EEG Biofeedback Treatment of ADD
A Viable Alternative to Traditional Medical Intervention?

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ABSTRACT: Literature searches dating back to 1968 were conducted through Psychlit and Medline services to review the scientific literature on EEG biofeedback treatment of ADD. While anecdotal and case reports cite promising evidence, methodological problems coupled with a paucity of research precludes any definitive conclusions as to the efficacy of enhanced alpha and hemisphere-specific EEG biofeedback training. One of the more promising EEG biofeedback treatment paradigms involves theta/beta training. Studies have reported that academic, intellectual, and behavioral gains have been attained with this approach. Significant behavioral and cognitive changes have also been reported following SMR training. However, research into these treatment approaches has also been marred by methodological inadequacies and lack of sufficient follow-up studies. A number of recommendations for future research into this treatment approach are made.

KEYWORDS: ADD; ADHD; Biofeedback; EEG biofeedback.

INTRODUCTION

In treating adults with residual Attention Deficit Disorder (ADD) symptomatology, the more traditional treatment approaches have included stimulant medication and/or cognitive behavioral psychotherapeutic techniques. While predominantly focusing on children and young adolescents, over the past twenty-five to thirty years research has surfaced positing EEG biofeedback as yet another viable technique in the treatment of this disorder.1–3 Biofeedback techniques use external instrumentation to provide a subject with information regarding psychophysiological processes, which are usually outside of one’s awareness and ability to control.4 The usual procedure involves establishing reinforcer-response contingencies with rewards being supplied for desired changes in internalized physiologic responses.5 Thus, the clinician sets desired thresholds on the biofeedback equipment which are based on treatment goals and, as the client’s physiologic changes approach and surpass those thresholds, the equipment provides either auditory or visual feedback which serves
as reinforcement for the desired changes. As an example, as a client decreases theta and increases beta waves during EEG biofeedback treatment of ADD, reinforcement is provided to encourage the client to become more aware of what he is doing to achieve this desired state and to continue in the same vein.

Given that research involving the application of EEG biofeedback training in the treatment of either ADD and/or learning disability (LD) almost exclusively involves children, this chapter critically reviews some of the more pertinent studies of children, particularly those with potential relevance to the adolescent and adult populations. Literature searches were conducted through Psychlit and Medline services dating back to 1968 in order to locate relevant articles.

This review is divided into four sections. The first section (after the introduction) describes the application of sensorimotor rhythm (SMR) training, which is generally utilized in the treatment of hyperactivity. While this is certainly less of an issue when dealing with adult residual ADD phenomenology, this section is included to provide an overview of the EEG biofeedback procedures most often utilized in dealing with ADHD (Attention Deficit/Hyperactivity Disorder). The second section focuses on theta/beta training. The third area involves a discussion of alpha training. Finally, we describe the utilization of hemisphere-specific EEG biofeedback training to ameliorate the behavioral and/or cognitive difficulties typically associated with ADD and ADHA (used somewhat interchangeably). In reviewing the efficacy of each approach, four important questions are applied:

1. Did biofeedback training result in the intended EEG changes?
2. Did significant modifications in cognition and behavior ensue inside and out of the laboratory?
3. Could these changes be reliably linked to biofeedback treatment?
4. And lastly, are these changes retained over time?

Owing to their extent and complexity, theories accounting for the results found in these studies have been excluded other than to mention that EEG biofeedback is generally hypothesized to affect the brain abnormalities associated with ADD and related disorders, and to promote those changes in maladaptive behavior and inattention typical of such disorders.

In EEG biofeedback training, feedback is typically furnished concerning the deficiency or overabundance of a specific amplitude and/or frequency of brain wave electrical activity. In the treatment of ADD, feedback is usually provided to promote a reduction in theta waves (4–8 Hz), typically evident when drowsy or dreaming, either alone or in combination with the enhancement of beta waves (16–20 Hz), which are associated with higher levels of arousal, problem-solving, and anxiety. In hyperactive states, sensorimotor rhythms (SMR) (12–15 Hz), believed to be related to somatomotor inhibition, are also an integral part of the treatment plan. In addition, alpha rhythms (8–12 Hz), which are related to states of wakeful relaxation and hemispheric discrepancies, are also targeted. The overall aim of these approaches is to produce desired changes in behavioral and cognitive states, and are summarized in Table 1.

Research dating back 35 years has established the presence of various degrees of EEG abnormalities in children with ADD and/or LD. Preliminary studies found that 63% of children with LD showed EEG abnormalities as opposed to 20% of matched
controls. Later, other investigators specified these abnormalities. As an example, Winkler, Dixon and Parker detected greater diffuse, slow wave theta activity, less fast wave beta SMR activity and more abnormal transient discharges in a sample of children evidencing scholastic and behavioral problems. In addition, Lubar, Bianchini, Calhoun, Lambert, Brody and Shasbin reported that LD-ADHD children exhibited significantly greater amounts of theta and more EMG activity (muscular tension) than normal matched controls. They could also predict group membership (LD-ADHD vs. non-LD) by better than 97% when a combination of EEG parameters were utilized, and by more than 80% when restricting predictors to variables concerning increased theta activity in frontal-temporal locations alone. In another study, Mann, Lubar, Zimmerman, Miller and Muenchen found additional evidence that children with pure ADD exhibited increased theta activity in frontal and central locations, and decreased beta activity in frontal and temporal regions as compared to matched controls. It has been suggested, however, that at least some of the EEG abnormalities seen in younger ADHD children may not be generalizable to adults with ADD as they are pathognomonic of a maturational lag in cerebral development which normalizes with advancing age, in particular during the latter stages of adolescence. While intriguing, there are those who believe that EEG abnormalities continue to be clearly evident in adolescents and adults with ADD.

In addition to EEG abnormalities found in those individuals with ADD-related difficulties, numerous other investigations have yielded additional evidence of cerebral dysfunction. As an example, Zametkin, Nordal, and Gross studied and compared PET scans of adults with histories of childhood hyperactivity and persisting complaints of restlessness and inattentiveness with those of normal controls. These individuals were naive to stimulant medication and were the biological parent of a hyperactive child. They found that the hyperactive adult group exhibited 8.1% lower global cerebral glucose metabolism than did normal controls. They also reported significant hypometabolism in 30 of 60 brain regions, particularly, but not exclusively, in areas of the frontal and central cortex, areas previously found to demonstrate the greatest theta elevations and beta reductions in children with ADD.

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<tr>
<th>Rhythm</th>
<th>Phenomenology</th>
<th>Training</th>
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<tr>
<td>Sensorimotor rhythm (SMR)</td>
<td>Movement causes blockage of SMR</td>
<td>Increase SMR</td>
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<tr>
<td>Theta rhythm</td>
<td>Abundant when drowsy</td>
<td>Decrease theta</td>
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<td>Beta rhythm</td>
<td>Associated with attentional and/or memory processes</td>
<td>Increase beta</td>
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<tr>
<td>Alpha rhythm</td>
<td>Associated with a relaxed, but alert state</td>
<td>Increase/decrease alpha</td>
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<td>Hemispheric rhythm training</td>
<td>Still experimental</td>
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Note that EEG biofeedback training often involves the training of either increases or decreases involving more than one type of brain wave activity.

Some debate continues as to whether alpha should be increased or decreased.
Further research has also shown that children with ADHD and LD exhibit less alpha attenuation while engaging in cognitive activities, including listening and arithmetic, and immediate recall tasks, in conjunction with more incorrect responding, as compared with normal controls. These findings are noteworthy because of the inverse relationship previously cited between alpha, attention, and cognitive activity. Discrepancies have also been noted in the cerebral functioning of these populations. In particular, right hemisphere dysfunction has been demonstrated in children and adolescents with ADD with and without hyperactivity and in children with ADHD demonstrating comorbid LD.

SMR BIOFEEDBACK

The use of SMR biofeedback training for children with hyperactivity stemmed, in part, from a body of literature which demonstrated that conditioned increases in SMR brain wave activity achieved via biofeedback training produced marked decreases in seizure activity in epileptics as well as motor inhibition. Wyricka and Sternman found that cats reinforced for the production of increased levels of SMR obtained that reinforcement by assuming motionless positions. Chase and Harper also reported reduced muscular tension in cats following SMR training. In humans, Sternman et al. described heightened SMR activity in the EEGs of paraplegics and quadriplegics and a decrement of the rhythm in epileptics, particularly when motor symptomatology was displayed. Following this work, Lubar and Bahlert reported increased motor control in a 14-year-old subject who was both epileptic and hyperactive in conjunction with increased production of SMR and decreased slow wave activity during SMR training. The investigators were tentative in drawing conclusions from these findings because of the high number of potential confounding factors, including maturational changes, which could account for such results.

In examining the efficacy of SMR biofeedback in individuals with histories uncomplicated by seizure activity, Lubar and Shouse investigated the application of SMR training with an 11-year, 8-month-old hyperkinetic male. The child underwent five phases of training, baseline periods of No Drug (I) and Drug only (II) conditions in the absence of EEG biofeedback, a feedback phase (III) during which the production of 12–14 Hz and the inhibition of 4–7 Hz EEG activity were reinforced, a contingency reversal condition (IV) in which the reinforcement paradigm was reversed and finally a reinstatement of the original feedback phase (V). During conditions II to V, the child was on a steady regimen of methylphenidate (10 mg/day). Training occurred three times per week, 40 minutes each time, with sessions consisting of two 5-minute baseline periods and two 15-minute feedback phases. It was found that SMR values consistently increased as training progressed, returning to pre-treatment levels only temporarily during the contingency reversal phase and following a three-week lapse in training. Furthermore, a statistically significant inverse relationship between SMR and electromyography (EMG) (motor tension) was also reported during each training phase, fortifying the link between SMR and motor inhibition as well as baseline shifts in SMR activity, providing evidence for a carry-over effect of learning. Finally, thirteen behavioral categories adapted from Wahler’s
category system were observed in the child’s classroom setting. Six, including out of seat behavior, cooperation, object play, sustained non-interaction, sustained school work and opposition, showed even greater improvement with SMR training and medication than with medication alone. Improvement was also noted in two other behaviors (i.e., self-stimulation and sustained attention) not affected by drug therapy. As with SMR, these behavioral gains reversed in large part during the counterconditioning phase. In a later paper, Lubar\textsuperscript{18} reported that follow-ups conducted several years post treatment showed the subject to have maintained the gains achieved during SMR training and to be performing well academically and without medication.

Using a similar design and similar selection criteria, Shouse and Lubar\textsuperscript{2,34} trained four hyperkinetic children with pronounced classroom misconduct in combination with substantial deficits in SMR and arousal. In addition to those of the design described above, experimental conditions included a sixth phase, which consisted of SMR training in the absence of medication. Relative to initial and training-phase baselines, three of the four subjects demonstrated increases in SMR during training, including the final SMR alone condition, and temporary reinstatement of near pre-training levels of SMR during counterconditioning. These same subjects also showed modest initial baseline shifts in SMR, providing evidence for the carry-over of training effects. One subject who, as his principal difficulty, evidenced the highest levels of pre-treatment SMR and distractibility, as opposed to hyperactivity, failed to acquire the SMR task and was dropped from the study after six months. As in their earlier study, all four subjects showed behavioral improvement in six of thirteen classroom behaviors with medication.\textsuperscript{33} Yet, it was solely those demonstrating increases in SMR, who exhibited additional improvements in those same behaviors in both the training and medication as well as training alone conditions. Amelioration of two behaviors (i.e., self-stimulation and sustained attention) which had not been helped by medication alone, was also noted. In addition, there were trends for these improvements to temporarily reverse during the counterconditioning phase.

Tansey and Bruner\textsuperscript{1} applied both SMR biofeedback and EMG training with a 10-year-old boy diagnosed as ADD with hyperactivity, a developmental reading disorder and ocular instability (i.e., problems with saccadic fixation and ocular smooth pursuit movements). Typically, in EMG training for general relaxation, a reduction of muscular tension is achieved via EMG feedback monitored over the central forehead region. Such techniques have reportedly demonstrated some utility in the treatment of hyperactivity.\textsuperscript{35} After being weaned off Ritalin, the child first underwent 3 EMG training sessions (1 per week) and subsequently 20 weekly SMR sessions, in addition to daily practice of relaxation techniques in which he had been trained. In conjunction with decreased EMG readings following training, subjective reports showed him to be in greater control of his behavior and no longer diagnosable as hyperactive. Gains superseded those achieved with medication alone. Furthermore, with an increase in SMR following biofeedback, the child’s ability to read and comprehend improved significantly and his ocular instability improved. Specifically, he was able to keep his head stationary while tracking smoothly along a horizontal axis as well as retaining focus on an object while moving his head from side to side. While originally scheduled to be retained in a fourth grade class for the perceptually impaired prior to biofeedback, after training the child was enrolled in a
normal fourth grade class where he attained better than passing grades in all areas throughout the academic year. These improvements were maintained on 24-month follow-up as well as 10 years post-treatment, when the subject was reported as continuing to exhibit normal social and academic functioning and EEG activity comparable to that of 24 children who had also undergone biofeedback training and were no longer deemed as mixed ADHD and learning disabled. In addition, Tansey applied SMR biofeedback training with six LD boys using a reversal (A-B-A) design. Besides biofeedback, the subjects were not involved in any additional form of treatment for their learning difficulties other than academic training including resource room and special class assignments. The investigator found mean increases in SMR amplitudes ranging from 38–300%, with a mean group increase of 137.6% over baseline. He also reported that all of the subjects demonstrated improvements of at least one standard deviation in their WISC-R Full Scale IQ scores. Those individuals evidencing significant pre-treatment verbal/performance discrepancies (15 points or greater) showed substantially greater improvement in the lower of their two scores following training. Replicating their prior findings, those subjects with additional oculomotor dysfunction exhibited improvement of these difficulties.

Lubar and Lubar also trained six males with specific learning disabilities and, in some cases, hyperactivity using a combination of SMR and subsequent beta biofeedback in addition to academic training twice a week over a 10–27 month period. All children exhibited increased production of SMR and beta activity as well as decreased EMG and slow wave (theta) activity. Subjects also evidenced improved academic performance, as demonstrated by achievement test scores or grades, and greater behavioral control. Notably, five of the six children had previously been in academic training and special resource classes prior to biofeedback training without significant improvement having being noted. Tansey also utilized SMR training with six neurologically impaired, one perceptually impaired, and one hyperkinetic child. Simultaneously recording five frequency bands of brain activity (5, 7, 10, 12, and 14), he found a mean group SMR amplitude change of 76% over baseline at the end of training in conjunction with a reduction in average slow wave (theta) activity for those with an IQ between 76 and 85 and an increase across all frequency bands for those within the 102–116 range, with slow wave activity increasing the least. All subjects demonstrated WISC-R IQ score improvements of at least one standard deviation, with those exhibiting verbal/performance discrepancies of greater than 14 points evidencing a greater increase in the lower of their two scores. As in their past work, amelioration of ocular motor dysfunction was also found. Lastly, Tansey trained eleven neurologically impaired children, eleven perceptually impaired, and two with ADD for a mean of 27.9 sessions utilizing SMR biofeedback techniques. All subjects exhibited significant mean energy decreases in slow wave (5 and 7 Hz) and increases in fast wave (12 and 14 Hz) activity. In addition, elevations in Full Scale IQ of at least (or close to) one standard deviation were achieved as well as normalization of verbal/performance discrepancies.

Overall, in all seven studies presented, increased production of SMR was demonstrated and, in some cases, coincided with increased beta and decreased EMG and theta activity. One subject was reported as unable to increase SMR production and four to evidence elevations as opposed to decrements in theta activity, although six of Tansey’s subjects were neurologically impaired in an unspecified manner.
Behaviorally, subjects were noted to demonstrate less hyperactivity and greater behavioral control following SMR and EMG training as well as amelioration of a number of maladaptive classroom behaviors and remediation of oculomotor dysfunction. Cognitively, individuals evidenced improved abilities in reading and comprehension, better grades, increased WISC-R IQ scores and resolution of verbal/performance discrepancies. Presumably, the better grades and test scores are attributable to their ability to stay on-task for longer periods of time. While such findings are quite compelling, control groups were not utilized in any of the above studies and designs, at times, mixed a variety of biofeedback techniques (such as SMR, EMG, and beta biofeedback) as well as academic training and medication. These factors make a definitive link between biofeedback training and the resulting changes difficult to forge. On the other hand, studies utilizing reversal designs reported a remediation of EEG and behavioral gains upon counterconditioning and lapses in training. Moreover, the gains achieved with SMR training were above and beyond those resulting from medication and/or resource classroom instruction and were maintained in the absence of other forms of treatment. Finally, the single subject who failed to enhance SMR production also failed to attain the behavioral and electrophysiological improvements demonstrated by other subjects. Only two case studies included follow-up reports to ascertain whether these results were maintained. In each, EEG, behavioral and academic gains were reported to have been retained up to ten years post-treatment.

In general, while some of these findings are promising, these studies suffer from methodological flaws consistently evident across many of the studies examined. Specifically, the use of control groups are lacking and subjects carrying different diagnoses as well as some with comorbid disorders are often lumped together, thus assuming that they share common neuropathological processes.

THETA/BETA BIOFEEDBACK

A number of studies were also found targeting theta and/or beta brain wave levels to produce desired cognitive and behavioral changes in ADD and LD children. Lubars described a demonstration project (conducted in Knox County, Tennessee and surrounding area schools) in which 37 ADD children treated with EEG biofeedback to increase beta and decrease theta (in addition to resource classroom training) were compared to 37 matched controls (who received resource classroom training alone for reading disabilities associated with ADHD). Those undergoing EEG biofeedback demonstrated significantly greater improvements in Grade Point Average (GPA) and on their Metropolitan Achievement Test scores than controls and continued to show improvements of better than 1.5 GPA levels over control subjects one year post-treatment. More recently, Lubar, Swartwood, Swartwood, and O’Donnell trained 23 children and adolescents with ADHD to decrease theta waves by either reinforcing decreased theta or increased beta activity over a two- to three-month summer program. All participants were trained under identical treatment protocols and none were on medication for the duration of the study. Twelve of nineteen subjects showed significant decreases in theta across sessions with no significant differences having been found in pre-treatment levels of theta across subjects. In
addition, those individuals who demonstrated theta reductions (EEG change group) evidenced a significantly greater improvement in TOVA test performance, a visual continuous performance task. Specifically, they showed improved functioning across three of four TOVA scales as opposed to those who did not exhibit such EEG changes (no change group). The EEG change group also demonstrated a significant elevation in post-treatment WISC-R Verbal, Performance and Full-Scale IQ scores. The significance of these findings though are compromised by the fact that no analyses were reported comparing the pre- and post-treatment WISC-R scores of the no change group. Finally, significant behavioral differences were not found between the EEG change and no change groups possibly, according to the authors, because parents may have overemphasized the positive gains made by their children when responding on subjective measures. Given that this was not a tightly controlled study, alternative explanations are also possible. As an example, perhaps the children comprising the no change group exhibited genuine behavioral improvement because of the individualized attention they were getting within the context of the treatment, or the fact that it was summer and they were relieved of school-related pressures. While the authors may indeed be correct in their explanation for the lack of behavioral differences between the change and no change group, the lack of experimental controls certainly opens up the possibility of alternative explanations.

Alhambra, Fowler and Alhambra\textsuperscript{43} reported that 30 of the 36 ADD/ADHD children and adolescents they had trained with EEG biofeedback showed significant overall improvements across behavioral measures, quantitative EEGs and TOVA scores, with significant correlations being found between changes in EEG and TOVA score elevations. Moreover, of the 24 subjects treated with medication, five were completely removed from medication, eleven were maintained on a reduced dose and four, though on the same dose, showed greater overall improvement on that dose following training. In another study, Pratt, Abel and Skidmore\textsuperscript{44} trained 19 ADD or ADHD subjects to decrease theta and increase beta in the presence or absence of background music. They reported that even though pre- and post-test ratios of theta and beta were not significant, to varying degrees, all subjects were subjectively rated as having improved their focusing capability, impulsivity, social skills and control of moods, with 70\% of individuals maintaining these gains six months post treatment. Furthermore, children with ADD trained in the presence of background music were reported to have achieved even greater gains in focusing behavior. Given that pre-and post-test ratios of theta and beta were not significant, it is unclear as to what the results of this study signify other than the possible efficacy of background music in ameliorating some of the symptoms which typify ADD and ADHD. Lastly, Linden \textit{et al.}\textsuperscript{6} compared two groups of children with ADD/ADHD and, in some cases, learning disabilities, who were randomly assigned to either forty sessions of training to suppress theta and enhance beta activity (over a six month period) or a waiting list/control group. None of the children were undergoing any other form of treatment for the duration of the study. The authors found that while neither of the groups were significantly different in terms of IQ, inattention, overactivity or aggressive-defiant behavior prior to treatment, the experimental group showed a significant increase on their K-BIT IQ scores (average increase of 9 points), improved attention, and a non-significant decrease in hyperactivity as compared to controls following treatment. It should be noted that, while the average increase in K-BIT IQ scores
represents statistical significance within the context of this study, it only represents an actual increase of less than one standard deviation, thus perhaps limiting its functional significance. In addition, because of methodological difficulties, the authors did not analyze their EEG data, thus precluding their ability to correlate the improvements found with conditioned brain wave changes.

Adding to the limited number of follow-up reports, Lubar\textsuperscript{11} described a retrospective telephone survey completed on 52 patients, 50\% of which were assessed between one and ten years following treatment. Sixteen items from the Conner's Parent-Teacher Questionnaire were utilized to assess results. The greatest changes found were in areas including homework, improved grades, family interactions and attitude. The authors also reported that virtually all parents interviewed had attributed these changes to EEG biofeedback training, though no information was provided as to whether subjects had been treated with SMR or theta/beta training. While encouraging, we do not know whether the demand characteristics of a telephone survey may have influenced parental response, nor whether the confounding effects of history itself were contributory. Specifically, perhaps intervening remedial and behavioral strategies which followed the original biofeedback treatment were responsible for the gains cited.

In the above reports, only four provided information concerning the attainment of intended theta/beta alterations during training. One study\textsuperscript{42} reported that twelve of nineteen individuals were able to decrease theta, another described an improvement in EEG activity\textsuperscript{43} and the last,\textsuperscript{44} a non-significant difference between pre- and post-theta/beta ratios. In terms of behavioral and cognitive changes, increases in focusing capabilities, social skills, family interactions and attitude as well as significant increases in IQ and academic performance were evident. The authors also reported non-significant decreases in hyperactivity. One investigation\textsuperscript{43} also noted that a large proportion (20 of 24) of the individuals in their study who were on medication had their doses decreased or eliminated altogether following training.

Although the data are interesting, one cannot firmly establish a direct link between theta/beta biofeedback and the changes reported because of the lack of EEG data in some studies\textsuperscript{6,18} as well as non-significant EEG changes cited in others.\textsuperscript{44} Still, two controlled studies\textsuperscript{6,18} demonstrated that experimental subjects showed significantly greater behavioral and cognitive changes than control subjects. In addition, two of the works cited indicated that none of their subjects had been on medication\textsuperscript{42} or any other form of treatment\textsuperscript{6} during biofeedback treatment.

Three studies provided follow-up data\textsuperscript{6,18,44} in which cognitive and behavioral changes were reported to have been maintained up to six months, one year, and even ten years post-treatment for a proportion of subjects trained.

\textbf{ALPHA BIOFEEDBACK}

Four studies were located pertaining to the application of alpha biofeedback with children having ADD and LD, three of which trained subjects to increase alpha while the last trained them to decrease or attenuate alpha. O'Malley and Connors\textsuperscript{45} trained a dyslexic adolescent to unilaterally increase alpha production in the left hemisphere and beta or theta in the right over a five-day period. While the subject did demonstrate
increased levels of alpha as compared to baseline, ratings of corresponding changes in behavioral and cognitive functioning were not obtained. Unfortunately, while demonstrating the ability to train unilateral brain wave changes, the functional concomitants of such changes was not addressed. In addition, Nall investigated the association between increased alpha levels and amelioration of overactive and maladaptive behaviors and overall achievement in hyperactive children with learning disabilities. She utilized three matched groups ($n = 16$), an experimental group which was provided with correct alpha feedback, a control group given false feedback and a no-treatment control group. Following biofeedback, she reported that similar numbers of subjects in the experimental and false feedback groups (9 of 16 and 7 of 16) achieved increased alpha levels and that, though there was a trend in favor of the experimental group, no behavioral differences resulted between any of the three groups. It was also reported that, in terms of achievement measures, the experimental group significantly outperformed the other two groups in reading comprehension alone.

Gracenin and Cook also investigated the utilization of alpha biofeedback training in achieving improved oral reading and reading comprehension scores in LD children. Experimental subjects ($n = 9$) received a ten-minute training session to increase alpha productivity one time/week for ten weeks. The investigators reported that, following treatment, most subjects evidenced gains in alpha amplitudes and duration which were accompanied by slight improvements in hyperactive and maladaptive behaviors, as well as significant improvements in comprehension as compared to controls.

Lastly, Jackson and Eberly trained five mentally challenged individuals between the ages of 22 and 29 to suppress alpha while engaging in a cognitive task across ten sessions spaced over two weeks time. A significant attenuation in alpha ensued in conjunction with an increase in correctly answered problems and attention as indexed by head turning responses. Though the population utilized in this experiment is a great deal older and diagnostically different than those usually studied within this context, this study was included because it demonstrates that even those individuals who may be mentally challenged can be trained to alter brain wave patterns within an EEG biofeedback training paradigm.

In reviewing these few studies of alpha biofeedback training, a proportion of individuals were shown to have successfully increased alpha. Slight to no differences in hyperactive behaviors though were reported and enhanced reading comprehension abilities proved to be the only significant cognitive change. Unfortunately, no follow-up data were supplied. Because of the scant number of studies found, the limited number of subjects (total $n = 31$ for all four studies combined), diagnostically mixed subject populations, and the small though significant changes in alpha, no links or conclusions can be drawn about the efficacy of this approach with LD and/or ADD children at this time. Furthermore, in terms of alpha attenuation biofeedback, though all mentally challenged subjects involved in the one study cited successfully decreased alpha (in conjunction with increasing attention and correctly answered questions), assessing the efficacy of this approach needs to be delayed until additional research with sufficient numbers of subjects and control groups accumulates which is specific to the ADD and LD populations.
HEMISPHERE-SPECIFIC BIOFEEDBACK

Five studies were also located which applied unilateral or bilateral biofeedback to alter brain waves in the cerebral hemispheres of LD and ADD children/adolescents. Murphy and Darwin trained three groups of LD adolescents who manifested cerebral dysfunction and accompanying depression and significant (15 points or greater) verbal-performance IQ score differences. Subjects were placed into either an alpha, beta or no training (control) group. Training occurred over ten sessions and targeted the left hemisphere, specifically the left occipital-temporal region. It was reported that alpha training resulted in such affective changes as reduced anxiety, improvements in emotional disinhibition, and increased expressed warmth as well as enhanced arithmetic performance on the Wide Range Achievement Test (WRAT). These same authors also trained one individual who evidenced right cerebral dysfunction and a significantly depressed performance relative to verbal IQ score (30 points) with thirty-five sessions of varied EEG biofeedback which resulted in a reduction of right hemispheric frequencies, an elimination of his cerebral brain wave asymmetry, a significant performance IQ elevation, and academic improvement. Similarly, Patmon and Murphy trained hyperactive and LD adolescents to increase or decrease dominant left occipital-temporal EEG frequencies in eight 21-minute sessions and found that increased frequency training was accompanied by corresponding dominant EEG alterations and reduced muscular tension (EMG), but no changes across hyperkinetic or cognitive indices. On the other hand, while decreased frequency biofeedback resulted in improved reading scores, no behavioral or physiological (including EEG) changes were noted. Cunningham and Murphy tested the effectiveness of bilateral EEG biofeedback with twenty-four LD adolescent boys evidencing cerebral dysfunction. They defined such dysfunction as verbal IQ scores that were at least twelve points lower than performance IQ scores. The authors divided the sample into three groups, training one group to increase dominant EEG frequencies in the right hemisphere while decreasing dominant frequencies in the left and a second to decrease dominant frequencies in both hemispheres over eight weekly twenty-one minute sessions. A third group, which acted as a control group, received no training. Following treatment, both biofeedback groups showed decrements in dominant left hemisphere EEG frequencies, but no changes in dominant right hemisphere frequencies. In addition, no cognitive changes ensued besides an improvement in arithmetic performance evidenced in those individuals trained to increase dominant frequencies in the right hemisphere while decreasing those in the left hemisphere.

Lastly, Carter and Russell utilized left hemisphere training to alternately produce alpha or beta activity in three LD boys with depressed verbal WISC-R IQ scores and one with depressed performance IQ scores in biweekly sessions over a period of eight weeks. Though statistical analyses were not attempted due to the small sample size and limited data, the authors reported that the three subjects with depressed pretreatment verbal IQ scores showed an elevation in their verbal scores as well as a reduction of their verbal/performance discrepancies. On the other hand, the single subject who demonstrated depressed performance IQ scores showed no change following treatment. In addition, Russell and Carter applied EMG and EEG hemisphere specific beta biofeedback to LD diagnosed individuals and found
significant elevations in verbal IQ accompanying left hemisphere training and performance IQ accompanying right hemisphere training.

In terms of attaining intended EEG changes with this approach, results are varied. For example, one study reported a single case to have successfully decreased right hemispheric frequencies and eliminated cerebral asymmetry following biofeedback. Another described both their experimental groups as having attained a decrement in dominant left hemispheric frequencies, but no intended alterations in right frequencies. Finally, another study reported attainment of increased dominant left hemisphere frequencies and decreased EMG in their increased frequency group, but no physiological changes (EEG or EMG) for those in the decreased frequency group.

With respect to cognitive and behavioral changes, decreased anxiety and increased expressed warmth as well as elevated arithmetic, reading and verbal/performance IQ scores have been reported following hemisphere-specific biofeedback training, although no follow-ups were reported. Though significantly positive results were reported in a number of these studies, links between achieved gains in hemispheric-specific biofeedback as well as conclusions regarding the efficacy of this approach cannot be made at this time because of the small number of studies found, the variability in resulting EEG alterations, heterogeneity of treatment approaches utilized (i.e., area and EEG activity targeted), and limited sample sizes. It should be noted that most of the work cited focused on adolescents as opposed to children with ADD and LD.

**SUMMARY AND CONCLUSIONS**

While anecdotal and case study reports cite promising evidence, no definitive conclusions can be drawn about the efficacy of enhanced alpha and hemisphere specific biofeedback training as an effective technique in treating ADD, ADHD, or LD. While desired EEG alterations were achieved by a proportion of individuals trained, minimal to no corresponding behavioral or cognitive changes emerged, with some studies even failing to measure such changes. This is further compounded by the small number of studies found utilizing this approach, the general lack of control groups, no follow-up data, and mixed diagnostic subject pools. In general, the efficacy of alpha biofeedback as a rehabilitative technique with ADD and LD individuals has yet to be studied in a scientifically rigorous fashion.

The hemisphere specific biofeedback studies cited report variable degrees of positive EEG, cognitive and behavioral findings. However, a paucity of studies employing this technique, coupled many methodological shortcomings, severely limits any positive conclusions as regards this therapeutic approach. Perhaps future rigidly controlled studies will prove hemispheric specific biofeedback to be useful, however, at present, it should be considered a highly experimental procedure.

A promising treatment technique for this population involves theta/beta biofeedback training. Given evidence of the association between increased beta and cognitive functions such as attention as well as between theta activity and drowsiness, a treatment paradigm which involves increasing beta while decreasing theta activity has inherent face validity. Studies have reported significant academic, intellectual and behavioral gains having been attained with this approach, even in the absence of other
treatment modalities, such as medication. Follow-ups have also provided some evidence for the longevity of results, although the number of follow-up studies are rather limited. In order to link these gains to biofeedback and to draw more definitive conclusions, more controlled research utilizing these techniques is necessary. Moreover, this research needs to be guided by stricter methodologies including larger and more homogenous populations, unified treatment protocols, valid and reliable outcome measures, follow-up reports, etc.

The approach with the most published research \( (n = 7) \) located through our Psychlit and Medline searches involves SMR biofeedback training. For the most part, subjects were shown to successfully increase SMR production and to exhibit significant behavioral and cognitive changes during and following SMR training, with a reversal of these gains being evident during a counterconditioning procedure. In several investigations, these changes were shown to be superior to those attained by alternative treatments including medication and resource classroom instruction and to be maintained even when these alternative treatments were discontinued. While the links between achieved gains and SMR biofeedback training is the strongest amongst the treatment paradigms reviewed, a paucity of controlled studies utilizing this approach continues to hinder wider acceptance of this procedure. Similarly to theta/beta biofeedback then, further research endeavors with stricter methodologies needs to be conducted within this treatment paradigm.

**SUGGESTIONS FOR FUTURE RESEARCH**

Future work investigating the efficacy of SMR, theta/beta, alpha and hemisphere specific biofeedback techniques with ADD and LD children as well as adolescents and adults is obviously necessary. Such work needs to include larger sample sizes, more homogeneous populations and treatment protocols, control groups and follow-up reports. The cost effectiveness of EEG biofeedback treatment relative to standard treatment options, including medication, also needs to be investigated. Research endeavors which utilize promising but, as of yet, unsubstantiated biofeedback procedures including alpha attenuation training with ADD and LD individuals may also prove fruitful. Furthermore, as Linden et al.\(^6\) suggest, investigations consisting of a multiple group design which compare children with ADD or LD treated with EEG biofeedback with a control group as well as those receiving other forms of treatment (e.g., behavior modification) would be of critical import in assessing the efficacy of these techniques versus other available treatment options. We would like to see future studies which employ double blind, placebo controlled designs with serial longitudinal follow-up at one, three, five, ten, and even fifteen years in order to firmly establish the efficacy of EEG biofeedback training as a treatment technique in ameliorating symptoms of ADD.

While random assignment to experimental versus control groups is logistically easier to accomplish than matching samples, the very limited sample sizes in many of the cited studies makes matched samples a more robust methodological approach. In addition to matched samples, such studies need to focus on children, adolescents, and adults, as the possibility of maturationally mediated brain wave changes needs to be considered. The establishment of consistent standardized procedures across
studies, including electrode placement, type of brain wave activity reinforced, response, as well as the number, frequency and length of treatment sessions criteria is also imperative for further research and clinical endeavors.

Yet another area in need of further investigation involves the underlying mechanisms of EEG brain wave formation. Influences on brain wave activity which have been proposed include excitatory and inhibitory postsynaptic potentials, thalamocortical relays, brainstem reticular formation relays, amongst others. The possible effects of individual variability on EEG biofeedback training must also be subjected to further research as certain EEG patterns may portend different underlying processes across individuals. As Walsh states, significant variability exists in respect to subjective reports associated with EEG alpha biofeedback training.

It may very well be that EEG biofeedback treatment represents an efficacious treatment option for individuals who suffer from ADD. However, its acceptance as a viable alternative to, or augmentation of, standard intervention with medication remains to be seen. Unfortunately, EEG biofeedback is often “perceived” as esoteric, much in the same way that acupuncture was once frowned upon by the medical establishment. In order to assure this perception, those proponents in the forefront of the EEG biofeedback research movement must increase the amount of supportive data obtained within the context of rigidly controlled studies. Once accomplished, it may well lead to wider acceptance of EEG biofeedback as an efficacious treatment modality for individuals afflicted with ADD and ADHD.

ACKNOWLEDGMENT

The authors would like to thank Dr. L. Eugene Arnold for reading this manuscript and providing helpful suggestions.

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Clinical Utility of EEG in Attention Deficit Hyperactivity Disorder

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Electrophysiological measures were among the first to be used to study brain processes in children with attention deficit hyperactivity disorder (ADHD; Diagnostic and Statistical Manual of Mental Disorders [4th ed.], American Psychiatric Association, 1994) and have been used as such for over 30 years (see Hastings & Barkley, 1978, for an early review). More recently, electroencephalography (EEG) has been used both in research to describe and quantify the underlying neurophysiology of ADHD, but also clinically in the assessment, diagnosis, and treatment of ADHD. This review will first provide a brief overview of EEG and then present some of the research findings of EEG correlates in ADHD. Then, the utility of EEG in making an ADHD diagnosis and predicting stimulant response will be examined. Finally, and more controversially, we will review the results of the most recent studies on EEG biofeedback (neurofeedback) as a treatment for ADHD and the issues that remain to be addressed in the research examining the efficacy this therapeutic approach.

Key words: EEG biofeedback, diagnosis, treatment, neurotherapy

Electroencephalography (EEG) measures reflect the correspondence between intracranial electrical currents and the resulting voltages on the scalp reflecting certain facets of brain electrical function and processing, such as how electrically active various brain regions are and how responsive they may be to stimuli or during cognitive tasks. Early EEG studies found that children with attention deficit hyperactivity disorder (ADHD) exhibit EEG abnormalities such as excess slow wave activity and epileptiform spike and wave activity (Satterfield, Cantwell, & Satterfield, 1974). These findings were interpreted as indicating abnormal brain processes among children with ADHD, specifically a maturational delay marked by underarousal. Recent advances in technology have resulted in more accurate quantification of EEG activity by allowing computation of amplitude and power values for specific frequency bands of activity, source localization, and brain electrical activity mapping. Electrophysiological techniques (event-related potential [ERP] and EEG) are non-invasive, are less sensitive to movement artifact, do not include radioactive isotopes, and offer excellent temporal resolution (EEG can measure changes in the brain to the millisecond). The spatial resolution (i.e., where the EEG signal is coming from), however, is sometimes difficult to determine because electrical currents recorded from the cortex do not always bear a direct relation to any specific underlying brain structure and are affected by many sources of electrical artifacts.

When examining EEG activity, scientists and clinicians often look at the activity within a specific frequency band. Frequency refers to the number of oscillations (or cycles) within a given time period (e.g., four cycles per second). Note that EEG waveforms are a mixture of several different frequency bands, which are transformed and quantified for further analysis. In addition, although it is possible to decompose the EEG signal into different frequency bands, they are part of a dynamic milieu that acts in concert. Thus, certain cognitive or behavioral characteristics have been associated with a frequency band, but it is also the relationship among frequencies in other areas of the brain that produce complex behaviors. Details about the specific frequency bands are presented in the table below along with a brief summary of some findings concerning ADHD and its subtypes.

In addition to looking at activity in the individual frequency bands, theta/beta and theta/alpha ratios have also been examined and are thought to reflect level of
cortical arousal and maturational delay, respectively. Though these may seem redundant measures of the individual frequency bands, they have been proposed to be a better way to capture the relative levels of these off-setting brain activation patterns (Monastra, Lubar, & Linden, 2001).

**EEG Correlates in ADHD Children**

**Comparisons of ADHD and Normal Children**

Early reviews of studies of electrophysiological measures collected on hyperactive or ADHD children concluded that the disorder was most likely associated with problems of underreactivity to stimulation and task demands with less evidence supporting resting underarousal in the disorder (Hastings & Barkley, 1978; Rosenthal & Allen, 1978). Recent studies have helped to support, clarify, and further refine these early studies (for a comprehensive review of EEG findings, see Barry, Clarke, & Johnstone, 2003). Current research findings show that most children with ADHD display fairly consistent EEG differences in brain electrical activity when compared to normal children, particularly regarding frontal and central theta activity, which is associated with underarousal and indicative of decreased cortical activity (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998, 2001a; El-Sayed, Larsson, Persson, & Rydelius, 2002; Lazzaro et al., 1998). In the largest EEG study of ADHD to date (with a sample of over 400 children), Chabot and Serfontein (1996) found that children with ADHD displayed increased theta power, slight elevations in frontal alpha power, and diffuse decreases in beta mean frequency. Increased theta power is the most consistent finding in this ADHD EEG literature, indicating that cortical hypoarousal is a common neuropathological mechanism in ADHD.

It has also been suggested that the theta/beta ratio is associated with cortical arousal that has also been shown to consistently differentiate between ADHD and normal samples (Bresnahan & Barry, 2002; Clarke et al., 2001a; Monastra et al., 2001). Furthermore, this measurement has been shown to be stable over time; EEG recording from a single electrode at the vertex (Cz) yielded that a 1-month reliability of the theta/beta ratio was .96, $p < .05$ (Monastra et al., 2001). The theta/beta ratio has been found to discriminate between individuals with ADHD and normal controls across the age range (Bresnahan, Anderson, & Barry, 1999) and theoretically makes sense given that frequency bands are part of a milieu rather than occurring in isolation.

Aside from the findings for theta and the theta/beta ratio, results for other frequency bands such as beta and alpha have been more variable among children with ADHD. The findings for beta (indicative of heightened cortical arousal) activity have been less consistent, with several studies finding decreased beta activity in frontal and central regions (Chabot & Serfontein, 1996; Clarke et al., 1998, 2001a; Lazzaro et al., 1998; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992) and others not (Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995; Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Satterfield, Schell, Backs, & Hidaka, 1984). There also appears to be a small group of ADHD children (~15%) who show an excess of beta activity (Chabot & Serfontein, 1996; Clarke et al., 2001c). With the exception of slightly elevated levels of temper tantrums and moodiness, this group of children appears to be behaviorally similar to other ADHD children, although they are more likely to be ADHD–Combined Type (Clarke et

<table>
<thead>
<tr>
<th>Frequency Band</th>
<th>Delta</th>
<th>Theta</th>
<th>Alpha</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Associated feeling states</td>
<td>&lt; 4</td>
<td>4–7</td>
<td>8–12</td>
<td>&gt;13</td>
</tr>
<tr>
<td>Findings in ADHD</td>
<td>Mixed findings: Increased in some ADHD, normal or decreased levels in others</td>
<td>Increased in frontal and central area, continues into adulthood</td>
<td>Mixed findings: May depend on age, gender, or subtype</td>
<td>Decreased in some but not all ADHD children, may normalize with in adults</td>
</tr>
<tr>
<td>ADHD subtype</td>
<td>Increased in ADHD–Combined Type</td>
<td>Increased in ADHD–Combined Type</td>
<td>Increased in ADHD–Inattentive Type</td>
<td>Decreased in ADHD–Combined Type</td>
</tr>
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Note: ADHD = attention deficit hyperactivity disorder.

**Table 1. Summary of EEG Frequency Bands and ADHD**

<table>
<thead>
<tr>
<th>Frequency Band</th>
<th>Delta</th>
<th>Theta</th>
<th>Alpha</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cycles per second (Hz)</td>
<td>&lt; 4</td>
<td>4–7</td>
<td>8–12</td>
<td>&gt;13</td>
</tr>
<tr>
<td>Associated feeling states</td>
<td>Sleep, unconscious</td>
<td>Drowsiness, unfocused</td>
<td>Eyes closed; relaxed, but alert</td>
<td>Mental activity, concentration</td>
</tr>
<tr>
<td>Findings in ADHD</td>
<td>Mixed findings: Increased in some ADHD, normal or decreased levels in others</td>
<td>Increased in frontal and central area, continues into adulthood</td>
<td>Mixed findings: May depend on age, gender, or subtype</td>
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</tr>
</tbody>
</table>

Note: ADHD = attention deficit hyperactivity disorder.
EEG Differences Among ADHD Subtypes

Another possible explanation for the variability in EEG findings may lie in patterns of EEG activity according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM–IV], American Psychiatric Association, 1994) ADHD (Inattentive, Hyperactive–Impulsive or Combined) subtypes. Subtype differences, however, have not been well studied, with only a handful of groups examining this question. Chabot and Serfontein (1996) looked at different groups of children with attention disorders (normal, attention problems [but subthreshold ADHD], ADHD) and found that differences were more quantitative than qualitative, with the children having the most severe symptoms showing the greatest EEG abnormality. Another group that has systematically studied subtype differences found that children with ADHD–Combined Type (CT) exhibited more absolute and relative theta, and higher theta/alpha and theta/beta ratios when compared to those with ADHD–Inattentive (I) type (Clarke et al., 1998, 2001a; Clarke et al., 2003). The differentiation between ADHD subtypes by the theta/beta power ratio was not independently replicated (Monastera et al., 2001); thus more study is required. Nonetheless, these results suggest that it is the ADHD–CT children who show the classic pattern found in earlier studies of greater underarousal and maturational delay than ADHD–I children. In contrast, ADHD–I children exhibited more relative alpha in the posterior regions than those with ADHD–CT, which is consistent with reports of slower cognitive processing and increased rates of daydreaming among these kids. Developmental studies suggest that there are actually two distinct components in ADHD that may be quantifiable with EEG. The first is a hyperactive–impulsive component that appears to normalize with increasing age and the second is an inattentive component that does not normalize with increasing age and therefore represents a deviation from normal development (Barry et al., 2003). This hypothesis is consistent with the clinical phenomenology of ADHD, where the hyperactive symptoms often decrease substantially with age, but inattention and disorganization remain problematic longer into development (Hart, Lahey, Loeber, Applegate, & Frick, 1995).

It is likely, however, that the use of the DSM–IV approach to ADHD subtyping will not be the most fruitful means of clinical description since diagnosis does not inform treatment or predict treatment response (Pelham, 2001). Furthermore, the different ADHD subtypes are based on behavioral criteria with no consideration of underlying pathophysiology. An alternate way of classifying subgroups of ADHD children may be according to their EEG patterns, which may reflect CNS abnormality. In studies examining whether there are distinct EEG defined subgroups of ADHD children, studies have found that both the ADHD–CT and ADHD–I have at least two distinct clusters with similar EEG profiles: a hypoaroused group and a maturational lag group (Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002). Further work is needed to replicate these subgroups in other ADHD samples and to determine whether this approach to ADHD diagnosis can aid in treatment response and prediction. These findings may be consistent with research that suggests that a subset of ADHD children (perhaps 30%–50%) now classified as Inattentive Type may have qualitatively different problems with attention, cognitive, social, and academic functioning as well as treatment response profiles (Milich, Ballentine, & Lynam, 2001). This subset is referred to as having Sluggish Cognitive Tempo (SCT; McBurnett, Piffinner, & Frick, 2001) and some have suggested that it may constitute a separate, distinct disorder from ADHD. More work is needed to determine whether one of these EEG defined subgroups is associated with the SCT subgroup.

EEG Findings Among Adolescents and Adults With ADHD

Studies that have examined developmental trends among normal and ADHD individuals have also documented EEG abnormalities across the lifespan in ADHD samples. Developmental studies of EEG among normal samples have found decreasing theta and increasing beta activity with age, with alpha activity initially increasing into adolescence and then decreasing into adulthood (Bresnahan & Barry, 2002; Gasser, Verleger, Bacher, & Sroka, 1988; John et al., 1983). The theta/beta ratio also decreases with increasing age among normal samples (Bresnahan et al., 1999;
Similar to children with ADHD, adolescents with ADHD had higher levels of theta activity and higher theta/beta ratios across development, which remained abnormally high into adulthood. Beta activity was also significantly reduced among adolescents with ADHD when compared to normal samples; however, this normalized into adulthood, except in posterior locations (Bresnahan et al., 1999; El-Sayed et al., 2002; Hermens et al., 2004). Thus, these findings have led some to hypothesize that frontal–central theta activity is related to impulsivity and frontal–central beta activity with hyperactivity. This might mirror the clinical phenomenology of decreased gross motor activity as ADHD children get older; however, at the level of behavior ratings, hyperactivity and impulsivity form a single dimension of child behavior, not two distinct domains as suggested by these results (Achenbach, 2001; DuPaul, Power, Anastopoulos, & Reid, 1999). Attentional systems in the posterior regions of the brain (Levy & Swanson, 2001; Mirsky, 1996; Posner & Dehaene, 1994) may be related to EEG abnormalities in the alpha or beta bands seen in parietal regions.

**Diagnostic Utility of EEG in ADHD**

**Sensitivity and specificity of EEG.** To study the diagnostic utility of any new instrument (in this case EEG), one should compare its ability to correctly identify those with a diagnosis (made with the “gold standard”) and those with no diagnosis. Sensitivity tells you what percent of ADHD children have an abnormal EEG and specificity tells you what percent of non-ADHD children have a normal EEG. In several studies, the EEG has demonstrated good sensitivity (90%–97%) and specificity (84%–94%; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Monasta et al., 2001; Monasta et al., 1999). This means that, when you have a group of children with ADHD, a high percentage of kids will have a corresponding abnormal EEG (increased theta or high theta/beta ratio); in a comparison group of children with no ADHD, a high percentage of those children will have a normal EEG (lower levels of theta or lower theta–beta ratio). But more important from the standpoint of clinical diagnosis is positive (PPP) and negative predictive power (NPP). PPP tells you whether an abnormal EEG can correctly predict which children will receive a diagnosis of ADHD and NPP tells whether a normal EEG correctly predicts who will be normal or non-ADHD. The PPP and NPP for EEG have been reported to be 98% and 76%, respectively (Monasta et al., 2001), meaning that when there is an abnormal EEG (high theta/beta ratio in this case) it is highly likely that the child is ADHD. However, when the EEG is within the normal range, 24% of those children go on to be diagnosed as ADHD using other clinical methods. Most clinicians would consider this to be an unacceptably high rate of misdiagnosis for clinical purposes. Furthermore, a two-group comparison (ADHD vs. normal) of EEG diagnostic validity is not the most appropriate way to examine predictive power because most ADHD children referred to clinics have at least one if not two other comorbid disorders. Thus, the issue facing the clinician is not whether the referred case is disordered or normal, but rather, which disorder or set of disorders the case manifests among the various possible disorders (e.g., learning, depression, anxiety, etc.) occurring in a clinical practice.

**Differentiating ADHD and other comorbid disorders.** More instructive, therefore, are studies examining whether EEG can discriminate among ADHD, learning disorders, and other psychiatric disorders. Chabot and Serfontein published two papers (Chabot et al., 1996; Chabot & Serfontein, 1996) reporting discrimination between normal children and those with learning disabilities (LD) and ADHD. When comparing an ADHD sample to a normative database of normal and LD children (John et al., 1983), EEG was sensitive (93%–97%) and fairly specific (84%–90%) in differentiating ADHD from LD. Including the possibility that children may be normal lowers these values considerably with correct classification rates of 76% of normals, 89% of ADHD–ADD, and 70% of LD children (Chabot et al., 1996). There are also methodological issues that may affect the generality of these results. Children were diagnosed ADHD using only parent and teacher behavior rating scales, which may have led to misdiagnosis. In addition, the ADHD sample was compared to a normative database where LD children were not specifically screened for ADHD and included a very broad and heterogeneous group of children with learning difficulties (either low IQ or normal IQ with an achievement score less than 90).

When large samples of ADHD and LD children were directly compared, the discriminant validity of EEG appear high enough to be potentially useful (Chabot & Serfontein, 1996). Though the classification of ADHD alone and LD alone was good (97% and 84%, respectively), classification of ADHD children with and without learning disorders was not reliable (i.e., a split half replication was less than 60%). The best initial classification they obtained was 65% of ADHD only and 70% of ADHD + LD children. Although these classification
rates are significantly higher than chance, they still result in unacceptably high rates (20%–35%) of misclassification and therefore misdiagnosis. These studies do not support the clinical utility of EEG alone in differentiating between ADHD patients with and without learning disabilities.

In addition to learning disorders, most ADHD children referred to clinics have at least one if not two other comorbid psychiatric disorders, such as other disruptive behavior disorders, depression, anxiety, and substance abuse disorders (Barkley, 1998). It is hard to anticipate how these other disorders might affect the EEG measures and their capacity to discriminate ADHD from normal cases as well as those involving other disorders. More systematic work in this area is needed. To date, all of the studies conducted thus far have examined children with ADHD who do not have other comorbid psychiatric conditions or where the proportion of comorbid disorders goes unspecified. Similarly, none of the studies to date have examined whether EEG can differentiate or accurately classify children having the different ADHD subtypes. Until EEG research addresses its utility in this context of diagnostic comorbidity, it should not be used clinically in the diagnosis of ADHD.

**EEG and medication response.** Relatively few EEG studies have examined medication response among ADHD children. ADHD children who are medication responders have been reported to have excessive slow wave activity (Clarke, Barry, McCarthy, & Selikowitz, 2002; Satterfield et al., 1984), supporting the theory that ADHD children are cortically hypoaroused. In addition, stimulant medication appears to “normalize” the EEG patterns and evoked potentials of children with ADHD (Jonkman et al., 1997; Verbaten et al., 1994; Winsberg, Javitt, & Silipo, 1997) and to decrease slow wave EEG (theta) activity and increase fast wave (beta) activity depending on the task and electrode location (Clarke, Barry, Bond, McCarthy, & Selikowitz, 2002; Loo, Teale, & Reite, 1999; Lubar, White, Swartwood, & Swartwood, 1999; Swartwood et al., 1998). Using EEG alone or a combination of behavioral and EEG measures, several studies have reported correct identification of 70%–80% of stimulant responders (Chabot et al., 1996; Prichep & John, 1992; Suffin & Emory, 1995). Similar predictive power rates (PPP and NPP ~70%) have been reported using ERP components such as the P3 (Sangal & Sangal, 2004). Though this may seem to be a relatively impressive predictive power of the EEG for predicting medication response, it is actually no better than the base rate one would have guessed in the absence of any EEG information. Research repeatedly finds that ~70% of ADHD children placed on a single stimulant demonstrate a positive response (Barkley, DuPaul, & McMurray, 1991; Cantwell, 1996; Findling, Short, & Manos, 2001). Unless the EEG can significantly surpass the prediction from the base rate, its utility in this respect is unimpressive.

**Summary**

Collectively, the EEG findings in children, adolescents, and adults with ADHD are increased slow-wave activity in frontal regions, suggesting cortical hypoarousal, especially in the ADHD Combined subtype. Several researchers have reported that EEG measures discriminate well between children with and without ADHD and others have asserted that the EEG works well in determining medication responders from nonresponders. There is preliminary evidence that EEG can differentiate ADHD subtypes, at least at the group level of comparison, but the requisite information on accuracy of individual classification is lacking.

Our conclusion, then, is that EEG alone, if used for diagnosis or prediction of treatment (i.e., stimulant) response, results in unacceptably high rates of misdiagnosis and misclassification. Although rates of 70%–80% classification are interesting at the research level and may be comparable to other assessment tools alone (e.g., rating scales or computerized tests), in a clinical setting, it means that 20%–30% of children will not receive the correct diagnosis or treatment. This suggests that the use of diagnostic instruments such as a structured or semistructured clinical interview, well-standardized behavior rating scales of ADHD symptoms, and information collected from multiple sources (parent, teacher, child) are still required. Because such measures must still be collected in evaluating anyone for ADHD, regardless of whether an EEG has been conducted, the EEG findings remain an interesting but nonessential piece of information in the diagnostic process. Though these findings indicate some promise for EEG as a diagnostic tool, additional systematic research to empirically validate its classification accuracy is needed.
EEG Biofeedback (Neurofeedback): The EEG as Treatment Device

Given the excess of theta and decreased beta activity observed among children with ADHD, it is easy to understand the theoretical basis for examining whether altering these problems through treatment would result in improvements in ADHD symptoms. This is the basic goal of EEG biofeedback, neurofeedback, or neurotherapy—to train the patient to decrease their slow wave activity and/or increase their fast wave EEG activity, often using behavioral principles such as operant conditioning (i.e., positive reinforcement) in the process. Typically, a neurofeedback therapist places one to three electrodes on the patient’s head, which are connected to a computer. The computer detects the EEG information and provides a visual or auditory display of activity in the targeted frequency band(s). When the person is producing the desired EEG pattern (there are differential training programs for alpha or theta reduction and sensorimotor rhythm [SMR] or beta increase), the computer will give a positive response or reward, usually in the form of points earned. The person is then given a reward (e.g., money or other reinforcers) for earning a certain amount of points within each session. After many sessions of training, between 20 and 50 as currently practiced, it is hypothesized that a person will be able to produce the desired EEG brain waves on their own through increased awareness of their own physiological processes. Such conditioned EEG changes have been reported to be associated with improved or normalized symptoms of ADHD, to generalize outside the treatment setting (such as at home, school, or work) even when the treatment is withdrawn (Monastra, Monastra, & George, 2002) and to be maintained into adulthood in most treated cases (Lubar, 1991). Of note is the fact that no other treatment approach for ADHD has been able to demonstrate such generalization or maintenance effects (Pelham, Wheeler, & Chronis, 1998; Smith, Barkley, & Shapiro, in press).

This treatment has stirred up quite a controversy between the clinical and scientific communities working with ADHD. Recent reviews of EEG biofeedback have generally concluded that preliminary studies of EEG biofeedback are promising, but require further study in rigorous scientifically controlled studies (Arnold, 2001; Nash, 2000; Ramirez, Desantis, & Opler, 2001). Proponents of EEG biofeedback feel that their studies have been overly criticized and that the scientific community has been unfairly biased against neurofeedback treatment, despite large numbers of participants who have reportedly experienced positive outcomes. Critics of EEG biofeedback, however, contend that the published studies have suffered from significant methodological weaknesses that make interpretation of the results and conclusions about the actual effect of EEG biofeedback impossible.

Many of these flaws were identified a decade ago by Barkley (1992) and the same problems with scientific methodology that existed then continue to exist with these newer studies. The flaws included no control groups, the confounding of several different treatments within the EEG biofeedback group, use of small numbers of participants, diagnostic uncertainty about the children in the study, lack of placebo control procedures, absence of blindness of the evaluators to the treatment received by the cases, and practice effects with the measures being used to evaluate the ADHD children. Crucial yet lacking in most studies of EEG biofeedback has been the randomized assignment of cases to treatment and no-treatment (or placebo) groups. Instead, treatment groups are often constructed retrospectively from a series of clinical cases that have been previously treated or not with EEG biofeedback. Furthermore, there may exist a conflict of interest in these findings because EEG biofeedback studies are typically conducted by clinicians who are being paid to provide the treatment and are published in neurotherapy journals that do not have rigorous peer review. As Chambless and Hollon (1998) pointed out in their guidelines for defining empirically supported therapies, treatment efficacy must be demonstrated in controlled research where it is “reasonable to conclude that benefits observed are due to the effects of the treatment and not to chance or confounding factors such as the passage of time, the effects of psychological assessment, or the presence of different types of clients in the various treatment conditions.” Thus, these are not petty or simply annoying issues that can be ignored. They are central to any demonstration of treatment efficacy. In the following paragraphs, we will review the most recent controlled studies of EEG biofeedback and offer a summary of where the state of EEG biofeedback lies currently.

EEG Biofeedback Versus No Treatment

The first controlled study was completed by Linden, Habib, and Radjojic (1996) and utilized small samples of ADHD patients; nine cases were randomly assigned to receive EEG biofeedback and nine were placed on a wait-list. Importantly, no other treatment was provided simultaneously including stimulant medication. The dependent measures included an IQ test
and two parent rating scales of ADHD symptoms and aggression. The neurofeedback group showed a significant increase in IQ and a significant decrease in parent ratings of inattention. There was no significant effect of EEG biofeedback on hyperactive–impulsive or aggressive behavior ratings.

This study is often cited as support for EEG biofeedback and does incorporate some important methodological controls such as random assignment, wait-list control, and treatment integrity. Noteworthy, however, is that no pre- and posttreatment comparisons in EEG power were reported to show that the treatment had altered the EEG parameters associated with ADHD. Also important to consider is that (a) no placebo control group was used to control for therapist time, attention, and other demand characteristics of the treatment environment; (b) parents evaluating the children before and after therapy were not blind to the condition (nor were the children); and (c) the improvement on the IQ test is irrelevant to the demonstration of efficacy of this treatment for ADHD. IQ is not a measure of ADHD. Just as important, the overall or omnibus statistical analysis (multivariate analysis of variance, or MANOVA) of IQ and behavior ratings reported in this article did not find a significant effect of treatment group but rather a nonsignificant “trend” for time (pretreatment to posttreatment), meaning that all children, regardless of whether they had treatment or not, showed similar levels of improvement from the pretreatment to posttreatment evaluations. Though this may have been due to low power because of the small sample size, follow-up univariate tests of a nonsignificant MANOVA are not recommended and increase the risk of Type 1 (false positive) error (Weinfurt, 1998).

EEG Biofeedback Versus Placebo Biofeedback

Most EEG biofeedback studies suffer a glaring oversight and that is the failure to incorporate a placebo control condition. There have been many reasons put forth for not using a placebo control, such as difficulty designing a sham biofeedback that is not detectable by clinicians and patients, ethics of giving a placebo for 6 months when other effective treatments are available, and feasibility of doing a placebo control condition within the context of a private clinical practice, which is where these studies have been conducted. Nonetheless, there is no other way to control for the effects of patient–therapist time, expectations generated by applying electrodes and being connected to a computer, ancillary support given to parents, and motivation and investment needed to complete treatment.

Only one study has used a placebo control group and is noteworthy for the degree of scientific rigor in its design. This was the unpublished paper by Fine, Goldman, and Sandford presented at the American Psychological Association meeting in 1994. In this study, 71 patients were randomly assigned to biofeedback, a no-treatment wait-list control group, or a placebo control group involving computerized cognitive training protocol. The authors collected 51 different measures, including 30 lab measures and parent ratings. Examiners doing the testing were blind to the treatment group assignment of these children; however parents were not. There were significant group differences on 12 measures, eight of which came from parent ratings. Of the four lab measures, just one favored the biofeedback group whereas the other groups did better on the remaining three. On the parent ratings, both treatment groups exceeded the wait-list control group on eight subscales from the three global rating scales. The biofeedback group was slightly better than the placebo group on two scales whereas the opposite was the case on the third rating scale, that being the Child Behavior Checklist (Fine, Goldman, & Sandford, 1994). In what is the most methodologically sound study on EEG biofeedback treatment outcome, using random assignment to groups and a placebo control group with examiner blindness to treatment assignment, no compelling evidence of efficacy for EEG biofeedback was evident.

Additionally, Heywood and Beale (2003) employed a single-subject design with a placebo control condition applied to a small sample of children (N = 7). The effects of EEG biofeedback were contrasted with a placebo (noncontingent) feedback condition. Outcome measures included parent and teacher behavior ratings as well as several cognitive tests (auditory and visual continuous performance tests, or CPTs, paired associate learning task, and verbal fluency task) during each of the conditions. Behavioral ratings and performance on cognitive tasks during active and placebo feedback conditions were compared and the results appear to support the effects of active EEG biofeedback on the dependent measures. These effects disappear, however, when controlling for overall trend of the data (which helps to account for maturation and nonspecific treatment effects) and including treatment noncompleters (known as an intent-to-treat design). Furthermore, the effects of active and placebo biofeedback do not result in changes in the treatment outcome measures that differ significantly from baseline measures. Thus, one might mistakenly conclude that there is a significant
treatment effect of EEG biofeedback only if maturation and nonspecific effects as well as treatment noncompleters are ignored.

Overall, of the three treatment outcome studies comparing EEG biofeedback to either no-treatment or placebo control conditions, two fail to support an active treatment effect. These studies are, methodologically speaking, the three strongest. Though the small sample sizes in the Linden et al. study may have limited statistical power for comparisons, the Fine et al. study had large sample sizes providing sufficient statistical power to detect differences between conditions had they been present.

EEG Biofeedback Versus Other Treatments (Medication and Psychological)

There have been three studies that have compared neurotherapy to other treatments, all including psycho-stimulant medication that is the gold standard in treatment for ADHD. If EEG biofeedback treatment demonstrated treatment effects that are similar (or not significantly inferior) to stimulant medication treatment, this might be taken as an indicator of equivalence in efficacy (Chambless & Hollon, 1998). Unfortunately, none of these studies used random assignment. Instead, they reconstructed their treatment groups after the fact of treatment (months or years) using samples of clinically treated patients or allowed patients to self-select into the treatment they preferred. Also, these studies failed to report psychiatric or learning disorders that often are comorbidities with ADHD, and did not incorporate evaluators who were blind to the patient’s treatment condition. In addition, only one study tested EEG biofeedback by itself without confounding it with additional treatments.

Rossiter and LaVaque (1995) were the first to compare EEG biofeedback to stimulant medication in groups (23 in each group) of children and adults with ADHD (ages 8 to 21 years). Rather than randomly assigning cases to each treatment group, the authors matched the cases of those who previously received EEG treatment against those who had received stimulant therapy (ages 5–45 years) using age as a matching criterion. Again, the absence of random assignment to treatment groups is an important methodological oversight here because such randomization helps to minimize inherent biases such as self-selection into the various treatments and experimenter bias in choosing the patients in treatment group assignment that confound efforts to draw conclusions from group comparisons. The patients were assigned to treatment groups based in part on their preferences, in part on whether they had previously failed stimulant therapy, and in part on insurance coverage for biofeedback. Furthermore, medication (to five of the EEG cases) and additional treatments were provided to all cases, confounding the two treatment groups and making interpretation of individual treatment effects (medication and EEG biofeedback) impossible. The authors reported using the Test of Variables of Attention (TOVA), a continuous performance test assessing inattention and impulsiveness, and a parent rating scale of behavioral problems, though not the same one for all participants. Cases and their parents were not blind to their treatment condition, nor were the examiners testing the cases on the lab measures blind to such assignment. Also, no information is provided as to just how these cases were selected from the larger pool of clients likely treated in this practice. Were all available cases within a specified period of time reclassified into these post hoc treatment groups or just some? If not all, why were some chosen for inclusion in these analyses and others not?

From pre- to posttest, the EEG biofeedback group showed significant improvement on the TOVA and on parent ratings of inattention, hyperactivity, and internalizing symptoms. So did the medication group, with no differences between them in the degree of change shown. Important to note is that, here again, pre- and posttreatment EEG measures were not reported so as to show that the biofeedback had changed the important parameters of the EEG believed to mediate the changes in ADHD symptoms. Although the authors conclude that, for children who do not respond to medications, EEG biofeedback is a good treatment choice, the significant scientific design problems (i.e., absence of random assignment to treatments, confounding of treatments, and lack of reported EEG changes) prohibit making such a conclusion.

In their study on EEG biofeedback, Monastra et al. (2002) reported results from samples of 51 ADHD children (6 to 19 years old) who received comprehensive clinical care (CCC; medication, parent counseling, academic support) with EEG biofeedback for 1 year and 50 who had received CCC alone (no biofeedback). Again, patients were not randomly assigned to the treatment groups. The fact that groups were found to not differ on pretreatment scores on either the measures of ADHD (ratings) or EEG measures is not very reassuring given that many other variables can operate to bias treatment studies such as this one absent random assignment to treatment groups before initiating therapy. Results of the study indicate that children in the CCC + EEG bio-
feedback (CCC + B) group were better at posttreatment on behavior ratings of attention and hyperactivity–impulsive behaviors (on and off medication), as well as on the TOVA (only when tested off medication), when compared to the CCC group. In addition, at posttreatment those in the EEG group had lower theta/beta ratios than the CCC group. These results indicate improved functioning in the CCC + B group even when off medication; however, the significant differences are primarily due (surprisingly) to virtually no improvement in the CCC group. Close examination of the pre- and posttreatment behavior rating scale scores (on and off medication) indicate that the CCC alone group appear to have received a degraded version of the CCC or are treatment nonresponders. This atypical patient group as a comparison coupled with the lack of random assignment, variation in individual treatment components, failure to control for the amount of time spent with a therapist, and lack of information as to just how patients were chosen from the larger treated pool prohibits interpretation concerning efficacy of any specific treatment component (EEG biofeedback without all of the other treatments).

The Fuchs et al. (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003) study is the only study thus far that involves a direct comparison between EEG biofeedback (N = 22) and stimulant medication (N = 11) where the treatments are not confounded (i.e., stimulants given to the EEG biofeedback group). As with the previous studies, the sample description lacks important information regarding ADHD subtype and psychiatric or learning disorder comorbidity. In addition, the readministration of the WISC intelligence test within such a short time period (12 weeks) invalidates the results of the posttreatment test. Methodological issues (no random assignment, no control for additional therapist time, small sample size, no information on the larger pool of treated patients from which these cases were selected and why) notwithstanding, these results may suggest that EEG biofeedback and methylphenidate result in similar levels of short-term change in ADHD behaviors. Yet those methodological issues are crucial to being able to say anything about such a treatment effect. Replication of these findings with increased scientific controls and larger sample sizes (at least 25–30 respondents per condition; Chambless & Hollon, 1998) will be a necessary step toward establishing EEG biofeedback as an equivalent treatment to medication. To demonstrate that EEG changes are responsible for treatment effects, reporting of actual EEG changes and correlation with treatment outcome must be shown.

Is EEG Conditioning the Active Ingredient in Biofeedback?

The reason we come back over and over again to scientific methodology is that proper experimental controls makes it possible to discern whether training EEG patterns is the active ingredient in the treatment. In fact, one of the biggest issues that the EEG biofeedback treatment literature needs to address is whether or not it is actually the training of the EEG patterns that leads to improvement in ADHD symptoms. Though the goal of EEG biofeedback is the “unconscious conditioning of underlying neurological systems [to] learn balance through reinforced practice” (Monastra, 2004), none of the studies thus far have demonstrated that the EEG changes are the actual mediator of treatment outcome. In an earlier EEG biofeedback study (Lubar, Swartwood, Swartwood, & O’Donnell, 1995), ~60% of children showed EEG changes with biofeedback treatment. The children who showed EEG changes (decreased theta) also exhibited significantly greater improvement on the TOVA (three of four scales improved) when compared to those whose EEG did not change (one of four scales improved). And yet, there are significant overall treatment effects in several studies. This indicates that other nonspecific or unintentional factors are present in the treatments that are helping bring about behavioral and cognitive improvement. But if it isn’t EEG change, what else might be at work to elicit the behavioral and cognitive improvements reported in these studies?

First, there are several nonspecific factors that may result in ADHD symptom improvement. Children in EEG biofeedback conditions, based on the study descriptions, received additional time with a therapist ranging from 17 to 40 hours across studies than did cases not receiving biofeedback. Failure to control for the amount of treatment time means that the EEG biofeedback group may have improved simply because they spent more time with a therapist, are more invested in treatment and therefore more motivated to change, or may have more stability and support from mental health professionals rather than the EEG biofeedback per se, or may simply have been more likely to want to please the therapist.

Another possibility is that biofeedback is simply another form of cognitive–behavioral training that just happens to employ the use of electrodes placed on the head. Under this scenario, it is not anything to do with the electrodes or EEG that necessarily produces the treatment effect. Instead, it is whatever conscious cognitive or behavioral actions the individual is actively
employing to alter the EEG activity that is being conditioned. For instance, in some studies, children are told to focus their concentration on some object or on some imagined condition, such as being a heavy rock. In others they are told to find some mental activity that results in a change in their performance of the videogame being used to give them feedback about their EEG status. In others, they are told simply to try to do better at the videogame. Even advocates of EEG biofeedback concede that “attentional training through behavioral methods cannot be ruled out” (Linden et al., 1996) and that the “factors that are essential in teaching attention/concentration remain an empirical question” (Monastra, 2004). Noteworthy here is that cognitive behavioral training has not been found to be effective in treating ADHD (DuPaul & Eckert, 1997; Pelham et al., 1998). Yet, rarely have children had to perform this sort of sustained practice (for 30–50 hr) and received such salient rewards (up to $150) for successful performance. Research studies have repeatedly shown that ADHD children’s performance on cognitive tests can be normalized with immediate and salient reinforcers (Firestone & Douglas, 1975; Oosterlaan & Sergeant, 1998). This is also likely to lead to stimulus generalization when children come into the lab for posttreatment EEG and TOVA assessments (Heywood & Beale, 2003). Thus, when children are completing the posttreatment session, they may be expecting similar rewards for performance; these behaviors may not continue, however, in other settings and if performance is not continually rewarded. Similarly, it has been suggested that altered breathing patterns may minimize theta activity, which may be a separate but correlated mechanism for treatment effects thought to be the result of EEG biofeedback (Heywood & Beale, 2003). This is consistent with some neurotherapy treatment protocols that encourage the patient to relax, which most likely leads to deeper breathing and increased oxygenation of blood cells in the brain. Perhaps it is the conditioning of deeper breathing and therefore increased cerebral perfusion that improves ADHD symptoms.

If it is not the reinforced conditioning of EEG activity per se, then the use of computers, electrodes, and amplifiers is unnecessary, similar to what was found for the EMG treatments in the 1970s—teaching muscle relaxation proved sufficient. This should lead to a less expensive, more targeted treatment focused on the active ingredient of this treatment. If, as proponents of EEG biofeedback state, it is the EEG conditioning that produces the balance among the underlying neurological systems, then additional studies are needed to demonstrate this specific effect. While EEG biofeedback studies with seizure patients have demonstrated correlation between EEG changes and clinical symptomatology (Sterman, 2000), this has not yet been demonstrated in ADHD.

Summary

Although the existing studies of EEG biofeedback claim promising results in the treatment of ADHD, the promise of EEG biofeedback as a legitimate treatment cannot be fulfilled without studies that are scientifically rigorous. Undoubtedly, treatments for ADHD would benefit greatly from a nonmedication alternative that is efficacious and cost effective. But there is much work to be done to demonstrate that EEG biofeedback provides that alternative and that actually changing the EEG is the mechanism of change in ADHD symptoms (as opposed to just more time with a therapist). Without such demonstrations, the changes in behavior cannot in fact be attributed to this specific treatment mechanism. It must also be shown that treatment effects can generalize to nontreatment settings and can persist over time. Even with such demonstrations it must also be shown that treatment is cost effective in managing the symptoms of ADHD relative to the prevailing empirically supported approaches.

Future Directions for Research on the Clinical Utility of EEG in ADHD

If EEG is to be used as a diagnostic tool for ADHD, there has to be much greater clarity on its ability to differentiate ADHD from normal children, ADHD subtypes from each other, and to assess for differential diagnoses as well as ADHD comorbidities. Work documenting correlations between EEG and ADHD symptoms and subtypes is needed. Two studies (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001b) have identified EEG-defined subtypes within ADHD, with one group exhibiting a higher than normal beta power in both samples. Replication of these subtypes and greater description of how they relate to current diagnostic subgroups and treatment outcome seems warranted, particularly among the excess beta group. Though some work has been done to examine the diagnostic utility of EEG in ADHD, more systematic study needs to be done using rigorous diagnostic procedures (i.e., structured or semistructured diagnostic interviews), careful identification of comorbid diagnoses (including specific learning disorders) and impact of these disorders on EEG characteristics. In addition, studies examining EEG correlates of stimu-
lant response should incorporate double-blind medication titration and reporting of EEG differences according to varying doses of medication.

As for EEG biofeedback as a treatment for ADHD, there are clearly many issues that need to be addressed adequately in future research. The first and foremost is addressing the methodological problems that have plagued this treatment outcome research from the start. Proper scientific controls are crucial to demonstrating that there is a real treatment effect due to EEG biofeedback and that EEG conditioning is the effective ingredient within the treatment. This will require clinical trials that incorporate random assignment to treated and untreated groups, placebo conditions, larger sample sizes, evaluators that are blind to treatment condition, clear and comprehensive sample description (particularly with regard to psychiatric and learning disorder comorbidity), appropriate data analytic (statistical) procedures, and documentation of EEG changes that correlate with treatment outcome. These methodological difficulties compromise the internal validity of most of the studies reviewed here, making interpretation of the results and conclusions about the actual effect of treatment impossible.

Finally, side effects of EEG biofeedback must be monitored systematically and reported in studies. All truly effective treatments produce some side effects in some percentage of the population. This has to be so because individuals differ in their physiological makeup, particularly brain organization and functioning. Those individual differences are sufficient to result in the treatment producing adverse effects in a subset of the population. Moreover, clinical ineptitude in the delivery of the treatment in some cases and as a consequence of comorbid disorders in other cases always ensures that some patients will not respond well to the intervention as delivered. This is as true for behavioral interventions as it is for medications. Hence any claim that a treatment is effective yet has absolutely no associated side effects is oxymoronic. The former cannot exist without the latter. This may be a telling piece of information about whether neurofeedback is actually effective for the management of ADHD.

**Conclusion**

The clinical utility of EEG in ADHD has yet to be proven. Though there are some promising results that require further study, the threshold for using EEG clinically has not been met. Of the possible uses reviewed here (diagnostic utility, prediction of stimulant response, and EEG biofeedback), the diagnostic utility of EEG appears most promising although considerable work is needed for this promise to be realized. The EEG biofeedback studies with the most rigorous methodologies to date have not supported the efficacy of EEG biofeedback when compared to no-treatment control or placebo feedback. Methodological flaws of previous EEG studies have hampered firm conclusions regarding its usefulness and precision. Though the field of ADHD would benefit greatly from a single diagnostic test and an effective nonmedication treatment alternative, we cannot recommend the use of EEG in a clinical setting based on the current empirical data.

**References**


The effectiveness of EEG-feedback on attention, impulsivity and EEG: A sham feedback controlled study

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a r t i c l e   i n f o

Article history:
Received 4 February 2010
Received in revised form 7 May 2010
Accepted 8 May 2010

Keywords:
Neurofeedback
EEG
Double-blind
Sham
Impulsivity
Attention
ADHD

a b s t r a c t

EEG-feedback, also called neurofeedback, is a training procedure aimed at altering brain activity, and is used as a treatment for disorders like Attention Deficit/Hyperactivity Disorder (ADHD). Studies have reported positive effects of neurofeedback on attention and other dependent variables. However, double-blind studies including a sham neurofeedback control group are lacking. The inclusion of such a group is crucial to control for unspecified effects. The current work presents a sham-controlled, double-blind evaluation. The hypothesis was that neurofeedback enhances attention and decreases impulsive behavior. Participants (n = 27) were selected on relatively high scores on impulsivity/inattention questionnaires (Barrat Impulsivity Scale and Broadbent CFFQ). They were assigned to a neurofeedback treatment or a sham group. (sham)Neurofeedback training was planned for 15 weeks consisting of a total of 30 sessions, each lasting 22 min. Before and after 16 sessions (i.e., interim analyses), qEEG was recorded and impulsivity and inattention was assessed using a stop signal task and reversed continuous performance task and two questionnaires. Results of the interim analyses showed that participants were blind with respect to group inclusion, but no trend towards an effect of neurofeedback on behavioral measures was observed. Therefore in line with ethical guidelines the experiment was ceased. These results implicate a possible lack of effect of neurofeedback when one accounts for non-specific effects. However, the specific form of feedback and application of the sham-controlled double-blind design may have diminished the effect of neurofeedback.

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Neurofeedback is a method with which individuals can learn to alter their brain activity by means of feedback of this activity and rewarding of appropriate brain activity. Neurofeedback may be a potential treatment for Attention Deficit Hyperactivity Disorder (ADHD) [23]. However, no solid sham feedback (fake feedback) controlled studies have been conducted aimed at evaluating the effectiveness of neurofeedback on alleviating core symptoms of ADHD. The inclusion of a sham-control group is crucial to control for unspecified factors and to be able to estimate the effectiveness of neurofeedback above placebo.

Different neurofeedback protocols do elicit specific EEG changes and positive effects on behavior [2]. In healthy subjects, Egner and Gruzelier [12] showed that uptraining Sensory Motor Rhythm (SMR) (12–15 Hz) was associated with increased perceptual sensitivity and a decline in commission errors on the Conner’s Continuous Performance Test (CPT), suggesting increased sustained attention and decreased impulsivity. High beta uptraining (15–18 Hz) resulted in the opposite effect. In a second study, Egner and Gruzelier [11] also found protocol specific effects. They concluded that increasing SMR results in a general attention enhancing effect and 16–20 Hz training results in an arousal enhancing effect. In a study of Vernon et al. [27], SMR uptraining and theta training were investigated in healthy volunteers. The effect of SMR training on attention was replicated; however, in contrast to SMR training, theta training did not affect the EEG, working memory performance, or measures of attention.

Neurofeedback has been investigated as an alternative treatment for ADHD. In these patients, anomalies have been found on EEG measures of brain activity. In short, one group of patients shows decreased fast wave beta (12–22 Hz), less alpha (8–12 Hz), and increased slow wave theta (4–7 Hz) activity [6,8]. The other group presents an excess of beta activity [7]. The effectiveness of...
neurofeedback in reducing ADHD symptoms has been assessed in several studies. A review of Monastra et al. [24] summarizing research on (1) SMR enhancement and theta (4–8 Hz) suppression, (2) SMR enhancement and high beta suppression (22–30Hz), and (3) beta-1 (16–20Hz) enhancement and theta suppression shows results comparable to methylphenidate [23]. Using fMRI in children with ADHD, Lévesque et al. [18] showed normalization of activity in the anterior cingulate cortex (ACC) during a cognitive control task following neurofeedback as compared to a waiting list. The ACC is an area implicated in deficiencies in the control of attention. Furthermore, two recent studies showed that neurofeedback, using theta/beta and SCP feedback, was superior to a computerized attention skills training in reducing ADHD symptomatology [14,15].

Although previous studies strongly suggest a positive effect of neurofeedback in ADHD [2], these studies did not implement a double-blind sham-controlled design, which is necessary to assess whether neurofeedback training outperforms sham feedback training. Indeed, there are ethical concerns for implementing a sham control in clinical research if a standard treatment exists [16,28]. Several alternatives have been proposed [28]. One possibility is to investigate the effects of neurofeedback on a variable implicated in pathology using a sample of healthy subjects. In this way, a sham-controlled design does not violate ethical principles. In one such study, albeit not directly related to attention and impulsivity, the effect of neurofeedback on an EEG component (alpha/beta ratio), targeted in the adjuvant neurofeedback therapy for alcoholism, was investigated [13]. Specifically, a sample of healthy participants was used and a sham-controlled design was implemented to test the idea that alpha/beta feedback significantly modulates power within these frequency bands. Comparing real feedback with mock feedback, an effect was indeed found on EEG, but not on subjective measures.

The goal of the current study was to investigate with a similar sham-controlled design the specific effect of neurofeedback on measures of impulsivity and attention in healthy volunteers who scored relatively high on self-reported impulsivity and inattention. First, it was expected that neurofeedback would decrease inattention and impulsivity as assessed by the adult self-report questionnaire and the state impulsivity questionnaire, respectively. Secondly, it was expected that a decrease in inattentiveness and impulsivity would also be evident using objective measures. Specifically, neurofeedback, relative to the sham group, should reduce the standard deviation of reaction time (SDRT) in the R-CPT (reflecting sustained attention [19,20]), the stop signal reaction time (SSRT) in the Stop Signal Task (SST), and the false-alarm rate in the R-CPT (both related to impulsivity). Lastly, it was expected that neurofeedback would have an effect on the power in the trained frequency bands.

This study was a collaboration of Utrecht University with the Neurofeedback Institute Nederland B.V. To account for electro-physiological heterogeneity, individualized protocols were used. A double-blind, sham-controlled between-subjects design was chosen. The study was approved by the Utrecht Medical Center, Medical Ethical Committee and conducted in accordance with the declaration of Helsinki. Medical Ethical Committee (IB) guidelines were to cease the experiment if no trend towards improvement in behavior was found at the interim analyses (after 16 sessions).

To estimate the sample size for this study group × time effect sizes (cohen’s d) were calculated based on a study by Egner and Gruzelier [11]. For behavioral variables, the group × time effect size was 1.25. Power analysis with d at 1.25, alpha at 0.05 and an average power of 80% renders a sample size of at least 24 participants (12 in each group) sufficient. Furthermore, to ensure a sample with enough room for improvement on attention and impulsivity variables, we selected healthy volunteers, who scored relatively high on rating scales of impulsivity (Barratt Impulsiveness Scale questionnaire; BIS [4]) and inattention (Broadbent’s Cognitive Failure questionnaire; CFQ [5]), from a sample of 455 psychology students at Utrecht University. Specifically, for both questionnaires a higher score denotes more problems and participants with a score of <1 S.D. below the average sample score on both questionnaires were excluded, to ensure a large enough sample size (>N=24) and a sample with room for improvement. Other exclusion criteria were (a history of) psychiatric disorders, epilepsy, and use of psychoactive drugs. Eventually, 29 participants with relatively high scores on the questionnaires (BIS: M=66.14 S.D.=6.66, CFQ: 50.52, S.D.=9.08) were included in the present study. After baseline measurements, two participants were excluded, one fell asleep during data acquisition, the other showed epileptiform activity in the EEG. Participants were randomly assigned to either the treatment (n=14) or sham group (n=13). To prevent group differences, participants were matched on handedness, age, gender, IQ, State Impulsivity Questionnaire (STIMP) score, SSRT on the SST, and SDRT on the R-CPT task. One participant from the sham group dropped out after 14 sessions for personal reasons not related to this study. In the final sample (n=26) the mean age in the sham group was 20.7 years (S.D.=2.72), one was male. For the treatment group the mean age was 21.1 years (S.D.=2.30), two were male. Participants received 3.50 euro per hour for participation.

The Adult Self-Report (ASR) [1], State Impulsivity (STIMP) Questionnaire and two computer tasks were used for baseline and interim assessment. The ASR questionnaire yields two scores, one for attentional problems (ATT score) and one as indication for ADHD symptoms (ADHD score). The STIMP was used to assess impulsivity. On all questionnaires, a higher score indicates more dysfunction. The computer tasks consisted of the R-CPT and stop signal task (SST). The R-CPT consisted of one uniquely randomized block of 608 trials comprised out of 16 letters (A, B, C, D, E, F, H, I, L, M, N, O, T, Y, Z, X); each black letter was presented at the center of the screen on a gray background for 1000 ms at a 1000 ms interval, and each letter occurred in total 38 times. The goal was to react as rapidly and accurately as possible by pressing a button on a pad after presentation of each letter except the X. Variables of interest were the standard deviation of mean reaction time (SDRT) on Go trials and the proportion of false alarms (i.e., responses to the X divided by total number of X stimuli). In the SST, participants were presented sequences of square-wave black-white grating presented on a gray background. The gratings were 6 × 6 in size and consisted of high or low fundamental spatial frequency, 3.62 cpd and 0.46 cpd respectively. After a fixation cross was presented for 500 ms a grating was presented for 750 ms. The duration from the end of presentation of a grating to the presentation of the fixation cross was variable between 1000 ms and 1250 ms. The task consisted of two types of trials, go trials and stop trials. In a go trial the grating was not followed by a tone and a response had to be made. Participants had to respond by pressing the right button when presented with a high frequency grating and the left button when presented with a low frequency grating. Stop trials were trials in which the grating was followed by a binaural tone (1000 Hz, 80 dB, and 400 ms in duration), and a response had to be withheld. In total 630 trials were presented in five blocks of 126 trials. Blocks consisted of a unique randomized sequence of trials per participant and contained 76 go trials and 50 stop trials. Each block had an equal amount of high and low frequency gratings. No more than three stop trials were presented consecutively. The task started with a practice block followed by the four experimental blocks. For reliable SSRTs, about 50% correct inhibitions need to be made, therefore the Stimulus Onset Asynchrony (SOA; i.e., go/stop interval) was adjusted after each block using a tracking algorithm [9,22]. Only in the first block the SOA was set at 250 ms. The Go-stop interval was jittered within a 250 ms range to prevent
response strategies [25]. Due to apparatus failure, the tone occurred unilateral for 12 participants; therefore this data was not used in the analysis of the SST. The SST main outcome is the Stop Signal Reaction Time (SSRT) which is thought to be a measure of motor inhibition [3,10]. The SSRT was calculated in accordance with De Jong et al. [10] and Tannock et al. [26].

For QEEG assessment, the Deymed Truescan 32 acquisition device was used. Recordings were done at 19 channels and a rate of 128 samples per second was chosen. 10/20 EEG caps were used and electrodes were referenced to linked ears. Impedances were kept around or under 5 kΩ. Each recording lasted approximately 9 min (eyes closed since norm data also consisted of eyes closed recordings). QEEG analyses needed for determining the individual treatment-protocols were conducted by the Neurofeedback Institute Nederland B.V. (NIN). Segments with eye movements and artifacts were removed. By comparing the individual QEEGs (after artifact rejection) to the Neuroguide database deviations from the norm in the EEG were identified (FFT maximal z scores). Individualized treatment-protocols (STOP/GO, Table 1) were based on this assessment and aimed at normalizing power within the frequencies at one specific location of interest. For each participant, the protocol could consist of feedback based on the real EEG signal or a simulated EEG signal. The simulated EEG signal consisted of a random signal relatively similar to real EEG and simulation provided similar feedback experience.

15 weeks of neurofeedback training consisting of 30 sessions, each lasting 22 min, were planned, but the experiment was ceased after 16 sessions (since no trend towards an effect of neurofeedback on behavior was evident in the interim analyses, and ethical guidelines were to cease the experiment). Before (Baseline) and halfway (interim, after 16 sessions) participants were assessed with respect to measures of attention and impulsivity and qEEG was recorded. Prior to the experiment and after receiving adequate information, participants were asked to sign the informed consent form. After baseline measurements, (sham)neurofeedback sessions were planned and proceeded as follows. Participants entered their planned twice a week attendance in an online calendar. It was advised to have one day between sessions. If a participant could only attend for one session in a week, they received three sessions the following week. Upon first arrival, the participant was instructed that the brightening of the movie screen and the audio clicks are good signs and that the learning process is mostly unconscious so no specific effort is needed. Afterwards the participant sat down on a comfortable chair in front of a 17-in. monitor, and locations were prepped with Nuprep. The Brainmaster Atlantis II module in combination with Brainmaster 2.5SE software was used for 22 min of (uninterrupted) sham) neurofeedback. All electrode impedances were kept under 6 kΩ. One electrode was placed at the scalp at the location of interest (Table 1) using Ten20 paste and a hair band. The reference was placed on the mastoid contralateral to the electrode at the scalp or at the right mastoid if the scalp electrode was placed on the middle of the scalp. The ground was placed on the opposite mastoid. EEG was recorded at 256 samples per second with a 50 Hz Notch filter. Online artifact rejection analysis was conducted with a threshold of 240 V. The 17-in. monitor and speaker sets were used for video/audio feedback. If power within the specific frequencies was above or below threshold, participants were rewarded with audio clicks and a brightening as opposed to darkening of the movie. Feedback thresholds were automatically and dynamically adjusted every 30 s to keep power 80% of time above or below threshold (depending on whether feedback consisted of up or down training). After 16 weeks of neurofeedback, the interim analysis took place within two weeks after the last session, subsequently, the experiment was ceased. Finally, participants were asked to indicate whether they thought they had received sham or real treatment.

Except for initial assessment by the NIN, eyes closed EEGs recorded at baseline and interim, were analyzed in Brain Vision Analyzer. A high pass filter of 0.5 Hz, 12 dB/oct, notch filter of 50 Hz, and low pass filter of 40 Hz, 12 dB/oct was used. The first and last parts of the EEG was discarded to remove possible artifacts; the segment of 20–500 s was used for analyses. This segment was divided in 2 s epochs resulting in a resolution of 0.5 Hz. Automatic artifact rejection was used with a difference criterion of 100 V, effectively removing artifacts and possible remaining eye movement (even though the recordings were eyes closed). A full segment baseline correction was performed to remove DC drift, and epochs were transformed to the frequency domain with Fast Fourier Transform. For each participant and session (baseline/interim), power within each specific frequency band (see Table 1) was calculated for each 2 s epoch. Since individualized protocols were used, effect sizes were calculated for the treatment group using the mean and standard deviation of power across segments for each individual participant, frequency band, and time point (baseline versus interim). Effect sizes were recoded (a negative value to positive and vice versa) for frequency bands in which power were expected to be reduced. Since multiple frequency bands were often targeted within participants, effect sizes were then averaged yielding one global effect size per participant (Table 1).

A Repeated Measures General Linear Model was chosen to test the interaction between the effect of the within-subjects factor time and that of the between-subjects factor group. Dependent variables were SSRT, SDRT and proportion false alarms, the STIMP score, and the ADHD and ATT score from the ASR. A nonparametric (chi2) test was used to test whether participants could guess to what group they were assigned. A one-sample t-test was used to test whether the effect sizes in the treatment group deviated significantly from zero.

With respect to behavioral data, there were no significant time × group interactions, or trends towards significance, for any of the dependent variables (SSRT, F(1,12)<1; SDRT, F(1,24)<1; false alarms, F(1,24)<1; STIMP, F(1,24)<1; ATT score on ASR, F(1,24)=1.790, ADHD score on ASR, F(1,24)<1). Furthermore, neurofeedback treatment did not seem to affect EEG. After the exclusion of one outlier (>2 S.D.), only a trend towards significance was evident (t(1,12)=2.098, p=0.058).

Irrespective of group inclusion, most participants thought they were in the sham group. For the treatment group, 10 out of 14 participants thought they received sham feedback. 10 out of 12 participants in the sham group thought they were in the sham group. Inclusion in group did not significantly influence guessing (chi2(1,26)=0.516, p=0.473).
In summary, the findings from the present double-blind, randomized, sham-controlled neurofeedback study in healthy volunteers could not confirm our hypothesis that neurofeedback outperforms sham feedback treatment in enhancing attention and decreasing impulsive behavior. Previous studies do suggest an effect of neurofeedback on measures of attention and impulsivity [2], but none of the studies conducted a double-blind, sham-controlled trial. This study incorporated a sham group-controlled double-blind design to control for unspecified effects and to investigate the effect of neurofeedback above placebo. Since no interaction proved to be significant at the behavioral level, there may be no treatment effect at all, or the effect is rather small and irrelevant. In other words, the present findings suggest that neurofeedback may have no effect on behavior when accounting for unspecified factors. This is in marked contrast to previous studies accounting for unspecified effects by applying different protocols within one sample [11,12,27].

Several factors may account for the contrasting results. One issue concerns the use of the double-blind sham-controlled design. In our study, most participants thought they received sham feedback irrespective of group inclusion. Part of the instruction to participants was that no specific effort was necessary for feedback to have an effect. In combination with quite liberal and continuously changing feedback thresholds, it may be possible that participants merely watched the movie. Similarly the movie itself could have distracted participants from the feedback process. Awareness of feedback may be necessary for neurofeedback to have an effect. In short, the specific application of the double-blind sham-controlled design may have diminished the effectiveness of the treatment. Future research should elucidate which format of feedback is most effective and whether awareness of treatment is necessary for training to have an effect. If future research indeed suggests that the double-blind sham-controlled design diminishes the effectiveness of neurofeedback, other designs should be considered. Indeed, several alternatives have been put forward [28]. If awareness is important in neurofeedback training, we would suggest a more direct and simple form of feedback devoid of distracting elements such as presumably in our movie-feedback. For example, Egner and Gruzelier [11] used simply an altering size of geometrical figures to represent power in a specific frequency band. In order to maintain a sufficient amount of awareness of an effect, the control group could receive feedback on muscle activity (electromyographic feedback).

Another limitation concerns the sample. Even though the participants scored relatively high on measures of inattention and impulsivity, the sample consisted of academic students. Perhaps insufficient room for improvement could partly account for the lack of effect. Also, since our sample consisted of healthy subjects, the results cannot be generalized to the clinical ADHD population. Subclinical impulsivity and problems with attention may differ from that of the clinical ADHD population. Lijffijt et al. [21], show that in contrast to the ADHD population, subclinical impulsivity does not correlate positively with decreased motor inhibition. Lansbergen et al. [17] found a lack of association between subjective impulsivity and motor inhibition as measured by the SST. Moreover, they showed that opposite to what is seen in ADHD patients, long SSRTs (indication of decreased motor inhibition) were associated with low theta/beta ratios. The authors argue that participants with higher theta/beta ratios may be more motivated to increase performance on the task.

Another issue is the use of individualized protocols in our electrophysiologically heterogeneous sample. Use of one standard protocol would have been better in terms of standardization, but would have necessitated, with respect to ethical principles, participant selection based on qEEG. For practical reasons it was chosen not to include this procedure. It could be that the effect of standard protocols used in previous studies is superior to our heterogeneity of protocols. Indeed, the effectiveness of many of our implemented protocols has not been evaluated yet in standardized research. Furthermore, we incorporated protocols aimed at inhibiting and protocols aimed at both inhibiting and enhancing power. It may be argued that there is a difference in effectiveness between these types of feedback. Unfortunately in our dataset it is not possible to separately assert the effect of specific protocols or only protocols aimed at inhibiting power, since the sample would become too narrow. There are two possibilities future studies could consider to resolve the issue with individualized protocols, and to successfully combine an individual approach with standardization. One possibility is to assert the effect of (limited) individualized protocols on an electrophysiologically heterogeneous sample of sufficient size, making it possible to assert the effect of specific protocols in associated electrophysiologically similar subsamples. Secondly, an electrophysiologically homogeneous sample may be selected through qEEG selection, and the effect of one specific protocol can be evaluated on that specific sample.

To conclude, the current results suggest a lack of effect of neurofeedback above placebo when one accounts for non-specific effects. However, the format of feedback in combination with the specific application of the double-blind sham-controlled design may have diminished the effect of neurofeedback.

Acknowledgments

This research was done in collaboration with the Neurofeedback Instituut Nederland B.V. I thank Drs. G. Loots, the psychophysiologist of the NIN for his help with the neurofeedback equipment and for providing the training in neurofeedback intervention.

I also thank the master students of Psychology under supervision of Prof. Dr. L. van Doornen: Z. Molnar-Logemann, BSc., Drs. M. Halfhuid, M. Laub, BSc., S. Leeuwen, BSc., for providing the (sham)neurofeedback sessions and research on the clinical questionnaires as part of their internships.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.neulet.2010.05.026.

References


