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Table of Contents

Article	**Page No.**
Business	**III**
Instructions to Authors	
Announcements	**IV**
Guest Editorial Norman C. Moore	**V**
Editorial Frank H. Duffy	**V**
A Review of EEG Biofeedback Treatment of Anxiety Disorders Norman C. Moore	**1**
An EEG Biofeedback Protocol for Affective Disorders J. Peter Rosenfeld	**7**
The Treatment of Addictive Disorders by Brain Wave Biofeedback a Review and Suggestions for Future Research David L. Trudeau	**13**
Self Regulation of Electrocortical Activity in Schizophrenia and Schizotypy: a Review John Gruzelier	**23**
Treatment of Attention Deficit Hyperactivity Disorder with Neurotherapy John K. Nash	**30**
EEG Operant Conditioning Biofeedback and Traumatic Brain Injury Robert W. Thatcher	**38**
Basic Concepts and Clinical Findings in the Treatment of Seizure Disorders with EEG Operant Conditioning M. Barry Sterman	
Abstracts	**56**

Treatment of Attention Deficit Hyperactivity Disorder with Neurotherapy

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Key Words

Attention Deficit Hyperactivity Disorder
EEG Biofeedback
Neurofeedback
Neurotherapy
Quantitative EEG

INTRODUCTION

An increasing number of clinicians use operant conditioning of electroencephalographic activity (termed neurotherapy, neurofeedback, or EEG biofeedback) as a method of helping children and adults with Attention Deficit Hyperactivity Disorder (ADHD) learn improved control of their attention and activity levels. The present review examines 1) the nature of ADHD, 2) the scientific and public health rationale for the use of neurotherapy, 3) the nature of the neurotherapy process and 4) the published outcome studies on EEG and behavioral change following neurotherapy.

THE NATURE OF ADHD

ADHD is a psychiatric disorder characterized by clinically significant levels of symptoms of either inattentiveness, hyperactivity/impulsivity or both, Persons with this pattern are easily bored, distractible, disorganized, often stimulation seeking and impulsive. Epidemiological studies have estimated 6 to 9% of our children have ADHD.[1,2] Children with ADHD show lowered vigilance, sustained attention, impulse inhibition, executive functions such as organization and complex problem solving and verbal learning and memory.[3-5] These deficits persist into adolescence and adulthood.'[6]

ADHD traits show significant heritability[7] strongly suggesting this pattern is a genetic predisposition. It is likely that societal factors, including economic and physical adversity, dissolution of traditional family and group/tribal structures contribute to the pathological expression of the ADHD phenotype as well as other childhood mental disorders.[8] One might suspect our desire for standardized, rather regimented educational processes (prescribed sequences of study, timed learning periods, siding for long periods of time) might also play a role in the pathological expression of ADHD traits.[9,]

THE RATIONALE FOR NEUROTHERAPY

Public Health Concerns

Although the effectiveness of psychostimulants in the treatment of ADHD is well documented, medications fail to produce the desired improvement in behavior in a significant percentage of patients, as high as 30-40%.[10] Further, gains from pharmacotherapy are "state dependent," i.e., are often lost when the medication wears oft or is discontinued While short-term side effects are considered minimal according to Barkley,[10] some mixture of appetite loss, weight loss, slowed growth, tics, headaches and stomach aches may appear in 20-50% of patient[11] and may lead to discontinuation of treatment. Although considered a rare problem, in some cases adolescents or even their parents may abuse the stimulants, selling their medications on the street or "snorting" higher doses of them personally.[12-13] A study of a sample of 161 school children who had taken Ritalin for 5 years showed 16% reported being asked to give, sell or trade their medication.[14] Comorbid anxiety, occurring in as many as 30% of ADHD youths, may limit the success of stimulant therapy. These limitations have led to the rapid adoption in clinical practice of alternative and adjunctive medications despite the current lack of controlled research on safety and efficacy[15] So while medications carry considerable benefits for ADHD patients, there are also considerable shortcomings and risks.

Despite research indicating a relatively low rate of side effects and the apparent short-term safety of these medications, many parents have safety concerns. Psychostimulants elevate heart rate and systolic blood pressure., the response is variable between individuals and is dose dependent[16] This may be a concern in patients with elevated risk for hypertension and heart disease. No long-term study has evaluated potential effects on the cardiovascular system or on the brain, One early CT scan study documented apparent cortical atrophy in a group of 25 young adult males with a history of ADHD and psychostimulant medication use.[16] Although this study could not directly relate the atrophy to psychostimulant use, the authors noted there may be a relationship, This somewhat alarming finding has never been followed up with contemporary imaging techniques. Newly identified unmedicated adult patients with ADHD should be compared to adults with a history

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of medical psychostimulant use as children. The recent NIH Consensus Conference on ADHD[18] pointed out the longest follow-up with methylphenidate is 14 months, and this did not involve imaging.

As the psychosocial difficulties associated with ADHD have been noticed, and as the only partial success of medical management has become apparent, there has been increasing interest in the possibility that persons with attentional hyperactivity and impulse control difficulties can learn to regulate their central nervous systems with neurotherapy and thereby ameliorate symptoms of ADHD. It would be highly desirable if neurotherapy produced long lasting improvements in CNS function with concomitant improvements in behavior, without side effects. Ideally, medication might be used as a safe, immediate short-term aid, with trained self regulation of the attention, impulsivity and hyperactivity developing in the longer term. The idea of neurotherapy is not to "cure* ADHD, but to afford the individual with the ADHD trait a means of learning to counterbalance his or her genetic tendencies with learned skill.

Scientific Rationale for Neurotherapy

In 1971 Satterfield and Dawson[19] suggested underarousal of frontal cortex might explain ADHD symptoms. Niedermeyer and Naidu[20] have recently discussed this lazy frontal brain" ADHD hypothesis further. Niedermeyer presents a detailed, integrative review of frontal lobe function that shows neuroanatomical possibilities for the variety of behavioral manifestations seen in ADHD.[21] Lowered average arousal levels, poor connectivity and perhaps structural differences in the brain regions that subserve the deployment and persistence of attention, planning, the examination and regulation of impulses the ability to notice impulses and "think better of it" would create most of the ADHD pattern.

As long ago as 1938 Jasper, Solomon and Bradley[22] showed evidence that a behaviorally divergent group of youngsters had EEG "slowing." Recent literature in quantitative electroencephalography (QEEG), single photon emission computed tomography (SPECT) and positron emission tomography (PET) lend further support to the relative underarousal hypothesis.

QEEG

Digitization EEG signal coupled with normative databases and discriminant functions has permitted the development of a 'neurometric` approach to the examination of EEG in psychiatric publications,[23] In a comprehensive review of this literature Hughes and John[24] discuss the range of over 500 QEEG articles published just in the last decade relating specific patterns of abnormality to particular diagnoses. This literature documents reliable EEG differences between ADHD and non ADHD children and shows QEEG discriminant analysis to be sensitive and specific to subtypes of ADHD, ADHD versus normals and ADHD versus specific developmental learning disorders (SDLQ) individuals.

Elevated frontal theta activity and diminished beta activity in the resting EEG have been documented in children with ADHD. Mann et al.,[25] studying matched groups of 25 9-12 year old boys with and without ADHD (inattentive type) documented increases of theta during tasks that suggests children with ADHD actually become less activated when presented with cognitive challenge. Discriminant analysis of QEEG variables correctly classified 74% of controls and 80% of ADHD participants. Matsuura et al[26] have also reported this frontal theta/alpha excess with alpha slowing. Lazzaro et al.[27] have reported similar QEEG results in 26 nonmedicated adolescents with ADHD diagnoses versus 26 matched controls showing elevated absolute frontal theta and diminished relative posterior beta in the participants with ADHD.

Chabot and Serfontein[28] compared the QEEGs of 407 children with ADHD diagnoses with 310 normal children. QEEG discriminated between the groups with 93.7% sensitivity and 88% specificity. ADHD children showed increased theta absolute and relative power, particularly in frontal regions. Marked interhemispheric frontal and parietal hypercoherence, posterior asymmetries and frontaloccipital as well as centralparietal hypocoherence also distinguished ADHD from normal children's QEEG. These findings were consistent with deviations from normal development as opposed to maturational delay, i.e., these were not patterns that would be normal at an earlier time in life. Two distinct neurophysiological ADHD subtypes, one showing elevated theta/alpha, the other also showing elevated theta/alpha but also slowing of the alpha mean frequency, were seen, suggesting the "underarousal' hypothesis may not be adequate to describe the complexity of the subtypes of ADHD.

Chabot et al.[29] went on to show that QEEG discriminant functions classified learning disabled, ADHD and normal children with high sensitivity and specificity. The same paper showed QEEG discriminates between methylphenidate versus dextroamphetamine responders. This is clinically important, since results showed that if initial diagnosis had included QEEG, 27 cases of adverse medication response could have been prevented. Suffin and Emory[30] showed that patients with frontal theta excess responded well to methylphenidate whether their diagnosis was attentional or affective disorder. Patients with elevated alpha responded well to Prozac. An earlier study by Simeon et al[31] showed ADHD children with elevated alpha responded well to bupropion. These findings have led Chabot and others to call for routine use of QEEG in diagnosing ADHD.

SPECT, PET Corroboration of Frontal Abnormalities In ADHD

Amen and Carmichael,[32] using high resolution SPECT imaging showed regions of prefrontal hypometabolism in 87% of a group of medication free children and adolescents with ADHD, compared with 5% of youths with other diagnoses. Lou et al[33] have also reported prefrontal hypoperfusion in SPECT studies on children with ADHD and SDLD Seig et al.[34] reported SPECT findings on 10 ADHD patients, with less uptake on left frontal and left parietal compared to controls. The PET studies of Zametkin et al.[35] have been widely cited as evidence for hypometabolism of frontal cortex in ADHD patients. This population showed worsening of the hypometabolism during a cognitive task, paralleling both the SPECT findings of Amen and Carmichael[32] and the QEEG findings of Lubar,[35] who describes increased theta during rest and even greater abnormality during cognitive challenge.

The findings described above emphasize the importance of moving toward objective neurophysiological diagnoses for psychiatric disorders. Given the present lack of objective measures to make differential diagnoses of ADHD, it appears time to integrate this powerful tool into standard clinical practice.

NEUROTHERAPY: OPERANT CONDITIONING OF THE EEG

The public health concerns and QEEG studies outlined above form a strong scientific rationale for neurotherapy. The QEEG abnormalities seen in ADHD are common, appear in most identified patients, and are not subtle when the proper technology is used to observe them.

As documented in the previous section, ADHD is often marked *on the average* by elevations of slow wave theta and/or alpha activity over frontal or in parietal regions (the latter more prevalent in inattentive ADHD). When the spontaneous EEG is observed, however, it is clear that there are moments when abnormal appearing EEG records 'clear up." When these moments occur the person appears focused on the task. When asked 'What are you doing?" a patient will usually respond "Nothing. I'm just concentrating." Patients will often note a clearing of the mind, reduction in distracting thoughts, or less .cognitive noise" as one of my patients puts it. These moments of improved EEG can be considered 'operants of alertness."

Operant conditioning of the EEG is called EEG biofeedback, neurofeedback or neurotherapy. Neurotherapy helps the person learn to recognize the small shifts in state they go through during the course of the session, as well as during the course of their day. Patients gradually begin to notice and influence their ambient state of alertness. Neurotherapy is a "finegrained" form of cognitive behavior modification in which improvements in cortical function are operationalized as changes in the EEG.

Neurotherapy involves presentation of auditory and/or visual signals that are proportional to the relevant EEG measure (e.g., amplitude, correlation, symmetry, etc.) A tone may come on when theta amplitude drops below a preset threshold; a second tone may come on when sensorimotor rhythm (SMR) or beta amplitudes rise above a given value with the goal of decreasing theta and increasing SMR or beta. A wide range of feedback presentations is currently in use, some of them quite creative. Computer displays exist in which various 'games' can be played, e.g., PacMan advances when certain parameters are met, an airplane flies to the top of the screen and an American tag lights up when parameters are met, etc. The specific values of the parameters are determined individually, such hat reinforcement (tones on, airplane up) occurs 60-80% of the time. Training is usually done twice weekly for 20-40 sessions.

Control must be obtained over muscle tension artefact since tense muscles can produce artefactual "beta" activity. Many people equate a certain amount of tension with "paying attention" and frown, lift their eyebrows or clench their jaws when trying to concentrate. A quiet body and alert mind is the explicit target state.

Cognitive tasks, including reading, listening, visual attentional tasks are used concurrently with auditory neurofeedback to promote generalization. The aim is to promote improved self monitoring of the onset of ADHD behaviors and improved conscious self regulation of the internal state at such moments. Like any operant conditioning program, neurotherapy requires generalization and establishing a reinforcing environment to support small, progressive changes in behavior. Parents and teachers should be trained in basic behavior modification methods. With neurotherapy small gains usually begin to be apparent after approximately 10 sessions. 'It is critical that parents and teachers be prepared to meet small improvements with reinforcement.

Sessions must occur regularly, typically twice weekly, although some summer programs will train daily. If the family cannot comply with this, putting oft treatment for a better time should be considered. If there is major family conflict, illness, tragedy, or a hostile relationship between divorced custodial parents or criminal conduct, these issues should be dealt with before a course of neurotherapy is considered. Further, although there are no studies that report untoward side effects from neurotherapy, patients should be screened for psychotic disorders. Mental retardation, severe depression or bipolar illness, or severe conduct/oppositional defiant disorder may prevent successful therapy[35] Occasionally patients report headaches, but this is likely due to tensing up during training and disappears rapidly when muscles are relaxed. Tiredness after the initial sessions may occur, presumably due to the intensity of the "workout." On the other hand, many patients will feel more alert and more awake after neurotherapy sessions. Lubar[36] has published a detailed account of the procedures involved in neurotherapy treatment.

Outcomes Studies of the

Treatment *of* ADHD with Neurotherapy

The 1970s and 1980s

The use of EEG operant conditioning to treat ADHD developed out of the work of M.B. Sterman and colleagues with epileptic patients[37,38] in the 1970s and the early literature showing slow wave excess in ADHD. Sterman demonstrated reduction in epileptiform background activity following treatment that successfully reduced seizure frequency. This healthier background EEG persisted even during sleep. This observation suggests neurotherapy produces increased cortical stability.

The first case study of applying Sterman's methods to ADHD was by Lubar and Shouse[39] who reported EEG and behavioral improvement in an 8 year 11 month old "hyperkinetic" child following application of neurofeedback. Lubar and Shouse rewarded increases in SMR and lower theta amplitude. Two independent observers documented reduced oppositional behavior and increased cooperation in the classroom. Self stimulation, object play and out-of-seat behavior decreased and attentive and school work behaviors increased. All of these changes were reversed over a 4 week period when increases in theta and decreases in SMR were reinforced. Finally, return to SMR enhancement and theta decrease resulted in a second and apparently long lasting improvement in the child's behavior. Medication was permanently discontinued by the end of treatment, and follow-up several years later revealed the child continued to function well with no medication.

The Lubar and Shouse[39] study was in the classic style of applied behavior analysis using an ABA reversal design. The importance of this design must be highlighted, since there have been criticisms directed at the studies of neurotherapy for lack of large double blind, placebo controlled designs, which have high internal validity, but are uncommon in the scientific study of behavior modification and often lack external validity. The ABA reversal design with subject as his or her own control has been an acceptable method in the scientific investigation of behavior for several decades.

Further studies involving more children were undertaken by Shouse and Lubar using the same blind, ABA design with SMR enhancement, theta suppression.[40,41] Lubar noted a decrease in hyperactivity with reduced theta and increased SMR, with less of an effect on attentiveness. Again, the effects were reversible, showing statistically significant changes dependent on the stage of the design.

These early studies could be criticized largely for limited generalizability due to small sample size. Followups that are reported indicate sustained improvements in social and academic behavior for substantial periods of time after treatment, something that cannot be said for psychostimulants. The finding of apparent academic improvement is most interesting given the general lack of any such effect from psychostimulants.

Lubar and Lubar[42] published a group of six case studies that used a combination of reinforcement of lowered theta and increased SMR along with reinforcement of elevated beta amplitudes. Feedback was shut oft during moments of elevated theta activity, EMG activity or gross motor movements. Reading, arithmetic and spatial tasks were conducted concurrent with the feedback. Training was conducted twice weekly for 10 to 27 weeks. No reversal of conditions was done, as these were clinical cases. The children were described as having a variety of learning, hyperactivity and attentional difficulties. Letter grades improved and were sustained. The Metropolitan Achievement Test, Peabody, Stanford Achievement Test and California Achievement Test were administered pre and post treatment and systematic improvements were seen. EEG changes were noted in all children (decreases in theta, increases in SMR or beta). This methodology has become the basis for the most common clinical practice style today. This study demonstrates a clinically useful procedure that produces consistent results and likely promotes generalization to real world situations.

In the same era as the initial Lubar studies a private practice psychologist, Michael Tansey, Ph.D., published his first report on improvements in ADHD following neurotherapy using a narrow band of EEG centered on 14 Hz.[43] His patient was diagnosed *perceptually impaired" and hyperactive and had been on Ritalin since age 7. The boy was in a special education class for fourth grade, which he had failed and was scheduled to repeat fourth grade. Tansey used 3 initial EMG training sessions followed by amplitude and frequency modulated auditory feedback to increase 14 Hz (SMR band). Tansey and Bruner[43] indicate Ritalin was stopped by the pediatrician who no longer saw him as hyperactive prior to the onset of EEG treatment, but after the three EMG sessions. The family moved and placed the boy in a normal fourth grade class because of the improvement in reading comprehension and behavior seen over the summer. Evidence of remediation was the boy's grades, which were 3 As, 3 Bs and 4 Cs the first quarter, improving to 4 As, 5 Bs and 1 C by the end of the third quarter. A 10 year follow-up on this boy showed he had successfully completed high school, was attending college and had remained medication free.[44]

A single case study like this naturally raises many questions of causality. Nonetheless, one has something of a time series design, with 3 years of special education producing failing results and a sudden, post treatment turn of events. The stability of the reported improvement is encouraging. The studies of this era were carefully reviewed by Joel Lubar in 1991.[45]

The 1990s – Contemporary Studies

Lubar, Swartwood, Swartwood and Timmerman[46] reported a series of studies further defining the QEEG and auditory event related potential (AEP) characteristics of ADHD, methylphenidate effects and the efficacy of neurotherapy. The AEP and methylphenidate literatures are complex and beyond the scope of the present review.[47] Briefly, these authors found no significant effects of Ritalin on theta/beta ratios. Lubar et al.[46] also reported habituation of P2 ERP amplitude in ADHD children that may correspond to the degradation of their performance on continuous performance tasks. Of most interest to the present review is the fourth study in this report. Seventeen children diagnosed with ADD/HD were given 30 to 45 sessions of neurofeedback training to decrease theta and increase beta activity. Two groups emerged: 11 were successful in either lowering theta percentage or power or increasing beta percentage; 6

were unable to produce these changes in their EEGs. As a result, the 11 successful learners showed reduced theta/beta ratios, compared with the unsuccessful learners whose theta/beta ratios did not change over their baseline recordings. Interestingly, the lowered theta/beta ratios appeared over a wide range of scalp locations, not just in the region from which the feedback had been derived, suggesting global activation of the EEG can occur as a result of neurofeedback at a single placement. Unfortunately teacher or parent report data for this study group (successful learner versus unsuccessful learner) were not presented.

Lubar, Swartwood, Swartwood and O'Donnell[48] presented data on 19 participants in an intensive summer neurofeedback training program. Patients were given daily 1 hour training sessions for 8 10 weeks, with the goal of accomplishing 40 training sessions during the summer months. Neurofeedback was given during two 5 minute onscreen periods, followed by a 5 minute reading and 5 minute listening periods during which auditory feedback was given simultaneous with the cognitive task. A key methodological point future researchers should pay attention to is that all neurofeedback therapists had bachelors or master's degrees in health care or education and at least 1 year experience providing neurotherapy for ADHD.

Outcome measures were theta amplitude, TOVA continuous performance test, WISC-R and Attention Deficit Disorder Evaluation Scale (ADDES). Twelve of the 19 patients showed significant lowering of theta across sessions. These 12 showed improvement on an average of three TOVA scales, while the group that showed no lowering of theta improved on an average of 1.5 TOVA scales. Pre-post ADDES showed significant improvement ($p<.001$) for inattention, hyperactivity and impulsivity for all patients. The criticism that such improvements are merely the result of parents reporting improvement simply because of the length and intensity of treatment is not intuitive. One might expect that parents who saw no improvement after substantial investment of time and money might well be expected to have quite the opposite reaction if there were not substantial changes.

WISC-R tests were administered post treatment by an independent neuropsychologist for 10 children who had WISC-R data from approximately 2 years prior to treatment. All children in this group showed reductions in theta activity during the course of their neurotherapy. Significant improvements ($p<.005$) in Verbal, Performance and Full Scale IQ were found. Mean Verbal IQ increased from 113.3 to 122, Performance from 109.6 to 116.1 and Full Scale from 112.4 to 122.1.

The objective reduction in theta amplitude in about two thirds of the cases suggests neuromodulation was taking place. The fact that patients who did not show theta or beta change still apparently improved behaviorally may be accounted for by changes in the cortical EEG that were not measured, e.g., coherence (known to be abnormal in ADHD), or by subcortical change that cannot be observed by scalp recorded EEG. Further, TOVA data are known to be stable on retest and if anything, second TOVAs may produce lower scores due to boredom,[47] so the TOVA data is supportive of a treatment effect. The improvement in Wechsler IQ scores is consistent with a globally more activated, less 'lazy," brain.

Alhambra et al.[49] reported on the results of 20 sessions of neurofeedback on TOVA and QEEG measures in a case series. Questionnaires were also sent to patients who received at least 30 sessions of training. Decreased theta and increases in beta or SMR were reinforced. Feedback was provided by a PacMan style display, where meeting threshold criteria on the relevant EEG measures caused PacMan to advance. Sessions were 30-45 minutes long. Of 43 surveys sent, there were 36 responses, 26 for males and 10 for females. Of the 36 respondents, 31 (86%) showed "some overall improvement" from the treatment. Improvement was judged "significant" in 30 of these. Three respondents noted no improvement and 2 were uncertain.

Rossiter and LaVaque[50] compared 23 patients who received EEG biofeedback as part of their treatment for ADHD with 23 age matched controls drawn from a pool of patients in the author's practice who were treated with psychostimulants and did not receive neurotherapy (MED group). This is an important comparison, since for general acceptance neurotherapy must be shown to have at least equivalent effects to those of the stimulants, with some additional advantages such as a long lasting effect after treatment and no negative side effects. Five of the EEG group were taking psychostimulants before and during the course of the treatment. All participants discontinued medication 2 days prior to pre and posttesting consisting of EEG measures and the TOVA. Behavior Assessment Scales for Children (mothers' ratings) were obtained for the EEG group, pre and post treatment. Pretest intelligence testing was obtained. Analysis revealed the groups to be well matched, with no significant differences between group in age, gender distribution, intelligence, frequency of ADHD vs 'undifferentiated" ADD, frequency of secondary/tertiary diagnoses, or LD/EBD placements. The groups did not differ on baseline TOVA measures. More of the EEG (N=17) than the MED group (N=10) had been treated with psychostimulants previously.

The EEG training protocols were varied depending on age, symptoms, baseline results and responses of the patient to the treatment. Generally, 4.8 Hz theta was trained down in children through adolescents, while 12-15 Hz SMR and/or 16-20 Hz beta was trained up in adolescents and adults. Twenty sessions of EEG training were completed prior to retesting. Posttreatment TOVA scores indicated highly significant improvement for both the EEG and medication groups on all scales. The BASC scales also demonstrated highly significant improvements on all scales.

The medication group improved significantly and the size and significance of the changes were not significantly different from the EEG group. Members of the EEG group were retested without medication; the MED group was retested during the optimum "window" 1.5-2.5 hours after taking their medication. The study demonstrated good experimental control of a wide range of potential confounding variables. Comparison of the gains made by the two groups was limited to the TOVA test results, which are machinegenerated and less likely than rating scales or other tests to be subject to observer bias. If patients showed improvement after 20 sessions, another 20 sessions were recommended to consolidate and "overlearn" the skills. This procedure has become a common practice model in neurotherapy.

Linden, Habib and Radojevic[51] have reported the results of a waiting list control study of the effects of neurotherapy. Eighteen children were randomly assigned to either the experimental group or a waiting list control condition. No psychotherapy patient received any medication or other during the study. Forty twice weekly 45 minute sessions were oven to the experimental group across 6 months. Participants received 10 minutes of visual and auditory feedback in a video game-like format, 10 minutes of auditory feedback during reading and 10 minutes of auditory and visual feedback while listening to age appropriate material being read to them. Reductions in 48 Hz theta and increases in 1620 Hz beta were reinforced. Recording was from a bipolar montage at Cz/Pz. The treatment group showed a significant 10 increase (mean 101.1 increased to 110.4, p=.02 relative to controls 99.1 to 100.0, n.s.) on the KBit 10 Composite. The treatment group also showed reduced inattentive behaviors pre to post (p=.04), but the groups did not differ on aggressive/defiant behaviors.

This study supports Lubar's 1995 study[48] that documents an apparent 10 increase following neurofeedback treatment, consistent with a generally improved cortical functioning.

Another single case study with similar IQ results was reported by Tansey.[52] One would like to have seen preposttreatment data for a continuous performance task in the Linden study in order to compare these results with previous studies.

Rossiter[53] reports essential normalization on the TOVA test following 30 sessions of home-based, patient-directed training in six patients. Patients received 10 clinic sessions during which they or their parents were trained to run the Lexicor POD-2 software. Feedback was aimed at increasing 15-18 Hz beta or 12-15 Hz sensorimotor rhythm and reducing amplitudes of delta, theta and 22-30 Hz beta.

Boyd and Campbell[54] reported improvements on WISC-III Digit Span and TOVA Inattention and Hyperactivity scales in five of six students. The students received SMR training during twenty 30-minute training sessions conducted in a school environment. Operational problems in either a home or school environment may prove daunting: training, time commitment, equipment issues can arise. Nonetheless, out-of-clinic training is an interesting possibility.

Further research should address several issues: the potential importance of basing training protocols on deviations from normative databases, the relevance of abnormal QEEGs during tasks, and the mechanisms of action of neurotherapy. Appropriate controls might be designed by random assignment of children/families desiring neurotherapy to an initial 20 sessions of neurotherapy or an initial 20 sessions of twice weekly cognitive therapy and family therapy, in a crossover design. A blind "placebo" controlled study could easily be designed, for example, by attaching an EMG electrode to the shoulder region of all patients and feeding back the EMG signal instead of the EEG signal to the controls unknown to the neurotherapist or the patient. Ethical questions arise from false-feedback designs, due to the commitment required from the children and their families and the potential for discouragement. Ideally, participants in a study would be off medication entirely for the course of the study. Precast IQ, QEEG, auditory and visual CPU, and parent/teacher ratings should be obtained. Follow-up of a year or more would be very desirable.

SUMMARY

Significant public health concerns exist regarding our current level of success in treating ADHD. Medication management is very helpful in 60-70% of patients. Side effects, lack of compliance and the fact that stimulant medications cannot be given late in the day limit the benefits largely to school hours. While stimulants improve behavior and attention, less of an effect has been noted on academic and social performance. Continuing concerns exist about long-term safety, and studies on long-term cardiovascular and neurophysiological effects have not been carried out. Neurotherapy for ADHD offers an effective alternate for patients whose treatment is limited by side effects, poor medication response and in cases in which the patients and/or their parents refuse to consider medications. Studies indicate clinical improvement is largely related to measurable improvements in the EEG signature, evidenced by declining theta/beta ratios over frontal/central cortex and/or reduced theta/alpha band amplitudes.

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