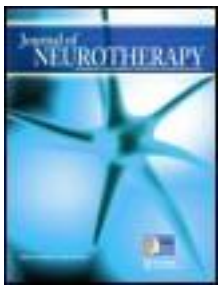

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Neurofeedback for AD/HD: A Ratio Feedback Case Study and Tutorial

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[a] EEG biofeed-back and the diagnosis and treatment of AD/HD

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SCIENTIFIC ARTICLES

Neurofeedback for AD/HD: A Ratio Feedback Case Study and Tutorial

Thomas Rossiter, PhD

ABSTRACT. *Introduction.* The case study of a 13-year-old AD/HD male treated with neurofeedback is the subject matter for a tutorial on Ratio feedback.

Method. Neurofeedback was conducted at C3 (increase 15 to 18 Hz, decrease 2 to 10 Hz) and C4 (increase 12 to 15 Hz, decrease 2 to 7 Hz). Protocols provided visual and auditory feedback based on the ratio of slow wave activity to be suppressed divided by fast wave activity to be enhanced (Ratio feedback).

Results. The patient demonstrated marked improvement in processing speed and variability on the Test of Variables of Attention-Auditory, a 19-point increase in IQ on the Kaufman Brief Intelligence Test, significant behavioral improvement based on parental (Behavior Assessment System for Children) and patient (Brown ADD Scale) reports, and a 7.5 grade equivalent increase in reading scores (Kaufman Test of Educational Achievement-Brief Form). At the 17-month follow-up parent questionnaires indicated that the patient's behavioral gains had been

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maintained or were slightly improved. EEG data showed significant declines in the C4/SMR Ratio (10*2 to 7 Hz/12 to 15 Hz) and 2 to 7 Hz amplitude, a tendency toward an increase in 12 to 15 Hz amplitude, a significant increase in 8 to 11 Hz amplitude, and a decline in 22 to 30 Hz amplitude. Beta activity (15 to 18 Hz) was unchanged. An unexpected finding was that C3/Beta (10*2 to 10 Hz/15 to 18 Hz) and C4/SMR protocols had similar effects on the EEG even though they targeted different bands to enhance and suppress. It appears that suppression of slow wave activity (2 to 7 Hz) may be the active component in both Ratio protocols and that fast wave enhancement either plays a minor (12 to 15 Hz) or no role (15 to 18 Hz).

Discussion. The findings cast doubt on the assumption that the C3/Beta and C4/SMR protocols have unique effects on EEG activity. Nevertheless, they may have differential effects on brain functions related to the training sites employed. It would be useful to analyze EEG changes in successfully treated individual AD/HD patients as a first step toward understanding the effects of various treatment protocols. What the protocols are intended to do, and the actual effects on the EEG may be different. If there are active components common to the various AD/HD treatment protocols reported in the literature, this is one way of beginning to recognize them. Brain maps collected before, during, and at the conclusion of treatment would enhance our understanding of treatment effects of various neurofeedback protocols, lead to more focused and productive research, and ultimately facilitate the development of more efficient treatment paradigms.

KEYWORDS. AD/HD, neurofeedback, ratio feedback, tutorial

INTRODUCTION

Rossiter and La Vaque (1995), using active control methodology (La Vaque & Rossiter, 2001), demonstrated that 20 sessions of EEG biofeedback effectively reduced the symptoms of Attention Deficit/Hyperactivity Disorder (AD/HD) in 19 of 23 patients. A matched stimulant drug group (methylphenidate, dextroamphetamine, or pemoline) achieved similar results with 20 of 23 patients showing significant improvement. The neurofeedback and stimulant drug groups showed comparable improvement on the Test of Variables of Attention (TOVA), a 22.5-minute

continuous performance test. Both of the groups improved significantly over pretreatment baselines on measures of attention (errors of omission), impulse control (errors of commission), processing speed (response time), and variability in attention (variability in response time). They exceeded the largest placebo response rate of 39 percent reported in double blind placebo studies of stimulant drugs (Barkley, 1990). Fuchs (1999) replicated the Rossiter and La Vaque (1995) study using random assignment of subjects and an active control group treated with methylphenidate. His results verify the Rossiter and La Vaque finding that neurofeedback is as effective as stimulant drugs in controlling AD/HD symptoms.

In addition to the two active control studies, outcome studies with samples as large as 1089 (e.g., Thompson & Thompson, 1998; Kaiser & Othmer, 2000; Kaiser, 1997; Lubar, Swartwood, Swartwood, & O'Donnell, 1995) have reported significant reduction in AD/HD symptoms with children, adolescents, and adults treated with neurofeedback. Both the active control and outcome studies assessed treatment effects using objective test data (e.g., Test of Variables of Attention, Wechsler Scales) and/or physiological data (Quantitative EEG) in addition to behavioral ratings. Although the AD/HD neurofeedback studies referenced above employed different neurofeedback equipment and protocols and treated patients of varying ages, they obtained very similar results. The combination of active control and large sample outcome studies offers compelling evidence that neurofeedback is an effective treatment for AD/HD and is sufficient to justify ongoing clinical use as additional controlled studies are conducted.

In spite of the evidence that EEG biofeedback is effective in ameliorating the symptoms of AD/HD, there is no consensus about which specific elements in the treatment have therapeutic value. EEG biofeedback to treat AD/HD has employed a variety of treatment protocols with different segments of the EEG spectrum being enhanced and/or inhibited. Reduction in slow wave activity (usually in the 2 to 8 Hz range) and/or increases in fast wave activity (usually in the 12 to 20 Hz band) are often hypothesized to be the key components (Lubar, Swartwood, Swartwood, & O'Donnell, 1995). However, to date there have been no published studies to test these, or competing hypotheses. In a single issue of the *Journal of Neurotherapy*, positive outcomes were reported using four very dissimilar protocols to treat AD/HD (Fenger, 1998; Ramos, 1998; Wadhwani, Radvanski, & Carmody, 1998) or to improve attention in a normally functioning individual (Norris, Lee, Cea, & Burshteyn, 1998). The treatment protocols described in the four articles

variously sought to increase 16 to 20 Hz, 13 to 15 Hz, 8 to 13 Hz, and/or 4 to 8 Hz activity while decreasing 4 to 8 Hz or 2 to 7 Hz activity. One protocol did not attempt to reduce slow wave activity.

A review of the literature on neurofeedback for AD/HD indicates that there is no evidence that one neurofeedback protocol is more effective than another. Treatment typically benefits 70 to 80 percent of those treated regardless of the specific equipment, treatment protocol, or patient population. The fact that a broad range of AD/HD neurofeedback protocols is effective in treating AD/HD suggests they may have as yet unidentified therapeutic element(s) in common. Research is needed to determine what specific elements make neurofeedback for AD/HD effective and how they can be used most efficiently. One potentially fruitful approach would be to closely examine the EEG changes that take place over the course of successful treatment with individual patients and determine whether the observed changes are consistent with the expected effects of the treatment protocols being used. That information might serve as a basis for determining which elements of the treatment protocols should be subject to further study.

The purpose of the following case study is to: (a) demonstrate the use of Ratio feedback protocols for AD/HD, and (b) provide a sufficiently detailed description of the author's assessment and treatment methodology and the underlying rationale to allow replication. One advantage of the individual case study is that it can provide a thorough account of clinical procedures and treatment outcomes leading to an in-depth understanding of the individual patient and the response to treatment. This type of information is lost in studies using averaged group data.

METHODS

Participants

The patient is a 13-year-old male initially evaluated when he was seven years old and in the first grade. At that time, he was diagnosed with Attention Deficit Disorder with Hyperactivity (DSM III-R, 314.01) and demonstrated reading, spelling, and mathematics skills that were significantly poorer than expected based on his grade placement and intelligence. Intelligence test results indicated that he was functioning in the Average range (Full Scale IQ = 101) but showed a 26 point higher Verbal than Performance IQ on the Wechsler Intelligence Scale for Children-Revised. This pattern is often observed among children diag-

nosed with a non-verbal learning disability (Rourke, 1989). The patient was treated medically with Ritalin until he was ten years old. His parents report that the medication was helpful in controlling the AD/HD symptoms. However, the patient experienced side effects including reduced appetite, slow growth, headaches, stomachaches, and increased frustration and anger. The Ritalin was ultimately discontinued due to the side effects.

When the patient returned at age 13, he was experiencing adjustment problems at home and at school. His parents reported that he was inattentive, distractible, impulsive, restless, quick to anger, impatient, and easily frustrated. The patient's impetuous behavior sometimes placed him in potentially dangerous situations. Concerns about his impulse control and quick temper prompted the parents to consider neurofeedback. At school, the patient received learning disability services for mathematics and language arts but not specifically for reading. He did not like to read, did only the minimum amount of reading required for completion of his schoolwork, and engaged in little or no recreational reading. The patient also had behavior problems at school related primarily to his restlessness and poor impulse control. Nevertheless, he was well liked by his teachers and peers. The patient suffered from eczema, particularly in the winter months. During previous winters, a rash spread over both legs and parts of his arms. He used a prescription lotion to control the discomfort.

Evaluation

The patient and his mother were seen for a diagnostic interview to update his history and review current symptoms. In addition, the pretreatment evaluation included the Kaufman Brief Intelligence Test (K-BIT), Test of Variables of Attention-Auditory (TOVA-A), Behavior Assessment System for Children (BASC), Brown ADD Scales, and the EEG Spectrum Structured Interview. The author's AD/HD assessment protocol called for re-evaluation after the twentieth and fortieth EEG biofeedback appointments. The re-evaluations included the TOVA-A, the Parent form of the BASC completed by the patient's mother, and the Adolescent Brown ADD scale completed with the patient. The K-BIT and the KTEA-Brief were re-administered after treatment was completed following the forty-fifth session of neurofeedback.

A current measure of intelligence was obtained in order to interpret the data from the Test of Variables of Attention-Auditory (TOVA-A). The TOVA-A was used to provide an objective measure of the cogni-

tive deficits associated with AD/HD and to assess response to neurofeedback midway through and at the end of treatment. The TOVA-A, rather than the visual TOVA, was administered because the visual TOVA results obtained during the diagnostic evaluation six years earlier had been within normal limits. The patient's TOVA-A performance was compared to the norms for males of his age and intelligence. In general for individuals of average intelligence, the author considers discrepancies of one standard deviation (Mean = 100, SD = 15) or more between intelligence test and TOVA scores to be clinically significant. Using this criterion, the patient demonstrated significant deficits in Processing Speed and Variability in Attention on the TOVA-A. The Attention score based on the number of targets missed, and the Impulsivity score based on the number of non-target stimuli responded to, were within normal limits. The TOVA-A results are consistent with, but not diagnostic of AD/HD. It should be recalled that while the TOVA and TOVA-A assess various aspects of the ability to pay attention, they are not tests of AD/HD per se. In order to establish the AD/HD diagnosis, it is necessary to confirm that the patient manifests not only the cognitive deficits but also the behavior patterns associated with AD/HD. Deficits in the ability to focus and maintain attention are not specific to AD/HD and are characteristic of many psychiatric disorders including anxiety and depression.

A standardized description of the patient's behavior was obtained using the Behavior Assessment System for Children (BASC). The patient's mother completed the Adolescent form of the BASC. The BASC results indicated that during the six months prior to the re-evaluation, the patient was extremely impulsive and restless, as well as markedly hostile in his dealing with others. In addition, the mother reported mild to moderate levels of acting out behavior, somatic complaints, and problems with focusing and maintaining attention. The patient was administered the Adolescent version of the Brown ADD Scales, an interview format symptom checklist with versions for adolescents (13 to 18 years) and adults (19 years and over). The results indicated that a diagnosis of an attention deficit disorder was "probable but not certain." The results of the pretreatment evaluation are consistent with a diagnosis of Attention Deficit/Hyperactivity Disorder, Combined type (DSM-IV, 314.01). A more detailed description of the evaluation process and the tests used can be found in Rossiter and La Vaque (1995) and Rossiter (1998).

The EEG Spectrum Structured Interview was completed using information provided by the patient's mother. The interview surveys:

(a) symptoms that the patient is currently or has previously experienced in the areas of attention, emotion, behavior, and sleep, as well as cognitive, immune, endocrine, autonomic, neurological, and motor functioning; (b) developmental history; (c) family (genetic) history; and (d) treatment history. Items in each category are classified according to the theory proposed by Othmer, Othmer, and Kaiser (1999) that many of the disorders being treated with neurofeedback are the result of disregulation of cortical arousal. They propose that these disorders can be understood as falling on a continuum of over activation, under activation, or unstable cortical activation. Treatment protocols based on the disregulation model are generally symptom driven. The information gathered from the Structured Interview is used in conjunction with the baseline psychological test data to determine the treatment protocol(s) to be used and the relative amount of time spent with each if more than one is indicated. For example, inattention, daydreaming, poor sustained attention, and lack of motivation are considered symptoms of left hemisphere under-arousal and suggest training at C3 (International 10-20 System) using an increase 15 to 18 Hz and decrease 4 to 7 Hz protocol (Othmer, 1999). By contrast, impulsivity, distractibility, and stimulus seeking are seen as symptoms of right hemisphere over-arousal and suggest training at C4 using an increase 12 to 15 Hz and decrease 4 to 7 Hz protocol (Othmer).

The disregulation model has considerable heuristic value. It provides a conceptual framework for understanding the relationship between a broad range of patient symptoms and cortical arousal. This allows the therapist to develop treatment protocols based on the totality of the patient's symptoms, whether or not they fall under a single diagnostic category. This is particularly important since a majority of AD/HD patients are diagnosed with at least one additional psychiatric disorder (e.g., Weiss & Hechtman, 1993). Thus, it is not uncommon for the practitioner to be treating an AD/HD child or adolescent who is also diagnosed with a learning disability, oppositional defiant disorder, anxiety, depression, and/or a conduct disorder. When treating AD/HD with a co-morbid depressive disorder, for example, the therapist needs to know what effect the AD/HD protocol may have on the depressive disorder and how to develop an integrated treatment approach that addresses both disorders.

One alternative to using the disregulation model is to develop a protocol based on the results of a Quantitative EEG assessment used in conjunction with a normative database (e.g., Thatcher, 1999). This is an attractive alternative because it allows the therapist to use a treatment

protocol specifically tailored to each patient's EEG. However, it requires that the patient's EEG be identified as "abnormal" in some respect in order to identify targets for remediation. In the long run, this may prove to be the preferred method but will require the development of normative databases for children and adolescents that incorporate a variety of cognitive challenges. A database that uses only eyes closed, resting EEG data may be less effective in identifying specific targets for training. A third alternative would be to use a combination of empirically based, and possibly incompatible, protocols developed to treat discrete disorders.

In this case, the disregulation model utilizing the patient's history and test data suggested that both left sided beta (15 to 18 Hz) and right sided SMR (12 to 15 Hz) training would be needed (Othmer, 1999). The need for left-sided training was suggested primarily by the patient's poor TOVA-A scores on measures of processing speed and variability in attention as well as behavioral reports of attention and motivational deficits. Right-sided SMR training was indicated principally by the patient's history of poor impulse control, hyperactivity, significantly lower Performance than Verbal IQ during the initial evaluation at age seven, and his tendency to be easily frustrated and angered.

Procedure

EEG data were acquired with a NeuroSearch-1620 digitizing EEG system (Lexicor Medical Technology, Boulder, CO) and a Pentium 200 MHz computer. A sampling rate of 128 Hz with two-second epochs was used. Twelfth order digital filters were used to define the steepness of the bands with the following bandwidths and default band analysis times: Delta 0.5 to 4.0 Hz (500 ms), Theta 4.0 to 7.0 Hz (500 ms), Alpha 8.0 to 11.0 Hz (250 ms), SMR 12.0 to 15.0 Hz (165 ms), Beta 15.0 to 18.0 Hz (125 ms), Beta2 22.0 to 30.0 Hz (79 ms), 2 to 7 Hz (500 ms) and 2 to 10 Hz (500 ms). Biolex version 2.38 software provided EEG biofeedback. Three (Grass E5GH) gold plated electrodes with 48-inch leads were used. The active electrode was placed at C4 or C3 (10-20 International System). The reference electrode was placed on the outside of the earlobe ipsilateral to the active electrode with the ground electrode on the outer surface of the contralateral earlobe. The reference and ground electrodes were mounted in plastic ear clips (Grass GRM-5636). The patient's skin was prepared according to the manufacturer's directions using a mildly abrasive skin prep gel (Nuprep). Skin impedance was less than 5 Kohms. The electrode cups were overfilled to form a

mound of conductive EEG paste (Ten20) and seated firmly against the skin. A cotton ball was placed over the active electrode to hold it in place.

The patient was seen three times a week for 45 treatment sessions over a period of four months. Generally, the author sees AD/HD patients for 40 office sessions of neurofeedback. However, when the patient was re-evaluated after 40 sessions, there were still indications of mild hyperactivity. At the author's suggestion, the patient and his parents agreed to an additional 10 sessions of neurofeedback. However, treatment was discontinued after the forty-fifth session at the patient's request.

Each treatment session consisted of two 18-minute biofeedback segments conducted in a well-lighted room. The patient was seated in an upright recliner facing a 17-inch computer monitor (Hitachi SuperScan Pro 620) three feet ahead of him. All training was conducted eyes open and the patient was provided with simultaneous visual and auditory feedback (Labtech LCS-1224 speakers). No cognitive challenges (e.g., reading, drawing, listening, etc.) were used during the training. At the end of each 18-minute segment, the cumulative numerical data and the graphs showing changes in the EEG during the segment were reviewed with the patient. Reductions in the magnitude and variability of the Ratios (10*2 to 7 Hz/12 to 15 Hz or 10*2 to 10 Hz/15 to 18 Hz) were considered the best indicators that the patient was learning to shift his level of cortical arousal in directions consistent with the intent of the training protocols. The session graphs were first viewed with a smoothing factor of one hundred to judge the general direction of changes in the Ratio and the component frequency bands. Ideally, the Ratio line declined over the 18-minute segment or initially declined and then flattened indicating that the heightened level of cortical activation was being maintained. A smoothing factor of ten was used to assess variability in the Ratio. The goal was to reduce the variability in the Ratio both within and between treatment sessions. Decreased variability was demonstrated by reductions in the range of the "peaks and valleys" in the Ratio graph. When viewing the graphs, the height of the Y-axis was set just above the highest elevation in the Ratio.

Treatment protocols were modeled after those developed by Othmer (1999) and adapted for the Lexicor equipment. Othmer's basic protocols call for left hemisphere training at C3 using a 15 to 18 Hz enhance band and 2 to 7 Hz, 4 to 7 Hz, or 8 to 11 Hz (usual) Inhibit bands. Right hemisphere training at C4 uses a 12 to 15 Hz enhance band and 2 to 7 Hz or 4 to 7 Hz (usual) Inhibit bands. The author combined the standard In-

hibit bands into a 2 to 10 Hz Inhibit band for left hemisphere training and a 2 to 7 Hz Inhibit band for right hemisphere training. This decision was based, in part, on EEG data obtained from previous patients indicating that the 4 to 7 Hz Inhibit at both C3 and C4 generally resulted in reduction in both the 0.5 to 4 Hz and 4 to 7 Hz bands. The broader Inhibit bands targeted those changes more directly. However, instead of providing the patient with separate feedback for the slow wave band being inhibited (2 to 7 Hz at C4 or 2 to 10 Hz at C3) and the fast wave band being enhanced (12 to 15 Hz at C4 or 15 to 18 Hz at C3), concurrent visual and auditory feedback was based solely on the Ratio of the amplitude of the slow wave band divided by the amplitude of the fast wave band. When the active electrode was at C4, the Ratio was based on the amplitude of 2 to 7 Hz divided by the amplitude of 12 to 15 Hz. At C3, the Ratio was determined by 2 to 10 Hz amplitude divided by the 15 to 18 Hz amplitude. In each case, the Ratio was multiplied by ten in order to make it easier for the patient to detect small changes in the cumulative averages, particularly toward the end of the 18-minute segment.

Only the Biolex Game displays (Lexman, Lexwoman, F-14 Tomcat and D'Gizmo) were used with the Ratio protocols. The game figures move up or down with corresponding changes in the magnitude of the Ratio. The game displays provide an additional source of feedback in the form of a reinforcement symbol (e.g., star, flag, and balloon) that appears at the top of the screen when the Ratio remains below the Inhibit level for a therapist specified minimum amount of time. In this case, the minimum was set at one second. A multiplier (e.g., X3 for 3 seconds) appears next to the symbol for each additional full second the Ratio is below the Inhibit. The patient was encouraged to produce the reinforcement symbol and then to extend the multiplier.

The only information provided to the patient about the targeted slow wave (2 to 7 Hz or 2 to 10 Hz) and fast wave (12 to 15 Hz or 15 to 18 Hz) bands were the cumulative session averages shown by the monitor at the left side of the screen. A reward Inhibit level was set at 1.2 to 1.3 times the average Ratio obtained during the first three to four segments with each training protocol. The Y-axis of the visual game display was set at the multiple of five closest to three times the Inhibit level. For example, if the Ratio Inhibit was set at 62, the height of the Y-axis of the display screen was set at 185. The visual display provided the patient with information about both the magnitude and variability of the Ratio. When the Ratio was below the Inhibit level and there were no artifact conditions, the patient received continuous auditory feedback of a middle C flute sound.

The use of Ratio feedback originally developed out of the author's home neurofeedback program (Rossiter, 1998). It has the advantage of being easily understood by even a young child, requires no interpretation, and provides equivalent auditory and visual feedback. When the flute sound is present and the game figure is flying beneath the triangular Inhibit marker on the Y-axis of the game display, the patient knows that he is achieving his training goal. Of all of the EEG data available to the patient, the Ratio is the best single measure of the level of cortical activation.

Ratio feedback has an additional advantage, particularly for home neurofeedback use, because it eliminates the need for daily pre-session baselines to make adjustments in the Inhibit (2 to 10 Hz or 2 to 7 Hz) and Threshold (12 to 15 Hz or 15 to 18 Hz) levels for the EEG bands being suppressed or enhanced. Such adjustments were often necessary because of fluctuations in the EEG patterns within and between days (Kaiser & Sterman, 1994). Pre-session baselines with non-Ratio feedback were needed to insure that the patient was receiving accurate feedback about acceptable levels of the bands being enhanced or suppressed and to maintain the desired level of reinforcement (70 to 90 percent). As the patient's Ratios decreased over time, changes in the Ratio Inhibit settings were made between sessions when the reinforcement rate approached 90 percent. The patient was instructed to reduce the Ratios but was discouraged from experimenting with different strategies to accomplish this goal. He only needed to "want Lexman to fly low and steady, the sound to stay on, and the Ratio average to decrease." The patient was encouraged to be aware of the cumulative Ratio average and to set interim goals of reducing it by 0.3 to 0.5 points. When the current goal had been attained, a new, lower goal was established. Visual feedback based on the Ratios is inherently more volatile than feedback based on the component EEG bands. The use of digital filters adds to the volatility of the game figure. The rapidly changing visual display is an advantage when the purpose of training is cortical activation as opposed to low arousal training. Momentary spikes in the Ratio often cause short-term increases in the cumulative Ratio average. For this reason, the patient is encouraged to keep the game figure flying low and steady. The up and down movement of the game figure provides the patient with an immediate sense of the variability in his level of cortical activation. In many real life situations (e.g., in the classroom, driving, carrying on a conversation, etc.), a highly focused state of attention is not always necessary. A moderate, but consistent level of attention is

often sufficient and may be preferable because it can be maintained with less effort.

Sustaining the level of motivation needed to successfully complete treatment is a crucial issue. Although neurofeedback involves operant conditioning, it is not sufficient to simply expose the patient to the feedback. Unless the desired changes in EEG activity result in feedback (reward) that is meaningful and seen as positive by the patient, little learning may occur. Sterman and his colleagues (Wyrwicka & Sterman, 1968) trained cats to increase 12 to 20 Hz amplitude and duration by following the desired response with a food reward. Many AD/HD adolescents and adults undergoing neurofeedback find the computer generated positive feedback about their performance intrinsically reinforcing because it indicates movement toward personal goals of symptom reduction and control. However, some individuals who appear to be sufficiently motivated at the onset of treatment quickly tire of the process. Once the initial novelty wears off, neurofeedback can become a tedious and boring experience, particularly for children who frequently do not fully understand the nature of AD/HD, how it adversely affects their lives, and the potential long term benefits of neurofeedback.

The patient was highly motivated from the outset to gain control over his AD/HD symptoms. Nevertheless, to provide more immediate incentives, a reward system was implemented after a performance baseline had been established. The patient's goal was to obtain a C4/SMR or C3/Beta Ratio during each 18-minute neurofeedback segment that fell at or below the median for his previous sessions. In addition to meeting the Ratio goals, he was required to maintain Movement artifact (0.5 to 4.0 Hz) at less than 5.0 percent and EMG artifact (22.0 to 30.0 Hz) at less than 10.0 percent. Movement artifact was initially triggered by Delta (0.5 to 4.0 Hz) activity two times greater than the baseline average. EMG artifact was initially defined as Beta2 (22.0 to 30 Hz) activity exceeding 1.7 times the baseline average. Artifact conditions were made more stringent as Delta and Beta2 amplitudes decreased over the course of treatment. The patient earned (a) 100 points each time he met or exceeded the Ratio and artifact goals for an 18-minute training segment, (b) an additional 50 points if he met the Ratio goals for both daily training segments, and (c) a 100-point bonus every time he established a new low for either of the Ratios. The points were exchanged for $5.00 gift certificates from a store of his choosing when he had accumulated 500 points.

The Ratio reward goals were revised as needed based on the updated medians for all training sessions. Over 63 segments of C4/SMR train-

ing, it was necessary to revise the Ratio Inhibit only six times. Both Ratios were adjusted downward based on improved performance as evidenced by a reduction in the median, but were never raised. It was anticipated that the patient would reach his goals approximately 50 percent of the time. However, he was very successful in reducing his Ratios and achieved the reward goal for 78 percent of the 18-minute segments. During the first nine sessions of treatment, the patient received 18 minutes of training using the C3/Beta protocol followed by 18 minutes using the C4/SMR protocol. Treatment days 10 through 22 involved two 18-minute segments of C4/SMR Ratio training at C4. Days 23 through 40 began with a C3 training segment and finished with C4. Days 41 through 45 involved two segments of SMR training at C4.

RESULTS

Cognitive

The patient's performance on the TOVA-A showed significant improvement in Processing Speed and Variability in Attention after both the twentieth and fortieth days of neurofeedback (Table 1). Both scores were well above average by the end of treatment. There were no significant changes (plus or minus 7.5 points) in the Attention (errors of omission) scores from the baseline evaluation to the end of treatment, nor were any expected due to ceiling effects. Errors of omission (failure to respond to the target stimuli) are a relatively infrequent occurrence except among children. Adolescents and adults, even those with AD/HD, seldom fail to respond to the target stimuli. The eight-point decline in

TABLE 1. Test of Variables of Attention-Auditory Scores

TOVA Variables	Baseline	20 Sessions Neurofeedback	40 Sessions Neurofeedback	Change From Baseline
Attention	99	103	104*	+5
Impulsivity	102	105	94	−8
Processing	52	77	133	+81
Variability	75	84	115	+40

Standard scores with Mean = 100 and SD = 15
* Maximum possible score for age

the Impulsivity (errors of commission) score is significant. However, it is not a matter of concern because of the corresponding 81-point increase in Processing Speed. It is not uncommon for Impulsivity and Processing Speed scores to move in opposite directions. As the speed of responding increases, so does the probability that the patient will respond to the non-target stimuli. In spite of the modest decline, the Impulsivity score remained within normal limits for an individual of the patient's age and intelligence.

Intelligence

The patient demonstrated a 19-point increase in IQ from the baseline evaluation (Composite IQ = 89) to the end of treatment (IQ = 108). These results should be viewed with caution since the K-BIT is a screening instrument that does not possess the same degree of reliability over time as the longer Wechsler Scales. Nevertheless, the increase in IQ is well within the range reported by other researchers following neurofeedback for AD/HD (e.g., Thompson & Thompson, 1998). Contrary to what might have been expected in view of the preponderance of training at C4 vs. C3, the greatest gains were made on the language subtests rather the visual-spatial subtest of the K-BIT. If there were to be differential effects, the opposite pattern might have been expected in a right-handed individual.

Behavioral

A comparison of BASC scores obtained from the mother prior to treatment and again after 20 and 40 sessions of neurofeedback (Table 2) indicates generalized improvement in the patient's emotional and behavioral adjustment. Although only the changes in the Hyperactivity and Aggression scales reach statistically significant levels ($p < .01$) as determined by the BASC scoring software, there is a consistent trend for scores on the Clinical Scales to move downward from the Clinically Significant (T score 70 and above) and At Risk (T score 60 to 69) ranges toward the Normal range (T score 40 to 59). By the fortieth session of treatment, only two of the clinical scales (Hyperactivity and Attention Problems) remained in the At Risk range and none were in the Clinically Significant range. The fact that the Attention Problems scale showed no significant change over the course of treatment is curious in view of the marked improvement in this area documented by the TOVA-A. The continued mild elevation on the Hyperactivity scale, however, accu-

TABLE 2. Behavior Assessment System for Children Scores from Baseline to the End of Treatment

BASC Variables	Baseline	20 Sessions Neurofeedback	40 Sessions Neurofeedback	Change From Baseline
Hyperactivity	107	78	69	−38*
Aggression	78	57	55	−23*
Conduct Prob	66	51	51	−15
Anxiety	56	42	50	−6
Depression	55	42	45	−10
Somatization	67	58	55	−12
Atypicality	50	50	41	−9
Withdrawal	37	37	41	+4
Attention Prob	68	65	65	−3

Standard scores with Mean = 50 and SD = 10
* Significant at < .01 level

rately reflects the fact that the patient still manifested a mild degree of restlessness although his impulse control was markedly improved.

BASC scores were obtained from both the mother and father at the end of treatment and 17 months later. The follow-up behavioral data confirm that the gains reported at the end of treatment were still present at follow-up. There were no statistically significant changes on any of the clinical scales at follow-up. However, the changes that did take place in the BASC scores at follow-up were consistently in the direction of lower scores (i.e., improved emotional and behavioral functioning). Four of the five BASC scales (three father, two mother) within the "at risk" category (t score 60 to 69) fell to within the Average range (t score 40 to 59) at follow-up.

The patient's self-report of AD/HD symptoms on the Brown ADD Scale fell from the "ADD probable but not certain" level at baseline (Raw Score = 53) to the "ADD possible but not likely" level (within normal limits) after 20 and 40 sessions of neurofeedback (Raw Score = 36). Being easily frustrated was the only symptom he continued to report as a frequent occurrence.

The patient reported a significant change from previous years in the distribution and severity of eczema during the fall and winter months. The rash was limited to a small spot on each thigh and did not spread over his legs and arms, as had been the pattern during previous winters.

In addition, he did not require the use of any prescription or over-the-counter medication to control the symptoms. While the improvement could have been due to a factor unrelated to the neurofeedback, Othmer (1999) reports success treating eczema with a C4 SMR (12 to 15 Hz) enhancement and theta (4 to 7 Hz) suppression protocol.

Although no formal data regarding the patient's adjustment at school were collected, there is anecdotal evidence suggesting significant improvement. The patient's school provides students with an opportunity to participate in a "reward day" at the end of each academic quarter by meeting specified academic and behavioral standards. Misbehavior, incomplete homework, tardiness, and other violations of school rules result in notations in the daily log that all students carry. Students who receive more than the allowed number of academic and/or behavior demerits during a quarter are not allowed to participate in the "reward day." The patient earned his first reward day in two years during the second quarter of the school year. Treatment was started at the end of the first quarter of the school year.

Academic

The screening version of the Kaufman Test of Academic Achievement (KTEA-Brief) was administered by the school psychologist in April of the previous school year and re-administered by the author at the end of treatment the following January. During that time, the patient's mathematics score increased slightly from a standard score 92 to 93 (Mean = 100, SD = 15) and his spelling score was unchanged with a standard score of 93. This indicates that his mathematics and spelling skills had improved as much as expected from the additional time in school time but that his position relative to his peer group had not changed. By contrast, his reading score increased by 7.3 grade levels (5.2 to 12.5) and 31 standard score points (90 to 121). The reading subtest includes both oral reading and silent reading for comprehension. In the context of the TOVA-A results, it is probable that the markedly improved reading scores are due to an increase in the patient's ability to focus and maintain attention and process information more efficiently. Many children and adults with AD/HD do not like to read because they have trouble maintaining attention, read slowly, and have to re-read in order to comprehend and remember what they have read. This makes reading a frustrating and unrewarding activity. As a result, many do not develop an interest in recreational reading.

During the four months of treatment, the patient received no remedial instruction in reading, nor was any attempt made to increase his recreational reading at home or at school. It was only after treatment was completed that the author recommended that the parents enforce a daily 20 to 30 minute period of recreational reading at home. During that time, he is allowed to read any materials that are of interest to him. The mandatory recreational reading is considered necessary if the patient is to realize that his reading skills have improved and he is to develop a more positive attitude toward reading. In the long run, it is hoped this will lead to an increase in voluntary recreational reading and further improvement in his reading skills.

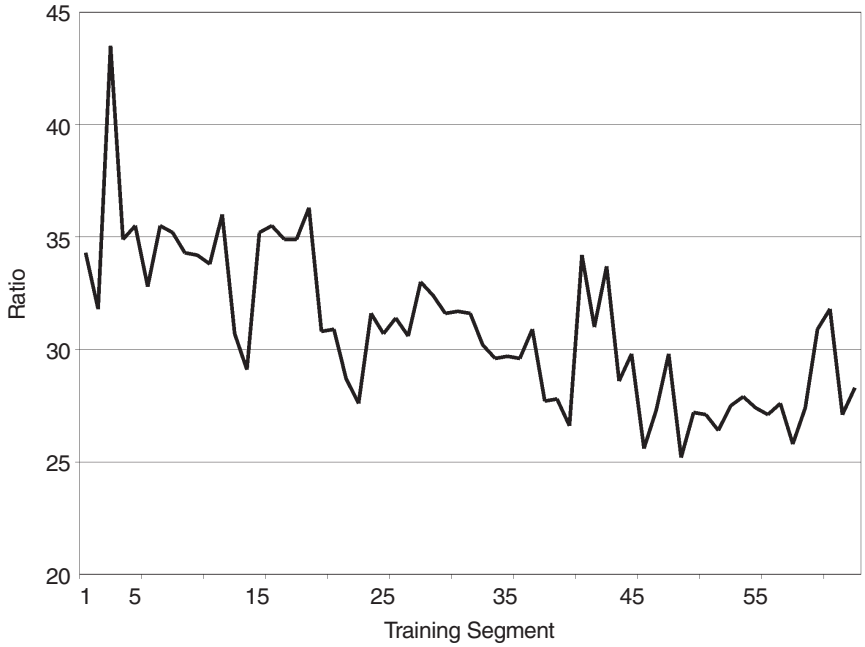
Electroencephalography: C4/SMR Training

One of the criticisms that can be leveled at studies evaluating the effectiveness of neurofeedback for AD/HD (Rossiter & La Vaque, 1995) is that they do not always provide data demonstrating changes in the EEG as a result of treatment. In the absence of changes in the EEG consistent with those expected from the training protocols, the possibility of a placebo response, spontaneous recovery, or some other uncontrolled factor resulting in clinical improvement cannot be ruled out (La Vaque, 1999). However, in this case, there are documented changes in EEG patterns that are generally consistent with expectations based on the C4/SMR treatment protocol. Since the patient was primarily trained to reduce the C4/SMR Ratio (10* amplitude 2 to 7 Hz/amplitude 12 to 15 Hz), a decrease in the average Ratio over the course of treatment would be expected and this is what occurred (Figure 1).

When the C4/SMR Ratios from the first and last halves of treatment are compared, there is a significant reduction in the Ratio averages (Table 3). The reduction in the Ratio was due to a decrease in 2 to 7 Hz amplitude (−11.6 percent) and, to a much lesser extent, an increase in 12 to 15 Hz amplitude (+ 3.3 percent). This is consistent with the author's previous experience that reductions in the Ratio and clinical improvement are more often associated with suppression of slow wave activity than an increase in the fast wave activity targeted for enhancement.

One factor that made it possible to document C4/SMR Ratio and EEG changes so clearly is the fact that training was done at the same time every day (4:00 PM). There are a number of factors that can influence the EEG including fatigue, illness, medications, time of day (Kaiser & Sterman, 1994), etc. A 10-year-old currently being treated by the author demonstrates the time of day effect very clearly. Ratios obtained

FIGURE 1. Changes in C4/SMR Ratio of 13-year-old AD/HD patient over 4 months.



at 9:00 AM average 20 to 30 percent higher than those obtained at 1:00 PM. Variations of this magnitude would almost certainly conceal smaller day-to-day reductions in the Ratio associated with neurofeedback. Where variations in the time of day or other confounding factors make day-to-day comparisons misleading, the best evidence that the patient is learning to change the level of cortical activation comes from changes in the Ratio that take place during individual training segments. Figure 2 illustrates both a decrease in the Ratio and a reduction in the variability of the Ratio from the first to the second half of a single C3/Beta training segment (10*2 to 7/15 to 18 Hz). The data were obtained during the sixth neurofeedback session of the 10 year-old referenced above.

Illness can also have a significant effect on the EEG. Figure 1 shows an increase in the 13-year-old patient's C4/SMR Ratios from the forty-first to the forty-seventh C4/SMR segments. During that period, he had a cold and his performance apparently suffered as a result. When the illness was resolved, the Ratios returned to previous levels and further progress was made.

TABLE 3. EEG Changes with C4/SMR Training

EEG Variables	Segments 1-31 Mean (SD)	Segments 32-63 Mean (SD)	Significance Level (p)*
C4/SMR Ratio	33.21 (3.01)	28.70 (2.24)	0.0001
2-7 Hz	17.18 (1.40)	15.18 (0.69)	0.0001
0.5-4 Hz	22.28 (2.07)	20.65 (1.07)	0.0002
4-7 Hz	11.14 (0.77)	9.79 (0.49)	0.0001
8-11 Hz	6.44 (0.47)	6.92 (0.74)	0.01
12-15 Hz	5.45 (0.32)	5.63 (0.43)	0.09
15-18 Hz	5.38 (0.29)	5.27 (0.35)	0.23
22-30 Hz	4.82 (0.24)	4.54 (0.28)	0.0001

*One tailed t-test with repeated measures, df = 30

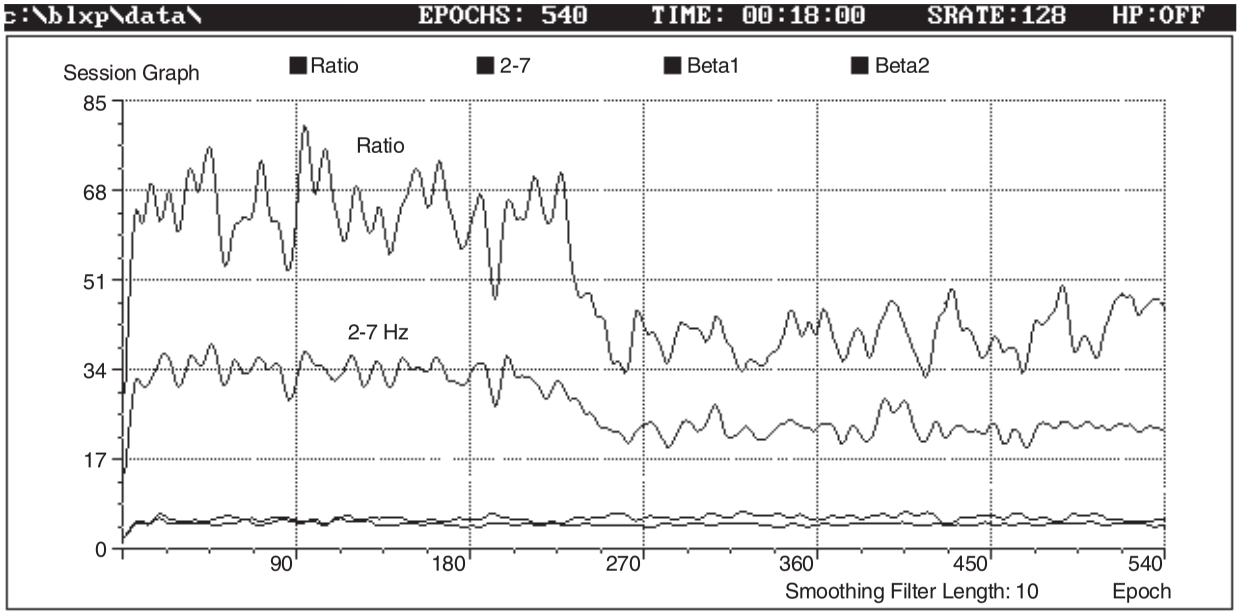
The training protocols contained both movement (0.5 to 4.0 Hz) and EMG (22 to 30 Hz) artifact Inhibits. The amplitudes of both bands decreased significantly over the course of treatment. The 12 to 15 Hz (SMR) amplitude tended to increase while the 15 to 18 Hz (Beta) band was essentially unchanged. The 12 to 15 Hz and 22 to 30 Hz session means moved in opposite directions and the differences increased as treatment progressed. These changes are consistent with the treatment protocol that sought to increase 12 to 15 Hz activity (+ 3.3 percent) while decreasing 22 to 30 Hz activity (− 5.8 percent).

The 22 to 30 Hz artifact condition was included in the treatment protocol to control the inclusion of data contaminated by EMG artifact. However, the relatively large and statistically significant correlation between the C4/SMR Ratio and Beta2 (Pearson r = 0.39, two tailed significance = .0015) was not anticipated and suggests that 22 to 30 Hz activity may play a direct role in influencing the 2 to 7 Hz/12 to 15 Hz Ratio. Whether the measured 22 to 30 Hz activity was due to EMG and/or heightened states of cortical activation associated with anxiety is unknown. In addition, it is not clear whether the decrease in 22 to 30 Hz amplitude would have been as large if there had not been stepwise reduction in the amplitude of 22 to 30 Hz activity allowed before triggering an artifact condition.

Electroencephalography: C3/Beta Training

Conclusions that can be drawn from the C3/Beta training are tentative because there were only 27 of the 18-minute training segments and

FIGURE 2. Changes in the magnitude and variability of the C3/Beta Ratio of a 10-year-old AD/HD patient during one training segment.



a gap of 35 days between the first nine and the last 18 segments. Unlike the C4/SMR training, the C3/Beta training did not result in a significant decline in the Ratio (Table 4). Only a tendency toward a decline in the C3/Beta Ratio was observed in spite of the fact that there was a significant decline in the 2 to 10 Hz amplitude. Inspection of the bands that contribute to the 2 to 10 Hz band provides a partial explanation. Both the Delta (0.5 to 4 Hz) and Theta (4 to 7 Hz) bands showed large and highly significant reductions. By contrast, the Alpha (8 to 11 Hz) band showed a significant increase in amplitude rather than the expected decrease.

The Alpha increase (+ 7.5 percent) had the effect of limiting overall declines in the 2 to 10 Hz Inhibit band (− 3.9 percent) thus minimizing reductions in the Ratio. It is significant that there was no increase in the Beta (15 to 18 Hz) amplitude over the course of C3 training even though the band was specifically targeted for enhancement. Thus, any reduction in the C3/Beta Ratio was due entirely to reductions in the 2 to 10 Hz band. There was, however, a tendency toward an increase in SMR (12 to 15 Hz) activity that parallels that observed with C4/SMR training. In addition, the amplitude of Beta2 (22 to 30Hz) tended to decrease (− 4.5 percent) from the first to the second half of training. The results obtained with the C3/Beta training suggest that attempts to suppress the 2 to 10 Hz band may be problematic, particularly if one of the goals is to bring about reductions in the low alpha range (8 to 10 Hz). An additional problem with this protocol is that it may be counterproductive

TABLE 4. EEG Changes with C3/Beta Training

EEG Variables	Segments 1-13 Mean (SD)	Segments 14-27 Mean (SD)	Significance Level (p)*
C3/Beta Ratio	38.55 (1.48)	37.17 (2.41)	0.15
2-10 Hz	18.53 (0.58)	17.81 (0.68)	0.01
0.5-4 Hz	21.39 (1.59)	19.94 (1.25)	0.0008
4-7 Hz	11.09 (0.33)	10.43 (0.33)	0.0006
8-11 Hz	6.51 (0.61)	7.00 (0.74)	0.03
12-15 Hz	5.25 (0.42)	5.50 (0.42)	0.13
15-18 Hz	5.01 (0.26)	4.99 (0.33)	0.74
22-30 Hz	4.71 (0.24)	4.50 (0.27)	0.07

*One-tailed t-test with repeated measures, df = 12

from a motivational standpoint. The patient did not like training with the C3/Beta protocol because success (i.e., reduction in the Ratio within and between training sessions) was much more difficult to obtain than with the C4/SMR protocol. The author was aware of the differential difficulty of the two protocols, but until the data were analyzed, attributed the difficulty to the fact that the patient was being asked to suppress a broader band of slow wave activity (2 to 10 Hz vs. 2 to 7 Hz) in the C3/Beta protocol. A review of EEG data from a number of other patients indicated similar difficulties with the 2 to 10 Hz Inhibit band. With patients who do not manifest excess levels of 8 to 11 Hz activity at baseline, the author is currently using a 2 to 7 Hz Inhibit band with C3/Beta training. Ratio feedback setup files currently being used by the author are available on request (t.rossiter@worldnet.att.net).

DISCUSSION

The positive treatment outcome with this 13 year-old with AD/HD is not surprising, except perhaps for the breadth and magnitude of the changes. He demonstrated: (a) marked improvement in his ability to process information efficiently and pay attention consistently (TOVA-A processing speed and variability), (b) a 19-point increase in IQ (K-BIT), (c) improvement in reading skills from the 5th to the 12th grade level (KTEA-Brief), and (d) generalized improvement in behavioral and emotional adjustment at home and at school (BASC & Brown ADD Scales).

As important as the improvement observed when treatment was completed, is the fact that the gains had not diminished 17 months later. The parents state that the patient is making good progress in school. The ready anger and impulsivity that were their primary concern prior to neurofeedback are no longer present. The patient is described by his mother as being much happier than he had been prior to treatment and a "joy" to his family.

This patient's sustained improvement at follow-up illustrates what may be the most important advantage that neurofeedback has over stimulant drugs in treating AD/HD. When neurofeedback is successful, as it is in 70 to 80 percent of the cases treated, the improvement lasts (Lubar, 1995). This is not surprising since neurofeedback involves the learning of new skills. Like learning to read or to ride a bicycle, the ability to shift the level of cortical arousal acquired through neurofeedback becomes a habit that is resistant to loss with the passage of time. The same

cannot be said for stimulant drug therapy. Although 65 to 75 percent of AD/HD children respond positively to stimulant drugs (Weiss & Hechtman, 1993) there is no lasting reduction in AD/HD symptoms once the medication is discontinued (Barkley, 1990).

Neurofeedback was conducted using Ratio feedback protocols. The patient received visual and auditory feedback based on the Ratio of 2 to 7 Hz/12 to 15 Hz or 2 to 10 Hz/15 to 18 Hz. Ratio feedback has a number of potential advantages. It eliminates the need for daily baselines and adjustments in Inhibit and Threshold levels that would otherwise be needed to provide accurate feedback and to maintain desired levels of reinforcement. More importantly, it provides clear and unequivocal feedback to the patient about his performance. No interpretation of the data is needed. The training goal is being met when the auditory feedback is on, the Biolex Game figure is flying below the Inhibit marker on the Y-axis of the visual display, and the cumulative average for the Ratio is declining. Ratio feedback makes no a priori assumption about whether brain activation is achieved by reducing slow wave activity and/or increasing fast wave activity. Based on experience with a limited number of patients to date, it is the author's impression that Ratio feedback may result in faster learning, particularly early in treatment and with younger children because it is easier for patients to grasp what the feedback means and what is expected of them.

Analysis of the changes in the patient's EEG patterns indicates that C4/SMR Ratio training generally had the expected effects. The targeted slow wave activity (2 to 7 Hz) declined and the fast wave activity (12 to 15 Hz) tended to increase. There was no significant change in the amplitude of the 15 to 18 Hz band. However, there was a significant increase in 8 to 11 Hz amplitude and 22 to 30 Hz activity declined significantly over the course of treatment.

The positive correlation between the C4/SMR Ratio and 22 to 30 Hz activity suggests that suppression of activity in the upper beta range played a role in treatment in addition to controlling EMG artifact. In retrospect, the relationship between the SMR and Beta2 bands is not surprising based on the physiological and psychological states associated with each. The SMR band is associated with reduced levels of muscle activation and an alert but relaxed state of mind. The upper Beta band, by contrast, is associated with heightened levels of muscle tension and anxiety.

An unexpected finding was that C3/Beta and C4/SMR protocols had essentially the same effect on the EEG, even though they targeted different bands to enhance and suppress. With both the C3/Beta and

C4/SMR protocols, amplitudes in the 0.5 to 4 Hz and 4 to 7 Hz bands decreased, 8 to 11 Hz increased, 12 to 15 Hz tended to increase, 15 to 18 Hz activity did not change, and 22 to 30 Hz activity decreased (C4/SMR) or tended to decrease (C3/Beta).

It appears that suppression of slow wave activity (2 to 7 Hz) may have been the active ingredient in both Ratio protocols and that fast wave enhancement either played a minor (12 to 15 Hz) or no role (15 to 18 Hz). The fact that both protocols led to a significant increase in 8 to 11 Hz activity suggests that further study of the role of alpha is warranted. Norris, Lee, Cea, and Burshteyn (1998) reported that enhancement of Pz alpha (8 to 13 Hz) without any slow wave suppression was effective in improving attention in a healthy college student. On the other hand, Lubar and Lubar (1999) report that many of their AD/HD patients above the age of 14 show excessive alpha activity and a lack of alpha blocking. It should be noted that the adolescent in this case did not show excessive pre-treatment alpha amplitude at Cz, C3, or C4.

The results obtained from this patient raise more questions about the effects of the C3/Beta and C4/SMR treatment protocols than they answer. Training at C4 with the goal of reducing 2 to 7 Hz amplitude and increasing 12 to 15 Hz generally appears to produce the expected changes in the EEG. Training at C3 to Inhibit 2 to 10 Hz and enhance 15 to 18 Hz activity does not. The C3/Beta protocol does not result in an increase in 15 to 18 Hz activity and the decrease in 2 to 10 Hz activity is limited by increases at the upper end of the band. In effect, the C4/SMR and C3/Beta training appear to have produced similar changes in the EEG, but the C3/Beta training may have done so less efficiently. There is also the possibility that the changes in EEG observed at C3 are the result of the C4/SMR training. If these results are not idiosyncratic to this patient, and data from other patients suggest that they may not be, they cast doubt on the author's assumption that the two training protocols have unique effects on EEG activity. However, even if the two Ratio protocols do not lead to unique changes in the EEG, they may still have differential effects on brain functions due to the differences in training sites.

Although treatment was very successful, a number of the author's assumptions regarding the effects of the Ratio protocols on EEG were incorrect. This suggests that during treatment, therapists monitor elements of the EEG other than those being targeted for change. Ideally, changes in the EEG could be examined both within and across individual treatment segments. This is easily done with the Biolex 2.38 software. The practitioner can define up to eight EEG bands in the 0.5 to 32.0 Hz

range. At the end of a training segment, average microvolt values are displayed for each of the defined bands. Changes in the band amplitudes can be tracked across successive treatment segments. Derived parameters are used to customize the feedback and gather information to show trends after the segment is completed. A maximum of five derived parameters can be defined in a treatment protocol. Derived parameters may include bands as well as mathematical expressions combining bands and/or algorithms (e.g., Ratios). When using Ratio setups, the derived parameters could include the Ratio, the bands being enhanced and suppressed, and two additional bands of interest. Given the results with this patient, the author would elect to monitor trends in the Alpha and Beta2 bands within segments.

It would be useful to analyze EEG changes in successfully treated individual AD/HD patients as a first step toward understanding the effects of different treatment protocols on well-defined groups of patients. However, defining patient groups in terms of observed behaviors as in done in DSM-IV is inherently unreliable and can lead to groups of patients that are heterogeneous not only in terms of their symptoms, but their EEG characteristics as well. It would be more useful to define group membership based on common EEG characteristics and use behavioral characteristics secondarily, if at all. It may be that what the treatment protocols are believed to do, and the actual effects on the EEG and on brain functions are not the same. If there are common active elements across the different treatment protocols, this may be one way of beginning to recognize them. Nineteen channel brain map data collected prior to treatment, during the course of training, and at the conclusion of treatment would enhance our understanding of how neurofeedback protocols actually work. It would be useful to know how different training protocols influence the EEG, not just at the site being trained but also across the brain more generally. This would allow for the development of more effective and efficient treatment protocols that, in turn, would make it easier to demonstrate the effectiveness of neurofeedback relative to stimulant drug therapy.

It is recommended that clinicians develop standard pre-treatment and post-treatment assessment protocols for use with their AD/HD patients. At a minimum, this would include documenting both the DSM-IV AD/HD and any secondary diagnoses, obtaining objective measures of cognitive abilities that may be compromised by AD/HD, using standardized behavioral questionnaires for parents, documenting changes in the EEG at the training site(s), and obtaining pre-and post-treatment brain maps, if feasible. Clinicians could use these data to improve the

efficiency of their neurofeedback protocols, build a treatment outcome database, and potentially add to the growth of the field by making individual or group case studies available to their colleagues through peer-reviewed journals or at professional meetings.

REFERENCES

Barkley, R. A. (1990). *Attention deficit hyperactivity disorder. A handbook for diagnosis and treatment*. New York: Guilford Press.

Fenger, T. N. (1998). Visual-motor integration and its relation to EEG neurofeedback brain wave patterns, reading, spelling, and arithmetic achievement in attention deficit disordered and learning disabled students. *Journal of Neurotherapy*, *3* (1), 11-20.

Fuchs, T. (1999). Aufmerksamkeit und neurofeedback: Evaluation eines psychophysiologischen eeg-biofeedback-behandlungs programms bei aufmerksamkeits und hyperaktivitatsstorungen in kinesalter. (*Attention and neurofeedback: An evaluation of a psychophysiological EEG biofeedback treatment program for childhood attention-deficit/hyperactivity disorders.*) Regensburg, Germany: Roderer.

Kaiser, D. (1997). Efficacy of neurofeedback on adults with attentional deficit and related disorders. (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436).

Kaiser, D., & Othmer, S. (2000). Effect of neurofeedback on variables of attention in a large multi-center trial. *Journal of Neurotherapy*, *4* (1), 5-15.

Kaiser, D., & Sterman, M. B. (1994). Periodicity of standardized EEG spectral measures across the waking day. Paper presented at the 7th Annual Summer Sleep Workshop Multi-Site Training Program for Basic Sleep Research, Lake Arrowhead, CA.

La Vaque, T. J. (1999). Neurotherapy and clinical science. *Journal of Neurotherapy*, *3* (3 & 4), 29-32.

La Vaque, T. J. & Rossiter, T. R. (2001). The ethical use of placebo controls in clinical research: The Declaration of Helsinki. *Applied Psychophysiology and Biofeedback*, *26* (1), 23-37.

Lubar, J. F. (1995). Neurofeedback for the management of attention-deficit/hyperactivity disorder. In M. S. Schwartz & Associates (Eds.), *Biofeedback: A practitioner's guide* (2nd ed., pp. 493-522). New York: Guilford Press.

Lubar, J., & Lubar, J. (1999). Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 103-143). San Diego, CA: Academic Press.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-Regulation*, *20* (1), 83-99.

Norris, S. L., Lee, C., Cea, J., & Burshteyn, D. (1998). Performance enhancement training effects on attention: A case study. *Journal of Neurotherapy*, *3* (1), 21-27.

Othmer, S. (1999). *EEG Biofeedback Protocol Update*. (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436).

Othmer, S., Othmer, S., & Kaiser, D. (1999). EEG biofeedback: An emerging model for its global efficacy. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 244-310). San Diego, CA: Academic Press.

Ramos, F. (1998). Frequency band interaction in ADD/ADHD neurotherapy. *Journal of Neurotherapy*, *3* (1), 28-46.

Rossiter, T. R., & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treating attention deficit hyperactivity disorders. *Journal of Neurotherapy*, *1* (1), 48-59.

Rossiter, T. R. (1998). Patient directed neurofeedback for AD/HD. *Journal of Neurotherapy*, *2* (4), 54-64.

Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model*. New York: Guilford Press.

Thatcher, R. W. (1999). EEG database-guided neurotherapy. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 29-64). San Diego, CA: Academic Press.

Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback*, *23*, 243-263.

Wadhwani, S., Radvanski, D. C., & Carmody, D. (1998). Neurofeedback training in a case of attention deficit hyperactivity disorder. *Journal of Neurotherapy*, *3* (1), 42-49.

Weiss, G., & Hechtman, L. T. (1993). *Hyperactive children grown up: ADHD in children, adolescents, and adults* (2nd Ed.). New York: Guilford Press.

Wyrwicka, W., & Sterman, M. B. (1968). Instrumental conditioning of sensori-motor cortex EEG spindles in the waking cat. *Physiology and Behavior*, *3*, 703-707.

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