

Research Recognition Award Paper

Discourse on the Development of EEG Diagnostics and Biofeedback for Attention-Deficit/Hyperactivity Disorders¹

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This article presents a review of work that my colleagues and I have been doing during the past 15 years developing a rationale for the diagnosis of attention-deficit/hyperactivity disorder (ADHD) and treatment of ADHD employing EEG biofeedback techniques. The article first briefly reviews the history of research and theory for understanding ADHD and then deals with the development of EEG and event-related potential (ERP) assessment paradigms and treatment protocols for this disorder, including our work and that of others who have replicated our results. Illustrative material from our current research and child case studies is included. Suggestions for future experimental and clinical work in this area are presented and theoretical issues

¹Over the years, many people have been involved both in my laboratory and at Southeastern Biofeedback Institute working with me in developing this area; I wish to acknowledge some of them. They are specifically Dr. Margaret Shouse and Dr. Chris Mann, who have been involved in the initial and recent stages of my research; Ms. Jennifer Samples, who has worked with us in the Institute for many years and has helped us in training many of the children that have benefited from EEG biofeedback. I would especially wish to acknowledge the skill and dedication of Judith O. Lubar, of Southeastern Biofeedback Institute, who has worked with me clinically in terms of developing treatment protocols for ADHD biofeedback and who has trained many of the children who have successfully completed EEG Biofeedback. I would like to acknowledge the generous help of the Lexicor Corporation of Boulder, Colorado who have provided support and instrumentation for recent studies in this area. Mr. Rod Bunn and Mr. Robert Muenchen, who have provided computer support, programming, and statistical assistance in evaluating data in various studies, are gratefully acknowledged. Some of this research was supported by a grant under the ESEA Title IV-C Program for the handicapped. I also gratefully acknowledge Children's Hospital of Knoxville, TN, who have provided essential contract support for our laboratory at the University of Tennessee.

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involving the understanding of the neurophysiological and neurological basis of ADHD are discussed.

KEY WORDS: ADHD; EEG biofeedback; children; event related potential.

In this article, I trace the work that I have done in association with my colleagues during the past 15 years to develop EEG neurodiagnostic procedures and establish the use of EEG biofeedback training for working with the attention-deficit/hyperactivity disorder (ADHD). The history of diagnosis and treatment of ADHD is long and complex. This is one of the most perplexing and pervasive disorders of childhood. At least 20% of children with ADHD continue to experience this disorder as adults. It is a condition that has been estimated to affect between 5% and 15% of the population depending on which review is cited and how the disorder is classified (Rie & Rie, 1980). I examine this disorder from different viewpoints rather than presenting an exhaustive review of the literature. It is important to discuss the problem both from a neurological and a behavioral context and show that there are EEG and event-related potential (ERP) differences that distinguish ADHD children from those without the disorder. These findings provide a firm basis for employing EEG feedback training as a potentially powerful modality for helping children ameliorate many symptoms of ADHD including improving learning skills, concentration, focusing attention, and reducing hyperactivity.

HISTORICAL PERSPECTIVE

Very little had been documented regarding ADHD during the previous century. One of the first accounts was a paper by an English physician, G. F. Still, published in the journal *Lancet* in 1902. He characterized what we now call ADHD as "abnormal defects in moral control . . . and wanton mischievousness and destructiveness." In the early part of the 20th century disruptive, hyperactive, and impulsive behavior associated with poor attention span was still primarily considered to be a defect in moral development. In the 1920s after the severe influenza outbreak of 1918, Hohman (1922), Kennedy (1924), and Stryker (1925) noted that antisocial behavior and behavior associated with decreased attentiveness, impulsiveness, and hyperactivity developed in a number of children who had influenza-related encephalitis.

In the 1930s research had led to the conclusion that there were a series of disorders of children, which might or might not be related, but

which seemed to all involve some type of minimal brain dysfunction syndrome. Some of these occurred as a result of brain injury and were associated with mental retardation. Others were associated with toxic reactions to heavy metals such as leaded paint. There may be genetic factors in that blood relatives of a child with ADHD including parents, grandparents, and cousins also have varying degrees of this disorder. In some cases perinatal complications were associated with behavioral dysfunctions, hyperactivity, and poor attentiveness later in life.

In an attempt to combine all of these factors under one rubric, the concept of minimal brain dysfunction syndrome (MBD) was developed by Strauss and Lehtinen (1947). The MBD syndrome was also referred to as the "Strauss syndrome" at that time. Some children appeared to exhibit decreased arousal rather than hyperkinesis. The first pharmacological attempt to treat this symptom complex was by Dr. Charles Bradley, who in 1937 administered amphetamine sulfate to some children. He found that there was a dramatic improvement in school-related behaviors and an increase in attentiveness. These effects were very drug and dosage dependent. At that time it was felt that stimulants simply increased arousal, perhaps through brain stem reticular activation. We shall return to this concept later.

During the 1940s and 1950s, psychometric measures began to be used to help differentiate children with MBD from normal populations. At that time such measures as the Bender Visual-Motor Gestalt Test, and the Stanford-Binet were the most popular metrics. Later, other tests were incorporated such as the Weschler Intelligence Scale for Children (WISC) and the revised version of this test (WISC-R), and a number of neuropsychological test batteries such as the Halstead Reitan and the Luria-Nebraska Neuropsychological Battery. More recently the Woodcock-Johnson Test of Cognition and Achievement has been employed in helping to differentiate children with these disorders from controls.

During the time that the concept minimal brain dysfunction syndrome was most popular neurologists searched for markers that could be employed in helping to clarify this diagnosis. As early as 1938 Jasper, Solomon, and Bradley presented evidence that there were EEG abnormalities in MBD children primarily in terms of slowing in the EEG. Later Knott, Platt, Ashby, and Gottlieb (1953) showed that there were EEG abnormalities in children with behavior disorders as well as those that later on developed psychopathic personality disorders. Some of these abnormalities were in the form of epileptogenic activity as well as generalized slowing. A number of other individuals such as Werry, Delano, and Douglas (1964), Cohn and Nardini (1958), and Pavy and Metcalf (1965) have also reported EEG abnormalities. Even though these early neurological studies helped to estab-

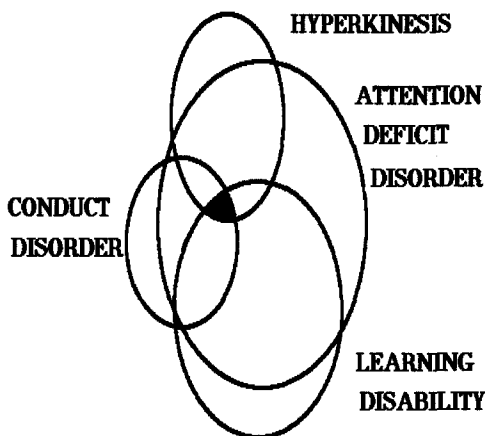


Fig. 1. Schematic diagrams showing the interrelationship between hyperkinesis, attention-deficit disorder, learning disability, and conduct disorder, emphasizing both the independence and overlap between them.

lish the concept of an MBD syndrome, the latter appeared too broad in that there were too many subtypes of this disorder to place under one classification. By the early 1970s it became clear that there were several different disorders that needed to be dealt with in more detail. These were specifically the hyperkinetic disorder, specific learning disabilities (LD), disorders of conduct, and disorders of attention.

In the most recent version of the Diagnostic and Statistical manual of the American Psychiatric Association (DSM III-R, 1987) four main categories were established: attention-deficit disorder with and without hyperactivity, conduct disorder, and learning disabilities. There are many subtypes within the general framework of learning disabilities divided according to hemispheric localization. One categorization by Hooper and Willis (1989) subdivided LD into a large number of categories involving sensory and recognition, perceptual including auditory, visual, tactile-kinesthetic, psycholinguistic disorders, and academic disorders. The latter encompasses different academic endeavors including mathematics, reading, spelling, writing, and cognitive intellectual disorders. There have been so many classifications of learning disability subtypes that it is beyond the scope of this article to review them in great detail. The reader is referred to the Hooper and Willis (1989) book as well as a recent book on right hemispheric learning disabilities by Pimental and Kingsbury (1989).

In order to use neurodiagnostics based on EEG analysis to determine appropriateness of EEG biofeedback as a modality for treating this disorder, it is important to know which of the major disorder categories best fits the individual. Figure 1 is a simplified diagram showing the relation among four disorders: attention-deficit disorder, hyperkinesis, conduct disorder, and specific learning disabilities. A child may fall into any one or a combination of these categories. Virtually all children who are hyperkinetic also experience Attention-Deficit Disorder. However, there are many children who have a pure attention-deficit disorder and yet are able to sit still for long periods, but have a short attention span and poor focusing and concentration skills. Similarly, there are many children with specific learning disabilities whether they be experiencing dyslexia, dyscalculia, or some of the right hemispheric syndromes such as aprosodia (a lack of the ability to understand the emotional content of language somewhat related to alexthymia) who are nevertheless able to concentrate and selectively focus their attention. Finally, there are children who have a conduct disorder (CD) which may or may not be related to neuropathology such as temporal lobe seizures or dyscontrol syndromes related to deep temporal lobe disorders. CD in children may be purely behavioral; that is, it may result from their being members of dysfunctional family systems, who, nevertheless, are neither hyperkinetic nor learning disabled nor have attentional problems.

It is of the utmost importance to be able to provide an accurate differential diagnosis for an individual before embarking on treatment, because the treatment undertaken might be very different depending on where on this map (Figure 1) one falls. Children with pure ADHD respond extremely well to EEG biofeedback training. Children with hyperkinesis, especially if they are good responders to stimulant medication, are also candidates for biofeedback treatment, but they may also require medication at least during the initial part of the treatment in order to obtain good control of their disorder. Children with specific learning disabilities appear to benefit less from biofeedback treatment as a primary modality; however, it may be a useful adjunctive technique when used with specialized educational approaches designed for their specific learning disorders. Children with (CD) often benefit most from a combination of behavior and family therapy approaches. Biofeedback probably plays a much more minimal role in treating CD than in the other three disorders. Psychometric, neurodiagnostic, rating scale, neuropsychological, and interview techniques are the best approaches for a prescreening of individuals to determine where they fall in this complex of disorders.

INITIAL RATIONALE FOR EEG BIOFEEDBACK RESEARCH AND TREATMENT

I would now like to trace in somewhat more detail the way in which we developed the use of EEG biofeedback for working with individuals with attention-deficit/hyperactivity disorders both with and without hyperkinesia and/or associated learning disabilities. In the early 1970s, a group of investigators in California developed a very elegant and simple theory for explaining the hyperkinetic syndrome. In two papers, one published by Satterfield and Dawson (1971) and another by Satterfield, Lesser, Saul, and Cantwell (1973), a hypothesis that became known as the "low-arousal hypothesis" of hyperkinesia was proposed. Based on measures of electrodermal activity, cortical evoked potentials, and behavioral observations of children with hyperkinesia, these researchers proposed that such children behaved as though they were experiencing a state of decreased sensory arousal. Essentially, they acted as if there were a type of "filter" blockading the impact of sensory information in each of the sensory modalities. This would mean that visual, auditory, somesthetic, vestibular, and gustatory inputs would not have the impact or the reinforcement value that they should. When children were placed in a room with very few objects except those representing each of the sensory modalities, a number of interesting behaviors would emerge. Objects that could be tasted were placed in the mouth, objects that could be shaken to produce noise were placed near the ears, those that were interesting visually would be stared at, others would be rubbed on the skin. A child would often spin around or jump around as if trying to increase the vestibular stimulation. After a flurry of hyperkinetic activity associated with sensory stimulation, very often the child would fall asleep.

Satterfield and his colleagues proposed that because of their low arousal, these children easily habituated to sensory stimulation and therefore constantly sought stimulation. This could explain the disruptive behavior often seen in hyperkinetic children both in and out of the classroom. They also suggested that there was a problem with reticular activation. Perhaps this low arousal was due to an abnormality in adrenergic neurotransmitter production or its utilization within the reticulo-thalamic axis. This axis includes the pontine and mesencephalic reticular activating system and the diffuse thalamic projection system, which acts to inhibit the former (Jasper, 1960). This dysfunction would also be reflected in disinhibition of the prefrontal lobes. Satterfield's hypothesis helped to explain the positive effects obtained by Bradley (1937) with amphetamine sulfate.

By the early 1970s it was well known but not well explained that stimulants had a paradoxical effect on hyperkinetic children. These children

tended to become less hyperkinetic and perhaps even showed improved concentration in classroom settings. Satterfield and his colleagues' hypotheses now make even more sense.

In the history of science there is a well-known philosophical rule known as Occam's Razor. Proposed by William of Occam in 1336, it states that "entities should not be multiplied unnecessarily." This is interpreted as requiring that the simplest of competing theories be preferred to complex explanations and that explanations of unknown phenomena be explained in terms of known quantities. The point is that a complex of behaviors as difficult to understand as hyperkinesis might be very easily explained by the relatively simple notion that these children are in a state of decreased arousal and that the use of sympathomimetics that increases arousal often decreases hyperkinetic behaviors.

After reading the work of Satterfield's group, it occurred to me that there may be a very simple neurological explanation for why these children act as they do. This explanation is based on the very well known early neurological studies by Berger in the 1920s and by Jasper, Soloman, and Bradley (1938). When one is resting, dominant EEG activity is in the alpha and theta range; but when one becomes excited, activity begins to shift toward the beta region above 14 Hz. My first hypothesis was that children with hyperactivity, especially where there was an attention deficit, might be less able to produce beta activity above 14 Hz, and would be experiencing excessive slow activity primarily in the theta region of 4-8 Hz.

In 1973 when I began to develop this view I also became aware of M. B. Sterman's work with sensorimotor rhythm (SMR) training for the management of seizures. During 1975 and 1976 I spent 9 months in Dr. Sterman's laboratory and at the same time was carrying out studies in our own laboratory using SMR training to help control seizures. In 1975 A.R. Seifert and I published our first article dealing with the reduction of seizures through EEG biofeedback training. In 1976 I published a series of extended case studies (Lubar & Bahler, 1976) showing that SMR training was very effective in reducing seizure activity, thus in part replicating Sterman's work and extending it further.

At the time that we were beginning to work with seizure patients using the SMR paradigm I observed that some of these seizure patients who were high school or college students had experienced increased attentiveness and were able to focus and concentrate better. Next, I engaged in a study with a former graduate student, Margaret Shouse, to determine whether EEG biofeedback training employing SMR might also be a viable modality for helping children with hyperkinesis. In 1976, we published a case study showing that there were EEG and behavioral changes in an 8 year 11 month old hyperkinetic child following SMR training with blockage

of reinforcement for SMR when 4-7 Hz theta was also present (Lubar and Shouse, 1976). This study had six components. We first measured the amount of SMR, theta, and chin EMG with and without methylphenidate (Ritalin). During SMR training the child tripled his production of SMR and submental EMG decreased by almost 50%. During this same period of time, this child showed a decrease in classroom measures of self stimulation, object play, and out-of-seat behavior and he also increased his sustained attention and sustained school work. He showed a decrease in oppositional behaviors and an increase in cooperative behaviors. These observations were made by two independent observers in the classroom. The child was not aware that they were there.

After the child was trained to produce SMR and inhibit theta we reversed the procedure in a blind fashion and now trained him to inhibit SMR and increase 4-7 Hz theta. Over 35 sessions the child regressed back to baseline levels of SMR and theta production with a concomitant deterioration of school performance. His out-of-seat behavior increased, he became more uncooperative, his attention span decreased, and his object play and self stimulation increased; the child had essentially lost all the previous gains. We then switched the child back to the initial condition of SMR production with theta inhibition for another 28 sessions. During this time the child regained the previously obtained successes and his school performance again significantly improved for all of the measures taken. The final portion of study involved removing the medication to see if the child could sustain this behavior. He was able to do this without any medication. Long-term follow-up over several years showed that the child maintained these gains and continued to do very well in school without medication.

Following this initial study we then published more extensive studies (Shouse & Lubar, 1978, 1979; Lubar, 1977), which employed more children using the same paradigm. In total, these early studies showed that the primary effect of SMR training was to improve the motor component of hyperkinesis with less of an effect on the attentive component, and that these gains were reversed when an ABA design was used. Statistically significant differences during the various phases of the study correlated with the type of reinforcement given. This blind crossover study provided the *first* clear evidence that EEG biofeedback training employing SMR with theta inhibition was a powerful modality for working with the hyperkinetic disorder.

After completing these controlled studies, I began treating hyperkinetic children in a clinical setting using EEG biofeedback training. In our initial clinical work in 1976, I employed an Autogen 120. In 1977 we obtained a Neuroanalyzer (Neurofeedback Instrument Inc.) similar to that used by Serman in his work with seizure patients, and used it with both seizure disorders and hyperkinetic children. In my work with the Autogen

120, I observed another phenomenon. Children who had attentive difficulties and problems in reading or spelling but were not hyperkinetic appeared to produce excessive theta activity in the 4-8 Hz range and were particularly deficient in beta production. In my early work with these children, I would observe them both during baseline conditions and while reading material at their grade level. Based on these early observations and my basic knowledge of the relation between higher frequency activity and activation in the nervous system, I hypothesized that perhaps children with attentional difficulties, especially if they had reading, spelling disorders, or other associated learning problems, might benefit from beta training as well as SMR training.

When we obtained our Neuroanalyzer, we initiated a paradigm that involved training children first to produce SMR with inhibition of theta activity and then to produce beta activity with inhibition of theta activity. We chose as our bandpass for beta 16-20 Hz. During the period from 1976 to the early 1980s I collected data on a number of patients and noted that not only did they learn to better control hyperkinetic behaviors with subsequent reduction of stimulant medications in many cases, but they also improved markedly in their ability to focus and concentrate. Many earned improved grades following SMR and beta training with theta and EMG inhibition. In 1981 I wrote a book with a pediatric neurologist William Deering of the Gunderson Clinic in La Crosse, Wisconsin which covered research and clinical work by our group and others (Lubar & Deering, 1981).

In 1984, Judith O. Lubar and I published a paper on six case studies which showed that a combination of SMR training followed by beta training with theta inhibition produced significant and sustained improvements in school performance and psychometric measures. We used the Metropolitan Achievement Test, Peabody, Stanford Achievement Test, and California Achievement Test for pre- and post-training assessments. Letter grades (GPAs) improved markedly. In that study learning curves were presented for 6 children showing that there was a significant improvement in both SMR and beta production with attendant decreases in the percentage of theta over many sessions.

During the period from approximately 1980 to the present we have seen over 250 children in our clinic, Southeastern Biofeedback Institute, with the diagnosis of attention-deficit/hyperactivity disorder with or without hyperactivity.

For example, in 1984, 37 of these 250 children who had been treated by EEG biofeedback for attention-deficit disorder were involved in a demonstration project in Knox County and surrounding county schools. They were compared with 37 matched controls in terms of age and IQ (ages

8-12) who received only resource classroom training for reading disabilities associated with ADHD. The children receiving biofeedback training to increase beta and decrease theta in addition to the resource training showed significant improvements compared with controls in grade point average and significant improvement in terms of Metropolitan Achievement Test Scores ($t = 2.21, p < .05$). The group of 37 children who received EEG feedback were followed for an additional year and continued to show grade point average improvements that were better than 1.5 GPA levels as compared with the children who did not receive the biofeedback training but only resource training. Overall, the 80% of the 250 children who we have worked with either in conjunction with school programs or in the clinic have shown grade point average improvements of approximately 1.5 levels (range 0-3.5).

During the last year, we have specifically been providing pre- and post-psychometric assessments employing a number of measures for all children seen clinically for EEG biofeedback. These include the WISC-R, the Woodcock Johnson, and the Wide Range Achievement Test revised (WRAT-R). Increases in IQ measures average 12 points for WISC-R verbal IQ and 8 points for performance IQ for nine cases completed with psychometric testing. The highest increase that we have seen so far is 25 points in one 13 year old male who has been trained employing the recently available Lexicor Biofeedback System. Currently we have seen new cases ages 8-17 who have received pre-training psychometric assessments and topographic brain maps. These children are currently being trained and will receive post-training psychometric testing and post-training topographic brain maps.

We have now had the advantage of being able to follow some of these children into adolescence and early adulthood. The question that is continually asked is whether the positive effects of training are permanent or temporary. I strongly believe that gains are permanent if the biofeedback skills are combined with academic training and incorporated into the classroom setting. The children then build on what they have achieved and continue to do better. The whole process becomes self-reinforcing not only within the school in terms of better grades but also within the family in terms of praise from parents and better family adjustment. Overall, the whole training program has a cascading effect in terms of positive outcomes.

Until the early 1980s, I felt that we had perhaps stumbled onto something very important in this work and were helping many children, but few people were trying to replicate our work. However, replications began to appear and many more are now in the process of occurring. Michael Tansey

and his colleagues have published a series of papers showing that SMR and beta training provides a powerful modality for helping children with learning disabilities and hyperactivity (Tansey & Bruner, 1983; Tansey, 1984, 1985a, 1985b, 1990). In his work he used a paradigm very similar to the one that we developed in the 1970s, that is, training activity primarily at 14 Hz, with simultaneous inhibition of slow activity in the 7-Hz range. He uses a 6.5×1.3 -cm electrode placed over the central cortex, whereas our placements using standard EEG electrodes (Grass Instrument Co.) have been either over frontal temporal locations (F7-T5, F8-T6 or F3-P3, F4-P4) for beta training or central temporal locations (C1 - C5) for SMR training. Later I will cover in more detail the topographic distribution of activity in children with attention-deficit/hyperactivity disorder and discuss further electrode location.

Carter and Russell (1985) showed that EMG biofeedback can be helpful for children with learning disabilities. The use of EMG as a modality is not unreasonable since ADHD and LD children often experience excessive motor activity. SMR training which is designed to help inhibit motor activity produces decreased EMG. Carter and Russell have also trained children to produce beta activity and reduce theta activity with larger increases in verbal IQ than performance IQ with left hemispheric beta training and larger increases in performance IQ than verbal IQ with right hemisphere training. This work is particularly important because it shows that the biofeedback effects can be hemisphere specific. Currently Othmer (personal communication) has been training children using our beta/theta paradigm with both changes in school performance and psychometric scores as well as obtaining EEG learning curves.

At the present time I am involved in coordinating a multicenter study employing beta and theta training. During the next 2 years it is hoped that more than 100 children will be trained using this paradigm with attendant psychometric evaluation before and after training and pre- and post-training topographic brain mapping.

I would next like to turn to two other important areas. The first encompasses a recent series of studies that we have carried out showing that children with ADHD and LD differ from controls in terms of event-related potential measures (ERP). The second is a recent study that we have just completed based on 16 channels of topographic brain mapping. This latest study shows that there are very significant differences in many cortical areas that differentiate children with pure ADHD from controls. I will also present some very recent case examples using a newly developed EEG feedback system.

EVENT-RELATED POTENTIAL STUDIES

The event-related potential (ERP) provides different information about the way in which the brain processes information than does the EEG. Although the EEG does contain information that relates to specific stimulus events, it primarily reflects changes in state as reflected by levels of awareness from deep coma to extreme alertness and therefore is extremely useful in the diagnosis of a number of neurological dysfunctions. The ERP can provide some information on how the brain processes *specific* stimulus information. In studying the event-related potential the EEG is time locked to the stimulus presentation and averaged until a clear pattern emerges. The electrical activity is recorded from the time the stimulus is presented to approximately one second or longer after the stimulus presentation.

During the past 5 years, we have carried out 3 studies in which we compared the ERPs of children with attention-deficit/hyperactivity disorder who also experienced reading difficulties with gifted children and matched controls. In our first study (Lubar, Gross, Shively, & Mann, 1990) we showed that a simple auditory stimulus presented using an "oddball" paradigm could lead to significant differences between groups. The target stimulus was of a high tone presented randomly 10% of the time; the common stimulus was a low tone. The target tones were randomly presented but intermixed with 300 common tones. EEGs were averaged for approximately 180 ms before the stimulus presentation and continued for 900 ms post-stimulus. Recordings were obtained from FZ, CZ and PZ, along with recordings of eye movements and EMG, which were used to provide artifact-free trials for averaging.

This study showed that there were significant differences between the ADHD-LD group and the other two groups in terms of the P2, P3, and late component amplitudes. Furthermore, the ADHD-LD group made significantly more errors to the target stimulus than either the control or gifted children. The ADHD-LD group differed significantly from the other two groups in terms of lower IQ scores as well. The P2 and N1 components of the ERP are believed to be associated with stimulus selection, based on the work of Picton and Hillyard (1974). Danier et al. (1981) and Warran and Karrer (1984) have proposed that increased amplitudes of an ERP are reflective of attentiveness. In our study it was found that the ADHD-LD groups differed primarily from the other groups in terms of a decreased P3 component and poorly developed late components, particularly at FZ. It is believed that the P3 is associated with understanding the meaning of the stimulus as compared with stimulus detection, whereas the P2 is involved in stimulus detection. The earlier components such as the N1 are involved primarily in selective attention. For all of the measures, it was clear that

the ADHD-LD's difficulty was with both stimulus discrimination and general attentiveness. Our work confirms and extends very early work by Satterfield and his colleagues (cited previously), who also showed that there were ERP differences between hyperkinetic children and controls.

The second study (Lubar, Mann, Gross, & Shively, 1991) involved a semantic stimulus. In this task children were asked to differentiate between nouns and animal names. When the target stimulus (animal names) were presented the child was required to press a button signaling that he understood the meaning of the stimulus, and the ERPs were averaged to common and target stimuli. Differences between the ADHD-LD and the other groups again occurred, particularly for P3 and the late components. Differences were also found in the N1 component. The results were very similar to those obtained in the auditory study, in that the ERPs associated with either attentive state or stimulus selection and comprehension were poor in the ADHD-LD group. Again the ADHD-LD children made significantly more errors in their response to target presentations than the other two groups. Many large significant differences were found in this study at locations FZ, CZ, and PZ. Most of the differences concerned the P3 component. However, for the P2 component significant differences were found between the gifted and normal groups as well as the normal and the ADHD-LD groups at F4. We also found differences between the gifted and normal groups for the central and posterior locations.

One of the reasons for using the unusual selection of stimuli for this study was to determine whether the ERP could become a useful tool for understanding cognitive processing. Since the results clearly indicated that there were many significant differences between the ADHD-LD group and the control and gifted groups in terms of their perception and evaluation of a cognitive stimulus, our results provided a basis for perhaps using the ERP in neuropsychological testing. There are many tests in which one is required to match a target stimulus to an array of stimuli containing the target stimulus (WISC-R), or in which one has to abstract the principle related to the organization of stimuli. The Halstead Reitan Battery Categories Test requires the latter. In the future we should be able to design ERP paradigms based on complex stimuli in order to better understand how the brain processes complex information and how this information processing relates to standard EEG measures of power, frequency, and amplitude.

In our third study, we evaluated the ability of children with ADHD-LD to perform an easy visual and a hard visual task. The easy visual task consisted of differentiating the letter O from the letter Z, where Z, the target stimulus, was presented 10% of the time. A novel stimulus, a random meaningless visual figure acting as a second target stimulus, was also pre-

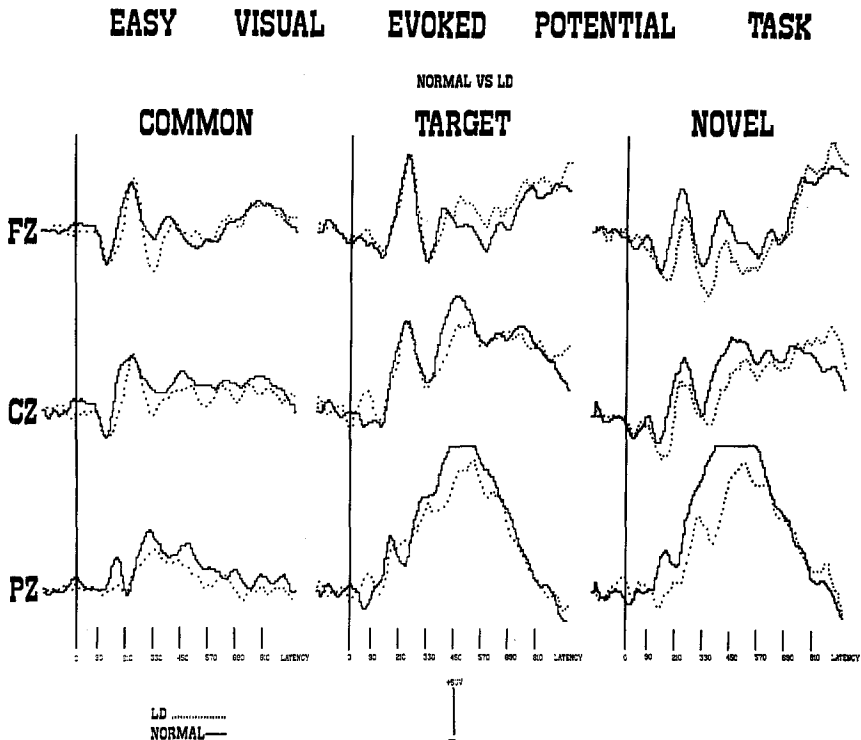


Fig. 2. Averaged event-related potentials (evoked potentials) for common target and novel stimuli for an easy visual task. The common stimulus was the letter O, the target stimulus the letter Z, the novel stimulus a meaningless visual figure.

sented. The hard visual task involved differentiating between the lower case letters b and d. These letters are often confused by children who have reading disabilities. Again a novel stimulus consisting of a meaningless visual pattern was employed 10% of the time.

Figures 2 and 3 show the averaged ERPs for common novel and target stimuli. Inspection of the figures clearly reveals that there are differences in the 300-500 ms post-stimulus region for the target and novel stimuli and to some extent there are differences for the common stimulus in the hard visual task at CZ. To briefly summarize, these differences are similar to those found in our other two studies for P3 and late components representing stimulus meaning and attention.

In terms of future directions, I propose that studies could be done to determine if ERPs also differentiate normal from highly gifted individuals or children with special areas of extreme talent, i.e., musical, artistic, or

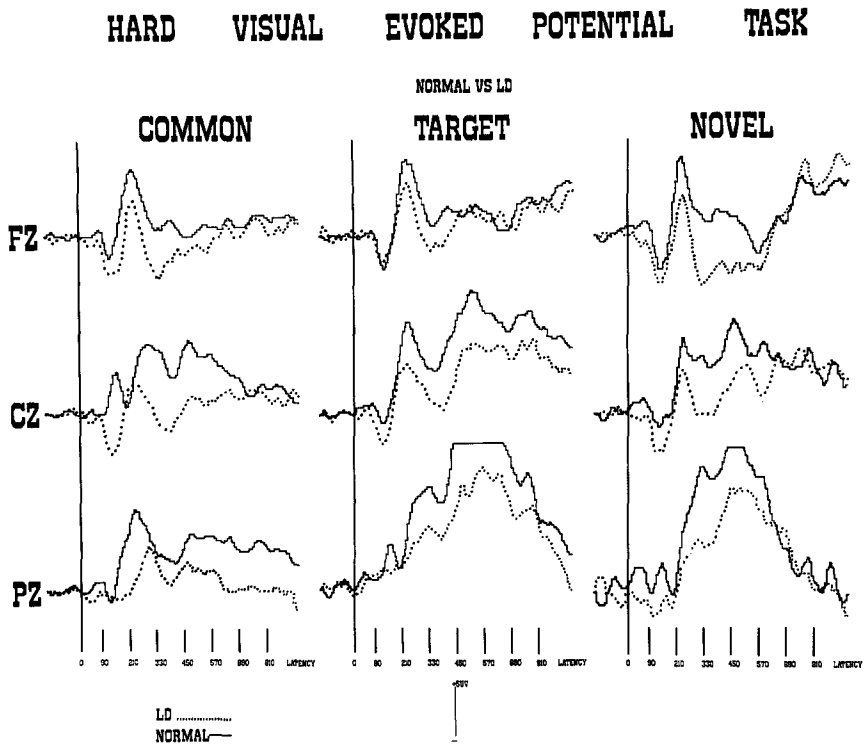


Fig. 3. Averaged event-related potentials for a hard visual task, the common stimulus was the letter b the target stimulus the letter d, the novel stimulus a meaningless visual figure.

mathematical. The field for brainstem-evoked potential studies is wide open, as very little has been done in this realm with LD-ADHD or highly gifted populations. I also propose that more complex stimulus paradigms be employed in the future in order to understand how LD children experience difficulties with certain types of information processing that might underlie dyslexia and dyscalculia or other specific learning disabilities.

TOPOGRAPHIC BRAIN MAPPING STUDIES

We have already seen that over the past few decades evidence has accumulated showing that children with ADHD, learning disabilities, and hyperkinesia may show differences in their EEGs as compared with matched controls. In the past five years we have carried out two studies that further clarified this issue. In our first study (Lubar, Bianchini, Cal-

houn, Lambert, Brody, & Shabsin, 1985) we examined the EEG of children with specific reading disabilities and attention-deficit disorders without hyperactivity under a variety of test conditions. These conditions included a baseline recording with eyes open, easy and hard reading tasks, easy and difficult arithmetic tasks, and a simple and more complex puzzle construction task. Recordings were obtained from six different cortical locations: frontal-temporal and central-temporal, and occipital-parietal for left and right hemispheres. We found large and significant differences between the groups in terms of excessive theta activity in the 4-8 Hz range. This study included 69 LD-ADHD children and 34 matched controls. Based on the results, it was possible to predict whether individuals fell into the LD-ADHD or control group based on a discriminant function analysis. The prediction was better than 97% when all the variables in the study were taken into account, and better than 80% when only variables concerning increased theta activity in frontal temporal locations were used as predictors. This was our first research study that provided a strong rationale for EEG biofeedback involving theta suppression.

In a very recently completed study, Chris Mann, a former doctoral student, and I examined the EEG over 16 locations using topographic brain-mapping techniques and accompanying statistics. The sample included 25 9- to 12-year-old right-handed males with pure attention deficit disorder (ADD) without hyperactivity or specific learning disabilities and 27 very carefully matched controls. There were no differences between the groups in terms of age or grade level. The groups did differ slightly but significantly in terms of verbal IQ scores; there were no differences in terms of performance or full scale IQ. The groups also differed significantly on the Wide Range Achievement Test (WRAT-R) revised for reading, spelling, and arithmetic. The ADD group had lower scores than the matched controls.

Sixteen exactly matched channels of EEG were recorded under three conditions: an eyes open baseline, a reading condition, and a drawing condition. The reading condition employed materials at grade level and drawing involved the copying of figures from the Bender Visual-Motor Gestalt Test. Using power spectral analysis and topographic brain mapping for display of the results, many significant differences were found. Specifically, increased theta activity was obtained in many locations particularly frontal and centrally, both in terms of increased absolute power and increased relative percent power. Decreased beta activity was found in many frontal and temporal locations. Many of the differences were significant at the .001 level. Based on the results of the new study, we obtained predictability of group membership of approximately 80% using a discriminant function analysis. Group predictability was higher for children within the study, but our results are

also predictive for children meeting the study's demographic criteria but who were not included in the study. This research (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1990) has been submitted for publication.

This new research is significant for several reasons: First it is the clearest demonstration yet that children classified as *pure* ADD without hyperkinesis, learning disabilities, or conduct disorder form a neurologically distinct group from controls. This research also provides a further rationale for treating children with pure ADD who are not on medication with biofeedback paradigms. We found that such children show greater increases in theta and decreased beta when challenged with academic tasks than during baseline readings. In many respects this is similar to the cardiac treadmill stress test model, in which some individuals show normal cardiac function at rest, but abnormalities develop under performance demands.

Another reason this particular study is important is because it correlates highly with a recent study published by Zametkin et al. (1990). Zametkin and his colleagues used positron emission tomography (PET) to compare regional brain glucose metabolism for 50 normal and 25 ADHDs who had childhood hyperactivity without any other neuropsychological disorder. The authors employed adults rather than children because of the concerns of exposing children to the relatively high levels of radiation involved in PET scanning. They found that the global metabolic rate was 8% lower in adults with hyperactivity than in controls. More important, the hypometabolism occurred in 30 of 60 brain regions studied bilaterally, particularly in regions located in the frontal and central cortex. This latter finding is most interesting because it correlates highly with the areas that we found in our topographic brain map study to show the greatest theta increase and beta decrease. All these results now begin to make sense in that for children who are hypoaroused we would expect to find a decreased cerebral metabolism, increased slow activity and academic difficulties.

The next question I addressed was, what is the best measure of cortical hypoactivation? Is it the increased theta activity, the decreased beta activity, or some combination of these? Age considerations are most important. As we age, the relative proportion of beta activity increases and theta decreases. Very excellent baseline studies showing the changes in EEG activity in different frequency bands as a function of age had been carried out previously by Gasser et al. (1988). They evaluated the development of EEG at rest from 6 to 17 years in normal individuals. They found that posterior regions mature earlier than anterior ones. This is interesting because in our studies, it appears that the EEG of children with ADHD are more "pediatric" in their appearance than matched controls, particularly in the frontal regions. These investigators also looked at more complex measures such as coherence between hemispheres for different

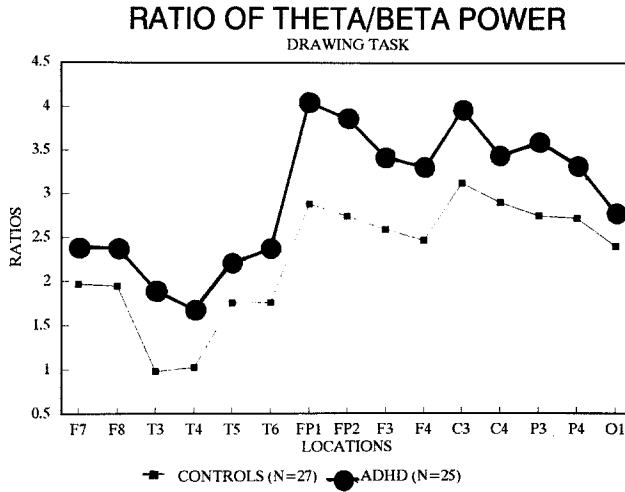


Fig. 4. Ratios of theta to beta activity for 16 standard EEG locations for 25 ADHD and 27 matched controls ages 8-11 during a drawing task.

frequencies and topographic distribution activity in different frequency ranges. Essentially, they found that beta activity increases with age later than theta decreases. Broad band coherences also increase with age; that is, the two hemispheres tend to show higher correlations in homologous regions in matched frequencies as we approach adulthood.

Based on Gasser's findings and our own observations, I decided that perhaps the better measure than simply the relative percentage of theta or beta or spectral power might be the ratio of theta to beta. This would take into account the fact that different individuals as a result of skull thickness or other factors have somewhat variable amplitude or power levels for the various EEG frequencies. However, the ratio of theta to beta should decrease in older individuals and should also be higher in individuals with ADHD than matched controls. Figure 4 shows an example of this. In this figure theta/beta ratios for 25 ADHD and 27 matched controls from our current study are displayed for 16 different locations. Examination of this particular metric taken during the drawing task shows no overlap between the groups. The largest theta/beta ratios are in frontal locations FP1, FP2, F3, and F4 and are much lower in the posterior locations O1 and O2. These ratio differences have important ramifications for the assessment of the effects of biofeedback training.

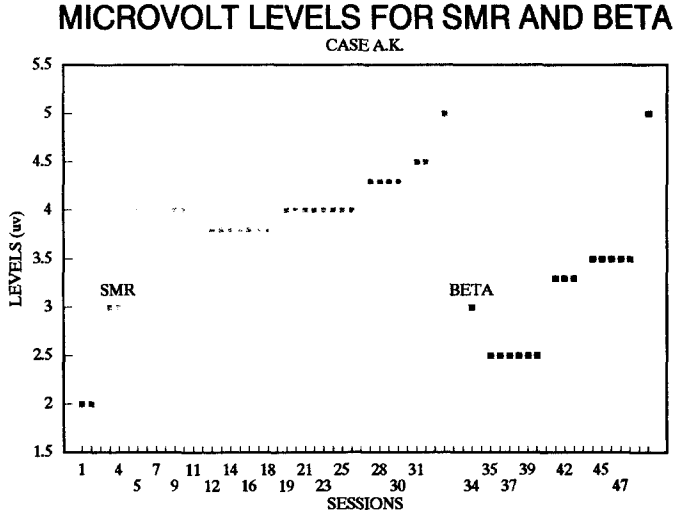


Fig. 5. Changes in reward criteria for SMR or beta over sessions. The requirements for reward increase over sessions.

CURRENT STUDIES OF BIOFEEDBACK TRAINING IN CHILDREN WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

In our present work, we are assessing the use of new instrumentation which has recently been developed by Lexicor Corporation of Boulder, Colorado. This instrumentation allows for 19 channels of topographic brain mapping and also for biofeedback from any location or combination of locations on the cranium.

There are two ways in which children can be trained. One is to use fixed criteria in terms of the levels of beta/theta, EMG activity, or movement for reward and inhibit circuits and to train the children to meet these criteria over a number of sessions. This produces learning curves but often requires longer training periods. The other technique is to vary the thresholds for beta, theta or in some cases SMR depending on how well the child is performing; we then track the child's performance and change the levels accordingly. This will lead to somewhat faster learning and less boredom and may be a better clinical approach. In Figures 5 and 6, examples are provided of how this would appear graphically. In this example a child is first trained to increase levels of SMR from 2 to 5 μ V and then to increase beta activity from approximately 2.5 μ V to 5 μ V. The graphs show on a session by session basis the criteria that were set for reinforcement of either

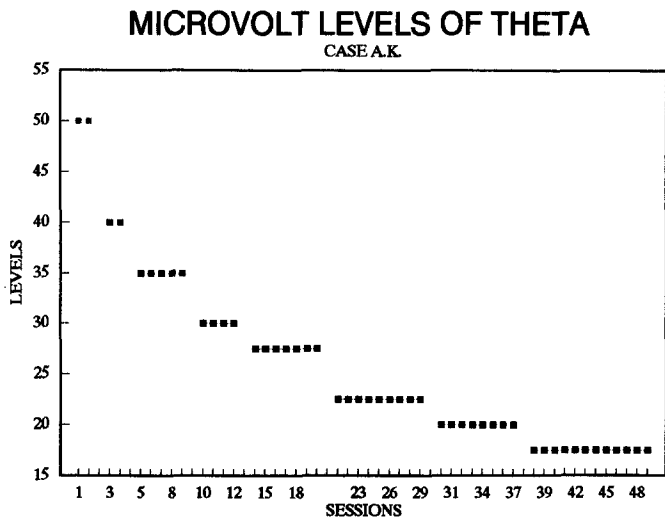


Fig. 6. Changes in theta inhibit criteria for SMR or beta reinforcement over sessions. The amount of theta allowed decreases over sessions.

SMR or beta (Figure 5) or for theta inhibit (Figure 6). Over the course of all 48 sessions the child was able to reduce the theta inhibit levels from 50 μ V to approximately 17 μ V. At the end of training, this child was producing relatively little theta and considerable beta. If we measure theta/beta ratios they would have clearly decreased over the course of training. Figures 7 and 8 show learning curves generated using the Lexicor instrumentation over 40 trials for a 12-year-old child trained under different fixed criteria conditions. These conditions involved a baseline, feedback only condition and conditions where the child obtained feedback while listening and reading. These figures show a clear increase in the percentage of beta activity and a decrease in the percentage of theta activity.

We have now completed work with three children using the Lexicor System and employing strict reward and inhibit criteria that are not changed over the course of the training paradigm. In these three children, we observed decreases in theta/beta ratios after training as compared with before training for many cortical locations. We have been able to observe in the multicolored topographic displays relative decreases for theta activity and increased beta activity after training. In addition to the three children that we are running according to strict criteria, we have additional children currently in shaping paradigms where the levels are changed as a function of the child's performance.

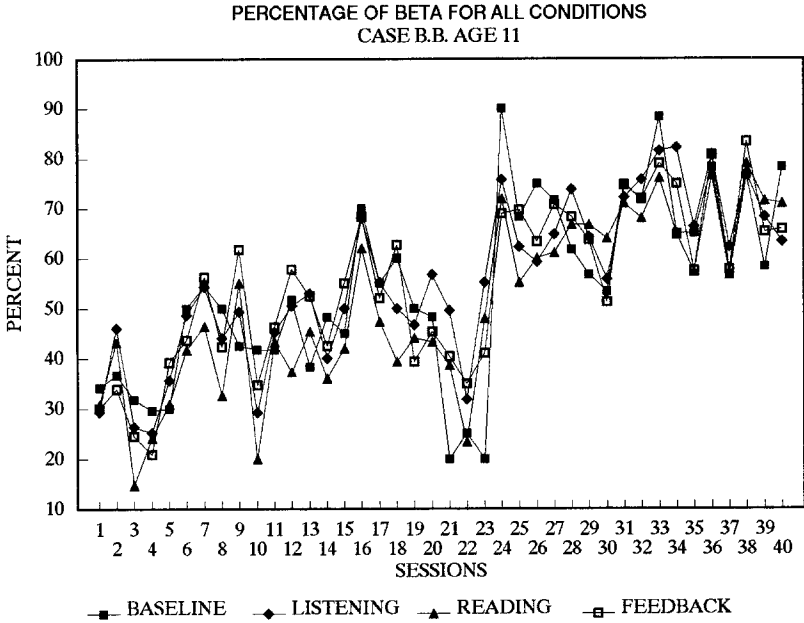


Fig. 7. Increases in percentage of beta activity for case B.B. over all conditions with fixed criteria for reward and inhibit over 40 sessions.

CONCLUSIONS

In this article, I have traced in some detail the history of our research endeavors in the area of attention-deficit/hyperactivity disorder. We have performed many studies over the past 15 years to show that there are differences between children with these and related disorders and matched controls in terms of EEG and ERP measures. We have shown that EEG biofeedback training when properly employed, although time consuming, can lead to very significant changes in these EEG measures as well as to improvements in psychometric test measures and school performance. We have worked with enough children to have confidence that the EEG biofeedback works very well.

Let me mention a practical issue. It takes many more sessions to work in this context than most biofeedback applications. Unlike the typical 10-20 session treatment for muscle contraction headaches, treatment for attention-deficit disorders may take between 40 and 80 sessions. The technique has to be integrated with academic work and children have to be phased out gradually and followed for a long period of time. Nevertheless, we have

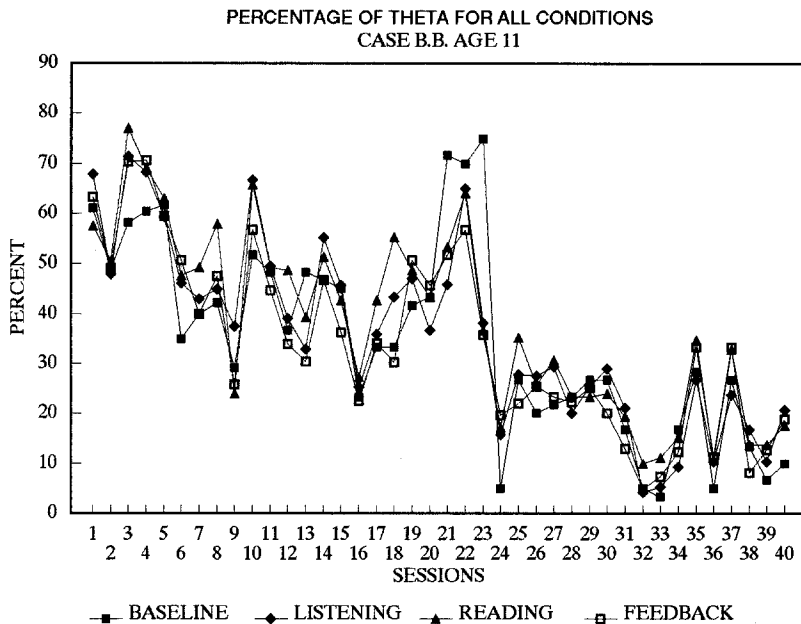


Fig. 8. Decreases in percentage of theta for all conditions for case B.B. during biofeedback training over 40 sessions with fixed criteria for reward and inhibit activity.

never seen a child become worse after treatment, and at least 20 of the children we treated before have been followed into adulthood and continue to function normally in college or other work settings. Because of the larger number of sessions, the expensive instrumentation involved, and the amount of time that has to be employed working with each child, the question of third party reimbursement often arises. It has been our experience that if one can provide documentation to a third party payor that for a particular client the technique works, they are much more likely to support the program for that child. This documentation should include, if possible, graphs showing the child's performance, measures of psychometric improvements, and changes in letter grade or grade point average. This can be supported by documentation from teachers and parents regarding the child's areas of improvement. It has been our experience that when third party payors are provided this level of documentation along with some references showing that other practitioners have obtained similar results, they are more likely to include treatment for ADHD using EEG biofeedback as a viable and supportable therapeutic approach.

Finally, I believe that the use of EEG biofeedback training for ADHD will soon become widespread. Instrumentation has now become available from at least four manufacturers that meet all the criteria for carrying out this type of work and documenting the results. I would encourage current investigators to look at ratio measures rather than only the microvolt, power levels, or percentages. They should also consider alpha/beta ratios. We have found that many ADHD children show persistence of alpha during academic tasks as opposed to normal alpha blocking. I would predict that ERP paradigms will be useful in the future, not only for assessment of EEG training related changes but also directly for ERP biofeedback.

The opportunity for treatment of attention-deficit/hyperactivity disorders, hyperkinesis, and learning disorders is wide open. It is an area that affects a large number of children for which previous treatment approaches have only been partially successful. The addition of EEG biofeedback training for these children will have a very significant impact on them and their families by helping to decrease the severity of these disorders, so that the children can achieve their educational potential and function better in a complex world.

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