Abstract

Electroencephalogram (EEG) biofeedback, also known as neurofeedback, is a promising alternative treatment for patients with attention deficit/hyperactivity disorder (AD/HD). EEG biofeedback therapy rewards scalp EEG frequencies that are associated with relaxed attention, and suppresses frequencies associated with under- or over-arousal. In large-scale clinical trials, the efficacy of EEG biofeedback for AD/HD is comparable to that of stimulant medications. Many different EEG biofeedback protocols for AD/HD are available. Single-channel protocols developed by Lubar and interhemispheric protocols developed by the Othmers are widely practiced and supported by large-scale clinical studies. (Altern Med Rev 2007;12(2):146-151)

Introduction

Attention deficit/hyperactivity disorder (AD/HD) affects approximately 3-5 percent of school-age children in the United States, and a majority of children diagnosed with AD/HD are treated with medications, primarily stimulants. It is estimated that 10 percent of 10-year old boys in the United States are currently being treated with prescription stimulants. Concerns about the cardiovascular toxicity of amphetamine and methylphenidate cause many patients and their families to seek alternative therapies. Well-established alternative therapies include dietary modifications and the administration of supplements, including vitamins, minerals, phytonutrients, amino acids, essential fatty acids, phospholipids, and probiotics. Another alternative to drug therapy for AD/HD is electroencephalogram (EEG) biofeedback, also known as neurofeedback, which is supported by extensive peer-reviewed literature, including large-scale controlled clinical trials. The purpose of this review is to summarize the evidence supporting the use of EEG biofeedback for treatment of AD/HD.

Background

The standard scalp EEG is recorded at 19 sites. Scalp EEG frequencies are broadly associated with various mental states, as shown in Table 1. With modern computerized systems, experts can map scalp EEG quantitatively by using spectral analysis. Quantitative electroencephalography (QEEG) studies demonstrate deviations from normal patterns in many neuropsychiatric conditions, including AD/HD.

Clinical EEG biofeedback originated with the observation by Sterman that cats conditioned to produce a specific EEG frequency (SMR; sensory-motor rhythm; 12-15 Hz) exhibited an elevated seizure threshold when exposed to the convulsant agent methylhydrazine. Subsequent studies by Sterman and others, conducted from the 1970s onward, demonstrated that approximately 80 percent of patients with medically intractable epilepsy experience a clinically significant (>50%) reduction in seizure frequency after a course of EEG biofeedback that rewards the SMR frequency. A different approach to treating patients with AD/HD is EEG biofeedback, which can be used as a stand-alone treatment or in combination with other therapies. EEG biofeedback is a relatively new treatment modality, and further research is needed to determine its long-term efficacy and safety.
Patients with AD/HD exhibit characteristic surface EEG disturbances. Specifically, 85-90 percent of patients with AD/HD 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 (Table 1). These patterns are typically observed over frontal and central midline brain regions. A smaller subgroup of AD/HD 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. The hyperaroused group tends to respond poorly to stimulant medications.

Lubar et al developed EEG biofeedback protocols to inhibit cortical slowing and reward higher frequencies in hypoaroused patients, with the goal of normalizing EEG activity in regions thought to be responsible for attention and behavioral control.4

Modern EEG biofeedback systems, sold by a number of manufacturers, consist of a set of EEG sensors and a signal transducer/amplifier, connected to a computer or computers with software capable of analyzing the EEG signals, performing various transformations, displaying relevant signals to the patient, and providing rewards or inhibitions in the form of visual and/or audio feedback. The client learns to enhance desirable EEG frequencies and suppress undesirable frequencies at the selected scalp location(s) by being rewarded (e.g., by progress in a video game) for increasing desirable frequencies and/or reducing undesirable frequencies. Scalp electrode placements along the sensory-motor strip (C3 and C4) and temporal lobes (T3 and T4) are widely used. A typical neurofeedback configuration involves the patient seated in a reclining chair, watching one 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 typical course of EEG biofeedback therapy involves at least 20 half-hour sessions, administered over a 6- to 12-week period. Although rates of progress vary from patient to patient, significant benefit is often observed within the first few weeks of therapy. Accreditation for EEG biofeedback practitioners is available through the Biofeedback Certification Institute of America.

### Table 1. EEG Rhythms and Associated Mental States

<table>
<thead>
<tr>
<th>EEG Rhythm</th>
<th>Frequency (hz)</th>
<th>Associated Mental States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delta</td>
<td>1 - 4</td>
<td>Sleep; dominant in infants</td>
</tr>
<tr>
<td>Theta</td>
<td>3 - 7</td>
<td>Drowsiness; “tuned-out;” inner-directed insights</td>
</tr>
<tr>
<td>Alpha</td>
<td>8 - 12</td>
<td>Alertness; meditation, dominant when eyes closed</td>
</tr>
<tr>
<td>SMR</td>
<td>12 - 15</td>
<td>Mentally alert; physically relaxed</td>
</tr>
<tr>
<td>Beta</td>
<td>13 - 21</td>
<td>Focused; sustained attention; problem solving</td>
</tr>
<tr>
<td>High Beta</td>
<td>20 - 32</td>
<td>Intensity; anxiety; hypervigilance</td>
</tr>
<tr>
<td>Gamma</td>
<td>38 - 42</td>
<td>Important in learning</td>
</tr>
</tbody>
</table>

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Clinical Trials

A recent review discusses the evidence supporting EEG biofeedback for AD/HD, collected in case studies and controlled-group trials. The studies reviewed all employed single-channel neurofeedback, based on the original work of Lubar et al. Case series demonstrating favorable outcomes with EEG biofeedback for AD/HD include one group of 111 and another of 186 subjects. In the case series of 111 patients, who were treated with 40 sessions of neurofeedback, Thompson and Thompson reported improvements in quantitative EEG and performance in a continuous performance task, as well as a mean gain in full-scale IQ of 12 points after neurofeedback. Kaiser and Othmer reported a series of 1,089 patients, 186 with AD/HD. They described significant improvement in measures of attentiveness and impulse control using a test of variables of attention (TOVA).

Five controlled-group studies that appeared in peer-reviewed journals between 1995 and 2003 were also reviewed. Rossiter and LaVaque compared the effects of 20 sessions of EEG biofeedback with the effects of stimulant medication in 46 subjects with AD/HD ages 8-21 years, who were divided into two matched groups. In this study, patients receiving EEG biofeedback demonstrated significant improvement in several psychometric test scores. There was no significant difference in response rates for patients treated with EEG biofeedback (83%) and medication (87%).

Linden et al studied 18 children (ages 5-15 years) with AD/HD who were randomly assigned to a “waiting list” or an EEG biofeedback treatment group. Patients treated with EEG biofeedback demonstrated a significant increase in IQ (9 points) when compared to the control group, and significantly reduced inattentive behaviors, as rated by parents.

Another randomized, waiting-list trial, involving 16 children (ages 8-10 years) with AD/HD, was conducted by Carmody et al. Patients treated with EEG biofeedback exhibited reduced impulsivity on psychometric testing and were rated more attentive by their teachers. However, follow-up QEEG testing did not demonstrate consistent patterns of electrophysiological improvement after EEG biofeedback.

The largest published controlled trial of EEG biofeedback for AD/HD was conducted by Monastra et al. A group of 100 patients (ages 6-19 years) was divided into two groups; one received methylphenidate and the other received methylphenidate plus EEG biofeedback. After one year of therapy, post-treatment assessments were conducted while patients continued to take methylphenidate, and then after a one-week medication washout. The EEG biofeedback-plus-medication group received an average of 43 sessions, which were designed to reduce cortical slowing to within one standard deviation of age peers. Statistical analysis demonstrated an independent beneficial effect of EEG biofeedback, with greater improvement in attention and less hyperactive behavior, reported by parents and teachers, in patients treated with both methylphenidate and EEG biofeedback. After medication washout, sustained improvement, as reported by parents and teachers, was seen only in the group that had been treated with methylphenidate and EEG biofeedback. Children whose parents followed the strategies taught in a concurrent parenting program had fewer attentional and behavioral problems at home, regardless of which treatment they received.

Fuchs et al compared EEG biofeedback with stimulant medication in 34 children (ages 8-12 years) with AD/HD. Treatment assignment was based on parental preference, and the two treatment groups were similar in pre-treatment measures of intelligence and severity of AD/HD. The EEG biofeedback group received 36 sessions over 12 weeks. Significant improvement in psychometric and behavioral test results, and in parent and teacher reports, were found in both treatment groups. The authors concluded that EEG biofeedback was efficient in improving some behavioral concomitants of AD/HD in children whose parents favored a nonpharmacological treatment.

A recent paper by Levesque et al evaluated the impact of EEG biofeedback on brain function in AD/HD by using brain functional magnetic resonance imaging (fMRI) in conjunction with psychometric tests. After EEG biofeedback therapy, children with AD/HD exhibited improved attentional performance, as well as distinctive activation of the right anterior cingulate cortex on fMRI, which were not observed in untreated control subjects.
In summary, controlled studies demonstrate that the efficacies of EEG biofeedback and stimulant medication are comparable in the treatment of AD/HD.

**Protocol Choice**
EEG biofeedback is a relatively new treatment modality, and the novice is confronted with a wide array of protocols to choose from. Although newer protocols may offer comparable or even better outcomes than the approaches used in the clinical trials described in the previous section, evidence to support the newer protocols tends to be more anecdotal.

**Quantitative Electroencephalographic Analysis**
One important question is whether pre-treatment QEEG is necessary and beneficial in guiding EEG biofeedback treatment. Of the five controlled-group studies discussed in the previous section, only one used QEEG improvement as a treatment endpoint. One other study found no consistent change in QEEG after EEG biofeedback, and the remaining three studies did not report QEEG data. Since the studies all found significant improvement for AD/HD with EEG biofeedback, independent of QEEG use, the case for QEEG is not compelling; furthermore, QEEG is relatively expensive. Avoiding QEEG testing can reduce the cost of EEG biofeedback. On the other hand, some of the leading experts in EEG biofeedback who routinely perform QEEG report excellent treatment outcomes.

**Interhemispheric EEG Biofeedback**
Interhemispheric EEG biofeedback was developed by the Othmers at The EEG Institute, based on re-evaluation of the original methods used in the controlled studies described earlier. In their clinical work using single-channel EEG biofeedback, the most common EEG disturbances encountered in patients were left hemispheric hypoarousal and right hemispheric hyperarousal. Single-channel EEG biofeedback aims to increase EEG frequencies in areas of hypoarousal and/or decrease them in areas of hyperarousal. The Othmers developed a new paradigm in which instability of state, as well as hypo- or hyperarousal, is addressed. Interhemispheric EEG biofeedback can be employed to simultaneously encourage increased left hemispheric frequency and decreased right hemispheric frequency, while also supporting left hemisphere-right hemisphere integration. Interhemispheric EEG biofeedback has become the Othmers’ method of choice for improving functional brain stability. Case study data indicate interhemispheric EEG biofeedback is comparable to single-channel EEG biofeedback in efficacy for treatment of AD/HD.

**Low Energy Neurofeedback**
A low energy neurofeedback system is another EEG biofeedback variation that employs direct weak electromagnetic stimulation at the sensor sites, instead of the customary visual and auditory feedback employed in other EEG biofeedback modes. At this time, no published research studies are available to evaluate this approach for treatment of AD/HD.

**Hemoencephalography**
Hemoencephalography, the newest outgrowth of neurofeedback, employs near-infrared sensors to monitor cerebral blood flow and guide feedback to the patient. Prefrontal sensor placement sites have been used in limited published studies of hemoencephalography to treat AD/HD. Because hemoencephalography has a direct impact on cerebral blood flow, it is contraindicated in patients with cerebrovascular disorders.

**Choosing a Protocol and Practitioner**
The data available do not allow a head-to-head comparison of standard single-channel EEG biofeedback and newer protocols. Selection of a neurofeedback practitioner should be based on level of experience and training, accreditation, the fraction of the therapist’s practice devoted to neurofeedback, positive reports from clients, and the therapist’s specific experience in treating AD/HD.

**Contraindications**
Case and controlled group studies did not include patients under age six years, or subjects with developmental delay or other significant medical, neurological, or psychiatric disease. Patients from families with significant marital discord that could interfere with participation in the treatment process were also excluded from the studies.
Adverse Effects
There is a potential for increased irritability, moodiness, and hyperactivity when stimulant medication and EEG biofeedback are combined. This can occur along with improvement in cortical activation, indicating the stimulant dosage might need to be reduced or eliminated. Occasionally, patients report transitory headaches, tiredness, and/or dizziness after treatment. The original work by Sterman clearly demonstrated that EEG biofeedback has the potential to decrease or increase seizure threshold, depending on the frequencies and sensor locations used. Patients with a history of epilepsy should only receive neurofeedback from practitioners who are well versed in EEG biofeedback therapy for seizure disorders.

Potential Synergies
EEG biofeedback therapy for AD/HD results in significant improvement in cognitive functioning for 75-85 percent of patients. It is possible faster and better outcomes might be achieved by combining other alternative therapies with EEG biofeedback. According to Schnoll et al, dietary modification plays a major part in the treatment of AD/HD and should be considered as part of the overall treatment protocol when EEG biofeedback therapy is employed. They also reviewed research demonstrating that patients with AD/HD and food sensitivities have changes in brain electrical activity after exposure to offending foods, suggesting that removing foods the patient is sensitive to could accelerate response to EEG biofeedback.

Another example of a potential synergy between EEG biofeedback and alternative therapies concerns omega-3 fatty acid supplements, which are incorporated into neuronal membranes and have stabilizing effects on mood and other aspects of mental functioning. Although it is possible omega-3 fatty acid therapy could "prime" the brain to respond to EEG biofeedback-augmented stabilization, no clinical research has been conducted to confirm such a hypothesis. Anecdotal evidence, however, from practitioners who prescribe dietary modification and nutritional supplements along with EEG biofeedback is impressive. Further research on combined approaches is warranted.

Use of EEG Biofeedback for other Disorders
Experienced practitioners treat a range of neuropsychiatric problems with EEG biofeedback. The strongest evidence-based justification for EEG biofeedback therapy exists for AD/HD and epilepsy. A growing body of evidence supports the use of EEG biofeedback in the treatment of mood disorders. A number of other conditions have been reported to respond to EEG biofeedback, including migraine, fibromyalgia, chemical dependency, and syndromes secondary to traumatic brain injury. EEG biofeedback protocols have also been developed to improve "peak performance" in healthy individuals. For example, conservatory students experienced improvements in artistic aspects of music performance equivalent to two class grades after EEG biofeedback.

Conclusions
EEG biofeedback is a well-established, non-drug treatment modality for AD/HD, with proven efficacy and minimal adverse effects. In seeking to engage neuronal plasticity for patient benefit, EEG biofeedback offers an optimistic, non-reductionist approach to neuropsychiatric problems. Although integrative treatment of AD/HD, including dietary modification, nutritional supplements, and EEG biofeedback, may offer patients the best chance for a favorable outcome, research on combining these therapies has not yet been conducted.

Disclaimer
This paper reflects the author’s opinions, which are not endorsed by the Forensic Laboratory Services Bureau of the Washington State Patrol.

References


21. Bradford Weeks, MD; Weeks Clinic, Clinton, WA – personal communication.


General References


