

# Electroencephalographic biofeedback (neurotherapy) as a treatment for attention deficit hyperactivity disorder: rationale and empirical foundation

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Attention deficit hyperactivity disorder (ADHD) is the most commonly diagnosed psychiatric disorder of childhood and adolescence, with prevalence rates ranging from 3% to 7% in the United States [1–4] and 2% to 29% in international studies [5–9]. Although characterized by enduring symptoms of inattention alone or in combination with evidence of hyperactivity and impulsivity, the range of comorbid psychiatric disorder, health-related problems, and functional impairments associated with ADHD is extensive. As reported in the National Institutes of Health Consensus Statement on the Diagnosis and Treatment of ADHD [10], the impact of this disorder is profound, and a considerable share of health care resources is dedicated for the treatment of these individuals.

Few patients with ADHD require treatment for this condition alone [11,12]. Comorbidity rates of 30% to 50% are reported for ADHD and conduct disorders, 35% to 75% for oppositional defiant disorders, 25% to 50% for anxiety disorders, and 15% to 75% for mood disorders [11–14]. Estimates of the rate of comorbid learning disabilities range from 8% to 39% (reading), 12% to 30% (mathematics), 12% to 27% (spelling), and 30% to 50% (written expression) [11,15,16]. Social relations also are highly problematic for children with ADHD, as parents, teachers, and peers consistently indicate that such children are more aggressive,

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disruptive, intrusive, controlling, and less able to communicate effectively than expected for their age [17–20].

Given this combination of attention, psychiatric, learning, and social problems, it is not surprising that the developmental course of this disorder is marked by lower average marks, more expulsions from school, a higher rate of retention in a grade, and fewer completed grades [21–23]. Other functional and health risks associated with ADHD include higher rates of criminal behavior, an employment history characterized by more jobs, more frequent “layoffs,” and an overall job status that is lower than that of peers with similar intelligence [21,24]. The risk for nicotine and other psychoactive substance abuse also is significantly greater for patients with ADHD [25,26], and rate of automobile accidents is elevated in the population [21,27].

Although once believed to be a condition that was outgrown by the adolescent years, longitudinal and retrospective studies of ADHD have not supported such a position. An overview of studies published during the past 25 years indicates that 30% to 80% of children diagnosed with ADHD continued to display a significant number of symptoms of inattention, impulsivity, and hyperactivity (and associated functional impairments) into adolescence and adulthood [14,25,26,28,29]. Because of the severity and enduring nature of the functional impairments associated with ADHD, thousands of scientific studies have focused on understanding the causes of ADHD and procedures for diagnosing and effectively treating this disorder. The preponderance of scientific findings supports a model that defines ADHD as an inherited disorder whose core symptoms are founded in neuroanatomic, neurochemical, and neurophysiologic anomalies of the brain.

## **Genetics and attention deficit hyperactivity disorder**

The results of twin and familial studies support the hypothesis that ADHD is a psychiatric condition with a high degree of inheritability. In twin studies, heritability indices of approximately 0.75 for ADHD are reported [30–32]. Family inheritance patterns also reveal elevated incidence rates. In families in which a child has been diagnosed with ADHD, more than 30% of siblings also have ADHD [33–35]. Other reports indicate that more than 50% percent of families that include a parent diagnosed with ADHD also have at least one child with this disorder [36].

Scientific investigations directed at identifying the genes responsible for such patterns of inheritance have focused primarily on dopaminergic alleles. This emphasis seems directly related to recent advances in molecular biology, which have demonstrated that stimulant medications primarily produce their clinical effects by occupying dopamine reuptake transporters, thereby increasing the availability of dopamine at the synaptic level [37,38]. Genetic studies of dopamine receptor and reuptake alleles have found increased incidence of anomalies of the dopamine receptor-4 gene [39,40], the dopamine receptor-2 gene [41], and the DAT1 gene [42,43]. The hypothesis derived from these studies was that such

anomalies would cause anatomic changes in the size of dopamine-rich brain regions and associated areas in patients diagnosed with ADHD.

### **Neuroimaging studies and attention deficit hyperactivity disorder**

As reported in several MRI and functional MRI studies [44–51], significant differences in the size and symmetry of brain regions essential for attention and behavioral disinhibition are evident in patients diagnosed with ADHD when compared with healthy age peers. Specifically, these studies have noted significant differences in the regions involved in behavioral inhibition (eg, basal ganglia and cerebellum) and attentional functions (eg, anterior cingulate gyrus, right frontal region, anterior and posterior regions of the corpus callosum, and caudate).

Although certain inconsistencies have been reported in the literature with respect to the specific region of the corpus callosum (rostrum versus splenium) and the hemisphere containing the “smaller” caudate (left versus right), there is substantial convergence of findings with respect to neuroanatomic differences in this patient population [52]. As summarized in their review of the literature, Giedd et al [53] asserted that volumetric studies “consistently point to involvement of the frontal lobes, basal ganglia, corpus callosum and cerebellum” in ADHD.

Similarly, the results of single photon emission tomography (SPECT) and positron emission tomography (PET) also have implicated the involvement of the right frontal basal ganglia circuitry and the moderating influence of the cerebellum in ADHD. Evidence of hypoperfusion in the prefrontal cortex and basal ganglia initially was reported during PET scans [54,55]. Subsequently, decreased cerebral blood flow in the right lateral prefrontal cortex, the right middle temporal cortex, and the orbital and cerebellar cortices (bilaterally) were reported on SPECT [56,57]. Consistent with SPECT findings, Zametkin et al [58] and Ernst et al [59] reported decreased glucose metabolism (an indicator of cortical deactivation) in PET studies that examined response to an intellectual challenge in adults and adolescents with ADHD.

### **Implications for the treatment of attention deficit hyperactivity disorder**

Collectively, neuroimaging (eg, MRI, functional MRI, PET, SPECT) studies suggest that ADHD is a result of underarousal in the regions of the brain responsible for sustained attention and behavioral planning and motor control. Such a model would predict that treatments intended to promote increased neuronal activity in these regions would be beneficial in the treatment of the core symptoms of this disorder. Consistent with this model, PET studies indicate that stimulants, such as methylphenidate, bind dopamine transporters, block reuptake

of the neurotransmitter dopamine, and increase neurotransmitter availability at the synaptic site, thereby increasing cortical arousal [37,38,60].

The effects of this process have been documented in multiple studies that have demonstrated increased cerebral blood flow to the prefrontal striatal region after administration of methylphenidate [57,61,62]. The impact of this increased cortical activation on attention and behavioral control has been examined in numerous randomized, controlled trials that have demonstrated the clinical efficacy of stimulants in the treatment of the core symptoms of ADHD [63]. As summarized by Jensen [64], “controlled studies of stimulants have shown their effect on reducing interrupting in class, reducing task-irrelevant activity in school, improving performance on spelling and arithmetic tasks, improving sustained attention and compliance, reducing overt aggression, reducing covert aggression, improving peer nominations, improving short-term memory, improving attention during baseball, and improving parent-child interactions.” As a result, stimulant medications have emerged as the primary type of treatment for the core symptoms of ADHD.

Despite the robust clinical effect of stimulant medications on the core symptoms of ADHD, however, there is mounting scientific evidence that a substantial percentage of patients do not respond to stimulants and that medication side effects can preclude use of clinically effective doses. Greenhill et al [63] reported in their review of 161 randomized controlled trials that 25% to 35% of the patients with ADHD did not demonstrate significant reduction in symptoms of hyperactivity and impulsivity after stimulant therapy. Other researchers [65] estimated that the percentage of patients who do not demonstrate significant improvements in attention may be somewhat higher (40%–50%). Severe side effects (sufficient to require discontinuation of medication) also have been reported in approximately 4% to 10% of patients [63], including sleep onset insomnia, loss of appetite, stomachache, headache, “jitteriness,” and increased irritability.

Because of the substantial number of patients who do not respond to or cannot tolerate stimulants, two types of nonpharmacologic treatments for ADHD have been investigated (ie, psychosocial treatments and electroencephalographic [EEG] biofeedback). Among the “psychosocial” interventions that have been evaluated, parental training [66], behavioral modification in classroom settings [67], and intensive social skills training programs [68] have been demonstrated to be effective in promoting adaptive functioning in school settings, improving behavioral and emotional control at home and school, and reducing oppositionalism [69–71].

As clarified in the report prepared by the research team that conducted the National Institute of Mental Health Multimodal Treatment Study of ADHD [12], however, none of these types of “psychosocial” treatments is considered effective in treating the core symptoms of ADHD in the absence of carefully titrated pharmacologic intervention. Although the combination of medication and intensive behavioral treatment produced improved outcome in patients diagnosed with ADHD with comorbid anxiety and disruptive disorders (relative to patients treated with behavioral or medication therapies alone), there is no indication that

this combined type of treatment was superior to medication management in treating the core symptoms of ADHD. The combined treatment was associated with additional reduction in symptoms of anxiety, depression, oppositionalism, and aggression and improvement in academic functioning, social skills, and parent-child relations.

In contrast, researchers who examined the effects of EEG biofeedback consistently reported significant reduction in the core symptoms of ADHD and improved academic and social functioning [72,73]. As noted in the National Institutes of Health Consensus Statement on the Diagnosis and Treatment of ADHD [10], EEG biofeedback is a nonpharmacologic treatment associated with clinical and functional improvements in patients with ADHD in case and controlled group studies. The purpose of this article is to summarize the neuro-anatomic and neurophysiologic foundations of EEG biofeedback, describe the procedures used in this type of treatment, evaluate the effectiveness of EEG biofeedback in treating the core symptoms of ADHD using guidelines for efficacy established by the Association for Applied Psychophysiology and Biofeedback and the International Society for Neuronal Regulation [74], and discuss the implications of current research for clinical practice.

### **Neuroimaging, quantitative electroencephalography, and attention deficit hyperactivity disorder**

The preponderance of data derived from functional MRI, MRI, PET, and SPECT studies supports a cortical “hypoarousal” model for ADHD. Collectively, these neuroimaging studies have revealed slowing of cortical blood flow and glucose metabolism, primarily in prefrontal and frontal cortical regions. MRI and functional MRI studies also have revealed anatomic differences between patients with ADHD and healthy peers in brain structures involved in attention, behavioral control, and judgment (eg, frontal lobes, basal ganglia, corpus callosum, and cerebellum).

Consistent with the results of neuroimaging studies, quantitative EEG (qEEG) research using power spectral analysis typically has revealed evidence of underactivity in patients with ADHD [75–80]. In studies that use power spectral analysis, EEG recordings are obtained during resting and academic tasks (eg, reading, listening, or writing). Multiple short periods of digitized EEG (90–180 seconds) are collected from scalp electrodes and are subjected to a fast Fourier transformation algorithm [81]. Quantitative analysis of various characteristics of the electrical output is conducted, including analysis of wave speed or frequency, assessment of absolute and relative power in specific frequency bands (eg, delta: 0.5–3.5 Hz; theta: 4–8 Hz; alpha: 9–11 Hz; sensorimotor rhythm [SMR]: 12–15 Hz; beta 1: 16–20 Hz; beta 2: 22–30 Hz), comparisons of power in slow (eg, 4–8 Hz) versus fast (eg, 13–21 Hz) frequency bands (ie, the theta-beta power ratio), and comparisons of the similarity of activity in different cortical regions (coherence).

As noted in PET and SPECT studies, evidence of electrophysiologic slowing has been recorded over frontal and central midline cortical regions in approximately 80% to 90% of patients with ADHD [75,77–79]. This slowing has been reflected in elevated relative theta power, reduced relative alpha and beta power, and elevated theta-alpha and theta-beta ratios, primarily over frontal, frontal-midline, and central-midline cortical regions. Using such qEEG measures as an electrophysiologic test for ADHD, Chabot et al [77] reported a test sensitivity of 94% and test specificity of 88%. Similar sensitivity and specificity levels have been subsequently reported [78,80].

Patients with ADHD who demonstrate such cortical slowing have been shown to respond positively to stimulants, whereas patients with ADHD who do not demonstrate cortical slowing on qEEG examination are typically stimulant non-responders [81,82]. Initial studies of patients diagnosed with other psychiatric (eg, oppositional-defiant disorders, anxiety disorders, and mood disorders) or learning disorders indicate that such patients do not display cortical slowing on qEEG [77,83,84], PET [85,86], or SPECT [87,88].

### **The rationale for electroencephalographic biofeedback for attention deficit hyperactivity disorder**

The rationale for the development of EEG biofeedback for ADHD is derived from neuroimaging studies that have indicated consistently the involvement of the frontal lobes, basal ganglia, corpus callosum, and cerebellum in ADHD [52] and a substantial body of neurophysiologic research [89] that has clarified the relationship between surface EEG recordings and the underlying thalamocortical mechanisms that are responsible for its rhythms and frequency modulations. As reviewed by Serman [89], variations in alertness and behavioral control seem to reflect the activity of specific thalamocortical generator mechanisms.

When a person is in an inattentive, unfocused state, evidence of slow EEG frequencies (3.5–8 Hz or theta) is predominant over the prefrontal and frontal cortex and at certain midline locations (eg, the vertex or Cz). In relaxed, wakeful states, alpha rhythms (9–11 Hz) begin to be noted in these same locations. As an individual shifts into a state of increased awareness and is preparing to engage in a planned or purposeful action, evidence of increased amplitude of the SMR (12–15 Hz) is apparent over the Rolandic (motor) cortex. Finally, during tasks that require focused attention and sustained mental effort, beta (16 to more than 20 Hz) is noted over prefrontal, frontal, and central midline regions. Because underarousal in these regions has been noted on PET and SPECT in patients with ADHD and such underarousal is evident on qEEG, neurophysiologists have examined whether laboratory animals and humans could learn to control the production of specific frequencies and if such self-regulation would promote the development of improved attention and behavioral control.

## **Electroencephalographic biofeedback treatment protocols for attention deficit hyperactivity disorder**

EEG biofeedback treatments for ADHD are founded on the groundbreaking research conducted by Sterman [90–93] and Lubar [94,95]. Initially, Sterman and colleagues conducted a methodical examination of EEG patterns associated with behavioral inhibition and identified the SMR over the Rolandic cortex. Subsequently, they demonstrated that laboratory animals could be trained to increase production of this rhythm and that patients with seizure disorders could develop improved control over epileptiform activity by learning self-regulation of the SMR [96].

Based on Sterman's earlier conditioning studies with cats, Lubar and his students at the University of Tennessee began to study children who demonstrated symptoms of impaired attention and lack of behavioral control. Initially, Lubar and Shouse [94] demonstrated improved behavioral control in a hyperactive child who had learned to reduce theta and increase production of SMR. Subsequently, Lubar and Lubar [95] reported that children diagnosed with an attention deficit disorder demonstrated improved attention and behavioral control after being trained to increase production of EEG activity in a fast frequency range (beta: 16–20 Hz) while learning to suppress activity at slower speeds (theta: 4–8 Hz).

These two primary training approaches (theta suppression/SMR enhancement; theta suppression/beta enhancement) provide the foundation for each of the protocols that have been examined in the controlled group studies of EEG biofeedback for ADHD. Although recent qEEG reports of a neurophysiologic subtype of ADHD, which is characterized by excessive beta activity over frontal regions, have been published [82,97] and protocols to suppress such excessive beta activity have been developed [98], the primary emphasis of EEG biofeedback research has been placed on examining treatment response in patients with ADHD who demonstrate cortical slowing on qEEG examination. The following summaries provide a description of the three EEG biofeedback protocols that have been investigated in controlled group studies to date.

### **Protocol 1: sensorimotor rhythm enhancement/theta suppression**

This type of EEG biofeedback is applied in the treatment of patients with ADHD who present with primary symptoms of hyperactivity and impulsivity. In this protocol, patients are encouraged to develop increased behavioral control by learning to increase their production of the SMR (12–15 Hz) over one of two sites (C3 or C4) positioned over the motor cortex while simultaneously suppressing the production of theta (4–7 or 4–8 Hz) activity. EEG recordings are obtained from one active site, referenced to linked earlobes, with a sampling rate of at least 128 Hz. Visual (eg, counter display, movement of puzzle pieces, graphic designs, or animated figures) and auditory (tones) feedback is provided



based on patient success in controlling microvolts of theta or SMR or the percentage of time that theta is below or SMR is above pretreatment thresholds. Typically, patients are informed via tone and visual feedback when they produce 0.5 seconds of desired EEG activity. Rossiter and LaVaque [99] used this protocol in the first published controlled group study of EEG biofeedback for ADHD.

### **Protocol 2: theta suppression/beta 1 enhancement**

This protocol has been examined in three of the four controlled group studies published to date [99–101]. In studies that use this procedure, patients are reinforced for increasing production of beta 1 activity (16–20 Hz) while suppressing theta activity (4–8 Hz). Recordings are obtained at Cz (central, midline) with linked ear references, at FCz-PCz (midline frontal, midline parietal) with single ear reference, or at Cz-Pz (midline, central, midline parietal) with ear reference. A variation of this protocol also has been reported in the treatment of patients diagnosed with ADHD, predominately inattentive type [98]. In this training protocol, theta suppression and beta enhancement are reinforced at C3. Sampling rate is at least 128 Hz. Feedback is provided contingent on a patient's ability to control amplitude of microvolts of beta and theta.

### **Protocol 3: sensorimotor enhancement/beta 2 suppression**

In this protocol, patients who are diagnosed with ADHD, predominately hyperactive/impulsive type, are trained to increase SMR (12–15 Hz) while suppressing beta 2 activity (22–30 Hz) [98]. Recordings are obtained at C4 with linked ear reference. Sampling rate is at least 128 Hz. In patients who are diagnosed with ADHD, combined type, this protocol is used during half of each session. During the other portion of each training session, protocol 1 is used (training site: C3). Reinforcement is contingent on patient ability to control microvolts of theta, SMR, beta 1, and beta 2.

### **Review of the scientific literature: case studies**

Numerous single case [94,102] and multiple case studies [103,104] have reported clinical benefits in patients diagnosed with ADHD [72,73]. In these studies, training has followed protocol 1 or 2, with minor variation in the definition of the SMR, theta or beta bands. The initial case study [94] described the results of training an 11-year-old boy who was diagnosed with hyperkinesis to increase production of SMR and reduce theta activity. This study was the first to demonstrate an electrophysiologic training effect in the laboratory that was associated with a decrease in off-task and oppositional behaviors and increased cooperation and completion of school work in the classroom.



By using an ABA treatment reversal design, Lubar and Shouse [94] showed that these changes in classroom functioning were associated with the type of training protocol. When the child received reinforcement for producing increased SMR and decreasing slow cortical activity (theta) attention, cooperation and completion of tasks increased. When the child received reinforcement for decreasing SMR and increasing slow cortical activity, however, significant deterioration of attention, compliance, and task completion occurred.

In the years after this initial paper, which described the amelioration of core symptoms of ADHD by training a child to regulate cortical activity, various other case reports have been written. The most extensive of these case studies was the study by Thompson and Thompson of 111 patients diagnosed with attention deficit disorder (with and without hyperactivity) [103] and Kaiser and Othmer's investigation of the effects of EEG biofeedback in the treatment of 1089 patients (186 diagnosed with ADHD) [104]. In each of these studies, participants were trained to suppress slow cortical activity while increasing production of the SMR or beta 1 (16–20 Hz).

In these studies, various improvements were described after EEG biofeedback. Clinical gains included significantly improved scores on continuous performance tests and behavioral ratings of attention and behavioral control. Thompson and Thompson [103] also reported an average increase of 12 points on the Wechsler Full Scale Intelligence Quotient; Kaiser and Othmer [104] noted an average increase in intelligence quotient of more than 10 points.

Another noteworthy multiple-case study was reported in the treatment of eight children diagnosed with ADHD who were treated with EEG biofeedback within a school setting [105]. As in prior clinical case studies, training protocols designed to suppress theta and enhance beta or SMR were used. To control for maturational effects, a wait list control design was used (four children were treated during the spring; four in the fall). To promote increased acceptance of the program within the school context, a group of eight age-matched peers (without ADHD) also received EEG biofeedback. All eight participants with ADHD completed the training. The number of sessions ranged from 36 to 48.

The results of this school-based EEG biofeedback study indicated improvement on a behavioral rating scale [106] and a continuous performance test [107]. On the behavioral assessment, a significant reduction in the frequency of inattentive behaviors was noted between ratings obtained before and after EEG biofeedback. No significant decrease in the rate of hyperactive or impulsive behaviors was noted. Analysis of the continuous performance test results revealed a significant reduction in the number of errors of commission (ie, impulsive responding to nontarget stimuli) in participants who had been diagnosed with ADHD. No consistent pattern of changes in amplitude of theta, beta, or SMR activity was noted.

Published follow-up case studies that examined the long-term benefits of EEG biofeedback are limited to a single case report [108] and a retrospective study of 52 patients who completed EEG biofeedback treatment for ADHD over a 10-year period [72]. In the single-case study, Tansey [108] reported that a child with

ADHD who had been treated during the fourth grade for hyperactivity was able to maintain sustained control over hyperactive symptoms for 10 years. Lubar [72] subsequently reported a broader range of clinical gains based on structured, follow-up telephone interviews conducted by an independent surveyor.

Using a rating scale that ranged from “no change” to “very much improved,” parents and former patients (who had reached adulthood) rated the degree of change after EEG biofeedback as “very much improved” in the following behaviors: fidgeting, being demanding or easily frustrated, restlessness or over-activity, excitability, inattention, failure to finish things, temper outbursts, and moody behavior. Such findings prompted Lubar [72] to comment, “Perhaps the most important finding in our retrospective study was that the greatest improvements occurred in the areas with which parents are most concerned—that is, behavior, attitude, homework and grades.” He also reported that “virtually all” of the raters attributed the gains to EEG biofeedback.

### **Critique of case studies**

Guidelines for determining the efficacy of treatments (including EEG biofeedback) have been published by the American Psychological Association [109], the American Academy of Neurology/American Clinical Neurophysiology Society [110], the Association for Applied Psychophysiology and Biofeedback, and the International Society for Neuronal Regulation [74]. As reflected in each of these position papers, information derived from case studies is not considered sufficient to demonstrate the efficacy of any treatment.

Despite such limitations, however, case studies serve an important role in the development of effective new treatments. Through such studies, clinical researchers are able to identify potentially beneficial interventions strategies and any patient health risks. Over the past 25 years, several training protocols have been developed and examined in case studies using various outcome measures to determine treatment effects. In these studies, patients diagnosed with ADHD have demonstrated improvement on behavioral ratings, continuous performance tests, and tests of intelligence. No significant adverse effects were reported in the case studies, although deterioration of clinical benefits and relapse has been reported in case studies in which training was discontinued before completion of treatment.

The primary limitation of case studies is their inability to clarify the degree to which treatment effects are caused by factors other than the specific treatment under investigation. Such nonspecific factors include therapist characteristics (eg, degree of compassion, understanding, displayed knowledge, or confidence), patient characteristics (eg, patient intelligence and capacity to learn new skills, severity of the disorder, degree of hope or expectancy, variations in patient motivation), and treatment characteristics (eg, administration of a pill, use of computerized EEG equipment), patient exposure to therapeutic experiences other

than the investigational treatment (eg, counseling, tutoring, variations in parenting style), and maturation.

The importance of monitoring and controlling the effects of such factors is illustrated in two controlled case studies, which highlight the moderating role of motivation and capacity to learn new skills in efficacy studies. In an EEG biofeedback study that examined the effects of theta suppression/beta enhancement on the core symptoms of ADHD in 17 children Lubar et al [76] noted that two groups emerged. One group of children ( $n = 6$ ) did not demonstrate a training effect on any of the EEG measures obtained during training. Another group ( $n = 11$ ) seemed to learn to increase cortical activation (by lowering the theta-beta power ratio).

Although these researchers did not assess directly the relationship between the degree of clinical response and the ability to increase cortical activation, their article illustrates the importance of assessing indicators of neurophysiologic learning in any evaluation of the efficacy of EEG biofeedback. If the goal of EEG biofeedback is to promote self-regulation of attention and behavior, then examination of the degree to which an individual is able to learn how to regulate cortical activity associated with sustained clinical improvements is of primary importance in evaluating the efficacy of this type of treatment.

A more recent study of EEG biofeedback, conducted by Heywood and Beale [111], provides further support for the importance of examining other nonspecific factors, such as failure to complete treatment, in assessing efficacy. In this study of seven children diagnosed with ADHD, bona fide biofeedback training protocols (eg, increase SMR, decrease theta and beta-2) and a noncontingent placebo condition were used. In the placebo training, a series of randomly determined bandwidths was reinforced or inhibited (eg, 12–29 Hz, 2–6 Hz, 2–18 Hz). To control for maturation and treatment sequence effects, a randomized design with an embedded ABAB reversal was used. Children were not informed of treatment condition.

As in previous case studies, the investigators initially analyzed the results of children who had completed the four treatment phases (five children). They found a significant positive effect of EEG biofeedback on neurophysiologic and behavioral measures of attention in treatment completers. When they included the data from the two children who discontinued treatment (and controlled for overall trend), however, the overall size of treatment gains diminished. Similar to other case studies, conclusions about the efficacy of EEG biofeedback cannot be drawn from this study. This experiment illustrated the importance of reporting the results of nonresponders and controlling for trend effects in studies that examine the efficacy of EEG biofeedback.

In general, researchers who have used case study designs to evaluate the effects of EEG biofeedback have reported significant improvement in the core symptoms of ADHD. Certain of these case reports also have suggested that more pervasive improvement in functioning (eg, improved ability to regulate affect, rate of completing school work, enhanced family and social relationships) occurs in patients treated with EEG biofeedback [72]. Like any treatment, however, it is

clear that a certain percentage of patients will not improve in response to this treatment. In reported case studies, that percentage is comparable to the rate of nonresponse to stimulant medications, which ranges from 29% [111] to 35% [76]. Heywood and Beale's findings [111] underscore the importance of evaluating and controlling expectancy and maturation in efficacy studies of EEG biofeedback.

### **Review of the scientific literature: controlled group studies**

A review of the literature indicates that four controlled group studies of the effects of EEG biofeedback in treating ADHD have been published in peer-reviewed journals. The research designs used in these studies attempted to control for maturational and other nonspecific factors (eg, age, intelligence, symptom severity before initiating treatment). Three of the four studies also compared the effects of EEG biofeedback with a bona fide treatment for ADHD that has been classified as efficacious (ie, stimulant medication).

The initial controlled group study was conducted by Rossiter and LaVaque [99]. In this study, the clinical effects of EEG biofeedback were compared with those obtained by children treated with stimulant medication (either methylphenidate or dextroamphetamine). Forty-six patients (aged 8–21 years) who were diagnosed with ADHD participated in the study. Two groups of 23 patients received the treatment of their (or a parent's) choice (either stimulant medication or 20 sessions of EEG biofeedback). EEG biofeedback protocols 1 and 2 were used in this study. None of the participants in either group discontinued treatment during the study.

Patients who participated in EEG biofeedback were seen three to five times per week (45- to 50-minute sessions that included 30 minutes of EEG biofeedback). Participants who were treated with stimulant medication participated in an assessment process to identify an effective medication dosage, including follow-up interviews and readministration of the Test of Variables of Attention (TOVA). Other counseling and support services were provided to participants in both groups (eg, school behavior modification programs, parental counseling in the use of reinforcement principles at home) but were not systematically analyzed in this study. The researchers did note that the two groups did not differ in the frequency of parents who received behavior management training.

The results of this study indicated significant improvements ( $P < 0.05$ ) on the TOVA and several subscales of the Behavioral Assessment System for Children [112] (eg, hyperactivity, attention problems, and externalizing behaviors). Comparison with a bona fide treatment for ADHD (stimulant medication) revealed no difference in the effects of these treatments after 20 sessions. Similarly, there was no significant difference in the percentage of patients who demonstrated clinical improvement with EEG biofeedback (83%) and stimulant medication (87%).

Linden et al [100] reported on the second controlled group study. In their study, 18 children (aged 5–15 years) who had been diagnosed with ADHD were

randomly assigned to either a waiting list condition (and received no psychological treatment or medication) or EEG biofeedback (protocol 2). Each group was composed of 9 children, 6 of whom were diagnosed with ADHD and 3 of whom were diagnosed with ADHD in combination with a learning disorder. Power analysis conducted before beginning the study indicated sufficient sample size to detect significant group differences. Patients who received EEG biofeedback participated in 40 training sessions (45 minutes each). None of the participants discontinued treatment during the study.

The results of the study revealed that children treated with EEG biofeedback demonstrated significant increases ( $P < 0.05$ ) on measures of intelligence [113] and a reduction in symptoms of inattention on a behavioral rating scale [114]. No adverse effects were reported.

The largest controlled group study reported in the literature was conducted by Monastra et al [101]. In their study, 100 patients (aged 6–19 years) participated in a multimodal treatment program conducted at an outpatient clinic that specialized in the diagnosis and treatment of ADHD. All participants received the following treatments: stimulant medication (dosage titrated based on the results of behavioral measures and the TOVA), a 10-week parenting program [115] with follow-up individualized parental counseling provided as needed, and academic support at school (provided via an individual education plan or 504 accommodation plan). None of the participants discontinued involvement in the study before completion.

In addition to these services, all patients were given the opportunity to receive EEG biofeedback (protocol 2) as part of their treatment program. Fifty-one families chose to include EEG biofeedback in their child's treatment (49 did not). All children were treated with stimulant medication (Ritalin). The average dose of Ritalin administered to the patients of both groups was 25 mg (typically 10 mg after breakfast, 10 mg at lunchtime, and 5 mg after school). The range of dosages was 15 to 45 mg per day for both groups.

Pretreatment screening included tests of intelligence, behavioral rating scales, a continuous performance test, and a qEEG assessment [78]. To be included in the study, participants had to demonstrate evidence of significant levels of cortical slowing on the qEEG measure (ie, attention index that was at least 1.5 standard deviations more than the mean for age peers as reported in Monastra et al [78]). Analysis of the results of prescreening assessment indicated that there were no significant differences on measures of intelligence, symptoms severity, degree of impairment on a continuous performance test (TOVA [107]), or the qEEG index of cortical slowing between participants who selected EEG biofeedback to be part of their treatment and those who did not.

EEG biofeedback was provided on a once per week basis during sessions that lasted 45 to 50 minutes. Biofeedback treatment was continued until the patient could demonstrate a degree of cortical activation on the qEEG measure that was within 1 standard deviation of age peers based on published normative data [78] and could maintain this level of arousal for 45 minutes during three consecutive training sessions. The average number of sessions needed to reach this goal was

43 (range, 34–50 sessions). All of the participants who received EEG biofeedback were able to achieve this goal.

In their published paper [101], this research team reported the results of a posttreatment evaluation that was conducted 1 year after initial evaluation under two conditions. First, patients were evaluated using behavioral ratings (parent and teacher), the TOVA, and a qEEG scanning process [78] while they continued to be treated with stimulant medication. Subsequent to this assessment session, medication was discontinued for 1 week. After this 1-week medication washout, participants were reevaluated. All of the participants remained in the study for the reassessment phase.

The results of this study supported the beneficial effects of stimulant medication and EEG biofeedback and indicated that parenting style was a moderating factor in both treatments. The biofeedback and the non-biofeedback groups demonstrated significant improvements on behavioral ratings and the TOVA when tested while using medication. After the medication wash-out period, relapse was noted on behavioral and CPT measures in each of the participants who had not received EEG biofeedback. Sustained improvement on the qEEG measure was not evident on the qEEG measure once stimulant medication was discontinued in patients who had not received EEG biofeedback.

Patients who received EEG biofeedback as part of their treatment demonstrated significant improvements on the qEEG measure of cortical arousal, behavioral ratings, and the TOVA that were sustained despite a week-long medication-free period ( $P < 0.01$ ). The moderating influence of parenting style also was noted by these researchers, because parents who systematically used the strategies taught in the parenting program had children who displayed fewer attentional and behavioral control problems at home [115].

To evaluate the persistence of the clinical gains associated with EEG biofeedback, Monastra and Monastra [84] conducted a systematic two-year follow-up study of these patients. Eighty-six of the participants of their initial study were re-evaluated 6, 12, and 24 months after the conclusion of their first year of treatment. Forty-three patients who had received the comprehensive clinical care program (ie, medication, parenting classes, school consultation) participated. Similarly, 43 patients whose treatment also included EEG biofeedback were re-evaluated. As in the initial study [101], follow-up assessments (eg, behavioral ratings, TOVA, qEEG scan) were conducted while taking stimulants and after a 1-week medication washout period.

The primary findings of this 2-year follow-up study were as follows:

1. There was no indication that the use of stimulant medication yielded any enduring benefits after 3 years (total) of pharmacologic treatment. Although patients who had never been treated with EEG biofeedback continued to demonstrate positive response on behavioral ratings, the TOVA, and the qEEG when tested with medication, relapse occurred in each of these participants when tested without medication 12, 18, 24, and 36 months after initial evaluation and treatment.



2. Patients whose treatment included EEG biofeedback continued to demonstrate significantly improved levels of cortical activation on the qEEG measure and sustained gains on the TOVA and behavioral ratings throughout the 3-year period, even when medications were withdrawn.
3. Thirty-four (80%) of the patients whose treatment included EEG biofeedback were able to decrease daily dosage of stimulant by at least 50%. By contrast, none of the patients who did not receive EEG biofeedback was able to reduce dosage (85% increased dose).
4. Parents who rated themselves as nonsystematic reinforcers of appropriate behaviors at the conclusion of the first year of treatment varied in their eventual response to our parenting program. The nonsystematic parents whose children participated in EEG biofeedback tended to return for “booster” parenting sessions and reported improved ability to follow through on recommended strategies for addressing child behavioral problems. Examination of their behavioral ratings at 2- and 3-year follow-up assessments revealed significant improvement in their child’s functioning at home.

Conversely, nonsystematic parents, whose children did not participate in EEG biofeedback, rarely returned for booster classes. Their primary reasons for contacting the clinic was to assist in adjustment of medication or revise the child’s IEP or behavioral program at school. Although their children continued to display improved attention and behavioral control at school (while taking medication), no indication of significant functional improvement at home was evident in these families.

The final controlled group study was conducted by Fuchs et al [98]. In this study, the effects of EEG biofeedback again were compared with a bona fide treatment for ADHD (stimulant medication). Thirty-four children (aged 8–12) participated in this study. Twelve children were treated with Ritalin (average dose: 10 mg, three times daily; range: 10–60 mg/day). Twenty-two children participated in EEG biofeedback sessions. Treatment selection was based on parent preference.

Two biofeedback protocols were used. Children diagnosed with an inattentive type of ADHD received training designed to increase production of beta 1 activity (and reduce theta) at C3. Children with the hyperactive or impulsive subtype participated in training intended to increase production of SMR and reduce beta 2 (22–30 Hz) at C4. Patients with the combined type of ADHD received both kinds of training. Sessions were conducted three times per week (30–60 minutes) for a 12-week period.

Pretreatment measures included a test of intelligence (Wechsler Intelligence Scale for Children-Revised [WISC-R]), computerized tests of attention (TOVA [107]; the attention endurance test [116]), and behavioral rating scales [112]. Statistical analysis of pretreatment measures indicated that the groups were comparable before treatment. Posttreatment analysis indicated that EEG biofeedback and Ritalin were associated with significant improvements on computerized



tests of attention and behavioral rating scales. The degree of clinical gains noted after EEG biofeedback was comparable to that associated with stimulant medication. No adverse effects were reported.

### **Critique of controlled group studies**

Collectively, the results of controlled group studies of EEG biofeedback for ADHD have indicated significant gains on measures of intelligence, behavioral rating scales that assess the frequency of the inattention, impulsivity, and hyperactivity, computerized tests of attention, and qEEG measures of cortical arousal. These studies also have compared the treatment outcomes after EEG biofeedback with that noted after stimulant medication. In each of the studies in which a direct comparison was made, response after EEG biofeedback was comparable or exceeded that obtained with stimulant medication. Such consistent reports of significant reduction of inattention, impulsivity, and hyperactivity after use of a nonpharmacologic treatment represent a significant step in the process of identifying effective psychological treatments for ADHD. To date, no other type of psychological treatment has been demonstrated to exert a significant effect on the core symptoms of ADHD.

A current review of the literature reveals that more than 75% of patients treated with EEG biofeedback in controlled group studies responded positively when the treatment was provided in an open trial in which patient choice determined type of treatment (eg, stimulant medication, EEG biofeedback, parental counseling). Follow-up studies also have provided evidence that unlike medication effects (which quickly dissipate when treatment is discontinued), EEG biofeedback seems to exert a far more enduring effect on the core symptoms of ADHD and associated functional problems [71,84]. Despite these positive findings, however, most controlled group studies of EEG biofeedback are potentially confounded by selection bias. Although a patient's right to select type of treatment is an essential aspect of ethical clinical practice, patient selection of treatment type during such open trials may inflate estimates of the effect size of EEG biofeedback.

Consequently, data from controlled group studies in which patients are randomly assigned to EEG biofeedback or comparison groups (eg, stimulant medication, non-contingent biofeedback, or a waiting list control group that has comparable amount of therapist contact) are needed. By controlling for selection bias and other nonspecific factors, clinicians will be in a better position to assess the robustness of EEG biofeedback in comparison to pharmacologic treatments. Such studies are underway at our clinic and other clinical research centers. Initial results from two recently completed RCTs have been presented during the past year [117,118] and merit citation in this review.

The study by deBeus et al [117] is the first randomized controlled trial using a double-blind paradigm that incorporates a "sham" biofeedback treatment. In their study, deBeus et al examined the effects of EEG biofeedback in the

treatment of 52 patients (aged 7–10) with a primary diagnosis ADHD. Fifty percent of the children were diagnosed with an inattentive type of ADHD; 50% with the combined type. Comorbid conditions (oppositional-defiant disorder, conduct disorder, depression, social phobia, and obsessive-compulsive disorder) also were present in 46% of the children.

Participants were randomly assigned to either a bona fide EEG biofeedback treatment (theta suppression, beta or SMR enhancement) or a “sham” biofeedback condition in which rewards (eg, movement on a Sony PlayStation game) were provided randomly. A total of 40 sessions were conducted. Because a Sony PlayStation interface was used, neither the participants nor the therapist was aware of treatment condition (ie, bona fide versus sham biofeedback). Monitoring of EEG activity was conducted in both types of treatment. Twenty-eight of the children (equally represented in the two groups) were being treated with stimulant medications during their participation in treatment.

Because of their interest in monitoring physiologic, behavioral, and functional changes in response to EEG biofeedback, these researchers conducted a power spectral analysis of qEEG data, assessed changes in event-related potentials, and evaluated intelligence, academic achievement, and attention (via continuous performance tests and behavioral ratings). A more extensive behavioral assessment also was conducted using the behavioral assessment system for children [112]. This system provides a comparison of a child with age-matched peers with respect to attention, hyperactivity, aggression, conduct problems, anxiety, depression, somatization, and adaptive skills (eg, leadership, social skills, adaptability, and study skills).

Statistical comparisons between the two groups revealed that the participants who received bona fide EEG biofeedback were rated as demonstrating significantly less hyperactivity at home and school, improved attention at home, less anxiety, less depression and fewer complaints of minor physical problems at home, better adaptability to change, improved ability to work with others, and improved peer interactions, organizational skills, study habits, and a better attitude toward school ( $P < 0.01$ ). On computerized tests of attention, the children who had received bona fide EEG biofeedback demonstrated significantly better scores than age-matched peers diagnosed with ADHD who received sham biofeedback ( $P < 0.01$ ).

Perhaps most significantly, demonstration of improvements in cortical arousal (reduced theta, increased beta or SMR) was evident only in the bona fide biofeedback groups ( $P < 0.01$ ). Within this group, approximately one third of the patients were able to reduce dosage of medication. A total of 6 children (11%) terminated their participation before completion of the study.

Although previous EEG biofeedback studies have described improvements on behavioral rating scales, neuropsychological tests of attention, and qEEG indicators of cortical arousal, none directly examined changes in those brain structures essential for cortical activation. In an effort to investigate changes in the neural substrate of executive function, Beauregard [118] conducted a study in which 20 children were randomly assigned to either a Waiting List control group

or received 40 sessions of EEG biofeedback, using a previously reported training protocol [103]. In addition to obtaining behavioral and neuropsychological measures, functional magnetic resonance imaging (fMRI) was conducted on each participant pre- and posttreatment during completion of the Counting Stroop Task and the Go/NoGo Task.

As anticipated, children who received EEG biofeedback demonstrated significant improvement on behavioral and neuropsychological tests of attention. However, more significantly, those children who had received EEG biofeedback demonstrated significant activation of the right anterior cingulate cortex, the left caudate nucleus, and in the lateral prefrontal cortex (bilaterally) in comparison to pretreatment findings. No such change in activation was noted in the control group. These findings provide one explanation for the enduring nature of clinical gains that have been reported following EEG biofeedback [72,84,108].

### **Assessment of the efficacy of electroencephalographic biofeedback**

Guidelines for the evaluation of the efficacy of treatments have been published by various scientific societies, including the American Psychological Association [109], the Association for Applied Psychophysiology and Biofeedback, the International Society for Neuronal Regulation [74], and the American Academy of Neurology and the American Clinical Neurophysiology Society [110]. Examination of each of these documents reveals a clear emphasis on the importance of supportive data derived from at least two randomized controlled trials conducted by independent research teams in order for a treatment to be considered efficacious. The efficacy criteria published by the Association for Applied Psychophysiology and Biofeedback and International Society for Neuronal Regulation are described later, because these scientific societies are primarily responsible for providing guidance for applied psychophysiology research.

### **Criteria for levels of evidence of efficacy**

*Level 1:* Not empirically supported. This classification is assigned to treatments that have been described and supported only by anecdotal reports or case studies in non-peer-reviewed journals.

*Level 2:* Possibly efficacious. This classification is considered appropriate for treatments that have been investigated in at least one study that had sufficient statistical power and well-identified outcome measures but lacked randomized assignment to a control condition internal to the study.

*Level 3:* Probably efficacious. Treatment approaches that have been evaluated and shown to produce beneficial effects in multiple observational studies, clinical studies, wait list control studies, and within-subject and between-subject replication studies merit this classification.

*Level 4:* Efficacious. To be considered efficacious, a treatment must meet the following criteria:

- In a comparison with a no-treatment control group, alternative treatment group, or sham (placebo) control using randomized assignment, the investigational treatment is shown to be statistically significantly superior to the control condition or the investigational treatment is equivalent to a treatment of established efficacy in a study with sufficient power to detect moderate differences.
- The studies have been conducted with a population treated for a specific problem, from whom inclusion criteria are delineated in a reliable, operationally defined manner.
- The study used valid and clearly specified outcome measures related to the problem being treated.
- The data are subjected to appropriate data analysis.
- The diagnostic and treatment variables and procedures are clearly defined in a manner that permits replication of the study by independent researchers.
- The superiority or equivalence of the investigational treatment have been shown in at least two independent studies [74].

*Level 5:* Efficacious and specific. To meet the criteria for this classification, the treatment must be demonstrated to be statistically superior to a credible sham therapy, pill, or bona fide treatment in at least two independent studies.

Review of the published scientific literature revealed controlled case and group studies on the effects of EEG biofeedback in treating the core symptoms of ADHD. These studies have examined the efficacy of well-defined treatment protocols in the treatment of patients diagnosed with hyperkinesia and patients diagnosed with each of the primary subtypes of ADHD (inattentive, hyperactive-impulsive, or combined). Clinical gains reported in these studies have included significant improvement on standardized tests of intelligence, attention, and behavioral control after EEG biofeedback. The study by Monastra et al [101] noted that increased level of cortical arousal was observed in patients who had received EEG biofeedback and that such gains were sustained without stimulant medication.

Multiple comparisons with a bona fide treatment for ADHD (stimulant medication) also have indicated equivalent or superior results with EEG biofeedback [98,99,101]. In each of these studies, however, patients (via parents or guardians) chose type of treatment, which increased the possibility that treatment effects were enhanced by selection bias. Although the results of the single, published, randomized, controlled trial, which used a waiting list control, illustrated several beneficial effects of EEG biofeedback [100], the classification of EEG biofeedback as “probably efficacious” for the treatment of ADHD is most consistent with the Association for Applied Psychophysiology and Biofeedback and International Society for Neuronal Regulation guidelines, because only one randomized controlled trial has been published to date. Once

the research findings of deBeus et al's double-blind, placebo-controlled study [117] and Beauregard's fMRI study [118] are subjected to peer review and are published, EEG biofeedback would meet criteria to be considered efficacious for the treatment of ADHD.

### **Integrating electroencephalographic biofeedback into clinical practice**

The decision to initiate a trial of EEG biofeedback is typically precipitated by one of three primary reasons. In my review of 800 patients treated with EEG biofeedback at our clinic during the past 10 years [84], the most common reason for a request of this type of treatment was a child's failure to respond to a series of at least two stimulant medications (68%), followed by severe adverse side effects, including irritability, aggressive behavior, significant weight loss, severe and persistent headaches, and insomnia (22%). An additional 10% of parents sought EEG biofeedback for their child because of a family history of addiction, a fear that their child would become addicted to stimulants, or concern about the safety of long-term use of stimulants.

Examination of service use patterns revealed that approximately 10% of patients who sought care at our specialized clinic selected EEG biofeedback (800/8390 patients evaluated and treated) during the past 10 years. A much larger percentage reported an interest in this type of treatment (61%); however, cost, lack of insurance benefits, transportation and scheduling problems, and questions about the permanence of clinical benefits of EEG biofeedback were commonly cited as reasons for selecting other types of services offered at our clinic (eg, monitoring of medication response, school consultation to develop educational and behavioral intervention plans, nutritional counseling, parental training, social skills training, individual and family therapy).

As with any clinical intervention, a decision to initiate a trial of EEG biofeedback proceeds from a comprehensive evaluation of data obtained from multiple sources. At our clinic we begin with a semi-structured, standardized clinical interview [119] to obtain relevant medical, psychiatric, developmental, social, and academic background information. Copies of medical and academic test results and report cards or transcripts are also requested and reviewed. Subsequently, patients with a history consistent with a diagnosis of ADHD are evaluated using a behavioral rating scale, such as the home and school versions of the Attention Deficit Disorders Evaluation Scale [120], a continuous performance test (eg, TOVA [107]), and a qEEG evaluation using power spectral analysis [78]. Although the diagnosis of ADHD is made using Diagnostic and Statistical Manual (fourth edition) criteria, we have found that evidence of cortical slowing on qEEG evaluation is commonly associated with positive response to stimulant medication, provided that other medical conditions that can cause such slowing (eg, anemia, hypoglycemia, diabetes, thyroid disorders, vitamin B deficiencies, mineral deficiencies of zinc and magnesium, sleep apnea, allergies, psychoactive substance abuse) are not present.

Because symptoms of inattention, hyperactivity, and impulsivity can be caused by medical conditions other than ADHD, we refer all of our patients to their physician for evaluation of these conditions before beginning treatment. Review of our clinical records from the past 20 years revealed that 335 of our 11,780 patients (3%) were screened and effectively treated for their ADHD symptoms by addressing one of the previously listed medical conditions. In instances in which cortical slowing and evidence of the core symptoms of ADHD persisted (despite clinical effective treatment for these other medical conditions), a diagnosis of ADHD was concluded in addition to these Axis 3 conditions.

Once screening for other medical conditions is completed, a multimodal treatment program is developed. Depending on areas of functional impairment, this plan includes nutritional counseling (to improve attention by insuring adequate consumption of dietary protein at breakfast and lunch), medication or EEG biofeedback (to treat the core symptoms of ADHD), parental counseling (to begin the process of improving social functioning at home using the program developed by Monastra [115]), school intervention (to develop, monitor, and revise individual education plans or 504 accommodation plans), and social skills training [67] (as adapted by our clinic).

The decision to proceed with a clinical trial of EEG biofeedback at our clinic typically follows a period in which stimulant medications for ADHD are prescribed but not well tolerated (eg, neuromuscular tics, significant weight loss, sleep disturbance, increased irritability, restlessness, or aggression). In cases in which there is a family history of addictions (and parental fear of promoting addiction in a child), we also accommodate a parental request for this type of treatment. We have not found the EEG biofeedback protocols examined in this article to be beneficial in the treatment of ADHD in children under the age of 6, children who have been diagnosed with mental retardation, children whose attentional problems are caused by another psychiatric disorder (eg, bipolar disorder, major depression, or psychosis) or neurologic condition (eg, seizure disorder; traumatic brain injury), or in instances in which an adolescent patient is abusing alcohol or psychoactive medications. We also do not initiate a clinical trial of EEG biofeedback in children whose families present with such significant marital discord that consistent participation in treatment is unlikely.

### **Adverse effects**

Although none of the case or controlled group studies that have been published to date describes adverse effects when EEG biofeedback is provided in the absence of stimulant medication, Monastra et al [121] and deBeus et al [117] noted that adverse effects can occur during the mid-phase of EEG biofeedback (sessions 20 or more) in children who are being treated with biofeedback and stimulant medication. Monastra and his colleagues reported that as children begin to demonstrate improved self-regulation of cortical arousal via EEG biofeedback, they may exhibit increased irritability, moodiness, and hyperactivity. They indi-

cated that reduction in medication dose (rather than increase) typically resolves these symptoms. In instances in which the primary residual symptoms are anxiety, irritability, or impaired anger control, antihypertensive agents (instead of stimulants) have proven helpful in clinical practice.

The development of symptoms of increased hyperactivity or loss of attention were also reported by deBeus et al [117] during the mid-phase of treatment in children who were being treated with stimulants and EEG biofeedback.

These symptoms responded to reduction in dose of stimulant medication. Other adverse reactions reported by Monastra et al [121] included headaches and dizziness. Such symptoms occur in approximately 1% to 3% of patients and respond to a brief resting period (30 minutes) or consumption of food.

### Adjunctive treatments

Consistent with the Consensus Statement on the Diagnosis and Treatment of ADHD [10], our clinical experience is that none of the traditional psychotherapeutic techniques that have been demonstrated in treating other psychiatric disorders has been effective in treating the core symptoms of ADHD in children treated at our center. Similarly, more recently developed cognitive-behavioral treatments are not used at our clinic, because multiple studies have not indicated that these treatments are effective in improving the behavior or academic functioning of children diagnosed with ADHD [122–124]. We have noted, however, that children and teens who are being subjected to parental neglect or abuse respond favorably to individual and family therapy provided at our clinic (and community-based interventions from child protective agencies) that address such issues.

### References

- [1] Pelham WE, Gnagy EM, Greenslade KE, Milich R. Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *J Am Acad Child Adolesc Psychiatry* 1992;31:210–8.
- [2] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th edition. Washington, DC: American Psychiatric Association; 1994.
- [3] Wolraich ML, Hannah JN, Pinnock TY, Baumgaertel A, Brown J. Comparison of diagnostic criteria for attention-deficit hyperactivity disorder in a country wide sample. *J Am Acad Child Adolesc Psychiatry* 1996;35:319–24.
- [4] Gadow KD, Sprafkin J. Child symptom inventory 4: norms manual. Stony Brook (NY): Checkmate Plus; 1997.
- [5] Anderson JC, Williams S, McGee R, Silva PA. DSM-III disorders in preadolescent children: prevalence in a large sample from the general population. *Arch Gen Psychiatry* 1987;44:69–76.
- [6] McGee R, Feehan M, Williams S, Partridge F, Silva PA, Kelly J. DSM-III disorders in a large sample of adolescents. *J Am Acad Child Adolesc Psychiatry* 1990;29:270–9.
- [7] Esser G, Schmidt MH, Woerner W. Epidemiology and course of psychiatric disorders in school-age children: results of a longitudinal study. *J Child Psychol Psychiatry* 1990;31:243–63.
- [8] Leung PWL, Luk SL, Ho TP, Taylor E, Mak FL, Bacon-Shone J. The diagnosis and prevalence of hyperactivity in Chinese schoolboys. *Br J Psychiatry* 1996;168:486–96.



- [9] Bhatia MS, Nigam VR, Bohra NJ, Malik SC. Attention deficit disorder with hyperactivity among paediatric outpatients. *J Child Psychol Psychiatry* 1991;32:297–306.
- [10] National Institutes of Health. Consensus statement on the diagnosis and treatment of attention-deficit/hyperactivity disorder. Bethesda: National Institutes of Health; 1998.
- [11] Barkley RA. Attention-deficit hyperactivity disorder: a handbook for diagnosis and treatment. 2nd edition. New York: Guilford Press; 1998.
- [12] Jensen PS, Hinshaw SP, Swanson JM, Greenhill LL, Conners CK, Arnold LE, et al. Findings from the multimodal treatment study of ADHD (MTA): implications and applications for primary care providers. *Dev Behav Pediatr* 2001;22(1):60–73.
- [13] Pliszka SR. Comorbidity of attention deficit hyperactivity disorder and overanxious disorder. *J Am Acad Child Adolesc Psychiatry* 1992;28:882–7.
- [14] 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.
- [15] Frick PJ, Kamphaus RW, Lahey BB, Loeber R, Christ MAG, Hart EL, et al. Academic underachievement and the disruptive behavior disorders. *J Consult Clin Psychol* 1991;59:289–94.
- [16] Faraone SV, Biederman J, Lehman BK, Spencer T, Norman D, Seidman LJ, et al. Intellectual performance and school failure in children with attention deficit hyperactivity disorder and in their siblings. *J Abnorm Psychol* 1993;102:616–23.
- [17] Campbell SB, Paulauskas S. Peer relations in hyperactive children. *J Child Psychol Psychiatry* 1979;20:233–46.
- [18] Johnston C, Pelham WE, Murphy HA. Peer relationships in ADHD and normal children: a developmental analysis of peer and teacher ratings. *J Abnorm Child Psychol* 1985;13:89–100.
- [19] Pope AW, Bierman KL, Mumma GH. Relations between hyperactive and aggressive behavior and peer relations at three elementary grade levels. *J Abnorm Child Psychol* 1989;17:253–67.
- [20] Barkley RA, DuPaul GJ, McMurray MB. A comprehensive evaluation of attention deficit disorder with and without hyperactivity. *J Consult Clin Psychol* 1990;58:775–89.
- [21] Weiss G, Hechtman L. Hyperactive children grown up. 2<sup>nd</sup> edition. New York: Guilford Press; 1993.
- [22] Mannuzza S, Gittelman-Klein R, Bessler A, Malloy P, LaPadula M. Adult outcome of hyperactive boys: educational achievement, occupational rank and psychiatric status. *Arch Gen Psychiatry* 1993;50:565–76.
- [23] Mannuzza S, Klein RB, Bessler A, Malloy P, LaPadula M. Adult psychiatric status of hyperactive boys grown up. *Am J Psychiatry* 1998;155:493–8.
- [24] Murphy K, Barkley RA. Attention deficit hyperactivity disorder adults: comorbidities and adaptive impairments. *Compr Psychiatry* 1996;37:393–401.
- [25] Mannuzza S, Klein RG, Addalli KA. Young adult mental status of hyperactive boys and their brothers: a prospective follow-up study. *J Am Acad Child Adolesc Psychiatry* 1991;30:743–51.
- [26] Claude D, Firestone P. The development of ADHD boys: a 12 year follow-up. *Can J Behav Sci* 1995;27:226–49.
- [27] Barkley RA, Guevremont DG, Anastopoulos AD, DuPaul GJ, Shelton TL. Driving-related risks and outcomes of attention deficit hyperactivity disorder in adolescents and young adults: a 3–5 year follow-up survey. *Pediatrics* 1993;92:212–8.
- [28] August GJ, Stewart MA, Holmes CS. A four-year follow-up of hyperactive boys with and without conduct disorder. *Br J Psychiatry* 1983;143:192–8.
- [29] Cantwell DP, Baker L. Stability and natural history of DSM-III childhood diagnoses. *J Am Acad Child Adolesc Psychiatry* 1989;28:691–700.
- [30] Silberg J, Rutter M, Meyer J, Maes H, Hewitt J, Simonoff E, et al. Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *J Child Psychol Psychiatry* 1996;37:803–16.
- [31] Levy R, Hay DA, McStephen M, Wood C, Waldman I. Attention deficit hyperactivity disorder: a category or a continuum? Genetic analysis of a large-scale twin study. *J Am Acad Child Adolesc Psychiatry* 1997;36:737–44.

- [32] Willcutt EG, Pennington BF, DeFries JC. Twin study of the etiology of comorbidity between reading disability and attention-deficit/hyperactivity disorder. *Am J Med Genet* 2000;96:293–301.
- [33] Welner Z, Welner A, Stewart M, Palkes H, Wish E. A controlled study of siblings of hyperactive children. *J Nerv Ment Dis* 1977;165:110–7.
- [34] Biederman J, Keenan K, Faraone SV. Parent-based diagnosis of attention deficit disorder predicts a diagnosis based on teacher report. *J Am Acad Child Adolesc Psychiatry* 1990;33:842–8.
- [35] Biederman J, Faraone S, Keenan K, Benjamin J, Krifcher B, Moore C, et al. Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. *Arch Gen Psychiatry* 1992;49:728–38.
- [36] Biederman J, Faraone SV, Mick E, Spencer T, Wilens T, Kiely K, et al. High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: a pilot study. *Am J Psychiatry* 1995;152:431–5.
- [37] Volkow ND, Ding YS, Fowler JS, Wang GJ, Logan J, Gatley JS, et al. Is methylphenidate like cocaine? Studies on their pharmacokinetics and distribution in the human brain. *Arch Gen Psychiatry* 1995;52:456–63.
- [38] Ding YS, Fowler J, Volkow N, Dewey S, Wang GJ, Logan J, et al. Clinical drugs: comparison of the pharmacokinetics of [11 C]d-threo and 1-threo-methylphenidate in the human and baboon brain. *Psychopharmacology (Berl)* 1997;131:71–8.
- [39] Smalley SL, Bailey JN, Palmer CG, Cantwell DP, McGough JJ, Del-Homme MA, et al. Evidence that the dopamine D4 receptor is a susceptibility gene in attention deficit hyperactivity disorder. *Mol Psychiatry* 1998;3:427–30.
- [40] Langley K, Marshall L, van den Bree M, Thomas H, Owen NM, O'Donovan M, et al. Association of the dopamine D4 receptor gene 7 repeat allele with neuropsychological test performance of children with ADHD. *Am J Psychiatry* 2004;161:133–8.
- [41] Comings DE, Wu S, Chiu C, Ring RH, Gade R, Ahn C, et al. Polygenic inheritance of Tourette syndrome, stuttering, attention deficit hyperactivity, conduct and oppositional defiant disorders: the additive and subtractive effect of the three dopaminergic genes—DRD2, DBH, and DAT1. *Am J Med Genet* 1996;67(3):264–88.
- [42] Cook EH, Stein MA, Kraskowski MD, Cox NJ, Olkon DM, Kieffer JE, et al. Association of attention deficit disorder and the dopamine transporter gene. *Am J Hum Genet* 1995;56:993–8.
- [43] Waldman ID, Rowe DC, Abramowitz A, Kozel ST, Mohr JH, Sherman SL, et al. Association and linkage of the dopamine transporter gene and attention-deficit hyperactivity disorder in children: heterogeneity owing to diagnostic subtype and severity. *Am J Hum Genet* 1998;63:1767–76.
- [44] Hynd GW, Semrud-Clikeman M, Lorys AR, Novey ES. Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. *Arch Neurol* 1990;47:919–26.
- [45] Hynd GW, Hern KL, Novey ES, Eliopoulos D. Attention deficit-hyperactivity disorder and asymmetry of the caudate nucleus. *J Child Neurol* 1993;8:339–43.
- [46] Castellanos FX, Giedd JN, Eckburg P, Marsh WL, Vaituzis AC, Kaysen D, et al. Quantitative morphology of the caudate nucleus in attention deficit hyperactivity disorder. *Am J Psychiatry* 1994;151:1791–6.
- [47] Giedd JN, Castellanos FX, Casey BJ, Kozuch P, King AC, Hamburger SD, et al. Quantitative morphology of the corpus callosum in attention deficit hyperactivity disorder. *Am J Psychiatry* 1994;151:665–9.
- [48] Aylward EE, Reiss AL, Reader MJ, Singer HS, Brown JE, Denckla MB. Basal ganglia volumes in children with attention-deficit hyperactivity disorder. *J Child Neurol* 1996;11:112–5.
- [49] Castellanos FX, Giedd JN, Marsh WL, Hamburger SD, Vaituzis AC, Dickstein DP, et al. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Arch Gen Psychiatry* 1996;53:607–16.
- [50] Castellanos FX. Toward a pathophysiology of attention-deficit/ hyperactivity disorder. *Clin Pediatr (Phila)* 1997;36:381–93.

- [51] Mostofsky SH, Reiss AL, Lockhart P, Denckla MB. Evaluation of cerebellar size in attention-deficit hyperactivity disorder. *J Child Neurol* 1998;13:434–9.
- [52] Swanson JM, Castellanos FX. Biological bases of ADHD: neuroanatomy genetics, and pathophysiology. In: Jensen PS, Cooper JR, editors. *Attention deficit hyperactivity disorder: state of the science. Best practices*. Kingston (NJ): Civic Research Institute; 2002. p. 7-1–7-20.
- [53] Giedd JN, Blumenthal J, Molloy E, Castellanos FX. Brain imaging of attention deficit/hyperactivity disorder. *Ann NY Acad Sci* 2001;931:33–49.
- [54] Lou HC, Henriksen L, Bruhn P. Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. *Arch Neurol* 1984;41(8):825–9.
- [55] Lou HC, Henriksen L, Bruhn P. Focal cerebral dysfunction in developmental disabilities. *Lancet* 1990;335(8680):8–11.
- [56] Sieg KG, Gaffney GR, Preston DF, Hellings JA. SPECT brain imaging abnormalities in attention deficit hyperactivity disorder. *Clin Nucl Med* 1995;20(1):55–60.
- [57] Kim B-N, Lee J-S, Shin M-S, Cho S-C, Lee D-S. Regional cerebral perfusion abnormalities in attention deficit/hyperactivity disorder: statistical parametric mapping analysis. *Eur Arch Psychiatry Clin Neurosci* 2002;252:219–25.
- [58] Zametkin AJ, Nordahl TE, Gross M, King AC, Semple WE, Rumsey J, et al. Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *N Engl J Med* 1990;323:1361–6.
- [59] Ernst M, Liebenauer LL, King AC, Fitzgerald GA, Cohen RM, Zametkin AJ. Reduced brain metabolism in hyperactive girls. *J Am Acad Child Adolesc Psychiatry* 1994;33(6):858–68.
- [60] Dresel S, Krause J, Krause KH, LaFougere C, Brinkbaumer K, Kung HF, et al. Attention deficit hyperactivity disorder: binding of [<sup>99m</sup>Tc]TRODAT-1 to the dopamine transporter before and after methylphenidate treatment. *Eur J Nucl Med* 2000;27:1518–24.
- [61] Matochek JA, Nordahl TE, Gross M, Semple WE, King AC, Cohen RM, et al. Effects of acute stimulant medication on cerebral metabolism in adults with hyperactivity. *Neuropsychopharmacology* 1993;8:377–86.
- [62] Krause KH, Dresel SH, Krause J, LaFougere C, Brinkbaumer K, Kung HF, et al. Increased striatal dopamine transporter in adult patients with attention deficit hyperactivity disorder: effects of methylphenidate as measured by single photon emission computed tomography. *Neurosci Lett* 2000;285:107–10.
- [63] Greenhill LL, Halperin JM, Abikoff H. Stimulant medications. *J Am Acad Child Adolesc Psychiatry* 1999;38(5):503–12.
- [64] Jensen PS. Current concepts and controversies in the diagnosis and treatment of attention deficit hyperactivity disorder. *Curr Psychiatry Rep* 2000;2:102–9.
- [65] DuPaul GJ, Barkley RA, Connor DF. Stimulants. In: Barkley RA, editor. *Attention deficit hyperactivity disorder: a handbook for diagnosis and treatment*. 2<sup>nd</sup> edition. New York: Guilford Press; 1998. p. 510–51.
- [66] Anastopoulos AD, Smith JM, Wien EE. Counseling and training parents. In: Barkley RA, editor. *Attention deficit hyperactivity disorder: a handbook for diagnosis and treatment*. 2<sup>nd</sup> edition. New York: Guilford Press; 1998. p. 373–93.
- [67] Pfiffner LJ, Barkley RA. Treatment of ADHD in school settings. In: Barkley RA, editor. *Attention deficit hyperactivity disorder: a handbook for diagnosis and treatment*. 2<sup>nd</sup> edition. New York: Guilford Press; 1998. p. 458–90.
- [68] Pelham WE, Hoza B. Intensive treatment: a summer treatment program for children with ADHD. In: Hibbs E, Jensen P, editors. *Psychosocial treatments for child and adolescent disorders: empirically based strategies for clinical practice*. New York: American Psychological Association Press; 1996. p. 311–40.
- [69] Pelham WE, Wheeler T, Chronis A. Empirically supported psychosocial treatments for ADHD. *J Clin Child Psychol* 1998;27:190–205.
- [70] MTA Cooperative Group. 14-month randomized clinical trial of treatment strategies for attention deficit hyperactivity disorder. *Arch Gen Psychiatry* 1999;56:1073–86.
- [71] Pelham WE. Psychosocial interventions for ADHD. In: Jensen PS, Cooper JR, editors. *Attention-deficit hyperactivity disorder: state of the science. Best practices*. Kingston (NJ): Civic Research Institute; 2002. p. 12-1–12-36.

- [72] Lubar JF. Neurofeedback for the management of attention deficit disorders. In: Schwartz MS, Andrasik F, editors. *Biofeedback: a practitioner's guide*. 3rd edition. New York: Guilford Press; 2003. p. 409–37.
- [73] Monastra VJ. Clinical applications of electroencephalographic biofeedback. In: Schwartz MS, Andrasik F, editors. *Biofeedback: a practitioner's guide*. 3rd edition. New York: Guilford Press; 2003. p. 438–63.
- [74] LaVaque TJ, Hammond DC, Trudeau D, Monastra VJ, Perry J, Lehrer P. Template for developing guidelines for the evaluation of the clinical of psychophysiological interventions. *Appl Psychophysiol Biofeedback* 2002;27(4):273–81.
- [75] Mann C, Lubar J, Zimmerman A, Miller C, Muenchen R. Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: controlled study with clinical implications. *Pediatr Neurol* 1992;8:30–6.
- [76] Lubar JF, Swartwood MO, Swartwood JN, Timmermann DL. Quantitative EEG and auditory event-related potentials in the evaluation of attention-deficit disorder: effects of methylphenidate and implications for neurofeedback training. *Journal of Psychoeducational Assessment Monographs* 1995;(Special ADHD Issue):143–60.
- [77] Chabot RA, Merkin H, Wood LM, Davenport TL, Serfontein G. Sensitivity and specificity of QEEG in children with attention deficit or specific developmental learning disorders. *Clin Electroencephalogr* 1996;27:26–34.
- [78] Monastra VJ, Lubar JF, Linden M, VanDeusen P, Green G, Wing W, et al. Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. *Neuropsychology* 1999;13(3):424–33.
- [79] Clarke AR, Barry RJ, McCarthy R, Selikowitz M. Electroencephalogram differences in two subtypes of attention-deficit/hyperactivity disorder. *Psychophysiology* 2001;38:212–21.
- [80] 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.
- [81] Chabot RA, Orgill AA, Crawford G, Harris MJ, Serfontein G. Behavioral and electrophysiological predictors of treatment response to stimulants in children with attention disorders. *J Child Neurol* 1999;14(6):343–51.
- [82] Clarke AR, Barry RJ, McCarthy R, Selikowitz M. EEG differences between good and poor responders to methylphenidate and dexamphetamine in children with attention-deficit/hyperactivity disorder. *Clin Neurophysiol* 2002;113:194–205.
- [83] Monastra VJ, Lubar JF. Using quantitative electroencephalography to differentiate attentional from other psychiatric disorders. Presented at the National Conference of the American Psychological Association. Washington, DC: August 5, 2000.
- [84] Monastra VJ, Monastra DM. EEG biofeedback treatment for ADHD: an analysis of behavioral, neuropsychological, and electrophysiological response over a three-year follow-up period. Presented at the Annual Conference of the Association for Applied Psychophysiology and Biofeedback. Colorado Springs: April 2, 2004.
- [85] Nordahl TE, Benkfelfat C, Semple WE. Cerebral glucose metabolic rates in obsessive compulsive disorder. *Neuropsychopharmacology* 1989;2:23–38.
- [86] Swedo SE, Schapiro MB, Grady CL. Cerebral glucose metabolism in childhood onset obsessive compulsive disorder. *Arch Gen Psychiatry* 1989;46:518–23.
- [87] Machlin SR, Harris GJ, Peralson GD. Elevated medial-frontal cerebral blood flow in obsessive-compulsive patients: a SPECT study. *Am J Psychiatry* 1991;148:1240–2.
- [88] Amen DG, Carmichael BD. Oppositional children similar to OCD on SPECT: implications for treatment. *J Neurotherapy* 1997;1:1–7.
- [89] Serman MB. Physiological origins and functional correlates of EEG rhythmic activities: implications for self-regulation. *Biofeedback Self Regul* 1996;21(1):3–49.
- [90] Roth SR, Serman MB, Clemente CC. Comparison of EEG correlates of reinforcement, internal inhibition, and sleep. *Electroencephalogr Clin Neurophysiol* 1967;23:509–20.
- [91] Serman MB, Wyrwicka W. EEG correlates of sleep: evidence for separate forebrain substrates. *Brain Res* 1967;6:143–63.

- [92] Wywricka W, Sterman MB. Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiol Behav* 1968;3:703–7.
- [93] Sterman MB, Wywricka W, Roth SR. Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Ann N Y Acad Sci* 1969;157:723–39.
- [94] Lubar JF, Shouse MN. EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): a preliminary report. *Biofeedback Self Regul* 1976;1(4):293–306.
- [95] Lubar JO, Lubar JF. Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback Self Regul* 1984;9(1):1–23.
- [96] Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clin Electroencephalogr* 2000;31:45–55.
- [97] Clarke AR, Barry RJ, McCarthy R, Selikowitz M. Excess beta activity in children with attention-deficit/hyperactivity disorder: an atypical electrophysiological group. *Psychiatry Res* 2001;103:205–18.
- [98] Fuchs T, Birbaumer N, Lutzenberger W, Gruzelier JH, Kaiser J. Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: a comparison with methylphenidate. *Appl Psychophysiol Biofeedback* 2003;28(1):1–12.
- [99] Rossiter TR, LaVaque TJ. A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. *J Neurotherapy* 1995;1:48–59.
- [100] 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(1):35–49.
- [101] 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(4):231–49.
- [102] Tansey M, Bruner RL. EMG and EEG biofeedback in the treatment of a 10 year old hyperactive boy with a developmental reading disorder. *Biofeedback Self Regul* 1983;8:25–37.
- [103] Thompson L, Thompson M. Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. *Appl Psychophysiol Biofeedback* 1998;23(4):243–63.
- [104] Kaiser DA, Othmer S. Effect of neurofeedback on variables of attention in a large multi-center trial. *Journal of Neurotherapy* 2000;4(1):5–28.
- [105] Carmody DP, Radvanskik DC, Wadhvani S, Sabo MJ, Vergara L. EEG biofeedback training and attention-deficit/hyperactivity disorder in an elementary school setting. *Journal of Neurotherapy* 2001;4(3):5–27.
- [106] McCarney SB. *The attention deficit disorders evaluation scale*. Columbia (OH): Hawthorne Press; 1989.
- [107] Greenberg LM, Dupuy TR. *Interpretation manual for the test of variables of attention*. Los Alamitos (CA): Universal Attention Disorders; 1993.
- [108] Tansey M. Ten-year stability of EEG biofeedback results for a hyperactive boy who failed the fourth grade perceptually impaired class. *Biofeedback Self Regul* 1993;18(1):33–8.
- [109] Chambless DL, Hollon SD. Defining empirically supported therapies. *J Consult Clin Psychol* 1998;66(1):7–18.
- [110] Nuwer M. Assessment of digital EEG, quantitative EEG, and EEG brain mapping: report of the American Academy and the American Clinical Neurophysiology Society. *Neurology* 1997;49:277–92.
- [111] Heywood C, Beale I. EEG biofeedback vs placebo treatment for attention-deficit/hyperactivity disorder: a pilot study. *J Atten Disord* 2003;7(1):41–53.
- [112] Reynolds C, Kamphaus R. *Behavioral assessment system for children*. Circle Pines (MN): American Guidance Service; 1994.
- [113] Kaufman A, Kaufman N. *K-BIT: Kaufman brief intelligence test manual*. Circle Pines (MN): American Guidance Service; 1990.
- [114] Atkins M, Milich R. IOWA-Conners teacher rating scale. In: Hersen M, Bellack A, editors. *Dictionary of behavioral assessment techniques*. New York: Pergamon Press; 1987. p. 273–5.

- [115] Monastra VJ. Parenting children with ADHD: lessons that medicine cannot teach. Washington, DC: American Psychological Association; 2004.
- [116] Brickenkamp R. Test d2, Aufmerksamkeits-Belastungs-Test. 8th edition. Göttingen: Hogrefe; 1994.
- [117] deBeus R, Ball JD, deBeus ME, Herrington R. Attention training with ADHD children: preliminary findings in a double-blind placebo-controlled study. Presented at the Annual Conference of the International Society for Neuronal Regulation. Houston: August 29, 2003.
- [118] Beauregard M. Effect of neurofeedback training on the neural substrate of executive deficits in AD/HD children. Presented at the Annual Conference of the International Society for Neuronal Regulation. Houston: August 27, 2002.
- [119] Barkley RA, Murphy KR. Attention-deficit hyperactivity disorder: a clinical workbook. 2nd edition. New York: Guilford Press; 1998.
- [120] McCarney SB, Arthaud RJ. The attention deficit disorders evaluation scale. 3<sup>rd</sup> edition. Columbia (OH): Hawthorne Press; 2004.
- [121] 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* 2004, in press.
- [122] Abikoff H, Ganeles D, Reiter G, Blum C, Foley C, Klein R. Cognitive training in academically deficient ADD-H boys receiving stimulant medication. *J Abnorm Child Psychol* 1988;16(4): 411–32.
- [123] Bloomquist ML, August GJ, Ostrander R. Effects of a school-based cognitive-behavioral intervention for ADHD children. *J Abnorm Child Psychol* 1991;19:591–605.
- [124] Brown RT, Borden KA, Wynne ME, Spunt AL, Clingerman SR. Compliance with pharmacological and cognitive treatment for attention deficit disorder. *J Am Acad Child Adolesc Psychiatry* 1987;26:521–6.