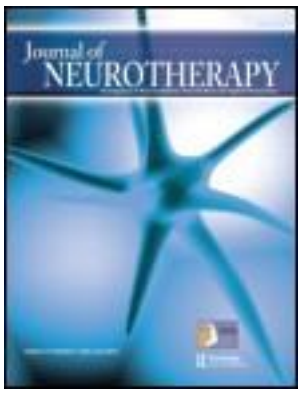

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EEG Biofeedback: A New Treatment Option for ADD/ADHD

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EEG Biofeedback: A New Treatment Option For ADD/ADHD

Marabella A. Alhambra, M.D., Timothy P. Fowler, and Antonio A. Alhambra, M.D.

Attention Deficit Disorder is commonly treated with stimulant medications such as ritalin (methylphenidate). However, this medication has short-term effects and numer - ous undesirable side-effects including insomnia and loss of appetite. This study explores using EEG biofeedback, with its minimal side-effects and long-term results, as an alter - native to pharmacological treatments for ADD.

Introduction

Attention Deficit Disorder with or without hyperactivity is a disorder commonly seen in children. It is estimated that ADD affects 5-15 percent of school age children (Linden, Habib, & Radojevic, 1993). It was previously thought that children outgrow the disorder, but it is now believed that 70 percent of children do not outgrow the problems associated with ADD (Linden, Habib, & Radojevic, 1993).

The causes of ADD and ADHD have a neurological basis. There is evidence that, in some ADD/ADHD children, there might be decreased levels of metabolism of catecholamine, brain chemicals related to adrenalin (epinephrine) and noradrenalin (norepinephrine) (Lubar, hand-out). Because of this, stimulant medications are often effective treatments for these children, especially in reducing hyperactivity. Children on the medication often show improved attentiveness and decreased impulsivity. However, a significant problem with the pharmacological treatment of ADD children is the state-dependent and short-lived effects. For example, the frequently prescribed medication Ritalin (Methylphenidate) lasts only for 3 or 4 hours in the nervous system. As soon as the medication wears off, full blown symptoms of ADD and ADHD appear (Lubar, hand-out). Furthermore, Ritalin has numerous undesirable side-effects such as insomnia, loss of appetite, inhibited growth, and depression. An ideal treatment is one with long-term results and minimal side-effects. EEG biofeedback is a non-pharmacological treatment with such characteristics.

The mechanism of how EEG biofeedback could help children with ADD/ADHD is based on the separation of certain brainwave patterns. The EEG frequency range has been divided into six (6) categories: delta: 0.5-4 Hz; theta: 4-8 Hz; alpha: 8-13 Hz; sensorimotor (SMR): 12-15 Hz; beta: 15-35 Hz; gamma: 35-50 Hz. Delta and theta are known as slow wave activity and are associated with states such as daydreaming and drowsiness. Alpha is associated with a relaxed state of unfocused attention. Beta is referred to as fast wave activity and is characterized by a state of high alertness, concentration, and focused attention (Linden, Habib, & Radojevic, 1993). Children with ADD and ADHD produce excess theta activity and lower amounts of beta activity (Lubar, 1991). Thus, these children are neurologically inclined to daydream, and less inclined to focus and concentrate. EEG biofeedback training functions to reverse this brain wave abnormality in ADD/ADHD children by inhibiting the amount of theta activities and simultaneously increasing beta activities.

Several studies (Linden, Habib, & Radojevic, 1993; Lubar & Shouse, 1976a, 1976b; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Tansey & Brunner, 1983) provided evidence that EEG biofeedback is a

beneficial method for treating the ADD condition. This paper is a report of a series of cases on the effects of EEG biofeedback on children with ADD/ADHD.

Method

Questionnaires were sent to patients who had completed at least 30 sessions of EEG biofeedback as a treatment for ADD/ADHD. A total of 43 questionnaires were sent. Thirty-two of the patients were male, 11 were female. Forty of the 43 patients fell between the ages of 7 and 15, and the remaining three were 17 years, 16 years, and one year.

The patients were diagnosed with ADD/ADHD based on the following: observations made by parents and teachers, Quantitative EEG, TOVA (Test of Variables Assessment, formerly known as the Minnesota Computer Assessment).

The questionnaire asked the parents or guardian of the patient to describe the child's symptoms, medication history, academic performance, school conduct, and social behavior before, during, and after EEG biofeedback treatment.

TOVA, QEEG, and Brainmapping results before and after 20 sessions were reviewed and changes were noted.

The EEG biofeedback equipment used is a product of Neurocybernetics. This system has a high-gain amplifier (10,000 x) and uses soft filters with a slope of 12 db/octave to filter the raw EEG into selected frequency bands.

The EEG monitoring was done by a monopolar electrode placement at Cz, according to 10/20 international electrode placement system, with two ear electrodes providing a reference and a ground. Each training session lasted for 30-45 minutes with the objective to inhibit theta activities while increasing SMR or beta waves. A 80486DX PC served as the computer interface which displaced the feedback signals. The feedback consisted of a Pac-man video game.

The sessions were conducted by a certified neurotherapist under supervision of a pediatric neurologist in a private clinic setting.

Effects of the EEG biofeedback training were evaluated by the following: 1) Observed changes by parents through questionnaires, 2) Comparison of TOVA scores before training and after 20 sessions, 3) QEEG changes at the end of sessions (30-60 sessions) in some patients.

Results

Of the 43 questionnaires sent, there were 36 responses; 26 responses were for male children (72%), and the remaining 10 were for females (28%). The ages of these 36 patients ranged from 6 to 17 years.

Thirty-one of the 36 respondents (86%) showed some overall improvement in their ADD/ADHD condition upon completion of the EEG biofeedback treatment. This improvement was judged to be significant in 30 patients, and slight in the remaining patient. Three of the 36 respondents showed no improvement after treatment. The remaining 2 responses indicated uncertainty as to whether or not there was any improvement.

All 36 patients had an initial TOVA test before starting EEG biofeedback. Thirty-three were abnormal, 3 were normal. Thirty-one of these patients with abnormal TOVAs had repeat TOVA tests after 20 sessions; 23 (74%) of these showed significant improvement of scores and 8 (26%) did not. All of those with increased TOVA scores improved clinically. Among the 8 patients without TOVA improvement, 4 (50%) showed clinical improvement and 4 (50%) did not. Four (80%) of the five patients who did not have clinical improvement also did not show improvement on the TOVA test. None was observed to be symptomatically worse after the sessions.

All patients in the study (36) had a QEEG before starting EEG biofeedback sessions. Only 10, however, were repeated at

the end of the sessions, and 9 of these improved clinically. Seven (78%) out of the 9 also showed improvement of the QEEG parameters. The QEEG changes observed are the following: decreased relative and absolute power of theta activities, less hemispheric asysemity, better poster and anterior hemisphere coherence, increased relative power of beta waves.

Table 1 shows the effects that EEG biofeedback had on the patients' pharmacological dependence. Twenty-four of the 36 patients who responded (66%) were on medication for their ADD/ADHD condition. Of this 24, 5 were able to be removed completely from their medication after the treatment. Eleven of the 24 showed a decreased dependence on their medication in that their dosage was able to be reduced. The remaining 8 initially on medication showed no change. Four of these 8, however, showed overall improvement, implying that this same dosage of drug was more effective. Twelve of the 36 respondents were not on medication before undergoing EEG biofeedback. Eleven of these 12 remained free from medication after treatment.

Table 2 shows the effects that EEG biofeedback had on conditions associated with ADD/ADHD. Four of the 36 respondents were suffering from seizures before treatment. After treatment 2 were no longer experiencing seizures, one was having fewer seizures, and one remained unchanged. Five of the 36 respondents reported headaches and/or abdominal pain. After the EEG biofeedback, all 5 improved, with 2 of the 5 no longer suffering from the condition. Two of the 36 patients suffered from nightmares before treatment, but not after treatment. There were 2 reports of bruxism (teeth-grinding), and this condition was resolved in one patient and improved in the other after treatment. Similarly, there were 2 reports of bed-wetting, with one case being resolved and the other improved after treatment. There were 2 reports of mood swings among the 36 respondents, with indication of improvement in both cases after EEG biofeedback. Likewise, the 2 reports of depression were judged to have improved after treatment. There were 3 reports of tics among the 36; 2 of these 3 reported improvement after treatment, and the remaining one reported no change. Finally, the one

Table 1
Effects of EEG Biofeedback on Medication Dependence

		After treatment		
Before treatment	n	On med.	No med	Decreased med.
Number	24	8*	5	11
Percentage		33%	24%	46%
Not on medication				
Number	12	1	11	—
Percentage		8	92	—

Note: "n" equals number of patients out of the 36 respondents.

* 4 of these 8 showed improvement; the same dosage was more effective.

Table 2
Effects of EEG Biofeedback on Problems Associated with ADD/ADHD

Problem	n	Status of problem after treatment		
		resolved	improved	unchanged
Seizures	4	2	1	1
HA & ABD pain*	5	2	3	—
Nightmares	2	2	—	—
Bruxism	2	1	1	—
Mood swings	2	—	2	—
Bed-wetting	2	1	1	—
Depression	2	—	2	—
Tics	3	—	2	1
Insomnia	1	—	—	1

Note: "n" equals the number of respondents experiencing the associated problem.

* "HA" and "ABD" refer to "headaches" and "abdominal."

report of insomnia among the 36 respondents was considered unchanged after EEG biofeedback.

Discussion

This study evaluates the effect of EEG biofeedback by subjective and objective parameters. Subjective observations from parents showed 86% improvement. There is a good correlation of observed clinical improvement to TOVA score improvement (74%) and changes in QEEG parameters (78%).

Clinical assessment of outcome was conducted 0 to 12 months after completion of the EEG biofeedback sessions. Long term effects or sustained benefits, therefore, could not be evaluated from this study.

Parameters predictive of benefits from EEG biofeedback will be helpful in clinical practice, especially during this era of managed health care. This is beyond the scope of this study. Future studies should address this important issue.

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Composite Biofeedback Conditioning and Dangerous Offenders: III

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* This report reflects the views of the author and not necessarily those of the Ontario Correctional Institute or the Ontario Ministry of the Solicitor General and Correctional Services.

Seventy-seven offenders, selected as subject to deep-brain complex seizures, were treated with varying amounts of a composite EEG-SMR and GSR-SCARS biofeedback conditioning procedure. Subjects were selected through successive screens, culminating in evidence of a perceptual-motor anomaly shown to predict to various types of dangerous criminal conduct. The lack of a recognizable prodrome that these subjects might use to cue voluntary self-regulation made it seem necessary to abandon the usual method of contin - uous analog biofeedback. An operant conditioning method was employed, which provid - ed discontinuous and contingent reinforcing feedback during all occurrences of EEG sen - sorimotor rhythm (SMR) and for successive 1K ohms increases in skin resistance (GSR). The mean duration of post-release follow-up was eighteen months. Criminal recidivism rates were shown to decrease roughly in proportion to the number of treatment sessions received. Recidivism rates varied from 65% for those receiving essentially no biofeedback treatment to 20% for those receiving more than 33 half-hour sessions. The results were interpreted as holding out hope for the identification and treatment modification of one subset of dangerous offenders.

Introduction

The difficulty of recognizing dangerous offenders has been well documented in the psychological literature (Quinsey & Maguire, 1986). The problem of treating them is even greater. If the variables controlling dangerousness cannot yet be specified in order to identify people who will manifest this attribute, it is obvious that those variables cannot yet be subjected to treatment modification to alter this human quality.

The psychological literature is replete with observations of borderline or atypical electrophysiological and neuropsychological indicators among offenders, and particularly among dangerous offenders. Unfortunately, few definitive criteria have appeared to permit any particular subgroup of offenders to be identified for specific investigation. Obviously, sub-groups of people have to be identified if the inferred causes which control their conduct are to be evaluated, for example, by discovering the effects of modifying those causes in treatment. The task of this paper is to investigate the relevance of a specific causal process to the dangerous criminal conduct of a definable sub-group of offenders.

Background Observations

The confluence of several separate observations provided a basis for recognizing one subset of dangerous offenders. Each of these observations requires brief comment.

Relevant Functional Neuroanatomy

There is a small area of the old brain, described by Olds and Milner (1954), and Olds and Olds (1965), called the 'drive cen-

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