier, Grunewald, & Rasche, 1975; Milstein, Stevens, & Sachdev, 1969).

Theta/Beta Activity

In the 1980s, researchers began to specifically point to excessive theta wave power in the brain as being linked to ADHD. Typically the "slow wave" theta rhythm is defined as 4-8 Hz and is associated with day-dreaming and vivid imagery (Lubar, 1989). The theta rhythm was named by Walter & Dovey in 1944 who proposed it originated in the thalamus (Mann, 1990). Andreassi (1989) noted that higher theta power has been associated with decreased arousal. Theta activity occurs more frequently in children than adults and decreases with age. By adulthood, theta rhythms are seen during drowsiness and stage 1 sleep (REM and dreaming) with only trace levels of theta present during normal wakefulness (Mann, 1990).

Significant differences in theta production have been found between learning disabled and control (non-learning disabled) children, with learning disabled children exhibiting more power in the theta bands (Ahn, Prichep, John, Baird, Trepetin, & Kaye, 1980; Lubar, Bianchini, Calhoun, Lambert, Brody, & Shabsin, 1985). Poor readers (both phonetic and dysphonetic) have been found to have lower beta activity than ADD controls who were normal readers (Ackerman, Dykman, Oglesby, & Newton, 1995). Mann (1990) found increased theta in frontal regions and significantly

reduced beta activity in temporal regions in ADHD children in comparison to controls. These differences were most pronounced during reading and drawing tasks.

Similarly, significant differences in theta and beta activity between ADD children and controls has been found, with ADD children having significantly more frontal theta activity and less temporal beta activity during a drawing task (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992). Beta, or "fast wave" activity ranges from 12-24 Hz and is associated with focused attention (Fein et al., 1984, John et al., 1988, Gasser et al., 1988b, Matsuura et al., 1985). Janzen (1992) found that across all tasks (baseline--eyes open, reading silently, drawing, doing arithmetic problems, and listening to a story) ADD children had more relative power than controls in the theta band and less relative power in the beta band. In a subsequent study, significant differences were found between ADD children and controls for the baseline, reading and drawing conditions, with ADD children demonstrating significantly more theta activity. Interestingly, significant differences did not exist between the two groups for beta activity (Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995).

Quantitative EEG Research

In 1977 John et al. proposed a new diagnostic method, neurometrics, based upon a large body of normative quantitative EEG (QEEG) data and demonstrated that a high proportion of children with learning disabilities or neurological dysfunction could be distinguished from normal children (John, Prichep, Fridman, & Easton, 1988). The researchers subsequently obtained normative data from subjects aged 17-

90 years and for depressive, alcoholic, schizophrenic and dementia patients and found that different disorders were characterized by distinctive EEG profiles. John et al. suggested that neurometrics provided a new diagnostic method which could provide objective evidence of specific abnormalities in brain electrical activity which could corroborate clinical diagnoses.

QEEG and developmental changes associated with aging of normal children and adolescents have been determined by a number of researchers. Matousek and Petersen (1973) provided normative EEG data based upon their study involving 561 subjects from 1-21 years. The researchers concluded that fast wave activity increased with age and that EEG parameters varied with age. In a major study involving 1416 subjects aged 6-39 years, brain wave activity at the C3, 01 and FP1 sites was examined and it was determined that the amplitude of delta, theta, alpha, and beta waves decreased from age 6 to approximately ages 18-21 when they reached a stable level (Matsuura et al., 1985). The researchers found that the least decline in amplitude occurred in the beta band, and they concluded that interindividual variability of the EEG is larger among children than adults.

Gasser, Verleger, Bacher, & Sroka (1988) examined the changes in EEG band power across age in a sample of 158 normal children and adolescents aged 6-17 years. They determined that all bands decreased in absolute power with age except for alpha 2 (defined as 9.5-12.5 Hz) and that, generally, with aging fast bands increased in relative power while slow bands decreased in relative power. The researchers also suggested that they found strong evidence for a "substituting process"

between theta activity and alpha 2 activity in which theta activity declines and alpha 2 activity increases with age.

In a second article, Gasser examined the development of topographic distribution of band power in the same subjects and concluded that band power between the left and right hemisphere does not change with age (Gasser, Jennen-Steinmetz, Sroka, Verleger, & Mocks, 1988). Gasser et al. also determined that slow waves (theta and alpha) mature first in the occipial regions, followed by the Pz site, and Cz, and, finally, in the frontal locations. Thus maturation of slow waves proceeds from posterior to anterior locations. Fast waves were found to develop first at the Cz site, followed by the Pz site, occipital, central and frontal locations.

The reliability or stability over time of QEEG measures of children has also been examined. Absolute power EEG measures of boys 9-13 years were found to be highly stable over 1-3 years (Fein, Galin, Yingling, Johnstone, & Nelson, 1984). However, relative power measures were found to be more variable and less reliable. Graap (1994) also found absolute power measures to be reliable over a eight day period.

Neurofeedback Training for ADD and ADHD

The use of EEG neurofeedback in the treatment of ADD and ADHD grew out of research on epilepsy. Sterman, LoPresti, & Fairchild (1969, cited in Lubar & Shouse, 1976) demonstrated that by training the sensorimotor (SMR) rhythm (12-14 Hz) of cats, their resistance to convulsant drugs was increased. This research was next extended to humans and epileptic subjects received SMR biofeedback training.

Studies demonstrated a reduction in seizure activity in subjects (Finley, Smith, & Etherton, 1975; Sterman & Friar, 1972; Sterman, Macdonald, & Stone, 1975).

Lubar & Bahler (1976) replicated Sterman's work and, again, demonstrated that SMR training was very effective in the reduction of seizures among epileptic subjects. Subjects in the study who were high school or college students also reported increased attention and ability to concentrate (Lubar, 1991). Lubar & Shouse (1976) next published a case study on an eight year old hyperkinetic child in which EEG and behavioral changes were evident after SMR training in which SMR was reinforced but only when theta activity was inhibited. Subsequent replication studies by Lubar & Shouse were published in which the same training protocol was used to successfully reduce motor activity in hyperkinetic children (Shouse & Lubar, 1978; Shouse & Lubar, 1979).

From these studies, Lubar concluded that SMR training with theta inhibition was an effective treatment for hyperactivity but had less of an effect on inattention (Lubar, 1991). Lubar began treating children in a clinical setting and found that children who had attentional problems but were not hyperactive seemed to produce excessive theta activity and also had low beta production (Lubar, 1991). He began training children with ADD with SMR training followed by beta training with theta inhibition and found significant improvement on academic achievement measures (Lubar & Lubar, 1984).

Replications of Lubar's studies were successfully conducted by other researchers with learning disabled and hyperactive children (Carter & Russell, 1985;

Tansey & Bruner, 1983; Tansey, 1985). Recently, Lubar, Swartwood, Swartwood, & O'Donnell (1995) demonstrated that ADHD children who learned to successfully decrease their theta activity through neurofeedback training showed significant improvement pre and post-training on the Test of Variables of Attention (T.O.V.A.), a parental behavioral rating scale, and the Wechsler Intelligence Scale for Children, revised (WISC-R). Tansey (1990, 1991) has also used the WISC-R as a measure of success in neurofeedback training with learning disabled children.

Conclusions from the Literature

Considerable research exists on ADHD/ADD and related issues. Much research has implicated a neurological etiology for ADHD, including right hemisphere or frontal lobe dysfunction, functioning of neurotransmitter systems, and abnormal brain wave activity. Research on abnormal brain wave activity has included the examination of epilepsy, hyperactivity, learning disabilities, ADD, and ADHD. Neurofeedback techniques have been developed out of the theoretical base established by abnormal brain wave activity research. Neurofeedback training protocols have been established for epileptic, ADD, ADHD and, most recently, alcoholic clients (see Penniston and Kulkoski, 1989).

Research studies which have specifically focused upon neurofeedback training for ADD and ADHD are still limited in number. Few studies have compared EEG measures from neurofeedback training data (namely EEG data collected during training sessions) with other measures such as parental behavioral rating scales. To date, no studies have compared neurofeedback training data and EEG data obtained

from QEEG assessments.

Purpose of the Study

This study examined data from three outcome measures of neurofeedback training: training data, QEEG assessments, and parental behavior ratings for their children. The neurofeedback training took place at the Cognitive Re-regulation Program at the University of Alberta. The study was exploratory in nature and sought to answer the general question: Can success in neurofeedback training be seen on any of three outcome measures? The specific research questions addressed in the study were:

1. Is there improvement across neurofeedback training sessions?

Improvement is defined as a decrease in theta microvolts, an increase in beta1 or beta2 microvolts, and/or a decrease in the theta-beta1 ratio or the theta-beta2 ratio across sessions.

2. Is there improvement on QEEG assessments after neurofeedback training?

Improvement is defined as a decrease in theta microvolts, and/or an increase in beta1 or beta2 microvolts.

3. Is there improvement in parental behavior ratings for their children before and after neurofeedback training?.

Improvement is defined as a decrease on the attention scale and an increase on the school scale of the Child Behavior Checklist.

CHAPTER THREE

METHODS

Sample

A sample of 19 subjects was chosen from participants in the Cognitive Re-regulation Program at the University of Alberta. Research has indicated that the EEG measures of pre-adolescent boys aged 9-13 years are stable when tested three years later (Fein et al., 1984). Thus the sample was originally limited to pre-adolescent males between the ages of 9-13 years. The sample limitations are important because they help to control for the potential effects of age, maturation, and gender. One subject who fell below the age range of 9-13 years was also included in the sample to increase the sample size to 19 subjects.

All participants in the Cognitive Re-regulation Program met the following criteria for inclusion in the training program: 1) behavior consistent with DSM-III-R/ DSM-IV criteria for ADHD or ADD; 2) pre-training EEG assessments with a pattern consistent with ADHD/ADD: namely, increased theta activity in frontal and central cortical locations and/or decreased posterior beta activity. None of the children in the Cognitive Re-regulation Program were on methylphenidate medication while in the training program. A standard psychometric battery was administered to all children before they begin the training program, consisting of the Wechesler Intelligence Scale for Children-Revised Third Edition (WISC-R) and the Wide Range Achievement Test-Revised (WRAT-R).

Informed consent was obtained from all parents before their children began the

training program (see Appendix B). The consent form informed parents that data collected both pre and post-training may be used for research purposes with the proviso that all data will remain confidential and no individual participants will be identified in any research reports. The proposed study complied with the ethical guidelines as outlined by the Department of Educational Psychology, University of Alberta.

Procedure

Neurofeedback Training

The majority of subjects were trained with Lexicor equipment and software. To increase the sample size, two subjects were included who received training with equipment and software by Autogenics. Two basic training protocol were used for subjects: electrodes were placed at the CZ-C3/CZ-C4 sites (alternated each session) for ADHD children, and at the FZ and PZ sites for ADD children (refer Appendix C for diagram of electrode placement). All subjects in the sample received bi-polar neurofeedback training. EEG's were sampled at a rate of 128 samples per second and gain was set at 32,000. All impedances measured below 10K ohms. Linked ear electrodes were used as the common reference for all recordings.

The goal in training at the C3-C4 sites was for the child to raise beta1 (12-16 Hz) and decrease theta (4-8 Hz) activity. The goal in training at the FZ-PZ sites was for the child to raise beta2 (16-20 Hz) and decrease theta activity. Five of the subjects in the study received CZ-C3/CZ-C4 training and fourteen of the subjects received FZ-PZ training. All subjects in the sample received bipolar training in

which electrodes are placed on both sites (CZ-C3/CZ-C4 or FZ-PZ) within each training session. During each training session, subjects did four trials: a practice trial, followed by a trial in which they read, followed by another practice trial, and lastly a trial in which they did arithmetic problems.

QEEG Assessment

Pre-training and post-training QEEG assessments were conducted at the University of Alberta. Post-training QEEG assessments typically occurred within a week after the completion of neurofeedback training. The Electro-Cap system (Electro-Cap International) was used in QEEG assessments. This system records from 19 active electrodes in a nylon cap which is worn by the subject. The electrode placement in the Electro-Cap is according to the International 10-20 system (Jasper, 1958; see Appendix C). Electrodes were attached using Electro-gel (Electro-Cap International). Linked ear electrodes were used as the common reference for all recordings. The Lexicor NeuroSearch-24 system (Lexicor Medical Technology Inc.) recorded EEG data. EEG's were sampled at a rate of 128 samples per second and gain was set at 32,000. Impedance was kept below 5K ohms and was checked at the beginning, middle and end of each recording session.

Before QEEG assessment began, the process was explained to the children. Children sat in a high-backed chair and were asked to produce artifacts by tensing their jaw, yawning and moving their eyes so that they could see on the computer screen how these movements affected their EEG signal. They were then asked to avoid these movements to limit the amount of artifacts produced. Also, electrodes

were placed on subjects' frontalis and masseter muscles in order that any muscle or eye movement could later be artifacted. Recording began when subjects were comfortable and relatively still. EEG recordings were taken during six conditions with each child:

Eyes Open: Subjects were asked to focus upon an X with a red dot in the centre on a piece of paper approximately three feet in front of them on a music stand.

Eyes Closed: Subjects were asked to close their eyes and still focus on the dot in front of them.

Reading Silently: Subjects silently read grade-appropriate material from the Canadian Achievement Test. Materials were placed on the stand in front of subjects.

Drawing: Subjects were asked to copy figures from the Bender-Gestalt Visual Motor Test (Bender, 1938). To provide a solid surface for copying the figures, the top half of a small wooden desk was placed on the subjects' laps.

Listening to a Story: Subjects were again asked to focus upon the dot three feet in front of them while a story was read to them.

Solving Arithmetic Problems: Subjects were asked to solve grade-appropriate arithmetic problems from the Canadian Achievement Test. Again, the desk top was placed on their laps to provide a solid surface to work upon.

All conditions proceeded in the same order and with the same instructions for each child, and all QEEG assessments took place between 0900-1200 hours or 1300-1600 hours. Five minutes of QEEG data were recorded for each condition. QEEG data was visually artifacted soon after the completion of recording sessions to remove

epochs with non-EEG activity such as muscle (EMG) or eye movement. Artifacted data were then saved to magnetic tape. Data was transformed using the Fast Fourier Transform. Frequency bands were defined in this study as:

Delta: 0-4 Hz

Theta: 4-8 Hz

Alpha: 8-12 Hz

Beta1: 12-16 Hz

Beta2: 16-20 Hz

In this study, beta1 was defined as 12-16 Hz and beta2 was defined as 16-20 Hz because most researchers recognize the lower limit of beta (beta1) as 12-16 Hz, and beta2 as 16-20 or 16-25 Hz (Fein et al., 1984, John et al., 1988, Gasser et al., 1988b, Matsuura et al., 1985). It should be noted that in earlier studies researchers such as Sterman & Friar (1972) and Lubar & Bahler (1976) referred to 12-14 Hz as the sensorimotor rhythm (SMR) band rather than beta1.

Statistical Analysis

Training data, QEEG data, and behavioral ratings data were analyzed with the SPSS program using means, standard deviations, paired T-tests, and Pearson Product-Moment Correlations.

Outcome Measures

Training Data

Data was collected during each training session for participants in the Cognitive Re-regulation Program. Participants received differing numbers of training

sessions based upon their progress (range of sessions= 36-73; mean number of sessions= 50). Thirty sessions were systematically sampled across time for each subject in the study, excluding the first three and last three sessions, to provide an even number of measures (30) for all subjects. Measures representing the beginning, middle and end of training sessions were analyzed for each subject. The third trial (practice) of each session was chosen for analysis because practice trials represent more of a baseline than either the reading or arithmetic trials. The first practice trial was not chosen because it was also the first trial in each training session and subjects sometimes used the first trial to "warm up". Theta, beta1 and beta2 were chosen for analysis because these frequency bands are targeted in neurofeedback training. Theta-beta1 and theta-beta2 ratios were also analyzed because Lubar (1991) has suggested that ratios may be a better measure of improvement across training sessions than beta or theta alone. Data are reported in magnitude measured in microvolts (uV) measured peak to peak.

QEEG Data

QEEG data were examined for the eyes closed and reading conditions. Additionally, *post hoc* analysis was conducted for the eyes open condition and for the standard deviations of the eyes closed, reading and eyes open conditions. These conditions were selected because in recent research they have been found to be highly reliable (Graap, Janzen, Norman, & Fitzsimmons, 1994). Beta1, beta2 and theta frequency bands were again analyzed. Data are reported in magnitude measured in

microvolts (uV) peak to peak. Five sites were examined: FZ, PZ, CZ, C3, and C4.

Behavior Rating Scales

Behavioral rating scales are often used in the assessment of children (McKinney, Montague, & Hocutt, 1993). One of the most commonly used instruments is the Child Behavior Checklist (CBCL) developed by Achenbach (1991). The CBCL covers ages 4 to 18 years and has 120 items. Parents rate their child on each item using a three point rating scale (0 = not true, 1 = somewhat or sometimes true, 2 = very true or often true) and raw scores are converted into T-scores. The CBCL has two sections: nine scales of behavior problems, including Attention Problems (formerly Hyperactive); and a social competency inventory comprised of three scales (Activities, Social and School).

CHAPTER FOUR

RESULTS

This chapter is organized according to this study's research questions outlined in Chapter Two. To summarize, the research questions are:

1. Is there improvement across neurofeedback training sessions?

Improvement is defined as a decrease in theta microvolts, an increase in beta1 or beta2 microvolts, and/or a decrease in the theta-beta1 ratio or the theta-beta2 ratio across sessions.

2. Is there improvement on QEEG assessments after neurofeedback training?

Improvement is defined as a decrease in theta microvolts, and/or an increase in beta1 or beta2 microvolts. *Post hoc* analysis of QEEG data is also presented at the end of the section.

3. Is there improvement in parental behavior ratings for their children before and after neurofeedback training?

Improvement is defined as a decrease on the attention scale and an increase on the school scale of the Child Behavior Checklist.

For the purposes of analysis of the beta frequency bands, two groups were created from the 19 subjects: ADHD Group (n=5) received CZ-C3/CZ-C4 training and ADD Group (n=14) received FZ-PZ training. Beta1 was analyzed for the ADHD Group and beta2 was analyzed for the ADD Group because these were the respective beta frequency bands each group was taught to increase in neurofeedback

training sessions. Admittedly these groups are small in size, particularly the ADHD Group. However, it was important to examine the beta frequency bands relative to subjects' training protocols. Theta was analyzed both for the ADHD Group and the ADD Group separately and for the groups combined. Because results for both types of analysis were similar, they are reported in this chapter for the groups combined only. As was discussed in the previous chapter, the goal in both CZ-C3/CZ-C4 and FZ-PZ training protocols is decrease theta activity and raise beta1 (CZ-C3/CZ-C4 training) or beta2 (FZ-PZ training). Theta-beta ratios are presented according to group (theta-beta1 for ADHD Group and theta-beta2 for ADD Group).

Psychometric Analysis

Data, with the exception of age, are presented in standard scores which are raw scores that have been transformed to have a given mean and standard deviation. The mean age of subjects in this study was 10.5 years, with a range of 7.10 to 13.11 years (see Table 1). The mean full scale cognitive functioning level as measured by the Wechsler Intelligence Scale for Children, Revised (WISC-R) was 107.2 (S.D.= 10.14) and fell within the average range. The mean verbal (103.9; S.D.= 10.49) and performance (109.3; S.D.= 11.74) IQs also fell within the average range. Academic achievement as measured by the Wide Range Achievement Test, Revised (WRAT-R) fell within the average range, although the mean standard scores for Spelling (87.0) and Arithmetic (87.9) were approaching one standard deviation below the population mean. The mean standard score for reading was higher (92.8). Generally, the sample's mean academic achievement levels were below the mean full scale cognitive

TABLE 1

Psychometric Data[a] on Subjects (Both Groups)[b]

Measure	M	S.D.
Age	10.5	1.50
WISC-R Full Scale	107.2	10.14
WISC-R Verbal	103.9	10.49
WISC-R Performance	109.3	11.74
WRAT-R Reading	92.8	13.34
WRAT-R Spelling	87.0	10.20
WRAT-R Arithmetic	87.0	11.38

[a] Data presented in standard scores, except age presented in years/months.

[b] $n = 19$.

functioning level.

Research Question 1: Training Sessions Analysis

The mean theta uV was analyzed by training session measures representing the beginning, middle and end of training (see Table 2 and Figure 1). Each session measure was compared to the other (i.e. beginning to middle, middle to end, and beginning to end) with paired t-tests. Theta magnitude did not decrease significantly between these session measures. Similarly, no significant differences were found in the mean magnitude of beta1 for the ADHD Group or beta2 for the ADD Group between the beginning, middle, and end of training sessions (see Table 3 and Figure 2). Further, neither the theta-beta1 ratio for the ADHD Group or the theta-beta2 ratio for the ADD Group decreased significantly between the beginning, middle, and end of training sessions.

Table 4 presents correlation coefficients of frequency bands for measures 1, 15 and 30. Correlations for the ADHD Group for beta1 ranged from .43 to .70, and theta-beta1 ratios ranged from -.01 to .66. Very strong positive correlations, ranging from .71 to .90, were found for beta2 and, similarly, strong positive correlations, ranging from .75 to .82, were found for the theta-beta2 ratio of the ADD Group. This is indicative of data which is strongly related and does not differ much from the beginning, middle, and the end of training sessions. Weaker positive correlations, ranging from .23 to .56, were found for theta, which may be indicative of data which differs more from measure to measure.

TABLE 2

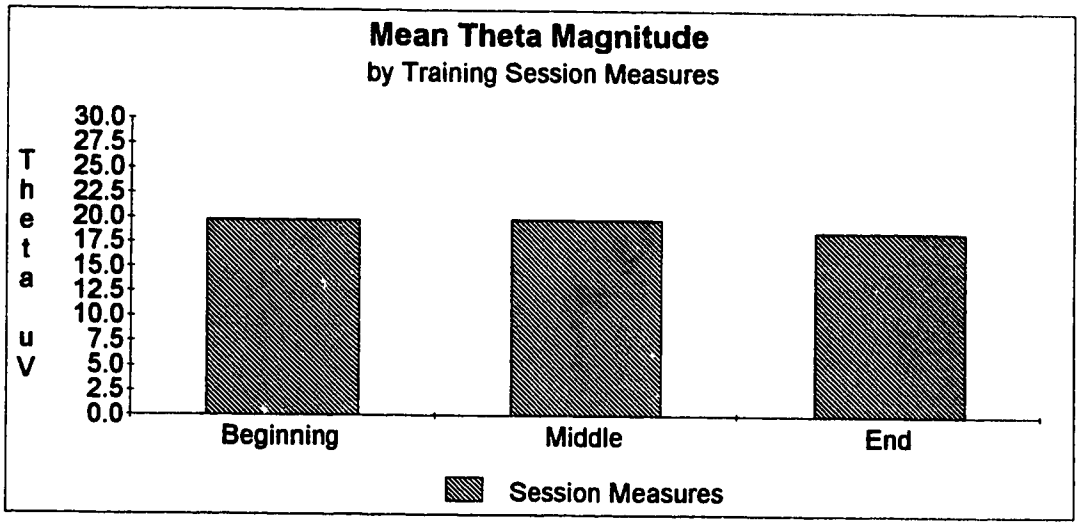
Mean Magnitude of Theta for Both Groups

by Training Session Measures

	Training Session Measure					
	Beginning		Middle		End	
Group	M	S.D.	M	S.D.	M	S.D.
Both[a]	19.7	5.33	19.8	7.17	18.6	6.15

No significant differences found between session measures.
[a] n= 19.

Figure 1



No significant differences found between session measures.
n = 19

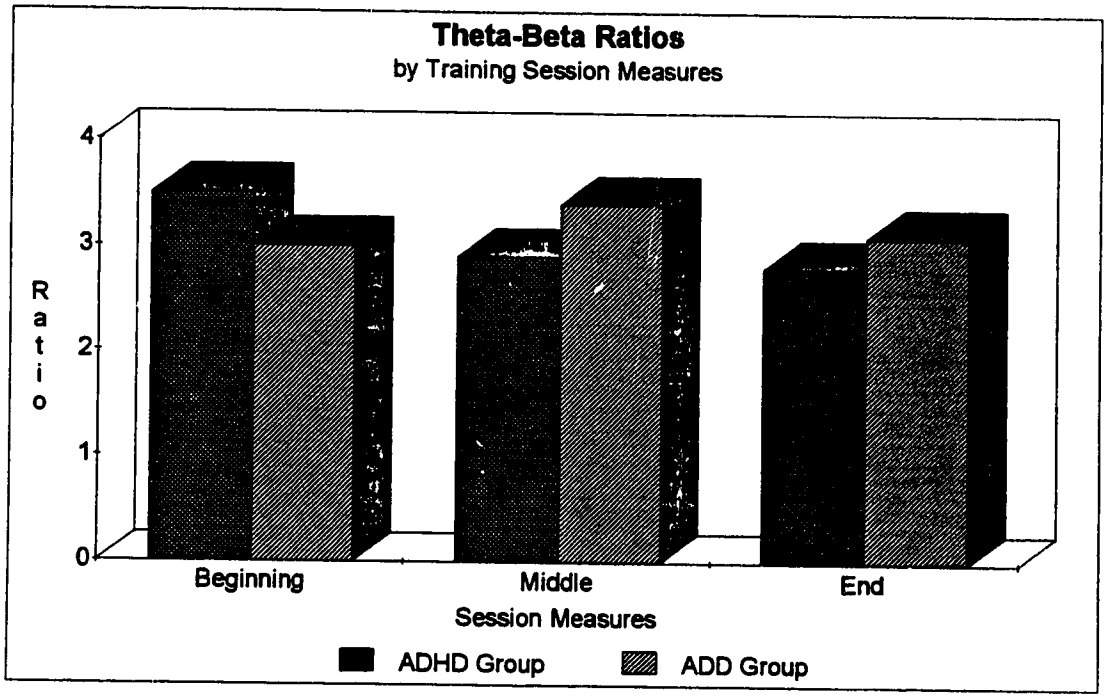
TABLE 3

Mean Magnitude of Beta1, Beta2, and Theta-Beta Ratios by Group by Training Session Measures

	Training Session Measure					
	Beginning		Middle		End	
Frequency Band/Group	M	S.D.	M	S.D.	M	S.D.
ADHD Group [a]						
Beta1	5.8	1.54	6.1	1.92	5.8	1.42
Theta-Beta1 Ratio	3.5	1.29	2.9	1.24	2.8	.64
ADD Group [b]						
Beta2	6.8	1.99	6.5	2.00	6.7	2.67
Theta-Beta2 Ratio	3.0	0.82	3.4	1.27	3.1	0.77

No significant differences found between session measures.
[a] n= 5.
[b] n= 14.

Figure 2



No significant differences found between session measures.
n = 19

TABLE 4

Correlations of Theta, Beta1, Beta2, and Theta-Beta Ratios for Both Groups and by Group for Training Session Measures

	Training Session Measure		
Frequency Band/Group	Beginning/Middle	Beginning/End	Middle/End
Both Groups [a]			
Theta	.23	.56*	.29
ADHD Group [b]			
Beta1	.46	.61	.71
Theta-Beta1 ratio	.07	-.01	.66
ADD Group [c]			
Beta2	.81***	.71**	.90***
Theta-Beta2 ratio	.75**	.82***	.76**

* $p < .05$, ** $p < .01$, *** $p < .001$

[a] $n = 19$.

[b] $n = 5$.

[c] $n = 14$.

Research Question 2: QEEG Analysis

Mean theta magnitude was analyzed for both the eyes closed and reading conditions pre and post-training (see Table 5 and Figures 3, 4). The mean theta magnitude decreased significantly at the sites FZ ($p < .01$), CZ ($p < .01$), C3 ($p < .05$), and C4 ($p < .05$) for the eyes closed condition. Although the mean theta magnitude also decreased at the PZ site from 29.2 microvolts (uV) to 27.7 uV, this decrease was not statistically significant. While the mean theta magnitude also decreased at all sites after training for the reading condition, differences were not statistically significant except at the CZ ($p < .05$) site where the mean theta magnitude decreased from 26.4 uV to 24.3 uV.

Differences in beta1 and beta2 mean magnitudes pre and post-training were also analyzed for the eyes closed and reading conditions. In the eyes closed condition, both beta1 and beta2 mean magnitudes increased or decreased slightly, or remained the same from pre to post-training (see Table 6). None of the differences were statistically significant. For the reading condition, beta1 and beta2 mean magnitudes also increased or decreased slightly, or remained the same from pre to post-training and no significant differences existed (see Table 7).

Because somewhat differing results were obtained between the eyes closed and reading conditions in terms of the decrease in theta pre and post-training, *post hoc* analysis was conducted to determine whether the theta frequency band would show any significant changes in another condition. Eyes open was chosen as the condition for analysis both because this condition has also been demonstrated to have high test-

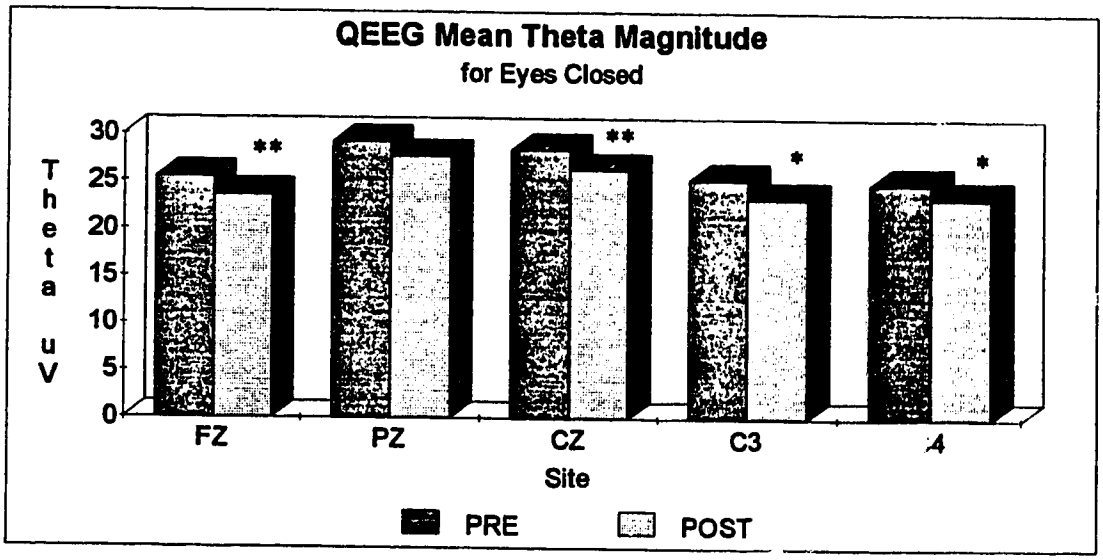
TABLE 5

QEEG Mean Theta Magnitude for Eyes Closed and Reading Conditions for Both Groups[a]

	Eyes Closed				Reading			
	Pre		Post		Pre		Post	
Site	M	S.D.	M	S.D.	M	S.D.	M	S.D.
FZ	25.5	5.99	23.6**	5.44	24.1	4.16	23.6	5.44
PZ	29.2	9.56	27.7	10.36	24.6	4.69	23.1	6.21
CZ	28.4	6.41	26.3**	6.83	26.4	4.55	24.3*	5.64
C3	25.3	5.43	23.3*	6.30	23.1	4.16	21.9	5.01
C4	24.9	5.88	23.4*	6.40	22.6	3.74	21.2	4.79

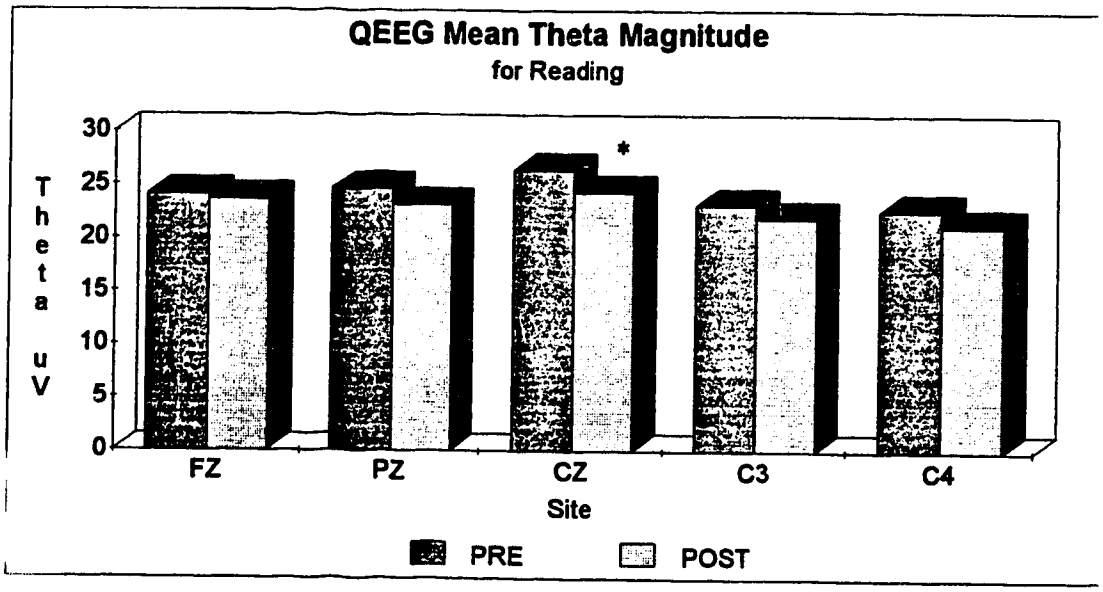
* $p < .05$, ** $p < .01$
[a] $n = 19$.

Figure 3



* p <.05, ** p .<.01
n = 19

Figure 4



* p .<.05
n = 19

TABLE 6

QEEG Mean Magnitude for Beta1 and Beta2 by Group for Eyes Closed Condition

	Beta1 (ADHD Group[a])				Beta2 (ADD Group[b])			
	Pre		Post		Pre		Post	
Site	M	S.D.	M	S.D.	M	S.D.	M	S.D.
FZ	9.4	.82	9.3	.56	7.6	1.56	7.6	1.97
PZ	10.5	1.28	11.7	.47	7.2	1.34	7.5	1.92
CZ	9.9	.62	9.9	.41	7.2	1.42	7.2	1.89
C3	9.7	.65	7.9	.94	7.0	1.39	7.1	1.80
C4	9.7	.58	9.8	.50	7.3	1.47	7.2	1.96

No significant differences found.
[a] n= 5.
[b] n= 14.

TABLE 7

QEEG Mean Magnitude for Beta1 and Beta2 by Group

for Reading Condition

	Beta1 (ADHD Group[a])				Beta2 (ADD Group[b])			
	Pre		Post		Pre		Post	
Site	M	S.D.	M	S.D.	M	S.D.	M	S.D.
FZ	8.8	1.47	8.9	.84	7.6	1.63	7.8	1.85
PZ	10.0	2.47	10.0	2.24	8.3	2.11	9.1	2.68
CZ	9.0	1.69	9.1	1.29	7.6	1.63	7.9	2.07
C3	8.4	1.40	8.5	.82	7.3	1.53	7.8	2.04
C4	9.0	1.68	9.4	.84	7.9	2.06	7.7	1.71

No significant differences found.
[a] n= 5
[b] n= 14.

retest reliability (Graap, 1994) and because it provided another condition in which subjects' eyes were open during the QEEG assessment as they were during the reading condition. Interestingly, the results of the eyes open condition (see Table 8 and Figure 5) are identical to those of the eyes closed condition: significant decreases ($p < .01$) pre and post--training were found in mean theta magnitude for all sites (FZ, CZ, C3, C4) except PZ.

Further *post hoc* analysis was conducted to examine whether significant differences existed between the mean standard deviations pre and post-training. It has been argued that the use of variability between subjects as data rather than as error is important in clinical biofeedback experimentation (Barlow, Blanchard, Hayes, & Epstein, 1977). The theta frequency band was chosen for analysis and data was collected on each subject's standard deviation of his theta magnitude at each site pre and post-training. The SPSS program then calculated the mean standard deviation for each site pre and post-training for each condition. Standard deviations provide a measure of the distribution of a group of scores, and a decrease in the mean standard deviation after neurofeedback training could indicate that subjects' individual magnitudes moved closer to the overall mean after training.

Theta standard deviations were examined for the eyes closed, reading, and eyes open conditions. In the eyes closed condition, the mean theta standard deviation decreased from pre to post-training but none of the differences were significant (see Table 9 and Figure 6). Mean theta standard deviations for the reading condition decreased slightly or remained the same from pre to post-training and, again, none of

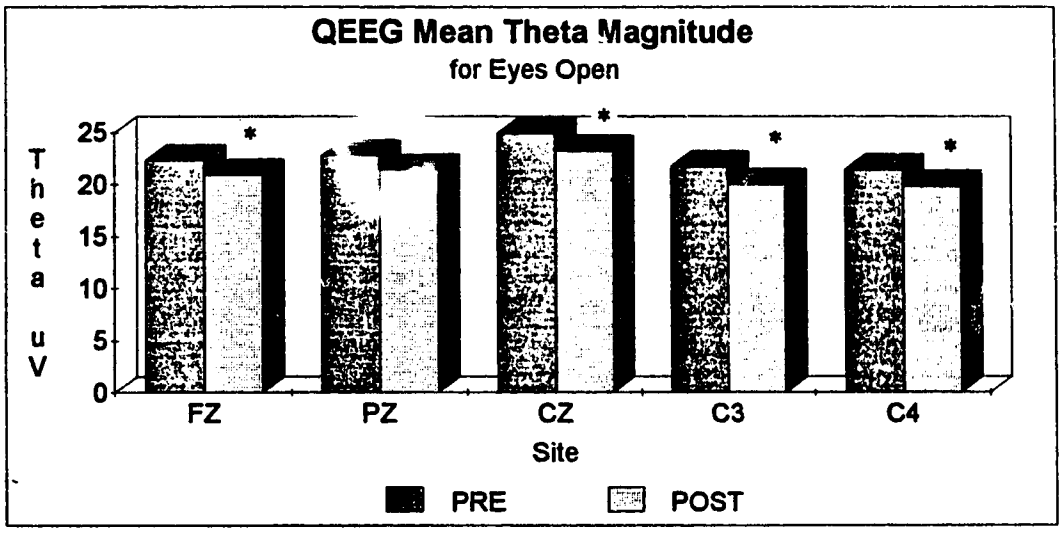
TABLE 8

Mean QEEG Theta Magnitude for Both Groups[a] for Eyes Open Condition

	Pre		Post	
Site	M	S.D.	M	S.D.
FZ	22.3	4.40	20.9*	4.11
PZ	22.8	4.63	21.4	4.92
CZ	24.9	4.66	23.2*	4.94
C3	21.7	3.44	20.0*	3.97
C4	21.4	3.71	19.8*	3.67

*$p < .01$.
[a] $n = 19$.

Figure 5



*.p <.01
n = 19

TABLE 9

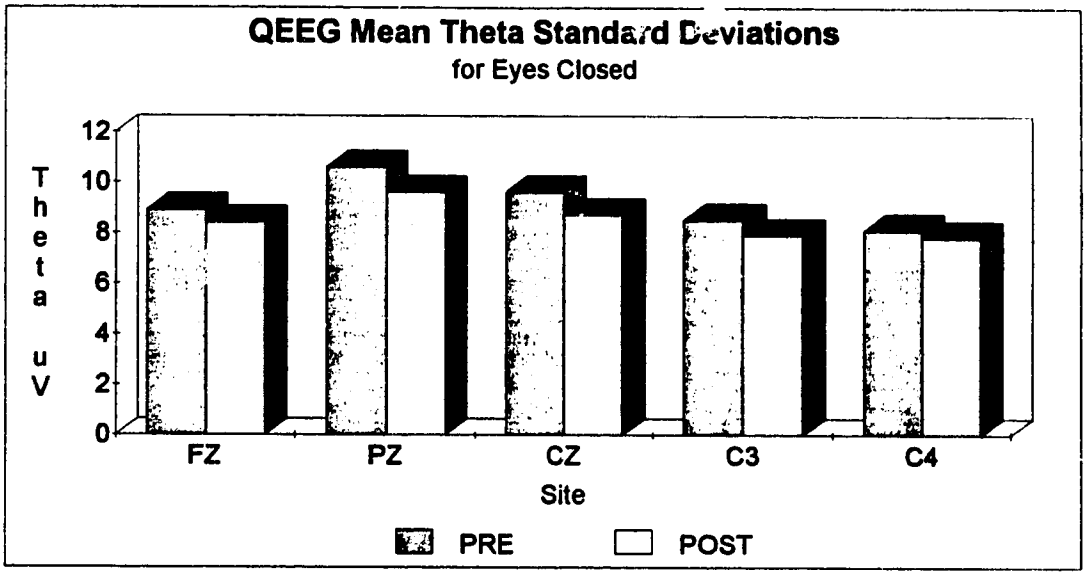
QEEG Mean Theta Standard Deviations for Both Groups [a]

for Eyes Closed and Reading Conditions

	Eyes Closed		Reading	
	Pre	Post	Pre	Post
Site	M	M	M	M
FZ	8.9	8.4	8.0	8.0
PZ	10.6	9.6	8.6	7.9
CZ	9.6	8.7	8.3	8.1
C3	8.5	7.9	7.3	7.3
C4	8.1	7.8	7.6	7.1

No significant differences found.
[a] n= 19.

Figure 6



No significant differences found.
n = 19

the differences were significant (see Table 9 and Figure 7). In the eyes open condition (see Table 10 and Figure 8), mean theta standard deviations also decreased from pre to post-training, and the difference was significant at the CZ site ($p < .01$).

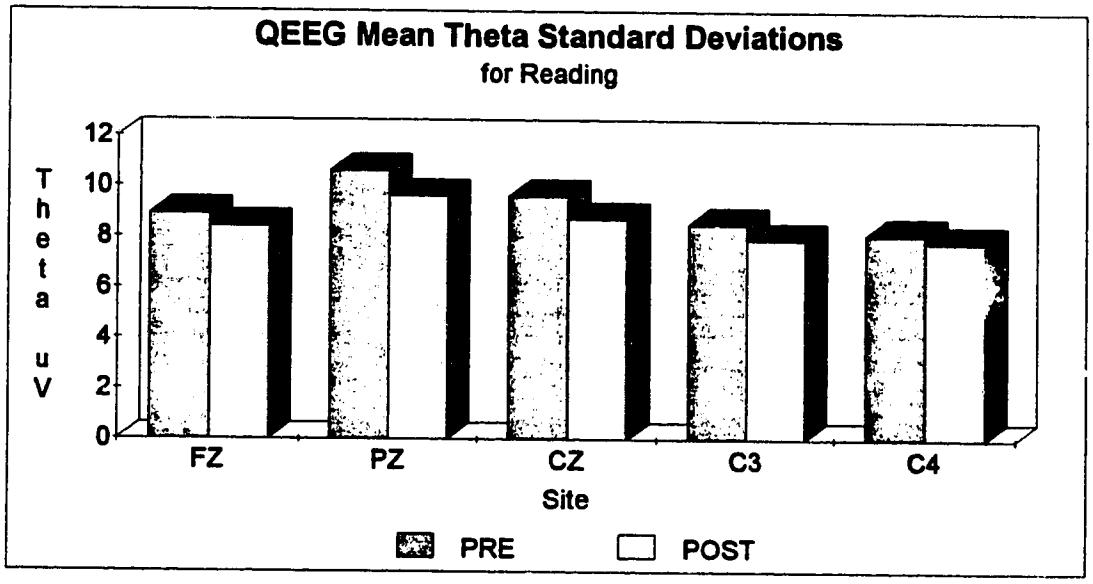
Research Question 3: Behavioral Ratings Analysis

The Child Behavior Checklist (CBCL) indicated that parents rated their children significantly improved on the attention scale from pre to post-training (see Table 11 and Figure 9. The mean T-score before neurofeedback training was T= 67.8 and after training was T= 62.4. This difference is significant at the $p < .01$ level. Parents did not, however, rate their children as significantly improved on the school scale (pre-training T-score= 32.2; post-training T-score= 34.4).

Summary

In summary, training data did not show significant change between the beginning, middle, and end of training sessions for any of the frequency bands beta1, beta2 or theta. Further, significant change was not evident on either theta-beta1 or theta-beta2 ratios. However, significant differences were found pre and post-training on QEEG assessments for the theta frequency band for both the eyes closed and eyes open conditions for the sites FZ, CZ, C3, and C4. PZ was not significant for either condition. Theta decreased significantly at CZ but not at other sites for the reading condition. The mean standard deviations of theta did not change significantly for the eyes closed or reading conditions but did decrease significantly at the CZ site for the eyes open condition. Neither beta1 or beta2 activity changed significantly from pre to post-training QEEG assessments. Lastly, parents rated their children as significantly

Figure 7



No significant differences found.
n = 19

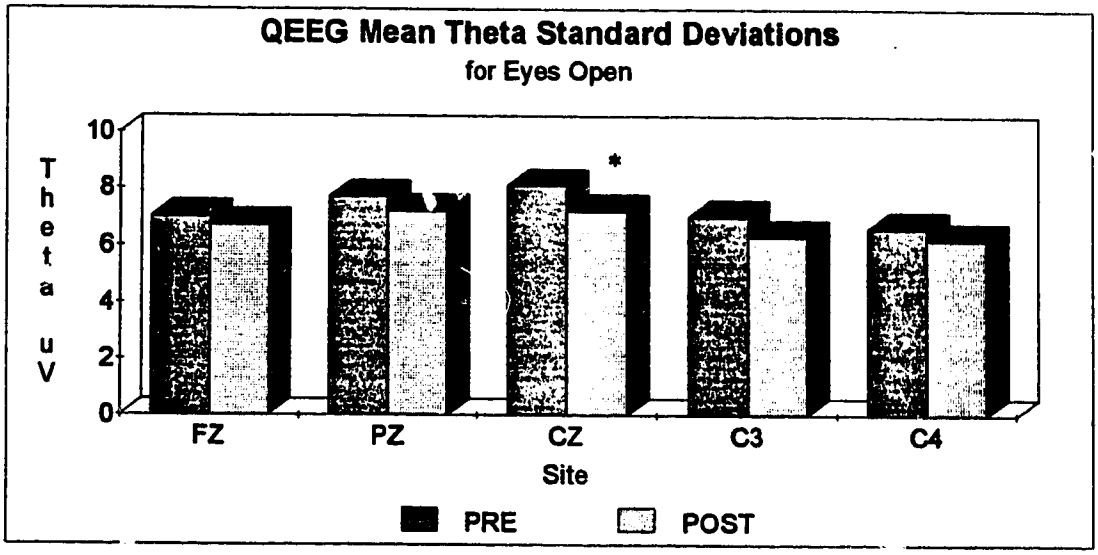
TABLE 10

QEEG Mean Theta Standard Deviations for Both Groups [a] for Eyes Open Condition

	Pre	Post
Site	M	M
FZ	7.0	6.7
PZ	7.7	7.2
CZ	8.1	7.2*
C3	7.0	6.3
C4	6.6	6.2

*$p < .01$
[a] $n = 19$.

Figure 8



* $p < .01$
$n = 19$

TABLE 11

Parental Behavior Ratings[a] for Children

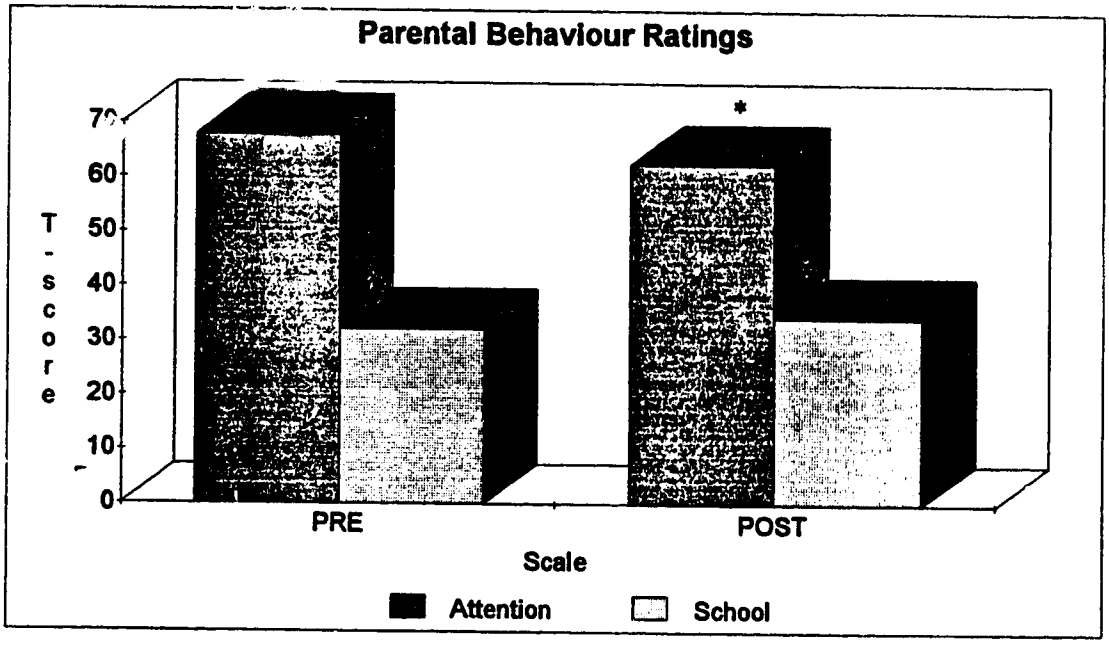
for Pre and Post-Training Periods for Both Groups[b]

	Pre		Post	
Scale	M	S.D.	M	S.D.
Attention	67.8	8.18	62.4*	9.41
School	32.2	7.02	34.4	6.24

* $p < .01$

[a] Data presented in T-scores.
[b] $n = 19$.

Figure 9



* $p < .01$
$n = 19$

improved on the attention scale of the CBCL but not on the school scale.

CHAPTER FIVE

DISCUSSION

This study examined three outcome measures of neurofeedback training for ADHD and ADD to determine if success was evident on any of these measures. Success was evident on two of the outcome measures, QEEG assessment data and parental behavior ratings. Success was not found on the third outcome measure, training data collected during each child's neurofeedback training sessions. The QEEG results of this study can not be compared to other studies because no known studies have compared QEEG measures before and after neurofeedback training. This chapter will discuss the results of each of the outcome measures, followed by a discussion of the study's limitations, and, finally, suggestions for future research will be provided.

Training Sessions

Beta

Neither of the three frequency bands examined in the training session data (beta1, beta2, theta) showed change associated with improvement between the beginning, middle, and the end of the training period. Further, qualitative analysis of each subject's data across time showed no consistent patterns in beta or theta activity. Improvement in the two beta bands is associated with an increase in uV from pre to post-training, while improvement in the theta frequency band is associated with a decrease in uV post-training. Increases in the sensorimotor rhythm (SMR) band (12-14 Hz; beta1 in the

present study) after neurofeedback training have been documented by several researchers using training session data as an outcome measure. Tansey (1990, 1991) utilized SMR training for learning disabled children, reporting their SMR activity increased. Lubar and Shouse (1976) reported a "substantial" increase in SMR activity in a case study of a hyperkinetic child who received SMR training in which training occurs at the C3-C4 sites. In a replication study, Shouse and Lubar (1979) again reported increases in SMR activity in hyperkinetic children after SMR training. However, in both of these studies, subjects received over 150 training sessions, whereas in the present study the mean number of training sessions subjects received was 50. It could be the case the changes in the beta1 frequency band take time to show in training data. Lubar and Lubar also (1984) found that SMR and beta1 (16-20 Hz; beta2 in the present study) increased with SMR training for six ADD children. In this study, children received fewer training sessions (20-54) than in Lubar's previous studies. However, training occurred over 10 to 27 months. Again, this time period is considerably longer than that of subjects involved in the present study, 23.2 weeks or approximately six months.

Theta

In the studies discussed in the previous section, concomitant with increased SMR activity was decreased theta activity. Most recently, Lubar et al. (1995) reported training ADHD children at the FZ-PZ sites in which success was defined as the reduction of theta uV. Subjects received 40 training sessions over two months. This study is of particular interest because of the much shorter training time period than Lubar's previous studies. A statistically significant reduction in theta uV across sessions was found for 12 of the

19 of the subjects involved in the Lubar et al. study but no change in theta was evident for the other subjects. Again, it may be the case that changes are not necessarily always apparent in training data in shorter time periods.

Lubar (1991) has suggested that theta-beta ratios may be a better measure of success in training data than theta or beta uV alone. In the present study, theta-beta ratios did not show significant decrease across training session measures. It must be noted, however, that only selected training session measures were chosen for analysis and these measures may not be entirely representative.

QEEG Assessments

Beta

Neither beta1 or beta2 increased significantly after neurofeedback training for either the eyes closed or reading conditions. Both beta1 and beta2 uV increased or decreased slightly, or remained the same. Interestingly, somewhat contradictory findings have arisen from QEEG assessments with regard to differences in beta activity between ADHD/ADD subjects and normal controls. Mann (1990) found decreased beta activity (12-21 Hz) in ADHD subjects when compared to matched controls. Janzen (1992) also found that ADD subjects had decreased activity in the 12-20 Hz range as compared to normal controls. However, Janzen et al. (1995) did not find significant differences in beta activity between ADD subjects and normal controls. Similarly, Lubar et al. (1985) did not find that the beta frequency band distinguished between learning disabled children and controls. It may be that beta does not distinguish between ADHD children and "normal" children and, as such, is not as important an indicator of success on QEEG

assessments as is theta. Because the literature does not clearly point in one direction with beta, it is difficult to make any firm conclusions regarding beta activity within ADHD/ADD populations.

Theta

Identical results were found in the eyes closed and eyes open condition for the theta frequency band. In both conditions theta uV decreased significantly at the FZ, CZ, C3, and C4 sites. While theta decreased in both conditions at PZ, the decrease was not significant. Ahn, Prichep, & John (1980) found that excessive theta waves in the parietal (PZ) and occipital regions distinguished learning disabled children from normal controls more than any other site or frequency band. Gasser et al. (1988b) determined that the theta band matures first in the occipital regions, followed by PZ, then the central sites mature, followed by the frontal sites. Perhaps in neurofeedback training, the effects of training for the theta frequency band are seen first in frontal areas since that area matured last. In other words, while the maturation of the theta band occurs from posterior to anterior regions, perhaps training moves in the opposite direction from anterior to posterior regions. This would explain the results of this study in which theta was significantly reduced post-training in frontal and central regions but not at a more anterior region (PZ). It would be interesting to conduct another QEEG assessment with each of the children involved in this study to determine if theta was now significantly reduced at PZ and occipital regions in comparison to their pre-training assessments.

The reading condition provided somewhat differing results from the eyes closed or eyes open conditions. Once again, theta was found to have decreased at all five sites

but the decrease was only significant at CZ. Mann (1990) found that increased theta was greatest between ADHD subjects and controls for reading and drawing conditions. It is not clear why only one site (CZ) showed a significant decrease in theta uV. A potentially confounding variable is that while the mean standard score of all subjects on the WRAT-R reading subtest was 92.8, seven of the subjects in the study (37%) had a standard score on the reading subtest which was at or below one standard deviation of the population mean. Further, in most of these cases, the subject's full scale IQ is at or above the population mean FSIQ. Thus over one-third of the sample in this study may be a poor reader or have a reading disorder. The comorbidity of ADHD with learning disorders was discussed in Chapter Two (see Biederman et al., 1991). It has been suggested that poor readers produce more theta activity than good readers (Galin et al., 1992; Lubar et al., 1985), although these findings have been disputed (Fein et al., 1986). If, in fact, reading ability is related to theta activity, the poor readers in the present study may have artificially elevated the overall theta uV means in the reading condition. Barlow, Blanchard, Hayes, & Epstein (1977) make an important point in regard to clinical biofeedback experimentation, stressing that statistical significance and clinical significance are not necessarily the same and that clinical significance may be apparent when statistical significance is not. Thus although significant differences were not found for most of the sites in the reading condition, the decreases post-training in theta uV may still be of clinical importance.

Variability between subjects as measured by theta standard deviations decreased from pre to post-training for all sites for both the eyes closed and eyes open conditions.

The decrease was only significant at CZ, however. Nonetheless, this suggests that variability between subjects in theta magnitude was reduced after neurofeedback training. Less change in the variability between subjects was evident in the reading condition in which mean theta standard deviations decreased or remained the same from pre to post-training. Again, perhaps the influence of poor readers in the sample is evident here.

An interesting pattern emerged in the present study. For every site, mean theta uV were the highest in the eyes closed condition and the lowest for the eyes open condition, with the reading condition falling in the middle. This pattern existed for both pre and post-training data. One would expect that mean theta uV would be highest in the eyes closed condition because as soon as the eyes are closed, theta and alpha activity increases. Thus the eyes closed condition is likely a good measure to use for evaluating if theta uV decrease post-training because theta magnitude can be expected to be maximized during this condition.

Behavioral Ratings

In this study, parents rated their children as improved on the attention scale of the CBCL from pre to post-training and the improvement was statistically significant. The CBCL attention scale has been shown to be a significant predictor of ADHD (Biederman et al., 1993; McConaughy et al., 1994). Lubar et al. (1995) also found that parents rated their ADHD children as significantly improved on the attention scale of a behavioral ratings scale (McCarney Attention Deficit Disorders Evaluation Scale) after neurofeedback training. The researchers further determined that children were rated as significantly improved on the attention scale regardless of whether their training data

showed significant improvement (decreased theta). Lubar et al. stressed that parents may tend to overstate gains made by their children as a result of a treatment such as neurofeedback training. Similarly, Drotar et al. (1995) and Reid et al. (1993) stressed the subjective nature of the CBCL. Thus although a significant improvement was found on the attention scale for subjects in this study, the subjective nature of this outcome measure and the fact that the subjects were not rated by their parents as significantly improved on the school scale, must be borne in mind.

Conclusion

This study sought to answer three research questions relating to outcome measures for neurofeedback training. While training data did not indicate success, QEEG data and behavioral ratings did. Thus two of the three outcome measures indicated success after neurofeedback training. As has been discussed, it could be that success takes quite a long time to be apparent on training data or that the training session measures chosen for analysis in this study were too selective. Also, behavioral ratings should be viewed as a somewhat subjective measure. Success was evident from QEEG outcome measures in terms of significantly reduced theta magnitudes in three conditions and reduced theta variability between subjects after neurofeedback training. Neurofeedback training clearly appears to affect brain wave patterns and, as such, has a very important role to play in ADHD/ADD.

Limitations

Several limitations to this study must be outlined. The *ex post facto*

design of the study could pose threats to internal validity. Also, the generalizability of the results is somewhat limited because of the sample size. Some further limitations relate specifically to neurofeedback training and QEEG assessment. Subjects in this study received bi-polar training in which there is no certainty as to which of the two sites is actually receiving training at any given time. Also, of late biofeedback studies have begun using electrodes under subject's eyes to measure eye muscle movement so that this movement can be artifacted. However, although facial electrodes were used in the QEEG assessments of subjects in this study, eye electrodes were not used and thus some eye muscle movement may have contributed to amplitude increase.

Future Research

The next step in examining outcome measures of neurofeedback training for ADHD/ADD would be a study utilizing a true or a quasi-experimental design with a matched ADHD or ADD control group which does not receive neurofeedback training. Such a design would help to control threats to internal validity more rigorously. Also, a larger sample size would be desirable to increase the generalizability of the results. While only parental behavioral ratings scales were used in this study, the use of teacher behavioral ratings would be useful in a subsequent study to obtain teachers' perspectives on subjects.

Clearly much research remains to be done which evaluates neurofeedback training for ADHD/ADD in terms of different outcome measures of success. A replication study which examines outcome measures from subjects trained with monopolar protocols would provide important information. In monopolar neurofeedback training, electrodes are

placed on one site (i.e. C3 or C4/ FZ or PZ) and alternated each session. Currently, the Cognitive Re-regulation Program at the University of Alberta is using monopolar neurofeedback training and is examining the relationship between monopolar training session data and QEEG data. In summary, it is hoped that the present study adds to the neurofeedback literature and, above all, that this research is of real benefit to ADHD and ADD children.

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Appendix A

DSM-IV Diagnostic Criteria for ADHD

Either (1) or (2):

(1) six (or more) of the following symptoms of **inattention** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.

(b) often has difficulty sustaining attention in tasks or play activities.

(c) often does not seem to listen when spoken to directly.

(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions).

(e) often has difficulty organizing tasks and activities.

(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).

(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools).

(h) is often easily distracted by extraneous stimuli.

(i) is often forgetful in daily activities.

(2) six (or more) of the following symptoms of **hyperactivity-impulsivity** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat.

(b) often leaves seat in classroom or in other situations in which remaining seated is expected.

(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).

(d) often has difficulty playing or engaging in leisure activities quietly.

(e) is often "on the go" or often acts as if "driven by a motor".

(f) often talks excessively.

Impulsivity

(g) often blurts out answers before questions have been completed.

(h) often has difficulty awaiting turn.

(i) often interrupts or intrudes on others (e.g., butts into conversations or games).

Appendix B

Parental Consent Form

Consent Form

I understand that the Cognitive Re-regulation Program at the University of Alberta is experimental, and that, while significant reduction in disability has been reported at other similar centres, no guarantee of improvement is made as a result of participation in this program.

I also understand the following items:

(a) that the Cognitive Re-regulation Program will receive $750. prior to commencement of initial dynamic EEG assessment.
(b) that following the initial dynamic EEG assessment, the Cognitive Re-regulation Program reserves the right to include or exclude any child on the basis of the results of the assessment.
(c) that the parent or the child have the right to revoke consent and withdraw from the program.
(d) that children included in the training phase of the program will participate in approximately 40 training sessions over a period of 20 weeks, or more if necessary, to complete the training.
(e) that if there is no progress, the team reserves the right to terminate the child's participation in the program.
(f) that children included in the program will be followed for one year after the completion of the training phase (questionnaires for parents/teachers, copies of report cards, one year post-dynamic EEG assessment).
(g) that the Cognitive Re-regulation Program will receive $3000. for 40 training sessions. There may be a charge of $75. per session for any in excess of that number.
(h) that children will adhere strictly to the training schedules by attending appointments and by arriving at least five minutes early for each session.

As the University is a teaching and research facility, I understand that participants in the program may be observed and trained by graduate students during the course of the individual's training.

I give permission for the data from the dynamic EEG assessment and training sessions to be used for current and future research purposes. I understand that the information will be coded in such a way that the identities of individual children will be kept confidential.

I also understand that records will be kept on each child, that information regarding the child's progress may be given to his/her school, and that parents will be provided feedback regarding assessment results and progress during training. Further, upon completion of the child's participation in the program, parents and participants will be given the opportunity to meet with the team to review the results of training and seek other information regarding the program.

I authorize Dr. George Fitzsimmons to request and receive information concerning the applicant from any or all schools attended by the applicant, persons having done assessments on the applicant (i.e. psychologists), and physicians who have seen the applicant, which by law might otherwise be considered to be confidential or privileged.

I have read the attached description of the Cognitive Re-regulation Program and understand the nature of this program, and the terms as outlined above. I give consent for the applicant on this referral to participate in the University of Alberta's Cognitive Re-regulation Program.

Parent's or Guardian's Signature _____ Date _____

Signature of Witness _____ Date _____

Child's Signature _____ Date _____

Appendix C

The International 10-20 Electrode System