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A Comparison of EEG Activity in Adults with Attention Deficit Hyperactivity Disorder and Normal Controls while Performing Tasks that Require Attention

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A thesis presented to the Faculty of Graduate and Postdoctoral Studies of the University of Ottawa as partial fulfillment of the requirements for the degree of Doctor of Philosophy

I would like to dedicate this work to the most important souls in my life, my wife Susan, my mother Yvonne, my father Jacob and all the pussycats with whom I have shared this journey; Fred, Dusty, Tabby, Snowball, Oliver, Oscar, Alex, Granby and William. To my little honey bee, I thank you for your endless energy, support, and love.

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TABLE OF CONTENTS

TABLE OF CONTENTS Cont.

TABLE OF CONTENTS Cont.

TABLE OF CONTENTS Cont.

LIST OF FIGURES

LIST OF TABLES

LIST OF ABBREVIATIONS

ADD-RT	Attention Deficit Disorder-Residual Type
ADD-H	Attention Deficit Disorder-Hyperactive Type
ADHD	Attention Deficit Hyperactivity Disorder
ADHDhy	Attention Deficit Hyperactivity Disorder- Primarily Hyperactivity
ADHDpi	Attention Deficit Hyperactivity Disorder- Primarily Inattentive
ADHDco	Attention Deficit Hyperactivity Disorder- Combined Type
ANOVA	Analysis of Variance
CD	Conduct Disorder
CNS	Central Nervous System
CPT	Continuous Performance Test
CT	Computerized Tomography
DFA	Discriminant Function Analysis
DSM	Diagnostic and Statistical Manual of Mental Disorders
EEG	Electroencephalography
EMG	Electro-Myography
EOG	Electro-Oculography
ERP	Event Related Potential
FFT	Fast Fourier Transform
GSR	Galvanic Skin Response
HRC	Hyperkinetic Reaction of Childhood
IVA	Intermediate Visual and Auditory
MANOVA	Multivariate Analysis of Variance
MBD	Minimal Brain Disfunction
MRI	Magnetic Resonance Imagery
ODD	Oppositional Defiant Disorder
PET	Positron Emission Tomography
RT	Reaction Time
TOVA	Tests of Variability of Attention
SD	Standard Deviation
SMA	Sensory Motor Area
SMR	Sensory Motor Rhythm
SPECT	Single Positron Emission Tomography
qEEG	Quantitative Electroencephalography
WAIS-R	Weschler Adult Intelligence Scale-Revised
WJ	Woodcock Johnson

ABSTRACT

Children with Attention Deficit Hyperactivity Disorder (ADHD) have been reported to have electroencephalographic (EEG) abnormalities in the form of increased levels of theta band activity and lower than normal levels of beta band activity. The purpose of the present study was to determine if these abnormalities can also be observed in adults with ADHD. There were 32 control subjects, 25 subjects with ADHD of the primarily hyperactive type (ADHDhy), and 17 subjects with ADHD of the primarily inattentive type (ADHDpi). For the purposes of analysis, the ADHDhy and ADHDpi groups were combined to form a CLINICAL group. The subjects were right handed males and females between the ages of 20 and 50 years of age. During the study, EEG activity was recorded from 19 electrode sites while subjects sat with their eyes open and eyes closed, and while they performed a variety of tasks including: the Tests of Variability of Attention (TOVA), a reading task, a mental rotation task, a selective attention task, and a listening task. The results of the discriminant function analyses produced functions that correctly classified an average of 60 out of 74 of the control and ADHD subjects during the TOVA, listening, and selective attention tasks ($p < .0005$). An ANOVA of inter-hemispheric activity revealed that only the ADHDpi group were significantly different from the control group with more right than left hemispheric activity in the delta band during the mental rotation task ($p < .006$). A paired t-test

analysis of inter-hemispheric activity showed that both the ADHDhy and ADHDpi groups had different percentage levels of right and left hemispheric activity ($p < .005$) during the performance of the eyes closed, T.O.V.A., mental rotation, and reading tasks; there were no significant differences in the control group in any of these comparisons. A MANOVA of regional (frontal, central, posterior, left temporal, and right temporal) activity revealed statistically significant differences in the theta/beta ratio during the eyes closed condition for the ADHDhy group with a higher ratio in the frontal region and right temporal area ($p < .006$). The results suggest that the differences between normal and ADHD children continues into adulthood but that the magnitude of the differences is reduced. A dysfunction in EEG activity caused by a maturational lag is supported by the results of this study. Based on the results of the analyses, recommendations are made as to which frequency bands and electrode sites should be targeted for use in neurotherapy.

CHAPTER 1

INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is considered to be one of the most perplexing and frequently diagnosed disorders among school aged children (Brown, Borden, Wynne, Schleser & Clingerman, 1986; Lubar, 1991; Wolraich, Lindgren, Stromquist, Milich, Davis & Watson, 1990) and recognized as a common reason for pediatric, neurologic, and child psychiatric referrals (Biederman, Munir, Knee, Habelow, Armentano, Autor, Hoge & Wateranux, 1986; Satterfield & Dawson, 1971). There is no agreement on the prevalence of this disorder, with some estimates varying from 5% to 15% of children (Biederman et al., 1986; Lubar, 1991; Lubar, Swartwood, Swartwood & O'Donnell 1995; Satterfield & Dawson, 1971) and others, using more rigorous criteria, ranging from 1% to 5% (Biederman et al. 1986; Greenburg, 1992; Satterfield & Dawson, 1971; Wolraich et al. 1990; Wender, 1995; Zametkin, Nordhal, Gross, King, Semple, Rumsey, Hamburger & Cohen, 1990). The exact percentages have been difficult to determine due to ambiguous diagnostic criteria and methodological problems such as sample size. It is, however, generally agreed that the age of onset is around seven years and that approximately four times as many boys as girls are diagnosed with the disorder (Biederman et al., 1986; Donnelly, 1989; James & Taylor, 1990). A high percentage of family members (35%) have also

been diagnosed with this illness and it is estimated that between 20% to 60% of children who suffer from ADHD will continue to experience it as adults (Biederman et al., 1986; Lubar, 1991; Lubar, Swartwood, Swartwood & O'Donnell 1995; Wender, 1995).

The difficulties experienced by the ADHD adult are not limited to those of attention, however, as ADHD is also thought to be a major precursor of adult psychopathology (Biederman et al. 1986) and has been associated with increased risk of alcoholism, sociopathy, and hysteria (Wender, Reimherr & Wood 1981). As adults, individuals with ADHD often experience interpersonal difficulties leading to high rates of divorce, difficulty holding jobs, and decreased potential for professional advancement (Lubar, 1985).

The Emergence of ADHD as a Disorder

One of the earliest references to behaviours that would currently be classified as ADHD was reported by G. F. Still (1902), who wrote that some children displayed "abnormal defects in moral control,... and wanton mischievousness and destructiveness". The "wanton mischievousness and destructiveness", referred to by Still (1902), are perhaps indications of the defining criteria for a diagnosis of Conduct Disorder (CD), a separate category in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders' (DSM-IV) Attention Deficit and Disruptive Behaviours classification (1994). Whereas,

"abnormal defects in moral control", appears to be a reference to impulsivity, one of the three defining features of ADHD in the DSM-IV, the other main characteristics of the disorder are inattention and hyperactivity.

The first researcher to note an organic cause related to ADHD behaviours was Kennedy (1924) who observed that most children who had suffered from epidemic encephalitis, following a severe influenza outbreak in 1918, displayed hyperactivity ranging from "slight twitches of the fingers" to marked changes in behaviour such as "running around and performing all kinds of strange acts" (p. 160). Similar observations were also noted by Stryker (1925) who reported that influenza related encephalitis had resulted in a number of cases of children who subsequently developed anti-social behaviours characterized by decreased attentiveness, impulsiveness, and hyperactivity.

The childhood behavioural disorders that had earlier been reported by Kennedy (1924) and Stryker (1925) were, by the 1930's, thought to be the result of some kind of 'minimal brain damage'. This 'brain damage' included a wide range of symptoms such as over activity, inattentiveness, impulsivity, affective lability and moodiness, temper outbursts, immaturity, poor peer relations, disobedience, defiance, hostility and other acting out behaviours (Wender, 1995). Researchers at that time were unclear as to whether these symptoms were related; however, there was a great deal of evidence to suggest that the syndrome was

organically based. For example, blood relatives displayed varying degrees of the behavioural dysfunctions and the disorder had been associated with brain injury, mental retardation, perinatal complications, and toxic reactions to chemicals (Lubar, 1991). By 1947 the wide range of minimal brain damage symptoms had been merged into one classification known as Minimal Brain Dysfunction Syndrome (MBD) or "Strauss Syndrome" (Strauss & Lehtinen, 1947). Medical research continues to support the view that the disorder is organically based (Lubar, 1999; Wender, 1995).

During the 1940's and 1950's psychometric measures began to be used to differentiate Strauss Syndrome patients from normal individuals; the most popular of these were the Bender Visual Motor Gestalt Test and the Stanford Binet Test. The classification of MBD or Strauss syndrome was used to describe a variety of symptoms such as hyperkinetic behaviour, learning disabilities, disorders of conduct, and disorders of attention resulting in a classification considered too broad (Lubar, 1991) and "vague, over-inclusive and of no prescriptive value" (Barkley, 1990). Because the core characteristic of MBD appeared to be hyperactivity the diagnostic term was changed in 1968 from MBD to Hyperkinetic Reaction of Childhood (HRC). A further refinement of the classification occurred when research suggested that deficits in attention, not hyperactivity, were primarily responsible for the behavioural and learning problems. As a result, the diagnosis

was changed to Attention Deficit Disorder with or without Hyperactivity in 1980; ADD and ADD-H respectively (DSM-III, 1980). The DSM-III described the adult version of the disorder as Attention Deficit Disorder-Residual Type (ADD-RT); however, the criteria continued to be vague and relied upon the clinician to apply childhood and adolescent diagnostic criteria to the adult (Wender, 1995). The diagnosis was further modified to Attention Deficit Hyperactivity Disorder (ADHD) in 1987 (DSM-III-R) but there were no specific measures for an adult diagnosis. The DSM-III-R stated that the diagnosis was to be applied when the patient had ADHD symptoms in childhood or adolescence and when these symptoms persisted into adulthood.

The DSM-IV (1994) provides three subtypes of ADHD for adults and children; Attention Deficit/Hyperactivity Disorder, Combined Type(ADHDco); Attention Deficit/Hyperactivity Disorder, Predominantly Inattentive Type (ADHDpi) and Attention Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type (ADHDhy). The adult criteria, however, are unchanged from the DSM-III-R giving the clinician or researcher no explicit set of descriptors (Wender, 1995).

Research studies of ADHD have been hindered by disagreements involving how the disorder is to be defined, its relationship to other disorders, and how the relevant dimensions should be measured. Notwithstanding the greater precision of

the DSM-IV's diagnostic criteria for children, there still appears to be some overlap between ADHD and other learning disorders, such as dyslexia, and clinical diagnoses such as CD or Oppositional Defiant Disorder (ODD) (Lubar, 1991; Wender, 1995). For example, approximately 50% of the children with a diagnosis of ADHD have a specific learning disorder and at least 50% of this group meet the criteria for ODD or CD (Fletcher, Morris & Francis, 1991). The overlap of ADHD with other disorders, as well as the vague diagnostic criteria for adults, increases the difficulty of reaching an accurate diagnosis. In fact, a number of clinicians have noted that the definition of ADHD has become one of exclusion, i.e., one that does not include primary visual or auditory disorders or mental retardation (Lubar, 1985; Lubar, Bianchini, Calhoun, Lambert, Brody & Shabsin, 1985).

The criteria for the diagnosis of ADHD have been frequently revised over the past 50 years as evidenced by the variety of terms used to describe it, i.e., Strauss Syndrome, MBD, HRC, ADD, ADD-H, ADD-RT, and ADHD. The revisions, however, have focused on the childhood diagnosis and have not assisted the clinician in arriving at an accurate adult diagnosis (Wender, 1995). Moreover, until recently, the clinical criteria for a diagnosis of ADHD, for either the adult or child, were based on questionnaires and diagnostic interviews with little empirical

support for the diagnosis. As a result, the diagnosis of ADHD is still perhaps controversial although the recent development and use of computerized tests of attention such as the Tests of Variability of Attention T.O.V.A. (Greenberg, 1992) have provided much needed objective criteria for this diagnosis.

Treatment of ADHD

There are two neurophysiological models that provide the basis for the treatment of ADHD. The first proposes that a low level of central nervous system (CNS) arousal is responsible for the behaviour of patients with ADHD (Satterfield & Cantwell, 1974). This research is supported by studies showing abnormally low Skin Conductance Levels (SCL) in hyperactive children (Satterfield & Dawson, 1971) but increased concentration and reductions in excessive motor activity following the administration of CNS stimulants such as dexamphetamine (Serfontein, 1991). The second model posits a maturational lag as the causal agent and is supported by studies showing ADHD children with EEG frequency distributions that resemble those of younger children (Mann, Lubar, Zimmermann, Millar, & Muenchen, 1992). Also, auditory evoked potential research shows hyperactive children with significantly lower amplitudes and longer latencies than age-matched controls, features that are commonly noted in younger children (Satterfield, Cantwell, Saul, Lesser, & Podosin, 1973; Satterfield, Lesser, Saul &

Cantfield, 1973). Although these models were developed 25 years ago they continue to provide a useful framework for research and treatment.

Researchers and physicians in the 1920's were unaware that hyperactive children had low, rather than high, levels of arousal, hence sedatives and hypnotics were the first drugs to be employed as a treatment for the behavioural disorders displayed by hyperactive and unruly children (Kennedy, 1924). Not surprisingly, these drugs were ineffective. The first successful pharmacological intervention was the administration of Benzedrine to institutionalized male and female children who presented with neurological and behavioural disorders (Bradley, 1938). The subjects showed dramatically increased interest in school material, improved comprehension and accuracy, distinctly subdued emotional responses, reduced motor activity, and a reduction of mood swings.

Since the 1930's, pharmacological agents such as methylphenidate, i.e., Ritalin, and amphetamine have been shown to produce positive changes in patients with ADHD. These adrenergic drugs affect the diffuse neurotransmitter systems and increase activity in the norepinephrine and dopamine systems. In particular, the administration of methylphenidate increases catecholaminergic activity by increasing synaptic release and by blocking pre-synaptic re-uptake. The effect on the patient is an increase in their sense of alertness and improvements in reaction time and vigilance tasks (Oken, Kishama & Salinsky, 1995). From a theoretical

perspective, however, because stimulant drugs act simultaneously on both the motor and arousal systems (Reticular Activating System), it is difficult to determine which system is responsible for the change in behaviour (Shouse & Lubar, 1978). Potahskin and Beckles (1990) suggest that the depletion of neurotransmitters within these systems may be a causal agent of ADHD. In particular, the monoamines, dopamine, norepinephrine and serotonin, play important roles in inhibition and, when depleted, may be responsible for the behavioural difficulties demonstrated by individuals with ADHD (Dustman, LaMarche, Cohn, Shearer & Talone, 1985; Wender, 1971).

Relative efficacy studies of drug interventions have been beset by methodological difficulties including inadequate controls, heterogeneous samples, and insufficient control of medications. In point of fact, long term drug treatment with hyperactive adolescents, without therapy, showed no lasting effects and as many as 33% of the subjects studied reported negative side effects such as weight and appetite loss, motor tics, growth suppression, emotional problems, and mood swings (Wolraich, Lindgren, Stromquist, Milich, Davis & Watson, 1990). To compound these difficulties, there is also a growing body of research on the problems of non-compliance to the medical regimens required for the treatment of disorders such as ADHD (Potahskin & Beckles, 1990).

Within the field of psychology, various 'traditional' approaches such as

cognitive, behavioural, and psychodynamic therapy have attempted to 'cure' or ameliorate the symptoms of ADHD but most of these approaches, even in conjunction with medication, have shown inconsistent results (Brown, et al., 1986). In addition, a variety of non-traditional treatments such as subliminal tapes (Swingle, 1994), herbal supplements, and chiropractic services are also available; none of these treatments, however, have received widespread acceptance. Notwithstanding the limitations and restrictions of drug interventions, traditional pharmaceutical interventions with drugs such as Methylphenidate or Dexedrine continue to be the main treatment modality for children and adults with ADHD.

The Measurement of ADHD

Due to the frequent overlap of ADHD with other psychiatric disorders, many clinicians and researchers rely on different sources of information when attempting to reach a diagnosis. In addition to the DSM-IV criteria, interviews with parents, teachers, and children are conducted (Gittleman, Mannuzza, Shenker & Bonagura, 1985) and rating scales, such as the Conners, Zukow or Werry-Weis-Peters Parent Rating Scale, Teachers Rating Scale of Attention and/or Impulsivity are also utilized (Brown, Borden, Wynne, Schleser & Clingerman, 1986; Potahskin & Beckles, 1990). Although these ratings can be effective with hyperactive children they are less effective with children who have no obvious motoric disturbances (Defrance, Smith, Schweitzer, Ginsberg & Sands, 1996). In the adult

population, DSM-IV criteria are similarly employed and questionnaires such as the Wender Utah Rating Scale or Brown Adult ADHD scales, take the place of teacher and parental ratings. Clinical determinations based on observational data, however, are often more difficult with adults as the hyperactivity of childhood has often abated by late adolescence.

In addition to psychiatric interviews and questionnaires, there are a number of objective measures that may be administered to detect the presence of an attentional disorder. Frequently used tests with children include either complete or selected sub-tests of the Wide Range Achievement Test Revised (WRAT-R), Woodcock Reading Mastery Test Revised (WRMT-R) and Weschler Intelligence Scale for Children Revised (WISC-R) (Janzen, Graap, Stephanson, Marshall & Fitzsimmons, 1995; Lubar, 1991; Tansey, 1984,1985,1991). For an adult assessment, age appropriate tests and selected sub-tests such as the Stroop or sub-tests of the Weschler Adult Intelligence Scale Revised (WAIS-R,) or Woodcock-Johnson (WJ) are employed. It should be noted that none of these tests were developed to measure attention deficits per se. A number of computerized diagnostic tests, such as the T.O.V.A. and the Intermediate Visual and Auditory Continuous Performance Test (I.V.A.) (Sandford & Turner, 1994), have been developed to test for ADHDhy and ADHDpi specifically; however, the diagnosis continues to be based mainly on the observations of parents, teachers, and mental

health professionals (Clarke, Barry, McCarthy & Selikowitz, 1998).

The use of interviews and tests suggests that there are two basic strategies, diagnostic and quantitative, which are used in classifying attention disorders and related disorders of behaviour and learning. Diagnostic classifications are clinically derived and are based on a consensus of practitioners and researchers. A diagnosis is given to an individual based on a set number of criteria which identify whether he or she belongs to a particular classification. For example, the DSM-IV uses a categorical system with 12 symptoms for each of the ADHDco, ADHDhy, and ADHDpi categories: If 6 of the 12 symptoms are identified, the person is classified as belonging to that group. Compared to the quantitative method of diagnosis, this diagnostic method tends to identify more members for each classification (Fletcher, Morris & Francis, 1991).

Quantitative approaches are based on the premise that there is a core set of dimensions present in all individuals. Within this method, empirically driven norms and statistical techniques are used to determine membership. This type of system tends to focus on only a few dimensions and, compared to the diagnostic approach, identifies fewer members for each classification (Fletcher, Morris & Francis, 1991). However, with the exception of tests such as the T.O.V.A. or I.V.A., many of the quantitative approaches, e.g. the WAIS-R, Stroop or WJ, have not been empirically validated with an ADHD population.

Within the research community, Electroencephalographic (EEG), Event Related Potentials (ERP), Computerized Tomography (CT), Magnetic Resonance Imagery (MRI), Single Proton Emission Cerebral Tomography (SPECT), and Positron Emission Tomography (PET) technologies have been employed as research tools to study ADHD. In particular, research with PET has lent support to the hypothesis that different levels of glucose metabolism may be responsible for the symptoms of ADHD (Zametkin et al. 1990, 1993). In this research, adult and teenaged subjects with ADHD were observed to experience decreased glucose metabolism in the left anterior frontal regions and prefrontal areas associated with the control of preparation for motor activity, motor activity, inhibition of inappropriate responses, and attention. In addition, reductions were also observed in the hippocampal, thalamic, right temporal, and posterior/temporal regions. The research using SPECT technology has shown that there may be five sub-types of ADHD characterized by features such as frontal lobe deactivation, temporal lobe dysfunction, homogeneous cortical suppression, increased activity in the anterior medial aspects of the frontal lobes, and hypofrontality at rest but not during cognitively demanding tasks (Amen, Paldi, & Thisted, 1993). Researchers using MRI have corroborated the PET and SPECT research by showing that many of the regions reported in these studies also had structural abnormalities in individuals with ADHDpi and ADHDhy (Castellanos, Giedd, & Eckburg, 1994; Giedd,

Castellanos, & Casey, 1994; Hynd, Semrud-Clikeman, Loryus, Novey & Eliopulos, 1990; Hynd, Semrud-Clikeman, Loryus, Novey, Eliopulos & Lyytinen, 1991). MRI has also detected a smaller rostral body of the corpus callosum in children with ADHD (Baumgardner, Singer, Denckla, Rubin, Abrams, Colli, & Reiss 1996) and a smaller posterior (splenial) corpus callosum region in children with the same condition (Semrud-Clikeman, Filipek & Biederman, 1994). Finally, abnormal regional blood flow has also been reported in hyperactive children, with hypo-perfusion within the right striatal region and hyper-perfusion within the sensory motor cortex (Lou, Henriksen, Bruhn, Borner & Neilson, 1989).

The CT, MRI, SPECT, and PET scans have been used infrequently as they use radiation, lack resolution, are usually unavailable, too expensive, or regarded as inappropriate for use with children (Torello & Duffy, 1985). Both the PET and SPECT are invasive (involving radiation) and have relatively low anatomical resolution. Although CT has now been replaced by the MRI, which produces relatively high resolution images of the structures of the brain, it does not provide a functional image. Recently, functional MRI techniques have become available providing an image of the areas of the brain which are active during different tasks: This technique will undoubtably be employed in future studies of ADHD. A major limitation for more frequent use of all these methods is the extremely high cost of the equipment, typically well over one million dollars per unit.

ERP and EEG research are far less invasive and expensive and have been frequently used to study attention and to test for the presence of attentional problems in children. Using ADHD and learning disabled (LD) children, researchers in the ERP field have reported differences in the form of decreased P300 components and poorly developed late components using 'oddball' auditory paradigms, semantic stimuli, and visual tasks with easy and difficult to distinguish letters (Lubar, Gross, Shively & Mann, 1990; Lubar. Mann, Gross & Shively, 1992) while others have reported differences in the P250, P350, and P500 components (Defrance, Smith, Scheitzer, Ginsberg & Sands, 1996; Holocomb, Ackerman & Dykman, 1985; Loiselle, Stamm, Maitinsky & Whipple, 1980; Robaey, Breton, Dugas & Renault, 1992). In the latter case, the differences were primarily with respect to the amplitude of the P500 component with no differences between groups in onset, peak latency, or topographic focus. In point of fact, Defrance et al. (1996), using tasks that were either passive or required effort, reported that the P500 alone was an effective discriminator between the control and ADHD subjects and the ADHDhy and ADHDpi subjects. A complete review of the ERP literature is beyond the scope of this paper; however, the studies reviewed above suggest that this technology has provided a powerful diagnostic tool.

Over the past twenty years, technological advances in EEG equipment and

software and the availability of low cost, high speed personal computers have led to the development of quantified EEG (qEEG), a technology that can be used to treat as well as identify brain dysfunction. Researchers have used this approach as a within subjects measure of pre- and post-treatment changes and as a measure to discriminate between ADHD children and normal controls. Studies comparing the brainwave activity of children with ADHD and normal controls have reported significant qEEG differences between these two groups. Compared to control subjects, children and adolescents with ADHD had higher levels of delta and theta wave activity but lower levels of beta wave activity (Janzen, Graap, Stephanson, Marshall & Fitzsimmons, 1995; Lubar, 1985, 1991, 1999; Lubar, Bianchini, Calhoun, Lambert, Brody & Shabsin, 1985; Mann, Lubar, Zimmermann, Miller & Muenhon, 1991; Tansey, 1984, 1985, 1991) and either increased or decreased levels of alpha wave activity (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy & Selikowitz, 1998; Defrance, Smith, Scheitzer, Ginsberg & Sands, 1996; Kuperman, Johnson, Arndt, Lindgren & Wolraich, 1996; Lazarro, Gordon, Whitmont, Plahn, Li, Clarke, Dosen & Meares, 1998). The increased levels of theta and delta and lower levels of beta activity in the ADHD subjects were most apparent in the frontal area but were also detected in comparisons of inter-hemispheric activity, i.e., more activity in the right than left hemisphere and at various individual electrode sites.

The importance of the frontal and pre-frontal areas to attentional processes is supported by research with neurological patients. For example, patients who have experienced damage to the frontal and pre-frontal areas of the brain showed deficits in tasks of concentration, sustained behavioural output, and the ability to inhibit immediate, but inappropriate, response tendencies. Meanwhile, these same patients showed no deficits in perception, construction, language, and directed spatial attention suggesting that the physical damage to the neural networks was responsible for the attentional deficits (Meslaum, 1986; Stuss, 1986). As Meslaum (1986) noted, however, these behavioural deficits could also have been caused by damage to neurotransmitter release or re-uptake processes.

To date, researchers have not examined an adult population of individuals who suffer from ADHDhy and ADHDpi through the use of qEEG analysis. The lack of EEG research with ADHD adults creates a number of theoretical and practical questions for researchers and clinicians. For example, as a normal child ages, slow wave activity is gradually replaced by faster wave activity (Gasser, Verleger, Bacher, & Sroka, 1988; Matsuura, Yamamoto, Fukuzawa, Okubo, Uesugi, Kojima & Shimazono, 1985). Researchers have not examined whether the EEG changes over time with ADHD subjects. In adults with ADHD, the presence or absence of differences, and the areas and frequencies in which these differences occur, in comparison to children with ADHD and a control group of normal adult

subjects, could assist in clarifying the possible role of a maturational lag or low arousal in this disorder.

There are an ever-increasing number of private practitioners who treat ADHD children and adolescents with EEG biofeedback (Lubar, 1985; Tansey, 1984, 1985, 1991). Published studies by these practitioners have shown that children with ADHD can learn how to regulate the abnormal theta and beta wave activity associated with this disorder through the use of auditory and visual EEG feedback (Lubar, 1985; Lubar & Lubar, 1984; Lubar, Swartwood, Swartwood & O'Donnell, 1995; Tansey, 1991). Furthermore, research has demonstrated that those children who were able to alter their brainwave activity, to more closely approximate that of a normal child, continued to experience profound and sustained changes in emotional and intellectual functioning at ten year follow-ups (Lubar, 1995; Tansey, 1993; Tansey & Bruner, 1983). For adults with ADHD it is unclear as to whether their EEG activity differs from that of normal adults. Importantly, a significant difference could indicate that the ADHD adult was capable of achieving the same behavioural improvements noted in ADHD children and that the same underlying physiological mechanisms responsible for childhood ADHD were present in the adult. To date, none of these issues and questions has been addressed in the literature.

The EEG

The use of the EEG began in 1929 when Hans Berger noted that there were two types of waveforms; alpha and beta. He observed that in a relaxed state, high amplitude, relatively slow and synchronous trains of waves (alpha) were produced, however, when subjects exerted mental effort, low amplitude, relatively fast and random activity (beta) was produced. Berger believed that the suppression of alpha and increased beta, was due to the subject attending to a stimulus. He noted that when the subject removed interest from the stimulus, the slower waveform returned.

Since Berger's first measurement of EEG activity, researchers have learned that the EEG is formed by the integration of electrical events in large numbers of brain cells and that this complex pattern of varying voltages and frequencies is related to cellular responses to stimuli both in the external environment and within the individual (Torello & Duffy, 1985). A number of researchers have reported that intra-cortical loops and thalamic 'pacemakers' are partially responsible for some of the activity displayed by the EEG (Lubar, 1997; Nunez, 1995; Thatcher, Krause, & Hrybyk, 1986).

The EEG has been used to examine the brain's functioning from both theoretical and clinical perspectives. Theoretically, the EEG has been used to better understand the activity and relationship between various neurological

pathways, neurotransmitters, and states of mind. Clinically, the EEG has been used to determine abnormalities in brain functioning, as a feedback device, and to provide supportive evidence to other diagnostic indicators of brain dysfunction (Chabot & Serfontein, 1996; Duffy, 1993; Defrance et al, 1996; Lubar, 1997; Prichep & John, 1986; Tansey, 1985; Tansey, Tansey & Tachiki, 1994). Compared to other brain imaging technologies, the EEG is easier to perform, less expensive, and more appropriate for research and clinical applications involving infants, children, and adolescents (Duffy, 1993; Kuperman et al, 1996).

The popularization of the EEG as a biofeedback device to assist individuals to operantly control changes in brainwave activity is a relatively new development. Although used experimentally in the 1960's and early 1970's, it was not until the availability of affordable personal computers in the 1980's that the use of the EEG by private practice clinicians became more prevalent. More recently, EEG biofeedback, more commonly called "neurofeedback", has been used by clinicians to treat clinical disorders such as epileptic seizures, post-traumatic stress disorder, alcoholism, ocular instability, and ADHD (Peniston & Kulkosky, 1989; Peniston & Kulkosky, 1991; Shouse & Lubar, 1978; Sterman, 1986; Tansey & Brunei, 1983; Shouse & Lubar, 1978). In addition to the wide range of applications, the acceptance of neurofeedback training appears to be due, in part, to the benign nature of the treatment; alternatives such as surgery, drug interventions, or

cognitive training are perceived as too invasive, dangerous, or ineffective (Kuperman et al, 1996).

Neurofeedback

Traditional biofeedback has been defined as the "technique of using equipment (usually electronic) to reveal to human beings some of their internal physiological events, normal and abnormal, in the form of auditory and visual signals in order to teach them to manipulate these otherwise involuntary or unfelt events by manipulating the displayed signals" (Basmagian, 1979). The EEG integrates the output of large numbers of individual neurons and extracts information about the activation of whole populations of cortical units. In this sense, the EEG may characterize the activity of the whole brain at a level appropriate to the study and modification of behaviour (Beatty, 1977). The rationale behind using neurofeedback is that various disorders may, in part, be caused by a disruption in the regulation of the neural processes which underlie electrical brain activity (Rochstroh et al., 1984).

The basis for the clinical application of neurofeedback with ADHD patients is the assumption that these individuals exhibit either excessive slow wave (theta) activity, reduced levels of fast wave (beta) activity or a significant difference in the fast to slow wave ratio. During training the client or subject learns techniques for directly impacting the brain state by modifying the specific EEG frequencies that

are characteristic of the disability (Tansey, 1991): This is a technique that has been validated by clinical studies performed by practitioners using children and adolescents (Lubar et al., 1985,1991; Tansey & Bruner, 1983, Tansey, 1984, 1985, 1991, 1993).

The operant conditioning programme used with neurofeedback focuses on the frequency, location, amplitude, and duration of the brain's electrical activity. Using either visual or auditory feedback, the EEG is first used to demonstrate the relationship between thoughts and feelings with the different electrical aspects of the EEG (Beatty, 1977; Rochstroh et al., 1984). Later, the EEG feedback is used as a means, through operant techniques, to increase or decrease particular brainwave activities. A crucial element of the neurofeedback program is the subjects' or clients' ability to discriminate, i.e., detect and quantify the different waveforms. In addition to discrimination, the other essential elements are the reinforcement schedules and the generalization or transfer of the response in question to non-feedback situations (Rochstroh et al., 1984). Training often requires from three months to a year on a once or twice a week basis (Lubar et al, 1995; Tansey, 1991).

As it is possible to employ a wide variety of scalp locations with neurotherapy, researchers and clinicians use a screening technique during which the EEG activity of the patient or subject is first observed at all 19 electrode sites.

The data from this screening are used to determine the area with the greatest theta, beta, or theta/beta ratio at which to monitor and treat the patient or subject. Due to the importance of the pre-frontal and frontal areas, however, clinicians often obtain the EEG output from one site, usually at or around the Sensory Motor Area (SMA) between the frontal and central areas (Tansey, 1984, 1985, 1991, 1993; Tansey & Bruner, 1983). The SMA is part of the pre-motor cortex and is a central, bilateral region that provides a crucial link for a wide range of perceptual motor functions and is used extensively in the treatment of ADHD children (Lubar et al, 1995; Lubar, Swartwood, Swartwood, & Timmermann, 1995; Tansey & Bruner, 1983, Tansey, 1984, 1985, 1991, 1993; Tansey, Tansey, & Tachiki, 1993). Of note, studies that used the SMA for neurofeedback have reported that training often resulted in generalized changes to other cortical sites. For example, training to reduce theta band activity at the central (Cz) electrode site resulted in changes at the left occipital site (O1) site. (Lubar et al, 1995; Lubar, Swartwood, Swartwood, & Timmermann, 1995).

The Development of Neurofeedback as Treatment for ADHD

One of the first reports of EEG abnormalities in children displaying ADHD symptoms such as hyperactivity, impulsivity, and academic difficulties, was that of Jasper, Solomon, and Bradley (1938). These researchers noted that, compared to controls, over 50% of the disordered group had abnormal EEGs with

predominantly slow wave activity from a variety of different sites. Similar results were reported by Knott, Platt, Ashley and Gottlieb (1953) who observed that the EEG abnormalities in behavior disordered children were primarily epileptogenic and generalized slowing. Furthermore, adults who demonstrated high risk and hyperactive behaviours, as well as children who exhibited unruly and hyperactive behaviours, were also noted to produce excessive slow wave activity (Cohn & Nardini, 1958).

The early reports of excessive slow wave activity in hyperactive adults and behaviour disordered children were unexpected as researchers had predicted greater than normal activity in these individuals (Cohn & Nardini, 1958; Jasper, Solomon, & Bradley, 1938; Knott, Platt, Ashley & Gottlieb, 1953). In point of fact, hyperactive, behaviourally disordered children were originally believed to be over-aroused (Satterfield & Dawson, 1971; Satterfield, Lesser, Saul & Cantwell, 1973). However, when observing some of the standard indices of arousal such as Galvanic Skin Response (GSR), researchers noted lower than usual levels of activity for the behaviourly disordered subjects and that they appeared to interact with their environment more intensely than control subjects (Satterfield et al., 1973). These children would move visually interesting objects closer to their eyes or move noise-making toys closer to their ears, suggesting the need for greater than normal sensory stimulation. Based on these observations Satterfield, Lesser,

Saul and Cantwell (1973) deduced that hyperkinetic children were experiencing decreased rather than increased sensory arousal. The causal basis of this decreased arousal was initially conceptualized to be a sensory 'filter' which was interfering with the impact of the visual, auditory, somaesthetic, vestibular and gustatory input, a concept which came to be known as the "low arousal" hypothesis.

Corroborating evidence of the low arousal hypothesis came from studies examining the effects of medications. Researchers noted that children who responded most favourably to stimulant drugs presented with signs of being physiologically under-aroused. Furthermore, children who demonstrated the most severe pre-treatment behavioural symptoms were those who were most likely to show dramatic improvements following medication (Shouse & Lubar, 1978). Following the administration of Ritalin, reduced CNS activation, as determined by EEG and GSR indices, was enhanced. Conversely, children who initially presented with heightened levels of EEG and GSR activity showed the least severe pre-treatment symptoms, the fewest neurological deficits, and responded less well or unfavourably to drugs.

The evidence from drug studies, as well as the supporting data from other research (Satterfield et al, 1973) suggested to Shouse and Lubar (1978) the presence of selective neurological and behavioural deficits in children with attentional problems. However, although Shouse and Lubar (1979) were the first

to recognize the potential for developing a neurofeedback treatment regimen for children with attentional problems, their initial research studies focused on seizure disorder patients (Tansey, 1991). These patients, prior to EEG biofeedback training, had exhibited decreased levels of EEG activity in the sensory motor area in the 12-14 Hz band. This suggested that a relationship existed between sensory motor rhythm (SMR) output and the presence of seizures. Once patients had completed neurofeedback training to increase SMR activity they reported fewer seizures and, surprisingly, improved focus and concentration. The evidence of a relationship between physical activity level and SMR activity prompted Shouse and Lubar (1979) to postulate that EEG biofeedback to increase SMR activity in the SMA would be a viable method of treatment for children who were demonstrating hyperactive ADHD behaviours. Since the Shouse and Lubar (1979) study, a number of studies have confirmed the presence of EEG abnormalities in ADHD children and biofeedback has become a popular treatment within the private sector (Lubar et al, 1995; Lubar, Swartwood, Swartwood, & Timmermann, 1995; Tansey & Brunei, 1983, Tansey, 1984, 1985, 1991, 1993; Tansey, Tansey & Tachiki, 1993).

Review of Neurofeedback with Children and Adolescents

The use of neurofeedback training by private practitioners has provided useful information regarding the EEG's of children with ADHD. The treatment

employed by these practitioners is typically neurofeedback training combined with cognitive therapy and usually involves auditory and/or visual feedback of theta or beta wave amplitude; the client is instructed and encouraged to inhibit theta wave production, increase beta and/or SMR wave production or both decrease theta while increasing beta/SMR. For example, using neurofeedback with operant conditioning techniques, Lubar & Lubar (1984), in a single case study, reported that an ADHD client was able to increase SMR activity from between 16-60%, successfully block theta wave activity, and increase beta wave activity. These changes persisted at a 12 month follow-up and were accompanied by increases in the subject's WISC-R verbal, performance, and full scale scores and improved pre- and post-T.O.V.A. indices. (Lubar, Odle, Swartwood & O'Donnell, 1995).

As a result of neurofeedback training, clients and research subjects have demonstrated the ability to reduce theta wave and EMG activity, increase test scores, and to decrease subjectively measured levels of overactivity and distractibility (Shouse & Lubar, 1979). Furthermore, increases in test scores such as the WISC-R are correlated with learned increases in SMR activity and decreased levels of theta wave activity (Tansey, 1984,1985) and continue even at 10 year-follow-ups (Tansey, 1993). These findings indicate a consistent ability of individuals with ADHD to decrease theta wave output, increase SMR output and

concurrently increase scores on tests such as the WISC-R (Tansey, 1991); changes that are accompanied by reports of improved behaviours from parents and teachers (Lubar & Lubar, 1984; Lubar, Odle, Swartwood & O'Donnell, 1995; Tansey, 1984, 1985, 1993; Tansey & Bruner, 1983)

There are, however, methodological concerns regarding these results. These studies have often used small subject pools ranging from 1 to 8 subjects (Lubar & Lubar, 1984; Tansey, 1984; 1985; 1993; Tansey & Bruner, 1983). Some of the research has shown mixed results, with as many as 40% of the subjects being unable to achieve meaningful changes in brainwave activity (Lubar, Odle, Swartwood & O'Donnell, 1995). Furthermore, contradictory evidence from research utilizing conventional EEG techniques, has shown EEG abnormalities for between 30% and 60% of hyperactive children (Small, 1993). In one study only 1 child in 11 showed EEG abnormalities (Phillips, Drake, Hitter, Andrew & Boner, 1993). There is also the possibility that extended periods of treatment (40 to 60 sessions) (Lubar, Odle, Swartwood & O'Donnell, 1995; Shouse & Lubar, 1979) may have been responsible for the observed changes, i.e., the subjects simply changed as a function of aging. The nature of the fee-for-service paradigm and the various cohorts, such as the socio-economic status of those who are able to afford these services, also present obstacles to a clear interpretation of these

results. In sum, these factors present difficulties in accurately determining the relative efficacy of the neurofeedback component of the treatment regimen and suggest that perhaps undetectable physiological dysfunctions or social factors may play a role not only in the emergence of this disorder but also in its treatment.

The Identification of ADHD During Resting Conditions

Many researchers use resting EEG activity recorded during eyes closed or eyes open conditions as a method of differentiating between groups of ADHDpi, ADHDhy, and ADHDco children and adolescents. The eyes closed condition is often used due to its simplicity and the relative uniformity of testing conditions (Thatcher, 1999). Using an eyes closed condition, Chabot and Serfontein (1996) reported that ADHDpi and ADHDhy subjects displayed increased absolute and relative theta, especially in the frontal area, slight elevations in relative alpha and diffuse decreases in alpha and beta mean frequencies. These researchers also reported two distinct neurophysiological subtypes in the ADHDco group. The 'first' type showed evidence of overactive theta and/or alpha generators with normal mean levels of alpha while the 'second' type showed excess theta and alpha but decreased mean levels of alpha. Chabot and Serfontein (1996) reported classification rates of 88%, using a stepwise discriminant function analysis.

Clarke et al. (1998), using regional comparisons based on an eyes closed

condition with ADHDpi and ADHDco children, reported greater levels of theta wave activity and deficiencies in alpha and beta wave activity in comparison with controls. These researchers reported theta band differences over all regions but significantly more midline frontal theta activity than control subjects. Both groups also showed increased levels of relative delta and decreased levels of absolute beta in the posterior regions. There was decreased relative alpha at all sites but the greatest differences occurred in the posterior sites. In addition, Clarke et al. (1998) reported differences between the ADHDco and ADHDpi groups with the ADHDco group producing more relative delta, theta, and absolute theta but less relative alpha and beta. Defrance et al. (1996), using both eyes open and eyes closed conditions with ADHDpi and ADHDhy children, reported greater relative theta across all scalp locations and reduced levels of alpha for the ADHD groups. There were no statistically significant differences in the beta band. These researchers, using only ERPs in their discriminant function analyses, achieved correct classifications of over 90%.

Lazzaro et al. (1998), using regional analysis in an eyes open paradigm with ADHDco subjects, reported increased absolute theta in the anterior region and decreased relative beta in the posterior region but increased relative alpha1 (8-9 Hz) in the right hemisphere. Kuperman et al. (1996), also using an eyes open

condition to examine ADHDco and ADHDhy groups, but with the combined activity of all of the electrode sites in each of the left and right hemispheres, reported higher relative levels of delta band and lower levels of beta band activity in the control subjects. These researchers also reported significant asymmetry between the left and right hemispheres of the ADHDco group with decreased left hemisphere delta and increased left hemisphere beta.

In summary, the comparison of children with ADHD to normal controls in resting eyes closed or eyes open conditions has provided a valuable database with which to compare adults with ADHD. Differences between adults and children may provide important links to understanding the maturational lag and under arousal models of this disorder.

The Identification of ADHD During on-task EEG Conditions

A problem with the usual eyes open-closed procedure is that the researcher has no control over the mental state of the subject. For example, one subject group may be engaged in thought or other cortically arousing activity while another group may be attempting to relax and fall asleep. Differences in EEG activity are thus difficult to interpret in such passive conditions. For this reason, researchers have also measured EEG activity during the performance of a task. In these paradigms, EEG data are collected while ADHD and control subjects

perform tasks that vary cognitive load. Lubar (1985) reported that during the performance of a reading task, the EEG of a normal child showed a blockage in alpha wave production and an increase in beta wave production. In ADHD individuals, normal blocking of alpha wave activity was at times observed. However, they also showed increased theta rather than beta activity and a greater tendency for alpha to persist as theta appeared (Lubar, 1985).

Increases in theta activity in the ADHD group when performing mathematical calculations, solving puzzles, and reading have also been reported (Lubar, 1991). In this study, the frontal/temporal areas were reported to be the most reliable locations at which to base a discriminate analysis. Using these locations, 98% of the ADHD and normal controls were correctly identified.

Janzen et al. (1995), measured the amplitude of different EEG frequencies in children and adolescents with ADHDpi. They noted that theta amplitude was 5-8 μV higher than controls during reading, drawing, and baseline tasks across widespread areas of the scalp. There were, however, no differences in the SMR (12-16 Hz) or beta1 (16-20 Hz) bands at any of the sites. The theta/beta ratio was statistically significantly different at the P3 and P4 sites and the authors reported that some of the central sites approached statistical significance (Janzen, et al., 1995). They concluded that ADHD and normal individuals have similar beta wave

profiles, suggesting that theta wave inhibition or theta/beta training could be useful in neurofeedback but that focusing on the control of beta wave activity alone would likely be ineffective.

Mann et al. (1990), using ADHDpi children, reported that there were increases in the percentage of theta band activity at frontal and central sites and decreases in beta activity in frontal and temporal sites during the performance of reading and drawing tasks. However, unlike the Janzen et al. (1995) study, there was a significant decrease in beta wave activity at frontal/temporal locations for the ADHD children. These researchers reported classification rates of 74% and 80% for control and ADHDpi children respectively.

The role of beta band activity in ADHD is not yet clear. Lubar et al. (1995) have suggested that this activity becomes more important during adulthood; a hypothesis that has not yet been empirically validated. Janzen et al. (1995), Lubar (Lubar et al., 1985; 1990) and Mann (Mann et al., 1990), however, indicate that there is a significant widespread increase of theta wave activity for ADHD subjects during on-task performance, particularly in the frontal areas.

Rationale

Practitioners of neurofeedback have assumed that ADHD, if untreated, will continue into adulthood. With this in mind, they often apply the same or similar

treatment techniques used with children to adults without a general base of knowledge about the EEG abnormalities they are treating. If shown to be a plausible treatment for adults, neurofeedback training could provide much needed assistance for ADHD adults who have not benefitted from drug based, psychological, or alternative treatments. Furthermore, due to the vague diagnostic criteria utilized by the DSM-IV and the lack of overt behavioural abnormalities that can assist in a diagnosis, there is a need for more quantitative measures of this disorder. Research using an adult population of ADHD subjects could provide useful basic EEG knowledge about this group to assist in classification and treatment. The review of the literature has indicated that there are differences in the EEG of ADHD children and normal controls, however, it is not known whether these differences will continue into adulthood.

There is some evidence that the abnormalities associated with ADHD children can also be observed in adults. Glucose metabolism, cerebral blood flow, and structural MRI studies show that there are differences in the physical structure and cerebral activity in children and adults with ADHDhy or ADHDpi. Furthermore, drug effectiveness studies have shown that adults with ADHD react in much the same fashion as children with ADHD, indicating that these drugs affect the same neurological systems in both children and adults.

Several areas of concern arise from a lack of EEG research with adults who have ADHD. For example, it is unknown whether the differences in theta, alpha, delta, and beta wave activity, observed in ADHDhy and ADHDpi children, is affected by age-related changes in EEG activity. It is also unclear whether the maladaptive behaviours of ADHD adults are related to or a result of the continuation of the EEG abnormalities noted in childhood or whether the EEG normalizes and the destructive intra- and inter-personal coping styles learned in childhood are carried into adulthood.

Goals of the Research

The aim of this study was to determine whether the EEG abnormalities observed in children with ADHDhy and ADHDpi continue into adulthood, to develop discriminate EEG criteria for ADHDhy and ADHDpi adults and to provide descriptive information concerning any EEG differences that may exist between ADHDhy and ADHDpi adults. In addition, to assist in providing more general information about individuals with attention deficits, the ADHDpi the ADHDhy groups were combined to form a 'CLINICAL' group. Finally, an important goal of this study was to determine the appropriateness of using EEG neurofeedback equipment for the treatment of adults with ADHD and to aid the clinician in the selection of

the most useful sites and bands in which to conduct neurotherapy on ADHDhy and ADHDpi adults.

In order to achieve the above-mentioned goals the present research used baseline measures of EEG activity obtained during eyes open-closed conditions, as well as EEG measures of on-task activity. The on task measures included the performance of the T.O.V.A., reading, listening, mental rotation, and selective attention tasks.

The eyes open and eyes closed conditions were selected because they formed the basis of many previous clinical studies using children and adolescents with ADHD (Chabot & Serfontein, 1996; Clarke et al., 1998; Defrance et al., 1996; Kuperman et al, 1996; Lazzaro et al., 1998). The data from the present research, therefore, can be compared to these earlier studies. Similarly, the reading and listening tasks were also selected as these were included in previous qEEG studies involving on-task measures of EEG activity (Janzen et al., 1995; Lubar, 1985; Lubar et al., 1990; Mann et al., 1991). The mental rotation task (Metzler & Shepard, 1974) was selected as a predominantly right hemisphere task that was similar to the drawing tasks used in other qEEG studies of ADHD (Janzen et al., 1995; Lubar, 1985; Lubar et al., 1990; Mann et al. 1991) but without the eye and body movement artifacts commonly associated with the performance of these

tasks.

The Tests of Variability of Attention (T.O.V.A.) was employed as it provides valuable interpretative information which can be used to compare the attentional status of the subject to populations of normal, ADHDhy, and ADHDpi adults. The results of the T.O.V.A. were used to discriminate the subject's group membership, ADHDhy, ADHDpi, or control, as well as serving as one of the on-task variables. The inclusion of the T.O.V.A. provided an opportunity to observe and analyze continuous on-task EEG activity and to observe EEG activity during a task that was specifically designed to test for ADHDhy and ADHDpi.

The advantage of using the selective attention task was that it provided a measure of distractibility, a factor that is rarely manipulated in most studies of ADHD. This particular paradigm was chosen in order to observe EEG activity during a task that was designed to deliberately interfere with attention without the eye movement artifacts that are often present during a visual search (Eriksen & Eriksen, 1974). Finally, a computerized reading task, using a stationary window, was also employed in order to observe EEG activity during reading. The advantage of using the stationary window is to drastically reduce the eye movement artifact commonly associated with reading text from a written page. This should allow a clearer measure of EEG activity during reading.

In addition to observing continuous EEG activity during the T.O.V.A. it was also of interest to observe changes or differences in the EEG activity of ADHDhy, ADHDpi, and normal subjects over an extended period of time (i.e. up to 80 or 90 minutes). For this reason, the tasks were presented sequentially during one session. The age range of the subjects selected for use in this study (20-50 yrs) was wider than that typically encountered in EEG research. There are risks associated with the use of this range of ages (Princhep & John, 1988); however, one of the goals of this study was to test whether ADHD subjects from different age groups shared similar enough EEG activity to be correctly identified in a clinical setting. In point of fact, this study is unique as it employs a wide age range of adult subjects, both male and female subjects, ADHDpi and ADHDhy groups, and relatively large sample sizes.

CHAPTER 2

METHODS

Subjects

Subject Recruitment

Subjects with ADHD were recruited from the University of Ottawa, Carleton University, Algonquin College, a local support group for adults with ADHD, and through referrals from learning specialists, psychiatrists, therapists, and physicians who practice in the Ottawa area and who specialize in the diagnosis and treatment of ADHD. The counsellors, therapists, and physicians responsible for recruiting the ADHD subjects were given a three page document explaining the rationale, purpose, and goals of the research (Appendix A) and a copy of the document to be used for recruiting prospective subjects (Appendix B). Subjects using medication to treat their ADHD were informed that they would, under the supervision of their physician, have to discontinue its use 72 hours prior to testing. In addition, subjects were also informed that they were not to consume caffeine, stimulants, or tobacco on the day of testing. Only clinical subjects who had received a medical diagnosis of ADHDpi or ADHDhy were accepted as participants in the study.

The control group was recruited from local corporations, businesses,

government, and the student body of the University of Ottawa and Carleton University through the use of posters and, in the case of the University of Ottawa, through classroom presentations to first and second year psychology students. Subjects were informed that they would have to refrain from caffeine, stimulant, or tobacco consumption on the day of testing. Participation was on a strictly volunteer basis. For both groups, only individuals with normal or corrected-to-normal vision and hearing were accepted.

Subjects

A total of 149 individuals volunteered to participate in the research. From the initial screening, six of the subjects were excluded because their data were contaminated by heartbeat artifact (n=4), excessive EMG (n=1), or stimulants consumed just prior to testing (n=1); in addition, two of the subjects withdrew due to discomfort from the EEG cap. Of the remaining 141 subjects, only individuals who had already been diagnosed by a physician as having ADHDhy or ADHDpi and also received a positive clinical diagnosis from the TOVA were accepted as clinical subjects. Similarly, only individuals who had presented as non-clinical subjects and also received a negative clinical diagnosis from the TOVA were selected as control subjects. Specifically, of the 67 control subjects, the TOVA detected 14 control subjects with ADHDhy and 10 controls

with ADHDpi. From the 84 ADHD subjects, the TOVA detected 10 subjects who would be classified as normal adults. This screening resulted in the loss of an additional 34 subjects. Finally, only those who were right handed, not taking psychotropic medication, had no head injury with cerebral symptoms, and were not suffering from a psychiatric illness were included in the analysis. Based on these criteria, the data from 74 subjects were selected; 32 control subjects, 25 ADHDhy subjects, and 17 ADHDpi subjects. Determination of group membership, control, ADHDhy, or ADHDpi, was determined solely by the results of the TOVA. There was, however, a statistically significant correlation between the TOVA diagnosis and the previously determined clinical diagnosis of either ADHDpi or ADHDhy $r(74) = .78$, $p < .01$). The ADHDpi and ADHDhy groups are treated as two distinct subgroups in the DSM-IV (1994); however, in order to describe more general information about the characteristics of attentional deficits, both these groups were merged to create an overall 'CLINICAL' group.

There were 21 males and 11 females in the control group, 15 males and 10 females in the ADHDhy group, and 11 males and 6 females in the ADHDpi group. The CLINICAL group was composed of 26 males and 16 females. The ratio of males to females was 1.9:1 for the control group, 1.5:1 for the ADHDhy group, 1.8:1 for the ADHDpi group, and 1.6:1 for the CLINICAL group. There were no

statistically significant differences in age (Table 1) between the control group and the ADHDhy, ADHDpi, or CLINICAL groups ($p > .40$). Similarly, there were no statistically significant differences with respect to the number of years of education (Table 1) between the control group and the ADHDhy, ADHDpi, or the CLINICAL group ($p > .30$).

Table 1

Summary of Demographics by Group

	Control	Clinical	$ADHD_{HY}$	ADD_{PI}
Males	21	26	15	11
Females	11	16	10	6
Average age & (SD)	31.9 (8.7)	30.1 (9.5)	30.3 (9.0)	29.8 (10.2)
Educated Yrs. & (SD)	15.6 (1.9)	15.2 (1.7)	15.1 (1.8)	15.4 (1.5)
Employed	17	12	6	6
Student	11	23	16	7
Unemployed	0	2	1	1
Family members*	3	10	5	5
1st Diagnosed & (SD)	N/A	4.3 (4.9)	4.9 (5.5)	3.5 (3.9)
Drug treatment**	N/A	13	7	6

* number of subjects who report family members diagnosed with ADHD.

** currently using medication prescribed for ADHD

The were notable similarities and differences in the demographics of the

groups. Almost twice as many subjects in the ADHDhy group were attending university on a full time basis compared to either the ADHDpi or control groups and the only two unemployed subjects were each members of the ADHDpi and ADHDhy groups. Seventeen of the 25 ADHDhy subjects, 14 of the 17 ADHDpi subjects, and 16 of the 32 control subjects consumed caffeine. As illustrated in Table 1, the groups were similar on the other demographic variables.

The majority of EEG studies of ADHD have examined individuals younger than 19 years, and focussed on children between the ages of 10 and 14 years (Lubar, 1992). Aging, however, is correlated with changes in the pattern of EEG activity throughout ones life. These changes are particularly pronounced before age 20, leaving some question as to the differences in brainwave activity between younger and older subjects in the ADHDpi or ADHDhy groups. In order to confirm that there was an approximately even number of older and younger subjects in each group, participants were classified as being either older or younger than 30 years and a chi square analysis performed. The results showed that there were no statistically significant differences in the number of older and younger subjects in each group (χ^2 (2) = 1.09, $p > .50$).

A similar concern to that of age was the distribution of gender within each of the groups. Previous studies of the EEG activity in ADHD groups have rarely

examined the effects of gender on the pattern of EEG activity (Serafontaine et al, 1995). In point of fact, most of the on-task research has examined only male subjects, leaving some question as to the composition of EEG activity in females with ADHDpi or ADHDhy. The control, ADHDhy, and ADHDpi groups in this study had approximately the same proportion of females; however, gender differences between each group may have influenced the overall results. A chi square analysis was performed in order to confirm that there was an approximately equal number of males and females in each group. The results showed that there were no statistically significant differences between the observed and expected frequencies of males and females in each group (χ^2 (2) = .074, $p > .95$).

Finally, there may have been differences in the distribution of younger and older, male and female subjects within each group which could have biased the results, e.g., all the ADHDhy males may have been younger than 30 years of age. In order to confirm that there was an approximately equal number of younger and older males and females in each group a chi square analysis was performed. The results showed that there were no statistically significant differences between the makeup of the groups (χ^2 (5) = 2.33, $p > .90$).

Procedure

All testing took place in a laboratory at the University of Ottawa. Subjects

were familiarized with the laboratory setting, experimental procedures explained, a background information questionnaire completed, and consent forms signed. Electrodes were then placed on the subject's scalp. The electrodes were attached to an elastic cap (Electro-Cap International) which was fitted to the subjects head. This required between 45 and 60 minutes and was performed in a room adjoining the testing room. Following the fitting, subjects were seated in a comfortable chair in a 3 x 4 metre, sound and light attenuated room. Once in the testing room, Electro-occulographic (EOG) electrodes were placed on the outer canthus and above the supra-orbital ridge of the left eye, and an EMG electrode placed on the masseter muscle of the right side of the face. The EOG and EMG channels served to identify eye movement and muscle artifact. Because the T.O.V.A. norms were developed with subjects tested before 13:00, all EEG recordings were held between 08:00 and no later than 12:00. The hour between 12:00 and 13:00 allowed for the eyes open, eyes closed, and T.O.V.A. to be completed before 13:00.

Instruments

EEG

The EEG measurements were recorded with a Lexicor Neurosearch-24 system (Lexicor Medical Technology Inc.). The NRS-24C is a 23 channel EEG

analyzer with 19 EEG channels, two EOG channels, two EMG channels, and a noise level of less than 2.5μV. pp at 32 Hz. The raw EEG was sampled in 2 sec epochs by using a 12 bit analogue-to-digital converter. The sampling rate was set to 128 Hz. The lowpass filter was set to 32 Hz and the highpass filter set to 2 Hz.

The 2 Hz high pass filter provided a means of removing most of the low frequency delta activity which included contamination by slow (0.5-2 Hz) eye movements and blinking. A drawback to using the highpass filter is the difficulty in comparing relative data results with previous studies that had the highpass turned off.

The EEG measures were recorded from 19 electrodes placed according to the International 10-20 System using the Electro-Cap system (Electro-Cap International). Electrodes were placed at the F7, T3, T5, Fp1, F3, C3, P3, O1, Fz, Cz, Pz, Fp2, F4, C4, P4, O2, F8, T4, and T6 sites. Electrodes were filled with a gel conductive agent (Electro-Cap International). The EMG and EOG electrodes were 9 mm, gold plated tin electrodes which were attached with conductive EEG paste and held in place with surgical tape.

All EEG sites were referenced to linked ears. Impedance at each of the electrodes was lowered to less than 5000 ohms (measured before and after recording). The data were analyzed using the following frequency bands; high

delta 2.0-4.0 Hz, theta 4.0-8.0 Hz, alpha 8.0-12.0 Hz, SMR 12.0-16.0 Hz, beta1 16-20 Hz, beta2 20-24 Hz, beta3 24-32 Hz. The data output from the NRS-24D was stored to a hard disk of a personal computer.

Subject Tasks

During each task an attempt was made to reduce artifacts due to eye movement, muscle tension, and body motion. Subjects were asked to refrain from movement and to keep eye movements and blinking to a minimum. Between tasks, one or two minute breaks were given while the data were stored and the next task prepared. The subjects completed the following 10 tasks:

Eyes Open (1st task, 0-3 min)

The subject was instructed to remain quiet and still while they observed a blank 8.5" x 11" inch sheet of paper on which a 2 cm wide black asterisk (*) had been printed. The paper was attached to the front of a computer monitor at eye level, at a distance of 1 metre. After 3 minutes the EEG recording was stopped and the subject was asked about their thoughts while staring at the asterisk. These thoughts were recorded on paper and later coded and analyzed.

Eyes Closed (2nd task, 4-6 min)

The subject was instructed to remain quiet and still as they closed their eyes and relaxed. The EEG recording began immediately before they closed their eyes.

After 3 minutes the recording was stopped and the subject was asked about the types of thoughts they were having while sitting with their eyes closed. These thoughts were recorded on paper and later coded and analyzed.

At the end of both the eyes open and eyes closed conditions, subjects were asked about the types of thoughts they were having while they were in these conditions. The subjects' thoughts were later coded into the following 5 general categories:

1. Relaxed- Feeling relaxed, thinking about nothing, meditating, dreaming....
2. Noises- Being aware of, and listening to, room noises...
3. Worry- Worrying about the experiment, eye movements, breathing, etc...
4. Concentrate- Fully concentrating on only the asterisk, "it looked like a....."
5. Outside- Thinking about outside, work, car, groceries, school, children etc...

T.O.V.A. (3rd task, 7-29 min)

The Tests of Variability of Attention (T.O.V.A.) was a 23 minute fixed interval visual continuous performance test during which two easily discriminated visual stimuli were presented for 100 milliseconds. Time between stimulus presentations was 2 seconds. Subjects were told to watch a video monitor and press a button whenever a square (the target) appeared at the top portion of an outer square (target stimulus): If the target appeared at the bottom portion of the

outer square (the foil), subjects were told to refrain from pressing the button. The targets are presented 2.5% of the time during the first half of the test and 77.5% of the time during the second half. The T.O.V.A. was presented on a personal computer.

Visual Reading Task (4th task, 30-34 min)

The visual reading task was a 1401 word story that had been approved by a learning specialist at the University of Ottawa (Appendix C) as appropriate for an adult population. The text was presented on a podium, at eye level, on 8.5 x 11 inch sheets of paper, while the subject sat at a comfortable reading distance (approx. 60 cm). The subject was asked to read silently and for meaning, informed that they would be permitted to read for 5 minutes and that they would be asked a few general questions about the text once they were finished (see Appendix D). After five minutes the subjects were told to stop reading and the number of words that had been read were counted.

Mental Rotation Task (5th task, 35-39 min)

During the mental rotation task subjects were shown two, two dimensional, irregular geometric forms that were either identical or mirror images of each other (see Appendix E). The forms were presented simultaneously, side by side, on the computer monitor at a distance of 120 centimeters. The geometric forms were

solid black in colour and presented on a grey background. The images remained on the monitor until the subject responded. The image on the right side of the monitor, the 'target' image, was presented at angles of (±) 0°, 30°, 60°, 90°, 120°, 150°, or 180° relative to the image on the left side of the screen, the 'base' image. The subject was instructed to observe the image on the left then to look at the image on the right, to mentally rotate the image on the right until it was at the same orientation as the image on the left, and then determine whether the target image was either identical to the base image or a mirror image. Subjects indicated their choice by pressing a left mouse button if the object was identical or a right mouse button if the object was a mirror image. Once the response was made the next pair of geometric forms appeared. Subjects were informed that it was important that they mentally rotated the object and that speed was not important. Each subject was given five practice trials during which they received feedback on their performance.

The mental rotation task was presented twice, after 34 minutes and after 45 minutes. During each presentation there were 88 pairs of images in each set, half the set were mirror images. Accuracy of performance and reaction time (RT) to each degree of rotation were measured. During the first presentation the target image was presented at rotations of 0°, 30°, and 60°. The images were presented

in randomized order on a personal computer.

Selective Attention Task (6th task, 40-41 min)

During the selective attention task, letter 'targets', H, K, or S, C, were presented to subjects above a marked fixation location on a computer screen. This task was presented on the video monitor at a distance of 60 cm. The viewing distance was closer than in other tasks because of the smaller size of the target stimuli. The subjects were instructed to press the left mouse button if they saw an H or a K, and the right mouse button if they saw an S or a C. The stimuli remained on the monitor until the subject responded. The targets were flanked by three distractor letters on either side that were 1) identical to the target, 2) the other member of the letter set, 3) members of the other response set, 4) not members of the set but having similar features, 5) dissimilar to the letter set, and 6) alone without distracters (Appendix F). Presentation of the letter set was followed by a 100 millisecond backward mask (#########). In addition to the different distractor sets, the separation between the target and the distracters was varied to be either .06°, 0.5°, or 1°. Subjects were given 10 practice trials during which they received feedback from the computer and the researcher on their performance. Subjects were instructed to perform the task as quickly but as accurately as possible.

This task was presented on two separate occasions, after the first mental rotation task at 39 minutes and after the second mental rotation task at 50 minutes. In the first presentation there were 90 trials with 35 of the targets presented at a 1° spacing, 35 at a 0.5° spacing, 10 presented with at a .06° spacing and 10 presented without distracters. The order of presentation for both sets was random. The reaction time and the accuracy of performance at the distractor spacings of .06°, 0.5°, or 1° were measured. The task was presented on a personal computer.

Listening Task (7th task, 42-46 min)

The auditory task was approved by a learning specialist at the University of Ottawa as appropriate for use with adults. The five minute listening task was a 1546 word story that was presented on a tape recorder (Appendix G). Subjects were asked to sit with their eyes open, to listen carefully and, in order to reduce eye movement, instructed to look at the same black asterisk used in the eyes open condition. The subjects were informed that they would be asked a few general questions about the story when it was completed (Appendix H).

Second Mental Rotation Task (8th task, 47-51 min)

The procedure, equipment, presentation, and stimuli for the mental rotation task have already been described above. During the second mental rotation, however, there were no practice trials and the target was presented at rotations of

(±) 0°, 30°, 60°, 90°, 120°, 150°, or 180°.

Second Selective Attention Task (9th task, 52-53 min)

The procedure, equipment, presentation, and stimuli for the selective attention task were previously described. During the second selective attention there were no practice trials and the number of trials were increased from 90 to 130 with 40 of the targets presented at a 1° spacing, 40 at a 0.5° spacing, 40 presented at the 0.06° spacing, and 10 presented alone.

Computer Reading (10th task, 54-56 min)

The computer reading text was a 260 word story that had been approved by a learning specialist at the University of Ottawa. During the computer reading task subjects read the story on a computer monitor. Each word of the story was presented at the centre of the video screen. The subjects controlled the pace of word presentation by pressing a mouse button, each button press resulted in the replacement of a word by the next word in the story. Subjects were informed that they would be unable to re-read a word or section once the mouse button was pressed and that they should read for meaning.

EEG Data Screening

Each two second epoch of raw EEG data was initially screened for eye blinks, eye movements, and EMG activity. After this initial screening, a qualified

EEG technician, who was blind to the subject's group and task condition, performed a second data screening. In addition to removing epochs that contained eye and muscle artifacts, the technician also screened for epileptic activity and abnormal EEG activity. Subsequent data analyses were restricted to individuals from whom a minimum of 60 seconds of artifact free data could be obtained, the minimum amount of data necessary to obtain reliable quantitative EEG measures (John, Prichep, & Ahn, 1986).

EEG Analysis

Fast Fourier transforms (FFT) were performed and numeric values plotted for each subject over each of the 19 standard locations and 7 frequency bands. The spectral values resulting from the FFT were used to form three types of data to be used in the analysis for each electrode, absolute data, relative data, and ratio data. The absolute data were the peak-to-peak amplitude in μV. The relative data were calculated from the absolute amplitude values by dividing the sum of all the amplitudes within all the frequency bands by each one of the bands at each of the electrode sites. The ratio data was calculated by dividing the values from one band into the other, e.g., the ratio of theta and beta1 activity (T/B) was formed by dividing theta activity by beta1 activity.

A preliminary examination of the absolute and relative data for each of the

frequency bands and for each of the groups showed that the data were skewed. In order to achieve approximately Gaussian distributions, the absolute data were transformed using log (X) and the relative data transformed using the formula log (X/(1-X)). These transformations are commonly conducted with qEEG data and have been shown to markedly reduce skew and kurtosis (Gasser, Bacher, and Mocks, 1982; John, Trepitin, and Kaye, 1980). Following the transformations, the majority of the sites were at or below a ± 1.00 level of skew and no site exceeded a ± 2.00, level. These levels of skew are considered to be from 'good' to 'excellent' (George & Mallery, 1998).

Multivariate and discriminant function analysis are particularly sensitive to extreme univariate and multivariate outliers. A data screening was thus performed to modify extreme outlying scores. For each of the seven frequency bands and the various ratios, univariate outliers ($Z > \pm 2.95$) within each group (control, CLINICAL, ADHDhy, and ADHDpi) were identified in each of the tasks. In a method outlined by Tabachnick and Fidell (1989), the transformed score of any univariate outlier was adjusted to be ten units greater (or less) than the next most extreme score in the distribution. Such a method maintained the rank position of the outlier and had the advantage that the sample size was maintained and the distribution was more normal in shape (Tabachnick and Fidell, 1989). In total, less

than 2% of the scores were modified using this method. When a subject's data were identified as extreme on more than 5% (8) of the site/band locations, the individual was deleted from that particular analysis. After the first screening, the data were re-screened until all univariate outliers had been removed.

Prior to the discriminant and multivariate analysis of each task, the EEG data were screened for multivariate outliers using Mahalinobis distance with $p < .001$ within each group (control, CLINICAL, ADHDhy, and ADHDpi) for each of the frequency bands and ratios in each of the tasks. Subjects who were identified as a multivariate outlier within a task were eliminated from the analysis. The multivariate screening was repeated until all multivariate outliers were identified and removed. Due to the sensitivity of discriminant analysis to both multivariate and univariate outliers, the univariate screening was run again after the removal of a multivariate outlier. The univariate and multivariate screening was repeated until no outliers remained. Following the data screening, Bartlett's test of sphericity, a measure of multivariate normality, was performed for each of the band, group, and task combinations and each of the variables was examined for fit between their distributions and the assumptions of multivariate analysis.

Statistical Analysis

In this study there were three independent groups, control, ADHDhy, and ADHDpi. The ADHDhy, and ADHDpi groups were also combined (labeled CLINICAL) for the purpose of comparisons with other studies and to provide more universal information about the general characteristics of ADHD. The total number of available subjects across groups was 74, although this number varied within each task due to the number of epochs rejected because of artifacts; within some tasks as few as 8 subjects within a specific group presented with artifact free data. The EEG was recorded from 19 electrode sites and each electrode site's activity divided into 7 frequency bands (high delta, theta, alpha, SMR, beta1, beta2, and the T/B ratio). The EEG was recorded during 10 different tasks; eyes open ("open", in which no task was performed), eyes closed ("closed", again in which no task was performed), TOVA, reading text ("read"), mental rotation 1 ("rot 1"), selective attention 1 ("sel 1"), listening to a story ("listen"), mental rotation 2 ("rot 2"), selective attention 2 ("sel 2"), and reading text on a computer ("comp"). There are thus (19 x 7 x 10) 1530 dependent measures. As the analysis of 1530 dependent measures is a daunting task, a more manageable approach would be to run a one-way, between groups ANOVA for each of these dependent measures; there would be, however, a considerable risk of chance significance.

Moreover, many of the dependent measures are correlated (i.e., not independent of each other). Another approach would be to run a between groups Multivariate Analysis of Variance (MANOVA) of the dependent measures, however, with the 'global' MANOVA there are far more dependent measures than subjects. The approach taken in this thesis was to run a separate analyses for each of the tasks which would reduce the number of dependent variables to 153 (19 x 7); even this number of variables is far more than the number of available subjects. In point of fact, there is no one analysis that can satisfy all statistical assumptions. For this reason, a variety of analyses were employed in an attempt to provide a complete view of the substantial amount of data that were collected in this study. In addition, due to the number of tests performed, familywise adjusted error rates were calculated for each of the analyses.

The EEG data collected during the performance of each task were analyzed in different ways. The main analyses were limited to those of relative and ratio data; however, at times, absolute data values were also used to assist in the interpretation of the relative data. There were seven main types of analyses performed on the data:

First, an analysis was performed to determine whether EMG artifact or the modification of outlying scores had affected the analyses. Due to the performance

of the data screening, certain sites might have been inadvertently biased resulting in abnormally high or low numbers of statistically significant results. In order to explore this possibility a phi-coefficient analysis was performed. This non-parametric analysis used data from individual t-test results to generate correlations between the sites and bands of the statistically significant results and the sites and bands that had been adjusted during the screening process.

The possibility of excess EMG affecting the results was addressed by recording and analyzing the EMG activity from the masseter muscle site and above the left eye. The ANOVAs were performed for each of the tasks using groups as the between subjects factor and the absolute as well as percentage data at these two sites as the dependent variables.

Second, for each of the tasks, the data in the T/B ratio, theta, beta1, and beta2 bands at the F3 site were summarized and presented as Figures in order to provide an overview of the results. In order to perform a correlational analysis, each of the tasks was coded by the order in which it was presented (e.g., eyes open was coded "1", eyes closed was coded "2", first quarter of the TOVA was coded "3".... up to the final task, computer reading, which was coded "13"). The correlations were then performed using the relative percentage of EEG activity from each of the bands for each of the ADHDhy, ADHDpi, and control groups

with the coded numbers of the tasks. The correlational and descriptive analysis performed on these data were used to better understand the EEG changes that occurred over the length of the study and during the performance of the various tasks.

Third, a series of stepwise discriminant function analyses were used to determine whether or not adults with ADHD could be reliably classified, which group could be best classified, and which of the tasks resulted in the highest classification rates. The percentage of EEG activity at each of the sites and each of the bands as well as the theta/beta ratios were used as the predictor variables and the groups were used as the dependent variables.

In this study, the number of correct classifications from the discriminant function analyses are reported as either the number of correct out of the total number of subjects in a group (e.g. 13/25) or as an average of the number of subjects that were correctly identified (e.g." for example, 13/25 would be expressed as "an average of 5 out of 10 subjects..."). Although the use of an exact percentage (52%) is simpler to read, it is misleading given the sample sizes used in this research.

Fourth, in order to examine if there were differences in the levels of activity between the left and right hemispheres during each task, a group (control,

ADHDpi, and ADHDhy) by hemisphere (Left and Right) Analysis of Variance (ANOVA) was conducted for each of the frequency bands (delta, theta, alpha, SMR, beta1 and beta2 and T/B ratio) using the ratio of the amplitude between the left to right hemispheres, within each of the bands, as the dependent variable. A more detailed examination of differences between the left and right hemispheres was then performed by pairing electrode sites from the left hemisphere with the corresponding electrode site on the right hemisphere. An ANOVA was then performed for each task using the ratio of the amplitude for each paired site, (Fp1/Fp2, F7/F8, F3/F4, T3/T4, C3/C4, T5/T6, P3/P4, O1/O2), in each band, as the dependent variable. Finally, in order to examine if there were different levels of activity between the left and right hemispheres within each group during the different tasks, paired-sample t-tests were performed between the total output from the left and right hemispheres for each group. The average percentage output from each hemisphere and each frequency band were used as the dependent variables for this analysis.

Fifth, to determine if there were regional differences in EEG activity - for each task in each of the frequency bands (delta, theta, alpha, SMR, beta1 and beta2) - a group (control, ADHDpi, and ADHDhy), by region (**Frontal**, the F3, F4, F7, F8, Fp1, Fp2, and Fz sites; **Central**, the C3, C4, and Cz sites; **Posterior**,

the P3, P4, Pz, O1, and O2 sites; **Left Temporal**, the T3 and T5 sites; and **Right Temporal**, the T4 and T6 sites) MANOVA was performed using the relative percentage of activity from the average of all electrode sites within each band as the dependent variables.

Sixth, in order to examine differences in EEG activity between adults and children with ADHD, planned t-test comparisons were conducted with previous research using children. In addition to providing information on maturational changes, the specific t-test information supplied in Appendices J-L may also assist clinicians in selecting the most advantageous sites and bands for neurotherapy.

Seventh, from a clinical perspective it is important determine which sites and frequency bands provided the largest inter-group differences. A 'Clinical' section, therefore, was added to aid the therapist in selecting an appropriate site and frequency band in which to perform neurofeedback. For this analysis, t-tests were performed at each of the sites in each of the bands for each of the tasks. This allows for the observation of the total number of statistically significant differences for each group within each band or site, over the entire testing period. The use of multiple t-tests is relatively common in evoked potential and qEEG research; it is used to reduce the large number of variables that are typically available in these kinds of analyses to more manageable levels (Chabot & Serfontein, 1996; Defrance

et al.,1996) and as a post hoc method of analysis (Rasey et al., 1999). In the present research, the results from these analyses provides specific information concerning the magnitude of the differences at each of the sites and bands.

CHAPTER 3

RESULTS

EEG Abnormalities and Artifacts

None of the raw EEG records of the 74 individuals included in the analysis contained epileptic or abnormal EEG activity.

One of the most basic requirements of EEG research is a 60 second sample of brain wave activity that is not contaminated by EMG, eye blinks, or other artifacts. In the present research, some of the subjects had difficulties producing the 60 seconds of artifact free EEG data required for the analysis of each task, especially during the T.O.V.A.. The problem was not limited to a few individuals but rather the result of an interaction between task type and subject, i.e., some individuals had trouble during the reading task while others had difficulties during the mental rotation task etc... By the end of testing all of the subjects had performed each of the tasks but only 5 ADHDpi, 13 ADHDhy, and 15 control subjects had complete data sets for all tasks. For example, although the data from 21 control subjects were analyzed in both the third quarter of the T.O.V.A. and the first selective attention task, only 15 control subjects had artifact-free data in both tasks. There were enough subjects in each group to satisfy the statistical requirements necessary to perform the multivariate, univariate, and non-

parametric analyses. Nevertheless, the number of subjects within each task did vary. For this reason, repeated measures analyses across tasks were not performed.

Data Outliers

Following the FFT, the data were screened for univariate and multivariate outliers. Less than 2% of the univariate distributions contained outliers and the multivariate screening procedure revealed that there was, on average, fewer than one multivariate outlier per task. The univariate outliers were modified as described in the Methods section and the very few multivariate outliers were eliminated from the analysis.

EMG Artifacts

During the raw EEG screening, care was taken to remove any epochs that were contaminated by EMG as even small amounts of this high frequency activity can contaminate the beta1 and beta2 bands. However, in order to further assess the presence of EMG activity, an analysis was performed using the artifacting leads on the supra-orbital ridge of the left eye and on the right masseter muscle. The site directly above the left eye is well situated to detect frontalis EMG without contamination from EEG activity (Duffy, 1993). A one way analysis of variance (ANOVA) was performed among the control, ADHDhy, ADHDpi, and

CLINICAL groups for each of the 10 tasks. With a Bonferroni adjusted error rate of .016 (.05/3), the results of the analysis showed no statistically significant differences in the percentage of EMG activity among the four groups.

The masseter muscle artifacting site was employed to detect EMG activity that may have contaminated sites at the temporal locations. A one way ANOVA was performed between the three groups for each of the 10 tasks using a Bonferroni adjusted error rate of .016 (.05/3). There were no statistically significant differences between the control group and the ADHDpi, or CLINICAL groups. There were, however, differences between the ADHDhy and the control group with the percentage of EMG significantly higher for the controls during the eyes open (F (2,55) = 5.16, p = .009), eyes closed (F (2,54) = 5.07, p = .01), and the second mental rotation task (F (2,52) = 6.34, p = .004).

Phi Coefficient Screening

Due to the sensitivity of discriminant function analysis to the inclusion of outliers, care was taken to modify any outlying scores following the transformations described in the Methods section. It is possible, however, that any statistically significant differences revealed during the analyses might be due to the influence of adjustments to the outlying scores. In short, there might not have been any statistically significant differences in the scores if the outliers not been

adjusted. This potential confound could create difficulties by artificially affecting the levels of statistical significance at the various sites and bands used in the analyses. One method to detect the presence of this type of confound is to perform non-parametric tests of the correlations between the locations at which statistically significant results occurred and the sites that were modified (George & Mallory, 1999). This is accomplished through the computation of the Phi coefficient (φ) which was performed for each of the groups in each of the tasks. To perform this test, t-tests were first performed at each of the 19 electrode sites in each of the 7 frequency bands resulting in 153 (7 x 19) t-tests for each one of the tasks. For each task, if a statistically significant t-test result was obtained at one of the 153 site/band locations it was coded as a "1" and if a non-significant result was obtained it was coded as a "2". Next, each one of the sites that had outliers that had been modified were similarly coded as "1" if a outlier had been modified and "2" if there was no outlier. The Phi-coefficient was then performed.

The appropriate test of φ against the null hypothesis is a Chi square test (χ^2). For the ADHDhy group, there were statistically significant correlations during the eyes closed condition ($\varphi = .26$, χ^2 (1) = 8.94, $p < .005$), the first quarter of the T.O.V.A. ($\varphi = .34$, χ^2 (1) = 15.44, $p < .005$), and the second mental rotation task ($\varphi = .27$, χ^2 (1) = 9.61, $p < .005$). These results suggest that for the ADHDhy

group, the modification of scores performed during the data screening may have resulted in a pattern of statistically significant t-test results during these tasks. There were no statistically significant correlations between the ADHDpi and CLINICAL groups.

Task Related Changes in the EEG

The purpose of this analyses is to present the data in a task-by-task fashion in order to observe the changes in EEG activity as they occur over time, during each of the tasks. As the tasks were presented in the same order for all of the subjects, this type of analysis can provide a useful overview as to how the passage of time and the different tasks differentially affected the groups. As it would be logistically difficult to present all of the activity from all of the bands over all of the tasks, only the T/B ratio and the percentage values of the beta1, beta2, and theta bands are presented. These particular bands were selected due to their importance in neurotherapy (Lubar, 1992). Similarly, it would be difficult to present the EEG activity during each task from all of the 19 sites used in the analysis, therefore, only the F3 site was presented although most of the activity in the other sites is similar to that of the F3 site. The data were originally analyzed as transformed values, however, the actual percentage values were used in this presentation. In order to perform this analysis, each of the tasks was coded from 1 to 13 (i.e., eyes

open = **1**, eyes closed =**2**, first quarter of the TOVA = **3**, second quarter of the TOVA = **4**.....up to the computer reading task = **13**) and correlations performed between the percentage values obtained during a task and the numbers (1 through 13) assigned to each task. For this analysis, the data from each quarter of the TOVA are presented as well as the data from the 9 other tasks. It should be noted that a major limitation of this type of analysis is that the correlations are based on only the F3 site and that differences in activity may not be due to the passage of time but rather tiredness, practice effects, or order effects. Nevertheless, the data presented here may provide a basis for future researchers to examine in closer detail changes in EEG activity over time.

Group Correlations with Tasks

As illustrated in Figure 1, the overall relative levels of theta wave activity were higher for the ADHDpi and ADHDhy groups than the controls. Only the control group's percentage of theta band activity was significantly correlated with the tasks r (12) = .645, p = .017 suggesting that as the number of tasks, and length of time, increased the level of theta band activity also increased.

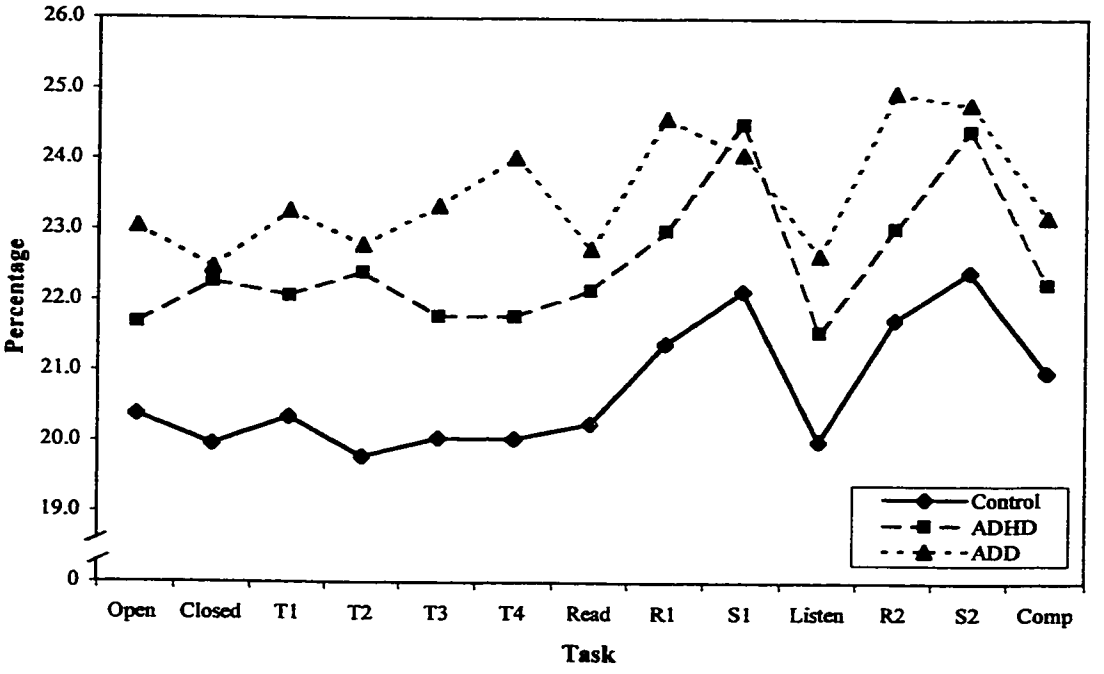

Figure 1. Percentage of theta wave activity at F3 across tasks.

Correlations Between Groups

In order to determine which of the three groups had the most similar pattern of EEG activity over time, or in response to the performance of a task, correlations were performed among the control, ADHDhy, and ADHDpi groups using the percentage values from each of the theta, beta1, beta2, and T/B ratio data.

Theta Band

In the theta band, the ADHDhy and controls had the highest correlation r (12) =

.90, $p < .01$), followed by the ADHDpi and controls r (12) = .810 $p < .01$), and the ADHDpi and ADHDhy r (12) = .67, $p < .05$) groups. The pattern of EEG activity of the ADHDhy and ADHDpi groups were more similar to the control group than to each other.

T/B Ratio

The correlation of T/B ratio activity between the control and ADHDhy subjects was the highest r (12) = .873, $p < .01$) followed by the ADHDpi and control group correlation r (12) = .648, $p < .05$). There was statistically significant correlation between the ADHDpi and ADHDhy groups. The T/B ratio data is presented in Figure 2.

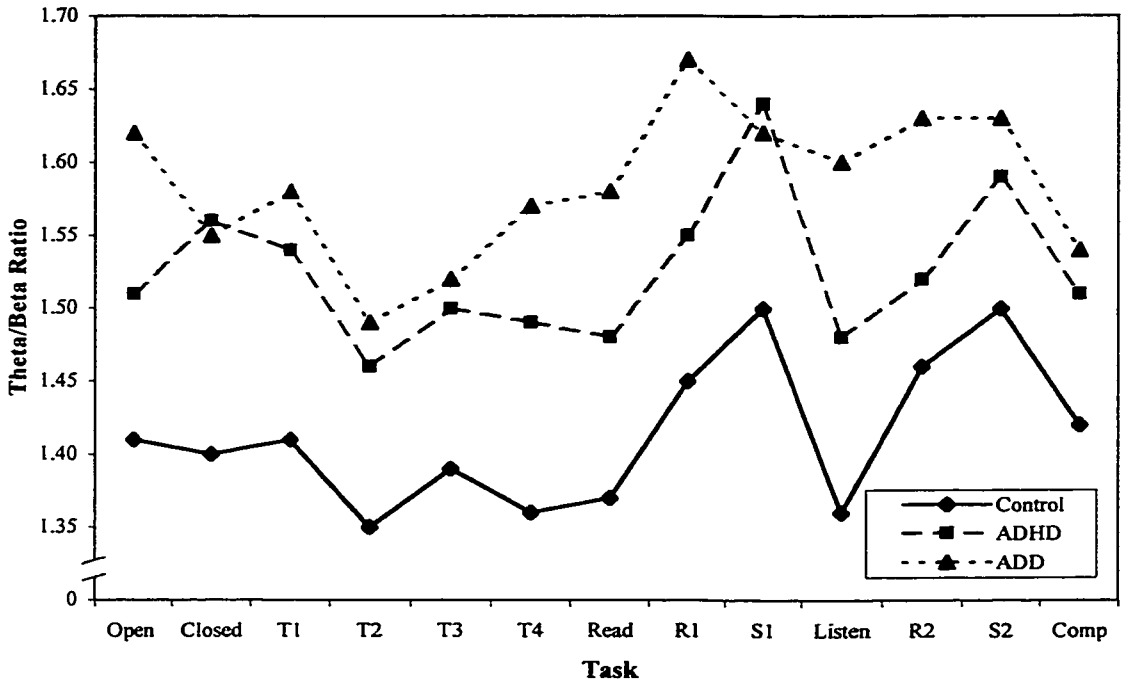

Figure 2. Theta/Beta ratio at F3 durings tasks.

Beta1 Band

In the beta1 band only the control and ADHDhy groups had a statistically significant correlation r (12) = .899, $p < .01$). The percentage levels of beta1 activity over the length of the study are illustrated in Figure 3.

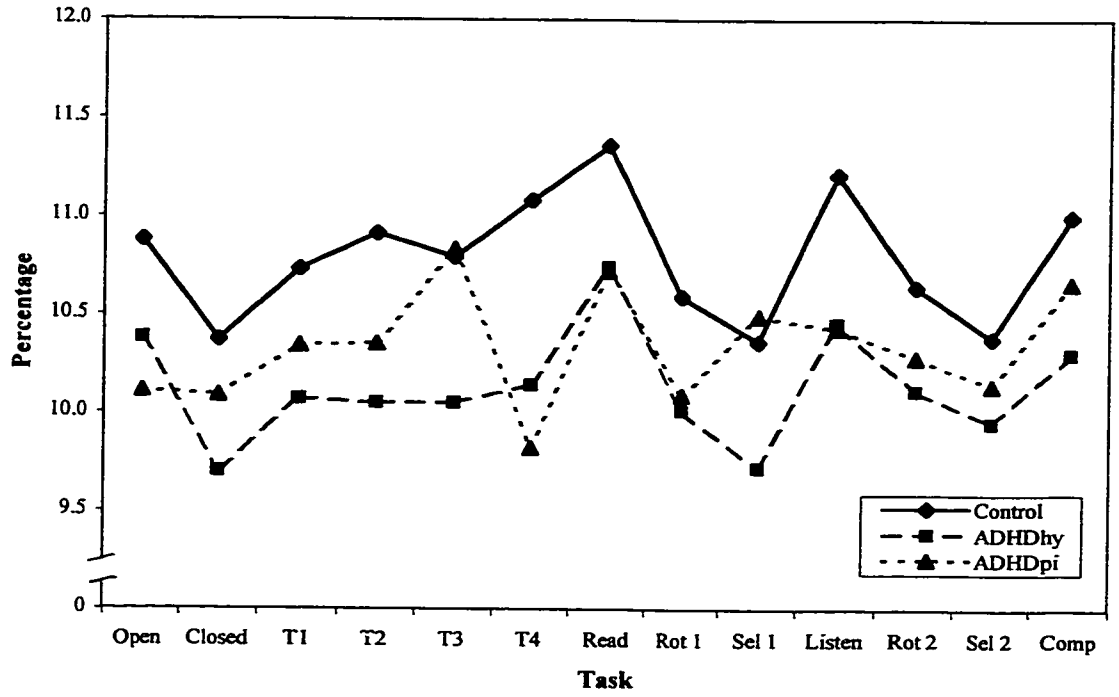

Figure 3. Percentage of beta1 wave activity at F3 across tasks.

Beta2 Band

Similarly, there was a significant correlation between the ADHDhy and the control group in the beta2 band r (12) = .644, $p < .05$) but no statistically significant correlation between the controls and ADHDpi group or between the ADHDhy and

ADHDpi groups. The pattern of changes in the percentage of beta2 activity are presented in Figure 4.

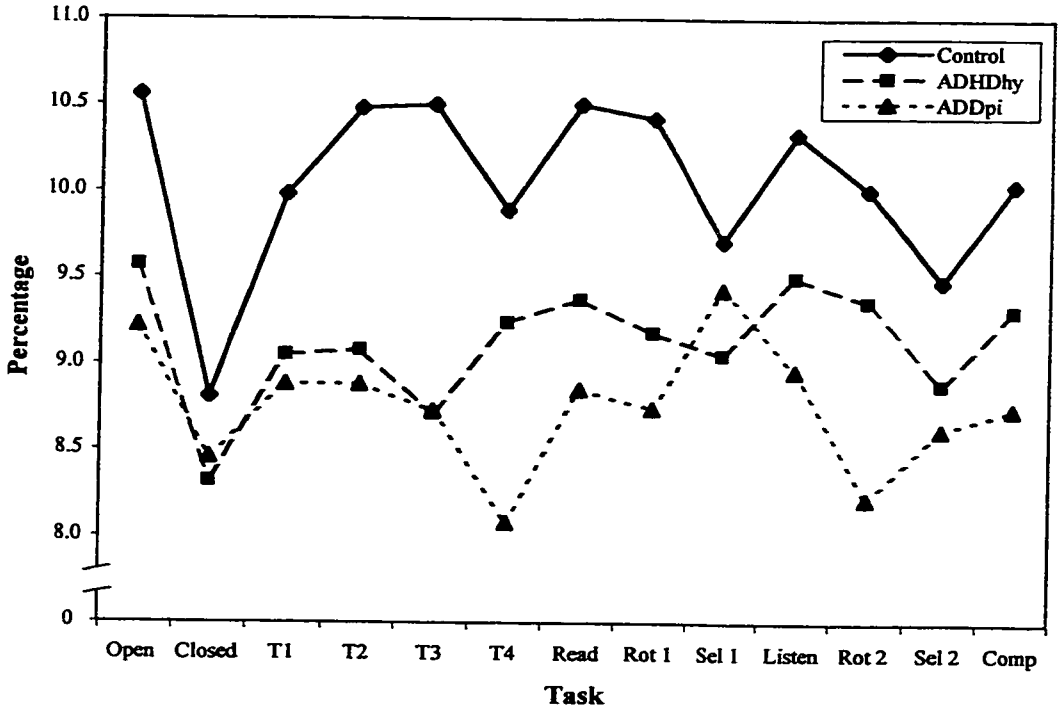

Figure 4. Percentage of beta2 wave activity at F3 across tasks.

In summary, the correlational analyses suggest that the ADHDhy and control groups share the most similar pattern of increases and decreases of theta, beta1, beta2, and T/B ratio activity over the length of the study. It also important to note that the pattern of increased theta and T/B for the ADHD groups and increased beta1 and beta2 for the control group, with few exceptions, is consistent throughout the length of the study.

Discriminant Function Analysis

Disciminant Function Analyses (DFA) are commonly employed in clinical research studies of ADHD children to provide a means of identifying other children with this disorder. The main purpose of DFA is to predict an individual's membership in a group based on a set of predictors. This is done by creating a mathematical 'function' comprised of the best predictors of the subject's group. In this study, the predictor variables are the EEG measures (transformed percentage values) obtained from the subjects at the 19 electrode sites and the 7 frequency bands and the T/B ratio.

This section sets out the results of the discriminate function analysis of the control, CLINICAL, ADHDhy, and ADHDpi groups using sites and frequency bands as the predictor variables. There were six frequency bands and the theta/beta (T/B) ratio for each of the 19 electrode sites, resulting in a total of 133 (7 x 19) predictor variables available for each of the tasks. This number of variables far exceeds the maximum subject/variable ratio recommended for use in a multivariate analysis. Generally, the number of variables should be less than the number of subjects (Tabachnick and Fidell, 1989).

In order to reduce the number of variables the data were analyzed in two stages. The first stage was comprised of separate discriminant analyses using the

19 electrode sites in each of the T/B ratio, and the high delta, theta, alpha, SMR, beta1, and beta2 bands as the predictor variables for each of the tasks. There were thus 70 DFA in this initial 'run'. Each one of these 70 DFA produced a 'function' that was comprised of a smaller number of dependent variables than the 19 that were originally entered into the analysis. In the second stage, this smaller group of variables that comprised the discriminant functions from each of the bands and T/B ratio analyses was combined. This 'combined' set of variables was thus comprised of predictor variables from each of the frequency bands and T/B ratio. There was one 'combined' set of variables for each of the tasks. With an aim to improving the classification rates, this combined set of predictor variables was then used to perform a DFA for each of the tasks. Separate two group DFA were performed for each task between the control and ADHDhy, control and ADHDpi, and control and CLINICAL groups. Finally, a three group - control, ADHDhy, and ADHDpi - analyses was also performed for each of the tasks. For all of these analyses, the data collected during the performance of the TOVA was analyzed for each of the 4 quarters, and each of the 2 halves. These 6 TOVA analyses and the analyses of the 9 other tasks resulted in a total of 15 tasks being analyzed.

Prior to each of the discriminate function analyses the data were screened for univariate and multivariate outliers and the assumptions of normality assessed.

Subjects who had produced less than the 60 seconds of artifact free data required for entry into the analysis were dropped. Bartlett's test of sphericity was then used to determine whether the set of variables being used in the analysis were from a multivariate normal distribution. Following the analysis, an evaluation of multicollinearity and singularity was performed by reviewing the tolerance tests at each step and the homogeneity of the variance-covariance matrices checked through the results of a Box's M test.

Cross Validation

An asset in DFA is the availability of a cross validation group with which to assess the generalizability of the discriminant functions. Unfortunately, it was not possible to form separate cross-validation groups because of the small sample sizes. To reduce the bias in classification a 'jackknifed' procedure was used whereby each subject's discriminant scores are removed before the classification function is calculated (Chabot & Serfontein, 1996; Lubar, Bianchi, Calhoun, Lambert, Brody, & Shabsin, 1985; Mann et al., 1991; Princhep & John, 1986). This classification gives a more realistic estimate of the ability of the predictors to separate the groups (Tabachnick & Fidell, 1989).

The success of a discriminant function is based on its ability to correctly identify members of different groups. In addition, the chi-square value (χ^2), Wilk's

Lambda (λ), and the eigenvalue can also used to assess the strength and utility of a function. Chi-square is a measure of whether the levels of the function significantly differ from each other. A high chi-square indicates that the function discriminates well. The chi-square value in this research is based on Wilk's Lambda, i.e., the ratio of the within groups sum of squares to the total sum of squares: The lower the Wilk's Lambda value, the better the function. The eigenvalue is the ratio of the between group's sum of squares divided by the within group's sum of squares. It is also a measure of the strength of the discriminant function with values exceeding 1.0 considered to be acceptable, and higher values indicating a stronger function. Finally, the canonical correlation is a measure of the correlation between the discriminant scores and the levels of the dependent variable. A high correlation indicates that the function discriminates well. In the present research, only functions that correctly classified an average of more than 8 out of 10 of the ADHD and control group subjects, with eigenvalues greater than 1, canonical correlations greater than .70, and significance levels of $p < .001$ are presented in Table 2.

Table 2

Number of Subjects Correctly Classified, Eigenvalues, Canonical Correlations, Task, and Type of Variable in the Most Successful Disciminant Functions

Number of Subjects Correctly Classified					Canonical Correlation	Eigenvalue	Task	Type of Variables
ADHDpi	ADHDhy	Clinical	Control	Overall				
—	—	27/35	22/28	49/63	.729	1.134	TOVA 2/2	Combined
10/13	—	—	19/21	29/34	.840	2.404	Select 1	Combined
—	—	34/39	20/30	54/73	.712	1.026	Listen	Combined
12/13	—	—	18/21	30/34	.757	1.338	TOVA 3/4	Combined
—	—	14/20	17/21	31/41	.756	1.335	TOVA 4/4	Combined
—	12/13	—	21/22	32/35	.882	3.505	TOVA 3/4	Alpha
—	9/13	—	17/20	22/33	.748	1.268	TOVA 4/4	Alpha
—	9/13	—	16/20	25/33	.714	1.043	TOVA 4/4	SMR
—	—	21/25	16/21	37/46	.733	1.159	TOVA 3/4	Combined
9/16	—	—	27/30	36/46	.765	1.408	TOVA ½	Combined
11/15	—	—	20/24	31/39	.852	2.650	TOVA 2/4	Combined

Eleven of the analyses produced functions that exceeded the chi-square, eigenvalue, canonical correlation, and classification criteria outlined above. Notably, nine of these functions were 'developed' during the T.O.V.A., suggesting that this test created the most advantageous conditions to detect differences in qEEG activity. The term 'developed' is used here because the EEG values were a result of the subject performing a particular task and not based on an existing value (e.g., height, weight, income etc... would be considered existing values). The function with the highest eigenvalue (3.51), lowest Wilk's Lambda (.22), highest canonical correlation (.88), and the highest number of correct classifications (32 of 35 ADHDhy and control subjects correctly identified) was developed using alpha band values while the subject performed the third quarter of the T.O.V.A.. The best function for correctly classifying ADHDpi subjects was developed during the second quarter of the T.O.V.A. using the combined variables. It should be noted that the classification rate is lower for this function than either the selective attention or the third quarter of the T.O.V.A. functions; however, the higher eigenvalue and canonical correlation suggest that this is the most robust of these functions. The function that was most successful in identifying CLINICAL group subjects was developed during the third quarter of the T.O.V.A. using the combined variables.

The least accurate functions were developed during the three group DFA with an average correct classification rate of 5 out of 10 subjects compared to an average of 3 out of 10 that would have been expected by chance alone. As expected, the combined functions, on average, predicted membership more accurately than the functions based on variables derived from individual frequency bands.

The age, gender, and group of the subjects who were misclassified can provide useful data regarding the weakness of a discriminant function. To this end, a summary of the subjects who were misclassified, based on the functions noted above, is presented in Appendix I. None of the female ADHDhy subjects were misclassified while the mean age of the misclassified ADHDpi subjects appear to be older than the correctly classified subjects (37.3 yrs vs 29.8 yrs, respectively).

Results from the Analyses of Inter-hemispheric Activity

Previous research has shown differences in brainwave activity between the left and right hemispheres of children with ADHD and control subjects. By comparing the results of the present research with previous research with children it is possible to see whether the differences between ADHD and normal children are present in ADHD and normal adults. This section describes and summarizes the results of the analyses of inter-hemispheric activity in the control, ADHDhy,

and ADHDpi groups. There were three separate analyses of inter-hemispheric activity in this section. The first two analyses examined differences in inter-hemispheric activity among the groups while the third analysis examined the differences in inter-hemispheric activity within each group.

Overall Differences in Inter-hemispheric Asymmetry

The first set of comparisons examined differences between the percentage of activity in the left and right hemispheres between each group, within each of the six frequency bands, during each of the tasks. For this set of analyses, the data collected during the performance of the TOVA was analyzed for each of the 4 quarters, and each of the 2 halves. These 6 TOVA analyses and the analyses of the 9 other tasks resulted in a total of 15 tasks being analyzed. For each task and within each frequency band, a ratio of left and right hemispheric activity was computed by dividing the total percentage output of all electrodes in the left hemisphere by the total percentage output of all electrodes in the right hemisphere. Thus, a ratio greater than 1.0 would indicate greater activity in the left hemisphere whereas a ratio less than 1.0 would indicate greater activity in the right hemisphere. The resulting ratio was then analyzed through a one-way ANOVA with the ratio as the dependent variable and groups as the between subjects factor. Separate ANOVAs were run for each of the 6 frequency bands (and the T/B ratio)

in each of the 15 tasks. The Bonnferroni adjusted error rate was set at $p < .007$ to compensate for the fact that there were 7 analyses in each task (i.e., the .05 alpha level was divided by the number of analyses, 7). The adjustment was made only for the number of frequency bands and the T/B ratio and not both the number of frequency bands and tasks as such an alpha level would have been overly conservative ($p < .0007$). Overall, there were 105 ANOVAs performed in this analysis. There was a statistically significant difference during the second mental rotation task ($F(2,62) = 5.66$, $p = .006$) with the ADHDpi subjects producing more left hemispheric activity in the delta band compared to the control group, 1.03 vs .99. None of the ADHDhy or CLINICAL comparisons were statistically significant.

Paired-Site Analyses of Inter-hemispheric Activity

In order to perform a more detailed examination of inter-hemispheric differences, the ratio of activity between individual sites in the left and right hemispheres were computed. The ratios were calculated for the Fp1/Fp2, F3/F4, C3/C4, P3/P4, O1/O2, F7/F8, T3/T4, T5/T6 sites by dividing the percentage output from a particular band in the left hemisphere site by the output of the same band in the right hemisphere. A ratio greater than 1.0 would indicate greater activity in the left hemisphere whereas a ratio less than 1.0 would indicate greater

activity in the right hemisphere. The data collected during the performance of the TOVA was analyzed for each of the 4 quarters and each of the 2 halves. These 6 TOVA analyses and the analyses of the 9 other tasks resulted in a total of 15 tasks being analyzed. A one-way ANOVA was then performed for each of the 8 sites using group membership as the between subjects factor. As there were 8 paired sites formed for each band, the Bonferroni adjusted error rate was set to $p < .006$ (i.e., alpha of .05 divided by the number of paired sites, 8). In total, there were 120 ANOVAs performed in this analysis. As noted in the previous analyses, using the total number of tasks (15) and the number of frequency bands (6) in the calculation of the error rate would have been too conservative. There were no statistically significant differences between the ADHDhy, ADHDpi, or CLINICAL groups at any of the sites in any of the frequency bands.

Within Groups Differences in Inter-hemispheric Activity

In order to further explore differences in inter-hemispheric activity among the groups an analysis was performed on the left and right hemispheric activity within each of the groups. In this analysis, for each of the bands and the T/B ratio, the percentage output from all of the electrodes in the left hemisphere was compared to the percentage output of all of the electrodes in the right hemisphere. These data were analyzed using a paired-samples t-test of left vs right hemispheric

activity in each of the bands (and T/B ratio) and each of the 15 tasks (the TOVA was once again analyzed for each of the 4 quarters and each of the 2 halves). The Bonnferroni adjusted familywise error rate was set to $p = .007$ to compensate for the number of analyses (7) for each task (.05/7). The adjustment was made only for the number of frequency bands and the T/B ratio and not both the number of frequency bands, T/B ratio, and tasks as such an alpha level would have been overly conservative ($p < .0007$). In total, there were 105 t-tests performed in this analysis. There were significant differences between the percentage of activity in the left and right hemispheres for the ADHDhy group during the eyes closed, the third quarter of the T.O.V.A., and second mental rotation tasks. During the eyes closed condition, the ADHDhy subjects generated a higher T/B ratio ($t(22) = -3.34$, $p = .003$) and a higher percentage of alpha band activity in the right hemisphere ($t(22) = -4.11$, $p < .0005$), but a lower percentage of beta1 ($t(22) = 4.26$, $p < .0005$) and beta2 activity ($t(22) = 4.04$, $p = .001$) in the left hemisphere. During the third quarter of the T.O.V.A., the ADHDhy subjects had a lower percentage of beta1 activity in the left hemisphere ($t(12) = 3.34$, $p = .005$). Finally, during the second mental rotation task the ADHDhy subjects had a higher percentage of alpha band ($t(21) = -4.07$, $p = .001$) and delta band activity ($t(22) = -2.88$, $p = .005$) in the right hemisphere but a lower percentage of beta1 activity in

the left hemisphere ($t(22) = 3.48$, $p = .005$). There was only one statistically significant difference in the ADHDpi group, during the computer reading task; the percentage of theta activity in the left hemisphere was greater than that of the right hemisphere ($t(22) = 3.20$, $p = .006$). There were no statistically significant differences within the control group during any of the tasks in any of the bands.

Results from the Analyses of Regional Activity

Previous research with ADHD children has shown regional differences in brainwave activity between this group and a group of normal children. By comparing the results of the present research with previous research using children it is possible to see whether the differences between ADHD and normal children are present in ADHD and normal adults. This section describes and summarizes the results of the analyses of the neuro-anatomically distinct regions of the scalp. To facilitate this comparison, five regions were formed; **Frontal**, the F3, F4, F7, F8, Fp1, Fp2, and Fz sites; **Central**, the C3, C4, and Cz sites; **Posterior**, the P3, P4, Pz, O1, and O2 sites; **Left Temporal**, the T3 and T5 sites; and **Right Temporal**, the T4 and T6 sites. For each of the 5 regions, an average percentage value for each of the frequency bands was computed by summing the values from each of the sites and dividing by the number of sites. These 5 values in each of the frequency bands and the T/B ratio served as the dependent variables.

A one-way MANOVA with a single between groups (ADHDhy, ADHDpi, and control) factor was run on the dependent variables (i.e., the average percentage of activity within each of the regions). A separate MANOVA was run for each frequency band and the T/B ratio for each of the 15 tasks (6 from the TOVA and the 9 other tasks) resulting a total of 105 MANOVAs in this analysis. The Bonferroni adjusted error rate for these comparisons was set at $p < .007$ (.05/7). The adjustment was made only for the number of frequency bands and the T/B ratio and not both the number of frequency bands and tasks as such an alpha level would have been overly conservative ($p < .0007$).

During the eyes closed condition, there was a significant group difference for the T/B ratio ($F(10, 128) = 2.71$, $p = .005$). Compared to the control group the ADHDhy group had a significantly higher T/B ratio in the right temporal region and the frontal region. None of the other regional comparisons reached statistical significance.

Planned t-test Comparisons

Other researchers have used t-tests to analyze differences between normal and ADHDhy, ADHDpi, and CLINICAL children and adults. These t-tests were conducted at each of the sites and in each of the bands and the T/B ratio during the performance of a variety of tasks. In order to compare the results of this study

with previous studies using children and adults, t-test were conducted between the control and ADHDpi, ADHDhy, and CLINICAL groups. By comparing the results it is possible to determine if the differences reported between children with ADHD and normal controls subjects are present between adults with ADHD and normal control subjects. For all of the t-test analyses the percentage values at each electrode site within each of the 6 frequency bands and the T/B ratio served as the dependent variables. This resulted in a total of 133 (7 x 19) t-test per task.

For the CLINICAL group, t-tests were performed on the data collected during the eyes closed condition. For the ADHDpi group, t-tests were performed on the data collected during the eyes open, reading text, and mental rotation tasks. For the ADHDhy group, t-tests were performed on the data collected during the eyes open, eyes closed, and the TOVA tasks. The results of these analyses are presented in Appendices J-L.

CLINICAL Group

During the eyes closed condition, the statistically significant differences between the controls and the CLINCAL group were numerous in many different sites with higher T/B ratios and higher percentage levels of theta and delta band activity but lower percentage levels of beta1 and beta2. Many of the results were below the $p < .01$ and $p < .001$ level. Notably, the lowest number of significant t-

test differences were in the beta2 band (Appendix J1).

ADHDpi Group

The only statistically significant difference between the control and the ADHDpi group was in the eyes open condition in the T/B ratio (Appendix K1). During the reading task the ADHDpi group had a significantly higher T/B ratio, a higher percentage of theta band activity, and a lower percentage of beta2 (Appendix K2). During the mental rotation task the ADHDpi group had a significantly higher T/B ratio, a higher percentage of delta and theta band activity, but a lower percentage of beta2 activity; none of the differences noted above were significant below the $p < .01$ level (Appendix K3).

ADHDhy Group

During the eyes open condition, the ADHDhy group had a higher T/B ratio, a higher percentage of delta and theta band activity, and a lower percentage of beta1 and beta2 activity compared to controls (Appendix L1). The majority of the significant differences were in the T/B ratio data with only two statistically significant differences in the theta and delta bands. During the eyes closed condition the ADHDhy group had a higher T/B ratio, a higher percentage of delta and theta band activity, and a lower level of beta1 activity. The differences were numerous with many significant at or below the $p < .01$ level (Appendix L2).

During the performance of the T.O.V.A., the ADHDhy group had higher T/B ratios, a higher percentage of delta and theta band activity, and a lower percentage level of beta1 and beta2 activity (Appendix L, Tables L3-L6).

In summary, for all of the statistically significant t-test results, the ADHDhy, ADHDpi, and CLINICAL groups had higher T/B ratios and higher percentage levels of delta and theta band activity while the control subjects had higher percentage levels of beta1 and beta2 activity. For all three groups, there were no statistically significant differences in the SMR band and only one statistically significant difference in the alpha band.

CLINICAL SECTION

Summary Observations

The results depicted earlier in Figures 1 to 4, suggest a general and consistent trend across all tasks. For this reason, in order to provide an overview of the theta, beta1, and beta2 activity, this section provides a summary of EEG activity over the entire testing period lasting approximately 56 minutes (Table 3). These averages were calculated for each of the 19 sites by summing the values from each task and dividing by the number of tasks. The control group had the highest percentages of beta1 and beta2 activity and the ADHDpi group had the highest percentage levels of theta band activity, followed by the ADHDhy and

control groups.

Table 3

Summary of Theta, Beta1, and Beta2 Activity Collapsed Across Tasks and Sites

Wave Band	*p-p Amp. or % values	Control	$ADHD_{HY}$	$ADHD_{PI}$
Beta1	%	11.13	10.37	10.56
	p-p Amp.	5.85μV	5.15μV	4.97μV
Beta2	%	9.91	9.42	9.16
	p-p Amp.	5.09μV	4.66μV	4.23μV
Theta	%	18.47	19.81	20.63
	p-p Amp.	9.50μV	9.72μV	9.81μV

* p-p Amp. is peak-to-peak amplitude.

Due to the importance of theta and beta band activity in the neurotherapy treatment of ADHD, it is important to know the sites at which the highest percentage of activity occurs. The two sites with the highest relative levels of theta wave activity were Fz and Cz sites respectively for all three groups, but at the F4 site for the ADHDpi and ADHDhy subjects and the F3 site for the control subjects. The two sites with the lowest percentage of theta wave activity were at the T4 and T3 sites respectively for all groups, but at the Fp1 site for the controls, the O2 site for the ADHDhys, and the T6 site for the ADHDpi subjects. These data are presented in Table 4.

Table 4

The Three Sites with the Highest and Lowest Percentages of Theta, Beta1, and Beta2 Activity

Wave Band	Highest/ Lowest	Control Sites			ADHDhy Sites			ADHDpi Sites		
Theta	Highest	Fz	Cz	F3	Fz	Cz	F4	Fz	Cz	F4
	Lowest	T4	T3	Fp1	T4	T3	O2	T4	T3	T6
Beta1	Highest	T4	T3	F8	T3	T4	F7	T4	T3	F8
	Lowest	Cz	Fz	Pz	Cz	Fz	Pz	Cz	Pz	P4
Beta2	Highest	T4	Fp1	Fp2	T3	T4	Fp1	T4	T3	Fp2
	Lowest	Pz	O2	P4	Pz	P4	Cz	Pz	P4	O1

Based on the previous analysis, the midline frontal (Fz, Cz) sites would probably be chosen as the most likely sites at which to provide feedback. If the goal of treatment, however, is to change the ADHD subject's EEG activity to more closely resemble a normal subject, consideration should be given to an alternative method of site selection. Specifically, a site that produces a level of activity that is most dissimilar to the control group's should also be considered as a target area for neurofeedback. The highest percentage differences between the ADHD groups and the control group in the theta, beta1, and beta2 bands are illustrated in Table 5.

Table 5

The Three Sites with the Greatest and Least Differences in the Percentage of Theta Beta1 and Beta2 Activity

Wave Band	Largest/Smallest	ADHDhy Sites			ADHDpi Sites		
Theta	Largest	F3	Fp1	T3	Fp1	F3	Fp2
	Smallest	T6	O2	P4	T4	T6	T3
Beta1	Largest	Fp2	T4	Fp1	Fp1	O1	Fp2
	Smallest	T3	O1	O2	T4	T3	Cz
Beta2	Largest	F3	Fp1	Fp2	F3	Fp1	F4
	Smallest	O1	T6	O2	T4	T3	T6

These results suggest that the frontal electrode sites (Fp1, Fp2, F3, and F4) in the ADHD group are the most different from the control group. There are, however, sites that are specific to each of the ADHD groups. For example, the ADHDhy group has large differences in the percentage of activity in the temporal area (T4 and T6) while the ADHDpi group has large differences in the left occipital (O1) area.

Relative Data t-tests

This section summarizes the results of t-tests performed between the ADHDhy, ADHDpi, and CLINICAL groups. As described in the Methods section, the t-tests were performed in order to provide the clinician with a reference of the sites at which the percentage differences were largest during the

various tasks used in this research. In addition, these analyses also provide a descriptive format in which to screen for differential patterns of activity between groups. The data collected during the performance of the TOVA was analyzed for each of the 4 quarters and each of the 2 halves. These 6 TOVA analyses and the analyses of the 9 other tasks resulted in a total of 15 tasks being analyzed. For each of the 15 tasks, t-tests were performed at the 19 electrode sites in the high delta, theta, alpha, SMR, beta1, and beta2 bands as well the T/B ratio. This resulted in 133 t-tests per task (7 bands x 19 sites), and 1,995 tests overall (133 t-tests x 15 tasks) for each of the ADHDhy, ADHDpi, and CLINICAL groups; the entry criteria for inclusion in the analysis was $p < .05$, two-tailed. Prior to analysis, the data were screened according to the criteria outlined in the Data Screening section. Levine's test for Equality of Variances was performed for each of the t-tests and, in the case of unequal variances, the t-score and probability values were adjusted. In order to provide an overview of activity within each of the frequency bands or electrode sites, the number of statistically significant t-test results at each site and within each frequency band were summed. For example, there were 7 t-tests performed at each site, one for each of the six frequency bands and one for the T/B ratio. These 7 tests were repeated for each of the 15 tasks resulting in 105 t-tests (7 x 15) at each site. Similarly, within each frequency band, there were 19

t-tests in each task, one for each of the 19 electrode sites. These 19 tests per frequency band were repeated for each of the 15 tasks resulting in 285 t-tests per frequency band. The purpose of grouping the t-test results in this manner is to permit the observation of consistent patterns that may not be apparent in task-by-task examination, to aid in selecting the appropriate frequency bands and sites for neurotherapy, and to assist in comparisons with previous research.

Frequency Band Activity

The number of times a statistically significant difference occurred within a particular frequency band may indicate whether there are persistent differences between the groups in that band and thereby assist in selecting the bands that are most suited to neurofeedback. This section provides a closer look at the differences between the groups by reporting the number of statistically significant results within each of the frequency bands and ratio data, collapsed across tasks and sites, with a $p < .05$ level for inclusion into the analysis. In addition, the number of times a statistically significant difference occurred at the $p < .01$ and $p < .001$ level, is also reported.

Summary of t-test results between the Control and CLINICAL Groups

The t-test comparisons of the control and CLINICAL groups indicate that most of the statistically significant differences were in the T/B ratio data, followed

by the theta, beta1, beta2 and delta bands. The differences were a result of a higher T/B ratio and higher percentages of delta and theta band activity in the CLINICAL group, whereas there was a higher percentage of beta1 and beta2 activity in the control group. As set out in Table 6, there were no statistically significant differences in the alpha band and only one significant difference in the SMR band.

Table 6

Summary of t-test Results in Bands Collapsed across Tasks for Control and CLINICAL Subjects

	Bandwidth							
Error	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta/ Beta	Total
p≤.05	0	62	48	43	1	58	59	271
p≤.01	0	17	12	18	0	18	60	126
p≤.001	0	1	1	0	0	9	26	37
Total	0	80	61	61	1	85	145	434

Control and ADHDhy Groups

The highest number of statistically significant differences between the control and ADHDhy groups, as indicated in Table 7, also occurred in the T/B ratio, followed by the theta, beta1, beta2, and delta bands. The differences were a result of higher T/B ratios and higher percentages of delta and theta band activity in the ADHDhy group but higher percentages of beta1 and beta2 activity in the control group. There was one statistically significant difference in the alpha band

and none in the SMR band.

Table 7

Summary of t-test Results in Bands Collapsed across Tasks for Control and ADHDhy Subjects

Error	Bandwidth							Total
	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta/ Beta	
p≤.05	1	27	16	25	0	35	49	153
p≤.01	0	3	3	1	0	7	22	36
p≤.001	0	0	0	0	0	1	3	4
Total	1	30	19	26	0	43	74	193

Control and ADHDpi Groups

The results of the comparisons between the control and ADHDpi group are similar to those of the CLINICAL and ADHDhy groups with the majority of the statistically significant differences in the T/B ratio, theta, and delta bands. The differences were the result of higher T/B ratios and higher percentages of delta and theta band activity in the ADHDpi group but higher percentages of beta2 activity in the control group. The most notable difference between the results from the ADHDpi group and the results from the ADHDhy and CLINICAL groups, as presented in Table 8, was the absence of statistically significant differences in the beta1 band. Again, there were very few differences in the alpha band and SMR bands.

Table 8

Summary of t-test Results in Bands Collapsed across Tasks for Control and ADHDpi Subjects

Error	Bandwidth							Total
	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta/ Beta	
p≤.05	2	1	33	13	1	33	69	152
p≤.01	0	0	1	7	0	5	10	23
p≤.001	0	0	0	0	0	0	0	0
Total	2	1	34	20	1	38	79	175

In summary, the majority of the statistically significant differences across all of the tasks occurred in the T/B ratio, high delta, theta, beta1, and beta2 bands. There were very few statistically significant differences in the alpha and SMR bands and the ADHDpi group set itself apart from the ADHDhy and CLINICAL groups with a notable lack of significant differences in the beta1 band.

Site Activity

The choice of the most appropriate site at which to perform neurotherapy may play an important role in the success of treatment. This section examines the differences between the groups by reporting the number of statistically significant differences at each of the 19 electrode sites, collapsed across bands and tasks with entry into the analysis set at a $p < .05$ level.

Control and CLINICAL Groups

A comparison of the differences between the CLINICAL and control

groups indicates that the five sites with the highest number of differences were F3, F4, Fp2, Fz, and C3, respectively. As indicated in Appendix M, Table M1, four of the five sites were in the frontal area; two over the left hemisphere, two over the right hemisphere, and one over the central area. At a $p < .01$ level, the seven sites with the highest number of differences were F3, O1, F4, Fp1, Fp2, Fz, and T5.

Control and ADHDhy Groups

As indicated in Appendix M, Table M2, the Fp2, F3, Fp1, T5, and Fz sites, had the highest number of statistically significant differences between the controls and the ADHDhy group. Four of the five sites yielding the highest number of statistically significant differences were over the frontal area, results which were similar to those of the CLINICAL group. Three of the sites were over the left hemisphere one in the right hemisphere and one over the central area. An examination at the $p < .01$ level indicated that the T5 and Fp1 sites had an equal number of highly statistically significant differences, followed by the F3, Fp2, and T4 sites, also with an equivalent numbers of statistically significant differences.

Control and ADHDpi Groups

A comparison of the differences between the ADHDpi and control groups, as presented in Appendix M, Table M3, indicates that the greatest number of statistically significant differences were at the F3 site followed by the O1, F7, Fp1, T5, and P3 sites, respectively. Three of the sites were frontal, with one each over the occipital, parietal, and temporal areas. It is noteworthy that only 50% of the largest number of statistically significant differences in the ADHDpi group were in

the frontal area compared to the 80% observed in the CLINICAL and ADHDhy groups. The highest number of statistically significant differences at the $p < .01$ level were at the O1, Fp1, F3, C3, O2, and Fz sites, respectively. The absolute and relative t-test results between the control and: ADHDhy, ADHDpi, and CLINICAL groups are presented in Appendices N-R and the percentage differences between the ADHDhy and ADHDpi groups in Appendix S.

CHAPTER 4

Subject Task Results

The focus of this research has been on the analysis of EEG data recorded while subjects performed various tasks. The data from the performance of these tasks is of importance and is presented in this section.

Eyes Open And Eyes Closed

As noted in the Methods, at the end of the eyes open and eyes closed conditions, subjects were asked about the types of thoughts they were having while they were in these conditions. These thoughts were recorded and later coded into 5 general categories:

1. Relaxed- Feeling relaxed, thinking about nothing, meditating, dreaming....
2. Noises- Being aware of, and listening to, room noises...
3. Worry- Worrying about the experiment, eye movements, breathing, etc...
4. Concentrate- Fully concentrating on only the asterisk, "it looked like a....."
5. Outside- Thinking about outside, work, car, groceries, school, children etc...

Eyes Open

As illustrated in Table 9, a greater proportion of the ADHDhy subjects (11 of 25) reported worrying about the experiment than either the ADHDpi (6 of 17) or control subjects (12 of 32) while a greater proportion of ADHDpi subjects (9 of 17) reported thinking about the fixation point compared to either the ADHDhy (7 of 25) or control subjects (12 of 32). Finally, a greater proportion of control subjects (11 of 32) reported thinking about the outside world than either the

ADHDhy (5 of 25) or the ADHDpi subjects (0 of 17).

Table 9

Distribution of the "Type of Thought" Reported by Different Groups Following the Eyes Open Condition

Type of Thought*		Control	ADHDhy	ADHDpi
1	Relaxed...	1	1	2
2	Noises...	0	1	0
3	Worry...	8	11	6
4	Concentrate...	12	7	9
5	Outside...	11	5	0

Note* See task results, Eyes Open and Eyes Closed section.

Eyes Closed

At the end of the eyes closed condition subjects were asked about the types of thoughts they were having while they sat with their eyes closed. As presented in Table 10, a greater proportion of control subjects (8 of 32) reported feeling relaxed than either the ADHDhy subjects (3 of 25) or the ADHDpi subjects (1 of 17). Notably, a greater proportion of the ADHDhy group (11 of 25) and the ADHDpi group (7 of 15) continued to think about the asterisk than the control group (11 of 32).

Table 10

Distribution of "Type of Thought" Reported by Different Groups Following the Eyes Closed Condition

Type of Thought*		Control	ADHDhy	ADHDpi
1	Relaxed...	8	3	1
2	Noises...	2	1	0
3	Worry...	6	8	7
4	Concentrate...	11	11	7
5	Outside...	4	1	2

Note* See task results. Eyes Closed section

TOVA

The TOVA diagnoses for the ADHD subjects were generally consistent with the diagnosis the subject had received from their physician (74 of 84). For control subjects the diagnoses were not as consistent as the ADHD group. Many of the control subjects were given a diagnosis of ADHD by the TOVA (24 of 67). Later it was discovered that many of the control subjects, whom had been diagnosed as ADHD by the TOVA, had entered the study in order to investigate suspicions of attentional problems.

Reading Printed Text

After five minutes the subjects were told to stop reading and the number of words that had been read were counted. The subjects were then asked five general questions about the story. Separate t-test were performed between each of the groups using the number of words recalled as the dependent variable. The ADHDhy subjects read significantly more words (mean = 952, SD = 304) than the

ADHDpi subjects (mean = 672, SD = 263), t (40) = 3.16, p = .003. The control subjects read an average of 829 words (SD = 212). The control subjects correctly answered an average of 3.8 questions (SD = .8), the ADHDhy group correctly answered an average of 3.8 questions (SD = .8), and the ADHDpi group correctly answered an average of 3.7 questions (SD = .8). There were no statistically significant differences among the groups on the number of questions correctly answered.

Mental Rotation Tasks

During the mental rotation tasks, subjects were presented with two images on a monitor. The subject had to mentally rotate the image on the right side and determine whether this image was a identical match or mirror image of the image on the left side. A one-way between subjects ANOVA using the percentage of correctly identified images as the dependent variable showed that there were no statistically significant differences between the percentage of correctly identified images among the control, ADHDhy and ADHDpi groups in either the first or second of the mental rotation tasks (Table 11).

Table 11.

Percentage of Correct Responses to Targets in the Mental Rotation Tasks

Task	Control (±SD)	ADHDhy (±SD)	ADHDpi (±SD)
1st Rotation	95.3%(±4.4)	95.3%(±3.8)	94.9%(±7.4)
2nd Rotation	92.0%(±7.4)	88.8%(±12.2)	91.1%(±13.1)

Similarly, a one-way ANOVA using the time required to complete the task

as the dependent variable indicated that there were no statistically significant differences among the groups in the time required to complete the task (Table 12) in either the first or second mental rotation tasks.

Table 12
Time Required to complete the Mental Rotation Tasks (sec.)

Task	Control (sec) (±SD)	ADHDhy (sec) (±SD)	ADHDpi (sec) (±SD)
1st Rotation	306(±124)	338(±141)	297(±134)
2nd Rotation	308(±123)	330(±150)	335(±172)

One-way ANOVAs using groups as the between subjects factor were then performed at each of the degrees of rotation (i.e., 0°, 30°, 60°, 90°, 120°, 150°, and 180°) using the time required to complete the rotation as the dependent variable. There were no statistically significant differences between the groups in either the first or second of the tasks.

Paired t-test analyses between the 0° and 30°, 30° and 60°, 60° and 90°, 90° and 120°, 120° and 150°, and 150° and 180° degree rotations using the RTs at each degree of rotation as the paired subject variable were then performed. During the first mental rotation tasks, there were statistically significant differences between the 0° and 30° rotations ($t(30) = 2.04$, $p = .049$) and 30° and 60° ($t(30) = 5.13$, $p < .0005$) rotations for the control group; between the 30° and 60° ($t(24) = 5.90$, $p < .0005$) rotations for the ADHDhy group; and between the 30° and 60° ($t(16) = 2.79$, $p = .013$) rotations for the ADHDpi group (Table 13).

Table 13

Time Required to Respond to Target Based on Angle of Rotation in the First Mental Rotation Task

Degrees of Rotation	Control (ms) (±SD)	ADHDhy (ms) (±SD)	ADHDpi (ms) (±SD)
0	3316(±1444)	3179(±1268)	3183(±1290)
30	3001(±1087)	2957(±935)	3409(±2071)
60	3585(±1434)	3812(±1437)	3951(±1863)

During the second mental rotation task there were statistically significant differences in the 0° and 30° ($t(30) = 3.05$, $p = .005$), 60° and 90° ($t(30) = 5.56$, $p = .005$), 90° and 120° ($t(30) = 2.50$, $p = .018$), and 120° and 150° ($t(30) = 3.47$, $p = .002$) rotations for the control group; between the 0° and 30° ($t(24) = 3.98$, $p = .001$), 60° and 90° ($t(24) = 3.85$, $p = .001$), and 120° and 150° ($t(24) = 2.36$, $p = .027$) rotations for the ADHDhy group; and between the 0° and 30° ($t(16) = 2.19$, $p = .043$) and 60° and 90° ($t(16) = 4.05$, $p = .001$) rotations during the second mental rotation task. In general, as the degrees of rotation increased the time required to perform the task also increased (Table 14).

Table 14.

Time Required to Respond to Target Based on Angle of Rotation in the Second Mental Rotation Task

Degrees of Rotation	Control (ms) (±SD)	ADHDhy (ms) (±SD)	ADHDpi (ms) (±SD)
0°	2216(±925)	1837(±846)	2136(±1201)
30°	2678(±1082)	2488(±717)	2597(±1465)
60°	2630(±866)	2730(±918)	2809(±1264)
90°	3475(±1384)	3756(±1574)	4060(±2074)
120°	3819(±1645)	3778(±1269)	4208(±2313)
150°	4348(±2122)	4207(±1471)	4705(±2981)
180°	4184(±1788)	4414(±1866)	4353(±2911)

Selective Attention Tasks

During the selective attention tasks subjects had to identify a target letter that was surrounded by distractor letters. The distractors letters were presented at spacings of .06°, 0.5°, or 1°. As the distractors were moved farther away from the target letter the RTs were expected to decrease. A one-way, between groups ANOVA using the time required to complete the task as the dependent variable showed that there were no statistically significant differences in the time required to complete the task in either the first or second selective attention task (Table 15).

Table 15

Time Required to Respond to Target Based on Separation of Distractors During the First and Second Selective Attention

Task	Degrees of Separation	Control (ms) (±SD)	ADHDhy (ms) (±SD)	ADHDpi (ms) (±SD)
1st Selective Attn.	0.06	985(±217)	1067(±217)	1161(±217)
	0.5	847(±217)	841(±217)	957(±217)
	1.0	865(±217)	872(±217)	1030(±217)
2nd Selective Attn.	0.06	871(±217)	978(±217)	1016(±217)
	0.5	752(±217)	768(±217)	804(±217)
	1.0	770(±217)	787(±217)	838(±217)

A between groups, one-way ANOVA using the number of correct responses as the dependent variable showed that there were no statistically significant differences in the number of correct responses in either the first or second selective attention task (Table 16).

Table 16

Percentage of Correct Responses to Targets in the Selective Attention Tasks

Task	Control (±SD)	ADHDhy (±SD)	ADHDpi (±SD)
1st Select	96.1%(±1.2)	96.2%(±1.7)	96.3%(±4.3)
2nd Select	98.7%(±1.7)	97.9%(±2.6)	98.7%(±1.6)

An one-way ANOVA at each of the 1° , 0.5°, and .06° distractor spacings using RT as the dependent variable was then performed. During the first selective attention task at the 1° spacing there was a statistically significant difference

between the groups (F (2,70) = 3.32, p = .042). During the second selective attention task at the .06° spacing there was also a statistically significant difference between the groups (F (2,70) = 3.53, p = .035). None of the Bonnferroni adjusted individual comparisons were statistically significant.

Paired t-tests were then performed for each group using the RTs between the .06° and 0.5°, and the 0.5° and 1° distractor spacings as the dependent variables. In the control group there were statistically significant differences in the RTs between the .06° and 0.5° distractor spacings in the first (t (30) = 6.43, p = .0005) and second (t (30) = 7.81, p = .0005) selective attention tasks. The results were similar for the ADHDhy group with statistically significant differences in the RTs between the .06° and 0.5° distractor spacings in the first (t (24) = 5.88, p = .0005) and second (t (24) = 7.50, p = .0005) selective attention tasks. The ADHDpi group also had significant differences in the RTs between the .06° and 0.5° distractor spacings in the first (t (16) = 2.87, p = .017) and second (t (16) = 3.84, p = .023) selective attention tasks but also a significant difference between the 0.5° and 1° distractor spacings in the first selective attention task (t (16) = 2.53, p = .023). In general, as the distractors were moved farther apart, the RTs dropped. The results of the selective attention tasks are summarized in Table 15.

Listening Task

Following the presentation of the story on audiotape, subjects were asked five questions about the story. The control group correctly answered an average of 4.4 questions (SD = .7), the ADHDhy group correctly answered an average of

4.5 questions (SD = .7), and the ADHDpi group correctly answered an average of 4.6 questions (SD = .6). A one-way ANOVA with groups as the between subjects factor was performed using the average number of correct responses from each group as the dependent variable. There were no significant differences between the number of correct answers for the control, ADHDhy, and ADHDpi subjects.

Computer Reading

In this task subjects read text that was presented on a computer monitor. Time to complete the text varied significantly between the ADHDpi (mean = 214 sec, SD = 82 sec) and control groups (mean = 160 sec, SD = 42 sec)(t (47) = 2.345, p = .030). There were no statistically significant differences between the controls and ADHDhy subjects (mean = 155 sec, SD = 55 sec).

CHAPTER 5

DISCUSSION

Introduction

The aim of this study was to determine whether the EEG abnormalities observed in children with ADHDhy and ADHDpi continue into adulthood, to develop discriminate EEG criteria for ADHDhy and ADHDpi adults and to provide descriptive information concerning any EEG differences that may exist between ADHDhy and ADHDpi adults. In addition, to assist in providing more general information about individuals with attention deficits, the ADHDpi the ADHDhy groups were combined to form a 'CLINICAL' group. Finally, an important goal of this study was to determine the appropriateness of using EEG neurofeedback equipment for the treatment of ADHD and to aid the clinician in the selection of the most useful sites and bands in which to conduct neurotherapy.

The general results from the different analyses indicate that the qEEGs of ADHD adults are significantly different from that of normal adult subjects. Compared to the control group, adults with ADHD have higher T/B ratios, higher percentage levels of theta and delta wave activity, and lower percentage levels of beta wave activity. Although the magnitude of the differences between the groups is less than that reported for children with ADHD, the accuracy of the discriminant function analyses indicates that the EEG could be used to assist in diagnosis. Furthermore, that the ADHD adults continue to have some of the qEEG abnormalities observed in childhood suggests that neurofeedback, either alone or

in an adjunctive capacity, could be an effective treatment for adults with attentional difficulties.

Due to the exploratory nature of this research and the non-experimental design, it is difficult to arrive at any firm conclusions regarding the cause of the differences between the adult groups. It is plausible, however, that maturation played an important role in the differences observed in the present research. For this reason, the developmental EEG data provided by Gasser et al. (1988) and Matsurra et al. (1985) are used as a framework with which to integrate, compare, and contrast the results of this study.

The discussion is presented in sections corresponding to the analyses performed in the Results sections. Within each of the sections the research groups are discussed separately and, when appropriate, compared and contrasted with the other groups or previous research.

Task Related Differences

Throughout all the tasks, the statistically significant differences between the control group and the ADHDhy and ADHDpi groups are the result of higher T/B ratios and higher percentage levels of delta and theta band activity for the ADHD group but higher percentage levels of beta1 and beta2 for the control group. Task demands had a consistent influence on EEG activity. The highest levels of theta activity and highest T/B ratio but the lowest levels of beta1 and beta2 activity were observed in the selective attention and mental rotation tasks. The different levels of activity during the various tasks suggest that the increases and decreases

in activity are due to the particular task being performed.

The reasons for the changes in EEG activity during the selective attention and mental rotation tasks are difficult to interpret. What may set them apart from the other tasks is that they are both novel and difficult. For example, while the reading and listening tasks are both difficult they are not particularly novel whereas the T.O.V.A., although novel, is not difficult.

It would be expected that the pattern of increases and decreases in theta, beta1, and beta2 activity would be more similar between the ADHDhy and ADHDpi groups than with the control group; however, the pattern of correlations between the groups shows the strongest relationship between the ADHDhy and control groups, the ADHDpi and controls, and the ADHDhy and ADHDpi groups, respectively. The fact that the ADHDpi and ADHDhy groups are more highly correlated with the control group than with each other may indicate that ADHDhy and ADHDpi are distinct disorders with more qEEG commonalities with the control group than with each other.

In summary, the increases and decreases of theta, beta1, beta2, and T/B ratio activity indicate a consistent pattern of responses with all three groups showing an increase or decrease in activity based largely on task type. Compared to the control group, the ADHDpi and ADHDhy groups both begin and continue to maintain a higher percentage of theta band activity, whereas the control group has higher percentages of beta1 or beta2 activity that are maintained at a higher level throughout the length of the study. As beta activity is commonly associated

with cognitive activity and theta band activity is often associated with being tired and sleepy. The results of this study suggest that control subjects are more cognitively active and less tired than the ADHD subjects throughout.

Discriminant Function Analyses

The discriminant function analyses provide classification rates that correctly identify an average of approximately 8 out of 10 of the control or ADHD subjects. These results compare favourably with those reported elsewhere in the literature (Chabot & Serfontein, 1996; Clarke et al., 1998; John et al., 1988) and exceeds those reported by Mann et al. (1992). Some of the tasks, however, are better suited to eliciting a pattern of qEEG activity that produces higher correct classification rates. For example, in the ADHDhy group the performance of the T.O.V.A. provides a discriminant function that correctly classifies approximately 9 out of 10 of the subjects in the control and ADHDhy group. In point of fact, the third quarter of the T.O.V.A. is the most effective discriminant function of the ADHDhy group and the best overall function of all three groups. The high target to foil ratio of the third quarter of the T.O.V.A. follows the low target to foil ratio of the first half of the test, hence it is unclear whether the 'change up' to a higher rate of target presentations, the higher rate of presentations, or a combination of these factors, plus others such as fatigue, contributed to the strength of the function. The effectiveness of the third quarter of the T.O.V.A. with the ADHDhy group is not surprising, however, as both the third and fourth quarters of the T.O.V.A. are designed to detect impulsivity, one of the defining features of

ADHDhy.

The highest proportion of the discriminant function's variables, in the third quarter of the T.O.V.A., are in the alpha band and located over the frontal area. The activity from the right central (C4) alpha variable, however, is the discriminant variable with the highest pooled within groups canonical correlation. Research with normal (Zametkin et al., 1990;1993) and brain damaged (Meslaum, 1986) subjects has shown the importance of the frontal lobes in response inhibition. The higher proportion of discriminant variables in the frontal area during a task that specifically requires response inhibition may indicate an important relationship between this region, response inhibition and the alpha band activity in ADHD adults. Furthermore, due to the difficulty that ADHDhy adults experience with impulsivity, the frontal area may be particularly important to target for neurotherapy.

A topographic map of the EEG activity occurring during the third quarter of the T.O.V.A. is presented in Figure 5. The maps were derived from two individual subjects with high discriminant probability values ($p > .90$) of belonging to either the ADHDhy or control group. Each of the following topographic maps shows the μV amplitude of peak-to-peak EEG activity in each band, relative to the other two bands. A comparison of the maps clearly shows the excess theta activity in the central region and the reduced levels of alpha activity that are associated with ADHDhy.

In conjunction with the T.O.V.A., the alpha based qEEG discriminant

function of the ADHDhy subjects could be a highly effective diagnostic tool for researchers and clinicians. For example, following a brief baseline measure taken during the eyes open and eyes closed conditions, a client would be asked to perform the T.O.V.A., then the results from the qEEG analysis and the T.O.V.A. used to further refine the clinician's or researcher's diagnosis.

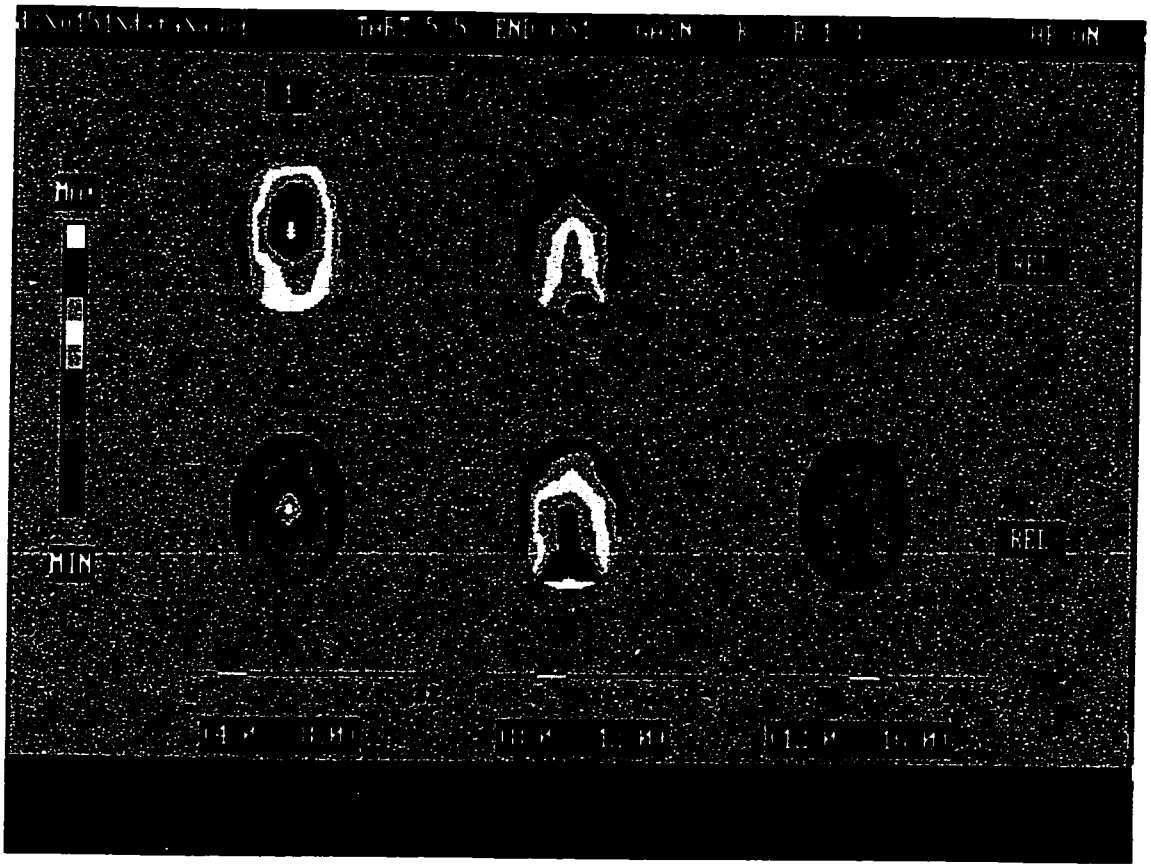

Figure 5. Relative levels of theta (Band 1), Alpha (Band 2), and SMR (Band 3) activity during the performance of the third quarter of the T.O.V.A. with violet to blue areas indicating relatively lower levels of activity and red to white areas indicating higher levels of activity. The control subject's data is in the lower row and the ADHDhy subjects data in the upper row.

For the ADHDpi subjects, the first selective attention task and the T.O.V.A. are both tasks that generate the highest correct classification rates, an

average of approximately 8 out of 10 subjects. Of these two tasks, the first selective attention task, with the highest correct classifications, appears to be the most effective at discriminating between the groups. The discriminant variables with the highest correlations with the discriminate function, during the selective attention task, were in the left hemisphere, occipital or frontal area, and based on the T/B ratio and theta band activity. The unique qualities of this task, such as the self controlled rate of target presentation and the use of distractor letters to interfere with processing, may be responsible for the success of the discriminant function. The selective attention task, however, occurs approximately 40 minutes into the testing. This suggests that mental fatigue may also differentially affect the ADHDpi group's performance and contribute to the success of this function. Future research that varies the time at which the selective attention task is presented could assist in clarifying the role of fatigue in the results of the present research.

A topographic map of the EEG activity occurring during the selective attention task is presented in Figure 6. The maps were derived from two individual subjects with high discriminant probability values ($p > .87$) of belonging to either the ADHDpi or control group. The low levels of beta activity in the occipital area and the excess theta in the central region are apparent for the ADHDpi subject.

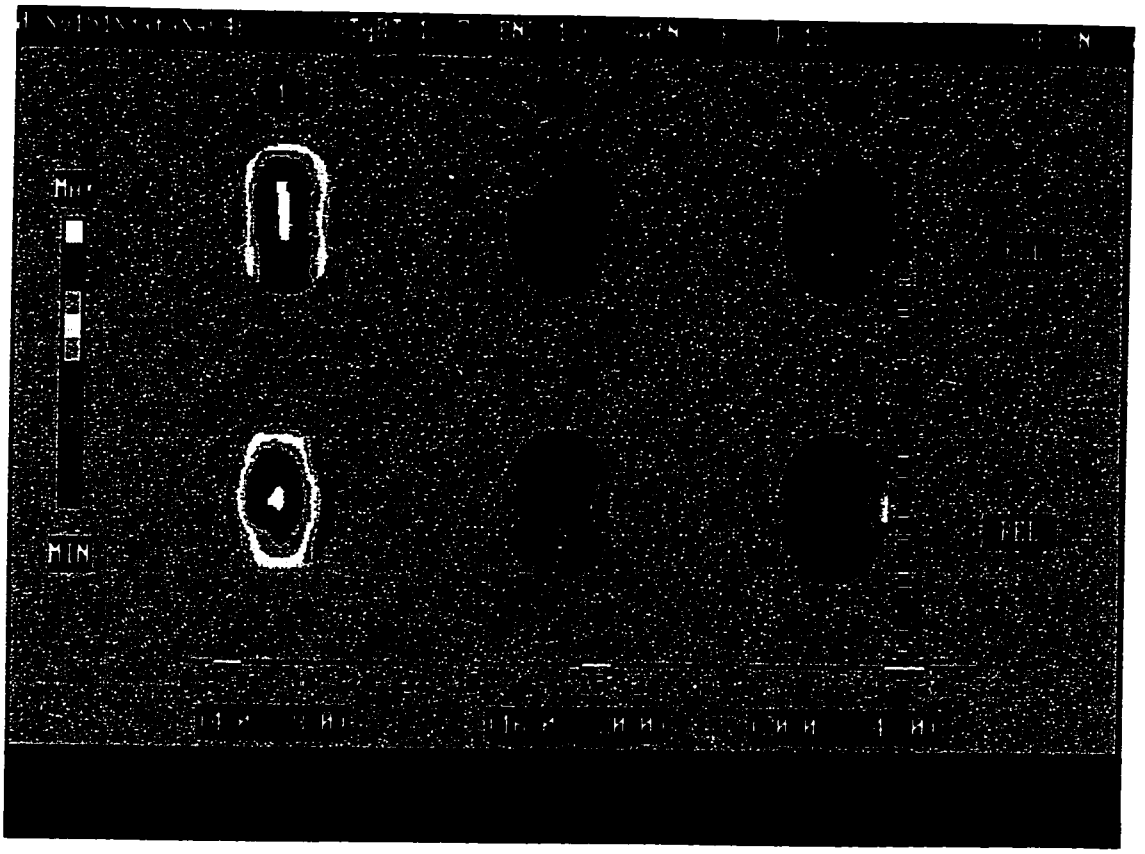

Figure 6. Relative levels of theta (Band 1), Beta1 (Band 2), and Beta2 (Band 3) activity during the performance of the selective attention task. The control subject is in the lower row.

When using the EEG to determine the clinical status of a patient, the selective attention task may not be as useful as the T.O.V.A. due to the length of time that elapsed before its presentation, the variety of tasks that were performed before it was used and because the results from the TOVA can also be used to assist in a diagnosis. When the goal is classifying individuals in a clinical setting, a more useful approach might be to use the T.O.V.A.. The second quarter of this task provides a discriminant function with a slightly lower classification rate but with a higher eigenvalue and canonical correlation than the selective attention task.

It is not surprising that the second quarter of the T.O.V.A. provides one of

the best classification rates, as the first and second quarters are both designed to detect inattention, a major distinguishing feature of the ADHDpi group. Interestingly, the fact that the performance of the T.O.V.A. generates the best discriminant functions for the ADHDhy and the ADHDpi groups, during parts of the test that are designed to separate the groups, perhaps illustrates the sensitivity of the relationship between task type and EEG activity, i.e., the qEEG appears to be effective at detecting differences in cognitive activity that are associated with each group.

In the CLINICAL group, the performance of the T.O.V.A. and the listening task both provide discriminant functions with high classification rates; however, the performance of the third quarter of the T.O.V.A. produces the most effective discriminant function for these subjects through the T/B ratio, beta1, beta2, theta, and delta band variables in the frontal and parietal areas. A major demand of this task is to inhibit responses, hence, the results suggests a relationship between beta band activity, the frontal lobes, and response inhibition. A topographic map of the EEG activity occurring during the third quarter of the T.O.V.A. is presented in Figure 7. The maps were derived from two individual subjects with high probability values ($p > .90$) of belonging to either the CLINICAL or control group. The T/B ratio is of particular importance in the determination of group membership with the CLINICAL and control group as the control subject has levels of theta that might suggest he is a member of the CLINICAL group. It is important to note, therefore, that the control subject also

has correspondingly higher levels of beta1 activity.

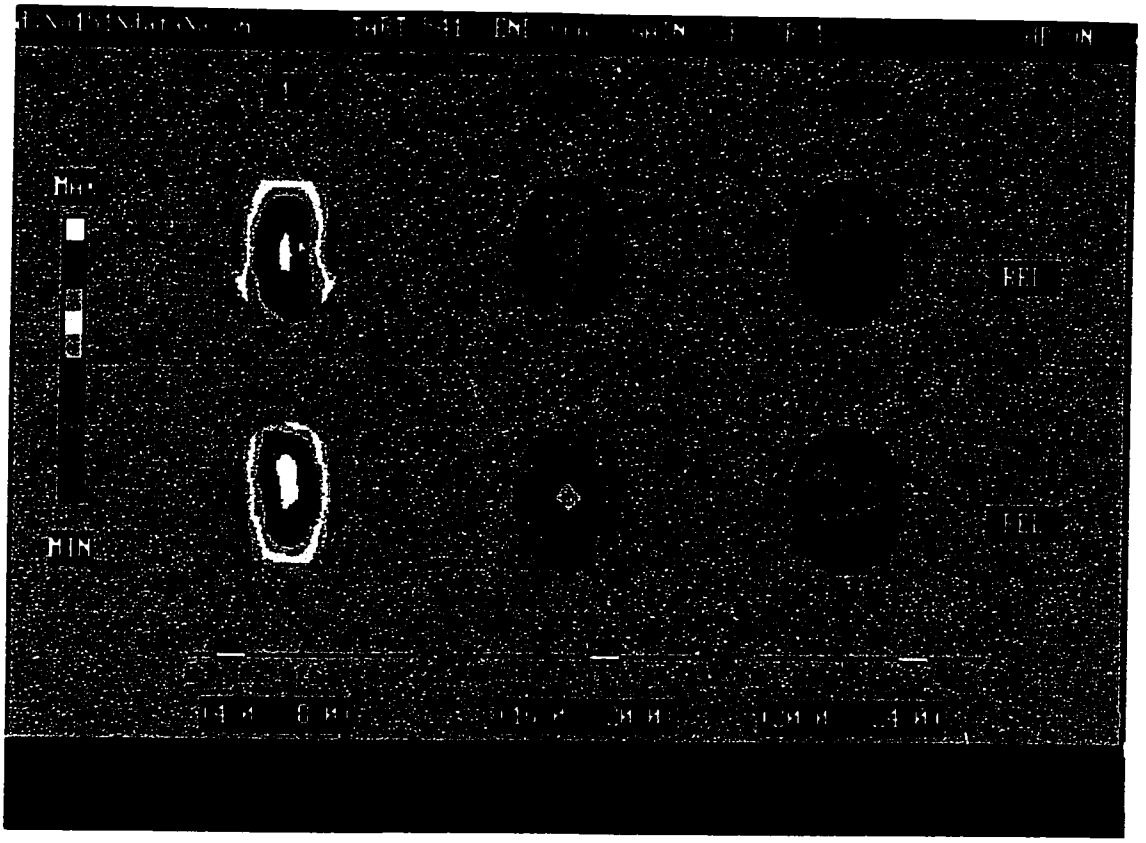

Figure 7. Relative levels of theta (Band 1), Beta1 (Band 2), and Beta2 (Band 3) activity during the performance of the third quarter of the T.O.V.A. The control subject is in the lower row and the ADHDhy subject in the upper row.

The overall correct classification rate for the CLINICAL group is less than the rates for the ADHDhy or ADHDpi groups but perhaps useful as an initial screening tool to assist in determining if a client is presenting with attentional problems. Following the screening, using the results of the qEEG discriminant and the T.O.V.A., additional analyses, using the qEEG discriminant functions for the ADHDhy and ADHDpi classifications could be employed to more precisely determine group membership.

Attempting to discriminate between the three groups was less successful as

only an average of 5 out of 10 of the ADHDhy, ADHDpi and control subjects were correctly classified. Chabot and Serfontein (1996) report similar but slightly higher average rates, approximately 6 out of 10 of the children in their study were correctly classified. They note, however, that most of the qEEG variance is accounted for by the normal/ADHD dimension and not by the ADHDpi and ADHDhy factor. This would also appear to be the case with the adult ADHD subjects as, within each of the tasks, some of the ADHDhy subjects are usually misclassified as ADHDpi subjects, and vice versa. The classification rates during the three group discriminant function are statistically significant and higher than the average rate of 3 out of 10 correctly classified that would be expected by chance but questionable for use in a diagnostic capacity.

The misclassifications from the discriminant function analyses are of particular interest as this research used mixed gender samples within each of the groups with a wider range of ages than is typically encountered in qEEG research. Princhep and John (1988) note that there is a risk in using subjects having different age ranges to construct norms because the obtained mean values may only be applicable to those individuals in the mid-portion of the age range. They further report that the standard deviation will be wider, compared to that of a more restricted age range, and that there will be a tendency for older subjects in one group and younger subjects in the other group to be misclassified. Overall, an average of approximately 8 out of 10 of the control group, 9 out of 10 of the ADHDhy group, and 8 out of 10 of the ADHDpi group were misclassified during

one task or another. In the functions noted in Appendix I, the average ages of the misclassified control, ADHDhy, and CLINICAL subjects are close to the average age of the subject's group suggesting that within each group the EEG activity is relatively similar. The ADHDpi misclassifications, however, do appear to be older than the average age of the correctly classified ADHDpi subjects. This is perhaps due to the fact that a higher proportion of ADHDpi subjects are over the age of 30 years, 10 out of 17 Based purely on chance one would expect more of these older subjects to be misclassified. Interestingly, none of the females were misclassified in the ADHDhy group, perhaps indicating that the qEEG activity of females with ADHDhy is closer to the average ADHDhy subject's qEEG profile.

In summary, the discriminant function analyses produced a number of functions with classification rates well above chance and effective enough to be used to assist in the diagnosis of attentional difficulties in general or ADHDhy or ADHDpi specifically. The T.O.V.A. appears to be particularly effective in eliciting EEG activity that can be used effectively in discriminant function analyses. The specificity of the test's design in evoking differential EEG activity in different clinical groups also lends support to the use of on-task EEG testing in diagnosis. The fact that the discriminant functions were effective, in spite of the small groups sizes, mixed genders and the wide age range of subjects, suggests that the differences between the groups are substantial. Future research using increased group sizes within narrower age ranges and all male or female groups would increase precision in classification and eventually replace the discriminant functions

developed in the present research.

Inter-hemispheric Activity

The comparison of inter-hemispheric EEG activity during the various tasks used a between groups analysis of the ratio of EEG activity in each of the frequency bands in each of the tasks as well as paired site comparisons. Analyses of this type can be useful in highlighting general differences between the groups.

Overall Differences in Inter-hemispheric Activity

The only statistically significant between groups difference indicates that the ADHDpi subjects have small but significantly increased levels of delta band activity over the left hemisphere during the second mental rotation task. The higher percentages of delta wave activity in the ADHDpi groups is difficult to interpret but is consistent with the cognitive difficulties reported for these individuals, i.e., increased delta is often interpreted as showing decreased arousal (Tansey & Bruner, 1983; Lubar & Lubar, 1984). These differences between groups, however, may in fact be due to different strategies employed by members of each group. For example, when comparing the images in the mental rotation task, some subjects reported mentally overlapping the images or mentally 'flipping' them while others reported that they "just knew" intuitively by looking at them. In spite of the fact that all of the subjects were given the same instructions on how to perform the mental rotation task, the differences illustrated in the example above suggest that different strategies actually employed by the subjects may have been partially responsible for the differences between the groups. Unfortunately, the

strategy employed by the subject was not systematically recorded, thus it cannot be determined if differences across groups are due to the strategy that was employed. Future research that closely monitors these strategies could clarify the differences observed in the present research.

Although the greater percentage levels of delta band activity over the right hemisphere for the ADHDpi subjects during the second mental rotation task are statistically significant, the analyses of the on-task results showed that there are no statistically significant differences between the groups on either the response times or the error rates while performing this task. This suggests that qEEG differences are not always indicative of statistically significantly poorer task performance.

Within Groups Differences in Inter-hemispheric Activity

The analyses of differences in left and right hemispheric activity within each group reveal that there are statistically significant differences in the ADHDhy and ADHDpi groups, but not in the control group. The ADHDhy group differences occur during the third quarter of the T.O.V.A., the eyes closed, and second mental rotation tasks. The lower levels of beta1 activity in the left hemisphere during the third quarter of the T.O.V.A. are difficult to interpret but are consistent with the use of this quarter for the diagnosis of ADHDhy; perhaps suggesting that beta1 activity plays an important role in response inhibition.

During the second mental rotation task for the ADHDhy group there are higher percentages of delta and alpha band activity in the right hemisphere. Again, interpretations are difficult to develop, but as there are no significant differences in

the control or ADHDpi group, these results may indicate a different strategy being employed by these subjects. The analysis of task performance, however, showed no statistically significant differences in the time required to perform the task or the number of errors committed during the task compared to the other two groups. Again, this suggests that higher levels of delta and alpha band activity will not always equate to measurable differences in performance.

The differences in hemispheric activity during the eyes closed condition for the ADHDhy subjects are of a higher T/B ratio and alpha band activity in the right hemisphere and lower beta1 and beta2 activity in the left hemisphere. These differences are difficult to resolve but perhaps indicate different types of cognitive activity than either the ADHDpi or control subjects while in a resting condition. For example, based on the questions asked following the eyes closed condition, a greater proportion of ADHDhy subjects continued to think about the asterisk during the eyes closed condition while a greater proportion of control subjects reported being relaxed. These different types of cognitive activity may have been responsible for the differential levels of left versus right EEG activity observed in the ADHDhy group.

The ADHDpi group displayed significantly higher levels of theta band activity in the left hemisphere during the computer reading task. The left hemisphere plays a central role in language processing and the attentional requirements necessary to perform a task such as reading, hence the increased levels of theta may be associated with a particular reading strategy employed by

this group. The analysis of the reading speed showed that the reading times for the ADHDpi groups were significantly slower than the control group.

In summary, there was only one significant between groups difference in inter-hemispheric activity, the ADHDpi group during the mental rotation. This finding was, however, consistent with previous research showing higher levels of slow wave activity ADHD children. In the within groups comparisons of inter-hemispheric activity there were no statistically significant differences in the percentages of EEG activity between the left and right hemispheres for the control group. There were, however, three differences observed for the ADHDhy group and one noted for the ADHDpi group. Notably, the differences were in the frequency bands commonly associated with ADHD in children. These EEG differences may be the result of different strategies employed by these subjects or a result of changes in EEG activity in response to processing demands.

Regional Activity

In the regional analyses, the surface of the scalp was divided into frontal, central, posterior, left temporal, and right temporal areas. An analysis of between groups differences for all five regions was then performed for each of the frequency bands and each of the tasks. The regional comparisons provide a more detailed view of the differences between the groups than is available from the inter-hemispheric comparisons. These comparisons reveal statistically significant differences in the T/B ratio in the right temporal and frontal areas for the ADHDhy group compared to the control group during the eyes closed condition. There

were no inter-group differences in the more cognitively demanding tasks or the eyes open condition. This suggests that when ADHDhy subjects are forced to maintain attention and to carry out a task, regional EEG differences dissipate.

A problem with the eyes closed condition is that there is little control over the subject's mental state. For example, based on reports of what the subject was thinking about during the eyes closed condition, a greater proportion of the control subjects report being relaxed than the ADHDhy subjects. In the ADHDhy group, however, a greater proportion of subjects report continuing to concentrate on the afterimage of the asterisk viewed during the eyes open condition. The differences between the groups may have been partially, therefore, a result of the ADHDhy group's focusing of attention on the afterimage of the asterisk while the control subjects remained more relaxed.

The between groups regional differences observed during the commonly used eyes closed condition raises an important issue regarding qEEG research that uses this task to classify and identify disorders. Tansey et al. (1994) demonstrated that different thoughts are responsible for different levels of EEG activity in different bands. For example, a meditative state resulted in increased levels of alpha activity while focussing on the outside surface of the body resulted in increased beta activity. None of the EEG studies of ADHD children have reported their subjects' thoughts while in the eyes closed condition. As stated previously, being unable to account for the cognitive behaviour of subjects during a resting eyes closed, or resting eyes open condition, may introduce a confound into the

analysis. The solution would appear to be either specific instructions on what to think about and a careful review of a subject's thoughts following the eyes open or eyes closed condition or the use of standardized on-task testing. In the case of on-task testing, the performance of a task can at least be verified through a comparison of the results to a normative database and, if necessary, the subject's strategy for performing the task recorded.

In summary, there were only two statistically significant differences in regional activity among the three groups. These differences, however, are consistent with previous research with children showing regional differences in an eyes closed condition.

Planned T-test Comparisons

Differences in Frequency Band Activity

The comparisons presented in this section are based on the planned t-test results obtained during the eyes closed condition (Appendix J).

Clinical Group

Beta Band

The electrode sites showing beta band differences in the ADHD adults are similar, but not identical, to those reported elsewhere in research that uses combined groups of ADHDpi and ADHDhy children in an eyes closed condition (Chabot & Serfontein, 1996; Clarke et al., 1998). Chabot and Serfontein (1996) report lower levels of beta band activity in the ADHD group, in the frontal and temporal areas, whereas the adult CLINICAL subjects present with differences

primarily in the frontal area.

Interestingly, the reduction in the number of beta1 and beta2 differences in all but the frontal area, between ADHD children and the CLINICAL adults, is consistent with the pattern of EEG changes that occur during the normal aging process (Gasser et al., 1988; Matsurra et al., 1985). In normal subjects, these researchers report increases in alpha, SMR, and beta band activity that occur earliest in the central, followed by parietal, occipital, and frontal areas. The presence of statistically significant differences in only the frontal region suggests that the CLINICAL group subjects have reached normal adult levels of beta band activity in the central, parietal, and occipital regions. Furthermore, the beta band differences appearing in the area predicted to mature last, i.e., the frontal region, suggest that the posterior to anterior maturation predicted by Gasser et al. (1988) and Matsurra et al. (1985) may have occurred in this group. If the lack of differences in all but the frontal area are a result of maturational processes then the CLINICAL group may have experienced normal, but delayed, changes in beta band activity. Regardless of the cause, the lack of significant differences in all but the frontal region, may signal an improvement in the baseline levels of cognitive functioning for the CLINICAL group.

Theta Band

The theta band differences between children with ADHD and control subjects are localized in the frontal region (Chabot & Serfontein, 1996; Clarke et al., 1998). The CLINICAL group subjects, however, have higher percentage

levels of theta band activity than the control group at all of the electrode sites, suggesting that, compared to ADHD children, the CLINICAL adults experience an increase in the range of sites affected by excess theta. The comparative increase in the number of sites affected by excess theta is perhaps due either to the CLINICAL group having more theta or the control group having less theta. Interestingly, the latter of these two possibilities fits well with maturation that occurs in a normal but delayed fashion for the CLINICAL group. Gasser et al. (1988) and Matsurra, et al. (1985) point out that theta band activity declines with age. Therefore, if the control group has experienced a normal reduction in theta band activity, then the increase in the number of sites at which there were statistically significant theta band differences could be the result of decreased levels of theta in the control group, rather than increased levels in the CLINICAL group.

If maturation is not the cause of the increase in the number of areas with excess theta activity, then there may have been a worsening of the condition as defined by EEG parameters. This seems unlikely, however, as the CLINICAL group has raw μV and percentage levels of theta activity that are only 10% to 12% higher than those of the control group, compared to ADHD children who reportedly have 300% to 500% more theta band activity than controls.

Delta Band

In the delta band, the differences between normal and ADHD children that are characterized as 'scarce' by Chabot and Serfontein (1996) and located primarily in the posterior area by Clarke et al. (1998), are evident at all of the sites

in the CLINICAL group. These results suggest that the excess delta band activity of ADHD children continues into adulthood and that it appears in a wider range of sites. The reasons for the relative increase in the number of sites affected by excess delta is unclear; however, it may be due to the same maturational effects hypothesized to be influencing the pattern of results in the theta band, i.e., normal changes in the control group but delayed maturation in the CLINICAL group. If delayed maturation is not a factor, then the increase in baseline levels of delta band activity may indicate more emotional difficulties and a wider range of cognitive problems than those experienced by children with ADHD. As noted previously in the theta band section, this hypothesis is unsupported by the changes in the raw μV levels of EEG activity.

Alpha Band

It is surprising that there are no differences between the CLINICAL and control groups in the alpha band considering the well documented presence of alpha band differences in children (Chabot & Serfontein; Clarke et al., 1998; Defrance et al., 1996). As noted earlier, Gasser et al. (1988) report a systematic decrease in slow wave activity and an increase in fast wave activity with increasing age, in particular with a decrease of theta wave activity and an increase of alpha wave activity. The present results support the hypothesis that adults with ADHD have experienced normal, albeit delayed, maturation and suggest and that the alpha band activity of the ADHD subjects has reached normal levels. A second possibility is that one or both of the ADHD groups are producing significantly

different levels of alpha band activity, e.g. very high and very low levels, and that by combining the groups the effects are canceled out. The analyses, however, failed to reveal any statistically significant differences between the groups, indicating that the alpha band differences reported by Chabot and Serfontein (1996) and Clarke et al. (1998), are not associated with the ADHDpi or ADHDhy dimension in ADHD adults. The lack of alpha band differences during the eyes closed condition suggests that the control and ADHD subjects are producing similar levels alpha wave activity.

SMR Band

There are no statistically significant differences in the percentage of SMR activity at any of the locations during the eyes closed condition. Comparisons of these results with previous research are difficult as other researchers (Clark et al., 1998) sometimes include SMR activity (12-16 Hz) under a broader beta1 band (13.5-20.5 Hz) and the only other research that used a 12-16 Hz SMR band (Janzen et al. 1995) reported results in raw μV and not in percentage values. These data and those from the alpha band comparison suggest that, compared to ADHD children, the CLINICAL group's levels of EEG activity between 8-16 Hz has increased to normal adult levels: A finding that fits with a normal but delayed increase in fast wave activity.

T/B Ratio

The higher percentage levels of theta band activity and the lower percentage levels of beta1 activity result in higher T/B ratios at all of the sites in

the CLINICAL group. These findings are consistent with research by Janzen et al. (1995), Lubar (1992), and Tansey (1995) showing ADHDpi and ADHDhy children with higher than normal T/B ratios; however, there are a wider range of sites affected by higher T/B ratios in adults. The importance of the ratio of activity between the beta1 and theta bands appears to be particularly salient in the case of on-task testing of ADHD subjects as increased beta and reduced theta is expected during the performance of cognitively demanding tasks. As Lubar (1992) notes, the presence of excess activity in one frequency band may be less important than the patterns of activity between the bands. It is difficult to determine the cause of the increase in the number of areas affected by higher T/B ratios without longitudinal data showing changes over time; this type of study would be invaluable to understanding the progress of this disorder.

In summary, the lack of significant differences in the alpha and SMR bands and the pattern of differences in the delta, theta, beta1 and beta2 bands may correspond to a developmental lag in children and adults with ADHD. For example, in the CLINICAL group, the lack of beta1 differences in the posterior area and the overall lack of differences in the alpha and SMR bands indicate that the brainwave activity between 8-16 Hz has reached normal adult levels and that beta1 activity is maturing in a normal posterior to anterior direction. The difficulty with this hypothesis is that there are no corresponding decreases in theta and delta band activity in the CLINICAL group's results. As noted earlier, however, this could be due to a normal decline in the control groups level of delta and theta

activity. If this were the case then a decline in delta and theta band activity in the CLINICAL group may not have occurred, with the result that they had relatively higher levels of theta and delta band activity. Although highly speculative, this would imply that the maturation of the faster bands in the CLINICAL adult occurs normally but at slower rate than normal adults, while theta and delta band activity appear to remain at stable levels or mature at a much slower rate.

Planned Comparisons with the ADHDpi Group

This section discusses the ADHDpi group's results compared to research performed by Mann et al. (1991) who recorded the EEG activity of ADHDpi children with their eyes open, during reading, and while drawing.

During the eyes open condition there are no statistically significant differences in any of the frequency bands for the adult ADHDpi subjects compared to the control group; results that are similar to those reported by Mann et al. (1991). The lack of differences in the eyes open condition appears to indicate that while the brain is "resting" there are no appreciable EEG differences between the activity of the ADHDpi group and control group. There are statistically significant differences in the T/B ratio but comparisons with Mann et al. (1991) were not possible as this researcher did not test for differences in the T/B ratio.

While performing the reading task, the ADHDpi adults have higher T/B ratios, higher levels of theta band activity, and lower levels of beta2 activity. As these differences are located only in the frontal area, however, increased eye movement might be responsible. Mann et al. (1991) also report absolute and

relative differences in the frontal area but attribute these to differences in eye movement between the groups; the same conclusions are drawn here.

The eyes open and reading tasks employed by Mann et al. (1991) are similar to those employed in this research. There is no drawing task in the present research, but as the mental rotation and drawing tasks are both performed primarily in the right hemisphere, the results from the first mental rotation task are substituted and compared in place of the drawing task. During the mental rotation task, the ADHDpi subjects had higher T/B ratios, higher percentages of delta and theta band activity and lower levels of beta2 activity mainly in the frontal areas, but also in the central, parietal, occipital and temporal regions. The lack of significant differences during the eyes open condition then the presence of statistically significant differences during this task supports the hypothesis that the performance of the mental rotation task was responsible for the changes in the ADHDpi groups' EEG activity. Unlike the reading task, there is little or no eye movement involved in the performance of the mental rotation, hence significant between groups differences are likely due to the performance of the task and not to eye movement. Mann et al. (1991) also report statistically significant differences in the frontal, parietal, central, and occipital areas, with generalized increases in slow wave activity in the frontal area and decreases in fast wave activity in the posterior and temporal areas.

While performing the mental rotation task, the increase in delta and the decrease in beta2, compared to the resting eyes open condition, suggests a similar

response to that reported in the Mann et al. (1991) research. The majority of the differences in the Mann et al. (1991) study are in the theta and beta1 range, whereas the differences in the adult ADHDpi group are in the delta and beta2 bands. The similarity of the regions affected during this task suggest that the ADHDpi adults continue to experience the qEEG differences noted in childhood but that there is a shift in the frequency bands that are affected. These changes may be the result of the particular demands of the mental rotation task or related to developmental changes. Future research using a drawing task could assist in clarifying the differences reported in the present study.

To summarize, the lack of statistically significant differences in the alpha, SMR, and beta1 bands suggests that ADHDpi adults may have reached normal adult levels of EEG activity at rest and during the performance of cognitively demanding tasks. The lack of differences in these bands also supports the hypothesis that maturational changes are responsible for the differences between the adults and children with ADHD and normal subjects.

Planned Comparisons with ADHDhy Subjects

During the eyes open condition, there are only a few statistically significant differences between the ADHDhy and control subjects in the beta1, delta, and theta bands but a high number of T/B ratio differences. The lower number of statistically significant differences and the lack of a clear pattern of differences in the frequency bands suggest that these results may be due to type I error. In a similar eyes open condition using regional comparisons with ADHDhy children,

Lazzaro et al. (1998) report decreased relative beta activity in the posterior region and increased alpha activity in the right hemisphere for the ADHDhy subjects. None of the differences reported by Lazzaro et al. (1998) are present in the adult group, either in regional, hemispheric, or t-test comparisons, suggesting that ADHDhy adults no longer have the significant differences in the beta or alpha bands that are apparent during the resting eyes open condition in children. Interestingly, the failure of Lazzaro et al. (1998) to detect statistically significant differences in the theta and delta bands suggests that the mechanisms that are responsible for this activity in ADHD children are not active during the resting eyes open condition.

The t-test comparisons of the T.O.V.A. reveal that for the ADHDhy group there are statistically significant differences in a range of electrode sites and frequency bands. During this task, the greatest number of significant differences occur during the second and third quarters in the T/B ratio, and the theta, delta, and beta bands. In the only other research using adult ADHDhy subjects during the performance of a task, Rasey et al. (1999) report statistically significant differences in the beta, theta, and alpha bands but only at the Fz, Cz, and Pz sites, respectively; these differences occur during the performance of the auditory component of the Intermediate Visual and Auditory Continuous Performance Task (I.V.A.). During the visual component of the I.V.A., the only statistically significant differences were in the theta band at the F3, C3, and Fz sites. Interestingly, the beta band differences in the frontal area during a task that

requires subjects to inhibit responses, is similar to the region and frequency band noted for the ADHDhy and CLINICAL subjects during the T.O.V.A..

The lower number of significant differences in the Rasey et al. (1999) study, compared to the results of the present study, could be due to methodological differences between the I.V.A. and the T.O.V.A.. For example, the auditory and visual components of the I.V.A. are only 6.5 minutes in length and each section was presented on different days. The majority of the statistically significant differences in the T.O.V.A. occurred during the second quarter of the test from 5.5 to 11 minutes, at which time the I.V.A. would have been completed. This suggests that the subjects in the Rasey et al. (1999) study may not have been attending for a long enough period of time to obtain the results observed during the T.O.V.A..

In summary, with respect to the ADHDhy subjects, the comparisons with previous research are inconclusive concerning the hypothesis of a maturational lag in the EEG development of ADHDhy subjects. The majority of the t-test differences in the T.O.V.A., however, occurred in the frontal and temporal areas with no significant differences in the parietal or occipital areas. These results fit with a posterior to anterior maturational process and suggest that the ADHDhy group may have experienced the same changes noted in the CLINICAL and ADHDpi groups.

A Comparison of the ADHDpi and ADHDhy groups

This section discusses the similarities and differences between the ADHDhy and ADHDpi groups based on the results of the planned t-test comparisons. The adult ADHD groups have a number of similarities such as the lack of significant differences with control subjects in the alpha and SMR bands, lower percentage levels of beta2 activity, higher T/B ratios, and higher percentage levels of delta and theta wave activity. The main difference between the ADHDhy and ADHDpi groups is in the beta1 band. The lack of beta1 differences for the adult ADHDpi group, but not for the ADHDhy group, suggests that from a maturational perspective the ADHDpi child's EEG may develop more rapidly than that of the ADHDhy child. In point of fact, the results of the planned t-test comparisons indicate that, by adulthood, the ADHDpi group's EEG has become statistically indistinguishable from the normal individual's EEG in the 8-20 Hz range. The ADHDhy group's EEG is indistinguishable from that of the control group in the 8-16Hz band but in the 16-20 Hz band there are significant differences between the control group in the frontal and temporal area. It is important to note that the ADHDhy group's beta1 differences are focussed in the frontal, central, and temporal areas, a pattern that fits well with the posterior to anterior maturation reported by Gasser et al. (1985).

It could be argued that the lack of significant differences in the beta1 band for the ADHDpi group could be the result of increased EMG activity affecting this

group's beta2 activity. This explanation seems unlikely, however, as there were no statistically significant differences in relative levels of EMG activity between the control and ADHDpi subjects at either of the artifacting leads. There are statistically significant differences in EMG activity between the ADHDhy and control groups during the eyes open, eyes closed, first quarter of the T.O.V.A., and the second mental rotation task. In all cases, the percentage levels of EMG activity were lowest for the ADHDhy group and highest for the control subjects. These findings appear to confirm Satterfield's (1973) observations of under-arousal and extend these findings into an adult ADHDhy population. The results also suggest that in the aforementioned tasks the beta2 differences between the control and ADHDhy groups may have been a result of higher, but normal, levels of EMG in the control group.

In summary, explanations for the differences between the control group and the ADHDpi and ADHDhy groups are difficult to generate due to the unequal sample sizes, mixed genders, and the wide range of ages in all three groups. Compared to the ADHDpi group, however, the pattern of differences for the ADHDhy group is a better fit with the developmental lag model, i.e., there are no differences in the SMR or alpha bands, delta is reduced, theta and beta1 are primarily frontal, indicating posterior to anterior maturation, and beta2 is reduced. Furthermore, the correlations reported during the performance of the tasks indicates that these two groups react in a more similar fashion than either the

control and ADHDpi group or the ADHDhy and ADHDpi groups.

The ADHDpi groups results are also supportive of a maturational lag with no significant differences in the alpha, SMR, or beta1 bands. Unlike the ADHDhy group, however, the beta2 activity is widespread and does not indicate any posterior to anterior maturation and the pattern of correlations, although significant, are weaker than those of the ADHDhy group.

CLINICAL SECTION

Neurotherapy Site and Frequency Band Selection

The purpose of this section is to provide the clinician with the electrode sites and frequency bands that are most useful for neurotherapy work with ADHD adults. The sites and bands suggested are provided with the full recognition of the importance of individual differences between patients. They may, however, be useful to clinicians who do not have the equipment necessary or the time required to perform an analysis of all 19 electrode sites. The strategy adopted for selecting an appropriate frequency band or electrode site at which to provide neurotherapy is based on the patterns of differences between ADHDhy, ADHDpi, and normal subjects over the course of the tasks and conditions described in the Methods section. As previously described, the number of statistically significant differences for each of the sites, frequency bands and ratios, as well as comparisons of the overall percentage levels of activity, aid in the selection of the most advantageous location or band to target for neurofeedback.

Band Selection-ADHDpi Group

Children with ADHDpi or ADHDhy are reported to have 3 to 5 times as much theta wave activity as the normal child, hence treatment protocols often focus on the reduction of this activity (Lubar, 1992; Tansey 1993). The results of the present research indicate that the theta band differences between normal and ADHD adults, though highly significant, are in the order of only 8% to 12%. In

addition, the widespread and highly significant differences in the T/B ratio, more than in any one of the frequency bands, indicates that in order to be effective, more than just the theta band should be targeted.

The lack of statistically significant differences in the beta1, SMR, and alpha bands during any of the tasks suggests that ADHDpi subjects have reached normal adult levels in these frequency bands. The highest number of statistically significant differences occurs in the theta band which indicates that this frequency should be a focus for neurofeedback treatment. However, many of the highly significant differences, $p < .01$, in the ADHDpi group are focussed in the delta band which suggests that a lower 2-4 Hz, 2-6 Hz, or 2-8 Hz treatment should also be considered for these patients. For beta band neurofeedback, the many statistically significant t-test differences between the normal and ADHDpi group in the beta2 band suggest that this frequency band, but not the beta1 band, should be the focus of fast wave treatment for adults with ADHDpi.

The beta2 band and a combination of high delta (2-4 Hz) with either low theta (4-6 Hz) or theta (4-8 Hz) would appear to be an appropriate treatment protocol to use with ADHDpi adults. The strategy of targeting the theta and beta bands is not new and is often used with children, but for adults with ADHDpi it would appear to be necessary to use the beta2 band along with high delta and low theta. Successful treatment using these two bands should result in an EEG profile that approximates a normal adult. Given the complexity in modifying two bands

simultaneously, a schedule that alternates the focus of treatment from one band to the other on a within session, session-to-session, or week-to-week basis could be used with these patients (Lubar, 1999).

Band Selection-ADHDhy Group

The widespread highly significant differences between the ADHDhy and the control group, in the T/B ratio, indicates that in order to be effective, both the theta and beta1 bands should be targeted. In the adult with ADHDhy, the highest number of statistically significant differences occurs in the theta band, indicating that this frequency should be a focus for neurofeedback treatment. There are differences between the normal and ADHDhy subjects in both the beta1 and beta2 bands but the greater number of these differences appear in the beta1 band, suggesting that this band or a protocol using an extended 16-24 Hz band would be an effective treatment. The same process described for the ADHDpi subjects, with alternating bands within or between sessions should also be employed for this group.

Selection of a Neurotherapy Site

Researchers and clinicians typically target the area between Pz and Fz to perform neurofeedback with children (Lubar, 1992; Tansey, 1990). The results of the present research suggest, however, that there may be other factors to consider before using the standard locations. For example, the clinician should also consider selecting a site based on the degree to which the site's activity deviates

from that of a normal person. The premise of this selection criteria is that the goal of neurotherapy is for the patient to produce EEG activity that more closely resembles that of a normal person. This site is determined by comparing the ADHDpi or ADHDhy groups' absolute and relative levels of EEG activity to the levels of activity in the control group. In addition, the selection of an appropriate site is also based on the number of statistically significant t-test differences that occurred at the site. The following sections describe the site selection process for ADHDpi and ADHDhy groups.

Site Selection-ADHDhy Group

The selection of an appropriate site for the ADHDhy subjects is determined by reviewing the t-test results and calculating the distribution and location of the five sites with the greatest number of statistically significant differences. These sites are in the frontal and temporal areas and, combined, represent the locations at which over 40% of the statistically significant differences occurred. Four of these locations, F3, Fp2, Fp1, and T5, also have the highest number of highly significant t-test differences, $p < .01$, but the T5 location would appear to be a particularly important region to target for neurofeedback treatment. It is the site that shows the greatest differences from control subjects in terms of raw μV value in the theta band; one of the five locations with the highest number of statistically significant t-test differences; one of two locations with the highest number of highly significant, $p < .01$, t-test differences; the highest number of T/B differences, and the highest

number of differences below the p <.001 level. In addition to these factors, the T5 site is unique in that none of the statistically significant differences were in the beta1 band and only one was in the beta2 band. These results indicate that the beta1 and beta2 activity of ADHDhy subjects are most like the controls at this location but least like the controls in the theta band, indicating that the T5 site would be ideal for theta or delta band neurofeedback. A compressed spectral array (CSA) from the T5 site showing the distribution of energy of a control and ADHDhy subject is presented in Figure 8.

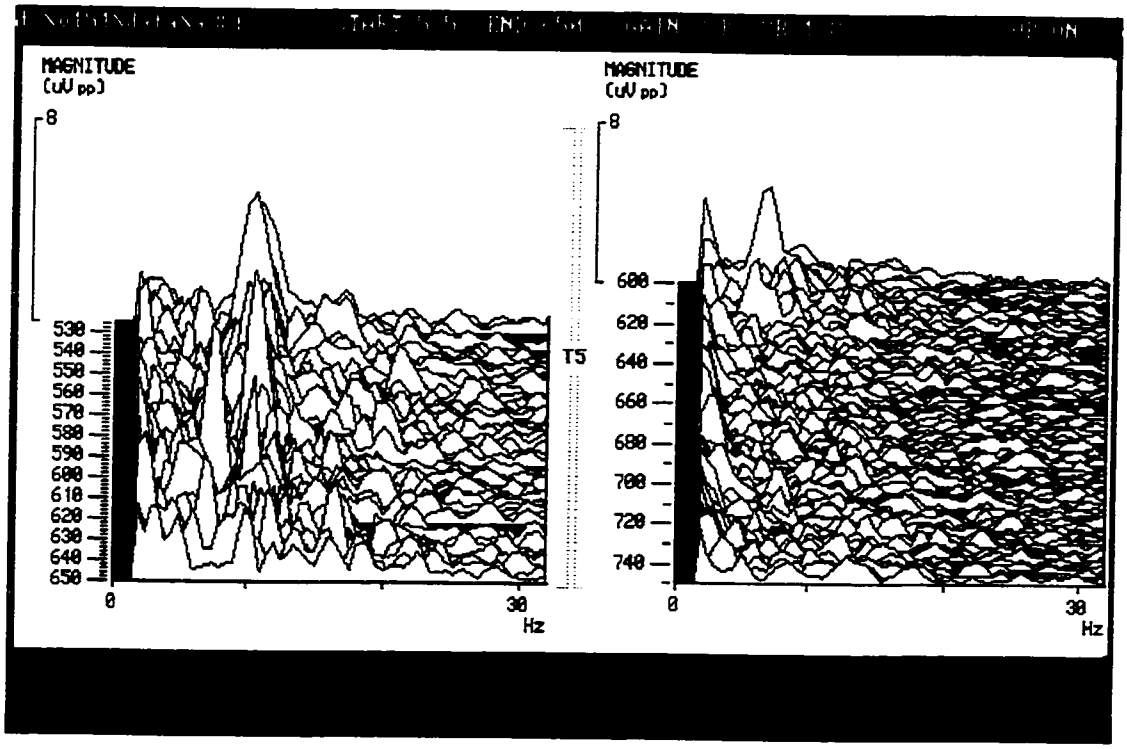

Figure 8. Compressed Spectral Array from the T5 site of a control (left) and ADHDhy subject (right) highlighting the spectral differences between these two individuals at this site.

As previously noted, the frontal area may be a particularly important region to target for neurotherapy due to its role in response inhibition as well as attention. Based on the statistical analyses, the Fp2 site is a complimentary location at which to provide beta1, beta2, or 16-24 Hz training for ADHDhy subjects. The Fp2 site is most dissimilar to the control group in raw μV and the percentage of activity in the beta1 band and one of three sites with the highest difference in raw μV and the percentage of activity in the beta2 band. In addition, it is the site with the largest number of statistically significant differences, one of four sites with the largest number of highly significant differences $p < .01$, and the site with the second highest number of T/B ratio differences. The Fp2 location is also unique in that the majority of the beta1 and beta2 band differences occur at this site making it the most potentially useful area to target for beta band neurofeedback. Caution, however, should be exercised when using this site as it is particularly vulnerable to changes in frontalis EMG which may affect the beta1 and beta2 bands. The ADHDhy group have lower levels of EMG hence apparent gains in beta1 or beta2 may simply be a result of increased EMG. In order to control for EMG contamination, this activity should be monitored and inhibited during training while only the beta1 band is targeted; beta2 feedback should be avoided.

Simultaneous inhibition of EMG and increases in beta1 may be too difficult for some clients. In this case, the F4 site would appear to be a useful compromise due to its proximity to Fp2. The F4 site has moderately high raw μV and

percentage differences in the beta1 and beta2 bands in addition to a high number of statistical differences in these bands. A compressed spectral array (CSA) from the F4 site showing the differences in activity between a control and ADHDhy subject is presented in Figure 9.

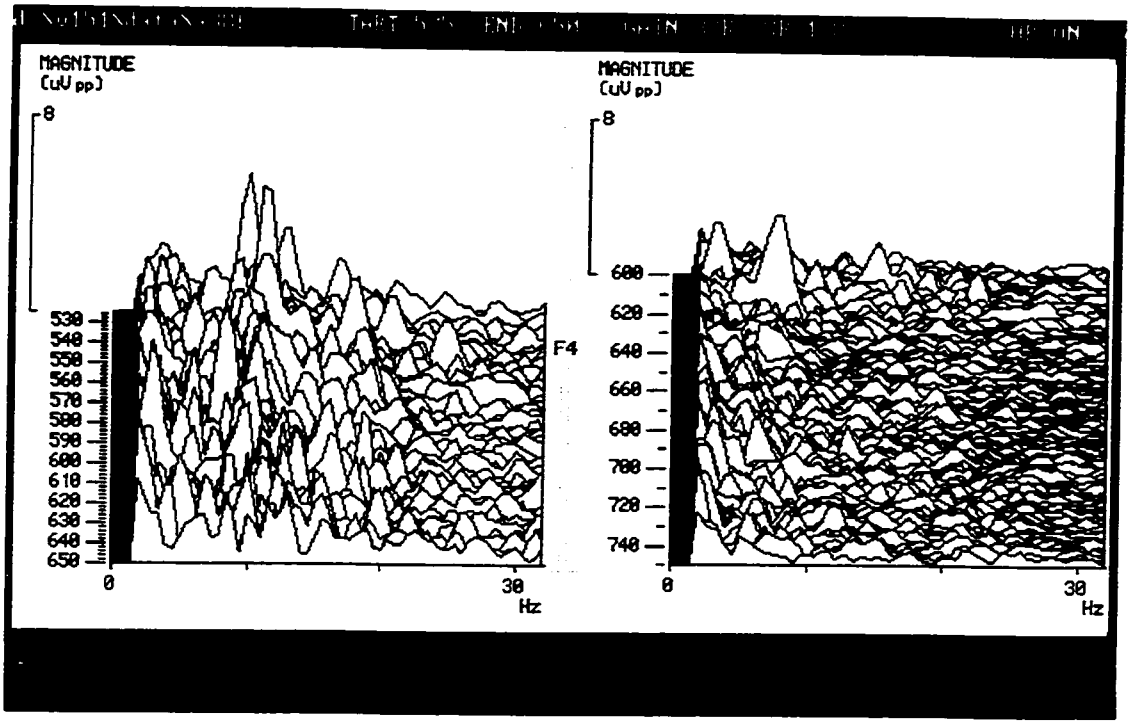

Figure 9. Compressed Spectral Array from the F4 site of a control (left side) and ADHDhy subject highlighting the differences between these subjects at this site.

Site Selection-ADHDpi Group

The process of selecting an appropriate site for the ADHDpi group begins by considering the six sites with the highest number of statistically significant t-test differences. In ADHDpi subjects these sites are in the left hemisphere and include frontal, temporal, parietal, and occipital locations and account for approximately

50% of the between group t-test differences. Three of these sites, F3, Fp1, and C3 also have the highest number of differences at the highest levels of significance, $p < .01$, but the F3 site appears to be the most advantageous location to target for neurofeedback. Compared to the control group, the F3 site has the highest percentage differences and the third highest raw μV difference in the beta2 band, the second highest number of statistically significant differences, the highest number of T/B differences, and the second highest number of highly significant, $p < .01$, differences. In addition, the F3 location also has the largest number of statistically significant t-test differences in the percentage of beta2 band activity suggesting that this would also be an ideal site to target for 16-24 Hz neurofeedback. A compressed spectral array (CSA) from the F3 site showing the differences in activity between a control and ADHDpi subject is presented in Figure 10.

Compared to the control group, the F3 site also has the second highest percentage of differences and the highest raw μV difference in the theta band and the second highest number of statistically significant differences in the theta and T/B ratios. These results suggest that theta band feedback would also be appropriate for this site. The O1 site is a suitable, complementary location at which to perform only theta or theta/delta wave neurofeedback. The O1 site has the highest number of T/B differences, the highest number of highly significant $p < .01$ differences, and the second highest number of theta band differences; qualities

that indicate that theta, delta, or 2-6 Hz neurofeedback training would be appropriate at this location.

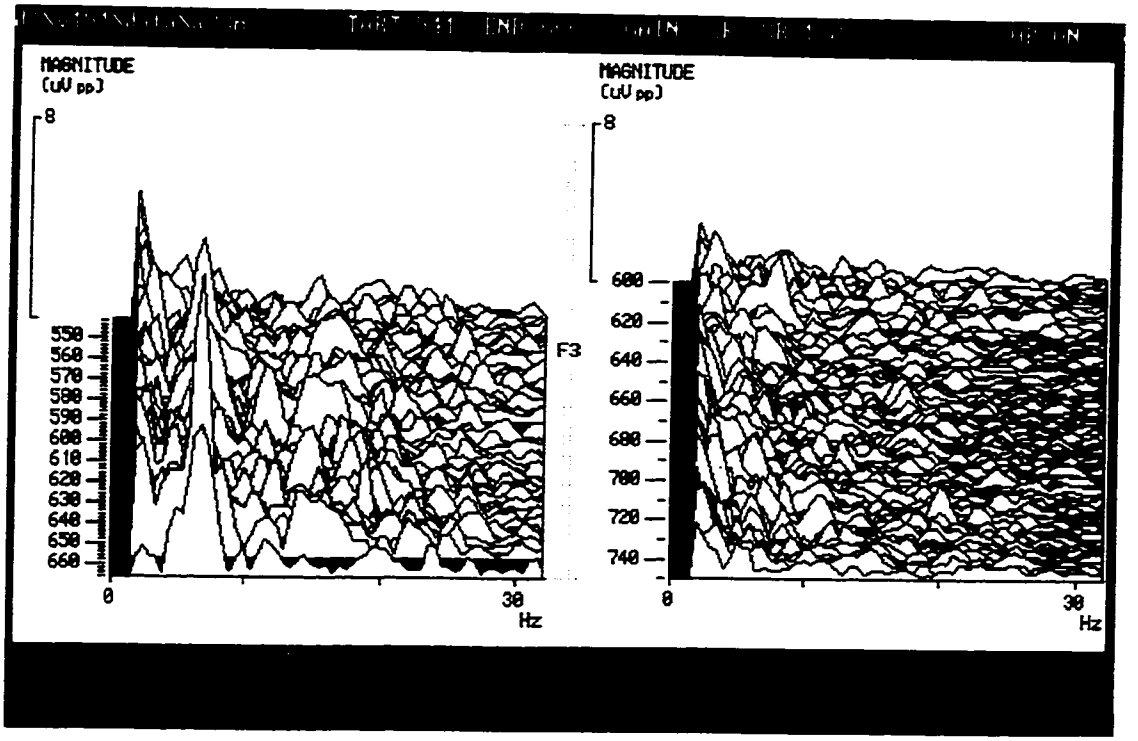

Figure 10. Compressed Spectral Array of the F3 site, control (left) and ADHDpi subject (right) highlighting the differences between these subjects at this site.

A cautionary note to providing theta band feedback from this location is that treatment of alcoholism and agitated drug addiction problems typically involves increases in theta band activity at this site (Peniston & Kulowsky, 1989). The clinician should, therefore, monitor the subject's progress carefully when using this site. A compressed spectral array (CSA) from the O1 site showing the differences in activity between a control and ADHDpi subject is presented in Figure 11.

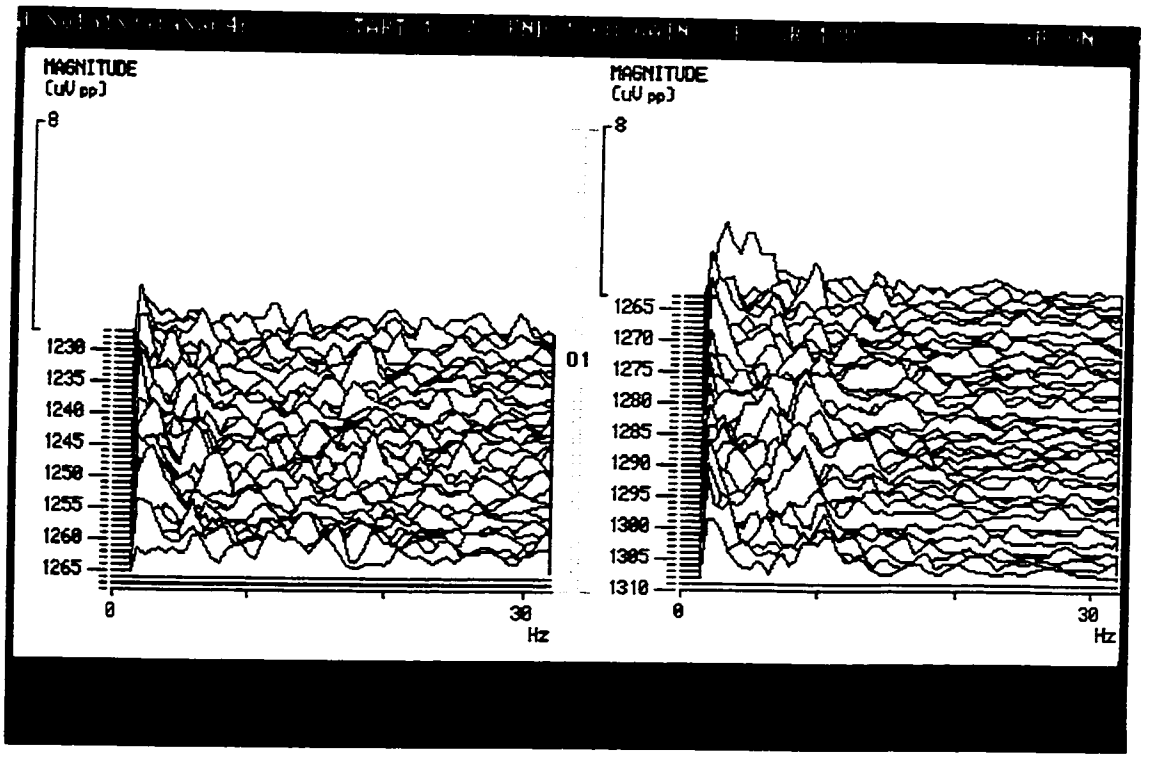

Figure 11. Compressed Spectral Array from the O1 site, of a control (left) and ADHDpi subject (right) highlighting the differences between them at this site.

In summary, the selection of the T5 site for theta and the Fp2 site for beta1 and beta2 for ADHDhy subjects, and the F3 site for beta2 or theta and the O1 site for theta and delta for ADHDpi subjects are quite different from the sites that would have been selected if maximum output μV or percentage of output data were used as the selection criteria. If these values had been used the Cz and Fz sites would have been selected for theta band feedback and the T3 or T4 sites would have been used for beta1 and beta2 neurofeedback.

The present research has shown that there may be important differences in the criteria for site selection for the ADHDpi and ADHDhy groups. The

widespread gains reported in children using only the Pz to Fz sites may have been due to the presence of 3 to 5 times as much theta band activity in these children. The differences in slow wave activity in ADHD adults, although statistically significant, appear to be substantially lower than that in the attention disordered child. Nevertheless, at the sites suggested for neurotherapy, the ADHDpi or ADHDhy adult has, on average, approximately 8% to 12% more theta and between 13% and 23% less beta1 or beta2 activity than control subjects at the same sites. The greater percentage differences in beta activity suggest that training in this band may be more important than reductions in slow wave activity, a possibility noted earlier by Lubar et al (1995).

Summary

The results from the present research suggest that a maturational lag may be in part responsible for the EEG differences between the ADHD and control groups. Although by no means conclusive, these results lend support to other studies that have based their hypotheses on studies with children with ADHD (Clarke et al. 1998; Satterfield et al. 1973). Research using a cross sectional design and larger subject groups could assist in clarifying this hypothesis.

The following observations may be useful to future researchers with regard to the effectiveness of various tasks to produce EEG differences among the groups. For example, there were very few differences in the analyses of regional or inter-hemispheric activity. In addition, many therapists use reading as the 'gold standard' to detect excess theta in children with ADHD (P. Swingle, September, 1997, personal communication); however, by adulthood, the reading and listening tasks appear to have become too well practiced and automatic to be useful as an on task measure.

The selective attention task is the most promising as a short, easy to administer, and low cost method of evoking significant qEEG differences between the normal and ADHDpi subject. The effects of fatigue on the performance of this task have yet to be determined hence future research that more closely examines the effects of this task on EEG activity should do so immediately following a baseline measure of EEG activity. As outlined and discussed previously, the

T.O.V.A. appears to be the most useful task for eliciting abnormal qEEG activity.

Significant qEEG differences are apparent between the control and ADHD groups. The differences, however, are only in the order of 10% to 20%, compared to the 300%-500% differences observed in children. Moreover, that the differences in the alpha, SMR, and beta1 bands have all but disappeared by adulthood may lead to the conclusion that neurotherapy would be of reduced or limited value. However, the smaller differences in EEG activity may indicate that the underlying neural regulatory mechanisms have 'set' at these levels in the adult population. This condition could indicate that changes to the regulatory mechanisms through neurofeedback would be more difficult to achieve than with children, but that the outcomes could be equally or perhaps more behaviourly apparent. Neurotherapy research using a matched groups design, with adult subjects tested pre- and post-treatment in a control (only neurotherapy) and clinical (neurotherapy and counseling) design could assist in determining not only the effectiveness of this treatment with adults but also the role of counselling in patient change.

The uniqueness of this study is the use of a wide age range of both male and female adult subjects. The use of a more heterogeneous sample allows the results to be applied to a broader range of individuals than is typically encountered in this type of research. In spite of unequal sample sizes and the varied makeup of the groups the significance levels of the results indicate that the differences were

unlikely to have occurred by chance. However, in order to gain greater precision, research using an experimental design with only male or female subjects, narrower age ranges, larger groups, and greater control over variables such as intelligence could add to the understanding and treatment of this pervasive and complex disorder.

Many of the ADHD subjects who participated in this research reported significant difficulties in personal and professional relationships, as well as problems completing homework and meeting deadlines at work. Some of those who were receiving medication praised the transformative effects of the drugs while others reported limited and sometimes negative effects from these same drugs. Virtually all the subjects, however, were hopeful that alternatives to medication would soon be found and many were interested in neurotherapy as a form of relief from their day-to-day problems.

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Appendix A

Recruitment handout for physicians

Dear Sir/Madam

As part of a doctoral thesis and with the approval of the School of Psychology at the University of Ottawa, I am conducting research with adults who have been diagnosed with Attention Deficit Disorder. This research is being performed under the direct supervision of Dr. P. G. Swingle (Ph.D. C. Psych) and with the approval of the School of Psychology's ethics committee.

The goal of this study is to determine if differences exist between the brainwave activity of adults with Attention Deficit Disorder, with (ADHD) or without hyperactivity (ADD) and a control group of adult subjects who do not present with this disorder. The brainwave activity will be measured with an electroencephalograph (EEG) and require that the subject have 19 electrodes attached to the scalp. These electrodes are held in place by a rubberized cap which fits over the subject's head and a conductive paste between the electrode and the scalp. The subject's EEG will be measured under a number of different conditions. Initially, the subject will be asked to sit with their eyes open for three minutes while fixating a 8.5 x 11 inch sheet of plain paper then asked to sit for three minutes with their eyes closed. Following each of these measures the subject will be asked to rate their level of relaxation and mental activity. The subject will then be asked to perform a reading and listening task; a computerized perceptual

matching task; a selective attention task; and a computerized diagnostic test of attention. The fitting of the recording equipment takes approximately 20-25 minutes and the tests take approximately 45-50 minutes to complete. The subject is free to leave at any time before or during the procedure.

The goal of this study is to better understand the relationship between the adult EEG and ADHD/ADD, to integrate these findings into current models of this disorder, and to develop adult diagnostic EEG criteria. From a practical viewpoint the aim is to determine the appropriateness of the currently used EEG biofeedback equipment as a diagnostic and treatment modality for adults.

I am requesting that you make available to your patients or clients the attached form (Appendix B) which outlines the procedure and goals of this study. There is no risk of physical or psychological damage, except a possible redness around the area of the scalp to which the electrode is fitted, the results are completely confidential and there is no use of deception in this study. Subjects who you feel may be appropriate for this research should be over 18 and have been diagnosed as primarily ADHD or ADD. It is necessary that the subject be free of stimulant medication for a period of 72 hours prior to testing and that he has not consumed caffeine or antihistamines 12 hours before testing.

If you have any questions or concerns please feel free to contact me, Mike de Jong, at 562-5800, ext 4430, my supervisor, P. Swingle, at 562-5800, ext 5979 or Jean D'Arc at the School of Psychology of the University of Ottawa.

Appendix B

Recruitment handout for potential subjects

Dear Sir/Madam

I am graduate student in the School of Psychology at the University of Ottawa and I am conducting research with adults who have been diagnosed with Attention Deficit Disorder. I am performing this research under the direct supervision of Dr. P. G. Swingle (Ph.D.. C. Psych) and with the approval of the School of Psychology's ethics committee.

The goal of this study is to determine if differences exist between the brainwave activity of adults with Attention Deficit Disorder with (ADHD) or without Hyperactivity (ADD) and a control group of adult subjects who do not present with this disorder. The brainwave activity will be measured with an electroencephalograph (EEG) and require that 19 electrodes be placed and held to the surface of your scalp by means of rubber cap and electrode paste. Following a detailed briefing on the experimental procedure and the completion of a short questionnaire you will be asked to perform a number of tasks. For example, initially, you will be asked to sit with your eyes open for three minutes while fixating a 8.5 x 11 inch sheet of plain paper then asked to sit for three minutes with your eyes closed. You will then be asked to read a short passage of text and then answer some questions about the passage, listen to a story and then answer some questions about the story, play a computerized game in which you match shapes, play a computerized game where you respond to a shape while distracting objects appear on the screen, and finally play a computerized game where you discriminate

between the colours of different objects. We are interested primarily in the activity of the brain while engaged in these tasks. Fitting the equipment takes about 20-25 minutes and the tests take about 45-50 minutes to complete.

There is no physical evidence of participation, except a possible redness around the area of the scalp on which the electrode is placed. In addition, there is no deception, no risk of physical or psychological injury, the results are completely confidential, and you may withdraw at any time for any reason without explanation.

Because of the nature of this type of research it is important that you do not consume any prescription medication 72 hours prior to testing. If you are receiving Ritalin, Methylphenidate or any other substance for the treatment of attentional problems please consult with your physician before arranging to participate in this study. I will make every attempt to accommodate your needs in this respect by, for example, arranging testing on a convenient day. Also, it is important that no caffeine or antihistamines be consumed prior to testing and that you are in good health.

By agreeing to participate as a research subject, you will assist in the understanding of how attentional processes operate and, possibly, how best to develop non-drug methods of treatment for attention deficit disorders. If you have any questions or concerns please feel free to contact me, Mike de Jong, at 562-5800, ext 4430, my supervisor, P. Swingle, at 562-5800, ext 5979 or Jean D'Arc at the Department of Psychology of the University of Ottawa at 562-5800, ext 5801.

Appendix C

Copy of the text used in the reading task

THE FLOOD

It had been an unusually long winter with heavy snow that had fallen on land still saturated from autumn's torrential rains, and fallen, and fallen until even the bison fled to more hospitable ground. Then, all at once, spring arrived and the Red River, named for the red-brown silt it carries, rose and kept on rising until there was nothing to do but abandon everything and run.

Before that spring in 1826, it had seemed a tall tale when native people talked of how, some 50 years earlier, the normally placid river had turned into a sea. For almost two months, the story went, the only land visible for kilometers around the junction of the Red and Assiniboine rivers, which would become The Forks in the heart of Winnipeg, was Stony Mountain and Birds Hill, about 20 kilometers to the north and northeast.

But here it was, May, and the normally 100-metre-wide river, which can threaten to dry up in mid-summer, was spread across the prairie. When its crest crossed the fork of the rivers, it stood at 36.5 feet above the river bed. "The shrieks of children, the lowing of cattle and the howling of dogs added terror to the scene," wrote fur trader and historian, Alexander Ross, in his memoir.

Almost everyone, including 400 people that Lord Selkirk had coaxed into settling around Fort Garry about a decade before, were forced to "fly from their homes." Governor George Simpson predicted the 1826 flood would be "a death blow to the colony." He was wrong.

When the waters struck with a fury again in 1852, Ross was one of the "wretched inhabitants" who had been stubborn enough to stay in this swamp in the middle of a flood plain. The 1852 deluge crested two feet (Manitoba still measures the river's crest in feet) lower than the 1826 mark, but the population had grown and with few preventive measures and little warning, havoc prevailed. "Dwelling houses and barns were floating in all directions like sloops under sail, with dogs, cats and poultry in them. Outhouses, carts, carioles, boxes, cupboards, tables, chairs, feather beds and every variety of household furniture drifting along added to the universal wreck," wrote Ross. Damage was estimated at 25,000 pounds sterling (approximately $2 million in 1997 dollars).

The Red River's waltz with its human inhabitants had begun in earnest. Flooding in the valley of silty loam left behind by the retreat of Lake Agassiz 7,700 years ago is an annual ritual. Some years are simply more memorable than others. In terms of crest levels, 1826 remains the worst in recorded Manitoba history. By 1861 when another great flood hit (32.5 feet), the settlers, who had fretted the smaller freshets each spring, knew to clear out before the water rose.

Throughout the 1800's, improved rail and water transport helped Winnipeg grow while a land drainage program in the valley, the first megaproject launched by the Manitoba government, enticed more farmers to cultivate the once forsaken marshland. By 1881, 320 kilometers of drainage ditches had been etched throughout the valley. Today, the figure is in the tens of thousands of kilometers.

The drainage system, coupled with consistently favourable weather, helped grant southeastern Manitoba a reprieve from severe floods between 1916 and 1948, when a near-flood jogged the memory of the Red's old dance partners. On May 1 of that year, the water at The Forks reached 23.4 feet. Hastily constructed dikes protected low-lying areas.

Two years later, on April 11, citizens were warned that a repeat of 1948 was pending. Again, the dikes went up. Information was relayed to authorities by phone from river stations between Fargo, N.D., and Winnipeg, with additional data provided via aerial reconnaissance. A week-long cold snap slowed the river's flow and authorities began to downplay the threat. But a burst of warm air and heavy rains followed and, by May 19, the waters measured 30.3 feet in the provincial capital - population 400,000. With the help of 5,000 military personnel, almost 80,000 fled from the city and close to 20,000 people were evacuated from rural areas. About 13,000 homes and farms were flooded and final damage estimates stood at $125 million ($606 million in 1997 dollars).

The massive flooding triggered a call for a comprehensive plan to tame the Red. In response, almost 800,000 cubic metres of Manitoba's dense clay were piled alongside the Red and Assiniboine rivers in the Winnipeg area, a total length of 109 kilometers, and 31 pumping stations were built to keep the city sewer system from flooding. The dikes, comprising an artificial secondary bank at 26.5 feet-james (the elevation at the James Avenue pumping station downtown Winnipeg), became a permanent part of the urban landscape. In 1968 work was completed on the $63 million Red River Floodway, a 47 kilometre long channel that diverts the Red around the eastern edge of the city where it continues on to its final destination, Lake Winnipeg. To control the flow of the Assiniboine into the city, the Shellmouth Dam and the Portage diversion were built upriver.

By the early 1970s, permanent dikes, serving as raised railways and roads, were built around eight towns in the valley between the U.S. border and Winnipeg and then around some 700 rural homesteads. Many are as high as the eaves troughs of town buildings. All were put to their first big test in 1979 when waters, similar to 1950 levels, raced through the valley. Many of the low-lying areas had been built up and the dams and dikes, by and large, did their job. This time, 7,000 people were forced to flee the Red. In the aftermath, many of the dikes were raised still further as an extra precaution.

Then came the spring of 1997. Record levels of snow fell on ground saturated through a wet autumn. In early April, just days after runoff had begun, a major storm dumped an additional 50 to 70 centimeters of snow and freezing rain

on top of a near-record snowpack of 250 centimeters.

Still, Manitobans, particularly the 662,000 Winnipegers protected by the floodway, remained cautiously optimistic that they were prepared for the worst. There were those massive, modern engineering projects and smaller helpers, sandbags, sump pumps, and sewer back-up valves, available to almost everyone. And flood forecasting had become a high-tech affair. The confidence gave way to nervous anticipations as television pictures started carrying images of the disaster in Grand Forks, N.D., and Manitoba authorities started to adjust their estimates.

Alf Warkentin, Manitoba's Natural Resource's flood forecaster for 27 years, is the province's modern-day Paul Revere, except that he rarely had to leave his paper-littered River Forecast Centre in a strip mall office in suburban Winnipeg to get his warnings out.

Back in February, snow and soil measurements, gathered by aircraft and satellites, were applied to run-off formulas based on decades of historical data. Additional information was generated as the melt began, combining data from a dozen automatic water level gauges installed throughout Manitoba's portion of the river basin, field measurements of flow, and manual gauge readings. By the time the state of emergency was declared on April 23, Warkentin, a grey-haired, somewhat rumpled hydrologist, was working flat out producing daily water level predications. Warkentin's calculations were sent out by phone, fax and via the centre's website.

His "inexact science," as Warkentin calls it, proved remarkably accurate. The only trouble spot was in trying to estimate how the flow over the banks of the Red River would affect communities such as Grande Pointe, a suburb of 150 homes a few kilometre southeast of Winnipeg, which had seldom flooded since a diversion was built in 1967. The water rose two feet higher that predicted a week before the crest hit. North Dakota authorities underestimated the crest at Grand Forks by almost five feet days before it struck.

Without the floodway, dams and dikes, estimates are that the 1997 crest would have measured 34.3 feet, almost five feet higher than the 1950 deluge. Almost 80 percent of Winnipeg would have been underwater and more than 550,000 city dwellers evacuated. Instead, the floodway kept the water level at 24.5 feet and even controlled the river's descent, preventing banks from collapsing under the weight of six million sandbags piled to protect several hundred houses along the river side of the primary dikes. In the end, 28,000 Manitobans were evacuated (6,000 from Winnipeg), more than 202,500 hectares of farmland flooded under a 2,000 square kilometre "Red Sea," and 2,500 properties between the U.S. border and Winnipeg damaged. About two dozen Winnipeg homes were damaged. While the ring-diked valley towns escaped serious damage, costs of repairs to roads, bridges, farms and homes is estimated at about $150 million.

Now, as has happened time and again, Manitobans have returned to their lives with tall tales of their latest round with the Red. And, once again, they are

exploring what more can be done. There is talk of turning temporary dikes into permanent ones, of reinforcing old ones, of setting up a cross-border commission to seek joint solutions. Discussions are being held with American authorities on whether or not dams or diversions might help south of the border, where most of the Red's drainage basin lies.

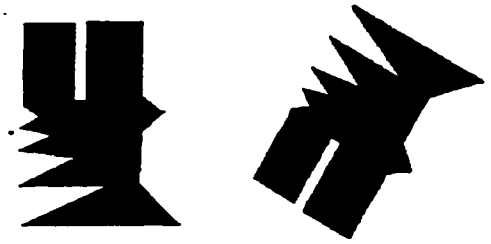
But there is one question no one can answer. For all anyone knows, the 877 kilometre Red River could again fall into one of its 30 year sleeps. Or, then again, it just might feel like dancing again next spring.

Appendix D

Questions asked after the reading task

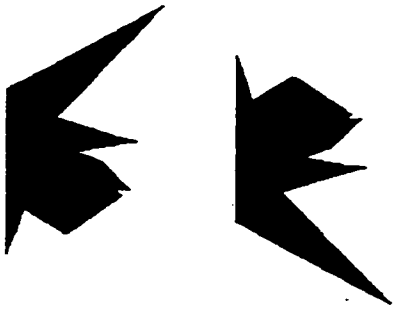
1. What was the main theme of the story?
2. Who was Selkirk/ George Simpson?
3. In which century did the worst flooding occur?
4. What, according to the text, was the advantage of flooding?
5. According to the story, what were the early ways of dealing with the flood?

Examples of images used in the mental rotation task

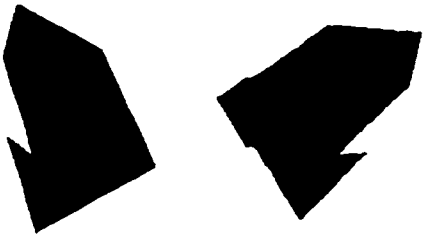


Press 1 for match. Press 2 for mirror.

Press 1 for match. Press 2 for mirror.



Press 1 for match, press 2 for mirror.



Press 1 for match. Press 2 for mirror

Appendix F

Example of selective attention task targets and distractors

Examples of target, in this case the letter H, and distracters for the selective attention task.

Target letters are H or K press left button, S or C press right button.

Distractor letters are:

1) H H H *H* H H H	Identical to target.
2) K K K *H* K K K	The other letter of the same set.
3) S S S *H* S S S	Members of the other response set.
4) N W Z *H* N W Z	Members that have similar features to the response set.
5) G J Q *H* G J Q	Members that do not have similar features.
6) *H*	Presented alone.

Appendix G

Story used in listening task

THE OCEAN

Sometime next season, if all goes well, a revolutionary new undersea vessel will be lowered gently into the waters of Monterey Bay for its maiden voyage. Named Deep Flight I, the 4 metre long 1,315 kg vehicle is shaped like a chubby, winged torpedo but flies like an underwater bird. Compared with the hard-to-manoeuvre submersibles that now haul deep sea explorers sluggishly around the oceans, Deep Flight is an aquatic F-16 fighter. It can perform barrel rolls, race a fast moving pod of whales or leap vertically right out of the sea. With a touch on the controls, a skilled pilot, who lies prone in a body harness, his or her head protruding into the craft's hemispherical glass nose, can skim just below the ocean's surface or plunge to 1,000 metres below.

But Deep Flight I is just a pale prototype of what's to come. Back in their Point Richmond, California workshop, the craft's designers have already drawn blueprints for its successor, Deep Flight II, an industrial strength submersible capable of diving as far as 11 km straight down, to the Mariana Trench, the aquatic equivalent of Mount Everest or the South Pole or the moon.

More than 35 years after the bathyscaphe Trieste took two men, for the first and last time, 10,912 metres down to the deepest spot in the world, the Mariana Trench's Challenger Deep just off Guam in the Western Pacific, undersea adventurers are preparing to go back. Last March a Japanese robot scouted a tiny section of the bottom of the 2,550 km long crevasse and sent back the first real time video images of deepest sea life. And in laboratories around the world, engineers are hard at work on an armada of sophisticated craft designed to explore, and in some cases exploit, the one great unconquered place on earth: the bottom of the sea.

The irony of 20th century scientists venturing out to explore waters that have been navigated for thousands of years is not lost on oceanographers. More than 100 expeditions have reached Everest, the 8,848 metre pinnacle of the Himalaya; manned voyages to space have become commonplace; and robot probes have ventured to the outer reaches of the solar system. But only now are the deepest parts of the ocean coming within reach. "I think there's a perception that we have already explored the Sea," says marine biologist Sylvia Earle, a former chief scientist at the National Oceanographic and Atmospheric Administration and a co-fonder of Deep Ocean Engineering, the San Leandro, California, company where construction of Deep Flight I began: "The reality is we know more about Mars than we know about the oceans."

That goes not only for the sea's uttermost depths but also for the still mysterious middle waters five or six kilometers down, and even for the "shallows"

60 to 90 metres deep. For while the push to reach the very bottom of the sea has fired the imagination of some of the world's most daring explorers, it is just the most visible part of a broad international effort to probe the oceans' depths. It's a high-sea adventure fraught with danger, and, because of the expense, with controversy as well.

But the rewards could be enormous: oil and mineral wealth to rival Alaska's North Slope and California's Gold Rush; Scientific discoveries that could change our view of how the planet, and the fife-forms on it, evolved; natural substances that could yield new medicines and whole new classes of industrial chemicals. Beyond those practical benefits there is the intangible by real satisfaction that comes from exploring earth's last real frontier.

There's a lot to explore. Oceans cover nearly three quarters of the planet's surface, 1.4 billion cubic km of water that reaches an average depth of 3,700 metres. The sea's intricate food webs support more life by weight and a greater diversity of animals than any other ecosystem, from sulphur eating bacteria clustered around deep-sea vents to fish that light up like New York City's Times Square billboards to lure their prey. Somewhere below there even lurks the last certified sea monster left from pre-scientific times: the 20 metre long giant squid.

The sea's economic potential is equally enormous. Majestically swirling ocean currents influence much of the world's weather patterns; figuring out how they operate could save trillions of dollars in weather related disasters. The oceans also have vast reserves of commercially valuable minerals, including nickel, iron, manganese, copper and cobalt. Pharmaceutical and biotechnology companies are already analyzing deep-sea bacteria, fish and marine plants looking for substance that they might someday turn into miracle drugs. Says Bruce Robison, of the Monterey Bay Aquarium Research Institute (MBARI) in California: "O can guarantee you that the discoveries beneficial to mankind will far outweigh those of the space program over the next couple of decades. If we can get to the abyss regularly, there will be immediate payoffs."

Getting there, though, will force explorers to cope with an environment just as perilous as outer space. Unaided, humans can't dive much more that 3 metres down, less than one three-thousandth of the way to the very bottom, before increasing pressure starts to build up painfully on the inner ear, sinuses and lungs. Frigid subsurface water rapidly sucks away body heat. And even the most leathery of lungs can't hold a breath for more that two or three minutes.

For these reasons the modern age of deep-sea exploration had to wait for two key technological developments: engineer Otis Barton's 1930 invention of the bathysphere, essentially a deep-diving tethered steel ball, and the invention of scuba (short for "self-contained underwater breathing apparatus") by Jacques Cousteau and Emile Gagnan in 1943. Swimmers had been trying to figure out how to get oxygen underwater for thousands of years. Sponge divers in ancient Greece breathed from air filled kettles; bulky helmeted diving suits linked by hose

to the surface first appeared in the 1800s. But it wasn't until scuba came along that humans, breathing compressed air, were able to move about freely underwater at depths of more than 30 metres.

Even the most experience scuba divers rarely venture below 45 metre, however, owing to increasingly crushing pressure and the laborious decompression process required to purge the blood of nitrogen (which can form bubbles as a diver returns to the surface and cause the excruciating and sometimes fatal condition known as the bends). And pressurized diving suits make it possible for humans to descend only to 440 metres, far short of the deepest reaches of the oceans.

Underwater vehicles date back at least to 1620. but it wasn't until Barton's bathysphere came along that scientists could descend to any respectable depth. The Bathysphere eventually tool Baron and zoologist William Beebe to a record 923 metres, off Bermuda. But it wasn't at all manoeuvrable; it could only go straight down and straight back up again. Swiss engineer Auguste Piccard solved the mobility problem with the first true submersible, a dirigible like vessel called a bathyscaphe, which consisted of a spherical watertight cabin suspended below a buoyant gasoline filled pontoon. (A submersible is simply a small, mobile undersea vessel used for science.)

The Trieste, which took U.S. Navy Lieut. Don Walsh and Piccard's son Jacques into the Challenger Deep, was only the third bathyscaphe ever built, and unlike modern submersibles, which bristle with advanced underwater cameras, grabbers, collection baskets and manipulator arms, it carried nothing but its passengers. Its mission was to test whether humans could reach the abyss, the first step toward developing a fleet of manned submersibles. "At the time, people were still flying across the Atlantic in prop planes," recalls Walsh, now a consultant on underwater technology. "Criticizing the Trieste mission for not carrying cameras and other instruments is like chastising the Wright brothers for not carrying passengers."

In the wake of Trieste's successful dive, the number of submersibles expanded dramatically. The American Woods Hole Oceanographic Institution's workhorse, the three person Alvin (still in operation), was launched in 1964. And the first robots-on-a-tether, the so called remotely operated vehicles, or ROVS, were developed several years later. The Soviet Union, France and Japan began building their own submersibles, either for military or scientific reasons. and for the first time scientists could systematically collect animals, plants, rocks and water samples rather than study whatever they could dredge up in collection baskets lowered from the surface.

Thus began a remarkable period of undersea discovery that transformed biology, geology and oceanography.

Appendix H

Questions used in listening task

1. What was the main theme of the story?
2. What was the name of the vehicles/ some of the areas discussed in the story?
3. According to the text, what are the advantages of undersea exploration?
4. According to the text, what sorts of animals were discussed?
5. What were some of the comparisons made with other types of explorations?

Appendix I

Summary of the Gender Age and Group Membership of Subjects Who Were Misclassified

Task	Wave Band	Group	Control			ADHD		ADD			Clinical		
			Male	Female	Age	Male	Age	Male	Female	Age	Male	Fem	Age
TOVA3/4	Comb	Clinical	3	2	27.8 (5.9)	2		2					33.5 (8.5)
TOVA1/2	Comb	ADD	1	1	25.5 (3.8)			4	3	37.8			
TOVA3/4	Alpha	ADHD		1	38	1	31						
TOVA3/4	Comb	ADD	3		22.5 (1.4)			1		43			
TOVA2/2	Comb	Clinical	2	4	28.3 (8.1)						5	3	32.8(11.3)
Select 1	Comb	ADD	1	1	45.9 (1.9)			2	1	32.6 (17)			
Listen	Comb	Clinical	2	7	31.6 (8.3)						2	4	31.9 (9.0)
TOVA4/4	Comb	Clinical	3	3	28.1 (7.3)						4		28.3 (2.4)
TOVA4/4	Alpha	ADHD	2	1	25.8 (6.9)	4	27.3 (7.3)						
TOVA4/4	SMR	ADHD	2	2	25.8 (6.9)	4	29.7 (5.7)						
TOVA2/4	Comb	ADD	1	1	37.6 (9.6)			2	2	35.8 (12.3)			
Totals			20	23		7		11	6		16	7	
x̄ age					30.6		29.33			37.3			32.2
SD					7.04		1.87			4.36			3.0
median					28.7		29.7			36.8			32.5

Appendix J

T-test results of the CLINICAL group during the eyes closed condition.

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.933 p=.005		t=-2.773 p=.007	t=-3.221 p=.002
C4		t=2.039 p=.045		t=-3.151 p=.002		t=-2.830 p=.006	t=-3.509 p=.001
CZ				t=-2.886 p=.005		t=-2.388 p=.020	t=-3.146 p=.002
F3		t=2.092 p=.040		t=-3.163 p=.002		t=-3.337 p=..001	t=-3.679 p=.000
F4		t=2.394 p=.020		t=-3.076 p=.003		t=-3.103 p=.003	t=-3.616 p=.001
F7		t=2.058 p=.044	t=2.556 p=.013	t=-3.130 p=.003		t=-3.982 p=.000	t=-3.565 p=.001
F8		t=2.486 p=.015		t=-2.781 p=.007		t=-3.445 p=.001	t=-4.148 p=.000
FP1		t=2.555 p=.013	t=2.042 p=.045	t=-2.716 p=.008		t=-3.541 p=.001	t=-4.295 p=.000
FP2		t=2.366 p=.021		t=-2.696 p=.009		t=-3.424 p=.001	t=-3.886 p=.000
FZ		t=2.421 p=.018		t=-2.956 p=.004		t=-3.003 p=.004	t=-3.914 p=.000
O1				t=-2.902 p=.005		t=-3.646 p=.001	t=-4.116 p=.000
O2				t=-2.402 p=.019		t=-2.847 p=.006	t=-3.223 p=.002
P3		t=2.050 p=.044		t=-2.443 p=.017		t=-2.905 p=.005	t=-3.686 p=.000
P4				t=-2.552 p=.013		t=-2.577 p=.012	t=-3.737 p=.000

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
PZ				t=-2.475 p=.016		t=-2.578 p=.012	t=-3.249 p=.002
T3				t=-2.190 p=.032		t=-3.137 p=.003	t=-3.076 p=.003
T4				t=-2.450 p=.017		t=-2.480 p=.013	t=-2.797 p=.007
T5				t=-2.707 p=.009		t=-4.165 p=.000	t=-4.244 p=.000
T6				t=-2.063 p=.043		t=-2.309 p=.024	t=-3.112 p=.003

* Note. For Theta/Beta Ratio df=65 (30/37)

Appendix K

Table K1

T-test results of the ADHDpi group during eyes open condition.

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
CZ							t=-2.147 p=.037
F3							t=-2.659 p=.034
F4							t=-2.479 p=.039
FZ							t=-2.273 p=.033
O1							t=-2.299 p=.032
P4							t=-2.322 p=.025
PZ							t=-2.253 p=.029

* Note. For Theta/Beta Ratio df=45 (30/17)

Appendix K

Table K2

T-test results of ADHD pi group during the reading task.

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
F3			t=2.132 p=.039			t=-2.026 p=.049	t=-2.173 p=.044
F4							t=-2.156 p=.014
F7			t=2.586 p=.013			t=-2.560 p=.014	t=-2.403 p=.028
F8						t=-2.060 p=.048	t=-2.310 p=.026
FP1			t=2.614 p=.012			t=-2.891 p=.006	t=-2.808 p=.012
FP2						t=-2.205 p=.036	t=-2.844 p=.007

* Note. For Theta/Beta Ratio df= 41 (29/14)

Appendix K

Table K3

T-test results of the ADHDpi group during the mental rotation task.

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C3			t=2.236 p=.031	t=-2.072 p=.045			t=-2.282 p=.028
C4			t=2.133 p=.039				
F3			t=2.594 p=.013				
F4			t=2.479 p=.018				
F7				t=-2.100 p=.042		t=-2.133 p=.039	t=-2.635 p=.012
F8							t=-2.072 p=.074
FP1							
FP2			t=2.386 p=.022	t=-2.119 p=.043			t=-2.446 p=.019
FZ			t=2.574 p=.014				
O1							t=-2.547 p=.021
O2							t=-2.378 p=.029
P3			t=2.308 p=.026	t=-2.050 p=.047			t=-2.204 p=.042
P4			t=2.446 p=.019				t=-2.210 p=.040
T5							t=-2.364 p=.032

* Note. For Theta/Beta Ratio df= 38 (26/14)

Appendix L

Table L1

T-test result of the ADHDhy group during the eyes open condition.

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C4				t=-2.044 p=.047			t=-2.061 p=.044
CZ		t=2.077 p=.043					t=-2.038 p=.047
F3							t=-2.245 p=.029
F7							t=-2.039 p=.047
FP2							t=-2.043 p=.046
O1							t=-2.579 p=.013
P3							t=-2.538 p=.014
PZ				t=-2.049 p=.045			t=-2.139 p=.037
T4		t=2.268 p=.027					t=-2.210 p=.032
T5						t=-2.466 p=.017	t=-2.717 p=.009

* Note. For Theta/Beta Ratio df= 52 (30/24)

Appendix L

Table L2

T-test results of the ADHDhy group during the first quarter of the T.O.V.A..

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
F3						t=-2.292 p=.026	t=-2.235 p=.030
FP1		t=2.803 p=.007	t=3.714 p=.001			t=-2.638 p=.011	t=-3.300 p=.002
T3		t=2.357 p=.023				t=-2.392 p=.021	t=-2.317 p=.025

* Note. For Theta/Beta Ratio df= 49 (29/22)

Appendix L

Table L3

T-test results of the ADHDhy group during the second quarter of the T.O.V.A..

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C4							t=-2.044 p=.048
F3			t=2.316 p=.026	t=-2.358 p=.023		t=-2.574 p=.014	t=-2.570 p=.014
F4			t=2.105 p=.041	t=-2.709 p=.010		t=-2.210 p=.033	t=-2.639 p=.012
FP1		t=2.107 p=.036					t=-2.551 p=.015
FZ		t=2.395 p=.021					t=-2.042 p=.048
T3				t=-2.356 p=.023		t=-2.299 p=.027	
T4							t=-2.108 p=.041
T5							t=-2.028 p=.049

* Note. For Theta/Beta Ratio df= 40 (24/18)

Appendix L

Table L4

T-test results of the ADHDhy group during the third quarter of the T.O.V.A..

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C4			t=2.113 p=.042	t=-2.114 p=.041			
CZ				t=-2.089 p=.045			
F3			t=2.926 p=.006	t=-2.355 p=.026			t=-2.064 p=.047
F4			t=2.544 p=.016	t=-2.233 p=.032			t=-2.290 p=.029
F8			t=2.106 p=.044	t=-2.372 p=.025			
FP1		t=2.240 p=.032	t=2.364 p=.025				t=-2.511 p=.017
FP2		t=2.332 p=.026					t=-2.500 p=.018
FZ			t=2.615 p=.022	t=-2.225 p=.033			
T3				t=-2.336 p=.026		t=-2.307 p=.028	
T4				t=-2.056 p=.048			

* Note. For Theta/Beta Ratio df= 31 (20/13)

Appendix L

Table L5

T-test results of the ADHDhy group during the fourth quarter of the T.O.V.A..

Site	Alpha	Beta 1	Beta 2	Delta	SMR	Theta	Theta / Beta Ratio*
C3							t=-2.100 p=.044
F3							t=-2.357 p=.025
F4							t=-2.185 p=.037
F7	t=2.836 p=.009						
FP2					t=2.223 p=.034		
P3						t=-2.316 p=.023	t=-2.272 p=.030

* Note. For Theta/Beta Ratio df= 30 (19/13)

Appendix M

Table M1

Summary of T-test Results at Sites for Control and CLINICAL Subjects

	Sites																			
Error	C3	C4	Cz	F3	F4	F7	F8	Fp1	Fp2	Fz	O1	O2	P3	P4	Pz	T3	T4	T5	T6	Total
p≤.05	19	19	11	16	18	15	23	15	18	18	10	12	18	19	15	3	2	11	9	271
p≤.01	11	6	6	11	10	4	4	6	10	10	7	5	7	5	5	4	1	10	4	126
p≤.001	0	1	0	6	2	2	2	6	2	2	6	1	2	3	0	0	0	2	0	37
Total	30	26	17	33	30	21	29	27	30	30	23	18	27	27	20	7	3	23	13	434
Percent Below p≤.01	36.9	26.9	35.3	51.5	40.0	28.6	20.7	44.4	40.0	40.0	56.5	50.0	33.3	29.6	25.0	57.1	33.3	52.1	30.8	37.6

Appendix M

Table M2

Summary of T-test Results at Sites for Control and ADHDpi Subjects

	Sites																			
Error	C3	C4	Cz	F3	F4	F7	F8	Fp1	Fp2	Fz	O1	O2	P3	P4	Pz	T3	T4	T5	T6	Total
p≤.05	9	7	3	16	11	14	7	7	10	10	11	6	13	9	4	1	0	12	2	152
p≤.01	3	1	0	3	1	1	0	3	1	2	5	2	0	0	0	0	0	1	0	23
p≤.001	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	12	8	3	19	12	15	7	10	11	12	16	8	13	9	4	1	0	13	2	175
Percent Below p≤.01	25.0	12.5	0	15.8	8.3	6.7	0	30.0	9.0	16.7	31.2	25.0	0	0	0	0	0	7.7	0	13.1

Appendix M

Table M3

Summary of T-test Results at Sites for Control and ADHDhy Subject

	Site																			
Error	C3	C4	Cz	F3	F4	F7	F8	Fp1	Fp2	Fz	O1	O2	P3	P4	Pz	T3	T4	T5	T6	Total
p≤.05	9	9	6	14	11	4	11	9	15	12	9	4	5	2	5	6	8	9	5	153
p≤.01	1	2	1	3	2	1	1	4	3	1	2	2	2	2	1	1	3	3	1	36
p≤.001	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	2	0	4
Total	10	11	7	17	13	5	13	14	18	13	11	6	7	4	6	7	11	14	6	193
Percent Below p≤.01	10.0	18.1	14.3	17.6	15.4	20.0	15.4	35.7	16.7	7.7	18.2	33.3	28.6	50.0	16.6	14.3	27.2	35.7	16.7	20.7

Appendix N

T-Tests - Eyes Open Task ADD/ADHD Combined (df = 67 (31/38))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.041 p=.045		t=-2.339 p=.022				t=-2.599 p=.012
C4		t=2.260 p=.027		t=-2.466 p=.016				t=-2.416 p=.019
CZ		t=2.646 p=.010		t=-2.260 p=.027				t=-2.680 p=.009
F3		t=2.250 p=.016		t=-2.384 p=.020			t=-2.714 p=.008	t=-3.097 p=.003
F4		t=2.127 p=.037	t=2.373 p=.021	t=-2.216 p=.030			t=-2.134 p=.037	t=-2.642 p=.010
F7		t=2.334 p=.023						t=-2.512 p=.015
F8				t=-2.086 p=.041			t=-2.116 p=.038	t=-1.996 p=.050
FP1								t=-2.177 p=.033
FP2								t=-2.205 p=.031
FZ		t=2.444 p=.017	t=2.143 p=.036	t=-2.207 p=.031			t=-2.086 p=.041	t=-2.743 p=.008
O1		t=2.630 p=.011		t=-2.057 p=.044				t=-3.330 p=.001
O2		t=2.163 p=.034						t=-2.588 p=.012
P3		t=2.948 p=.004		t=-2.307 p=.024				t=-3.195 p=.002
P4		t=2.607 p=.011		t=-2.480 p=.016				t=-2.645 p=.010

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ		t=2.779 p=.007		t=-2.466 p=.016				t=-2.849 p=.006
T5		t=2.328 p=.023					t=-2.082 p=.041	t=-3.031 p=.003
T6		t=2.533 p=.014						t=-2.231 p=.029

* Note. For Theta/Beta Ratio df=66 (30/38)

T-Tests - Eyes Closed Task ADD/ADHD Combined (df = 66 (31/37))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.933 p=.005			t=-2.773 p=.007	t=-3.221 p=.002
C4		t=2.039 p=.045		t=-3.151 p=.002			t=-2.830 p=.006	t=-3.509 p=.001
CZ				t=-2.886 p=.005			t=-2.388 p=.020	t=-3.146 p=.002
F3		t=2.092 p=.040		t=-3.163 p=.002			t=-3.337 p=..001	t=-3.679 p=.000
F4		t=2.394 p=.020		t=-3.076 p=.003			t=-3.103 p=.003	t=-3.616 p=.001
F7		t=2.058 p=.044	t=2.556 p=.013	t=-3.130 p=.003	t=2.306 p=.023		t=-3.982 p=.000	t=-3.565 p=.001
F8		t=2.486 p=.015		t=-2.781 p=.007	t=2.596 p=.011		t=-3.445 p=.001	t=-4.148 p=.000
FP1		t=2.555 p=.013	t=2.042 p=.045	t=-2.716 p=.008	t=3.075 p=.003		t=-3.541 p=.001	t=-4.295 p=.000
FP2		t=2.366 p=.021		t=-2.696 p=.009	t=2.199 p=.029		t=-3.424 p=.001	t=-3.886 p=.000
FZ		t=2.421 p=.018		t=-2.956 p=.004			t=-3.003 p=.004	t=-3.914 p=.000
O1				t=-2.902 p=.005			t=-3.646 p=.001	t=-4.116 p=.000
O2				t=-2.402 p=.019			t=-2.847 p=.006	t=-3.223 p=.002
P3		t=2.050 p=.044		t=-2.443 p=.017			t=-2.905 p=.005	t=-3.686 p=.000
P4				t=-2.552 p=.013			t=-2.577 p=.012	t=-3.737 p=.000

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ				t=-2.475 p=.016			t=-2.578 p=.012	t=-3.249 p=.002
T3				t=-2.190 p=.032			t=-3.137 p=.003	t=-3.076 p=.003
T4				t=-2.450 p=.017			t=-2.480 p=.013	t=-2.797 p=.007
T5				t=-2.707 p=.009			t=-4.165 p=.000	t=-4.244 p=.000
T6				t=-2.063 p=.043			t=-2.309 p=.024	t=-3.112 p=.003

* Note. For Theta/Beta Ratio df=65 (30/37)

T-Tests - TOVA First Half Task ADD/ADHD Combined (df = 66 (31/37))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.108 p=.040	t=2.108 p=.040				t=-2.662 p=.010	t=-2.736 p=.008
C4		t=2.779 p=.007	t=2.462 p=.016				t=-2.072 p=.042	t=-3.183 p=.002
CZ		t=2.232 p=.029					t=-2.032 p=.046	t=-2.699 p=.009
F3		t=2.896 p=.005	t=2.782 p=.007	t=-2.480 p=.016			t=-3.521 p=.001	t=-3.744 p=.000
F4		t=2.900 p=.005	t=2.361 p=.021	t=-2.230 p=.029			t=-2.619 p=.011	t=-3.362 p=.001
F7		t=2.729 p=.008		t=-2.284 p=.026			t=-2.596 p=.012	t=-2.640 p=.010
F8		t=2.213 p=.030				t=2.037 p=.046	t=-2.084 p=.041	t=-2.394 p=.020
FP1		t=3.444 p=.001	t=2.201 p=.031	t=-2.103 p=.039			t=-2.929 p=.005	t=-3.935 p=.000
FP2		t=2.954 p=.004					t=-2.092 p=.040	t=-2.881 p=.005
FZ		t=2.904 p=.005	t=2.613 p=.011	t=-2.112 p=.038			t=-2.745 p=.008	t=-3.285 p=.002
O1		t=2.122 p=.038					t=-2.703 p=.009	t=-3.637 p=.001
O2		t=2.121 p=.038					t=-2.131 p=.037	t=-3.285 p=.002
P3		t=2.625 p=.011	t=2.114 p=.038				t=-2.469 p=.016	t=-3.351 p=.002
P4		t=2.670 p=.010	t=2.470 p=.016					t=-3.196 p=.002

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ		t=2.459 p=.017					t=-2.316 p=.030	t=-3.088 p=.003
T3		t=2.589 p=.012					t=-2.880 p=.005	t=-2.928 p=.005
T5		t=2.255 p=.028					t=-3.033 p=.003	t=-3.272 p=.002
T6		t=2.080 p=.042						t=-2.477 p=.016

* Note. For Theta/Beta Ratio df= 65 (30/37)

T-Tests - TOVA Second Half Task ADD/ADHD Combined (df = 62 (29/35))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.108 p=.039	t=2.678 p=.009	t=-2.369 p=.021			t=-2.202 p=.031	t=-2.369 p=.021
C4		t=2.419 p=.018	t=2.638 p=.011	t=-2.592 p=.012				t=-2.810 p=.007
CZ		t=2.220 p=.030	t=2.069 p=.043	t=-2.478 p=.016				t=-2.511 p=.015
F3		t=2.953 p=.004	t=3.415 p=.001	t=-2.641 p=.010			t=-2.796 p=.007	t=-3.526 p=.001
F4		t=2.646 p=.010	t=2.350 p=.022					t=-2.977 p=.004
F8								t=-2.106 p=.039
FP1		t=2.716 p=.009						t=-2.716 p=.009
FP2		t=2.674 p=.010						t=-2.696 p=.009
FZ		t=3.056 p=.003	t=2.589 p=.012	t=-2.145 p=.036			t=-2.459 p=.017	t=-3.424 p=.001
O1							t=-2.085 p=.041	t=-2.741 p=.008
O2								t=-2.247 p=.028
P3		t=2.376 p=.021	t=2.251 p=.028	t=-2.187 p=.033			t=-2.545 p=.013	t=-3.247 p=.002
P4		t=2.538 p=.014	t=2.085 p=.041	t=-2.658 p=.010			t=-2.000 p=.050	t=-3.346 p=.001
PZ		t=2.224 p=.030		t=-2.247 p=.028			t=-2.016 p=.048	t=-2.563 p=.013

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5		t=2.205 p=.031					t=-2.898 p=.005	t=-3.017 p=.004
T6		t=2.231 p=.029						t=-2.670 p=.010

* Note. For Theta/Beta Ratio df= 61 (28/35)

T-Tests - Reading Task ADD/ADHD Combined (df = 65 (30/37))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3								t=-2.082 p=.041
C4								t=-2.256 p=.027
F3			t=2.094 p=.040		t=2.385 p=.018		t=-2.583 p=.012	t=-2.697 p=.009
F4							t=-2.258 p=.027	t=-2.643 p=.010
F7							t=-2.052 p=.044	t=-2.038 p=.046
F8							t=-2.120 p=.038	t=-2.171 p=.034
FP1		t=2.212 p=.031	t=2.697 p=.009		t=2.406 p=.016		t=-3.435 p=.001	t=-3.612 p=.001
FP2		t=2.091 p=.040					t=-2.394 p=.020	t=-3.015 p=.004
FZ								t=-2.355 p=.022
O1								t=-2.214 p=.031
P4								t=-2.168 p=.034

* Note. For Theta/Beta Ratio df= 64 (29/37)

T-Tests - First Rotation Task ADD/ADHD Combined (df = 58 (27/33))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.075 p=.042	t=-2.106 p=.040				t=-2.254 p=.028
C4			t=2.232 p=.030					t=-2.161 p=.035
CZ			t=2.051 p=.048					
F3			t=2.733 p=.009		t=2.089 p=.043		t=-2.262 p=.027	t=-2.582 p=.012
F4			t=2.984 p=.004	t=-2.508 p=.015				t=-2.219 p=.030
F7		t=2.306 p=.025		t=-2.402 p=.021			t=-2.250 p=.028	t=-2.716 p=.009
F8		t=2.736 p=.008	t=2.026 p=.047	t=-2.893 p=.005			t=-2.328 p=.023	t=-2.714 p=.009
FP1			t=2.173 p=.034	t=-2.005 p=.005			t=-2.094 p=.041	t=-2.310 p=.025
FP2		t=2.670 p=.010	t=3.068 p=.003	t=-2.645 p=.010	t=2.233 p=.030		t=-2.470 p=.020	t=-3.192 p=.002
FZ			t=3.117 p=.003	t=-2.161 p=.035				t=-2.129 p=.038
O1				t=-2.064 p=.044			t=-2.849 p=.006	t=-3.632 p=.001
O2		t=2.248 p=.028		t=-2.393 p=.020			t=-2.360 p=.022	t=-3.578 p=.001
P3		t=2.153 p=.036	t=2.238 p=.029					t=-2.776 p=.007
P4		t=2.631 p=.011	t=2.353 p=.022	t=-2.276 p=.027				t=-2.855 p=.006

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ		t=2.206 p=.031		t=-2.148 p=.036				t=-2.570 p=.013
T5							t=-2.057 p=.044	t=-2.790 p=.007
T6		t=2.161 p=.035						t=-2.691 p=.009

* Note. For Theta/Beta Ratio df= 57 (26/33)

T-Tests - First Select Task ADD/ADHD Combined (df = 47 (21/28))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.697 p=.010				t=-2.023 p=.049
C4			t=2.058 p=.045	t=-2.180 p=.035				t=-2.235 p=.030
F3					t=2.352 p=.026			
F4			t=2.584 p=.013		t=2.149 p=.036		t=-2.095 p=.042	
F8							t=-2.253 p=.029	t=-2.069 p=.044
FP2							t=-2.079 p=.043	t=-2.110 p=.040
FZ			t=2.364 p=.022					
O1				t=-2.511 p=.016			t=-2.697 p=.010	t=-2.701 p=.010
O2				t=-2.677 p=.010			t=-2.064 p=.045	t=-2.448 p=.018
P3				t=-2.618 p=.012				
P4				t=-2.349 p=.023				t=-2.114 p=.040
PZ				t=-2.342 p=.024				
T6				t=-2.258 p=.029				

* Note. For Theta/Beta Ratio df= 46 (20/28)

T-Tests - Listening Task ADD/ADHD Combined (df = 69 (31/40))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.053 p=.044					t=-2.663 p=.010
C4								t=-2.418 p=.018
CZ								t=-2.039 p=.045
F3		t=2.134 p=.036	t=2.425 p=.018				t=-2.420 p=.018	t=-3.214 p=.002
F4								t=-2.478 p=.016
F7		t=2.014 p=.048					t=-2.129 p=.037	t=-2.455 p=.017
FP1		t=2.441 p=.017					t=-2.396 p=.018	t=-2.963 p=.004
FP2		t=2.387 p=.020						t=-2.599 p=.011
FZ								t=-2.571 p=.012
O1								t=-2.651 p=.010
P3								t=-2.797 p=.007
P4								t=-2.437 p=.017
PZ								t=-2.352 p=.022
T3								t=-2.190 p=.032

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-2.040 p=.045	t=-2.824 p=.006

* Note. For Theta/Beta Ratio df= 68 (30/40)

T-Tests - Second Rotation Task ADD/ADHD Combined (df = 61 (29/34))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.884 p=.005					
C4			t=2.249 p=.028					
CZ			t=2.155 p=.035					
F3			t=2.064 p=.043					
F4			t=2.698 p=.009					
F7								t=-2.218 p=.030
F8		t=2.119 p=.038	t=2.159 p=.035	t=-2.274 p=.026	t=2.279 p=.025		t=-2.318 p=.024	t=-2.516 p=.015
FP1								t=-2.022 p=.048
FP2		t=2.335 p=.023	t=2.066 p=.043	t=-2.027 p=.047	t=2.240 p=.029		t=-2.309 p=.024	t=-2.598 p=.012
FZ			t=2.743 p=.008					t=-2.175 p=.034
O1								t=-2.196 p=.032
P3			t=2.258 p=.028					t=-2.014 p=.048
P4			t=2.127 p=.038					

* Note. For Theta/Beta Ratio df= 60 (28/34)

T-Tests - Second Select Task ADD/ADHD Combined (df = 68 (30/40))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.024 p=.047	t=2.569 p=.013					t=-3.273 p=.002
C4		t=2.265 p=.027						t=-3.015 p=.004
CZ		t=2.201 p=.031						t=-2.966 p=.007
F3					t=2.279 p=.022		t=-2.529 p=.014	t=-2.298 p=.025
F4		t=2.264 p=.027	t=2.273 p=.026				t=-2.117 p=.038	t=-2.975 p=.004
F8							t=-2.159 p=.034	t=-2.493 p=.015
FP1		t=2.027 p=.047					t=-2.284 p=.026	t=-2.493 p=.015
FP2							t=-2.038 p=.046	t=-2.337 p=.022
FZ							t=-2.038 p=.045	t=-2.880 p=.005
O1							t=-2.844 p=.006	t=-3.578 p=.001
O2							t=-2.519 p=.014	t=-2.880 p=.005
P3		t=2.446 p=.017						t=-3.613 p=.001
P4		t=2.407 p=.019						t=-3.341 p=.001
PZ		t=2.205 p=.031						t=-3.190 p=.002

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5		t=2.173 p=.033					t=-2.048 p=.045	t=-3.012 p=.002
T6								t=-2.643 p=.010

* Note. For Theta/Beta Ratio df= 67 (29/40)

T-Tests - TOVA First Quarter Task ADD/ADHD Combined (df = 65 (30/37))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3			t=2.373 p=.021		t=2.390 p=.023		t=-2.634 p=.011	t=-2.584 p=.012
F4			t=2.145 p=.036					t=-2.166 p=.034
F7							t=-2.542 p=.013	t=-2.165 p=.034
F8							t=-2.165 p=.034	
FP1		t=2.619 p=.011	t=2.997 p=.004		t=2.169 p=.034		t=-3.178 p=.002	t=-3.650 p=.001
FP2		t=2.165 p=.034	t=2.052 p=.044				t=-2.579 p=.012	t=-2.681 p=.009
FZ			t=2.041 p=.045					t=-2.281 p=.026
O1								t=-2.199 p=.031
T3		t=2.171 p=.034					t=-2.201 p=.031	t=-2.461 p=.017

* Note. For Theta/Beta Ratio df= 64 (29/37)

T-Tests - TOVA Second Quarter Task ADD/ADHD Combined (df = 56 (25/33))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.118 p=.039				t=-2.458 p=.017	t=-2.365 p=.022
C4			t=2.189 p=.033					t=-2.528 p=.014
F3			t=2.643 p=.011	t=-2.404 p=.020			t=-2.900 p=.005	t=-2.855 p=.006
F4			t=2.144 p=.036	t=-2.188 p=.033			t=-2.373 p=.021	t=-2.651 p=.010
F8								t=-2.055 p=.045
FP1								t=-2.603 p=.012
FP2								t=-2.154 p=.036
FZ			t=2.431 p=.019				t=-2.314 p=.024	t=-2.309 p=.025
O1				t=-2.131 p=.037			t=-2.166 p=.035	t=-2.773 p=.008
O2				t=-2.334 p=.023				t=-2.417 p=.019
P3								t=-2.735 p=.008
P4				t=-2.096 p=.041				t=-2.424 p=.019
PZ								t=-2.376 p=.021
T3							t=-2.236 p=.029	t=-2.020 p=.048

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-2.752 p=.008	t=-3.003 p=.004
T6								t=-2.140 p=.037

* Note. For Theta/Beta Ratio df= 55 (24/33)

T-Tests - TOVA Third Quarter Task ADD/ADHD Combined (df = 45 (22/25))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.535 p=.015	t=-2.356 p=.023			t=-2.168 p=.036	
C4			t=2.531 p=.015	t=-2.467 p=.018				
CZ			t=2.070 p=.044	t=-2.461 p=.018				
F3			t=3.318 p=.002	t=-2.869 p=.006	t=2.469 p=.018		t=-2.793 p=.008	t=-2.713 p=.010
F4		t=2.065 p=.045	t=2.906 p=.006	t=-2.725 p=.009	t=2.198 p=.032		t=-2.342 p=.024	t=-2.642 p=.012
F8			t=2.387 p=.021	t=-2.215 p=.032			t=-2.254 p=.029	t=-2.103 p=.041
FP1		t=2.118 p=.040	t=2.236 p=.030	t=-2.194 p=.033			t=-2.506 p=.016	t=-2.873 p=.006
FP2		t=2.312 p=.025					t=-2.050 p=.047	t=-2.847 p=.007
FZ			t=2.828 p=.007	t=-2.562 p=.014	t=2.019 p=.050			
O1								t=-2.017 p=.050
P3				t=-2.066 p=.045			t=-2.724 p=.010	
P4				t=-2.180 p=.035				
T3				t=-2.065 p=.045			t=-2.377 p=.022	
T4				t=-2.169 p=.035				t=-2.343 p=.024

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-2.667 p=.011	

* Note. For Theta/Beta Ratio df= 44 (21/25)

T-Tests - TOVA Fourth Quarter Task ADD/ADHD Combined (df = 39 (20/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3								t=-2.556 p=.015
C4			t=2.232 p=.032				t=-2.798 p=.008	t=-2.573 p=.014
CZ							t=-2.139 p=.039	t=-2.214 p=.033
F3		t=2.230 p=.032					t=-2.170 p=.036	t=-2.742 p=.009
F4								t=-2.598 p=.013
FP1		t=2.436 p=.020						t=-2.566 p=.014
FP2		t=2.150 p=.038				t=2.108 p=.042		t=-2.356 p=.024
FZ								t=-2.358 p=.024
O1							t=-2.416 p=.021	t=-2.301 p=.027
O2								t=-2.065 p=.046
P3							t=-3.304 p=.004	t=-3.093 p=.004
P4							t=-2.801 p=.008	t=-2.681 p=.011
PZ							t=-3.319 p=.002	t=-2.861 p=.007
T4							t=-2.219 p=.032	

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-3.254 p=.002	t=-3.032 p=.004
T6								t=-2.366 p=.023

* Note. For Theta/Beta Ratio df= 38 (19/21)

T-Tests - Computer Reading Task ADD/ADHD Combined (df = 65 (32/35))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.740 p=.008					t=-2.215 p=.028
C4			t=2.309 p=.024					
F3			t=2.543 p=.013		t=2.180 p=.035			t=-2.054 p=.044
FZ			t=2.191 p=.032					
O1							t=-2.618 p=.011	t=-2.561 p=.013
P3			t=2.191 p=.032					t=-2.226 p=.030
P4			t=2.106 p=.039					t=-2.006 p=.049
T5		t=2.300 p=.025	t=2.047 p=.045				t=-2.501 p=.015	t=-3.062 p=.003

* Note. For Theta/Beta Ratio df= 64 (31/35)

Appendix O

T-Tests - Eyes Open Task Control/ADD % (df = 46 (31/17))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
CZ								t=-2.147 p=.037
F3								t=-2.659 p=.034
F4								t=-2.479 p=.039
FZ								t=-2.273 p=.033
O1								t=-2.299 p=.032
P4								t=-2.322 p=.025
PZ								t=-2.253 p=.029

* Note. For Theta/Beta Ratio df=45 (30/17)

T-Tests - Eyes Closed Task Control/ADD % (df = 46 (31/17))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.712 p=.010				
C4				t=-2.775 p=.008				t=-2.111 p=.040
CZ				t=-2.643 p=.011				
F3				t=-2.751 p=.008			t=-2.299 p=.026	t=-2.426 p=.019
F4				t=-2.748 p=.009			t=-2.199 p=.033	t=-2.393 p=.021
F7				t=-2.411 p=.021			t=-2.504 p=.016	t=-2.274 p=.028
F8				t=-2.226 p=.031			t=-2.202 p=.033	t=-2.641 p=.011
FP1							t=-2.794 p=.008	t=-2.958 p=.005
FP2				t=-2.063 p=.045			t=-2.604 p=.012	t=-2.563 p=.014
FZ				t=-2.778 p=.008			t=-2.190 p=.034	t=-2.715 p=.009
O1				t=-2.986 p=.005			t=-2.863 p=.006	t=-2.967 p=.005
O2				t=-2.884 p=.006			t=-2.586 p=.014	t=-2.246 p=.030
P3				t=-2.276 p=.028			t=-2.139 p=.038	t=-2.323 p=.025
P4				t=-2.270 p=.028				t=-2.426 p=.019

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ				t=-2.263 p=.029				
T5				t=-2.461 p=.019			t=-2.995 p=.004	t=-2.589 p=.017

* Note. For Theta/Beta Ratio df=45 (30/17)

T-Tests - TOVA First Half Task Control/ADD % (df = 45 (31/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3							t=-2.095 p=.042	
C4								t=-2.459 p=.018
F3			t=2.529 p=.015				t=-2.609 p=.012	t=-2.676 p=.010
F4			t=2.127 p=.039				t=-2.186 p=.034	t=-2.341 p=.019
F7							t=-2.816 p=.007	t=-2.390 p=.021
F8							t=-2.120 p=.040	
FP1							t=-2.506 p=.016	t=-2.562 p=.019
FP2							t=2.108 p=.040	t=-2.165 p=.042
FZ							t=-2.122 p=.039	t=2.388 p=.021
O1			t=2.181 p=.034				t=-2.639 p=.011	t=-3.382 p=.002
O2							t=-2.393 p=.021	t=-3.323 p=.002
P3							t=-2.063 p=.045	t=-2.569 p=.014
P4								t=-2.503 p=.016
PZ								t=-2.332 p=.024

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-2.104 p=.041	t=-2.084 p=.050
T6								t=-2.036 p=.048

* Note. For Theta/Beta Ratio df= 44(29/16)

T-Tests - TOVA Second Half Task Control/ADD % (df = 43 (29/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.109 p=.041		t=2.066 p=.045			
C4			t=2.139 p=.038					t=-2.144 p=.038
F3			t=2.130 p=.039		t=2.174 p=.035		t=-2.057 p=.046	t=-2.169 p=.036
FZ					t=2.035 p=.048			t=-2.183 p=.045
O1			t=2.251 p=.030				t=-2.504 p=.016	t=-2.642 p=.012
O2								t=-2.021 p=.050
P3			t=2.244 p=.030					t=-2.116 p=.047
P4								t=-2.409 p=.020
T5			t=2.251 p=.030				t=-2.143 p=.038	
T6								t=-2.028 p=.049

* Note. For Theta/Beta Ratio df= 42 (28/16)

T-Tests - Reading Task Controls/ADD % (df = 42 (30/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3			t=2.132 p=.039		t=2.200 p=.033		t=-2.026 p=.049	t=-2.173 p=.044
F4								t=-2.156 p=.014
F7			t=2.586 p=.013		t=2.100 p=.042		t=-2.560 p=.014	t=-2.403 p=.028
F8					t=2.137 p=.041		t=-2.060 p=.048	t=-2.310 p=.026
FP1			t=2.614 p=.012		t=2.606 p=.013		t=-2.891 p=.006	t=-2.808 p=.012
FP2					t=2.259 p=.029		t=-2.205 p=.036	t=-2.844 p=.007

* Note. For Theta/Beta Ratio df= 41 (29/14)

T-Tests - First Rotation Task Controls/ADD % (df = 39 (27/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.236 p=.031	t=-2.072 p=.045	t=2.264 p=.029			t=-2.282 p=.028
C4			t=2.133 p=.039					
F3			t=2.594 p=.013		t=2.647 p=.012			
F4			t=2.479 p=.018					
F7				t=-2.100 p=.042	t=2.479 p=.018		t=-2.133 p=.039	t=-2.635 p=.012
F8								t=-2.072 p=.074
FP1					t=2.636 p=.012			
FP2			t=2.386 p=.022	t=-2.119 p=.043	t=2.636 p=.012			t=-2.446 p=.019
FZ			t=2.574 p=.014		t=2.246 p=.030			
O1								t=-2.547 p=.021
O2								t=-2.378 p=.029
P3			t=2.308 p=.026	t=-2.050 p=.047				t=-2.204 p=.042
P4			t=2.446 p=.019					t=-2.210 p=.040
T5								t=-2.364 p=.032

* Note. For Theta/Beta Ratio df= 38 (26/14)

T-Tests - First Select Task Control/ADD % (df = 33 (21/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
CZ	t=2.277 p=.030							
FZ	t=2.273 p=.030							
O1				t=-2.184 p=.036				

* Note. For Theta/Beta Ratio df= 32 (20/13)

T-Tests - Listening Task Control/ADD % (df = 35 (31/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.602 p=.013					t=-2.721 p=.009
C4								t=-2.301 p=.026
F3			t=2.499 p=.016				t=-2.142 p=.038	t=-3.061 p=.004
F4			t=2.112 p=.040					t=-2.504 p=.016
F7								t=-2.448 p=.023
FP1							t=-2.047 p=.046	t=-2.482 p=.021
FP2								t=-2.426 p=.019
FZ								t=-2.239 p=.036
O1								t=-2.826 p=.007
O2								t=-2.468 p=.018
P3								t=-2.361 p=.028
P4								t=-2.465 p=.018
PZ								t=-2.409 p=.020
T5								t=-2.156 p=.043

* Note. For Theta/Beta Ratio df= 34 (30/16)

T-Tests - Second Rotation Task Control/ADD % (df = 41 (29/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.631 p=.012		t=2.029 p=.049			t=-2.310 p=.026
F3			t=2.439 p=.019		t=2.985 p=.005			
F4			t=2.085 p=.043		t=2.356 p=.023			
F7			t=2.529 p=.015		t=2.303 p=.026		t=-2.327 p=.025	t=-2.231 p=.039
F8					t=2.337 p=.024			
FP1					t=2.170 p=.036			
FP2					t=2.248 p=.030			
FZ			t=2.350 p=.024		t=2.292 p=.027			

* Note. For Theta/Beta Ratio df= 40 (28/14)

T-Tests - Second Select Task Control/ADD % (df = 44 (30/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3					t=2.044 p=.047			
FP1								t=-2.122 p=.040
FZ								t=-2.050 p=.046
O1								t=-2.661 p=.015

* Note. For Theta/Beta Ratio df= 43 (29/16)

T-Tests - TOVA First Quarter Task Control/ADD % (df = 43 (30/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3								t=-2.124 p=.040
F4								t=-2.061 p=.046
F7							t=-2.948 p=.005	
F8							t=-2.100 p=.042	
FP1					t=2.167 p=.036		t=-2.807 p=.007	t=-2.547 p=.020
FP2							t=-2.636 p=.012	t=-2.775 p=.008
FZ								t=-2.029 p=.049
O1								t=-2.401 p=.021
O2								t=-2.346 p=.024

* Note. For Theta/Beta Ratio df= 42 (29/15)

T-Tests - TOVA Second Quarter Task Control/ADD % (df = 38 (25/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.150 p=.038				t=2.327 p=.025	t=-2.113 p=.047
C4								t=-2.201 p=.034
CZ								t=-2.247 p=.031
F3			t=2.221 p=.032		t=2.074 p=.045			
F7							t=-2.055 p=.047	
O1		t=2.558 p=.015		t=-2.075 p=.046			t=-2.991 p=.005	t=-3.224 p=.005
O2				t=-2.312 p=.026			t=-2.456 p=.019	t=-2.828 p=.011
P3			t=2.541 p=.015				t=-2.380 p=.022	t=-2.410 p=.026
P4				t=2.357 p=.024				t=-2.230 p=.037
PZ				t=2.074 p=.045			t=-2.066 p=.046	t=-2.134 p=.045
T5				t=-2.656 p=.011			t=-2.815 p=.008	t=-2.688 p=.015

* Note. For Theta/Beta Ratio df= 37 (24/15)

T-Tests - TOVA Third Quarter Task Control/ADD % (df = 32 (21/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3					t=2.256 p=.031			
C4					t=2.438 p=.022			
F3			t=2.362 p=.024		t=2.817 p=.009			
F4			t=2.078 p=.046		t=2.357 p=.025			
F8			t=2.043 p=.049					
FZ					t=2.386 p=.023			
O1			t=2.069 p=.046				t=-2.345 p=.025	t=-2.207 p=.035
P3			t=2.154 p=.039					
T5			t=2.040 p=.049				t=-2.050 p=.048	

* Note. For Theta/Beta Ratio df= 31 (20/13)

Table B14

T-Tests - TOVA Fourth Quarter Task Control/ADD % (df = 39 (20/21))

No significant data

* Note. For Theta/Beta Ratio df= 38 (19/21)

T-Tests - Computer Reading Task Control/ADD % (df = 45 (32/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.671 p=.010		t=2.203 p=.033		t=-2.042 p=.047	
C4			t=2.198 p=.040					
F3			t=2.230 p=.038					
O1							t=-2.452 p=.018	t=-2.249 p=.037
P3			t=2.204 p=.033					t=-2.093 p=.050
P4			t=2.123 p=.039					
T4		t=2.014 p=.050	t=2.057 p=.046				t=-2.113 p=.040	t=-2.460 p=.024

* Note. For Theta/Beta Ratio df= 44 (31/15)

Appendix P

T-Tests - Eyes Open Task Control/ADHD % (df = 53 (31/24))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4				t=-2.044 p=.047				t=-2.061 p=.044
CZ		t=2.077 p=.043						t=-2.038 p=.047
F3								t=-2.245 p=.029
F7								t=-2.039 p=.047
FP2								t=-2.043 p=.046
O1								t=-2.579 p=.013
P3								t=-2.538 p=.014
PZ				t=-2.049 p=.045				t=-2.139 p=.037
T4		t=2.268 p=.027						t=-2.210 p=.032
T5							t=-2.466 p=.017	t=-2.717 p=.009

* Note. For Theta/Beta Ratio df= 52 (30/24)

T-Tests - Eyes Closed Task Control/ADHD Combined (df = 51 (31/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.087 p=.042				t=-2.378 p=.024
C4		t=2.204 p=.032		t=-2.369 p=.022			t=-2.302 p=.025	t=-3.098 p=.004
CZ				t=-2.109 p=.040			t=-2.016 p=.049	t=-2.789 p=.009
F3				t=-2.334 p=.024			t=-2.508 p=.015	t=-2.816 p=.007
F4		t=2.195 p=.033		t=-2.305 p=.025			t=-2.370 p=.022	t=-2.985 p=.004
F7				t=-2.321 p=.024	t=2.195 p=.033		t=-3.038 p=.004	t=-2.639 p=.011
F8				t=-2.235 p=.028	t=2.293 p=.026		t=-2.746 p=.008	t=-3.462 p=.001
FP1		t=2.349 p=.023		t=-2.341 p=.023			t=-2.902 p=.005	t=-3.557 p=.001
FP2		t=2.066 p=.044		t=-2.232 p=.030			t=-2.510 p=.015	t=-3.063 p=.004
FZ		t=2.097 p=.041		t=-2.129 p=.038			t=-2.242 p=.029	t=-3.144 p=.003
O1				t=-2.178 p=.034			t=-3.061 p=.004	t=-3.215 p=.002
O2								t=-2.695 p=.010
P3							t=-2.284 p=.027	t=-2.992 p=.004
P4							t=-2.187 p=.034	t=-3.081 p=.004

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
PZ							t=-2.308 p=.025	t=-2.954 p=.005
T3							t=-2.532 p=.014	t=-2.313 p=.027
T4		t=2.925 p=.005		t=-2.568 p=.014			t=-2.845 p=.007	t=-3.321 p=.002
T5				t=-2.181 p=.034			t=-3.472 p=.001	t=-3.438 p=.001
T6							t=-2.006 p=.050	t=-2.825 p=.008

* Note. For Theta/Beta Ratio df= 50 (30/22)

T-Tests - TOVA First Half Task Control/ADHD % (df = 53 (31/24))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3			t=2.115 p=.039				t=-2.465 p=.017	t=-2.550 p=.016
F4		t=2.053 p=.045						t=-2.282 p=.027
FP1		t=2.943 p=.005					t=-2.223 p=.031	t=-3.252 p=.002
FP2		t=2.118 p=.039						
FZ			t=2.098 p=.041					t=-2.022 p=.048
P3								t=-2.016 p=.049
T3		t=2.467 p=.017					t=-2.727 p=.009	t=-2.535 p=.014
T4		t=2.304 p=.025					t=-2.237 p=.030	
T5								t=-2.134 p=.038

* Note. For Theta/Beta Ratio df= 52 (30/24)

T-Tests - TOVA Second Half Task Control/ADHD % (df = 48 (29/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3		t=2.285 p=.027	t=2.690 p=.010	t=-2.026 p=.048				t=-2.512 p=.016
FP1		t=2.346 p=.023						
FP2		t=2.109 p=.040						
FZ		t=2.097 p=.042	t=2.019 p=.049					t=-2.105 p=.041

* Note. For Theta/Beta Ratio df= 47 (28/21)

Table C5

T-Tests - Reading Task Control/ADHD % (df = 51 (30/23))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3							t=-2.297 p=.026	t=-2.113 p=.040
F4								t=-2.182 p=.034
FP1			t=2.015 p=.049				t=-2.869 p=.006	t=-2.979 p=.004
FP2		t=2.034 p=.047						t=-2.340 p=.023
T3							t=-2.343 p=.023	

* Note. For Theta/Beta Ratio df= 50 (29/23)

T-Tests - First Rotation Task Control/ADHD % (df = 46 (27/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3	t=2.458 p=.018							
F3			t=2.707 p=.010					
F4			t=2.708 p=.010					
F7								t=-2.012 p=.050
F8		t=2.593 p=.013	t=2.069 p=.044	t=-2.504 p=.016			t=-2.176 p=.035	t=-2.405 p=.020
FP1			t=2.034 p=.048					
FP2		t=2.676 p=.010	t=2.582 p=.013	t=-2.088 p=.042			t=-2.193 p=.033	t=-2.891 p=.006
FZ			t=2.668 p=.011					
O1							t=-2.026 p=.049	t=-2.585 p=.013
O2								t=-2.628 p=.012
T4		t=2.235 p=.030						t=-2.139 p=.038

* Note. For Theta/Beta Ratio df= 45 (26/21)

T-Tests - First Select Task Control/ADHD % (df = 35(21/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3				t=-2.387 p=.023			t=-2.184 p=.036	
C4		t=2.288 p=.028					t=-2.058 p=.047	t=-2.324 p=.026
F3							t=-2.129 p=.040	
F4			t=2.319 p=.026				t=-2.407 p=.021	
FZ			t=2.264 p=.030					
O1				t=-2.229 p=.033			t=-2.659 p=.012	t=-2.584 p=.014
O2				t=2.889 p=.007			t=-2.260 p=.030	t=-2.234 p=.032
P3				t=-2.519 p=.017				
P4				t=-2.133 p=.040				
PZ				t=-2.129 p=.040				
T4		t=2.066 p=.047						
T5			t=2.468 p=.019				t=-3.026 p=.005	t=-2.398 p=.022
T6		t=2.534 p=.016		t=-2.457 p=.019				t=-2.292 p=.028

* Note. For Theta/Beta Ratio df= 34(20/16)

T-Tests - Listening Task Control/ADHD % (df = 53 (31/24))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3								t=-2.519 p=.015
FP1							t=-2.025 p=.048	t=-2.310 p=.025
FP2								t=-2.003 p=.050
P3								t=-2.082 p=.042
T5								t=-2.326 p=.024

* Note. For Theta/Beta Ratio df= 52 (30/24)

Table C9

T-Tests - Second Rotation Task Control/ADHD % (df = 49 (29/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.311 p=.025					
F4			t=2.550 p=.014					
F8		t=2.411 p=.020	t=2.236 p=.030	t=-2.188 p=.034			t=-2.071 p=.044	t=-2.313 p=.025
FP2		t=2.530 p=.015					t=-2.061 p=.045	t=-2.451 p=.018
FZ			t=2.349 p=.023					

* Note. For Theta/Beta Ratio df= 48 (28/22)

T-Tests - Second Select Task Control/ADHD % (df =53 (30/25))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.212 p=.031	t=2.053 p=.045				t=-2.024 p=.048	t=-2.911 p=.005
C4		t=2.490 p=.016						t=-2.951 p=.005
CZ		t=2.215 p=.031						t=-2.424 p=.019
F3					t=2.014 p=.049		t=-2.148 p=.036	
F4		t=2.087 p=.042						t=-2.541 p=.014
FZ								t=-2.131 p=.038
O1							t=-2.119 p=.039	t=-2.367 p=.022
O2								t=-2.062 p=.044
P3								t=-2.778 p=.008
P4								t=-2.664 p=.010
PZ								t=-2.354 p=.022
T3								t=-2.131 p=.038
T5							t=-2.546 p=.014	t=-2.844 p=.006
T6								t=-2.403 p=.020

* Note. For Theta/Beta Ratio df= 52 (29/25)

T-Tests - TOVA First Quarter Task Control/ADHD % (df = 50 (30/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3					t=2.081 p=.043		t=-2.292 p=.026	t=-2.235 p=.030
FP1		t=2.803 p=.007	t=3.714 p=.001				t=-2.638 p=.011	t=-3.300 p=.002
T3		t=2.357 p=.023					t=-2.392 p=.021	t=-2.317 p=.025

* Note. For Theta/Beta Ratio df= 49 (29/22)

T-Tests - TOVA Second Quarter Task ADD/ADHD Combined (df = 41 (25/18))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4								t=-2.044 p=.048
F3			t=2.316 p=.026	t=-2.358 p=.023			t=-2.574 p=.014	t=-2.570 p=.014
F4			t=2.105 p=.041	t=-2.709 p=.010			t=-2.210 p=.033	t=-2.639 p=.012
FP1		t=2.107 p=.036						t=-2.551 p=.015
FZ		t=2.395 p=.021						t=-2.042 p=.048
T3				t=-2.356 p=.023			t=-2.299 p=.027	
T4								t=-2.108 p=.041
T5								t=-2.028 p=.049

* Note. For Theta/Beta Ratio df= 40 (24/18)

T-Tests - TOVA Third Quarter Task Control/ADHD % (df = 32 (21/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4			t=2.113 p=.042	t=-2.114 p=.041				
CZ				t=-2.089 p=.045				
F3			t=2.926 p=.006	t=-2.355 p=.026				t=-2.064 p=.047
F4			t=2.544 p=.016	t=-2.233 p=.032				t=-2.290 p=.029
F8			t=2.106 p=.044	t=-2.372 p=.025				
FP1		t=2.240 p=.032	t=2.364 p=.025					t=-2.511 p=.017
FP2		t=2.332 p=.026						t=-2.500 p=.018
FZ			t=2.615 p=.022	t=-2.225 p=.033				
T3				t=-2.336 p=.026			t=-2.307 p=.028	
T4				t=-2.056 p=.048				

* Note. For Theta/Beta Ratio df= 31 (20/13)

T-Tests - TOVA Fourth Quarter Task Control/ADHD % (df = 31 (20/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3								t=-2.100 p=.044
F3								t=-2.357 p=.025
F4								t=-2.185 p=.037
F7	t=2.836 p=.009							
FP2						t=2.223 p=.034		
P3							t=-2.316 p=.023	t=-2.272 p=.030

* Note. For Theta/Beta Ratio df= 30 (19/13)

Table C15

T-Tests - Computer Reading Task Control/ADHD % (df = 51 (32/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5							t=-2.023 p=.048	t=-2.337 p=.024

* Note. For Theta/Beta Ratio df= 50 (31/21)

Appendix Q

T-Tests - Eyes Open Task Control/ADHD Raw (df = 53 (31/24))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4		t=2.358 p=.020					t=-2.150 p=.048	
F4			t=2.267 p=.027					
O1			t=2.006 p=.050					
O2			t=2.072 p=.043					
P3			t=2.314 p=.025					
P4			t=2.255 p=.028					
PZ			t=2.181 p=.034					
T4			t=2.114 p=.039					

* Note. For Theta/Beta Ratio df= 52 (30/24)

T-Tests - Eyes Closed Task Control/ADHD Raw (df = 51 (31/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4		t=2.131 p=.038						
F7			t=2.135 p=.038					
FP1			t=2.352 p=.023	t=-2.211 p=.032				
T4			t=2.505 p=.015	t=-2.111 p=.040				

* Note. For Theta/Beta Ratio df= 50 (30/22)

Table D3

T-Tests - TOVA First Half Task Control/ADHD Raw (df = 53 (31/24))

No significant data

* Note. For Theta/Beta Ratio df= 52 (30/24)

Table D4

T-Tests - TOVA Second Half Task Control/ADHD Raw (df = 48 (29/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F3			t=2.169 p=.035					

* Note. For Theta/Beta Ratio df= 47 (28/21)

T-Tests - Reading Task Control/ADHD Raw (df = 51 (30/23))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
FP1		t=2.155 p=.036	t=2.153 p=.036					

* Note. For Theta/Beta Ratio df= 50 (29/23)

Table D6

T-Tests - First Rotation Task Control/ADHD Raw (df = 46 (27/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F4			t=2.103 p=.041					
FP2			t=2.060 p=.045					

* Note. For Theta/Beta Ratio df= 45 (26/21)

Table D7

T-Tests - First Select Task Control/ADHD Raw (df = 35 (21/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F8							t=-2.115 p=.042	

* Note. For Theta/Beta Ratio df= 34 (20/16)

Table D8

T-Tests - Listening Task Control/ADHD Raw (df = 53 (31/24))

No significant data

* Note. For Theta/Beta Ratio df= 52 (30/24)

Table D9

T-Tests - Second Rotation Task Control/ADHD Raw (df = 49 (29/22))

No significant data

* Note. For Theta/Beta Ratio df= 48 (28/22)

Table D10

T-Tests - Second Select Task Control/ADHD Raw (df = 53 (30/25))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.042 p=.046						
C4		t=2.095 p=.041						

* Note. For Theta/Beta Ratio df= 52 (29/25)

T-Tests - TOVA First Quarter Task Control/ADHD Raw (df = 50 (30/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
FP1		t=2.203 p=.033	t=2.258 p=.028					
T3		t=2.027 p=.048						

* Note. For Theta/Beta Ratio df= 49 (29/22)

Table D12

T-Tests - TOVA Second Quarter Task ADD/ADHD Raw (df = 41 (25/18))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
F7				t=-2.278 p=.028				

* Note. For Theta/Beta Ratio df= 40 (24/18)

T-Tests - TOVA Third Quarter Task Control/ADHD Raw (df = 33 (22/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4			t=2.358 p=.024					
F3			t=2.640 p=.013					
F4			t=2.620 p=.013					
FZ			t=2.610 p=.014					

* Note. For Theta/Beta Ratio df= 32 (21/13)

Table D14

T-Tests - TOVA Fourth Quarter Task Control/ADHD Raw (df = 31 (20/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4			t=2.313 p=.028					
F4		t=2.065 p=.048						
FZ		t=2.092 p=.045	t=2.487 p=.028					

* Note. For Theta/Beta Ratio df= 30 (19/13)

T-Tests - Computer Reading Task Control/ADHD Raw (df = 51 (32/21))

No significant data

* Note. For Theta/Beta Ratio df= 50(31/21)

Appendix R

Table E1

T-Tests - Eyes Open Task Control/ADD Raw (df = 45 (30/17))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C4			t=2.487 p=.017		t=2.140 p=.036			
F3		t=2.041 p=.047	t=2.487 p=.017					
F4			t=2.199 p=.033					
FZ			t=2.667 p=.011		t=2.051 p=.046			
O1		t=2.418 p=.020	t=2.427 p=.019					
P3		t=2.577 p=.013						
P4		t=2.132 p=.038	t=2.623 p=.012					
PZ			t=2.064 p=.045					

* Note. For Theta/Beta Ratio df= 43 (29/17)

T-Tests - Eyes Closed Task Control/ADD Raw (df = 46 (31/17))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3	t=2.094 p=.043		t=2.347 p=.024			t=2.111 p=.042		t=-2.091 p=.043
C4	t=2.087 p=.044		t=2.704 p=.010					t=-2.069 p=.046
F3			t=2.086 p=.044					
F4			t=2.226 p=.031		t=2.231 p=.029			
F7			t=2.020 p=.049		t=2.461 p=.020			
FP1		t=2.329 p=.024	t=2.859 p=.006		t=3.258 p=.001			t=-2.109 p=.040
FP2		t=2.191 p=.034	t=2.820 p=.007		t=3.187 p=.003			t=-2.038 p=.047
FZ			t=2.174 p=.036		t=2.235 p=.029			
O1		t=2.766 p=.008	t=3.187 p=.003		t=2.601 p=.012	t=2.498 p=.017		t=-2.181 p=.034
O2	t=2.185 p=.035	t=2.532 p=.015	t=2.786 p=.008		t=2.163 p=.037	t=2.692 p=.010		t=-2.532 p=.015
P3			t=3.190 p=.003		t=2.697 p=.010	t=2.098 p=.043		
P4			t=2.694 p=.010		t=2.521 p=.014			
PZ			t=2.359 p=.023		t=2.089 p=.043			
T3						t=2.085 p=.044		

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5	t=2.090 p=.044	t=2.932 p=.006	t=3.656 p=.001		t=2.531 p=.015	t=2.718 p=.010		t=-2.595 p=.013
T6			t=2.581 p=.013					

* Note. For Theta/Beta Ratio df= 45 (30/17)

T-Tests - TOVA First Half Task Control/ADD % (df = 45 (31/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.685 p=.011		t=2.090 p=.043			
C4			t=2.403 p=.022					
F3			t=2.949 p=.006		t=2.513 p=.014			
F4			t=2.323 p=.025		t=2.011 p=.049			
FZ			t=2.497 p=.017		t=2.169 p=.033			
O1		t=2.066 p=.047	t=3.483 p=.001		t=2.831 p=.008			
O2		t=2.054 p=.048	t=2.830 p=.008		t=2.127 p=.040			
P3			t=3.142 p=.003		t=2.169 p=.037			
P4			t=2.744 p=.009		t=2.122 p=.040			
PZ			t=2.158 p=.036					
T5			t=2.639 p=.011					

* Note. For Theta/Beta Ratio df= 44 (30/16)

T-Tests - TOVA Second Half Task Control/ADD Raw (df = 43 (29/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.424 p=.021		t=2.753 p=.007			
C4			t=2.458 p=.018		t=2.691 p=.011			
F3			t=2.325 p=.026		t=2.346 p=.023			
FZ					t=2.216 p=.030			
O1			t=3.144 p=.004		t=3.102 p=.006			
O2			t=2.516 p=.017					
P3			t=2.680 p=.010		t=2.689 p=.009			
P4			t=2.491 p=.017		t=2.716 p=.015			
PZ					t=2.216 p=.039			
T4			t=2.733 p=.009		t=2.078 p=.046			

* Note. For Theta/Beta Ratio df= 41 (28/16)

T-Tests - Reading Task Controls/ADD Raw (df = 41 (28/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.925 p=.006	t=-2.311 p=.026	t=2.901 p=.006	t=2.049 p=.047		t=-2.097 p=.042
C4		t=2.226 p=.032	t=2.940 p=.005		t=2.603 p=.015			t=-2.034 p=.048
F3		t=2.292 p=.027	t=3.384 p=.002		t=3.451 p=.001			t=-2.419 p=.020
F4		t=2.168 p=.036	t=3.455 p=.001		t=3.716 p=.000			t=-2.153 p=.037
F7		t=2.672 p=.011	t=3.552 p=.001		t=3.529 p=.001	t=2.612 p=.013		t=-2.933 p=.005
F8		t=2.435 p=.021	t=3.571 p=.001		t=3.568 p=.001	t=2.274 p=.028		t=-2.698 p=.010
FP1		t=3.170 p=.004	t=3.864 p=.001		t=3.812 p=.000	t=2.643 p=.012		t=-3.320 p=.002
FP2		t=2.636 p=.014	t=2.913 p=.007		t=3.511 p=.001			t=2.711 p=.011
FZ			t=2.610 p=.013		t=2.633 p=.014			
O1		t=2.503 p=.016	t=2.690 p=.010		t=2.150 p=.037			
O2		t=2.295 p=.027	t=2.917 p=.006		t=2.239 p=.031			
P3			t=2.825 p=.007		t=2.801 p=.009			
P4			t=2.789 p=.008		t=2.290 p=.027			
T3						t=2.011 p=.050		

* Note. For Theta/Beta Ratio df= 40 (27/14)

T-Tests - First Rotation Task Controls/ADD Raw (df = 39 (27/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3	t=2.171 p=.036		t=2.948 p=.006		t=3.341 p=.002			t=-2.136 p=.039
C4			t=2.584 p=.014		t=2.391 p=.022			
CZ					t=2.316 p=.023			
F3			t=3.155 p=.004		t=3.100 p=.004			
F4			t=2.791 p=.008		t=2.491 p=.019			
F7		t=2.093 p=.043	t=2.232 p=.034		t=2.792 p=.007			t=-2.127 p=.040
FP1			t=2.098 p=.042		t=2.768 p=.009			
FP2		t=2.077 p=.047	t=2.839 p=.008		t=2.940 p=.006			t=-2.164 p=.036
FZ			t=2.769 p=.009		t=3.092 p=.004			
O1	t=2.272 p=.009	t=2.617 p=.013	t=3.327 p=.002		t=3.711 p=.000	t=2.275 p=.028		t=-2.694 p=.041
O2	t=2.260 p=.029	t=2.414 p=.021	t=3.187 p=.003		t=3.511 p=.001			t=-2.565 p=.014
P3	t=2.033 p=.049	t=2.089 p=.043	t=3.155 p=.003		t=3.816 p=.000			t=-2.293 p=.027
P4			t=2.964 p=.005		t=3.322 p=.002			
PZ			t=2.356 p=.024		t=2.910 p=.006			

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
T5		t=2.610 p=.013	t=3.255 p=.002		t=3.249 p=.002	t=2.570 p=.014		t=-2.850 p=.007
T6			t=2.144 p=.038					

* Note. For Theta/Beta Ratio df= 38 (26/14)

T-Tests - First Select Task Control/ADD Raw (df = 32 (21/13))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3	t=2.835 p=.008							t=-2.170 p=.038
C4	t=2.234 p=.033							
CZ	t=2.532 p=.016							
F3	t=2.662 p=.012				t=2.380 p=.024			
F4	t=2.140 p=.046		t=2.602 p=.014		t=2.531 p=.016			
F7	t=2.223 p=.033				t=2.261 p=.031			t=-2.148 p=.039
FP1	t=2.339 p=.026							
FP2	t=2.445 p=.020	t=2.036 p=.050	t=2.064 p=.047		t=2.440 p=.020			t=-2.110 p=.043
FZ	t=2.176 p=.043		t=2.077 p=.046					
O1		t=2.352 p=.025						t=-2.063 p=.047
O2		t=2.143 p=.040						
P3	t=2.353 p=.025							
PZ	t=2.083 p=.045							

* Note. For Theta/Beta Ratio df= 31 (20/13)

T-Tests - Listening Task Control/ADD Raw (df = 43 (30/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.185 p=.034	t=3.221 p=.002		t=3.081 p=.004	t=2.054 p=.046		t=-2.300 p=.026
C4		t=2.075 p=.044	t=2.959 p=.005		t=2.296 p=.027	t=2.209 p=.033		t=-2.334 p=.024
F3		t=2.389 p=.021	t=3.087 p=.004		t=3.088 p=.004			t=-2.394 p=.021
F4		t=2.014 p=.050	t=2.877 p=.006		t=2.081 p=.042			t=-2.100 p=.042
F7						t=2.019 p=.050		
FP1		t=2.439 p=.019	t=2.126 p=.039		t=2.271 p=.029	t=2.194 p=.034		t=-2.320 p=.025
FZ			t=2.537 p=.015		t=2.406 p=.017			
O1		t=2.282 p=.028	t=2.717 p=.009			t=2.182 p=.035		
O2		t=2.181 p=.035	t=2.040 p=.048			t=2.207 p=.033		
P3		t=2.104 p=.041	t=3.021 p=.004		t=2.282 p=.028	t=2.014 p=.050		t=-2.016 p=.050
P4			t=2.726 p=.009			t=2.014 p=.050		
PZ			t=2.222 p=.032					

* Note. For Theta/Beta Ratio df= 42 (29/15)

T-Tests - Second Rotation Task Control/ADD Raw (df = 41(29/14))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3		t=2.141 p=.038	t=3.145 p=.003		t=3.571 p=.001			t=-2.189 p=.034
C4			t=2.345 p=.024		t=2.534 p=.015			
F3			t=2.709 p=.010		t=3.499 p=.001			
F4			t=2.415 p=.020		t=2.934 p=.005			
F7		t=2.074 p=.044	t=3.130 t=.004		t=3.169 p=.004			
F8			t=2.078 p=.044		t=2.681 p=.011			
FP1			t=2.065 p=.048		t=2.349 p=.023			
FP2			t=2.186 p=.037		t=2.456 p=.018			
FZ			t=2.385 p=.022		t=2.998 p=.005			
O1		t=2.321 p=.025	t=3.197 p=.003		t=3.515 p=.001			t=-2.598 p=.013
P3		t=2.259 p=.029	t=3.233 p=.002		t=3.561 p=.001			t=-2.529 p=.015
P4			t=2.457 p=.018		t=2.601 p=.014			
PZ			t=2.221 p=.032		t=2.598 p=.014			
T5		t=2.859 p=.007	t=3.289 p=.002		t=3.550 p=.001	t=2.251 p=.030		t=-3.088 p=.004

* Note. For Theta/Beta Ratio df= 40(28/14)

T-Tests - Second Select Task Control/ADD Raw (df = 41 (28/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.619 p=.012		t=2.686 p=.011			
C4			t=2.111 p=.041					
F3			t=2.508 p=.016		t=2.983 p=.006			
F4			t=2.037 p=.048		t=2.201 p=.033			
FP1		t=2.099 p=.043	t=2.168 p=.038		t=2.813 p=.009			
FZ					t=2.301 p=.025			
O1		t=2.432 p=.019	t=3.180 p=.003		t=2.711 p=.010			
P3			t=2.716 p=.010		t=2.611 p=.012			
T5		t=2.592 p=.013	t=2.261 p=.029		t=2.165 p=.038	t=2.296 p=.027		

* Note. For Theta/Beta Ratio df= 40 (27/15)

Table E11

T-Tests - TOVA First Quarter Task Control/ADD Raw (df = 42 (29/15))

No significant data

* Note. For Theta/Beta Ratio df= 41(28/14)

T-Tests - TOVA Second Quarter Task Control/ADD Raw (df =37 (24/15))

No significant data

* Note. For Theta/Beta Ratio df= 36(23/14)

Table E13

T-Tests - TOVA Third Quarter Task Control/ADD Raw (df = 32 (21/13))

No significant data

* Note. For Theta/Beta Ratio df= 31(20/13)

Table E14

T-Tests - TOVA Fourth Quarter Task ADD/ADHD Raw (df = 25((19/8))

No significant data

* Note. For Theta/Beta Ratio df= 24((18/8)

T-Tests - Computer Reading Task Control/ADD Raw (df = 45 (32/15))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio*
C3			t=2.545 p=.015		t=2.701 p=.009			
C4			t=2.335 p=.024					
F3			t=2.104 p=.041					
O1			t=2.189 p=.034					
P3			t=2.473 p=.017		t=2.333 p=.024			
P4			t=2.188 p=.034					
T5			t=2.162 p=.036					

* Note. For Theta/Beta Ratio df= 44 (31/15)

Appendix S

Table F1

T-Tests - Eyes Open Task ADD/ADHD % (df = 39 (17/24))

No significant data

Table F2

T-Tests - Eyes Closed Task ADD/ADHD % (df = 37 (17/22))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio
T4		t=2.397 p=.022						t=-2.095 p=.043

Table F3

T-Tests - TOVA First Half Task ADD/ADHD % (df = 38 (16/24))

No significant Data

Table F4

T-Tests - TOVA Second Half Task ADD/ADHD % (df = 35 (16/21))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio
C3					t=2.225 p=.033			
C4					t=2.032 p=.050			

T-Tests - Reading Task ADD/ADHD % (df = 35 (14/23))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio
F7		t=2.672 p=.011	t=3.552 p=.001		t=3.561 p=.001	t=2.612 p=.013		t=-2.933 p=.005

Table F6

T-Tests - First Rotation Task ADD/ADHD % (df = 33 (14/21))

No significant data

Table F7

T-Tests - First Select Task ADD/ADHD % (df = 27 (13/16))

Site	Alpha	Beta 1	Beta 2	Delta	EMG	SMR	Theta	Theta / Beta Ratio
T4		t=2.415 p=.023						

Table F8

T-Tests - Listening Task ADD/ADHD % (df = 38 (16/24))

No significant data

Table F9

T-Tests - Second Rotation Task ADD/ADHD % (df = 34(14/22))

No significant data

Table F10

T-Tests - Second Select Task ADD/ADHD % (df = 39 (16/25))

No significant data

Table F11

T-Tests - TOVA First Quarter Task ADD/ADHD % (df = 35 (15/22))

No significant data

Table F12

T-Tests - TOVA Second Quarter Task ADD/ADHD % (df = 31 (15/18))

No significant data

Table F13

T-Tests - TOVA Third Quarter Task ADD/ADHD % (df = 24 (13/13))

No significant data

Table F14

T-Tests - TOVA Fourth Quarter Task ADD/ADHD % (df = 19 (8/13))

No significant data

T-Tests - Computer Reading Task ADD/ADHD % (df = 34 (15/21))

No significant data