EEG NEUROFEEDBACK – A NOVEL TREATMENT MODALITY FOR CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

ARUN MP

Assistant Professor, Dept. of Psychiatry, Academy of Medical Sciences, Pariyaram.

Correspondence: Assistant professor, Dept. of Psychiatry, Academy of Medical Sciences, Pariyaram, Kannur 670503.
Email: arunpalayat@gmail.com

ABSTRACT

EEG neurofeedback (EEG-NF) is a novel treatment modality. Many studies have assessed its efficacy for childhood ADHD. There is some evidence that it improves inattention and other behavioral and cognitive symptoms of ADHD. It is associated with minimal adverse effects, and can be used alone or in combination with medications to enhance their efficacy. Though double-blind placebo controlled studies have recently raised some doubts about its efficacy, EEG-NF remains an option to consider in ADHD management at least as an add-on to pharmacological interventions.

Keywords: Attention Deficit Hyperactivity Disorder, children, EEG neurofeedback

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common childhood and adolescent behavioral disorders.[1] A recent Indian study reported a community prevalence rate of 11.3%.[2] The illness predominantly affects boys,[3] and frequently leads to significant social and academic difficulties.[4] Without effective treatment, children with ADHD are at greater risk to develop academic and behavioral problems, beget co- morbid psychiatric conditions like mood and anxiety disorders or substance use disorders,[5,6] or incur accidental injuries.[7]

NEUROBIOLOGY OF ADHD

Current etiological theories of ADHD emphasize the central role of attentional and executive dysfunctions.[8,9] A growing body of evidence supports a model in which multiple genetic and environmental factors interact during early development to create a neurobiological susceptibility to the disorder, the expression of which is mediated by alterations in different neural networks and deficits in the neuropsychological functions they subserve.[10] Frontostriatal network is a likely contributor to the pathophysiology of ADHD, and reductions have been observed in the total cerebral volume and in the volumes of prefrontal cortex, basal ganglia (striatum), dorsal anterior cingulate cortex, corpus callosum and cerebellum in those affected by the condition.[11] A developmental trajectories study revealed

Please cite this article as: Arun MP. EEG neurofeedback – a novel treatment modality for children with Attention Deficit Hyperactivity Disorder. Kerala Journal of Psychiatry 2015; 28(1):59-65.

EEG Rhythm	Frequency(Hz)	Associated Mental States
Delta	1-4	Sleep. Dominant in infants
Theta	3-7	Drowsiness
Alpha	8-12	Alertness
SMR	12-15	Mentally alert
Beta	13-21	Focused
High Beta	20-32	Intensity
Gamma	38-42	Important in learning

Table 1: *Scalp EEG frequencies associated with various mental states*

a delay in cortical maturation, and demonstrated that different clinical outcomes may be associated with different developmental trajectories in adolescence and beyond.[12]

CURRENT TREATMENT OPTIONS

Both psychological and pharmacological therapies have been shown to benefit ADHD patients.[13] Medications like methylphenidate (MPH), atomoxetine and clonidine, and psychological interventions like behavioral therapy, parent training and social skills training are commonly recommended in ADHD treatment. Though medications are considered the most effective treatment, [14] there is insufficient evidence for their long term efficacy.[15] Studies show that 2-10% of patients prescribed MPH discontinue it due to adverse effects.[16] Stimulants cannot be used in those with previous sensitivity to them, glaucoma, symptomatic cardiovascular disease, hyperthyroidism, or hypertension. Besides, children with ADHD and their parents are concerned about possible long-term effects of medications.[17] Though psychological interventions are often advocated and used, according to a recent metaanalysis of RCTs, studies that employed blinded assessments have not found significant efficacy to most such interventions.[18] All these point to a need for other treatment modalities that are more effective and acceptable.

BRAINWAVES IN ADHD

Scalp electroencephalogram (EEG) frequencies have been broadly associated with various mental states (Table 1).[19] Scalp EEG can be quantitatively measured using spectral analysis, and Quantitative EEG (QEEG) studies demonstrate deviations from the normal pattern in many neuropsychiatric conditions, including ADHD.[20]

Children with ADHD have been found to have increased theta (slow wave) activity and decreased beta (fast wave) activity than normal controls.[21] QEEG studies of ADHD patients have consistently revealed increased frontocentral theta band activity and increased theta to beta (θ/β) power ratio during rest.[22] 85-90% of ADHD patients display signs of cortical "hypoarousal" — quantitatively described as elevated relative theta power, reduced relative alpha and beta power, and elevated theta/alpha and theta/beta power ratios. These changes are mainly seen over the frontal and central midline areas. A smaller

subgroup of patients exhibits an EEG pattern suggestive of "hyperarousal" — with greater relative beta activity, decreased relative alpha activity, and decreased theta/beta power ratios diffusely across multiple cortical recording sites.[23]

EEG NEUROFEEDBACK— AN INTRODUCTION

The concept of EEG neurofeedback (EEG-NF) originated from observation by Sterman that cats conditioned to produce a specific EEG frequency called Sensory Motor Rhythm (SMR; 12-15Hz) exhibited an elevated seizure threshold when exposed to the convulsant agent methyl hydrazine.[24] Subsequent studies by Sterman and others demonstrated that approximately 80% of patients with medically intractable epilepsy experience a clinically significant (>50%) reduction in seizure frequency after a course of EEG-NF that rewards the SMR frequency.[25]

Modern EEG-NF systems consist of a set of EEG sensors and a signal amplifier connected to computers with software capable of analyzing the EEG signals, displaying relevant signals to the patient, and providing rewards or inhibitions in the form of feedbacks through visuals, sounds, or both. A typical EEG-NF setup involves the patient seated in a reclining chair, watching a video display that provides video and audio feedback, while the therapist monitors a second video display that provides detailed, real-time data on the patient's EEG during the session. A course of EEG-NF consists of at least 20 half-hour sessions administered over a 6- to 12-week period. EEG-NF have been tried in psychiatric disorders like addiction.[26]

EEG-NF FOR ADHD

Several studies have shown that EEG-NF is an efficacious and promising treatment for ADHD. Here, children with ADHD learn to enhance the desirable EEG frequencies and suppress the undesirable frequencies at the selected scalp location(s). This is achieved by rewarding them, for example by allowing to progress in a video game, when they are able to increase the desirable frequencies and/or reduce the undesirable frequencies. For hypoaroused patients, Lubar JF have developed EEG-NF protocols that inhibit cortical slowing and reward higher frequencies, with the goal of normalizing EEG activity in regions considered responsible for attention and behavioral control.[27] Children with ADHD who received EEG-NF training have been able to decrease theta activity, which is associated with daydreaming or distraction, and increase beta activity, which is associated with sustained attention.[28]

EFFICACY OF EEG-NF IN ADHD

Multiple studies, of varying methodological vigor, have evaluated the efficacy EEG-NF in treatment of children with ADHD.

Case studies: Thompson and Thompson reported improvements in QEEG and performance in a continuous performance task, and a mean gain in full-scale IQ of 12 points, after 40 sessions of EEG-NF in111 ADHD patients.[29] Kaiser and Othmer reported that EEG-NF improved attentiveness and impulse control in186 patients with ADHD.[30]

Studies with a control group: Rossiter and La Vaque compared the effects of 20 sessions of EEG-NF with effects of stimulant medication (MPH or dextro-ampetamine) in 46 ADHD patients aged 8-21 years divided into two matched groups. The EEG-NF group demonstrated significant improvements in several psychometric test scores, though no significant intergroup differences were noticed.[31]

A randomized waiting-list trial involving 16 ADHD children aged 8-10 years was conducted by Carmody et al.[32] 12 patients treated with EEG-NF exhibited reduced impulsivity and were rated more attentive by their teachers. However, follow-up QEEG testing did not demonstrate consistent electrophysiological improvement after EEG-NF.

Linden et al studied 18 ADHD children aged 5-15 years after randomly assigning them to a "waiting list" or an EEG-NF group. The EEG-NF group demonstrated a significant increase in IQ (nine points) compared to the control group, and there was a significant decrease in their inattentive behaviors as rated by parents.[33]

The largest published controlled trial of EEG-NF for ADHD was conducted by Monastra et al.[34] Ten patients aged 6 to 19 years were divided into two groups. One group received MPH, while the other group received both MPH and EEG-NF. Post-treatment assessments were conducted after one year of therapy while patients continued to take MPH and then after a one-week medication washout. The EEG-NF-plus-medication group, which received an average of 43 sessions of EEG-NF designed to reduce cortical slowing to within one standard deviation of same age peers, had greater reductions in inattention and hyperactivity as reported by parents and teachers. Only this group demonstrated a sustained improvement after medication wash out.

deBeus et al used a double blind cross over design for 52 ADHD children aged 7- 10 years. Half of them had the inattentive subtype of ADHD, while the other half had the combined subtype. All subjects received 20 NF sessions and 20 sham feedback sessions in random assignments. Active treatment protocol in this study was theta suppression, and enhancement of SMR or beta. NF resulted in better ADHD ratings at home, better ability to work with others, and better results in computerized assessment of attention. [35]

Levesque et al evaluated the impact of EEG-NF on brain function in ADHD using functional magnetic resonance imaging (fMRI) in conjunction with psychometric tests.[36] After EEG-NF sessions, the children exhibited improved attentional performance and distinctive activation of the right anterior cingulate cortex on fMRI. Such changes were not observed in the untreated controls.

A recent Chinese study too evaluated the efficacy of combined MPH and EEG-NF. 40 ADHD patients aged 7 to 16 years were randomly assigned to the combination group (MPH and EEG-NF) or the control group (MPH and non-feedback attention training). Patients were evaluated at baseline, after 20 treatment sessions, 40 treatment sessions, and six months. The combination group had significantly better symptom reduction and improvements in related behavioral and brain functions.[37]

LIMITATIONS IN AVAILABLE RESEARCH

Majority of the available studies on efficacy of EEG-NF in ADHD have methodological flaws like lack of blinding or absence of a placebo arm. Methodologically sound studies are still lacking in this area. Incorporation of a placebo feedback condition can minimizes the effect of unspecific factors and help in blinding. However, use of a placebo group for a condition like ADHD may be unethical as other effective treatments are available.[38] It should also be noted that some studies that did include a placebo group or a blinded design did not find EEG-NF to be superior toplacebo-NF.[39, 40, 41]

ADVERSE EFFECTS

EEG-NF is associated with minimal adverse effects. There are chances of increased irritability, moodiness, and hyperactivity when stimulants and EEG-NF are combined.[42] Dose of stimulants might have to be reduced or eliminated once improvement is noticed after NF sessions. Patients occasionally report transitory headaches, tiredness, and/or dizziness after the sessions. EEG-NF also has the potential to decrease or increase the seizure threshold depending on the frequencies and sensor locations used, as reported by Sterman in his original studies.[43] Patients with a history of epilepsy should receive NF only from practitioners well-versed in EEG-NF for seizure disorders.

INDIAN SCENARIO

Not many centers in our country use this modality for children with ADHD yet. Subsequently, no Indian data on efficacy of EEG-NF in ADHD is currently available. Department of Neuropsychology, NIMHANS, Bangalore has the facility to use NF as an optional treatment for children with ADHD. Some private centers, like Medha Mind Enhancement Co (P) Ltd, Chennai, too use EEG-NF to treat some neurological disorders, autism, and ADHD.

SUMMARY AND CONCLUSIONS

EEG-NF is a novel therapeutic method for ADHD, and there is some evidence that it improves the patients' inattention and other behavioral and cognitive symptoms. EEG-NF is associated with minimal adverse effects, and can be used alone or in combination with medications to enhance their efficacy. Though double-blind placebo controlled studies have recently raised some doubts about its efficacy, EEG-NF remains an option to consider in ADHD management at least as an add-on to pharmacological interventions.

REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (4th ed). Washington, DC; 2000.
2. Barkley, RA. Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment (2nd ed.) New York: Guilford; 1990.
3. Clarke C, Prior M, Kinsella G. The relationship between executive function abilities, adaptive behaviour, and academic achievement in children with externalising behaviour problems. J Child Psychol Psychiatry 2002; 43:785-96.
4. Venkata JA, Panicker AS. Prevalence of attention deficit hyperactivity disorder in primary school children. Indian J Psychiatry 2013; 55:338-42.
5. Biederman J, Faraone S, Milberger S, Guite J, Mick E, Chen L, et al. A prospective 4-year follow-up study of attention-deficit hyperactivity and related disorders. Arch Gen Psychiatry 1996; 53:437–46.
6. Claude D, Firestone P. The development of ADHD boys: A 12 year follow-up. Can J Behav Sci 1995; 27:226–49.
7. Lahey BB, Pelham WE, Stein MA, Loney J, Trapani C, Nugent K, et al. Validity of DSM-IV attention-deficit/hyperactivity disorder for

younger children. J Am Acad Child Adolesc Psychiatry 1998; 37(7): 695–702.
8. Sergeant JA, Geurts H, Huijbregts S, Scheres A, Oosterlaan J. The top and the bottom of ADHD: a neuropsychological perspective. Neurosci Biobehav Rev 2003; 27:583-92.
9. Castellanos FX, Sonuga-Barke EJ, Milham MP, Tannock R. Characterizing cognition in ADHD: beyond executive dysfunction. Trends Cogn Sci 2006; 10:117-23.
10. Sonuga-Barke EJ, Halperin JM. Developmental phenotypes and causal pathways in attention deficit/hyperactivity disorder: potential targets for early intervention? J Child Psychol Psychiatry 2010; 51:368-89.
11. Emond V, Joyal C, Poissant H. Structural and functional neuroanatomy of attention-deficit hyperactivity disorder (ADHD). Encephale 2009; 35:107-14.
12. Shaw P, Rabin C. New insights into attention-deficit/hyperactivity disorder using structural neuroimaging. Curr Psychiatry Rep 2009; 11:393-98.
13. The MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 1999; 56(12):1073–86.
14. Araone SV, Buitelaar J. Comparing the efficacy of stimulants for ADHD in children and adolescents using meta-analysis. Eur Child Adolesc Psychiatry 2010; 19:353–64.
15. VandeLoo-Neus GH, Rommelse N, Buitelaar JK. To stop or not to stop? How long should treatment of attention-deficit hyperactivity disorder be extended? Eur Neuropsychopharmacol 2011; 21:584–99.
16. Kutcher S, Aman M, Brooks SJ, Buitelaar J, van Daalen E, Fegert J, et al. International consensus statement on attention-deficit/hyperactivity disorder (ADHD) and disruptive behaviour disorders (DBDs): clinical implications and treatment practice suggestions. Eur Neuropsychopharmacol. 2004; 14:11-28.
17. Berger I, Dor T, Nevo Y, Goldzweig G. Attitudes towards attention deficit hyperactivity disorder (ADHD) treatment. Parents and children's perspectives. J Child Neurol 2008; 23:1036-42.
18. Sonuga-Barke EJ, Brandeis D, Cortese S, Daley D. Nonpharmacological interventions for ADHD: systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. Am J Psychiatry 2013; 170(3):275-89.
19. Patrick N, Friel BS. EEG biofeedback in the treatment of attention deficit/ hyperactivity disorder. Alternative medicine Review, 2007; 12(2):146-51.
20. Hughes JR, John ER. Conventional and quantitative electroencephalography in psychiatry. J Neuropsychiatry Clin Neurosci 1999; 11:190-208.
21. Mann CA, Lubar JF, Zimmerman AW, Miller CA, Muenchen RA. Quantitative analysis of EEG in boys with attention-deficit hyperactivity disorder: Controlled study with clinical implications. Pediatr Neurol 1992; 8:30-6.
22. Monastra VJ, Lubar JF, Linden M. The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: reliability and validity studies. Neuropsychology 2001; 15:136-44.
23. Monastra VJ, Lynn S, Linden M, Lubar JF, Gruzelier J, LaVaque TJ. Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. Appl Psychophysiol Biofeedback 2005; 30:95-114.
24. Sterman MB. Effects of brain surgery and EEG operant conditioning on seizure latency following monomethyl hydrazine intoxication in the cat. Exp Neurol 1976; 50:757-65.
25. Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. Clin Electroencephalogr 2000; 31:45-55.
26. Burkett SV, Cummins JM, Dickson R, Skolnick MH. An open clinical trial utilizing real-time EEG operant conditioning as an adjunctive therapy in the treatment of crack cocaine dependence. J Neurother 2005; 9(2):27–47.
27. Lubar JF. Discourse on development of EEG diagnostics and Biofeedback for attention deficit / hyperactivity disorders. Biofeedback Self Regul. 1991; 16(3):201-25.
28. Mann CA, Lubar JF, Zimmerman AW, Miller CA, Muenchen RA. Quantitative analysis of EEG in boys with attention-deficit hyperactivity disorder: Controlled study with clinical implications. Pediatr Neurol 1992; 8:30-6.
29. Thompson L, Thompson M. Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD.

Appl Psychophysiol Biofeedback 1998; 23:243-63.
30. Kaiser DA, Othmer S. Effect of neurofeedback on variables of attention in a large multi-center trial. J Neurother 2000; 4:5-28.
31. Rossiter TR, La Vaque TJ. A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. J Neurother 1995; 1:48-59.
32. Carmody DP, Radvanski DC, Wadhwani S, Sabo MJ, Vergara L. EEG biofeedback training and attention-deficit/ hyperactivity disorder in an elementary school setting. J Neurother 2001; 4:5-27.
33. Linden M, Habib T, Radojevic V. A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. Biofeedback Self Regul 1996; 21:35-49.
34. Monastra VJ, Monastra DM, George S. The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/ hyperactivity disorder. Appl Psychophysiol Biofeedback 2002; 27:231-49.
35. deBeus, R. Progress in efficacy studies of EEG biofeedback for ADHD. Presentation at the Annual Meeting of American Psychiatric Association 2006; Toronto, Canada.
36. Levesque J, Beauregard M, Mensour B. Effect of neurofeedback training on the neural substrates of selective attention in children with attention deficit/ hyperactivity disorder: a functional magnetic resonance imaging study. Neurosci Lett 2006; 394:216-21.
37. Li L, Yang L, Zhuo CJ, Wang Y. A randomized controlled trial of combined EEG neurofeedback and methyl phenidate therapy for the treatment of ADHD. Swiss Med Wkly 2013; 143:w13838.
38. La Vaque TJ, Rossiter T. The ethical use of placebo controls in clinical research: the Declaration of Helsinki. Appl Psychophysiol Biofeedback 2001; 26:23–37.
39. Perreau-Linck E, Lessard N, Levesque J, Beauregard M. Effects of neurofeedback training on inhibitory capacities in ADHD children: a single blind, randomized, placebo-controlled study. J Neurother 2010; 14:229–42.
40. Arnold LE, Lofthouse N, Hersch S, Pan X, Hurt E, Bates B, et al. EEG Neurofeedback for ADHD: double-blind sham-controlled randomized pilot feasibility trial. J Atten Disord 2012; 17:410–19.
41. vanDongen-Boomsma M, Vollebregt MA, Slaats-Willemse D, Buitelaar K. A randomized placebo-controlled trial of electroencephalographic (EEG) neurofeedback in children with attention-deficit/hyperactivity disorder J Clin Psychiatry 2013; 74:821–27.
42. Hammond DC, Kirk L. Negative effects and need for standards of practice in neurofeedback. Biofeedback 2007; 35(4):139-45.
43. Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. Clin Electroencephalogr 2000; 31:45-55.

Source of support: None *Conflict of interest:* None declared