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EEG Findings in ADHD and the Application of EEG Biofeedback in Treatment of ADHD

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1. Introduction

As defined in the 4th edition of Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994), Attention deficit hyperactivity disorder (ADHD) is characterized by a persistent pattern of inattention, hyperactivity, and impulsiveness, though it can present with or without hyperactivity. ADHD is the most common childhood mental health disorder, with an estimated prevalence of 7% to 10% in boys and 3% in girls aged 4-11 years (Sgrok et al., 2000). This disorder substantially affects the individual's normal cognitive and behavioral functioning. For example, children with ADHD can have a great deal of difficulty focusing on lessons presented by their teachers and remembering how to do their homework. They may often be easily distracted whereby they pay attention to other things than what they should.

The numerous studies support a model that defines ADHD as an inherited disorder whose core symptoms are founded in neuroanatomic, neurochemical, and neurophysiologic abnormalities of the brain (Monastra, 2005). Deficits associated with ADHD support a hypothesis that anatomical and biochemical abnormalities of the prefrontal cortex constitute the physical basis of this disorder (Barkley, 1997). In this line, neurodiagnostic procedures (e.g., positron emission tomography [PET], single photon emission tomography [SPECT] and magnetic resonance imaging [MRI]) studies have provided evidence of the neurological basis of ADHD (Boutros, et al., 2009). Nevertheless, new theories on the pathogenesis of psychopathological phenomena conceptualize as a consequence of the failure to integrate the activity of different brains' areas (Boutros et al., 2009). It needs techniques tapping the dynamics of complex interaction over time among cerebral regions involved in the integration of cognitive processing.

Electrophysiological techniques enable monitoring brain processing in real time, providing the best methods to describe the time course of brain electrical activities. Growth of this field came from the newer and quantifiable techniques such as quantitative electroencephalography (QEEG). QEEG methods provide a set of non-invasive tools that are capable of quantitatively assessing resting and evoked activity of the brain with sensitivity and temporal resolution superior to those of any other imaging methods (Hughes & John, 1999).

QEEG studies have explored brainwave profile in children with ADHD, compared to normal children. These brainwaves could be trained via operant conditioning (called EEG

biofeedback or neurofeedback) and it is claimed that self regulation of brain electrical activity result in a therapeutic benefit in ADHD. The main purpose of this chapter is to look at one alternative method of treating children with ADHD. To fulfill this purpose, the present chapter will review:

• description of electroencephalography and QEEG
• QEEG findings in ADHD
• brief history and rationale for neurofeedback development
• description of neurofeedback in practice
• neurofeedback findings in the treatment of ADHD as supported by controlled studies.

Our brain is made up of many cells, including neurons and glial cells. There are about 100 billion neurons in the brain. Neurons are cells that send and receive information to and from the brain and nervous system. The language of these communications throughout the nervous system is electro-chemical signals. An electroencephalography (EEG) is a tool for measuring electrical activity generated in the brain. These electrical activities of neurons are very tiny. Hence, EEG activity always reflects the summation of the synchronous activity of thousands or millions of neurons; when many neurons shift towards being more ready to fire (excitatory) or to not fire (inhibitory) at the same time. The EEG signals are recorded using sensors (electrodes) placed on the scalp. Electrodes are attached to our head and hooked by wires to a computer and then the computer records our brain's electrical activity on the screen. Patterns of neuronal electrical activity recorded are called brainwaves.

An EEG signal is characterized by three major components: phase, frequency and amplitude. Traditional EEG displays waveforms in the time domain, and the interpretation is based on amplitude and dominant frequency. Each brainwave frequency is expressed in Hertz (Hz). One Hz means 1 cycle per second; it is the rhythm of the wave. Amplitude represents the height (intensity) of the brainwave, and is expressed in microvolt (mV). Brainwaves have traditionally been separated into different frequency bands (Drongelen, 2007):

• Delta rhythm (δ): 0.1–4 Hz
• Theta rhythm (θ): 4–8 Hz
• Alpha rhythm (α): 8–12 Hz
• Sensory-motor rhythm (SMR): 12 to 15 Hz
• Beta rhythm (β): 15–30 Hz
• Gamma rhythm (γ): the higher EEG frequencies, usually 30~70 Hz.

Conventional interpretation of the EEG is done visually by a trained specialist. The specialist will examine the EEG, by detecting features of waveshapes (morphology) of the brainwaves to identify certain characteristics that might indicate organic or neurological pathologies. Routine EEG is typically used in the following clinical circumstances:

• to distinguish epileptic seizures,
• to differentiate "organic" encephalopathy or delirium from primary psychiatric syndromes such as catatonia,
• to serve as an adjunct test of brain death,
• to localize the region of brain from which a seizure originates (Niedermeyer and da Silva, 2004).

A voluminous literature attests to the robustness of conventional EEG studies and their clinical utility in disorders of brain function (Hughes & John, 1999). However, many functional characteristics of brain activity could not be detected visually. Whereas, quantitative EEG (QEEG) transform the EEG into a format or domain that elucidates relevant information, or associate numerical results with the EEG data for subsequent review or comparison (Nuwer, 1997). Often, neurologically based disorders do not involve a structural abnormality, lesion, or disease process, but abnormalities are expressed in the way the brain evaluates information. These processes can be studied with QEEG techniques, but not with simple visual analysis of the raw EEG (Hoffman et al., 1999). Hence, one can say that QEEG might provide additional measurements and displays of EEG in many different ways that are not possible with visual inspection.

In fact, QEEG reflects the ability of a network to locally synchronize. Such ability to synchronization is related to the integrative capacities of a network and to the characteristics of its inputs. This can be strongly modified by the active state of the brain. Thus, impairment of cognitive processing (i.e. attention) can be monitored by QEEG (Nazari, 2008). Furthermore, QEEG enables precise comparison of the individual patient's record with normative and psychopathologic patient databases (Hughes & John, 1999). QEEG procedures involve the mathematical processing of digitally recorded EEG. The most commonly used method for EEG quantification is the spectral analysis by means of Fast Fourier Transformation (FFT) algorithm. It provides measures of the power at each frequency of the EEG bands, known as the power spectrum. The test-retest of power spectra has been shown to be high (Hughes & John, 1999).

The first step for doing a QEEG is digital EEG recording; a cap (usually 19 electrodes at standardized positions) placed on the head and two electrodes are placed on the ears. The electrodes are then made to conduct with the scalp and ears by using a conductive gel. Once this is achieved, a computer interfaces with the EEG machine, and a software program is used to display the traces of the brainwaves generated by the brain, and detected on the scalp. Data is recorded during resting states of eyes open, eyes closed, and in some instances during cognitive tests such as reading or attentional task. Approximately ten minutes of data are recorded in each state. A QEEG typically requires about an hour total in the clinic to complete the data gathering.

After recording the EEG data it is edited to remove artifacts which are distortions in the EEG signal due to muscle movement such as coughs, eye movement, and teeth clenching, muscle tension, pulse and other sources. Artifacts are electric potentials of non-brain origin that are in frequency and voltage range of EEG signals and that are detected by scalp electrodes (Boutros et al., 2009). Clinicians utilizing QEEG must be skillful in recognizing and minimizing artifacts, as well as in careful pre-recording preparation procedures to minimize artifacts in the EEG (Hammond and Gunkelman, 2001). Indeed, it needs to carefully study the raw EEG since abnormalities may be masked by the use of a QEEG alone (Hammond et al., 2004). During the editing process the data is examined visually to identify any patterns that might be of interest for training purposes or would suggest the need to refer to another specialist.

After the editing process is completed the EEG data is subjected to a variety of mathematical and statistical analyses. EEG recordings should be of sufficient quality and of sufficient length so that after artifacting there is a minimum of 40-50 seconds of artifact-free data

available for analysis (Hammond et al., 2004). A sample of artifact-free EEG data, usually 1 to 2 minutes, is analyzed, using the FFT to quantify the power at each frequency of the EEG averaged across the entire sample (Hughes & John, 1999). Results from each electrode can be represented as following measures:

- absolute power: amount of amplitude in each band (total μV^2),
- relative power: in each band percentage of absolute power/total power,
- power ratio: i.e. absolute power of theta/absolute power of beta (theta/beta ratio),
- coherence: a measure of synchronization between activity in two channels (similarity of frequency between two channels),
- symmetry (the ratio of power in each band between a symmetrical pair of electrodes (no similarity is called asymmetry).

The final analysis is the database comparison. This procedure allows for an individual's EEG to be compared to an 'average' EEG. One can use a reference EEG database to reveal the location and type of EEG feature abnormalities greater than two standard deviations from a normative group (Thatcher, 1998). This comparison data is derived from the analysis of EEG's gathered from hundreds of individuals; same sex, same handedness, approximate same age; who do not exhibit or report historically any significant mental health issues. Often the EEG will be compared to multiple databases. The aspects of an individual's EEG to be analyzed by the QEEG are:

- Does the individual's EEG features differ from the 'average' EEG?
- How does it look different (the level of statistical significance and the degree of difficulty)?
- Where (what areas of the brain) does it look different?

The QEEG data is used to generate a series of analyses presented in tables and graphics in brain map. Brain map is a computerized EEG topography that enables the construction of a bi- or three-dimensional matrix for a topographic representation of Q-EEG parameters, such as instant amplitude or band power (Boutros et al., 2009). Different algorithms have been proposed to localize underlying brain generators. Among the distributed source models, Low Resolution Brain Electromagnetic Tomography-LORETA (Pasqual-Marqui et al., 1994) has been proven to present the smallest localization error (Boutros et al., 2009). The LORETA is one of the QEEG topographic analysis method by which one can provide a 3-D analysis of the EEG identifying localized disruptions in brain activity within the interior of the brain.

An individual who has received specialized training in these fields (see Hoffman et al., 1999; Hammond et al, 2004; Hammond et al, 2011) could examine the QEEG results. Individuals conducting assessment utilizing quantitative EEG or any type of brain mapping should be able to gather reliable data. A much higher standard is required for someone to hold himself or herself out as competent to analyze and interpret QEEG data (Hammond et al., 2004). It is strongly recommended that the QEEG providers *should hold diplomate status in QEEG from the Quantitative Electroencephalography Certification Board or be certified by the EEG and Clinical Neuroscience Society (or a comparable neurology board in the case of physicians), or be analyzing data under the supervision of such a certified person, or at a minimum be able to demonstrate thorough education, training, and work product documenting their competence to interpret QEEGs. Otherwise, the QEEG data should be submitted for analysis by an individual with such certification*

(Hammond et al., 2011). For further details about standards and qualifications for doing QEEG and neurofeedback see Hoffman et al., 1999; Hammond et al, 2004 and Hammond et al, 2011.

Due to the non-invasive nature of the procedure, the convenience, not expensive, and specificity of the data the QEEG has been used extensively to examine a variety of aspects of brain function. As mentioned before, with the quantitative EEG and topographic brain maps, it is often possible to observe attributes of brain function that cannot be seen in the raw EEG signal. These processes can be observed and quantified through subtle frequency-related and coherence related activities in the QEEG brain maps that index the degree of difficulty of cognitive tasks (Hoffman et al., 1999). Furthermore, it is well known that a great many medications as well as psychoactive drugs can produce some alteration in the EEG (Boutros et al., 2009). The availability of QEEG let to the development of a new research field that named pharmaco-EEG. Pharmaco-EEG methods were included in preclinical studies to identify at early stages of drug development, the therapeutic indications of new drugs, determining onset, peak effect, and duration of drug effect on CNS, and predict therapeutically useful dosage of psychotropic drugs (Boutros et al., 2009).

In the clinical setting, many studies have been reported that QEEG can be useful for the evaluation and understanding of mild traumatic brain injury, learning disabilities, ADHD, alcoholism, depression, and other types of substance abuse (Hoffman et al., 1999). Specifically, QEEG studies have reported different brainwave patterns in children with ADHD than those of the normal population.

Most studies of the electrophysiological correlates of ADHD have compared the QEEG from ADHD sufferers with those of healthy children under resting conditions (for a review, see Barry et al., 2003; for a meta-analysis, see Snyder & Hall, 2006). However, the allocation of neural resources differs when the subject directs his/her attention to an experimentally controlled situation (Thatcher, 1998). It is therefore important to evaluate a neural network's ability to change from a passive to an active condition. Since inattentiveness and distractibility are the major symptoms of ADHD, assessment of these symptoms would require tasks specifically designed to highlight attentional deficits, such as the continuous performance task (CPT) or the go/no-go task. Hence, in a study, Nazari et al (2011) set out to establish the functional reactivity of frequency-specific EEG activities during eyes-open resting and CPT in children with ADHD. High-resolution EEG was recorded during eyes-open resting and CPT performance in 16 children meeting the DSM-IV criteria (APA, 1994) for ADHD and 16 age-matched controls. Significant CPT vs. eyes-open differences in EEG activities was observed in children with ADHD. In particular, switching to CPT induced an alpha power increase in children with ADHD and an alpha power decrease in controls. Lower alpha power at baseline (eyes-open resting condition) might be interpreted as meaning that children with ADHD are unable to attend to and process visual stimuli as efficiently as healthy children. Klimesch et al (1996) suggested that alpha synchronization during mental inactivity may be important for introducing powerful inhibitory effects, which could prevent a memory search from entering irrelevant parts of neural networks. Based on this explanation, we suggested that impaired inhibition of neural networks in children with ADHD at baseline alters not only energy demands but also control excitatory processes. Opposite alpha changes may also reflect a primary deficit associated with cortical hypoarousal in ADHD. These EEG results agree with behavioral findings leading the

authors to suggest that dynamic changes in neural network activities are impaired in children with ADHD (Nazari et al., 2011).

Lubar (1995) compared QEEG data for ADHD children with controls. He concluded, "Excessive theta activity and lack of beta activity are the primary neurological landmarks of ADHD" (p. 505). Furthermore, "during academic challenges, there were significant increases in slow (4-8 Hertz) theta activity along the midline and in the frontal regions and decreased beta activity, especially along the midline posteriorly" (p. 502). Lubar's review of the literature revealed the following:

> "*Abnormalities in EEG were reported in children now classified as ADD and ADHD as early as 1938 (Jasper, Solomon & Bradley, 1938). There is extensive literature, much of it reviewed in the supplement to the Journal of Child Neurology published in 1991. Basically, EEC studies show excessive slow activity in central and frontal regions of the brain. These studies are supported by recent PET [positron emission tomography] scan and SPECT [single photon emission computerized tomography] scan studies that also indicate abnormalities in cerebral metabolism in these particular brain areas" (p. 50I).*

Based on Lubar's finding, studies have repeatedly reported a QEEG pattern that might be present in ADHD but not in controls (normal children, adolescents, and adults). A considerable number of these studies have reported an increase in low-frequency power (predominantly theta) and a decrease in high-frequency power (especially beta) in children with ADHD compared with the age-matched control group (Barry et al., 2003; Snyder & Hall, 2006). Some researchers have tried to examine the theta/beta ratio as a measure of ADHD-related abnormality with a higher detection power. As reported by Snyder & Hall (2006) results of 9 DSM-IV studies and the results of 29 pre–DSM-IV studies support that a theta/beta ratio increase is a commonly observed trait in ADHD relative to controls. By meta-analytic statistical extrapolation, the effect size of 3.08 predicts a sensitivity and specificity of 94%, which is similar to values predicted by retrospective studies examining ADHD and normal controls in group comparisons (Snyder & Hall, 2006).

As emphasized by the committee of the Association for Applied Psychophysiology and Biofeedback (AAPB) and the Society for the Study of Neuronal Regulation (SSNR), QEEG should not be the only tool used for diagnosis of attention-deficit/hyperactivity disorder (Hoffman et al., 1999). *There is no single technique that can be solely relied upon for the diagnosis. Manifestations of ADD/ADHD reflect behavior problems, learning style, cognitive processing, social interaction, and many other developmental factors. The current diagnosis of ADD/ADHD depends also on the use of computerized continuous performance tasks, detailed history, school performance, and evaluation for learning disabilities and other comorbidities, as well as other measures. QEEG data complement these other findings by providing for a comparison of brain activity with databases for both normal and ADD/ADHD groups* (Hoffman et al., 1999).

Having diagnosed the locations in the brain that are producing high or low activity, it is now possible to intervene with training the brain to normalize the activity of the various locations in the brain. On the other words, the power in being able to define deviations of brain's electrical patterns within a normally distributed measurement set is that one can target deviant measures to "normalize" by a variety of intervention modalities. In fact, the EEG (as a physiological measure) is considered a form of behavior, which is subject to behavior modification through basic "operant conditioning" and "shaping" principles within

the formwork of learning theory. This brainwave training and learning self regulation of brain activity is called EEG biofeedback or neurofeedback. Neurofeedback postulates that normalizing the target signal will result in a therapeutic benefit. Definition of neurofeedback by the International Society for Neurofeedback and Research (ISNR) is the following:

> "*neurofeedback is a process in which sensors are placed on the scalp and devices are used to monitor and provide moment-to-moment information that is fed back to the individual about his or her physiological brain activity for purposes of improving brain functioning" (Hammond et al., 2001; p.55)*. For detailed information about neurofeedback see the website of the ISNR (http://www.isnr.org).

Figure 1 shows the neurofeedback procedure. During neurofeedback training, neuroelectrical activity is detected via surface electrodes (step 1). Note that no electrical current is put into the brain. This activity is then amplified (step 2) and processed by software programs (step 3) that provide contingent auditory or visual feedback to the patient on a computer monitor (step 4); brain activity is monitored and desired changes are rewarded similar to a videogame. The patient watches the dynamic display of the amplitude of the brainwaves in the areas where the electrodes are attached by a gel paste. The computer program gives a reinforcement each time the goal level of the EEG power (an optimal brain state) is reached. This processing continues during the neurofeedback session for a period of 15 to 40 minutes (step 5).
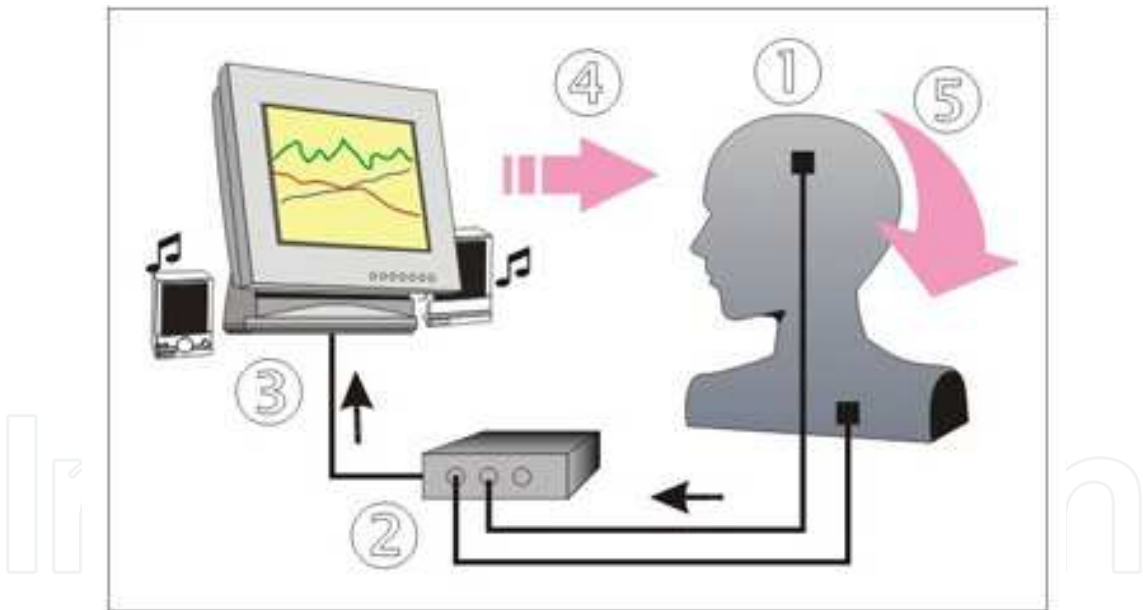

Fig. 1. Neurofeedback procedure

For example, there might be areas of the brain where there is an excess of neurons firing slowly during tasks requiring concentration. This is often the case with ADHD. On the basis of QEEG findings in ADHD, typically the EEG of a person with ADHD will reveal excess theta activity, but diminished beta activity. Hence, during the neurofeedback training a puzzle advances and sounds a tone whenever a child with ADHD maintains waves in the 15-18 Hz range above a certain amplitude threshold (beta increasing) while keeping waves in the 4-8 Hz range below a certain threshold (theta decreasing). Clients require 20 to 60

training sessions to achieve their goals. Training takes place 1-3 times for at least one hour of training per week. Once the original goals of treatment have been met, the client continues to train for an additional 5 to 10 sessions to prevent relapse (Demos, 2005).

Prior to beginning neurofeedback training an assessment is conducted to examine presenting problems, client history, contributing factors, current medications the patient may be taking, and other relevant information. Interviews, symptom checklists, computer based tests (i.e. CPT, TOVA, IVA), and review of relevant documentation are common components of the assessment. A pre- and post treatment objective assessment of the client's QEEG should be performed. The QEEG objectively assess the functioning of the brain in comparison with normative database (Hammond et al., 2011). One can use QEEG database and topographical brain maps to evaluate the location and type of EEG feature to target for neurofeedback training.

After reviewing the data gathered during the assessment a training protocol is developed. The neurofeedback protocols cover the following questions:

- Power of which frequency bandwidth targeted to be changed?
- Which areas of the brain are to be trained (electrodes location)?
- Which montage must be used (referential or bipolar)? Which locations are chosen for active electrode, reference and ground?
- How threshold levels are set for each client?

The rationale for neurofeedback protocols is based on solid research and clinical practice. Initially, neurofeedback treatments for ADHD are founded on the groundbreaking research conducted by Sterman (roth et al., 1967; sterman and Wyrwicka, 1967; Wyrwicka and sterman, 1968; sterman et al., 1969 and Lubar and Shouse, 1976; Lubar and Lubar, 1984). A brief history could be interesting. Sterman's research team conducted a systematic examination of EEG patterns and identified the sensory motor rhythm-SMR (12 -15 Hz) over the Rolandic cortex. They were able to train cats to increase production of this rhythm by providing food as an immediate reward. In later research those cats exhibited a significant improvement in stability when exposed to Hydrazine observed to evoke seizure activity in cats that had not received the SMR increasing. Subsequently, they demonstrated that patients with seizure disorders could develop improved control over epileptiform activity by learning self-regulation of the SMR (Sterman, 2000).

Sterman's procedure was replicated by Lubar who used the same training to reduce the symptoms exhibited by 'hyperkinetic' children. Initially, Lubar and Shouse (1976) reported some improvements in a hyperactive child who had learned to reduce theta and increase production of SMR. Subsequently, Lubar and Lubar (1984) reported that children diagnosed with an attention deficit disorder demonstrated improved attention and behavioral control after being trained to increase production of EEG activity in a fast frequency range (beta) while learning to suppress slow wave activity (theta).

These two primary training approaches provide the foundation for each of the protocols that have been examined in the controlled group studies of neurfeedback for ADHD. In a review study, Monastra (2005) has summarized three neurofeedback protocols that have been investigated in controlled group studies. These research-based protocols are the following (Monastra, 2005):

Protocol 1- SMR enhancement/theta suppression: in this protocol, patients (ADHD who present with primary symptoms of hyperactivity and impulsivity) instructed to increase their SMR (12–15 Hz) over one of two sites (C3 or C4) while simultaneously suppressing the production of theta (4–7 or 4–8 Hz) activity. EEG recordings are obtained from one active site, referenced to linked earlobes. Auditory and visual feedback is provided based on patient success in controlling power of theta below and SMR above pretreatment thresholds.

Protocol 2- Theta suppression/beta1 enhancement: In this protocol, patients are reinforced for increasing production of beta1 activity (16–20 Hz) while suppressing theta activity (4–8 Hz). Recordings are obtained at Cz with linked ear references, at FCz-PCz with single ear reference, or at Cz-Pz with ear reference. A variation of this protocol also has been reported in the treatment of ADHD, predominately inattentive type (Fuchs et al., 2003). In this training protocol, theta suppression and beta enhancement are reinforced at C3.

Protocol 3- SMR enhancement/beta2 suppression: in this protocol, children with ADHD, predominately hyperactive/impulsive type, are trained to increase SMR (12–15 Hz) while suppressing beta2 activity (22–30 Hz) (Fuchs et al., 2003) Recordings are obtained at C4 with linked ear reference. In ADHD, combined type, this protocol is used during half of each session. During the other portion of each training session, SMR enhancement/theta suppression at C3 is used. Selection of a neurofeedback protocol should be based on level of experience and training, accreditation, the fraction of the therapist's practice devoted to neurofeedback, reports from clients and objective assessments, and the therapist's specific experience in treating AD/HD (for more information see Monastra, 2005; Demos, 2005; Hammond et al., 2011).

Since the work of Lubar and Shous (1976), numerous studies have used neurofeedback approaches for treating ADHD and reported successful diminution of inattentivity and hyperactivity, and improvement in academic performance and concluded that despite some limitations, neurofeedback may be worthy of further consideration as a viable treatment approach for ADHD (Shouse and Lubar , 1979; Lubar and Lubar, 1984; Lubar et al., 1995; Rossiter and La Vaque, 1995; Linden et al., 1996; Thompson and Thompson, 1998; Kaiser and Othmer, 2000; Carmody et al., 2001; Monastra, 2002; Fuchs et al., 2003; Heywood and Beale, 2003; Cho et al., 2004; Heinrich et al., 2004; Rossiter et al., 2004; Xiong et al., 2005; Kropotov et al., 2005; Beauregard and Levesque, 2006; Levesque et al., 2006; Strehl et al., 2006; Gevensleben et al., 2010; Nazari et al., 2011; for review see Rossiter, 2004; ; Vernon et al., 2004; Monastra, 2005; Butnik, 2005; Friel, 2007; Toplak et al., 2008; John and Prichep, 2009; Coben and Evans, 2011). In an excellent meta-analytic study, Arns et al (2009) investigated results of 15 controlled studies. They concluded that neurofeedback treatment for ADHD can be considered "efficacious and specific" with a high effect size for inattention and impulsivity and a medium for hyperactivity (Arns et al., 2009).

Gevensleben et al (2009) conducted a randomized controlled trial encompassing 102 children with ADHD. In this trial behavioral and neurophysiological effects of neurofeedback, were analyzed in comparison to a computerised attention skills training (as a semi-active control group). They have shown neurofeedback to be superior to control group (Gevensleben et al., 2009). They reported follow-up behavioral data assessed 6

months after completion of the training (either neurofeedback training or attention skills training). Improvements in the neurofeedback group at follow-up were superior to those of the control group and comparable to the effects at the end of the training. They concluded that "though treatment effects appear to be limited, the results confirm the notion that neurofeedback is a clinically efficacious module in the treatment of children with ADHD" (Gevensleben et al., 2010).

In a clinical outcome study, Nazari et al (2011) investigated whether neurofeedback compared to methylphenidate achieves an equally effective outcome. Participants were 39 children: 13 children with ADHD were trained to enhance the amplitude of the beta1 activity and decrease the amplitude of the theta activity, 13 of which were treated with methylphenidate alone, and 13 healthy children did not receive intervention. Several behavioral, neuropsychological and experimental tests were administered before and after intervention. While behavioral measures were improved by both types of method, methylphenidate was significantly more effective than neurofeedback. Response inhibition (assessed by Stroop) was improved only by neurofeedback. Both neurofeedback and methylphenidate were associated with improvements on the variability and accuracy measures of computerized attention tests. Intellectual ability (measured by full version of WISC-III) increased also by both methods. Although averaged effect size for methylphenidate seems to be greater than for neurofeedback, the difference was not significant. In conjunction with other studies they concluded that neurofeedback can significantly improve several behavioral and cognitive functions in children with ADHD and it might be an alternative treatment for ADHD, particularly for those their parents favor a non-pharmacological treatment (Nazari et al., 2011).

Neurofeedback is contraindicated with subjects under age six years, or subjects with mental retardation, developmental delay or other significant medical, neurological, or psychiatric disease. Subjects from families with significant marital discord that could interfere with participation in the treatment process (Friel, 2007).

Side effect can sometimes occur during neurofeedback and practitioners should be aware that occasionally negative effects may occur (Hammond & Kirk, 2008; Hammond et al., 2001; Lubar & Shouse, 1976; Todder et al., 2010) if training is not being supervised by a knowledgeable and certified professional. Adverse effects that have been reported by some clinicians include increased anxiety and agitation, headaches, fatigue, sleep disturbance, anger and irritability, crying and emotional lability, enuresis, an increase in depression, increase in somatic symptoms (including tics and twitches), seizures, and temporary disorientation. These reports are uncontrolled case reports from which one cannot know the degree to which other confounding events in the patients' lives may have contributed to these negative symptoms (Hammond & Kirk, 2008). However, neurofeedback provider as a health-related profession should promote the welfare of their clients. Therefore, they should perform appropriate and objective assessments prior to, during and after providing neurofeedback to assess regularly the effectiveness of the services provided, and they inquire frequently about any side effects or adverse reactions. When it is observed that side effects or negative effects are occurring, providers document the details, discuss them with the client, and take appropriate action to remediate negative effects as quickly as possible. Such action may include modifying neurofeedback protocols, verifying the amount or frequency of treatment, utilizing adjunctive

treatments, and seeking consultation (Hammond et al., 2011). It should be mentioned that patients with a history of epilepsy should only receive neurofeedback from practitioners who are well versed in neurofeedback for seizure disorders.

Some people interested in alternative health react to the neurofeedback with hesitation. Neurofeedback has been considered as a relatively unstudied treatment, and the studies that have been conducted have reportedly been problematic, due to methodological problems such as confounded treatments, inconsistent use of dependent measures, small sample size, and a lack of clinically meaningful dependent measures (Kline, Brann, & Loney, 2002; Waschbusch & Hill, 2003; Loo and Barkley, 2005; Holtmann and Stadler, 2006). In this line, there are some fundamental questions:

- Does neurofeedback result in the intended EEG changes?
- Is there really an effect that leads to significant modifications in cognition and behavior?
- Could these changes be reliably linked to neurofeedback training?
- How does it compare to the current standard of treatment?
- Are these changes retained over time?
- How does neurofeedback work?

For validating purpose, some controlled studies on healthy subjects (i.e. Egner and Gruzelier, 2001; Egner et al., 2002; Vernon et al., 2003; Egner and Gruzelier, 2004) assessed specific cognitive, neuropsychological and electrocortical effects from training of specific frequency bands. They concluded that the modulation of specific frequency bands led to significant and protocol-specific effects. It seems that despite these validation works much remains to be done to provide a scientific basis for neurofeedback.

It has been argued that a potential explanation of the effects of neurofeedback could be cognitive-behavioral training effect as well as client-therapist relationship effect since children are engaging in a training for often 30-50 sessions. Such concerns could be addressed by double-blind controlled studies. Considering the ethical problem of including untreated patient or patient undergoing placebo and the difficulty of conducting a double-blind placebo controlled study in neurofeedback, some groups (Drechsler et al., 2007; Gevensleben et al., 2009) have still addressed these concerns by comparing neurofeedback group with a semi-active control group (can be considered a credible sham control). In these studies neurofeedback in comparison to this semi-active control group still had medium to large ES for inattention and impulsivity, and small to medium ES for hyperactivity (Arns et al., 2009).

La Vaque and Rossiter (2001) pointed out that, rather than comparing a new treatment (e.g., neurofeedback) to a no-treatment placebo, it should be compared to a protocol of 'known efficacy' to determine whether such an intervention would result in an equivalent effect. This type of design is often referred to equivalent study (Vernon et al., 2004). Regarding the well established efficacy of methylphenidate, several studies have compared the effects of neurofeedback and methylphenidate. Results revealed that although averaged effect size for methylphenidate was greater than for neurofeedback, both were in medium range and the difference was not significant (i.e. Nazari et al., 2011). None of the studies comparing neurofeedback with stimulant medication used random assignment. Although self-selection

the treatment may bias these findings, self selection potentially maximizes the effects of expectancy in both groups. However, more studies using randomization and larger sample sizes are needed to investigate further how neurofeedback compares to stimulant medication in the treatment of ADHD.

Several follow-up studies (Monastra et al., 2002; Strehl et al., 2006; Gani et al., 2008; Gevensleben et al., 2010) showed that improvements in behavior and attention turned out to be stable. Test results for attention and some of the parents' ratings once more improved significantly. Based on these researches, it can be concluded that the clinical effects of neurofeedback are stable and might even improve further with time. This, in contrast to stimulant medication where it is known that when the medication is stopped often the initial complaints will come back again and recent evidence showing that temporary treatment with stimulant medication is not likely to improve long-term outcomes (Molina et al., 2009).

Yet another domain in need of further investigation involves the theoretical basis and the underlying mechanisms of neurofeedback impact. Today, to understand "how does neurofeedback work?" is one of the most interesting and challenging tasks. It is not surprising that the field of neuroscience attracts a lot of researchers try to answer this question.

Despite some limitations, neurofeedback may be worthy of further consideration as a viable treatment approach for ADHD (See Vernon, 2005; Friel, 2007; Toplak et al., 2008; Yucha and Montgomery, 2008; Arns et al., 2009). On the basis of currently available research results, the success of this therapeutic method is indicated by widespread utilization, and reports of carefully designed studies suggest the utility of this method (John and Prichep, 2009). EEG biofeedback therapy for AD/HD results in significant improvement in cognitive functioning for 75-85 percent of patients. It is possible that faster and better outcomes might be achieved by combining other alternative therapies with EEG biofeedback (Friel, 2007). Neurofeedback meets the American Academy of Child and Adolescent Psychiatry criteria for clinical guideline for treatment of ADHD. As mentioned before, meta-analysis results of Arns and his colleagues (2009) demonstrated that neurofeedback treatment for ADHD can be considered "efficacious and specific".

Frank H. Duffy, M.D., Professor and Pediatric Neurologist at Harvard Medical School, stated in an editorial in the January 2000 issue of the Journal Clinical Electroencephalography that the scholarly literature suggests that neurofeedback should play a major therapeutic role in many difficult areas:

> *"In my opinion, if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used" (p. v). "It is a field to be taken seriously by all." (p. vii).*

Until 2005, neurofeedback was reportedly used by more than 1500 practitioners (Butnik, 2005) and the last years have seen a rapid growth of the field of neurofeedback in the US and at least 27 countries (Budzynski et al., 2009). There are more than 100 health-related professions in Iran that using neurofeedback in their routine clinical practice. All of them have been trained by the Biofeedback Foundation of Europe-BFE (www.bfe.org) instructors in the Paarand Specialized Center for Human Enhancement-PSCHE (www.paarand.org).

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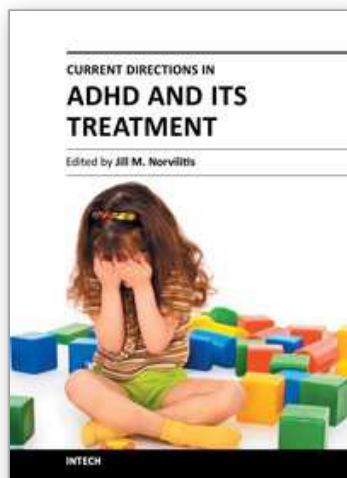
Current Directions in ADHD and Its Treatment

Edited by Dr. Jill M. Norvilitis

The treatment of Attention Deficit Hyperactivity Disorder is a matter of ongoing research and debate, with considerable data supporting both psychopharmacological and behavioral approaches. Researchers continue to search for new interventions to be used in conjunction with or in place of the more traditional approaches. These interventions run the gamut from social skills training to cognitive behavioral interventions to meditation to neuropsychologically-based techniques. The goal of this volume is to explore the state-of-the-art in considerations in the treatment of ADHD around the world. This broad survey covers issues related to comorbidity that affect the treatment choices that are made, the effects of psychopharmacology, and non-medication treatments, with a special section devoted to the controversial new treatment, neurofeedback. There is something in this volume for everyone interested in the treatment of ADHD, from students examining the topic for the first time to researchers and practitioners looking for inspiration for new research questions or potential interventions.

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