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ABSTRACT

This literature review examined 16 treatment studies which employed electroencephalographic (EEG) biofeedback to treat children with attention-deficit/hyperactivity disorder (ADHD). An introductory section reviews hallmarks of ADHD, its historical background, current diagnostic criteria, etiology, single treatment or symptom focused treatment, development of EEG biofeedback, neurophysiology, EEG frequencies and biofeedback, medication, cognitive-behavior therapy, relaxation training, comparative studies, past reviews, and studies covering construct design or definitions. The EEG biofeedback treatment studies are divided into case studies and multiple subject designs. Methodological considerations are examined, including subject variables, EEG reinforcement contingencies, length of treatment, EEG sampling, measures of hyperactivity, measures of attention, achievement measures, measures of IQ, measures of impulsivity, and variables infrequently studied. Fifteen of the studies reported positive results; however, small sample size and variable implementation of normal and ADHD controls are seen as limiting generalization. The studies also implemented three different EEG contingencies, further reducing comparisons. The paper concludes that, in the absence of any clearly defined and proven alternative treatment for ADHD children, EEG biofeedback should be viewed as an efficacious adjunctive treatment. (Contains approximately 120 references.) (Author/DB)

IS EEG BIOFEEDBACK EFFICACIOUS AS A TREATMENT FOR CHILDREN WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER?: A REVIEW OF THE LITERATURE

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by

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IS EEG BIOFEEDBACK EFFICACIOUS AS A TREATMENT FOR CHILDREN WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER?: A REVIEW OF THE LITERATURE

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This literature review examined 16 treatment studies employing electroencephalographic (EEG) biofeedback to treat children with Attention-Deficit/Hyperactivity Disorder (ADD/HD). Current formulation of ADD/HD was presented, along with primary forms of treatment: medication, cognitive-behavior therapy, and relaxation training. Although the hallmarks of ADD/HD remained stable, there remains no conclusive etiology. No single type of therapy has proven superior: therapies are symptom-specific. Treatment research remains in the preliminary or exploratory stage. Fifteen studies reported positive results; however, small sample size and variable implementation of normal and ADD/HD controls limit generalization. Studies implemented three different EEG contingencies, further reducing comparisons. In the absence of any clearly defined and proven alternative treatment for ADD/HD children, EEG biofeedback should be viewed as an efficacious adjunctive treatment.

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IS EEG BIOFEEDBACK EFFICACIOUS AS A TREATMENT FOR CHILDREN WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER?: A REVIEW OF THE LITERATURE

Introduction

A growing number of therapists treat children diagnosed with Attention-Deficit/Hyperactivity Disorder (ADD/HD) by implementing what has variously been termed electroencephalographic (EEG) feedback, EEG biofeedback, or neurofeedback. This review will focus on research concerning efficacy of EEG biofeedback treatment for children diagnosed with ADD/HD published between 1973 and the present.

No published review has addressed this body of literature within this 24-year span. This should not be surprising since there have been relatively few articles concentrating on this form of treatment. Review articles do exist that mention EEG biofeedback as a treatment for ADD/HD. These reviews, however, addressed only part of the relevant literature while considering the legitimacy of biofeedback as a whole (Lee, 1991; Richter, 1984; Cobb & Evans, 1981). This review is intended to assist the clinician who may be considering an investment in training for biofeedback or in making an informed referral. These areas of research will be summarized and their specific methodologies evaluated in terms of weaknesses and strengths. The course of the discussion

will proceed as follows: first, the introduction will cover the current defining characteristics of ADD/HD, followed by a brief description of the historical development of biofeedback. Next, current diagnostic formulation and etiology of ADD/HD will be discussed. After that, development of EEG biofeedback rationale and EEG frequencies of concern will be reported. Treatment comparisons, comparative studies, and studies contributing to construct design will be mentioned. A discussion and review of these topics will help introduce the treatment literature and paint a brief picture of the disorder.

Hallmarks of Attention-Deficit/Hyperactivity Disorder

Children with Attention-Deficit/Hyperactivity Disorder typically manifest their differences from peers around the age of 4 years when parents and caregivers notice them struggling with tasks of higher complexity. These tasks are frequently school related, requiring performance within the bounds of structure such as time limits and individual or group participation. An example of such a task is sitting in a seat for a teaching period, requiring the child to focus his or her attention for longer periods of time. Their inability to either remain focused on a task or control impulsive motor activity sabotages their ability to learn to succeed on these tasks, let alone remain focused long enough to complete their assignments.

Failure in school further isolates ADD/HD children from their peers, diminishes chances to experience success, and leaves them searching for

alternative and often negative ways to bolster self-esteem and act out frustration. Teachers devote an inordinate amount of time trying to maintain these children on task. Teachers may be torn between providing time for other children and redirecting the child who cannot focus attention and/or control hyperactive symptoms. Caregivers manage these children rather than teaching them, a painful situation not lost on either caregiver or child. Classroom management and school-centered treatments and resources are summarized by Durlak (1992).

Although the current standard for diagnosing Attention-Deficit/Hyperactivity Disorder was reformulated in 1994, the core hallmarks of the disorder have been stable (American Psychiatric Association, 1980, 1987, 1994). The three core symptoms of this disorder are inattention, impulsivity, and hyperactivity. Anastopoulos and Barkley (1992) note all three hallmarks or core symptoms are defined differently by various researchers (e.g., Lahey, Stempniak, Robinson, & Tyroler, 1978). Inattention is alternatively described as the behavior of not listening to instructions, the failure to stay on task, or the behavior of becoming easily bored and distractible. Impulsivity is referred to as inaccurate, rapid responding, or failure to inhibit or modulate behavior in social contexts. Hyperactivity is defined as either the motor or the verbal expression of behaviors that are off task and excessive in number. Barkley (1990) includes other associated symptoms such as difficulties with rule-governed behavior and inconsistent performance. Therefore, Attention-

Deficit/Hyperactivity Disorder is the diagnostic term describing children exhibiting a behavioral pattern of inattention and/or hyperactivity or impulsivity that is significantly more severe than children or adolescents who achieve expected developmental milestones.

Historical Background

The turn of this century ushered in formal observations of ADD/HD. G. F. Still (1902) first recorded hyperactive and impulsive symptoms associated with ADD/HD in a case study. These symptoms were attributed to variability of character and temperament such as defects in moral control, destructiveness and mischievousness. Relatively little attention was paid to children displaying these symptoms during the following years. Dr. Charles Bradley (1937) first reported a pharmacological treatment. He administered amphetamine sulfate to a group of children and noted increased attentiveness and improved school related behaviors. Strauss and Lehtinen (1947) developed the concept of minimal brain dysfunction syndrome (MBD), solidifying a conceptual formulation. According to Strauss and Lehtinen's model, impulsive and hyperactive symptoms resulted from brain impairment significant enough to lower the threshold of behavioral inhibition. Minimal brain dysfunction, though a constructive departure from moral defect etiology, fell under increasing criticism due to its over-inclusive definition and inability to provide descriptive or predictive clarification for those affected children. Increasingly, the hyperactive or

hyperkinetic symptomatology came to the fore, prompting a renaming of the disorder: Hyperkinetic Reaction of Childhood (American Psychiatric Association, 1968). Douglas (1972) introduced the attentional and impulsive component. His work is responsible for the current paradigm that considers deficits in attention as the primary reason for disorganized behavior, then expressed as hyperkinetic, impulsive behavior.

Current Diagnostic Criteria

The diagnostic criteria have changed as research accumulated and conceptualization of the disorder progressed. The current diagnostic criteria, taken from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) are listed below (American Psychiatric Association, 1994, pp. 83-85).

[Criterion] A. Either (1) or (2):

(1) six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes mistakes in schoolwork, work, or other activities
(b) often has difficulty sustaining attention in tasks or play activities
(c) often does not seem to listen when spoken to directly
(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often has difficulty organizing tasks and activities (e.g., toys, school assignments, pencils, books, or tools)
(g) often loses things necessary for tasks or activities (e.g., school assignments, pencils, books, or tools)
(h) is often easily distracted by extraneous stimuli

(i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situations in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often has difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative, or a Personality Disorder). . . .

Attention-Deficit/Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 months. . .

> Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 months. . .
>
> Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 months

An additional diagnostic category, Attention-Deficit/Hyperactivity Disorder Not Otherwise Specified, was kept in order to include manifestation of symptoms of inattention or hyperactivity-impulsivity that do not meet criteria for Attention-Deficit/Hyperactivity Disorder.

The DSM-IV provides one set of criteria with three subtypes (Combined Type, Predominantly Inattentive Type, Predominantly Hyperactive-Impulsive Type). A child's condition may be diagnosed as manifesting either predominantly attention-deficit or hyperactive-impulsive symptoms or a combination of both. Criterion A organizes the three hallmarks into two groupings: inattention and hyperactivity/impulsivity. Criterion B continues the description that ADD/HD symptoms are discovered early in life and run a strong chance of taking a chronic course. Criterion C was introduced in order to reduce the incidences of false-positive diagnoses.

Etiology of ADD/HD

Research to date has not established the etiology of ADD/HD or explicated its underlying pathology. Generally ADD/HD children do not show gross structural damage or CNS impairment as judged by mainstream neurological methods. Conversely, most neurologically disordered children

or brain injured children do not exhibit hyperactivity (Kaplan & Sadock, 1988). Hypersensitivity to food additives has not been validated as a cause of ADD/HD (Kaplan & Sadock, 1988). Genetic studies have shown some concordance with twins. Siblings of affected children are at a greater risk than half-siblings. Maturational delays may contribute to ADD/HD symptomatology and usually resolve around puberty (Kaplan & Sadock, 1988). Emotional deprivation, stressful psychic events interacting with temperament or genetic endowment may cause hyperactive symptoms. Oftentimes a change in these factors will cause the behavior to remit (Kaplan & Sadock, 1988).

Single Treatment or Symptom Focused Treatment

The former categories of Attention-Deficit Hyperactivity Disorder and Undifferentiated Attention-Deficit Disorder (without hyperactivity) were reintegrated into one encompassing category. This reintegration was based on field trials, literature reviews and data reanalysis that suggested the disorder was best conceptualized as a "unitary disorder with different predominating symptom patterns" (DSM-IV, p. 775). Organizing the symptom patterns into a single category implies a coherent and cohesive substrate, and lends credence to finding a treatment tailored to the underlying cause of the manifesting patterns, as this would be most efficient and effective.

Grouping symptom patterns in one diagnosis may distract from effective, symptom-based treatment. Lubar (1991) raised this objection in his

discussion of treatment difficulties. According to Lubar, children are often difficult to treat because more often than not their diagnostic picture is very complex.

Assigning the correct diagnosis or diagnoses and ensuring the most efficacious treatment or treatments is the chief difficulty in working with these children. Lubar listed four categories that are independent diagnostically, but may overlap. The child that presents to a clinician for diagnosis and treatment may have pure attention-deficit, conduct problems (approximately 75% of ADD/HD children), have associated aggressive or defiant symptoms (Kaplan & Sadock, 1988), hyperactive symptoms, or learning disabilities. Lubar (1995) cites two other independent/overlapping symptom categories: anxiety disorders and oppositional defiant disorder. It is also entirely possible that the child may exhibit a combination of these problems. Effective treatment, then, often depends on an array of treatments which address the particular complex of symptoms of the child. For the purposes of this review, the core behavioral components comprising ADD/HD will be treated more as independent categories in order to avoid confusion and facilitate meaningful statements about EEG biofeedback and diagnosis.

Development of EEG Biofeedback

Several streams of thought have merged to form the body of what is now multimodal biofeedback and EEG biofeedback. Biofeedback use stems

mainly from two rationales. The first starts in the work of learning theorists such as Meichenbaum and Miller (e.g., Meichenbaum, 1975; Miller & Dworkin, 1974). During the exploration of operant reinforcement and other forms of learning, researchers such as Kamiya (Kamiya, Callaway, & Yeager, 1969; Nowlis & Kamiya, 1970) showed that it was possible to track and learn from one's own bodily functions, even autonomic functions, if the feedback provided were salient and timely. It was therefore possible to influence or shape body states. Reviews of biofeedback such as Cobb and Evans (1981), reported success teaching children to be able to reduce muscle tension and modify other physiological states using various biofeedback modalities.

The very concept of EEG biofeedback treatment is steeped in learning theory. Inhibiting a certain wave such as theta, and encouraging or rewarding a wave such as beta are really means of arranging reinforcement contingencies. Research has successfully combined both learning theory and EEG correlates such as event-related potentials (ERP's). ERP's are averaged measures of selected EEG characteristics (e.g., wave amplitude and frequency) created after repeated trials using a selected stimulus. ERP research is ongoing and proceeds with the task of helping decode the role that psychological factors play in mediating sensory responses and even subsequent processing of information (Rosenfeld, 1990). It is important to note that brain waves are not independent variables in EEG paradigms. The reinforcement and/or inhibition contingency is the independent variable. When brain waves are

rewarded or inhibited by implementing EEG contingencies, what is thought to change is the neurological substrate generating that electrical activity.

Neurophysiology

The second body of thought arose from neurophysiology research and EEG studies. Formulations regarding etiology of ADD/HD, its hallmarks and specific EEG contingency rationales, came from these disciplines. The means for measurement, data on the use of EEG readings as measurements of brain function, and using EEG as a reliable index of arousal (paired with behavioral observations and mental state self report) came from EEG studies. Neurophysiological studies using electrical monitoring equipment such as EEG and/or neurotransmitter studies contributed to theories about mechanisms that may play a part in ADD/HD behavior. The major assumption of this research is that every change in a physiological state is represented by an appropriate change in mental state, or vice versa (Linkenhoker, 1983). The theory driving most research using EEG biofeedback with ADD/HD children subscribes to or reacts with the low arousal hypothesis that predicts children who manifest symptoms of ADD/HD are attempting to perform routine functions through the filter of decreased brain arousal. Competing etiological theories do exist. Neurotransmitter depletion theory was based on evidence that children who had difficulty modulating activity levels may have unbalanced inhibitory and

excitatory neurotransmitters (Wender, 1971). Amphetamines may work by balancing these systems through their effect on monoamines.

EEG studies in both awake and sleeping subjects revealed that the brain cycles through various levels of arousal (Kaplan & Sadock, 1988). Normal arousal levels ranged from alertness to sleep. As we go through our various behavioral patterns, there arise concomitant change in EEG patterns. Various brain functions are maximized by a certain state of arousal. For example, if the brain was in a state close to sleep (drowsiness), it would be very difficult to perform cognitive functions such as composing a paper or performing a calculation. Sterman (1996) argued that there is a confluence between neurophysiological research, learning theory, and clinical research.

"We now know clearly that relevant EEG rhythmic patterns reflect the unique properties of thalamocortical circuits, that these EEG patterns are topographically localized in relation to nervous system organization, and that the interaction between specific and nonspecific sensory and cortical influences determines their frequency and cortical expression" (Sterman, 1996, p. 4). In work with cats, Sterman found that operant conditioning of electrical potentials originating from the somatosensory cortex, called a sensorimotor rhythm (12 to 14 Hz or cycles/second), was possible. Cats were able to increase the production of these rhythms in response to rewards. Motor inhibition was the behavior associated with increasing the response of this wave. Cats conditioned to suppress motor behavior were found more

immune to seizures (Satterfield & Dawson, 1971; Satterfield, Lesser, Saul, & Cantwell, 1973). In response to this thalamocortical arousal hypothesis Robinson (1989, as cited in Tansey, 1991) reported evidence of increased intelligence in humans associated with balanced EEG. Robinson observed neural excitation (natural frequencies above 10 Hz) that is consistent with a state of middling arousability which coincides with high IQ: Low arousal would predict lower IQ (Tansey, 1991).

Rhythmic electrical activity begins in the somatosensory relay nuclei of the thalamus. These nuclei, known collectively as the Ventrobasal nuclei, change their firing patterns during operant conditioning of SMR activity. Normal firing patterns of the nuclei are characterized by a fast/random pattern of discharge. During operant conditioning, the pattern moves from the normal fast/random discharge to regular or systematic bursting activity. In addition, somatosensory information normally channeled through the nuclei were suppressed during SMR activity. The feedback between nucleic cells, "produces a recurrent oscillatory discharge which is entrained among many relay cells . . . . Corresponding synchronous volleys of discharge are projected to functionally related pools of cortical cells" (Sterman, p. 9). Neurophysiological studies focused on mechanisms regulating bursting or rhythm-generating activity have not gone beyond anesthetized animal preparations or isolated, single-cell experiments. Results from animal studies supported the hypothesis that sensory motor rhythm frequency varies as a

function of activation (studies by Bouyer, Dedet, Konya, & Rougel, 1974; Holcomb et al., 1979; Rougel, Bouyer, Dedet, & Debray, 1979 as cited in Sterman, 1996). Slower frequencies were correlated with drowsiness, while increasingly faster frequencies were associated with ever higher levels of activation (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992). Zametkin et al. (1990) found cerebral glucose metabolism, both global and regional, less in adults who were hyperactive since childhood than in matched normal controls. Furthermore, largest reductions were in premotor cortex and superior prefrontal cortex areas involved in control of attention and motor activity.

Additional clinical studies do support a relationship between directed activity and brain waves. Studies by Galletti, Battaglini, and Fattori (1993), and Stone and Lisberger (1990) found that: (a) body and eye movement suppressed 11 to 15 Hz activity in respective thalamocortical pathways (b) reduction in the activities increased the same wave activity (as cited in Sterman, 1996). "In both of these studies, cognitive engagement suppressed 7 to 13 Hz activity in cortical regions relevant to the task" (Sterman, 1996, p. 18). Sterman also stated that it was likely that cognitive engagement alone suppressed 7 to 11 Hz activity (Sterman, 1996).

Research has also found arousal differences in hyperkinetic children. Two subgroups of hyperkinetic children represent low CNS arousability and high CNS arousability (Satterfield et al., 1973). Assuming a normal level of

arousal, symptoms of hyperactivity may be seen as compensatory behavior in a low-aroused child. Such a child would evidence an increase in slow waves such as theta. Evidence has pointed to a correlation between low-arousal subjects and excessive, synchronized slow-wave activity, based on measures of electrical activity such as electrodermal activity, cortical evoked potentials, and behavioral observations of children with hyperactivity (e.g., Satterfield et al.; Stevens, Sachdeo, & Milstein, 1968). Such experiments suggest that children may act or seek out ways to increase stimulation due to quicker habituation to sensory stimulation. Highly-aroused subjects (based on indexes of measurement such as galvanic skin response [GSR]) showed the least amount of behavioral symptomatology and least effective response to medication (Satterfield et al.).

EEG Frequencies

Kaplan and Sadock (1988) list the major frequency bands that are of clinical interest: delta (< 4 Hz), theta (4 to 8 Hz), alpha (8 to 13 Hz), and beta (> 13 Hz). The abbreviation, Hz, describes the frequency in terms of number of cycles the wave alternates polarity per second.

Alpha waves are frequencies between 8 to 13 Hz. The wave has been associated with a state of relaxed wakefulness. Alpha feedback mainly assists in relaxation, and may not be accountable for increasing concentration, which has been posited to be more in the domain of beta (Linden, Habib, & Radojevic, 1996).

Beta waves have been divided into three distinct band classes. Most common is the 18 to 25 Hz band, followed by the less common 14 to 16 Hz band, and finally a rare 35 to 40 Hz band. In 97-98% of normal adults and children, the voltage in the awake EEG is less than 20 μV (microvolt); in 70% it is 10μV or less, as recorded between closely spaced scalp electrodes (studies by Eeg-Olofsson, 1971; Maulsby et al., 1968; Petersén & Eeg-Olofsson, 1971; as cited in Kellaway, 1990). Voltage or amplitude of a wave is considered a relative index of strength of that particular wave; however, Tyner, Knott, and Mayer (1983) caution that measurements of voltage vary with location of electrodes and the particular setting of the EEG.

Kellaway (1990) regards beta activity, with voltage of 25μV or greater, abnormal when seen on electroencephalogram; however, little is known about high voltage beta activity, and what significance this has. Developmentally, beta activity is a predominant feature of the EEG of the premature and term infant, and it is barely evident in the EEGs of young children. It may be increased in voltage and persistence in the precentral region in middle-aged and elderly females, but it tends to have a low voltage during old age, especially in males (studies by Frey & Sjögren, 1959; Harvald, 1958; Obrist, 1954; as cited in Kellaway, 1990).

In the presence of skull defects, beta activity in the area of the defect or adjacent to it may be enhanced as a consequence of the low-impedance pathway. Defects of dura, bone, and scalp enhance beta activity more than

other, lower frequency activity (Kellaway, 1990), and has led to erroneous identification of so-called "foci of fast activity" in patients with surgical or traumatic skull defects.

Beta activity of 18 to 25 Hz tends to increase during drowsiness, light sleep, and rapid eye movement (REM) sleep, and it usually decreases during deep sleep (Kellaway, 1990). Although beta activity should have the same voltage on both sides of the brain, normal individuals may have as much as a 35% voltage difference between sides. On the other hand, a consistently low voltage on one side (difference greater than 35%) may indicate trauma, structural abnormality, or status post focal epileptic seizure (Kellaway, 1990).

Beta activity in the 14 to 16 Hz band is usually most marked in the frontocentral region, but may show maximum voltage elsewhere, even in the occipital region. The location of the maximum potential field does not appear to have particular physiological or pathophysiological significance. Beta activity in this band, when present, is usually enhanced by hyperventilation and indeed may become clearly evident only in bursts (Kellaway, 1990).

Theta activity was defined as 4 to 8 Hz or 4 to 7 Hz (Lubar & Shouse, 1977). It has been associated with drowsiness or daydreaming (Kaplan & Sadock, 1988). Heightened emotional states enhance frontal and central theta activity in the 6 to 7 Hz (Kellaway, 1990). Some normal individuals show marked frontocentral rhythmic theta activity (with eyes open) while

performing some types of tasks. Generally, however, this frequency of activity tends to show greater incidence and higher amplitude with the onset of drowsiness, although 35% of young, non-drowsy, asymptomatic adults have low voltage (less than 15μV) 6 to 7 Hz activity in the frontocentral region in a quiet lab environment (Cohn & Nardini, 1958; study by Carels, 1959; as cited in Kellaway, 1990). In 10% of young adults, the voltage, or amplitude of theta is 15 to 25μV. Therefore there is variability in expected or normal theta amplitude. Though increased theta activity is linked with decreasing arousal, ascribing a particular theta level as abnormal is difficult (Mundy-Castle, 1951; Werre, 1957). "This problem is particularly important in children, who are especially prone to increased theta activity in highly emotional states and in whom frontal theta activity was once identified as an abnormality having a specific association with behavior disorders. Indeed, the presence of this 'abnormality' in the EEG was originally thought to be 'evidence of the organic nature of the behavior disorder present'" (Kellaway, 1990, p. 153).

Kellaway believes that clinical interpretation of theta rhythms in the EEG's of children has suspect validity since these rhythms appear just as likely to result from emotional upsets engendered by behavior problems and their consequences rather than from pathologically altered brain function (Kellaway, 1990). The correlation of anterior theta rhythm with behavior-disordered children was first reported in 1938 (Jasper, Solomon, & Bradley).

These authors compared children with behavior problems to normal, age-matched controls. Jasper et al. and subsequent investigators regarded this theta activity as evidence of "fundamental brain pathology." Lindsley and Cutts (1940) reported that although occasional brief runs of 5 to 8 Hz waves in the frontal and central regions are not unusual in normal subjects, the waves should be considered abnormal if they are present as much as 10% of the time in well-organized runs or bursts. The concept that young children with Attention-Deficit/Hyperactivity Disorder display abnormal theta activity on EEG forms one of the fundamental assumptions of neurofeedback (EEG biofeedback).

Kellaway (1990) emphatically stated that EEG interpretation should be based on informed qualitative inspection followed by directive quantitative searches, and not on a designated wave amplitude. In addition to wide EEG variability in "normals" (as much as 35% variance in beta activity), efforts to diagnose behavior disorders have not met with success (Deboskey, 1982; Kellaway, 1990; Pliszka, 1991). It could be argued from Lubar's work as well, that significant change in EEG measures remain a rather general, non-specific index of change. Also wide, intra-subject variability of EEG output is an additional factor. Technology is increasing, however, and conditions of testing are better controlled.

EEG Biofeedback

Lubar (1991, 1995) provided recommendations for comprehensive treatment using EEG biofeedback for children with ADD/HD. He recommended extensive evaluation to identify specific hallmarks exhibited by the ADD/HD child. Evaluation included both psychological and neuropsychological tests in addition to behavioral observation. He stated that behavior must be evaluated in different settings in order to uncover concomitant disorders (e.g., conduct disorder or learning disabilities) and treat them accordingly (Lubar, 1991). Many children have additional overlays of conduct disorder (Hinshaw, 1987; as cited in Kendall, 1993).

Lubar (1995) reported that children with pure ADD/HD responded extremely well to EEG biofeedback training. Children who displayed hyperkinesis benefited most from a multimodal regime of stimulant medication and biofeedback. Children on high doses of stimulant medication who showed poor behavioral control were unlikely candidates for EEG biofeedback. Medications can treat some ADD/HD comorbidity problems such as impulsiveness, depression, seizure disorders, and tic disorders.

Lubar (1995) recommended individual or family therapy to help the family manage children with ADD/HD. Individual therapy was helpful if the children were physically or emotionally abused. Family therapy was an especially helpful means of educating the family and child that it was not their fault that the child had ADD/HD.

It is important that EEG biofeedback training not be viewed as a cure-all or a single approach to treat children with ADD/HD. Lubar (1995) addresses four areas of concern regarding neurofeedback (EEG biofeedback) therapy: (a) specific criteria for feedback therapy, (b) symptoms improved with therapy, (c) results of treatment, and (d) treatment effectiveness.

> 1. Who is a candidate for neurofeedback therapy? Anyone with a primary diagnosis of ADD or ADHD, between the ages of 7 and 45, with low-average, average, or above-average intelligence is a candidate. Neurofeedback treatment should not be offered with comorbidity of: mental retardation; childhood psychosis; severe depressive or bipolar illness; significant seizure disorder where medications interfere with learning (i.e., sedating medications); hyperkinesis, where multiple medications or high dosages with monotherapy have been ineffective; learning disabilities without ADD or ADHD as a primary problem; [and] dysfunctional families who refuse to participate in indicated therapy.
> 2. What symptoms can be improved with neurofeedback? [Lubar lists] Attention, focus, and concentration; task completion and organizational skills; impulsiveness; [and] mild hyperactivity.
> 3. What are the results of treatment? [Lubar lists] Improved behavior and learning, improvement in school grades, increased self-esteem, better job performance, greater realization of innate potential, higher intelligence test scores, [and] improved scores on parent-teacher rating scales.
> 4. How effective is this treatment approach? When the above criteria are used to select candidates for therapy and treatment, the majority of patients completing treatment show marked improvement. (pp. 506-507)

Neurofeedback or EEG biofeedback has not been restricted to ADD/HD diagnoses, but instead it has been tried on an ever-widening list of difficulties. Neurofeedback has shown noted results for treatment of epilepsy (this appears to have been its first clinical application), where seizures were reduced concomitant with reduced chemotherapy use (Seifert & Lubar, 1975;

Lubar & Shouse, 1977; Sterman, 1996). It has been used to treat simple and complex tic (Tansey, 1986). It has also been applied to a stroke victim (Rozelle & Budzynski, 1995), with reported improvement in EEG pattern, increase in speech fluency, balance, attention, and concentration. It has also been tried with substance abuse patients (Peniston & Kulkosky, 1989).

Medication

Psychostimulants have been used to mitigate hyperactive behaviors. The medications used are primarily central nervous system stimulants. The drugs are dextroamphetamine sulfate (Dexedrine), methylphenidate (Ritalin), pemoline (Cylert), and certain anticonvulsant medications. Cylert is a central nervous stimulant which is different from the amphetamines and methylphenidate in that it has not been reported to have high sympathomimetic effects. Caution should be used in treating children with this drug if they have impaired renal function (Physicians' Desk Reference, 1994). Imipramine hydrochloride (Tofranil), an antidepressant, has been used with some success in treating ADD/HD, however the manufacturer has not advised it for use with children (particularly those under the age of six years) due to potential cardiotoxic effects (Kaplan & Sadock, 1988).

There is general agreement (e.g., Anastopoulous & Barkley, 1992; Kaplan & Sadock, 1988; Lubar, 1995; Swanson, McBurnett, Christian, & Wigal, 1995) that medication was helpful in reducing restlessness and impulsivity, and increasing attention span, concentration, and compliance with

commands. Central nervous system stimulants appeared to help approximately 75% of treated children (Kaplan & Sadock, 1988). The precise biological reasons for them working are not known; however, the drugs are known to affect both the arousal (reticular) and motor systems (Millichap, 1968, 1973; as cited in Lubar & Shouse, 1977).

Some additional difficulties have been reported with medication use. Behavior controlled by psychostimulants may not remain controlled once the medication is withdrawn (O'Leary, 1980; Weiss, Minde, Werry, Douglas, & Nemeth, 1971). Although behavioral symptoms are reduced and attention increased, academic performance is unchanged (study by Rapport et al., 1994, as cited in Campbell & Cueva, 1995). In addition medications can produce retarded growth (Safer, Allen, & Barr, 1972). In a double blind study (Brown, Borden, Wynne, Schleser, & Clingerman, 1986), 40 children diagnosed as attention-deficit disorder were divided into four comparison groups described as cognitive training and placebo, attention control and methylphenidate, cognitive training and methylphenidate, and attention control and placebo. The results indicated that, on several measures of attention and impulse control there were no significant behavioral effects that continued with the withdrawal of medication. Furthermore, the medication and cognitive therapy condition was not found to be any more efficacious than the other conditions. Caution is needed in giving the stimulants, due to their possible potential for abuse (Kaplan & Sadock, 1988). No real data has been reported

for use of phenytoin (an anticonvulsant); however, there have been some indications that carbemezapine is efficacious (Pliszka, 1991). In spite of the difficulties that medications offer, it is still the first line of treatment for most children diagnosed with ADD/HD (Walden & Thompson, 1981) and is recommended as part of a multi-modal treatment regimen for children with ADD/HD (Lubar, 1991, 1995).

Many commonly used drugs (e.g., barbiturates, benzodiazepines, chloral hydrate) increase beta activity (Frost, Carrie, Borda, & Kellaway, 1973). The incidence of beta rhythms with amplitudes much above 20μV is statistically low in normal individuals. Presence of such activity suggests the possibility of drug ingestion. Although the 18 to 25 Hz band is the one most generally affected, some drugs also increase the 14 to 16 Hz activity (Kellaway, 1990). No study indicates that CNS stimulants affect EEG activity. Feldman, Crumrine, Handen, Alvin, and Teodori (1989) studied 10 ADHD children with seizure activity given methylphenidate and reported medication response identical to non-affected ADHD children. There were no effects noted on EEG patterns.

Cognitive-Behavior Therapy

Behavior therapy has been a very popular and well researched form of treatment. Anastopoulos and Barkley (1992) summarized cognitive-behavioral treatment and research. Self-monitoring programs have resulted in improved on-task behavior and academic productivity of some children

with ADHD. Self-instructional training has shown equivocal results (Anastopoulos & Barkley, 1992), since expected generalization effects did not materialize. Behavior therapy has proven unreliable with other behaviors exhibited by medicated hyperactives, and it, too, has not emerged as a singular or superior form of treatment (Abikoff, 1991; Gittleman et al., 1980).

The National Institute of Mental Health initiated a five year multisite, multimodal treatment study of children diagnosed with ADD/HD. The purpose of this study is to answer under what circumstances and with what characteristics do which treatments or combinations of treatment (stimulants, behavior therapy, parent training, school-based intervention) have what impacts upon what domains of child functioning (cognitive, academic, behavioral, neurophysiological, neuropsychological, peer relations, family relations), for how long, to what extent, and why (Richters et al., 1995).

Cognitive-behavioral training can reduce one hallmark of ADD/HD: impulsivity (Kendall, 1993). Baer and Nietzel (1991) reported cognitive-behavioral interventions for impulsivity associated with improvements of one third to three quarters of a standard deviation relative to untreated control subjects. Their meta-analytic review included 36 outcome studies using treatments of self-statement modification, reinforcement contingencies, modeling, strategy training, problem-solving training, and various treatment combinations.

Relaxation Training

Relaxation training has been used primarily to reduce symptoms of hyperactivity, though studies have been hurt by methodological flaws such as imprecise definition of subject populations and heterogeneous use of dependent variables (Richter, 1984). Richter reported that relaxation training provided significant results when continued over a longer period of time and when it was part of other supports in school-based interventions. There was an additional report that this treatment can be as effective as EMG treatment (Denkowski & Denkowski, 1984), though treatment length was short.

Comparative studies

Comparison studies have not shown any treatment modality superior to another; however, conditions using multiple treatments have had some superiority over individual treatment conditions (Lubar, 1995). Kassel (1986) compared EEG biofeedback and EMG modalities on an ADHD population and reported inconclusive results. Biofeedback and medication (targeted to reduce hyperactivity) are the most frequently compared treatments. Hughes, Henry, and Hughes (1979) showed a direct relationship between muscle tension and activity levels, which indicates that any treatment targeted at reducing either muscle tension or activity level reduces the other. Potashkin and Beckles (1990) reported EMG biofeedback reduced muscle tension while personal attention and Ritalin did not reduce muscle tension. All three groups produced significant change with regard to improvement on behavioral

ratings of hyperactivity. This last result contradicts the direct relationship theory of Hughes et al. Lubar (1991, 1995) and others (Barkley, 1990) respond that the failure of one form of treatment to be superior to another points to evidence that different modalities likely address different components of hyperactivity. These results do little to help the clinician select a single treatment course. At present it is unwise to rely on a single treatment, although addition of modalities tailored to fit specific symptoms seems to be helpful in increasing response (Lubar, 1995).

Past Reviews

Many forms of biofeedback have been used to treat children manifesting symptoms of ADD/HD. The most cited form of treatment was electromyograph or EMG studies. Cobb and Evans (1981) reviewed 44 studies published between 1975 to 1978, covering the use of biofeedback with school-age children. They concluded that children had the ability to reduce frontalis muscle tension, showing that the children were able to move to a more relaxed state. Children also learned to modify other physiological processes such as skin temperature and alpha rhythm. Cobb and Evans pointed out methodological flaws including failure to include placebo groups, use of material reinforcers and additional concurrent treatments, lack of replications, and small sample size which made it difficult to draw the follow-up conclusion that biofeedback caused any reduction in targeted behavior (usually hyperkinesis).

Secondly, Cobb and Evans (1981), and Lee (1991) concluded that biofeedback was not shown to be superior to the more accepted forms of treatments for behavioral dysfunction. Lee found EMG and body movement biofeedback was efficacious in combination with other treatments. He did not, however, recommend biofeedback as the treatment of choice for hyperactivity. No review focused on biofeedback treatments for impulsivity or attention related symptoms.

Studies Covering Construct Design or Definitions

In spite of the objections of researchers such as Kellaway that ADD/HD children cannot be differentiated from normals on EEG measures, a great deal of research exists that shows EEG differences between groups of children who perform differently on measures of cognition and perception, performance tasks, and academics. Ackerman, Dykman, Oglesby, and Newton (1994, 1995) found, in two studies of 33 girls and 86 boys age 7.5 to 12 years, differences between poor readers and a normal-reading clinic control of ADD children. The combination of greater low beta and less theta power significantly predicted better reading and spelling in correlation analysis. This implies that adequate readers process stimuli more actively than poor readers. Lubar, Mann, Gross, and Shively (1992) reported similar results in gifted or normal reading children versus learning disabled, using the P3 component of ERP. However, they could not conclude whether results were due to attentional or processing deficits.

Ortiz, Exposito, Miguel, Martin-Loeches, and Rubia (1992) contrasted poor readers (dyslexic) and controls in an auditory phonemic discrimination paradigm. They found that, unlike the controls, the dyslexics showed an increase in alpha and a decrease in high beta during the experimental task, which they interpreted as under reactivity of poor readers to the linguistic demands of the task.

The EEGs of 91 age-matched children diagnosed with ADD/HD in psychiatric clinics in Japan, China, and Korea were significantly different from those of normal and deviant behavior groups, as measured by Rutter's questionnaire (1970). Matsuura et al. (1993) found more delta and fast theta waves and fewer alpha waves in ADD/HD children in all three countries. There were no group differences between the deviant children and the normal children. These results suggested that there is a biological difference between ADD/HD children and children displaying pure behavior dysfunction.

Janzen, Graap, Stephanson, Marshall, and Fitzimmons (1995) measured on task EEG performance of eight ADD children and eight normally achieving controls. They found significant amplitude differences in the theta band (4 to 8 Hz) in all tasks (baseline, eyes open, reading, and drawing). Amplitude differences in the beta band (12 to 20 Hz) were negligible. Studies such as these show that EEG measures can be successfully employed in order to assess group differences in processing and attention.

Real-time EEG measures may still be susceptible to variability of the individual and group; however, power spectra and ERP studies are not as susceptible to this variance and are useful in assessing group differences.

EEG Biofeedback Treatment Studies

Sixteen studies were reviewed where children with Attention-Deficit Hyperactivity Disorder were treated with EEG biofeedback. Publication dates ranged from 1973 to 1996. While treatment remained a focus for research, a growing number of studies were devoted to exploring group differences between clinical and normal populations (Matsuura et al., 1993), providing EEG reference criterion (Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995), and exploring the difference between processing and attention resources (Lubar, Swartwood, Swartwood, & O'Donnell, 1995). Studies such as these have modified Kellaway's objections that there are no significant differences between clinical populations such as children with ADD/HD and normal populations on measures of EEG. There remains a paucity of published treatment literature that used children with ADD/HD or its hallmarks (hyperactivity, inattention, impulsivity). Table 1 lists the treatment studies that met the inclusion criteria. The criteria for review inclusion were: 1) Empirical studies, 2) Use of EEG reinforcement contingency for at least one of the independent variables, 3) Inclusion of child population representative of ADHD diagnosis (ages 7-13), 4) Exclusion of pure adult or

Table 1

Studies Reviewed

Descriptor	n	EEG Contingency
Tansey and Bruner, 1983	1	SMR
Tansey, 1993	1	Follow up
Nall, 1973	48	Alpha
Lubar and Shouse, 1977	24	Theta/SMR
Lubar and Shouse, 1976	1	Theta/SMR
Shouse, 1977	24	Theta/SMR
Shouse and Lubar, 1979	4	Theta/SMR
Cartozzo, 1995	19	Theta/SMR
Parziale, 1982	16	Theta/SMR
Tansey, 1991	24	SMR
Tansey, 1990	24	SMR
Lubar, et al., 1995	23	Theta/Beta
Linden, Habib, & Radojevic, 1996	18	Theta/Beta
Lubar and Lubar, 1984	6	SMR/Theta or Beta/Theta
Schnoll, 1995	56	Theta/Beta
Bell, 1986	4	Theta/Beta

pure adolescent populations, 5) Inclusion of ADD/HD diagnosed population or population with diagnosed hallmark of ADD/HD (i.e., inattention, impulsivity, or, most commonly, hyperactivity), 6) Studies presented at conferences or society meetings were not included.

Studies included in the review were garnered from search of the electronic data bases. These sources included Medline, PsycInfo, ProQuest, and ERIC data bases. The Boolean search sequence which was the most productive was (EEG biofeedback OR Neurofeedback) AND (Attention Deficit Disorder OR hyperactivity). Additional search on the Internet yielded no additional peer reviewed publications; however, case studies were found on websites associated with the publication, MegaBrain (relevant issues available through the website: http://users.aol.com/eegspectrum/adhd.htm). Additional reports of successful outcome case studies and multiple subject designs are available and can be accessed through Compuserve's Mind/Body Forum (B. Hurwitz, personal communication, March 22, 1997). Although subscription to Compuserve is a prerequisite for access, a website giving general characteristics of the forum and membership information for Compuserve is freely available (http:directory.compuserve.com/Forums/MIND/Abstract.htm).

Case Studies

Three case studies reported EEG treatment. Two of the three represent the work of Tansey (Tansey, 1993; Tansey & Bruner, 1983). Tansey reported

the successful private practice use of EEG biofeedback with a child-team diagnosed, hyperactive 10-year-old boy who had failed a fourth-grade class for the perceptually impaired (Tansey & Bruner, 1983). He also published a ten year follow-up of the subject along with comparison to a matched group of children (Tansey, 1993). The third case study was reported by Lubar and Shouse (1976), and was intended to provide a preliminary report on a single subject who was part of a larger, doctoral study (Shouse, 1977).

Tansey (Tansey & Bruner, 1983) began treatment sequence with three electromyograph (EMG) sessions designed to reduce hyperactive behavior in children unsuccessfully treated by Ritalin. Next, 14 Hz EEG was monitored and conditioned (operantly) to increase amplitude. The subject increased both amplitude and frequency of 14 Hz (defined as Sensorimotor Rhythm or SMR) activity. Hyperactivity was reduced as reported by mother. Treatment was provided 40 minutes, once-a-week, and extended for twenty sessions. Post-treatment grades indicated that the subject was doing well in a regular fourth-grade class. A follow-up study (Tansey, 1993) indicated continued school and social-related success (2.50 grade point average during currently ending college semester) combined with stability of SMR frequency and amplitude. Electroencephalograph measures were reported to compare favorably with the gains the subject made in treatment.

The case study and follow-up reported by Tansey is significant for several reasons. First, the subject made rapid and long-lasting increases in 14

Hz activity over the course of only 20 sessions. This lends face validity to the notion that operantly reinforced brain wave change relates to behavioral change. Second, subjects' achievement increased during treatment, and long term follow-up revealed college level achievement.

Multiple Subject Designs

A number of studies utilized multiple subject designs. Treatment outcome was generally positive; however, the correlation between improved behavior and successful EEG training was not firmly established. One study reported no significant results with EEG training.

Nall (1973) investigated the effects of increasing alpha waves in a population of 48 hospitalized children, all but one having an abnormal EEG. Based on results from ANOVA's, she did not find significant improvement in post-treatment measures of hyperkinetic and maladaptive behavior, although the subjects were able to increase the amplitude of their alpha waves. This was the only study utilizing a matched control group that was given false biofeedback (alpha). Though well conceived, Nall's study (1973) used unique measures, reproduced with difficulty. In addition, subjects were hospitalized together, raising the possibility of inter-subject confounds.

Lubar and Shouse published a series of three studies based on their work with a sample of hyperkinetic males between 6 and 12 years (Lubar & Shouse, 1976, 1977; Shouse & Lubar, 1979). Instead of encouraging alpha EEG production, subjects were given a feedback contingency designed to increase

12 to 14 Hz (SMR) activity and inhibit 4 to 7 Hz (Theta) activity. Activity in the 12 to 14 Hz range was presumed to be analogous to the SMR (Sensorimotor) wave that Sterman (1996) associated with motor inhibition in cat studies and successful inhibition of epileptic seizure activity. The hyperkinetic group (n=12) and normal control (n=12) were given baseline measures which included EEG measures and behavioral measures taken from parents and from multiple classroom observations. Baseline data for the hyperkinetic group (n=12) was taken under a no-drug condition and a drug only condition. Based on their extreme behavioral scores, four subjects were drawn from the set of hyperkinetic children. This comprised the treatment group. The remaining hyperkinetic children served as additional controls.

Lubar and Shouse (1976, 1977) implemented four additional sequential conditions of biofeedback training that were given only to the treatment group. These conditions were inhibition of SMR and encouragement of 4 to 7 Hz (theta) in the presence of medication, a reversal back to increasing SMR and inhibiting 4 to 7 Hz activity, and finally maintaining SMR/4 to 7 Hz conditioning while withdrawing medication. The hyperkinetic control group (n=8) received medication and no biofeedback. The normal control group was given pre- and post-measures of EEG and behavioral measures.

Based on the mean number of teacher-reported symptoms, mean number of desirable and undesirable behaviors, and the mean number of

social behaviors and incidences of motor inhibition, Lubar and Shouse (1976, 1977) found superior effectiveness for SMR training (increasing SMR and decreasing theta) combined with medication administration. When SMR was inhibited and theta was encouraged (reversal condition), motor inhibition was decreased. High arousal subjects could not be differentiated from hyperkinetic subjects based upon auditory evoked cortical responses, the incidence of SMR, or the amount of theta and basal skin (GSR) resistance (Shouse, 1977). The preliminary case study (on a selected hyperkinetic child) reported reduced incidence of behavior combined with voluntary motor inhibition and increases in SMR rhythm (Lubar & Shouse, 1976, 1977). Shouse and Lubar (1979) replicated the results in three out of the four children rated as severely hyperkinetic (the hyperkinetic treatment group).

Results from Lubar's training are compelling in that symptoms of hyperkinesis returned during reversal condition. Although a hyperactive control group was included, subjects were assigned to conditions based upon degree of symptomatology rather than random assignment.

Using the same EEG contingency, Cartozzo (1995) tested the effectiveness of EEG biofeedback training on attention and concentration measures and on reducing measures of overactivity in children with ADHD. Subjects (n=19) were selected based on cognitive testing, parent and teacher behavior ratings scales, and response to Ritalin (25 to 30% were Ritalin nonresponders). Subjects received Ritalin dosage during 30 neurofeedback

sessions. Subjects were matched by age, learning disability, special education, and other psychotherapy. They were assigned to either a biofeedback training group (9 subjects, mean age = 8.9) or a control group (10 subjects, mean age = 9.2). The treatment group received 30 sessions of biofeedback training designed to reduce theta amplitude (4 to 7 Hz) while increasing SMR (sensorimotor rhythm, 12 to 15 Hz) using a Pac-Man Game as feedback. The control group played Pac-Man manually for 30 sessions. Both groups were reinforced for on-task behavior using a token economy. Results indicated that EEG biofeedback was effective in training individuals to reduce within-session theta. Reductions were not sustained across sessions. Biofeedback was not found to be effective in increasing SMR. Theta reduction was accompanied by increased attention and concentration as measured by cognitive testing. Behavior rating scales indicated general improvements in both groups.

Parziale (1982) used a sample of 16 hyperactive males ranging in age from 6 to 12 years. Subjects were placed in either a treatment or a placebo group. Treatment length was 15 sessions. Method of EEG contingency was theta inhibition with SMR enhancement. Dependent measures such as behavioral rating scales showed significant improvement. Biofeedback measures changed in the predicted direction.

Utilizing EEG biofeedback training, Tansey (1990, 1991) presented an office-setting based treatment regimen for learning disabilities. Children

(n=24) with "brainwave signature patterns" indicating brain-based learning disability were given EEG 14 Hz biofeedback training. Five frequency bands of brainwave activity (5 Hz, 7 Hz, 10 Hz, 12 Hz, and 14 Hz) from one active sensor recorded changes in the brainwave signatures. Results showed an increase in targeted 14 Hz production. "Thus, a cerebro-neural (brainwave) rectification was observed to occur wherein slow wave (7 Hz) activity decreased in overall energy (-16%) concomitant to an EEG biofeedback trained increase (+48.9%) in energy of 14 Hz activity" (Tansey, 1990, p. 57). There was significant (> 15 point) growth in Wechsler Intelligence Scale for Children-Revised (WISC-R) Full Scale, Verbal, and Performance IQ scores, reflecting improved brain function and resultant test performance, with a "normalization" of Verbal-Performance IQ anomalies. An inverse relationship was observed between energy levels at 5 Hz and 7 Hz and pretreatment FSIQ levels.

A three component parts study (Lubar et al., 1995) was performed in a clinical setting using 23 children and adolescents with ADD/HD ranging in age from 8 to 19 years. Feedback was targeted to production of 16 to 20 Hz (beta) activity without 4 to 8 Hz (theta) activity. Post-training changes in EEG activity, Test of Variables of Attention (TOVA) performance, Attention Deficit Disorders Evaluation Scale (ADDES) behavior ratings, and WISC-R performance were assessed. Part I showed that subjects who diminished theta activity showed significant improvement in TOVA performance; Part II

indicated that trained subjects showed improved behavior ratings; and Part III showed significant increases in WISC-R scores. Findings indicated that neurofeedback training can be an appropriate and efficacious treatment for children with ADHD. The researchers also used both objective and subjective measures under relatively controlled situations.

Linden, Habib, and Radojevic (1996) reported children showed significantly greater increase (mean of 9 points) on the Kaufman-Brief Intelligence Test (K-BIT) IQ composite compared to controls. Behavioral ratings also improved. Eighteen children with diagnoses of either ADD/HD or Learning Disabled comprised the subject pool. They ranged in age from 5 to 15 years. Treatment consisted of EEG biofeedback which encouraged increased beta wave (16 to 20 Hz) and decreased theta (4 to 8 Hz).

Schnoll (1995) implemented beta reinforcement and theta inhibition with 56 school-aged children in a school environment. Results showed statistically significant increased beta activity and reduction in theta. Multiple regression was used to analyze the relationship between subject variables and post-treatment beta and theta. Significant correlations between post-treatment EEG activity and pre-treatment beta, pre-treatment theta, and Verbal/Performance IQ differences were found. Variables of age, gender, IQ, number of treatment sessions, reading level, math level, attention span, and visual-motor integration skills were not significantly correlated with beta and theta post-treatment levels.

Bell (1986) attempted to increase academic performance by enhancing 8 to 15 Hz activity and inhibiting 3 to 7 Hz activity in four Learning Disabled (LD) diagnosed children aged 9 to 13. Treatment ran for 31 twice weekly sessions. Results indicated statistically significant improvement in the LD group compared to normal and LD controls in reading comprehension and on Bender Gestalt drawings. No other significant outcomes were noted in psychoeducational or neuropsychological tests (measures of attention); however, general improvement was noted for treatment group.

Lubar and Lubar (1984) provided six ADD/HD children with long-term neurofeedback and academic treatment for their attention-deficit disorder. Feedback was contingent for either increasing 12 to 15 Hz SMR or 16 to 20 Hz beta activity in the absence of gross body movement or theta (4 to 8 Hz) activity. Academic treatment included reading, arithmetic, and spatial tasks to improve attention. Subjects increased SMR or beta activity and decreased theta and EMG activity. All subjects improved school grades or achievement test scores.

Methodological Considerations

Subject Variables

A total of 232 unique subjects were studied by researchers who published the 16 treatment studies reviewed in this paper. Some of the treatment studies used the same subject pools. For example Tansey's 1993

report was a follow-up of his case study performed 10 years earlier (Tansey & Bruner, 1983). In addition Tansey (1990) published initial treatment results followed by an expansion of the dependent variables used in the original study (Tansey, 1991). Lubar and Shouse (Lubar and Shouse, 1976, 1977; Shouse and Lubar, 1979) published a series of studies (including one preliminary case study) drawing on the sample initially used for Shouse's doctoral work (Shouse, 1977). An additional, three part study (Lubar et al., 1995) used two unique sample groups and one sample group composed of a subset of those who participated in the previous two groups. Excluding duplicate studies and case studies, the 16 studies were based on 11 unique subject samples.

The 11 subject samples matched accepted ADD/HD clinical presentation (DSM-IV). Twenty-one of the subjects (9%) were female, although Nall (1978) and Linden et al. (1996) did not report gender data. This approaches the lower end of estimates for occurrence of ADD/HD diagnosed females in clinical populations (one female per nine males (DSM-IV). Although means for subject ages were not consistently reported, ages ranged from 5 to 19 years.

Most studies used populations originating in an outpatient treatment clinic. Schnoll (1995) received her referrals exclusively through the school system. Nall (1973) used 48 hospitalized children; however, specific diagnoses

were not reported. The number of subjects in the studies was relatively small, as are the number of studies.

A variety of sources established the diagnosis of ADD/HD in the sample population for the treatment studies. Professionals involved in diagnosis and referral included psychologists, pediatricians, nurses, and child-care treatment teams. Parents and teachers were the original source of referral, which is consistent with how the ADD/HD child impacts the environment.

Most treatment studies aimed to normalize hyperactivity or hyperkinesis. Tansey (1990, 1991) proved one of the exceptions in that he focused on cognitive and attentional components. Schnoll (1995) and Bell (1986) focused on attention span and cognitive and academic skills. The above studies (Tansey, 1990, 1991; Schnoll, 1995; Bell, 1986) addressed the treatment of inattention directly. Cartozzo (1995) and Lubar et al. (1995) emphasized both inattention and hyperactivity. Impulsivity, the final hallmark for ADD/HD, has not been as thoroughly investigated as hyperactivity and inattention. In fact it was not mentioned as a primary targeted symptom in any of the reviewed literature. In summary, EEG biofeedback contingencies were employed to address two of the three core symptoms of ADD/HD, hyperactivity and inattention.

EEG Reinforcement Contingencies

Three types of feedback contingencies were: alpha feedback alone, theta inhibition combined with SMR reinforcement, and theta inhibition combined with beta reinforcement. Alpha reinforcement has been associated with increases in relaxation and relaxed alertness (Kamiya, Callaway, & Yeager, 1969). Nall (1973) reported no significant reduction of hyperkinetic behavior or increase of academic achievement with alpha wave reinforcement.

Lubar and Shouse (Lubar & Shouse, 1976, 1977; Shouse, 1977; Shouse & Lubar, 1979), Lubar and Lubar (1984), Cartozzo (1995), and Parziale (1982) used theta inhibition combined with SMR reinforcement. Cartozzo found no improvement in behavior following 30 treatment sessions. Lubar and Shouse (1976, 1977) and Parziale reported positive changes in behavior, Lubar and Lubar noted improvement in grades and achievement test scores. SMR reinforcement alone has been associated with behavioral inhibition (Sterman, 1996). Theta or slow-wave activity has been associated with low-arousal.

Lubar et al. (1995), Linden et al. (1996), Schnoll (1995), Bell (1986), and Lubar and Lubar (1984) implemented theta inhibition combined with beta reinforcement. Outcomes were generally positive. Post-training EEG beta and theta improved (Lubar et al.). Linden et al. replaced EEG machines in mid-experiment and could not rely on EEG results. Schnoll found statistically

significant increased beta activity and a reduction in theta. Lubar and Lubar also reported successful increase of beta and reduction in theta. Finally, Tansey (1990, 1991) approached the problem of EEG contingencies using different reinforcement contingency and electrode placement. He only reinforced 14 Hz activity as measured by a single electrode centered over the Rolandic cortex. He reported improvement in 14 Hz activity and decrease in 7 Hz activity with this design.

Although treatment studies reported generally favorable outcomes, EEG contingencies were rarely used alone. Additional reinforcers included intermittent verbal reinforcement and repetition of instructions after three sessions of EMG training (Tansey & Bruner, 1983). Nall (1973) did not provide additional reinforcement; however, neurotherapy continued for the children. Shouse and Lubar (1979) varied two treatment conditions, medication and SMR enhancement combined with theta inhibition. Cartozzo (1995) and Parziale (1982) employed a token economy in addition to EEG reinforcement. Lubar and Lubar (1984) alternated the feedback portions of their sessions that were "chosen to be compatible with the child's academic needs and interest. During the later months of training, specific academic tasks — e.g., spelling, arithmetic, reading — alternated with biofeedback during each session"(p. 6). Lubar et al. (1995) utilized pure visual reward (color-wheel interactive game), while Pac-man Game was used as a visual reinforcer in another study (Cartozzo, 1995). Varied implementation of

contingency reward and use of other treatments keeps the treatment literature a collection of difficult to compare preliminary studies.

Length of Treatment

Length of treatment varied throughout the reviewed studies. Schnoll (1995) unexpectedly found no significant relationship between age, IQ, and number of treatment sessions (mean of 26.6 sessions) with post-treatment beta or post-treatment theta. Parziale (1982) had 15 sessions; Tansey and Bruner (1983), 20 sessions; Tansey (1990, 1991), a mean of 27.9 sessions; Cartozzo (1995), 30 sessions; Lubar et al. (1995), approximately 40 sessions; Linden et al. (1996), 40 sessions; and Nall (1973), approximately 60 sessions. Lubar and Shouse (1976, 1977) and Shouse and Lubar (1979) conducted 76 to 182 sessions.

EEG Sampling

Sampling procedures have improved with time. Nall (1973) reported an experimental design where subjects were given audio feedback and visual feedback in the form of a voltmeter. Now with computerization, EEG can be monitored, be given real-time visual representation, and be recorded and analyzed. Gaps in technology between the studies are certainly a significant form of non-equivalency; however, use of dependent measures such as behavioral rating scales help to sustain comparability of studies.

Kellaway (1990) comments on the importance of electrode location:

normal beta activity was reported to vary in location of propagation. Bilateral variability between hemispheres is normal (Kellaway, 1990). The body of treatment research is broken down into location recommendations to help increase the reliability and validity of measures to ensure outcome consistency. Specific placement would then be in accordance to the "ten-twenty electrode system" which is an internationally recognized standard for the placement of 21 electrodes on the scalp (Tyner, Knott, & Meyer, 1983). Recent data published in Gevins and Bressler (as cited in Niedermeyer & Lopes Da Silva, 1993) demonstrated that increasing the monitoring channels to 51 showed true distribution of electrical event-related potentials with increased resolution. Sixteen channel monitoring had insufficient spacial sampling, whereas 27 channel monitoring was marginal.

Implementation of changing reinforcement schedules is another factor decreasing comparability of test results. In order to implement a particular reinforcement contingency, baseline measures of waves to be reinforced or inhibited (e.g., beta reinforcement and beta inhibition) are objectively recorded. Reward and inhibition thresholds, however, are subjective, left to clinical judgment on a subject to subject basis. It is doubtful, judging from wide subject variability, that a reproducible reinforcement schedule is obtainable without loosing clinical utility.

Measures of Hyperactivity

Behavioral Rating Scales such as the Conners scale (Conners, 1969) and McCarney Attention Deficit Disorders Evaluation Scale (ADDES; McCarney, 1989) have been used to diagnose ADD/HD, provide baseline behavioral measures, and post-test measures. Potashkin and Beckles (1990) defined hyperactivity as an independent, quantitative dimension of behavior. Selection of behavior rating scales varied among investigators. Nall (1973) used an in-house symptom check list to track or to quantify behavior; Parziale (1982), the Conners behavior rating scale; Linden et al. (1996), the Iowa-Conners behavior rating scale (Atkins & Milich, 1987) and the Swanson, Nolan, and Pelham questionnaire (SNAP; Swanson, Nolan, & Pelham, 1982); Lubar et al. (1995), the ADDES (McCarney, 1989); and Lubar and Shouse (1976, 1977), an adapted version Wahler's Category System (Wahler, House, & Stanbaugh, 1975). Tansey (1990, 1991) did not use behavioral measures. Wahler's Category System (adaptation) and Nall's use of an in-house measure can not be judged as to reliability and validity. The ADDES has been criticized has having poor discrimination and criterion-validity (Adesman, 1991). This class of measure has also fallen under criticism due to inaccuracy of parent and teacher over- or under-reporting of behavior change and to the fact that it does not measure skill acquisition (Lipman & Kendall, 1992). Research into whether parents inaccurately report has found that maternal reports of

children's psychopathology were both reliable and accurate (Faraone, Biederman, & Milberger, 1995).

Measures of Attention

Measures of attention have been sparse and problematic to implement and define (study by Anthony, Mersky, Ahearn, Kellam, & Eaton, 1988, as cited in Riccio, Morris, Hynd, & Keith, 1996; Fletcher, Morris, & Francis, 1991). Neuropsychological tests such as continuous performance tests (Lezak, 1995) have finally been included in the dependent measures of the treatment literature. Test of Variables of Attention (TOVA; Greenberg, 1991) is, to date, the only continuous performance test used. TOVA purports to measure both impulsivity (by errors of commission) and attention (by errors of omission). Parziale (1982) used Bender-Gestalt and Digit Span Subtest (WISC-R) but reported no significant improvement following treatment. Nall (1973) used Digit Span Subtest (Stanford-Binet) and auditory related and auditory unrelated memory span (Detroit Test of Mental Ability). No significant improvement was reported.

Achievement Measures

Achievement measures substantially increase the validity of treatment studies. Since ADD/HD children have problems in school and expected outcome of successful treatment is improved grades, in a case study, Tansey and Bruner (1983) reported academic progress (fourth-grade grades by quarter). Nall (1973) used outcomes in reading comprehension losses (Gray-Votaw-

Rogers General Achievement Test) and total educational gains (GPA). Nall's subjects, however, did not show significant improvement in grades. Although Schnoll (1995) used measures of achievement (reading and math levels), these levels were examined to predict post-treatment EEG performance and were not used as indexes of change due to treatment. Lubar and Lubar (1984) reported "considerable improvement" in grades and/or achievement test scores.

Measures of IQ

Measures of cognitive or intellectual performance have been implemented to substantiate improvements in school-related skills and cognitive ability. Cognitive and problem-solving measures have been shown to be strong discriminators of hyperactive and non-hyperactive children (study by Homatides & Konstantareas, 1981, as cited in Kendall, 1993). Measures of cognition lend credence to the hypothesis that normalized brain wave patterns are associated with improved performance. Tansey (1990, 1991) noted very compelling IQ improvements. Linden et al. (1996) found mean increase in IQ of 9.3 ($p<0.05$) in treatment group, with no significant change in matched controls. Lubar et al. (1995) also noted significant IQ improvement. Only three studies have included IQ measures. The three showed compelling results. Future studies should include IQ measures.

Measures of Impulsivity

Measures of impulsivity are sparsely employed. There has been no study specifically designed to study and treat this core symptom. Achenbach and Edelbrock (1983) were unable to differentiate impulsivity from hyperactivity in a factor analytic study. Although TOVA purports to measure a dimension of impulsivity (errors of commission), only two studies used this instrument (Cartozzo, 1995; Lubar et al., 1995). Cartozzo and Lubar et al. noted substantial improvement on TOVA performance; however, neither reported outcomes using commission errors in isolation.

Variables Infrequently Studied

This section is devoted to variables that may be important in terms of treatment efficacy, but have received, as yet, little attention. The list discussed is not inclusive; however, it represents a judgment as to what may be helpful for clinicians to hear. The two questions that are clinically important, but infrequently studied, are: (a) how best to engage the cooperation of a hyperactive child and (b) what reward stimulus is most salient or effective. Foster (1991) reported that brain wave production can be influenced by the saliency of stimuli used. Lubar (1995) recommended a comprehensive treatment approach designed to meet the child's attention and control capacities at any given moment. Tasks of achievement and attentional training are rotated with EEG training based upon the child's interest and growing level of expertise. This form of treatment meshes well with the

child, but is not easily amenable to empirical replication. It is not surprising that numerous case reports were published in non-peer reviewed literature showing positive treatment outcomes (e.g., Othmer & Othmer, 1992; Othmer, Othmer, & Marks, 1991).

Discussion and Conclusions

Literature Status

Sixteen studies were reviewed that employed EEG biofeedback treatment contingency using children who have Attention-Deficit/Hyperactivity Disorder. Based on review of treatment literature, studies implementing EEG biofeedback for ADD/HD children are still in the preliminary or exploratory stage. The preliminary or exploratory status that this body of treatment literature retains is attributed to the design flaws that in many cases are attributed to the clinical demands of this population. Study size is small (16 studies), and the number of unique subject samples is even smaller (11 subject pools). As with any emerging field, subject selection and assignment has depended more on ensuring particular subject characteristics than on need for statistical control.

Newer studies, however, have increasingly used controls matched for age and other critical subject characteristics such as gender, similar diagnoses, and treatment equivalence. It is due to the relative dearth of samples across any one treatment formulation, the sacrifice of random assignment in favor

of increasing face validity, and the relatively small size of the subject samples that stronger correlations can not be made between EEG treatment and the largely uniform reports of positive outcomes. In a field with many uncertainties, it is these positive outcomes that merit further investigation.

The hallmarks of ADD/HD have been unevenly measured and researched. Two of the three major hallmarks of ADD/HD (hyperactivity and inattention) remain the primary focus of the sixteen studies. Impulsivity, the third major hallmark, has historically been difficult to operationalize across all treatment modalities. Tests of continuous performance (e.g., TOVA) that do provide some objective measure of impulsivity have not been implemented in such a way as to provide interpretable results. Some controversy has been noted that the diagnosis of ADD/HD may not address the underlying substrate; however, there is no proven etiology. The diagnosis of ADD/HD may overlap other diagnostic entities, and so may not reflect a unique etiology.

Treatment literature generally substantiates the neurophysiological model for ADD/HD outlined by Sterman (1996). There is a correlation between the EEG and levels of arousal based on indexes of physiological arousal and wave amplitude, though it is not always significant. Although Kellaway (1990) minimizes these differences (citing wide EEG variability in normals), brain mapping, power spectra studies and ERP studies have proven

effective in showing group differences between normals and ADD/HD and LD children on a wide range of tasks.

The effect has generally continued that children have been able to modify their brain waves. ERP and power spectra studies have suggested that there is a biological difference between ADD/HD children and children displaying pure behavior dysfunction. Studies found significant amplitude differences in theta band on tasks when comparing ADD/HD children and normally achieving controls. And increased beta and decreased theta were observed in better performing groups measured by different ERP and power spectra studies across different tasks.

Multiple subject designs employ three different EEG contingencies (alpha reinforcement, beta reinforcement + theta inhibition, SMR reinforcement + theta inhibition). Although the use of different treatment contingencies threatens generalizability, the observed positive treatment outcomes across contingencies may point to a robust treatment effect. Case studies have shown that individual subjects can make rapid and permanent modification in brain wave activity with concomitant positive behavior change and improved achievement. Comparative studies have not shown any single treatment modality superior to another.

Treatment Efficacy

EEG treatment should be considered an efficacious adjunctive treatment for children with ADD/HD at this time. Alternative treatments for

ADD/HD such as medication, cognitive-behavioral training, EMG biofeedback and other forms of relaxation do not address the varied and sometimes comorbid symptoms that present in the ADD/HD child. With the exception of medication, which has been shown to have effects primarily on hyperactive symptoms, no form of treatment has consistently outperformed another. In this milieu, where there is no single, effective treatment, research into a compelling area such as EEG biofeedback should proceed, and this form of treatment should be considered efficacious because of its preliminary positive results.

Some clinicians argue that controlled studies targeted to account for placebo effect and specific EEG contingency are not necessary and may even be counterproductive given the wide variability of EEG production and the absence of levels of any brain wave. Othmer and Othmer (1994) state that the most relevant outcomes are seen in amelioration of behavior problems and mental performance that can be measured by objective means. In response to Barkley's criticism (as cited in Othmer, Kaiser, & Othmer, 1995) that even the placebo effect of the apparatus may account for positive treatment results, they reply that such effects are more than counteracted by using the subject (patient) as his or her own control, based on multiple baseline measures. This argument has validity, especially when one considers the complexity of many children who present with ADD/HD. However, confounding treatment variables unique to each treatment case are most easily accounted

for in controlled, multiple subject designs. Also the very uniqueness and complexity of each case, along with outside variables that inevitably act in each case, would limit the generalizability of individual case studies with baseline controls. What is important to remember is that both designs are important and contribute to our knowledge about what part of EEG biofeedback accounts for its positive treatment effects.

Lubar (1995) provides specific recommendations for comprehensive treatment. Based on clinical acumen and research, he cites a robust positive effect in at least 3,000 subjects. Clinical reports contribute to positive treatment effect and support a neurophysiological basis, and the treatment studies provide partial support for the specificity of contingencies.

Directions for Future Research

There is noted difficulty in collecting large samples of ADD/HD children whose parents are willing to place them in a research study which may involve no treatment (Lubar, 1991). Treatment factors such as length of time (generally 40 sessions) and ethical considerations for the child mean that controlled studies will probably remain limited to waiting-list controls. Nevertheless, without more controlled studies the validity of this treatment remains questionable.

Clinicians working in the field generally do not have access to the resources which may enable the procurement of large samples or provide an ethical alternative for handling control group issues. It is time for this form

of treatment be studied at more university settings. Rasey, Lubar, McIntyre, Zoffuto, and Abbot (1996) provide an example of this approach. They found that normal young adults classified as learners (subjects able to increase beta and decrease theta) can achieve significant improvements on measures of attention after relatively short periods of training (mean of 20 sessions). Further investigation of EEG biofeedback as a possible booster for cognitive performance is worth investigating, particularly as it would take advantage of a larger sample base (college students). Preliminary investigation into EEG biofeedback (10 Hz) has been tried as part of focusing training to enhance psychomotor tasks; however, studies have reported variable success (Gillette, 1983; Matteson, 1981). Additional research is needed with normal populations to delineate the effects of EEG biofeedback training on brain functioning of non-patient populations, providing information with which to compare the effects of treatment of symptomatic conditions. One must keep in mind, however, that parameters of treatment efficacy for "normal" and "non-normal" populations may not be the same.

Double-blind studies are necessary to clarify the relationship between therapist, EEG contingency, and patient variables. It is technologically feasible to design EEG programs which can allow a true double-blind study. Use of multiple EEG measures such as that employed by Lubar et. al. (1995) should continue. Real-time or within-session EEG output is variable, which

decreases the amount of confidence. The inclusion of power-spectra and ERP studies increase inferential power.

In summary:

1. There is a wide-ranging and growing body of evidence of a connection between the neurophysiology of ADD/HD and the effects of EGG biofeedback training. In spite of design flaws and small subject pools, preliminary clinical and laboratory investigations support a positive relationship between increases in 16 to 20 Hz activity and improvement on measures of attention; a negative relationship between increases of 12 to 15 Hz activity and decreases in hyperactive behavior; and an inverse relationship between 4 to 7 Hz activity and mental efficiency.

2. Before recommending EEG biofeedback treatment, diagnostic issues need to be delineated. The diagnosis of ADD/HD should be based on comprehensive assessment which includes at least the following: (a) measures of attention, impulsivity, and hyperactivity; (b) measures of cognitive functioning and academic achievement; and (c) measures of emotional adjustment. Misdiagnosed children (e.g., depression and adjustment problems) with behavioral symptoms similar to those of ADD/HD children may not benefit from an EEG biofeedback training protocol designed for the ADD/HD child.

3. EEG biofeedback training appears to work best as part of a multimodal treatment approach including medication (when necessary), family

intervention, increased structure in the classroom, behavior modification techniques, tutoring for specific learning problems, social skills training, and supervised recreational activities (Barkley, 1990; Lubar, 1991, 1995).

REFERENCES

Abikoff, H. A. (1991). Cognitive training in ADHD children: Less to it than meets the eye. Journal of Learning Disabilities, 24, 205-218.

Achenbach, T.M., & Edelbrock, C. (1983). Manual for the Child Behavior Check-list and Revised Child Behavior Profile. Burlington, VT: University of Vermont, Department of Psychiatry.

Ackerman, P. T., Dykman, R. A., Oglesby, D. M., & Newton, J. E. O. (1994). EEG power spectra of children with dyslexia, slow learners, and normally reading children with ADD during verbal processing. Journal of Learning Disabilities, 27, 619-630.

Ackerman, P. T., Dykman, R. A., Oglesby, D. M., & Newton, J. E. O. (1995). EEG power spectra of dysphonetic and nondysphonetic poor readers. Brain and Language, 49, 140-152.

Adesman, A. R. (1991). Test and measurement review: The Attention Deficit Disorders Evaluation Scale. Developmental and Behavioral Pediatrics, 12, 65-66.

American Psychiatric Association. (1968). Diagnostic and statistical manual of mental disorders (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (3rd ed. rev.). Washington, DC: Author.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Anastopoulos, A. D., & Barkley, R. A. (1992). Attention deficit-hyperactivity disorder. In C. E. Walker & M. C. Roberts (Eds.), Handbook of clinical child psychology (2nd ed., pp. 413-429). New York: Wiley & Sons.

Atkins, M., & Milich, R. (1987). IOWA-Conners Teacher Rating Scale. In M. Herson & A. Bellack (Eds.), Dictionary of behavioral assessment techniques (pp. 273-275). New York: Pergamon.

Baer, R. A., & Nietzel, M. T. (1991). Cognitive and behavioral treatment of impulsivity in children: A meta-analytic review of the outcome literature. Journal of Clinical Child Psychology, 20, 400-412.

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

Bell, C. R. (1986). An investigation of the remediation of learning disabilities utilizing EEG biofeedback as measured by neuropsychological and psychoeducational tests, and EEG spectral analysis. Dissertation Abstracts International, 46(09), 3235B. (University Microfilms No. ACC 85-24113)

Bradley, C. (1937). The behavior of children receiving benzedrine. American Journal of Psychiatry, 94, 577-585.

Brown, R. T., Borden, K. A., Wynne, M. E., Schleser, R., & Clingerman, S. R. (1986). Methylphenidate and cognitive therapy with ADD children: A methodological reconstruction. Journal of Abnormal Child Psychology, 14, 481-497.

Cambell, M., & Cueva, J. E. (1995). Psychopharmacology in child and adolescent psychiatry: A review of the past seven years, Part I. Journal of the American Academy of Child and Adolescent Psychiatry, 34, 1124-1132.

Cartozzo, H. A. (1995). EEG biofeedback and the remediation of ADHD symptomatology: A controlled treatment outcome study. Dissertation Abstracts International, 56(05),1706A. (University Microfilms No. AAC 95-30291)

Cobb, D. E., & Evans, J. R. (1981). The use of biofeedback techniques with school-aged children exhibiting behavioral and/or learning problems. Journal of Abnormal Child Psychology, 9, 251-281.

Cohn, R., & Nardini, J. E. (1958). The correlation of bilateral occipital slow activity in the human EEG with certain disorders of behavior. American Journal of Psychiatry, 115, 44-54.

Conners, C. A. (1969). A teaching rating scale for use in drug studies with children. American Journal of Psychiatry, 126, 884-888.

Deboskey, D. S. (1982) An investigation of the remediation of learning disabilities based on brain-related tasks as measured by the Halstead-Reitan Neuropsychological Test battery [CD-ROM]. Abstract from: ProQuest File: Dissertation Abstracts Item: 8225330

Denkowski, K. M. & Denkowski, G. C. (1984). Is group progressive relaxation training as effective with hyperactive children as individual EMG biofeedback treatment? Biofeedback Self Regulation, 9, 353-364.

Douglas, V. I. (1972). Stop, look, and listen: The problem of sustained attention and impulse control in hyperactive and normal children. Canadian Journal of Behavioural Science, 4, 259-282.

Durlak, J. A. (1992). School problems of children. In C. E. Walker & M. C. Roberts (Eds.), Handbook of Clinical Child Psychology (2nd ed., pp. 497-510). New York: Wiley & Sons.

Faraone, S. V., Biederman, J., & Milberger, S. (1995). How reliable are maternal reports of their children's psychopathology? One-year recall of psychiatric diagnosis of ADHD children. Journal of the American Academy of Child and Adolescent Psychiatry, 34, 1001-1007.

Feldman, H., Crumrine, P., Handen, B. L., Alvin, R., & Teodori, J. (1989). Methylphenidate in children with seizures and attention-deficit disorder. American Journal of Diseases of Children, 143, 1081-1086.

Fletcher, J. M., Morris, R. D., & Francis, D. J. (1991). Methodological issues in the classification of attention-related disorders. Journal of Learning Disabilities, 24, 72-77.

Foster, D. S. (1991). EEG and subjective correlates of alpha frequency binaural beats stimulation combined with alpha biofeedback [CD-ROM]. Abstract from: ProQuest File: Dissertation Abstracts Item: 9025506

Frost, J. D., Jr., Carrie, J. R. G., Borda, R. P., & Kellaway, P. (1973). The effects of Dalmane (flurazepam hydrochloride) on human EEG characteristics. Electroencephalography and Clinical Neurophysiology, 34, 171-175.

Gillette, D. L. (1983). The effect of synchronized, multichannel EEG biofeedback and 'open focus' training upon the performance of selected psychomotor tasks [CD-ROM]. Abstract from: ProQuest File: Dissertation Abstracts Item: 8317366

Gittleman, R., Abikoff, H., Pollack, E., Klein, D. F., Katz, S., & Mattes, J. (1980). A controlled trial of behavior modification and methylphenidate in hyperactive children. In C. K. Whalen & B. Henker (Eds.), Hyperactive children: The social ecology of identification and treatment (pp. 221-243). New York: Academic Press.

Greenberg, L. M. (1991). T.O.V.A. interpretation manual. Minneapolis, MN: Author.

Hughes, H., Henry, D., & Hughes, A. (1979). The effect of frontal EMG biofeedback training on the behavior of children with activity-level problems. Biofeedback and Self-Regulation, 5, 207-219.

Janzen, T., Graap, K., Stephanson, S., Marshall, W., Fitzsimmons, G. (1995). Differences in baseline EEG measures for ADD and normally achieving preadolescent males. Biofeedback and Self-Regulation, 20, 65-82.

Jasper, H. H., Solomon, P., & Bradley, C. (1938). Electroencephalographic analyses of behavior problem children. American Journal of Psychiatry, 95, 641-658.

Kamiya, J., Callaway, E., & Yeager, C. L. (1969). Visual evoked responses in subjects trained to control alpha rhythms. Psychophysiology, 5, 683-695.

Kaplan, I. K., & Sadock, B. J. (1988). Synopsis of psychiatry (5th ed.). Baltimore: Williams & Wilkins.

Kassel, S. (1986). The effects of EEG biofeedback training on hyperactive and /or learning disabled children (Report No. RIENOV 87). (ERIC Document Reproduction Service No. ED 283 351)

Kellaway, P. (1990). An orderly approach to visual analysis: Characteristics of the normal EEG of adults and children. In D. D. Daly & T. A. Pedley (Eds.), Current practice of clinical electroencephalography (pp. 139-199). New York: Raven Press.

Kendall, P. C. (1993). Cognitive-behavioral therapies with youth: Guiding theory, current status, and emerging developments. Journal of Consulting and Clinical Psychology, 61, 235-247.

Lahey, B. B., Stempniak, M., Robinson, E. J., & Tyroler, M. J. (1978). Hyperactivity and learning disabilities as independent dimensions of child behavior problems. Journal of Abnormal Psychology, 87, 333-340.

Lee, S. W. (1991). Biofeedback as a treatment for childhood hyperactivity: A critical review of the literature. Psychological Reports, 68, 163-192.

Lezak, M. D. (1995). Neuropsychological assessment. New York: Oxford University Press.

Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. Biofeedback and Self-Regulation, 21, 35-49.

Lindsley, D. B., & Cutts, K. K. (1940). Electroencephalograms of "constitutionally inferior" and behavior problem children: Comparison with those with normal children and adults. Archives of Neurology and Psychiatry, 44, 1199-1212.

Linkenhoker, D. (1983). Tools of behavioral medicine: Applications of biofeedback treatment for children and adolescents. Developmental and Behavioral Pediatrics, 4, 16-20.

Lipman, A. J., & Kendall, P. C. (1992). Drugs and psychotherapy: Comparisons, contrasts, and conclusions. Applied & Preventive Psychology, 1, 141-148.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. Biofeedback Self Regulation, 16, 201-225.

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

Lubar, J. O., & Lubar J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. Biofeedback Self Regulation, 9, 1-23.

Lubar, J. F., Mann, C. A., Gross, D. M., & Shively, M. S. (1992). Differences in semantic event-related potentials in learning-disabled, normal, and gifted children. Biofeedback and Self-Regulation, 17, 41-57.

Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. Biofeedback and Self-Regulation, 1, 293-306.

Lubar, J. F., & Shouse, M. N. (1977). Use of biofeedback in the treatment of seizure disorders and hyperactivity. In B. B. Lahey & A. Kazdin (Eds.), Advances in clinical child psychology: Vol. 1 (pp. 203-265). New York: Plenum Press.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of EEG neurobiofeedback 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, 29, 83-99.

Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: Controlled study with clinical implications. Pediatric Neurology, 8, 30-36.

Matsuura, M., Okubo, Y., Toru, M., Kojima, T., He, Y., Hou, Y., Shen, Y., & Lee, C. K. (1993). A cross-national EEG study of children with emotional and behavioral problems: A WHO collaborative study in the western Pacific region. Biological Psychiatry, 34, 59-65.

Matteson J. H., II. (1981). A comparison of two feedback encephalography training programs (with self-regulation strategies) upon selected physiological, psychological, and motor performance correlates of arousal and attention. Dissertation Abstracts International, 42(06), 2590B. (University Microfilms No. 81-25869)

McCarney, S. (1989). Attention Deficit Disorders Evaluation Scale Home Version. Columbia, MO: Hawthorne Educational Services.

Meichenbaum, D. H. (1975). Self-instructional methods. In F. H. Kanfer & A. P. Goldstein (Eds.), Helping people change. New York: Pergamon Press.

Miller, N. E., & Dworkin, B. R. (1974). Visceral learning. In P. Obrist, A.H. Black, J. Brenner, & L. V. Cara (Eds.), Cardiovascular psychophysiology (pp. 312-331). Chicago: Aldine Press.

Mundy-Castle, A. C. (1951). Theta and beta rhythm in the electroencephalograms of normal adults. Electroencephalography and Clinical Neurophysiology, 3, 477-486.

Nall, A. (1973). Alpha training and the hyperkinetic child - is it effective? Academic Therapy Quarterly, 9, 5-19.

Niedermeyer, E., & Lopes Da Silva, F. (Eds.). (1993). Electroencephalography (3rd ed.). Baltimore: Williams and Wilkins.

Nowlis, D.P., & Kamiya, J. (1970). The control of electroencephalographic alpha rhythms through auditory feedback and the associated mental activity. Psychophysiology, 6, 476-484.

O'Leary, K. D. (1980). Pills or skills for hyperactive children. Journal of Applied Behavior Analysis, 13, 191-204.

Ortiz, T., Exposito, F. J., Miguel, F., Martin-Loeches, M., & Rubia, F. J. (1992). Brain mapping in dysphonemic dyslexia: In resting and phonemic discrimination conditions. Brain and Language, 42, 270-285.

Othmer, S., Kaiser, D., & Othmer, S. F. (1995). EEG biofeedback training for attention deficit disorder: A review of recent controlled studies and clinical findings. (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436)

Othmer, S., & Othmer, S. F. (1992). EEG biofeedback for attention deficit hyperactivity disorder. (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436)

Othmer, S., & Othmer, S. F. (1994). EEG biofeedback: Medicine, therapy, or learning? (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436)

Othmer, S. F., Othmer, S, & Marks, C.S. (1991). EEG biofeedback training for attention deficit disorder, specific learning disabilities, and associated conduct problems. (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436)

Parziale J. L. (1982). The effects of EEG biofeedback training on the behavior of hyperactive children. Dissertation Abstracts International, 43(03), 881B. (University Microfilms No. AAC82-17451)

Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and b-endorphin levels in alcoholics. Alcoholism: Clinical and Experimental Research, 13, 271-279.

Physicians' desk reference (48th ed.). (1994). Montvale, NJ: Medical Economics Data Production Company.

Pliszka, S. R. (1991). Anticonvulsants in the treatment of child and adolescent psychopathology. Journal of Clinical Child Psychology, 20, 277-281.

Potashkin, B. D., & Beckles, N. (1990). Relative efficacy of ritalin biofeedback treatments in the management of hyperactivity. Biofeedback and Self-Regulation, 15, 305-315.

Rasey, H. W., Lubar, J. F., McIntyre, A., Zoffuto, A. C., Abbott, P. L. (1996). EEG biofeedback for the enhancement of attentional processing in normal college students. Journal of Neurotherapy, 1, 15-21.

Riccio, C. A., Morris, J. C., Hynd, G. W., & Keith, R. W. (1996). Validity of the auditory continuous performance test in differentiating central processing auditory disorders with and without ADHD. Journal of Learning Disabilities, 2, 561-566.

Richter, N. C. (1984). The efficacy of relaxation training with children. Journal of Abnormal Child Psychology, 12, 319-344.

Richters, J. E., Arnold, L. E., Jensen, P. S., Abikoff, H., Conners, C. K., Greenhill, L. L., Hechtman, L., Hinshaw, S. P., Pelham, N. E., & Swanson, J. M. (1995). NIMH collaborative multisite multimodal treatment study of children with ADHD: I. Background and rationale. Journal of the American Academy of Child and Adolescent Psychiatry, 34, 987-1000.

Rosenfeld, J. P. (1990). Applied psychophysiology and biofeedback of event-related potentials (brain waves): Historical perspective, review, future directions. Biofeedback and Self-Regulation, 15, 99-119.

Rozelle, G. R., & Budzynski, T. H. (1995). Neurotherapy for stroke rehabilitation: A single case study. Biofeedback and Self-Regulation, 20, 211-228.

Rutter, M., Tizard, J., & Whitmore, K. (1970). Education, Health and Behavior. London: Longmans.

Safer, D., Allen, R., & Barr, E. (1972). Depression of growth in hyperactive children on stimulant drugs. New England Journal of Medicine, 287, 217-220.

Satterfield, J. H., & Dawson, M. E. (1971). Electrodermal correlates of hyperactivity in children. Psychophysiology, 8, 191-197.

Satterfield, J. H., Lesser, L. I., Saul, R. E., & Cantwell, D. P. (1973). EEG aspects in the diagnosis and treatment of minimal brain dysfunction. Annals of New York Academy of Science, 205, 274-282.

Seifert, A. R., & Lubar, J. F. (1975). Reduction of epileptic seizures through EEG biofeedback training. Biological Psychology, 3, 81-109.

Schnoll, E. R. (1995). EEG beta and theta training with learning disabled children: Variables associated with individual responsivity. Dissertation Abstracts International, 56(08), 4592B. (University Microfilms No. AAC95-40119)

Shouse, M. N. (1977). The role of CNS arousal levels in the management of hyperkinesis: Methylphenidate and EEG biofeedback training. Dissertation Abstracts International, 37(08), 4206B. (University Microfilms No AAC77-03685)

Shouse, M. N., & Lubar, J. F. (1979). Operant conditioning of EEG rhythms and Ritalin in the treatment of hyperkinesis. Biofeedback and Self-Regulation, 4, 299-312.

Sterman, M. B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: Implications for self-regulation. Biofeedback and Self-Regulation, 21, 3-33.

Stevens, J. R., Sachdeo, K., & Milstein, V. (1968). Behavior disorders of childhood and the electroencephalogram. Archives Neurology, 18, 160.

Still, G.F. (1902). The Coulstonian lectures on some abnormal psychiacal conditions in children. Lancet, 1008-1012.

Strauss, A. A., & Lehtinen, V. (1947). Psychopathology and education of the brain-injured child (Vol. 1). New York: Grune & Stratton.

Swanson, J. M., McBurnett, K., Christian, D. L., & Wigal, T. (1995). Stimulant medications and the treatment of children with ADHD. In T. H. Ollendick & R. J. Prinz (Eds.), Advances in clinical child psychiatry (Vol. 17, pp. 265-322). New York: Plenum Press.

Swanson, J. M., Nolan, W., & Pelham, W. (1982). The SNAP rating scale for the diagnosis of the attention deficit disorder. Resources in Education, 17(10), 157.

Tansey, M. A. (1986). A simple and a complex tic (Gilles de la Tourette's syndrome): Their response to EEG sensorimotor rhythm biofeedback training. International Journal of Psychophysiology, 4, 91-97.

Tansey, M. A. (1990). Righting the rhythms of reason: EEG biofeedback training as a therapeutic modality in a clinical office setting. Medical Psychotherapy, 3, 57-68.

Tansey, M. A. (1991). Learning disabilities via EEG biofeedback training in a private practice setting. Australian Journal of Psychology, 43, 147-153.

Tansey, M. A. (1993). Ten-year stability of EEG biofeedback results for a hyperactive boy who failed fourth grade perceptually impaired class. Biofeedback Self Regulation, 18, 33-44.

Tansey, M. A., & Bruner, R. L. (1983). EMG and EEG biofeedback training in the treatment of a 10-year-old hyperactive boy with a developmental reading disorder. Biofeedback Self Regulation, 8, 25-37.

Tyner, F. S., Knott, J. R., & Mayer, W. B., Jr. (1983). Fundamentals of EEG technology (Vol. 1). New York: Raven Press.

Wahler, R. G., House, A. E., & Stanbaugh, E. E. (1975). Ecological assessment of child problem behavior. New York: Pergamon Press.

Walden, E. L., & Thompson, S. A. (1981). A review of some alternative approaches to drug management of hyperactivity in children. Journal of Learning Disability, 14, 213-217.

Weiss, G., Minde, K., Werry, J. S., Douglas, V., & Nemeth, E. (1971). Studies on the hyperactive child VIII. Five-year follow-up. Archives of General Psychiatry, 24, 409-414.

Wender, P. (1971). Minimal brain dysfunction in children. New York: Wiley-Interscience.

Werre, E. F. (1957). The relationships between electroencephalographic and psychological data in normal adults. Leiden: Universitaire Presse Leiden.

Zametkin, A. J., Nordahl, T. E., Gross, M., King, A. C., Semple, W. E., Rumsey, J., Hamburger, S., & Cohen, R. M. (1990). Cerebral glucose metabolism in adults with hyperactivity of childhood onset. New England Journal of Medicine, 323, 1361-1366.

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