**The Utilization of Electroencephalography**

**For Brain Mapping and Neurofeedback**

**In Therapeutic Treatment of ADD and ADHD**

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Abstract

This work reviews attention deficit disorder in combination with a treatment approach utilizing EEG Neurofeedback, including the use of Neurofeedback as a process for normalizing brain wave function. A history of the EEG work is presented that shows the development of EEG Neurofeedback work with ADD/ADHD, providing support for the rationale of using Neurofeedback for successful treatment protocols for ADD/ADHD.

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According to the NIH Consensus Statement from the Consensus Development Program on the Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder (1998), attention deficit hyperactivity disorder (ADHD) is the most commonly diagnosed public health problem in children, with estimations that it affects 3 to 5 % of all school age children.  The NIH Consensus Statement described the core symptoms of ADHD as inappropriate levels of attention according to the individual's developmental level, problems with concentration, inappropriate activity, distractibility, and impulsivity.

## Impairments from ADHD

These symptoms have serious impairments that cause long-term problematic concerns not only in academic performance during childhood but also with vocational success as these individuals grow older.  ADHD individuals experience impairments that extend into every area of their lives including home, school, and work.  There are also social-emotional development problems that cause peer and social relationship difficulties.  All of these concerns have profound impacts on the lives of the individuals, their families, the schools they attend, and society.

Murphy, Barkley, and Bush (2001) state that ADHD has a childhood onset but is persistent over time with follow-up studies showing that up to 80% of the ADHD individuals diagnosed in childhood will continue to experience clinical diagnostic criteria into adolescence, and up to 67% of these individuals will continue to have symptoms of ADHD into adulthood.

## ADD/ADHD Progresses Into Adulthood

While ADD/ADHD is predominately considered to be a childhood disorder, a number of other researchers have also found that as these individuals grow and mature their childhood symptoms will continue to impair their levels of functioning into adulthood, and overall what was low functioning in childhood will continue to be low functioning into their adult vocational worlds (Barabasz & Barabasz, 1995; Hughes & John, 1999; Linden, Habib, & Radojevic, 1996; Lubar, 1991; Monastra, Lubar, & Linden, 2001; Nash, 2000; Tinius & Tinius, 2000; Rossiter & La Vaque, 1995).  Attention deficit disorder is described by Lubar, Swartwood, Swartwood, and O'Donnell (1995) as a pervasive lifelong condition existing in all countries and in all cultures, and is one of the most prevalent of childhood and adolescent disorders being treated by workers in mental health fields. According to a number of studies, ADD/ADHD in school age children may constitute as much as 50%, if not more, of child counseling, psychology, and psychiatry clinic populations (Cantwell, 1996; Culbertson & Krull, 1996; Linden, Habib, & Radojevic, 1993; Lubar, 1991; Whalen & Henker, 1991).

## Executive Functioning Impairments

According to Murphy, Barkley, and Bush (2001), ADHD individuals have cognitive deficits associated with difficulty in executive functioning.  These include problems with planning and forethought, a delay of gratification, difficulty in resisting temptation, and sustaining goal related behavior.  Murphy et al. expands the cognitive problems to include deficiencies in problem solving and flexibility in responding.  They also believe that problems with executive functioning include working memory; private self-directed speech; self-regulation; and future directed behavior.  In their study, they determined that executive functioning was most likely a result of an individual being ADHD and not the result of other common co-existing psychiatric disorders such as ODD or OCD.

## Problems of Impulsivity

Barabasz and Barabasz (1996) point out that ADD/ADHD children have difficulty sitting still within school classrooms, paying attention, following instructions given to them, focusing on studies, and completing tasks such as school assignments or home area responsibilities.  Typically these are the underachievers who are behind in grade levels and put into special resource and rehabilitation classes, and due to their impulsivity these children may respond too quickly without understanding what is required of them for doing a specific task.  This gives a high careless-error rate. The NIH Consensus Statement on ADHD (1998) states that children who have inabilities of sitting still in class and paying attention experience negative consequences of their disruptive behavior as well as peer rejection.  Lubar (1991) suggests that by acting impulsively, without being able to think things through as a normal individual would, ADHD individuals are often in situations that result in high-risk and/or dangerous behavior to themselves or others.  Destructive and physically aggressive antisocial behavior is often an additional element of the disorder, especially with boys and adult male populations.   According to the NIH Consensus Statement, these children experience higher injury rates while young and drug abuse and additional injuries and public involvement as they grow older.  The impact of this is that medical care is financial burdensome. Families experience higher levels of frustration, marital discord, and divorce.  Society also experiences this, as ADHD mixed with conduct disorders often results in violent crimes.   Financial cost in 1995 to public schools alone for ADHD related problems was estimated at over $3 billion.

## Differences Between Girls and Boys with ADD/ADHD

With regard to sex differences of attention deficit disorder, Murphy, Barkley, and Bush (2001) point out that neuropsychological research has found cognitive deficits of functioning with attention deficit disorders, and girls with the disorder may be more cognitively impaired in functioning than boys with the disorder, or control groups from either sex.  Murphy et al. think that sex difference is more factual if the samples were clinically referred instead of coming from larger community samples.  In studying the sex differences of this disorder, Barabasz and Barabasz (1996) found an internalizing of

symptoms with girls. The authors compared the proportions of males and females who suffer from attention deficit disorders. Barabasz and Barabasz point out the belief that males outnumber females in ratios ranging from 4:1 and 6:1 and believe these ratios were based upon the presentation of symptoms, as the majority of ADHD referrals to mental health clinics were boys with acting out behaviors. In contrast, ADD children without hyperactivity are more frequently shy, socially withdrawn, and moderately unpopular and do not receive clinical treatment in proportion to ADHD individuals. Barabasz and Barabasz concluded that symptoms outwardly expressed in males may differ somewhat from those inwardly expressed in females, and the occurrence of attention deficit problems in males and females may be equal as females present with problems in mood and emotion while showing much less aggression than boys. Their conclusion was that girls are more socially withdrawn and internalize their ADD symptoms through depression and anxiety, and as a result of being more withdrawn and not as aggressive in behavior. This circumstance results in the fact that parents and social organizations are not as aware of the condition in the girls as they should be, and as a consequence, girls have been denied treatment more frequently than boys.

## ADHD Usually has Multiple Complexities

The NIH Consensus Statement from the Consensus Development program (1998) stated that ADHD usually does not exist as an isolated disorder, and due to this, comorbidities may cause complications in reaching a diagnosis as well. Moreover these co-existing mental health problems may account for differences in research findings. Cantwell (1996) found in his literature review that additional mental and emotional problems often coexist with ADD/ADHD individuals, and as many as two thirds of elementary school-age children referred with ADD will have a clinical evaluation of at least one other diagnosable psychiatric disorder. Conduct disorders, oppositional defiant disorders, and learning disorders are highest in conjunction with ADHD in pediatric samples.

## Genetic Component with ADD and ADHD

In looking for a cause, Cantwell (1996) found that many researchers believe that there is a genetic component to the ADD/ADHD disorder that is passed down from parent to child.   Others believe the disorder occurs as a result of brain injury and/or mental retardation.   Prenatal complications have also been named as possible causes of ADD/ADHD.   Cantwell did find that there are some known causes of ADD such as fragile X syndrome, fetal alcohol syndrome, and low birth weight, but only a small percentage of ADD/ADHD children come with these problems.   Thus he found that overall the underlying causes of ADD are unknown, but the supposition is that most likely there are interplays of psychosocial, biological, and neurological factors that lead to the syndrome of ADD/ADHD.   In conjunction with this, one of the conclusions reached in the NIH Consensus Statement from the Development Program on ADHD (1998) was that after years of scientific research, knowledge about the cause of ADHD remains speculative, and as a result there is no preventative strategy for the future prevention of ADHD.

## Diagnosis and Treatment of ADD/ADHD

In describing controversial diagnostic procedures and treatments, the NIH Consensus Statement on ADHD (1998) said that a diagnosis of ADHD may be made through diagnostic interview methods.   However, there is no definitive independent valid test for ADHD, and both the diagnosing of ADHD, and the treating of ADHD have remained controversial among parents and physicians alike.   The Consensus Statement said that this is due to the need for more consistent diagnostic procedures and guidelines for treatment.   The many conflicting opinions have resulted in confusion regarding the achievement of a reliable diagnosis and what interventions are most appropriate.   One of the problems has been differentiating ADHD from other behavioral disorders as well as determining what the boundaries are between those with ADHD and those considered to be in the normal population.

In this same area of looking at diagnostic procedures, Cantwell's (1996) comprehensive literature review stated that there is no laboratory test that can make a definitive diagnosis of ADD/ADHD.   In his literature review of ADHD, Cantwell

described an extensive investigative method for determining a diagnosis of the disorder. Proper comprehensive diagnosing usually starts with parent and child interviews and then includes a wide variety of assessment rating scales. These should then be followed by cognitive assessments of ability and achievement. Next, a developmental history of the child should be taken along with medical and psychosocial histories. Finally, in looking further, an investigative search should be made to look for any neurological problems and/or sensory deficits. According to Cantwell, this constitutes a comprehensive method of diagnosing ADD/ADHD by looking at the many components of its various manifestations. Overall, this time intensive procedure covers many factors of the disorder from behavioral to neurological. However, Cantwell stated that for the most part this type of comprehensive testing for the disorder rarely happens as it is time consuming and costly, leading to the high concern expressed by professionals in the field that ADD/ADHD is often inaccurately diagnosed and medications are overly prescribed. The NIH Consensus Statement on ADHD (1998) also addresses this by saying that the lack of insurance coverage creates barriers for those making the diagnosis and providing appropriate treatment for ADHD and is a long-term cost for society.

## Psychostimulants

One of the most controversial issues has been the use of psychostimulants to treat ADHD. These include the use of amphetamines methylphenidate (MPH), dextroamphetamine, and pemoline. Because these are very available and have been prescribed most frequently, there is concern about overuse and abuse of these drugs, whereas the NIH Consensus Statement (1998) of the Development Program acknowledges that there is controversy over the use of psychostimulants, short-term trials have supported the efficacy of methylphenidate, dextroamphetamine, and pemoline. However, the medications did not normalize behavior problems across the board, and some ADHD children while under medication still manifested more behavioral problems than normal children. The NIH Consensus Statement said that despite the improvement of core symptoms of ADHD, there was little carry over into

improvement in academic areas and social activities.   This concurs with the findings of Lubar, White, Swartwood, and Swartwood (1999), who studied the effects of MPH through the use of Quantitative EEG (QEEG) and TOVA.   Their findings showed that MPH does not produce perceptible overall change in EEG such as increases in beta or decreases in theta.   What they did find during the use of MPH was an increase in fast wave beta that correlated with an increase in slower waves of delta and theta.   Lubar et al. speculated that this might be the reason that long-term use of MPH does not result in long-term improvement of cognitive or academic performance.   The findings are important due to the signature brain wave patterns of ADHD of low beta and high theta (Lubar, 1991).   Therefore, as Lubar et al. report, these findings are paradoxical in that under a performance task, the use of MPH would increase beta while also increasing delta and theta, thus limiting any useful gain of beta.   No such correlation occurred during the off-MPH condition.   Swanson et al. (1993) found that medication often had no effect in 25% to 40% of children with ADHD disorder.

## EEG Brain Waves Show Basis of ADD/ADHD Problems

Lubar (1991) reported that as early as the 1920's and 1930's, it was thought that abnormal impulsive and disruptive behaviors were the result of some kind of brain damage, and this was followed by the concept of minimal brain dysfunction syndrome (MBD).   Lubar stated that as early as 1938, Jasper, Solomon, and Bradley had found EEG abnormalities occurring in children with ADD/ADHD symptoms.   These were also considered to be in the MBD classification.   The specialized studies of Satterfield and Dawson (1971), and also Satterfield, Lesser, Saul, and Cantwell (1973), proposed the "low arousal hypothesis" due to the fact that hyperkinetic children appeared to experience a state of decreased sensory arousal.   According to their theory, this state of low sensory arousal resulted in the child's inability to experience sensory input as it was received.   In order to compensate for the problem of being unable to take in sensory information as it came in, the child would try to adapt by activating the vestibular system located in the inner ear, as this would then activate the reticular system and take incoming information to the cortex.   In Ayres (1983) work, she

stressed the importance of movement in the child's early stages.   Early movement (e.g., crawling) assists the maturation growth and developmental of specific parts of the brain, including the left and right hemispheres, to take in and process information. According to Lubar (1991), children try to compensate for their lack of ability to take in information, and they do this by what seems to be an inborn intuitive inclination for instinctively moving and being overly active by spinning and jumping around, as if trying to activate their vestibular systems and set in motion a domino chain reaction effect of taking information to the cortex.

## A Low Sensory Arousal with Hyperactivity

The work of Satterfield and Dawson (1971), and again in Satterfield, Lesser, Saul, and Cantwell (1973), suggested that due to a low sensory arousal problem occurring in hyperactive children, there is a need for movement to activate the vestibular and reticular activating systems.   According to Lubar (1991) this need to move (e.g., rocking, swaying, etc.) would then explain the disruptive behavior that children display as they strive to activate their sensory input systems through movement.   As a result of a lack of incoming informational processing, an additional maturational developmental delay would occur in the frontal lobes.   This would be due to many things, including a lack of informational accumulation resulting in a deficiency of the normal expansion of neural dendritic branching and synapse sites.

Lubar agrees with Satterfield's theory of low arousal and said that it helps to explain why stimulant medications work with hyperactive children, for inasmuch as these individuals are unable to raise their own levels of arousals due to insufficient movement processes, the stimulants in effect increase the arousal state for the ability to take in incoming sensory information.   When this happens, the child no longer has need for excessive movement to stimulate the vestibular system, resulting in a decrease of overactive movement.

## To Much Slow Theta and Not Enough Fast SMR

**In Brain Waves Causes Hyperactivity**

After reading Satterfield's work, Lubar (1991) made the hypothesis that the brains of hyperactive children might not be able to produce a sufficient amount of brain wave sensory-motor-rhythm (SMR) of 12 to 15 Hz, and beta above a 15 Hz level. Levels of 12 Hz and above are needed for active mental processing. Additionally, Lubar assumed that these hyperactive individual's brains might be stuck in producing an excessive amount of slow wave theta of 4 to 8 Hz. Having an excessive amount of theta would produce a state of underarousal. Lubar's research showed this to be true (Lubar, 1991; Lubar & Deering, 1981; Lubar & Lubar, 1984; Lubar & Shouse, 1976). This led to his conclusion that decreasing the theta and increasing the amount of SMR and beta would stimulate higher levels of arousal. This would consequently decrease the child's need for self-stimulation of the vestibular system by excessive movement activity. Neurological research studies have shown this to be true, as findings show that there is an overall decrease of physical activity occurring when theta is reduced and SMR is increased in brain waves of children with ADD/ADHD and behavioral/learning disorders (Barabasz & Barabasz, 1995, 1996; Hughes & John, 1999; Lubar, 1991, 1995; Lubar & Lubar, 1984; Lubar & Shouse, 1976; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Monastra, Lubar, Linden, 2001; Nash, 2000; Rossiter, & La Vaque, 1995; Tansey, 1983, 1990, 1993; Tinius & Tinius, 2000).

The brain is intricately composed of many specific specialized functions. Two of these are: 1) the motor cortex, located in the back of both the left and right frontal lobes, and 2) the sensory cortex, which is located behind the motor cortex and in front of the parietal lobes. These two combined areas are referred to as the sensorimotor strip. Lubar (1991) reports that over this area is recorded the EEG sensorimotor rhythm (SMR) of 12 to 14 Hz. in normal functioning modes: Should the amount of the SMR reading be excessively low, a condition of under-arousal would be occurring with the brain being in neutral, unable to take in information and process it appropriately, indicating the brain at this location is low functioning in comparison to normal processing ability.

**<u>Specific Brain Sites for ADD/ADHD</u>**

In addition to EEG brain wave progress in identifying neurological problems experienced by ADD/ADHD children, the neurological imaging technology of MRI, PET, and SPECT has also moved rapidly forward.   Due to new advancements in the field, neurologists have been able to identify several brain sites that are distinctly different in children with ADD/ADHD from those children who do not have the disorder, and the following areas and conditions have been identified as neurological concerns in ADD/ADHD children:

* Reduced volume in the rostrum and rostral body of the corpus callosum (Giedd, Castellanos, Casey, Kozuch, King, Hamburger, & Rapport 1994).
* Abnormal left and right asymmetries according to studies by Barabasz and Barabasz (1995).
* Decreased glucose metabolism in both frontal cortical regions (Zametkin, Nordahl, Gross, King, Semple, Rumsey, Hamburger, & Cohen 1990).
* Decreased noradrenergic activity (Zametkin et al., 1990).
* Focal cerebral hypofusion of striatum and hyperfusion in sensory and sensorimotor areas according to SPECT scans (Cantwell, 1996).

According to Hannaford's (1995) view, the corpus callosum, located between the two hemispheres, is underdeveloped in ADD/ADHD individuals and needs stimulation in order to properly myelinate the crossover nerve fibers that send electrical impulse signals between the left and right hemispheres.   The supposition is that there is a lack of sufficient stimulation occurring from what is needed to properly develop the fibers of the corpus callosum (Ayers, 1983; Hannaford, 1995), and this would result in the corpus callosum being smaller in size than is developmentally normal, which is what Giedd et al. (1994) found.   A decrease in the size of the corpus callosum could thereby result in: (1) insufficient flows of electrical signals crossing over and back and forth between the two hemispheres, (2) a lack of blood flow and glucose metabolism (Zametkin et al., 1990), (3) a loss of normal asymmetry between the left and right lobes (Hynd, Castellanos, Casey, Kozuch, King, Hambutrger, & Rapport, 1991), and (4) abnormal frontal lobes (Hynd et al., 1991).   These conclusions coincide with the findings of

reduced EEG brain wave activity in the above areas (Barabasz & Barabasz, 1995, 1996; Hughes & John, 1999; Lubar, 1991, 1995; Lubar, Linden, VanDeusen, Green, Wing, Phillips, & Fenger, 1999; Lubar & Lubar, 1984; Lubar & Shouse, 1976; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Monastra, Lubar, & Linden, 2001; Nash, 2000; Rossiter, & La Vaque, 1995; Sams, 2000; Tansey, 1983, 1990, 1993).   In an effort to increase the normal flow of electrical activity between the hemispheres, Dennison and Dennison (1995) created a physiological program of movement designed to stimulate the corpus callosum through the sensory-motor cortex and generate increased activity between the hemispheres.   However, the pioneering works of Lubar and those who followed him, from 1976 to the present time, have been focused on changing the electrical patterns of the brain to achieve this.   It is assumed that changing the electrical patterns would produce overall changes in the neural physiological functioning of the brain.

## The Utilization of EEG Neurofeedback
## for ADD/ADHD

Hughes and John (1999) state that new brain imaging processes are now giving information that unequivocally establishes the fact that psychiatric disorders have definite correlates with brain dysfunction.   Among these advanced techniques is quantitative electroencephalography (QEEG).   Chabot and Serfontein (1996) defined electroencephalography (EEG) is a measurement of fluctuating electrical energy activity occurring in the cortical areas of the brain, also referred to as brain waves.   QEEG is referred to as a quantitative neurometric assessment that enables the practitioner to make distinctions between the normal and neurophysiological dysfunctions occurring in the brain. Hughes and John state that of all the imaging technologies, EEG and QEEG studies have the greatest body of replicated evidence concerning psychiatric and developmental disorders, and during the past decade over 500 studies have been done that show EEG and QEEG abnormalities in a high percent of psychiatric patients. Continuing, they say that frequencies ranges have traditionally been divided into 4 bands that are typically defined as delta (1.5-3.5Hz), theta (3.5-7.5Hz), alpha (7.5-12.5

Hz), and beta (12.5-20 Hz).   In addition, results from each electrode may be shown as absolute power or relative power as a percent of the total power.   According to Hughes and John, the origins of the electrical activity in the frequency bands appears to be anatomically complex homeostatic systems, with the brainstem, thalamic, and cortical areas involving the neuronal populations and using all of the major neurotransmitters. The following is a detailed description by Hughes and John as to the characteristic changes that are seen in ADHD disorders:

> Activation of the mesencephalic reticular formation (MRF) causes inhibition of the nucleus reticularis by cholinergic and serotonergic mediation, which releases the thalamic cells from inhibition by the *n.*reticularis.   The dominant activity of the EEG power spectrum becomes more rapid, with the return of alpha activity and the higher frequency beta activity, and the flow of information through the thalamus to the cortex is facilitated.

> The cortex can activate *n.*reticularis directly by glutamatergic pathways to suppress the arrival of information to the cortical level and, by striatal projections, dopamine can inhibit the MRF.   Such inhibition of the MRF enables inhibition of thalamic neurons to occur and blocks the flow of sensory information through the thalamus to the cortex. (p. 192)

Hughes and John state that their model suggests that neurotransmitter deficiencies or excesses produce a departure from homeostatistically regulated EEG spectrum, and this is believed to contribute to pathophysiology of psychiatric disorders.

## Normative Data Bases in Software Distinguish
## Normal from Abnormal Brain Wave Patterns

From the early days to present times during EEG investigations, Hughes and John (1999) state that large samples of normally functioning individuals have been studied quantitatively to produce a standardized normative data-base of information. This normative information ranges from 1 to 95 years and is standardized to the International 10-20 System for electrode placement. Further investigations by Hughes and John showed a comparison of EEG studies to the normative data-bases, along with

a collection of psychiatric disorder data-bases, and revealed that as many as 64% to 68% of EEG's for psychiatric patients show evidence of pathophysiology.   In QEEG studies of ADD/ADHD children, Hughes and John stated that either excess theta or excess alpha has been seen in these individuals, and 90% of children with attention deficit problems show QEEG signs of cortical dysfunction. The majority of the QEEG's of these children show hypercoherence and abnormal interhemispheric asymmetry. Using QEEG, it has been possible to discriminate ADD/ADHD children from normal children with a specificity of 88% and sensitivity of 94%. The conclusion that Hughes and John reached is that numerous EEG and QEEG reports reveal abnormal brain electrical activity in individuals with ADD/ADHD, and this typically is an excess amount of theta and/or alpha in relation to what is considered as normal.

A National Institute of Mental Health research committee suggested that quantitative EEG be used as an excellent method to identify functional measures of child and adolescent psychopathology (Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996). This is due to the fact that for many years EEG research has focused on the brain and has brain mapped specific symptoms according to location of origination within the neural organization of the brain, and quantitative EEG procedures have identified brain electrophysiological differences in various psychiatric conditions such as schizophrenia, Alzheimer's disease, Parkinson's disease, alcoholism, autism, and attention deficit disorder (Hughes & John, 1999; Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Lubar, Linden, VanDeusen, Green, Wing, Phillips, & Fenger, 1999; Monastra, Lubar, & Linden, 2001).

**EEG Neurofeedback in Treatment of ADD/ADHD**

EEG researchers have been able to successfully identify specific brain wave patterns relative to disturbances in ADD/ADHD individuals, in addition, they have been able to design EEG computer generated software programs that enable the brain to retrain itself into brain wave patterns typical of those of normal individuals (Barabasz & Barabasz, 1995, Hughes & John, 1999; Lubar, 1991, 1995, 1996; Lubar & Lubar, 1984; Lubar & Shouse, 1976; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Monastra,

Lubar, Linden, 2001; Nash, 2000; Rossiter, & La Vaque, 1995; Sams, 2000; Tansey, 1983, 1990, 1993).

## Changing Abnormal Brain Waves into Normal Ones

EEG neurofeedback is an innovative treatment procedure that teaches children, and also adults, how to regulate their own brain waves toward more normal patterns. Primarily, the goal of the treatment process is to teach ADD/ADHD individuals to reduce the amplitude and/or duration of theta 4 to 8 Hz. waves and at the same time to increase the SMR 12 to 14 Hz activities. With sophisticated computer driven instruments and software, rewards are given during the session in the form of levels of accomplishments earned that are similar to those of computer games. The rewards given may be both auditory and visual with sound tones and interactive visual displays. Rewards are given when a high amount of theta 4-8 Hz activity decreases along with the increase of an SMR 12-15 Hz or beta 16-20 Hz activity. The treatment process is considered to be complete when children or adults are able to produce brain wave patterns that closely resemble those of normal individuals, according to normative data-bases (Barabasz & Barabasz, 1995, 1996; Hughes & John, 2000; Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995; Lubar, 1991, 1995; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Monastra, Lubar, Linden, VanDeusen, Green, Wing, Phillips, & Fenger, 1999; Nash, 2000; Sams, 2000; Tansey & Bruner, 1983; Tansey, 1990, 1993).

## Required Sessions

The typical number of EEG treatment training sessions required to achieve clinical improvement of normalization has usually been in the range of between 40 to 80 sessions, with sessions usually lasting from 40 minutes to an hour. However, Rossiter and La Vaque's (1995) research demonstrated that significant improvement may be achieved within 20 sessions. Also, Barabasz and Barabasz (1996) were able to reduce the number of standard sessions by about half, down to about 32 sessions for each child. This was accomplished through the use of individualized instructions prior to

each EEG training session.   Tinius and Tinius (2000) have also been successful in reducing treatment sessions to twenty or below.   Success for Tinius and Tinius was due to traditional methods for decreasing theta and increasing SMR plus specialized coherence training methods.

## A History of Research Reveals
## Neurofeedback ADD/ADHD Treatment Protocols

According to Robbins (1998), some of the early history of biofeedback and ADD/ADHD began with Barry Sterman of UCLA School of Medicine, and Sepulveda Veterans Hospital who developed processes of using EEG with SMR.   Sterman, professor of neurobiology and biobehavioral psychiatry, was the first to experiment with and identify the sensorimotor rhythm (SMR) at 12-15 Hz.   As he developed his research, it led him to focus on epileptic seizures with success in EEG augmentation training in 12-15 Hz frequencies.   The results of those studies found marked decreases in seizures following neurofeedback training.

Lubar reviewed his own work in 1991 and stated that he had been studying the work of Sterman when he conceptualized the idea that if increasing SMR frequencies would reduce motor activities of epileptic seizure, it might work with reductions of motor over-activity in ADHD.   Lubar proceeded to investigate this possibility, and in 1976, Lubar and Shouse reported their work using EEG training with a hyperkinetic child. The child that they worked with met the standard hyperkinetic population requirements of: being male, age range 6-12, diagnosed hyperkinetic by a pediatrician, and taking Ritalin.   After an EEG base line was established, the goal of EEG training was to reduce the individual's slow wave theta activity of 4 to 7 Hz and increase SMR 12 to 14 Hz.   Training occurred three times a week in 40-minute sessions over several months. Laboratory findings reported that EEG feedback produced orderly changes in SMR functioning, resulting in a reduction of undirected activities and oppositional behaviors with an increase in cooperation and academic improvement.   At the end of the research, the child's Ritalin medication was removed to determine if he could still sustain the same level of improved behaviors without medication.   Results were

positive.   Long-term follow up showed the child maintained the gains without further need for medication (Lubar, 1991).

In 1979, Shouse and Lubar reported the results of EEG biofeedback training with four hyperkinetic children.   These children were selected for training due to each child's pronounced misconduct, overactivity, and severe deficiency in SMR Hz.   Also, each one was on a Ritalin medication program that did not produce normalization of behavior.   Research was conducted similar to previous ones, except this research included gradual withdrawal of Ritalin toward the end of the experimental treatment period.   Of the four participants, one failed to acquire the SMR task of increased 12-14 Hz.   Of the remaining three subjects, the baseline SMR Hz pre-training percentages more than doubled in feedback and final baselines by end of training.   Results of this study showed SMR training exceeded those improvements obtained with medication alone, and these improvements were sustained following termination of medication.

Of the three subjects who succeeded with the SMR training, each had experienced more motor over-activity and less attentional deficit than the one who did not succeed.   The subject who did not succeed in EEG training had experienced more difficulties in staying focused (i.e., attention) than the other three subjects.   Thus, the SMR training proved beneficial to individuals who experienced difficulty in motor hyperactivity, and later studies would need to address attention problems.

Following those initial studies, Lubar and Lubar (1984) observed that children with attentional difficulties and reading or spelling problems, but without hyperkinetic concerns, produced excessive amounts of 4 to 8 Hz theta and were deficient in 16 to 32 Hz beta.   Lubar and Lubar hypothesized that these types of individuals would benefit from an inhibition of theta and an increase in beta training, as well as SMR training. Results supported those conclusions by substantially improved grades for many in the study.

**Taking Neurofeedback from the Lab to the Clinic**

For the most part, EEG neurofeedback training had been accomplished in the research lab.   In contrast, Lubar and Lubar (1984) decided to show the efficacy of EEG

work within a private clinical setting at the Southeastern Biofeedback Institute.   Lubar and Lubar's study worked with six male children who were experiencing learning difficulties, with origination of the problems coming from the left hemisphere.   Some also had hyperkinesis.   Each child was first treated with SMR training to decrease the hyperkinetic behaviors.  Next, each child was given beta wave training to increase focused attention and arousal.   The goal was to either increase the beta activity or decrease the theta activity.   Each child's length of time in treatment was individualized according to the needs of the child, as displayed by the beta/theta EEG ratio.   The time of the study ranged from 10 to 27 months depending on the individualized progress of each child.   Records were kept of grades and achievement test scores prior to, and at the end of, the EEG training.   In addition, an EEG Fast Fourier Power Spectral analysis was performed at the University of Tennessee's Laboratory for Brain Research and Neuropsychology, and this analysis provided a neurological baseline.   After the initial testing periods, each subject received two 40-minute trainings per week.   Five of the children had been in academic resource classes for many years with no significant improvement before referral for EEG training.   At the end of the training period, all of the children showed improvement in both limiting gross movement and in improving academic performance.   Only one child was still in a resource class, while all of the other children were performing well in normal classes.   The results of this study clearly indicated the potential for using EEG training within a private clinic setting for individuals with ADD/ADHD.

### Increased Scholastic GPA from Neurofeedback

In 1984, Lubar (1991) did a demonstration project in the local schools in which 37 children participated in beta/theta EEG training.   This training was in addition to resource classroom activities, and was compared to matched controls of 37 children who received only resource classroom training.   The children with the beta/theta training increased in GPA and in the Metropolitan Achievement Test Scores.   A one year follow up continued to show improvements of over 1.5 GPA increased levels as compared to those who did not receive the EEG neurofeedback training.

## Using Brain Waves to Predict LD and ADhD

In a later study, Lubar, Bianchini, Calhoun, Lambert, Brody, and Shabsin (1985) examined six different cortical locations of EEG in 69 LD-ADHD children and 34 matched controls. Lubar et al. found excessive theta activity in the 4-8Hz range between the groups, and based on a discriminant function analysis, Lubar (1991) reported that it was possible to predict children in the classification of LD-ADHD group better than 97% with all variables taken into account, and better than 80% when only considering increased theta activity in frontal temporal locations.

## An Increase in IQ Scores

During the year of 1990, Lubar (1991) along with his colleagues added pre- and post-psychometric assessments for all children seen clinically for EEG training. These included the WISC-R, the Woodcock Johnson, and the Wide Range Achievement Test revised (WRAT-R). Of the nine cases who completed the psychometric testing, increases in IQ averaged 12 points for WISC-R verbal IQ and 8 points for performance IQ. The highest increase was seen in a 13-year-old male who increased 25 points after training on the new Lexicor Biofeedback System.

## Theta Slow Waves in Ratio to Fast Beta in ADD/ADHD

In looking at the relevancy of age and ADD/ADHD, Lubar in his 1991 writings discussed the work of Gasser, Verleger, Bacher, and Sroka (1988), who found the posterior regions of the brain to mature earlier than the anterior ones, and beta increased with age at a later time than theta decreased. Lubar took this into consideration as many of the children worked with had more of a pediatric look in their apperanace than did those in the control groups. With this perspective, Lubar reached the conclusion that the ratio of theta to beta would probably decrease in older individuals, and it should be higher in individuals with ADHD than in non-ADHD matched controls. Lubar also found that the largest theta/beta ratios occurred in frontal locations and were much lower in the posterior locations. Therefore, with EEG

neurofeedback training, theta/beta ratios should clearly decrease over the course of the training.   Research findings showed that after using the Lexicor EEG training system with three children, theta/beta ratios decreased in many cortical areas, as compared to pre-training ratios.

The researching team of Mann, Lubar, Zimmerman, Miller, and Muenchen (1992), increased the number of matched EEG channels to sixteen.   The study also used the new power spectral analysis and Lexicor topographic brain mapping for displaying results.   Many significant differences were found between 25 pure ADHD males, without learning disabilities or conduct disorders, and 27 carefully matched controls.   Theta activity was found in many locations but primarily in the frontal and central areas.   Decreased beta was also found in many frontal and temporal areas. Researchers were able to predict group membership in ADHD approximately 80% using Discriminant functional analysis.   This early research was significant because it was found that ADHD children showed greater increases in theta, and decreases in beta, when challenged with an academic task, than they displayed during their baseline readings.   Lubar first discussed this concept in 1991 when he compared the increase in theta activity to that of an individual being tested on a cardiac treadmill who shows abnormalities under performance demands.   Barabasz and Barabasz (1995) also found that as the brain drops down into theta, the child shows characteristic behaviors of the wondering mind with non-vigilance and unfocused thought.   Barabasz and Barabasz accomplished their research without children being on medication.   This provided a rationale for treating ADHD children with EEG neurofeedback training without the use of medication.

### T.O.V.A Continuous Performance Computer Testing

In the summer of 1992, Lubar, Swartwood, Swartwood, and O'Donnell (1995) did a time intensive neurofeedback program that consisted of daily one hour training sessions Monday through Friday for up to ten weeks.   The goal was to provide 40 EEG training sessions, as this was considered to be the appropriate number of sessions necessary to achieve significant change.   To assess changes in performance, the Test of Variables

of Attention (TOVA) was used.   According to the TOVA performance rating, 12 children with significant EEG changes showed improvement of an average of three TOVA ratings, but for 7 children who showed no EEG changing, the TOVA ratings showed an average improvement of 1.5.   This is an objective assessment of the neurofeedback treatment for ADHD.   Inasmuch as the TOVA has been used following pharmacological interventions while blood levels are at therapeutic levels, the findings of significant changes in the TOVA following neurofeedback training provided evidence that neurofeedback improves TOVA performance.   However, the neurofeedback performance on the TOVA was achieved without receiving neurofeedback during the administration of the TOVA, showing a change without continual intervention.   Lubar et al. also reported the effect of neurofeedback on IQ scores with nine males and one female following the initial study.   Approximately two years prior to the study, the Wechsler Intelligence Scale for Children-Revised (WISC-R) was given by school psychologists.   Following the EEG training, the WISC-R was again administered. Whereas each of the participants in this study made significant improvements in decreasing his or her levels of theta, each also showed significant improvement in post IQ scores.

### A Case Study Showing Longevity of Treatment

In 1983, Tansey and Bruner initially presented a case study of a 10-year-old boy who they had treated with electromyographic feedback (EMG) and sensorimotor training (SMR).   The boy had previously experienced motor over activity, extreme impulsivity, short attention, low frustration tolerance, and high distractibility.   He also had difficulty with saccadic fixation and ocular pursuit movements.   He was unable to move his eyes smoothly horizontally while his head was still.   He could not keep his eyes on an object while moving his head side-to-side.   He had previously been diagnosed as Perceptually Impaired and hyperactive during the start of his second grade and was on Ritalin for the second, third, and fourth grade years.   Ritalin was removed prior to the EEG biofeedback training.   EEG conditioning included EMG, monitored over the central forehead area, and SMR for 12-14 Hz increases over the Rolandic cortex.   The EMG

referent is the amount of electrical discharge in muscle fibers, whereas, the SMR referent is the electrical neural discharge. EMG was used to obtain reduction of muscle tension, and SMR was used because hyperactivity, rather than distractibility, was the primary symptom for the boy. A 40-minute session was held once weekly. By the end of the first 3 weeks of EMG only trainings, the boy had dropped from the baseline reading of 60 microvolts down to 5 microvolts and was no longer considered as hyperactive by mother and therapist. Yet he still had ocular instability and developmental reading disorder, and was unable to understand or retain visual reading material. SMR was added and EMG continued, and by the end of 20 sessions, he tracked smoothly, skipped no words, and understood what he had just read. While the ocular instability had remained the same and was unaffected by the EMG training, it did normalize through the EEG 14-Hz SMR training.

In 1993, Tansey gave a 10-year follow-up report and analysis of this case study. Over the 10-year period, the boy had continued to have an absence of motor over-activity, distractibility, and low frustration levels. In analyzing the changes made during the EEG sessions, Tansey pointed out that the EMG sessions were best seen as introducing the boy to a state of physical calmness, after which the 14-Hz brainwave training proceeded to functionally change the abnormal brain state that was reflected by the hyperactivity. Permanency of the treatment can be recognized as the young man continued to succeed both personally and academically all through grade school, junior high and high school, and at the time of Tansey's report, had earned a 2.50 grade point average in college. A number of researchers have reported studies with maintained success (Hughes & John, 1999; Kaiser & Othmer, 2000; Lubar, 1995; Nash, 2000; Rossiter & La Vaque, 1995; Sams, 2000; Tinius & Tinius, 2000).

**Research Comparing**

**Effects of Psychostimulant Medication with Neurofeedback Sessions**

In the research of Rossiter and La Vaque (1995), the effects of EEG neurofeedback were compared with those of patients on psychostimulant medications. The participants, ages 8 to 21 years, were 46 outpatients at a mental health clinic who

were referred by parents, school, or physicians. After obtaining baseline evaluations, patients in the Medication Group were placed on psychostimulant medications by their personal physicians. The TOVA was administered and then periodically given again to determine each patient's level of functioning. Depending upon TOVA scores, medication was adjusted until optimal dosage was obtained for each individual patient. EEG patients participated in 45-minute EEG sessions three to five times a week, for a total of 20 sessions over a period of four to seven weeks. At the end of 20 sessions, patients were re-evaluated using the results of TOVA as the determining factor to indicate the patient's level of progress in treatment. When the TOVA results suggested the need for further EEG sessions, the patient would continue on for an additional 20 sessions to achieve additional progress. Post testing for the two groups showed no significant differences between the EEG group and the Medication group on the change scores. The EEG treatment program led to improvement on all four TOVA outcome variables, comparable to those obtained with the medication program. Due to the comparable outcomes in this study between medication and EEG trainings, Rossiter and La Vaque concluded that the EEG neurofeedback training may be a correct treatment plan in cases where (a) medication has proven ineffective, or (b) medication has unacceptable side effects, or (c) the individual does not comply with taking medication. For those patients who have a good response to medication, a medication program may be cheaper in the short term. However, overall the EEG neural training is cost effective in the long term, especially for patients who are among the 60 to 70% of those who will never outgrow the disorder and will most likely continue to have ADD/ADHD into adulthood.

## Sessions Without Increasing SMR Beta Waves

In contrast to protocols of previous studies, Linden, Habib, & Radojevic (1996) varied the protocols and did not have the expected success. Their study was with 18 subjects of which 12 were ADD/ADHD and 6 were LD. Subjects underwent EEG neurofeedback for a total of 40 sessions over a period of six months. The procedure employed beta training *without SMR training*. At the end of the training period there

were no significant differences reported on the Aggressive/Defiant behaviors. Linden et al. concluded that *SMR training should have been included* with the beta training. They also thought that individualizing the EEG protocols to each child's specific needs would have improved the results of the study. However, the attentional behavior problems were significantly reduced in the EEG neurofeedback group. A most important significant finding was that the EEG experimental group had an overall IQ increase on an average of 9 points greater than that experienced by the waiting list control group. This increase in IQ is in agreement with other researchers who also found an increase in IQ with EEG neurofeedback trainings sessions (Chartier & Kelly, 1991; Lubar, 1991; Lubar, Swartwood, Swartwood, and O'Donnell, 1995).

## **Larger Research Studies**

In a large study by Chabot and Serfontein (1996) in Sydney Australia, 439 children were placed in an experimental group, and of these, 407 EEG readings were obtained. There were 310 children in the normal population control group. Findings profiled children with ADD/ADHD as having increased theta power, especially in the frontal regions, slight elevations in alpha power, and diffuse decreases in alpha and beta mean frequencies. Chabot and Serfontein concluded that the patterns of QEEG abnormality represented deviations from the individual's normal development rather than maturational lag or delay in normal development. To minimize the influence of IQ as a confounding variable, children were placed into either a normal IQ group or a low IQ group. The largest EEG differences that Chabot and Serfontein found were between the low IQ groups and the normal IQ groups:

Frontal Regions: The low IQ group showed more increases in theta and alpha power, with greater decreases in alpha hypercoherence, and more extreme decreases in alpha mean frequencies than the normal IQ group.

Midline Regions: The low IQ children showed more decreases in alpha mean frequency and in coherence than the normal IQ group.

     <u>Posterior Regions:</u> The low IQ group showed more increased delta and decreased alpha power than the normal IQ group.

A Discriminant function resulted in the correct identification of 70.4% of the normal IQ and 70.5% of the low IQ children.   Also, a Discriminant function resulted in the correct classification of 71.4% of the ADHD and 70.3% of the ADD children.

     Chabot and Serfontein (1996) found that one third of the ADD/ADHD sample showed disturbed interhemispheric functioning of decreased coordination between hemispheres.   An interhemispheric power asymmetry was common, usually between the left and right posterior regions. The right hemisphere was eight times as likely to have excess theta power than the left hemisphere. Interhemispheric coherence was prevalent, primarily in the posterior and midline regions, indicating abnormal communications between those cortical areas. The intrahemispheric coherence indicated problems of disturbed cortical/cortical relationships via the thalamus and/or basal ganglia, with frontal/striatal and corpus callosum dysfunction.

**Marvin Sams Work on 6.5 Hz and 13 HZ**

     While all of the research on ADD/ADHD has primarily focused on decreasing the theta band from 4-8 Hz, and increasing SMR at 12-15 Hz, the work of Marvin Sams for 1995 and 2000 appears to have a different viewpoint.   Sams divided theta into a lower band of theta consisting of 3-5.5 Hz and a higher band of theta of 5.5-8 Hz. Earlier researchers had called this higher range by the name of Frontal Mid Theta Activity. This he shortened to "Theta2" and described it as rhythmic frontal activity centered at 6.5 Hz with amplitudes of up to 50-100 microvolts.   The location for the highest amplitude of Theta2 was found anterior to FZ electrode and extends to the midline and the central strip when an individual is performing some mental task such as math or thinking of a scene.   Theta2 relates to mechanisms of attention, or arousal, and is found in most extroverts who have low neurosis and anxiety.   Sams described two

methods for reducing slow wave activity and increasing Theta2 for improved attention. These involve three primary bands.   The first is the delta band of 0-3 Hz that is down trained. Sams found that if he first reduces slow wave delta while under task at CZ and Pz, this enables the faster wave SMR and beta to more rapidly increase and improve mental and attentional flexibility, suggesting that decreasing delta allows faster waves to become "unstuck" and become more available for use.   The second method decreases Theta1 at 3-5.5 Hz. and is followed by sessions of increasing Theta2 at 5.5-8 Hz while doing a task.   The rationale is that Theta2 increases with ADD/ADHD individuals when doing a non-verbal task, therefore, it needs to be retrained while performing a non-verbal task.   Sams believes the SMR band of 12 to 15 Hz to be the primary EEG frequency relating to performance of mental tasks and is displayed at central sites of C3, CZ, and C4.

This suggests to Sams that the SMR training that has been so effective with ADD/ADHD individuals may not be due to the existence of a sensory-motor-rhythm, but instead may be "the brain's affinity for a resonant frequency close to 13 Hz which is captured by the 12-15 Hz frequency…[and] An implication for neurotherapy is that if increased intelligence and mental efficiency is the objective, then a frequency band with a 13 Hz center should be used" (p. 4).   After the importance of 13 Hz, Sams believes that the next highest band for doing tasks is that of the alpha 10 to 12 Hz band, and it should never be decreased unless determination is made that it is excessively high.

## Sites Specific to ADD/ADHD

Swanson, Posner, Cantwell, Wigal, Crinella, Filipek, Emerson, and Nalcioglu (1998), describe location sites in the brain that appear to be related to a collection of symptoms specific to those of ADD/ADHD individuals.   For inattentive types of individuals who have poor sustained attention abilities and alerting qualities, Swanson et al. proposes the concept that the right frontal area correlates with this symptom.   For the problems of failing to have orienting and poor selective attention, Swanson et al. suggests that the posterior parietal is involved.   The third classification of impulsivity and executive control has to do with the anterior cingulate. Swanson et al. suggest

consideration of these areas when working with ADD/ADHD individuals.

Lazzaro, Gordon, Whitmont, Plahn, Li, Clarke, Dosen, and Meares (1998) examined increased anterior theta and reduced posterior beta in unmedicated ADHD adolescents as compared to controls.  In comparison, Lazzaro et al. found : (a) increased absolute EEG theta in ADHD adolescents particularly in anterior locations, (b) significantly larger left and right hemispheric activity, (c)  reduced beta activity in posterior regions, and (d) increased alpha activity.

## ADHD and Traumatic Brain Injury

Tinius and Tinius (2000) studied a combination of traumatic brain injury (BTI) and attention deficit disorder (ADHD) subjects with intent that subjects would complete treatment as quickly as possible.   Sessions consisted of simultaneous EEG biofeedback, based upon clinical symptoms and a brain maps, and cognitive retraining.  The protocol was to reduce theta if high, increase SMR if needed, and if individual was depressed, increase alpha/beta.   Objectives were met as treatment plans were combined with EEG coherence training: (a) in frontal areas starting with alpha and beta, (b) making short connections and moving to longer connections, and (c) working at CZ, C3, and C4 sites.

This concurs with Sterman (2000) who reported that suppressing slow frequencies must be accompanied by rewarding enhancement at higher frequencies. Increasing higher frequencies enables the client to acquire a feeling of control over the associated state.   According to Sterman, training exclusively to reduce frequencies without enhancing higher frequencies has been ineffective.   Sterman suggests that appropriate neurofeedback training should be a mixture of decreasing slow wave activity followed by increasing higher frequencies.    Brain maps provide the reasoning for a determination of how to increase or reduce the brain wave training and the frequencies that should be improved.

## Multiple Clinic Research

In a study by Kaiser and Othmer (2000), thirty-two individual clinics from across the nation participated in a trial to evaluate efficacy of SMR/beta training with attention deficit disorder.  Over 1089 subjects participated and were composed of 726 children and 363 adults.  Treatment of twenty sessions was followed by TOVA testing, and twenty additional neurofeedback sessions were added as needed according to TOVA indications. Results demonstrated improvement in attentiveness and impulse control scores of nearly two standard deviations.

## Establishing Neurofeedback Theta/Beta Ratios Guidelines
## As A Diagnosis of ADD/ADHD

Monastra, Lubar, Linden, VanDeusen, Green, Wing, Phillips, and Fenger (1999) conducted a study with 482 subjects.  Results showed that theta/beta ratios were significantly affected through age and diagnosis.  Compared with other age groups, the higher theta/beta ratios were seen in the 6-11 year old age group.  In age progression, there is a reduction in theta/beta ratios as age increases.  However, the power ratio was affected by type of task performed.  The four tasks were baseline, reading, listening, and drawing.  Inasmuch as there has not been a laboratory test that could provide a conclusive diagnosis for ADHD, Monastra et al. sought to establish guidelines for clinical researchers who are seeking the validity of a simplified indicator of QEEG as a means of providing a laboratory test for ADHD diagnosis.

In further studies Monastra, Lubar, and Linden (2001) investigated test reliability and validation issues in establishing QEEG as an assessment for ADHD. A series of studies were made to best determine what simplified procedure would qualify QEEG as an assessment.  Monastra et al. studied theta/beta power ratios of participants for performance in four experimental tasks.  Statistical analysis indicated that the magnitude of the theta/beta power ratio was affected by age and diagnosis.  The main effects indicated that the inattentive and hyperactive types of ADHD had higher theta/beta power ratios than peer control groups.  Assessment was standardized according to an individual's score, and if that score was 1.5 standard deviations above average, it was considered positive for ADHD. The positive predictor power was 98%

for the QEEG attention index with negative power at 76%.   Results indicated the QEEG was reliable.

## Summary and Conclusion

It may be summarized that a typical brain wave pattern for an ADD/ADHD child would be an excessive amount of slow-wave theta relative to a low amount of SMR. The research also seems to indicate that there are problems with hemispheric asymmetry between the right and left hemispheres with a predominance of EEG displayed in the right hemisphere.   Neurofeedback has been shown to be very effective in reducing the excessive amounts of slow waves and enabling the individual to return to normal functioning in mental and emotional processing.

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