

CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.3455153

Available online at: <u>http://www.iajps.com</u>

Research Article

EEG NEUROFEEDBACK AND ADHD IN CHILDREN

¹Kiran Chaudhary, ²Mohammed Ali

¹MBM 5553 Quantitative Research, Elena Gillespie, PhD., Saybrook University, July 30, 2015.

Abstract:

Attention Deficit Hyperactivity Disorder (ADHD) often manifests during childhood, and typically impacts children from the age of four to fifteen. There is a lack of optimum levels of catecholamine's like dopamine and norepinephrine present. Psychostimulants such as methylphenidate and phenethylamine, modulate these catecholamine's, which are the primary treatment option for ADHD in children.

These drugs remain the treatment of choice, but their long-term effect and safety is still under scrutiny. Nonpharmaceutical treatment options for this complex disorder may be of benefit in this population, with fewer side effects, whose incidence is 8-12% of children worldwide. This preliminary review of the literature is guided by two questions: 1) How electroencephalogram-neurofeedback (EEG-NF) reduces inattention, hyperactivity, and impulsivity in children diagnosed with ADHD and 2) what are some of the limitations of this treatment modality.

Hypovolemia of PFC is detected in ADHD and it is associated with the symptoms of inattention. Several studies in this review have suggested that NF training produced microstructural changes in the gray and white matter of the cerebral cortex. Increases in alpha wave and reduction in beta wave brain function are often found on the electroencephalogram in ADHD. Electroencephalogram-neurofeedback (EEG-NF) provides a continuous feedback. The study participants showed improved attention, cognitive functions, reduced distractibility, and improvement in IQ. This treatment modality utilizes operant conditioning and trained children to gain control of their cortical functions.

This paper explores the impact of NF on ADHD symptoms, it also provides a brief overview of neuroanatomy and neurotransmitters of this disorder, electroencephalogram, the even related potential, biomarkers of EEG, side effects of medical regimens, and limitations of NF.

Keywords: *EEG-neurofeedback, ADHD, QEEG, ERP, cortical regulation, brain wave pattern, psychostimulants, catecholamines*

Corresponding author:

Kiran Chaudhary,

MBM 5553 Quantitative Research, Elena Gillespie, PhD., Saybrook University, July 30, 2015.



Please cite this article in press Kiran Chaudhary et al., **EEG Neurofeedback And ADHD In Children.,** Indo Am. J. P. Sci, 2019; 06(09).

INTRODUCTION:

It is estimated that approximately 11% (6.4 million) of children between the ages of four and seventeen were diagnosed with ADHD in 2011 (Visser et al., 2014). Children diagnosed with ADHD have increased from 7.8% in 2003 to 9.5% in 2007, and to 11.0% in 2011 (Visser et al., 2014). Interestingly, the research showed a wide difference in the number of children diagnosed with ADHD throughout the country. For example, it was 5.6% in Nevada and 18.7% in Kentucky (CDC). Boys typically seem to have a higher incidence and prevalence than girls (Visser et al., 2014).

The core symptoms of ADHD are impaired attention, excessive motor activity and impulsivity. There is no single diagnostic test for this disorder and it often involves a multi-step process. Children with anxiety, depression, and certain type of learning disorder can also have symptoms of inattention, motor activity, and impulsivity in children. The prefrontal cortex (PFC) is connected to the amygdala and the basal structures ganglia. These are rich in neurotransmitters like dopamine (DA) and norepinephrine (NE). Both the structures and neurotransmitters control behavioral inhibition, working memory, and reward reversal symptoms of ADHD. Details are discussed under the heading of neuroanatomy.

Diagnostic Criteria of ADHD (DSM-5):

Six or more symptoms of inattention for children up to age 16, five or more for adolescents 17 and older. The symptoms of inattention must be present for at least 6 months and they are inappropriate for developmental level (DSM-5):

- 1. Often has trouble holding attention on tasks or play activities.
- 2. Often does not seem to listen when spoken to directly.
- 3. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., loses focus, side-tracked).
- 4. Often has trouble organizing tasks and activities.
- 5. Often avoids, dislikes, or is reluctant to do tasks that require mental effort over a long period of time (such as schoolwork or homework).
- 6. Often loses things necessary for tasks and activities (e.g. school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
- 7. Is often easily distracted.

8. Is often forgetful in daily activities.

Six or more symptoms of hyperactivity-impulsivity (HI) for children up to age 16, five or more for adolescents 17 and older, and adults. The symptoms of hyperactivity-impulsivity must be present for at least 6 months to an extent that is disruptive and inappropriate for the person's developmental level (DSM-5):

- 1. Often fidgets with or taps hands or feet, or squirms in seat.
- 2. Often leaves seat in situations when remaining seated is expected.
- 3. Often runs about or climbs in situations where it is not appropriate (adolescents or adults may be limited to feeling restless).
- 4. Often unable to play or take part in leisure activities quietly.
- 5. Is often "on the go" acting as if "driven by a motor".
- 6. Often talks excessively.
- 7. Often blurts out an answer before a question has been completed.
- 8. Often has trouble waiting his/her turn.
- 9. Often interrupts or intrudes on others (e.g., butts into conversations or games).

In addition, the following conditions must be met (DSM-5):

- 1. Several inattentive or hyperactive-impulsive symptoms were present before age 12 years.
- 2. Several symptoms are present in two or more setting, (e.g., at home, school or work; with friends or relatives; in other activities).
- 3. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, school, or work functioning.
- 4. The symptoms do not happen only during the course of schizophrenia or another psychotic disorder. The symptoms are not better explained by another mental disorder (e.g. Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

The American Psychiatric Association's Diagnostic and Statistical Manual, Fifth edition (DSM-5), stresses that only trained health care providers can diagnose or treat ADHD. This diagnostic standard put forth by DSM-5 ensures that people are appropriately diagnosed and treated for ADHD.

Neuroanatomy of ADHD:

Symptoms of this disorder are rooted in structural

and functional disturbances in the prefrontal cortex of the brain. The neuropsychological and imaging studies showed that ADHD was not only associated with the reduction in the prefrontal cortex (PFC) volume, but also its connections to the striatum and cerebellum. In addition, Castellanos et al. (2008) reported a lower functional connectivity (FC) between the anterior cingulate and posterior cingulate cortex regions in individuals diagnosed with ADHD. The connection between precuneus (posterior cingulate cortex), the ventromedial prefrontal cortex, and portions of posterior cingulate cortex were associated with attention and focus. This may explain the inability to focus in children with ADHD (Castellanos et al., 2008).

Itami & Uno (2002) suggested that lesions in the PFC produced symptoms similar to ADHD. This indicated that PFC regulates behavioral inhibition, working memory, and reward reversal. Not only the PFC was hypovolemic, but it also differed in its connections to striatum and cerebellum in individuals with ADHD (Itami & Uno, 2002). In addition, the PFC in the right hemisphere controls behavioral inhibition. Brennan & Arnsten (2008) showed that the loss of integrity of the PFC structures and lower levels of connectivity are responsible for distractibility, forgetfulness, impulsivity, poor planning, and motor hyperactivity.

Neurotransmitters of ADHD

The three major catecholamines are: 1) epinephrine (adrenaline), 2) norepinephrine (noradrenaline), and 3) dopamine. The balance between these catecholamines is crucial to focus and concentrate on a task at hand (Brennan & Arnsten, 2008). The striatum is a subcortical part of the brain, and it is made up of the putamen and the caudate nucleus. The striatum plays a role in planning and movement pathways (Dai et al., 2012). The changes in connectivity among these can be responsible for the motor hyperactivity in ADHD children.

There are a wide range of reasons for the children's inability to focus and pay attention in a classroom setting. These symptoms are closely linked to the diagnosis of ADHD and it seems logical for a teacher to associate a student's disruptive behavior to ADHD. This is why DSM-5 stresses that only trained health care providers can diagnose or treat ADHD. Suboptimal levels of both norepinephrine (NE) and dopamine (DA) heavily influence the PFC. Therefore, low levels of catecholamines result in fatigue and high levels of catecholamines lead to stress (Brennan & Arnsten, 2008).

Regulation of attention and behavior in PFC requires

an optimal balance of catecholamines. Fatigue is associated with insufficient catecholamines and stress is associated with excessive catecholamines. This imbalance may produce symptoms of inattention and hyperactivity similar to symptomatology of ADHD. These symptoms may also stem from epigenetics, genetics differences, lesions in the PFC, diet, and the psychosocial environment of children diagnosed with ADHD. Studies show a link between heterogeneity of catecholamine genes and ADHD (Matthews et al., 2012). Matthews et al (2012) also stated that catechol-o-methyltransferase (COMT) gene plays an integral role in working memory and genetic variation in COMT genes in children with ADHD. This gene can cause memory problems in ADHD.

The Cambridge Neuropsychological Test Automated psychomotor Battery (CANTAB) measures coordination and motor speed, reasoning and planning abilities, memory (spatial working memory, pattern recognition memory, spatial recognition memory), and attention. In a study done by Matthews et al (2008), the CANTAB established a direct link between COMT genotype and four aspects of memory that are involved in the inattention symptoms of ADHD. This explained why children with ADHD have a difficult time with spatial recognition and spatial working memory. Studies revealed that optimum levels of dopamine aided the met-COMT allele in its ability to improve working memory in ADHD.

Electroencephalogram:

An electroencephalogram (EEG) is a test that can help to measure electrical activity of the brain. It uses electrodes to detect the brain wave activity and these electrodes are attached to the head with a conductive gel. Electrodes placed on the forehead detect and transmit electrical signals from the frontal lobe of the brain to a polygraph that produce separate graphs on moving paper using an ink writing pen or on a computer screen. Analog-to-digital converters are used to extract various wave bands (alpha, beta, theta, and gamma). Hans Berger was the father of EEG and he described normal brain wave activity (Haas, 2005).

Sensorimotor rhythm (SMR) is an oscillatory idle rhythm of synchronized electromagnetic brain activity and it appears in spindles on EEG. Sensorimotor rhythm is mainly recorded in the sensorimotor region of the frontal lobe. During states of immobility, higher amplitudes of SMR were noted (Tansey, 1984).

Heinrich, et al (2004) described that the slow electro-

cortical potentials (SCPs) account for a significant fraction of the readiness potential (RP). Slow electrocortical potentials (SCPs) are the manifestation of common activity of different neuronal regions of the frontal cortex. Heinrich, et al (2004), also suggested that the individual potential shifts preceding selfinitiated movements. These potentials are classified as showing negative or positive shifts on EEG. The ongoing negative shifts of the SCPs facilitate selfinitiated movement but are not related to processes involved in motor decision. The negative or positive shift can be used in developing a personalized neurofeedback training program.

The event readiness potential is a measure of activity in the motor cortex of the brain leading up to voluntary muscle movement. It is the manifestation of cortical contribution to the pre-motor planning of voluntary movement (Kornhuber et al., 1990). The readiness potential is detected as a slowly rising negative wave (down ward deflection on EEG) in response to components of the event-related potential (Kornhuber et al., 1990).

The slow cortical potential (SCP) is a suitable measure of central arousal and it plays an important role in the management of ADHD, because it is often associated with sustained activation of the sympathetic nervous system (Heinrich, et al., 2004). These slow waves are ideally suited to capture the mental workload and amount of neural activation associated with a specific cognitive process (Drechsler et al., 2007).

The EEG is generated by changes in action potential (electrical activity) on the membranes of cells located in the cerebral cortex and the thalamus acts as a pacemaker of the cerebral cortex similar to sinoatrial node (SA) in the heart (Haas, 2005).

The cerebral cortex signals are grouped together by location, amplitude, frequency and duration of specific types of EEG activity. Amplitude is a measure of the magnitude of the EEG signal, either of the whole band wave or of a sub-band of the wave signal. Amplitude is measured in microvolts but is commonly described in terms of being low, medium or high with respect to age-appropriate EEG norms (Schwartz & Andrasik, 2003). Electroencephalogram (EEG) provides an objective and quantitative analysis of brain wave functions and EEG plays a critical role in neurofeedback treatment for ADHD. However, it is also important to consider that EEG changes with age. Bresnahan et al (1999), reported an increase in theta and a reduction in beta activity in the ADHD groups with age compared with the normal controls.

Electrode Placement:

Electrode placement can be done using the 10-20 system - standardized placement or 19 active electrodes and 2 references (earlobes) or an 8-channel (electrode) setting. As discussed above, the electrodes are placed to detect and transmit brain activity from different parts of the brain to a polygraph. The electrodes are placed from Nasion (depression on bridge of nose) to Inion (the back of the skull), and from right temple to the left temple region (Monastra et al., 2005). The newer systems are using wireless technology and eliminate the use of electrodes. The Play Attention System is an example of wireless technology and is discussed under the electroencephalogram-neurofeedback (EEG-NF) section of this paper.

Brain Wave Frequency Pattern:

The major brain waves recorded on an EEG are: delta (0.5-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (13+ Hz), and SMR (12-14 Hz) (Jasper, 1958; Lubar & Shouse, 1976; Schwartz & Andrasik, 2003; Tasney, 1984). Delta activity is most prominent during deep sleep or unconscious state and theta activity is produced as an individual begins to fall asleep. Alpha waves are associated with passive awareness or relax state and beta waves are linked to high degree of external focus or active awareness, and SMR spindles are prominent during motor inactivity.

The Event-Related Potential (ERP)

The event-related potential (ERP) measures the electrical activity of the cerebral cortex that represents a distinct phase of cortical processing. Selective attention and conscious discrimination are two components of ERP that correspond to the intensity of a stimulus. An ERP is recorded using the non-invasive EEG cap. The event-related potential (ERP) is often used as an investigative, diagnostic, and prognostic tool in neuroscience and clinical medicine (Patel & Azzam, 2005).

The P300 (P3) is a positive wave (upward deflection on EEG). It is elicited in the process of decisionmaking and it usually appears at 300 ms poststimulus. It is associated with decision-making processes. Modifications in P3 are often detected in conditions that impair cognitive function (Patel & Azzam, 2005). The P2 is also a positive wave and it is involved in memory functions. Variability in P200 peak amplitude suggests that there are differences in the anterior and posterior region of brain during memory tasks like encoding words (Sokhadze et al., 2012). This study also noticed that P200 was involved in a cognitive matching system that compares sensory inputs with stored memory.

N200 (N2) is a negative wave (downward deflection on EEG). It is related to response inhibition, response conflict, and error monitoring (Folstein et al., 2013). The N200 negative wave usually appears 200-350 ms post-stimulus. The N200 is typically evoked before the motor response. This suggests that it is linked to the cognitive processes of stimulus identification and distinction.

Patel and Azzam (2005), suggested that the P300 was not only involved in the processes of working memory, but it also interacted with the N200 in the control of motor response to external cues. Through the use of a "GO/NOGO" paradigm, requiring subjects to identify targets either with a motor response or a suppression of activity, it was possible to examine the processes associated with voluntary movement. Similarly, Sokhadze et al. (2012) found more significant delay in the appearance of P300, P200, and N200 waves post-stimulu in the children diagnosed with ADHD. The N200 and the P300 can explain the deficit in executive functions during an external performance task such as homework in ADHD children (Sokhadze et al., 2012). The N200 is typically evoked before the motor response, suggesting that it is linked to the cognitive processes of stimulus identification and distinction in ADHD (Sokhadze et al., 2012; Patel & Azzam, 2005). These ERP studies support that some aspects of the psychophysiology of ADHD could be due to changes such as post-stimulus appearance time and alteration in amplitude of P2, P3, and N2 in the frontal cortex.

The contingent negative variance (CNV) affects neuronal circuits underlying resource allocation during cognitive preparation (Walter et al., 1964). The contingent negative variation (CNV) consists of slow negative potentials on EEG. It depends on the association of preparatory and imperative stimuli during a warned reaction task requiring a response (Heinrich et al., 2007; Holtman et al., 2014).

Electroencephalogram (EEG) Biomarkers

Properties of an electroencephalogram can serve as a unique biomarker to administer individualized neurofeedback training. Arns et al (2012), described five unique EEG biomarkers: 1) excess fronto-central theta, especially on the midline region of the frontal lobe, 2) excess fronto-central alpha, especially during eyes open during a task, 3) excess beta or beta spindles, 4) Sensorimotor rhythm (SMR) spindles or SMR/theta, and 5) no clear EEG pattern. Targeting individual EEG biomarkers can improve the treatment outcomes.

Electroencephalogram-Neurofeedback:

Electroencephalogram-neurofeedback (EEG-NF) uses basic principles of EEG to establish brain activity in children diagnosed with ADHD. Electrode placement typically follows the International 10/20 System (Shouse & Lubar, 1997). During each session, an individual wears a helmet equipped with sensors that record alpha, theta and beta wave activity (Roja & Chan, 2005).

The Play Attentions System:

The Play Attention System is a computer based video game-like interface with different programs focusing on different learning tasks such as attention, auditory processing, social cues, and memory. A small hardware device that fits in an armband sleeve, which is placed on a user's arm. It is crucial that the threedry disk shaped sensors on the sleeve must be in direct contact with the skin. It is necessary to record and transmit brain signals via a wireless technology.

Neurofeedback Protocols:

Electroencephalogram biomarkers can be used to apply an appropriate neurofeedback.

Some of the neurofeedback protocols include SMR protocol, theta/beta protocol, beta-downtraining protocol, frontal alpha protocol, and alpha protocol (Arns et al., 2009) Neurofeedback resulted in large and clinically relevant effect sizes (ES) for ATT and impulsivity and a low to medium ES for hyperactivity (Arns et al., 2009).

A low amplitude of SMR on an EEG is indicative of hyperkinetic symptom profile and it would be most appropriate to apply SMR protocol during neurofeedback training so that user can learn to increase SMR amplitude to reduce symptoms of motor hyperactivity (Arns et al., 2009).

Electroencephalogram-Neurofeedback Training:

Electroencephalogram (EEG) electrodes are attached to the head with a conductive gel according to the 10-20 international electrode placement system or 8channel (electrode). During training, participants sit in a comfortable armchair. Brain-wave signals are amplified for processing by analog-to-digital converters and polygraphs to extract pure delta, theta, alpha, beta, and gamma bands of the EEG (Hardt, 2012).

The training starts with a baseline EEG to establish the wave pattern of trainees. EEG biomarkers are often included to create an individualized training program. Neurofeedback training is rooted in basic operant conditioning. Trainees receive continual feedback, which serve as reward for producing a specified EEG pattern. The reward is built into the training software. Upon producing the desired EEG brain wave such as beta wave, the trainee receives an auditory tone and/or a visual display on the computer screen as a reward. The software references the EEG amplitudes, typically in more than one spectral band simultaneously. This offers a continuous feedback to a trainee in comprehensible sensory forms (Schwartz & Andrasik, 2003).

The short-term goal is to cultivate attention so that a trainee can finish the task at hand without worrying about changing the brainwave. The initial phase of the training revolves around meeting the situational demands such as homework, transition between jobs, follow multiple step directions, and improve social interactions. Once the brain begins to exercise this ability, as with other types of learning, the assumption is that it will most likely hone this skill until the symptoms of ADHD are managed.

The Play Attention System is one of the aids to improve attention stamina, discriminatory processing, visual tracking, motor, and social skills through playing. For example, by improving focus and attention, a child can have an Orca swim to the bottom of the ocean, and as soon as the child loses focus, the Orca starts to swim away. This continuous feedback is also captured and stored on the computer. This data can be used later to generate progress reports. A reward system is used to motivate children. For example, children get points for maintaining focus and avoiding distractions. These points can be redeemed for a sleep over or buying a toy or a clothing item.

The long-term objective is to develop stateappropriate cortical regulation. During this phase, the purpose is to improve short-term memory, auditory and visual discrimination, and auditory sequencing. These skills are needed to engage effectively outside the training environment (laboratory/clinical office). The objective of the training is to assist children in managing their ADHD symptoms. It may help reduce the dose of medication or may eliminate the need of psychostimulants.

According to the research studies, a typical program offers 20 to 40 sessions, 2 to 3 times per week (Arns et al., 2009; Drechsler et al., 2007; Fuchs et al., 2003). However, in the presence of comorbid symptoms, more sessions may be needed. The cost varies and ranges from fifty to two hundred dollars (Lansbergen et al., 2011). The healthcare insurance companies seldom cover EEG biofeedback, but private clinics offer payment plans and special treatment packages (Baydala& Wikman 2001; Masterpasqua et al., 2003).

Seven controlled trials have assessed the efficacy of neurofeedback training (EEG and SCP) as a treatment option for ADHD in the pediatric population. These trials reported improvements in ADHD symptoms as well as cognitive functions such as enhanced attention and inhibition after neurofeedback training compared to the control group (Drechsler et al., 2007; 2009; Heinrich et al., 2004; Levesque et al., 2006). Similar improvements were also reported by controlled trials compared to psychostimulants (Fuchs et al., 2003; Rossiter, 2004).

Quantitative Electroencephalography (qEEG) is a procedure that processes the recorded EEG activity from a multi-electrode recording using a computer. This multi-channel EEG data is processed with various computerized algorithms. The digital data is statistically analyzed, sometimes comparing values with "normative" database reference values. The processed EEG is commonly converted into color maps of brain functioning called, brain maps (Arns, et al., 2012). QEEG neurofeedback resulted in large and clinically relevant effect sizes (ES) for ATT and impulsivity and a low to medium ES for hyperactivity (Arns et al., 2009).

Medical Regimens:

Optimum levels of catecholamine like dopamine (DA) and norepinephrine (NE) are essential in the PFC to achieve the peak attention and focus. A decrease or increase in the levels of these cathecholamines in the PFC is associated with the symptoms of ADHD. This relationship between between catecholamines and the functions of PFC is utilized by psychostimulants like methylphenidate (MPH). Methylphenidate is often the drug of choice in the treatment of ADHD in children. It works by increasing extracellular catecholamine by inhibiting the reuptake of catecholamine such as dopamine and norepinephrine (Arnsten & Dudley, 2005). Optimum levels of DA and NE can assist PFC in accomplishing executive functions like reasoning, organizing, problem solving, and planning (Arnsten & Dudley, 2005). Higher levels in catecholamines in the postsynaptic cleft stimulated the central nervous systems, which further improved the function of PFC in ADHD (Markowitz et al., 1999).

Stimulants:

Methylphenidate (MPH) is associated with improvements in alertness, reduction in fatigue, and

improvements in attention span. This makes MPH a useful medicine for ADHD. However, psychostimulant drugs (Concerta. Metadate. Adderall, and Ritalin) have numerous serious side effects. Markowitz et al (1999), pointed that MPH suppresses appetite, activates the sympathetic nervous system, and can also cause arrhythmias. Drugs in the stimulant class are also associated with increased risk of eating disorders and mood disorders (Markowitz et al., 1999). Methylphenidate (MPH) acts fast to improve the release and/or inhibit the reuptake of both DA and NE. The fast increase in DA and NE makes MPH a drug of choice as it works to improve the working-memory in ADHD children (Wigal, 2009). This is why NE and DA modulators play a big role in treating inattention symptoms of ADHD. The right dose of psychostimulants is usually found by a trial and error method (Wigal et al., 2006). The abuse and dependence are not common in US high schools. Nonetheless, the parents are often nervous that their teenagers may abuse these stimulants.

Non-Stimulants:

Modafinil is not a stimulant but acts like a stimulant such as methylphenidate and amphetamine. It promotes attention by increasing the activity in the frontal cortex (Wigal et al., 2006). The film-coated tables were well tolerated and provided overall relief from the symptoms of ADHD. Stimulants are most widely prescribed, but Wigal et al (2006) reported that about 30% of all children diagnosed with ADHD do not respond to them.

Antidepressants:

Antidepressants like bupropion (Wellbutrin,) and desipramine (Norpramin) are also used to treat ADHD in patients who are not able to tolerate stimulants or are unresponsive to them. Clonidine works as an alpha-2 (α 2) and an imidazoline receptor agonist. It binds to the α 2 receptors in the central nervous systems (CNS) and reduces calcium in the presynaptic cleft (Reis & Piletz, 1997). This leads to lower levels of NE, which reduces the activity of the sympathetic nervous system (Reis & Piletz, 1997).

Antidepressants are slower than stimulants and usually take longer to work. However, these drugs are a good alternative for ADHD children with an existing medical condition such as heart conditions, thyroid diseases, and hypertension (Reis & Piletz, 1997).

DISCUSSION:

With time, reduction in the severity of ADHD symptoms is often reported, but in 40-60% of all

cases, the symptoms of this complex disorder persist in late adolescent and adulthood (Lansbergen et al., 2011: Tsai et al., 2013). ADHD can lead to poor academic performance, poor socialization, and increased traffic accidents because of inattention (Markowitz et al., 1999). Psychostimulants, in particular, are the primary pharmaceutical treatment for ADHD. Approxmately 20% of all children with ADHD fail to respond to psychostimulants, regardless of the dose (Molina et al., 2009). The long-term use of psychostimulants has been linked to minor and major side effects (Arnsten & Li, 2013; Markowitz et al., 1999). A significant number of children start these medicines between 7 and 9 years of age (Arnsten & Dudley, 2005). The majority stopped taking psychostimulants within 2 years and they re-experienced the symptoms of ADHD(Masterpasqua et al., 2003). According to Charach et al (2007) and Tsai et al (2013), the side effects of psychostimulants included growth retardation, sleep disorders, and decreased appetite.

These studies further necessitate the importance of exploring non-pharmacological treatments. EEGneurofeedback studies suggest that it is a promising alternative or additional treatment without reported side effects (Arns et al., 2009; Gevensleben, et al., 2009; Heinrich et al., 2007; Holtman et al., 2014; Lansbergen et al., 2011; Monastra et al., 2005; Shouse & Lubar, 1997).

Shouse & Lubar (1997) found that conscious control over motor movements led to conditioned increases in the sensorimotor rhythm (SMR). Increases in SMR were associated with the reduction in the symptoms of hyperactivity. Although the study size was small (21), Shouse & Lubar (1997) suggested that SMR, in conjunction with medication, proved most effective to treat physical hyperactivity compared to medication alone.

The slow cortical potential (SCP) neurofeedback ADHD treatment differs from SMR, because SCP rewards changes in the polarity of the EEG (positivity vs. negativity). This discrete reward scheme is specific to SCP training (Heinrich, et al., 2004). Both the SCP and SMR play an important role in the regulation of cortical excitability, inhibition, and inattention characteristics of ADHD. These initial findings contributed to a considerable amount of research into the treatment of ADHD with EEG biofeedback or neurofeedback. Many clinicians are currently using this therapy in their clinics (Heinrich, et al., 2004). Leins et al (2007) found that both SCP provided Theta/Beta neurofeedback and improvements in behavior, attention, and IQ.Arns et al (2012), applied the digital quantitative EEG neurofeedback to understand the impact on inattention (ATT), hyperactivity/impulsivity (HI), and comorbid depressive symptoms. This study found that QEEG neurofeedback reduced ATT, HI, and symptoms of depression in children with ADHD. Changes in N200 and P300 amplitudes in pre and post-treatment QEEG neurofeedback were the reason for using individual psychophysiological markers of EEG Arns et al (2012).

Arns et al (2012), suggested that selecting individual EEG biomarkers could improve the treatment outcome, but more research is needed to establish the efficacy of EEG biomarkers. Individualized neurofeedback can also be beneficial in treating the comorbid symptoms in children diagnosed with ADHD (Arns et al., 2012).

Holtman et al (2014), conducted a randomized study to examine the efficacy of slow cortical potentials (SCPs) neurofeedback as a non-pharmacological treatment option for ADHD. This study recruited 144 children and the researchers used German adaptation of Kiddie-Sads-Present and Lifetime Version (K-SADS-PL). This instrument assisted the researcher to rule out comorbid disorders such as major depressive disorder, anxiety disorders, and behavioral disorders by interviewing parent/s in the presence of the child. This instrument also included probes and scoring criteria for symptom assessment based on DSM-III and DSM-IV (Kaufman et al., 1999).

Holtman et al (2014), provided 25 sessions (60 minutes each) of SCP neurofeedback to the experiment group. The clinical global impression scale (CGI) is used to estimate symptom severity (CGI-S) and improvement (CGI-I). The clinical global impression scale (CGI) was a subjective assessment tool, because it compared the severity of symptoms among the group. Testbattery for Attentional Performance (TAP) used the go/nogo and flexibility (non-verbal) to assess the ability to suppress a response to an irrelevant stimuli and ability to shift attentional focus respectively (Holtman et al., 2014).

Holtman et al (2014), suggested that the CNV could be associated with improved self-regulatory abilities in children and provided reduction in the ADHD symptomatology. Other studies have also found that the NF training generated a "more negative" CNV in children with ADHD compared to the control group (Heinrich et al., 2007). Holtman et al (2014), suggested that the CNV modulation is important in addressing impaired preparatory processes and responding to a stimulus in the treatment of inattention symptoms of ADHD. A recent study provided evidence for long term post EEGneurofeedback (6 month follow up) benefits in children with ADHD (Gevensleben et al., 2010).

These positive results after EEG neurofeedback training in the children with ADHD are welcome news, but it should be noted that the majority of these studies had serious methodological problems. The most common limitations included small sample size, non-randomized group assignment, and lack of control conditions (double-blind study). Only three studies randomized group assignments (Gevensleben et al., 2010; Heinrich et al., 2004; Levesque et al., 2006). There is an immense need for large size randomized control trials to establish the effectiveness and efficacy of EEG biofeedback. There is also a big need to refine and standardize the treatment protocols.

Lack of coverage for this time and labor-intensive technique, and high number of treatment session are some of the major obstacles to expand this field. Electroencephalogram (EEG) biofeedback equipment ranges from \$5,000 to \$20,000. The major criticism of EEG biofeedback treatment is the big time commitment from both parents and children (Holtman et al., 2014). A typical treatment includes on average, 20-40 sessions, which can be costly, because the majority of health insurance plans do not cover neurofeedback treatment (Masterpasqua, 2013).

CONCLUSION:

The complex interaction between PFC, neurotransmitters, and various tracks that connect PFC to amygdala and basal ganglia makes it challenging to develop a universal EEG protocol. Children diagnosed with ADHD experience an increase in their theta wave activity and reduction in their beta wave activity. With age the symptoms of hyperactivity diminishes and the symptoms of impulsivity persists. This information can help to create more effective neurofeedback training programs.

This preliminary review of the literature found that neurofeedback is effective in managing symptoms of inattention and hyperactivity. The slow cortical potential (SCP) and Beta/Theta neurofeedback also suggested improvement in IQ. Effective management of the symptomatology and an increase in IQ can significantly improve the academic performance of children diagnosed with ADHD. Neurofeedback affects both behavioral as well as neurophysiological parameters such as attention, preparation, time to process a stimulus, and inhibitory ERP components (CNV, P 300).

In the recent years, the number and the scientific quality of research on EEG-based neurofeedback for ADHD have grown significantly. Studies included in this review suggested that neurofeedback training improved the symptoms of ADHD. However, it is important to discuss if factors such as self-efficacy, relaxation, structured learning environment and routines might have caused the changes in brain activity.

Irrespective of cost, time commitment, and agerelated changes in EEG, neurofeedback assisted selfregulation can help a child diagnosed with ADHD better manage ADHD symptoms.

REFERENCE:

- 1. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th edition. Arlington, VA., American Psychiatric Association, 2013. Retrieved from http://www.cdc.gov/ncbddd/adhd/diagnosis.html
- Arns, M., Drinkenburg, W., & Kenemans, L. J. (2012). The effects of QEEG-informed neurofeedback in adhd: An open-label pilot study. *Applied Psychophysiology Biofeedback*. 37(3),171-180. doi: 10.1007/s10484-012-9191-4 Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/P MC3419351/
- Arns, M., Ridder, de S., Strehl, U., Breteler, M., Coenen, A (2009). Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta- analysis. *Clinical EEG and Neuroscience*, 40(3), 180-9. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19715 181/
- Arnsten, F. T. A., & Li, B. M. (2005). Neurobiology of Executive Functions: Catecholamine Influences on Prefrontal Cortical Functions. *Biological Psychiatry*, 57 (11), 1377-84. doi: 10.1016/j.biopsych.2004.08.019
- Baydala, L., & Wikman, E. (2001). The efficacy of neurofeedback in the management of children with attention deficit/hyperactivity disorder. *Pediatric Child Health*, 6(7), 451- 455. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2 807759/
- 6. Bresnahan, S. M., Anderson, J. W. & Barry, R. J. (1999). Age-related changes in

quantitative EEG in attentiondeficit/hyperactivity disorder. *Biological Psychiatry*, 46(12), 1690-7. http://www.ncbi.nlm.nih.gov/pubmed/10624551

- Brennan, A. R. & Arnsten, A. F. (2008) Neuronal mechanisms underlying attention deficit hyperactivity disorder: the influence of arousal on prefrontal cortical function. *Annals of the New York Academy of Sciences*,1129, 236-245. doi: 10.1196/annals.1417.007
- Castellanos, F. X., Margulies, D. S., Kelly C., Uddin, L. Q., Ghaffari, M., Kirsch, A., . . . Biswal, B. (2008). Cingulate-precuneus interactions: a new locus of dysfunction in adult attention-deficit/hyperactivity disorder. *Biological Psychiatry*, 63(3), 332-337. a. doi: 10.1016/j.biopsych.2007.06.025
- Charach, A, Figueroa, M., Chen, S., Ickowicz, A., & Schachar, R.. Stimulant treatment over 5 years: effects on growth. (2007). Journal of American Academy Child and Adolescent Psychiatry, 45, 415-421. doi: 10.1097/01.chi.0000199026.91699.20.
- Dai, D., Wang, J., Hua, J., & He, H. (2012). Classification of ADHD children through multimodal magnetic resonance imaging. *Frontiers in Systems Neuroscience*, 3, 6:63. doi: 10.3389/fnsys.2012.00063
- 11. Drechsler, R., Straub, M., Doehnert, M., Heinrich, H., Hans-Christoph, S., & Brandeis, D. (2007). Controlled evaluation of а neurofeedback training of slow cortical potentials in children with Attention Deficit/Hyperactivity (ADHD). Disorder 3. Behavioral and Brain Functions. 35. doi: 10.1186/1744-9081-3-35
- Folstein, J. R., Palmeri, T.J., & Gauthier, I. (2013). Category learning increases discriminability of relevant object dimensions in visual cortex. *Cerebral Cortex*, 23, 814-823. doi: 10.1093/cercor/bhs067.
- 13. Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Jochen Kaiser, J. (2003).
- 14. Neurofeedback treatment for attentiondeficit/hyperactivity disorder in Children: A comparison with methylphenidate. *Applied Psychophysiology and Biofeedback*, 28(1),
- 15. Retrieved from http://www.appliedneuroscience.com.au/resource s/Documents/Fuchs%202003%20neuro feedback%20comp%20with%20ritalin%20ritalin .pdf
- Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz., Studer, P., ... Heinrich, H. (2010). Neurofeedback training in children with

ADHD: 6-month follow-up of a randomized controlled trial. European Child & Adolescent Psychiatry, 19(9), 715-724. doi: 10.1007/s00787-010-0109-5

- 17. Haas, L. (2003). Hans Berger (1873–1941), Richard Caton (1842–1926), and electroencephalography. Journal of Neurology, Neurosurgery, and Psychiatry. 74(1), 9doi: 10.1136/jnnp.74.1.9
- Hardt, J. V. (2012). Alpha brain-wave neurofeedback training reduces psychopathology in a cohort of male and female Canadian aboriginals. *Advances in Mind Body Medicine*, 26(2), 8-12. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/23341412
- Heinrich, H., Gevensleben, H., Freisleder, F. J., Moll, G. H., & Rothenberger, A. Training of slow cortical potentials in attentiondeficit/hyperactivity disorder: evidence for positive behavioral and neurophysiological effects. *Biology of Psychiatry*, 55(7), 772-5. Retrieved from
 - http://www.ncbi.nlm.nih.gov/pubmed/15039008
- Heinrich, H., Gevensleben, H., & Strehl, U. (2007). Annotation: neurofeedback—train your brain to train behaviour. *Journal of Child Psychology and Psychiatry*. 48, 3-16. doi: 10.1111/j.1469-7610.2006.01665.x. http://www.ncbi.nlm.nih.gov/pubmed/17244266

21. Holtmann, M., Pneiwski, B., Wachtlin, D.,

- Holtmann, M., Phelwski, B., Wachulin, D., Worz, S., & Strehl, U. (2014). Neurofeedback in children with attention-deficit/hyperactivity disorder (ADHD) – a controlled multicenter
 - study of a non-pharmacological treatment approach. *BioMedical Central Pediatrics*, 2014; 14(202), 1-10. doi: 10.1186/1471-2431-14-202

http://www.ncbi.nlm.nih.gov/pubmed/25123917

- Itami, S. & Uno, H. (2002) Orbitofrontal cortex dysfunction in attention-deficit hyperactivity disorder revealed by reversal and extinction tasks. *Neuroreporter*,13(18), 2453-7. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12499848
- 23. Jasper, H. H. (1958). Report of the committee on methods of clinical examination in electroencephalography: 1957. *Electroencephalography and Clinical Neurophysiology*, 10 (2), 370–375. doi: 10.1016/0013-4694(58)90053-1
- 24. Kaufman, J., Birmaher, B., Brent, D., Rao, U., & Ryan, N. (1996). Kiddie-Sads-Present and Lifetime Version (K-SADS-PL). Retrieved from

http://www.psychiatry.pitt.edu/node/8233

25. Kornhuber, H. H., & Deecke, L. (1990).

Readiness for movement – The Bereitschaftspotential- Story, Current Contents Life Sciences 33: 14. Retrieved from http://www.garfield.library.upenn.edu/classics19 90/A1990CH18100001.pdf

- 26. Kovatchev, B., Cox, D., Hill, R., Reeve, R., Robeva, R., & Loboschefski, T. (2001). A psychophysiological marker of attention deficit/hyperactivity disorder (ADHD)-Defining the EEG consistency index. Applied Psychophysiology and Biofeedback, 26(2), 127-140. Retrieved from http://web.b.ebscohost.com.ezproxy.humanisticp sychology.org:2048/ehost/pdfviewer/pdf viewer?vid=5&sid=b56d3aaa-e9d6-4736-a19ac63f038d7f00%40sessionmgr112&hid=118
- Lansbergen, M. M., Dongen-Boomsma, van. M., Buitelaar, J. K., & Slaats-Willems, D. (2010). ADHD and EEG-neurofeedback: a doubleblind randomized placebo-controlled feasibility study. *Journal of Neural Transmission*, 118(2), 275-284.

a. doi: 10.1007/s00702-010-0524-2

- Leins, U, Goth, G., Hinterferger, T., Klinger, C., Rumpf, N, & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and Theta/Beta protocols. Application of Psychophysiology Biofeedback, 32, 73-88. Doi: 10.1007/s10484-007-9031-0
- 29. Levesque, J., Beauregard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attentiondeficit/hyperactivity disorder: a functional magnetic resonance imaging study. *Neuroscience Letters*, 394(3), 216-21. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16343769 ?dopt=AbstractPlus
- 30. Markowitz, J. S., Logan, B. K., Diamond, F., & Patrick, K. S. (1999). Detection of the novel metabolite ethylphenidate after methylphenidate overdose with alcohol congestions. Journal ofClinical Psychopharmacology, 362-6. 19(4), doi: 10.1097/00004714- 199908000-00013
- Masterpasqua, F., & Healey, K. N. (2003). Neurofeedback in psychological practice. American Psychological Association, 34(6), 652-656. doi: 10.1037/0735-7028.34.6.652
- Matthews, N., Vance, A., Cummins, T. D., Wagner, J., Connolly, A., Yamada, J.Bellgrove, M, A. (2012). The COMT Val158 allele is associated with impaired delayed-match-tosample performance in ADHD. *Behavior of Brain Function*, 28, 8-25. doi: 10.1186/1744-9081-8-25

33. Molina, S. B., Hinshaw, P. S., Swanson, M. J., Arnold, L. E., Vitiello, B., Jensen, S. P., Epstein, N. J. Houck, R. P. (2009). The MTA at 8 years: prospective follow-up of children treated for combined type ADHD in a multisite study. *Journal of American Academy of Child Adolescent Psychiatry*, 48(5), 484-500.

a. doi: 10.1097/CHI.0b013e31819c23d0

- 34. Monastra, J. V., Lynn, S., Linden, M., Lubar, F. J., Gruzeller, J., La Vaque, L., & Theodore, J. (2005). Electroencehalographic biofeedback in the treatment of attention-deficient/hyperactivity disorder. *Applied Physiology and Biofeedback*, 30(2), 323-42. doi: 10.1007/s10484-005-4305-x
- 35. Patel, S. H., & Azzam, P. N. (2005). Characterization of N200 and P300: Selected studies of the event-related potential. International Journal of Medicine, 2(4),147-154. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1 252727/
- 36. Reis, D. J. & Piletz, J. E. (1997). The imidazoline receptor in control of blood pressure by clonidine and drugs. American Journal of Physiology, 273 (5), R1569–R1571. Retrieved from http://ajpregu.physiology.org/content/ajpregu/27 3/5/R1569.full.pdf
- Rojas, N. L., & Chan, E. (2005). Old and new controversies in the alternative treatment of attention hyperactive disorder. *Mental Retardation and Development Disabilities Research Reviews*, 11, 116-130. doi: 10.1002/mrdd.20064
- Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating ADHD: Part II. replication. *Applied Psychophysiology and Biofeedback*, 29(4), 233-243. doi: 10.1007/s10484-004-0383-4
- Schwartz, M.S., & Andrasik F. (2003). Biofeedback: A practitioner's guide (3rd ed.). New York, NY: The Guilford Press.
- 40. Snyder, S. M., Hall, J. R. (2006). A metaanalysis of quantitative EEG power associated with attention-deficit. Journal of Clinical Neurophysiology, 23(5), 440-55. Retrieved from http://www.qeeg.com/qeegfact.html
- Sokhadze, E. M., Baruth, J. M., Sears, L., Sokhadze, G. E., El-Baz, A. S., Williams, E., ...5 Casanova1, M. F. (2012). Event-related potential study of attention regulation during illusory figure categorization task in ADHD, autism spectrum disorder, and typical children. *Journal of Neurotherapy*, 16(1), 12-31. doi: 10.1080/10874208.2012.650119.

- Shouse, M. N. & Lubar, J.F. (1979). Operant conditioning of EEG rhythms and ritalin in the treatment of hyperkinesis. *Biofeedback Self Regulation*, 4(4), 299-312. Retrieved from: http://www.ncbi.nlm.nih.gov/pubmed/526475
- 43. Tansey, M. A. (1984). EEG sensorimotor rhythm biofeedback training: some effects on the neurologic precursors of learning disabilities. International Journal of Psychophysiology, 1 (2), 163-77. doi:10.1016/0167-8760(84)90036-9
- 44. Tsai, C. S., Huang, Y. S., Wu, C. L., Hwang, F. M., Young, K. B., Tsai, M. H., Chu, S. M. (2013). Long-term effects of stimulants on neurocognitive performance of Taiwanese children with attention deficit/hyperactivity disorder. BioMedical Center of Psychiatry, 4, 13:330. doi: 10.1186/1471-244X-13-330
- 45. Visser, S., Danielson, M., & Bitsko, R. (2014). Trends in the parent-report of health care provider-diagnosis and medication treatment for ADHD disorder: United States, 2003–2011. *Journal of American Academy of Child and Adolescent Psychiatry*. 53(1), 3446.e2. Retrieved from http://www.cdc.gov/nchdd/adhd/features/key.

http://www.cdc.gov/ncbddd/adhd/features/key-findings-adhd72013.html

- 46. Walter, W. G, Cooper, R., Aldridge, V. J., McCallum, W. C., Winter, A. L. (1964). Contingent Negative Variation: an electric sign of sensorimotor association and expectancy in the human brain. *Nature*, 203(4943), 380-384. doi:10.1038/203380a0
- 47. Wigal, S. B. (2009). Efficacy and safety limitations of attention-deficit hyperactivity disorder pharmacotherapy in children and adults. *CNS Drugs*, 23, Suppl.1: 21-31. Retrieved from http://www.coping.us/images/Wigal_2009_ADH D_Med_Effects.pdf
- 48. Wigal, S. B., Biederman, J., Swanson, J. M., Yang, R., & Greenhill, L. L. (2006). Efficacy and safety of modafinil film-coated tablets in children and adolescents with or without prior stimulant treatment for attentiondeficit/hyperactivity disorder: Pooled analysis of 3 randomized, double-blind, placebo-controlled studies. Primary Care Companion of Journal of Clinical Psychiatry, 8(6),352-360. Retrieved from

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1 764520/