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Overcoming the barriers to effective treatment for attention-deficit/hyperactivity disorder: A neuro-educational approach

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Abstract

Despite specific diagnostic criteria, published practice guidelines for assessing patients, and the availability of effective pharmacological treatments for children diagnosed with attention-deficit/hyperactivity disorder (AD/HD), a review of prescription practices in the United States indicates that as few as 25–50% of these patients receive even minimal medical treatment for this condition. Because untreated children with AD/HD are at increased risk for psychoactive substance abuse, criminal behavior, and other social problems as adults, the provision of effective care during childhood is essential. In order to clarify the factors impeding treatment during childhood and develop a targeted intervention to overcome these barriers, two studies involving 1514 families were conducted. Each family included one child diagnosed with AD/HD. Factors associated with treatment failure or non-compliance with medical advice included: dissatisfaction with a diagnostic process limited to brief observation, interview, and review of behavior rating scales; fear of stimulant medication; lack of medication response within the first month; development of side-effects during the first month; lack of understanding of the reasons stimulants were being prescribed for a child, and insufficient clinical response. Based on these findings, an intervention program consisting of a comprehensive evaluation process (that included neuropsychological and neurophysiological tests of attention, and medical screening for other health problems associated with inattention and hyperactivity) and parent education about the medical causes of AD/HD, the biochemical action of medications, the relationship between dietary habits and attention, and the educational rights of children with AD/HD was conducted. Following completion of this three session intervention, 95% of patients complied with medical recommendations, initiated pharmacological treatment, and continued medication for a 2-year follow-up period. Three percent of the patients were diagnosed and treated for other medical conditions.

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1. Introduction

Both the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; [American Psychiatric Association, 1994](#)) and the International Classification of Diseases (ICD-10; [World Health Organization, 1994](#)) describe a syndrome of childhood onset that is characterized by symptoms of impaired attention and/or hyperactivity/impulsivity and is associated with significant functional impairment in social, academic and/or occupational settings.

Although differences are evident in the specific criteria needed to meet the diagnostic requirements for the DSM-IV diagnosis of Attention-deficit/hyperactivity disorder (AD/HD) and the ICD-10 diagnosis of a Hyperkinetic Disorder (HKD), it is clear that a substantial number of individuals throughout the world experience functional problems that are due to impaired attention and lack of behavioral inhibition ([Barkley, 1998](#); [Bird, 2002](#)).

Because of the pervasive nature of this disorder and concerns about “over” and “under” diagnosis of AD/HD ([NIH, 1998](#)), practice guidelines have been developed by organizations such as the American Academy of Child and Adolescent Psychiatry ([AACAP, 1997](#)) and the American Academy of Pediatrics ([AAP, 2000](#)). These professional

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societies recommend an evaluation process based on DSM-IV diagnostic criteria, emphasizing physician evaluation of evidence obtained from parents or caregivers, as well as classroom teachers (or other school personnel) regarding “the core symptoms of AD/HD in various settings, the age of onset, duration of symptoms, and degree of functional impairment” (American Academy of Pediatrics, 2000, p. 1158). These guidelines also stressed the importance of evaluating co-existing conditions.

The importance of early diagnosis and sustained treatment is underscored by research examining the long-term course and adult outcomes of children with AD/HD. As reviewed by Barkley (2002) and Hinshaw (2002), AD/HD is a disorder of childhood origin that endures into adulthood. Without effective treatment, these children are at increased risk to develop oppositional defiant disorder, conduct disorder, and anti-social personality disorders. They are also more likely to abuse alcohol and illegal psychoactive substances, suffer from academic underachievement, vocational underemployment, and are at increased risk for accidental injury.

Because of such adverse outcomes, and studies supporting the efficacy of pharmacological treatments for AD/HD (Swanson, 1993; Greenhill, 2002; Biederman and Spencer, 2002) the AAP and the AACAP, as well as the National Institutes of Health (1998), supported the use of stimulant medications (e.g. methylphenidate, mixed-amphetamine salts, pemoline), based on their review of the existing literature. Certain “psychosocial” or behavioral interventions (e.g. systematic use of reinforcement principles at home and school; parent training) were also cited as effective in the treatment of functional problems associated with AD/HD. Other types of pharmacological agents (e.g. bupropion, imipramine, desipramine, clonidine and guanfacine) were considered possibly effective in treating symptoms of impulsivity and hyperactivity in these patients (American Academy of Child and Adolescent Psychiatry, 1997).

However, despite the existence of practice guidelines for the assessment and treatment of AD/HD, a review of data from clinical practice suggests that a substantial percentage of children and adolescents who have been diagnosed with AD/HD either do not begin medical treatment or fail to continue treatment for their core symptoms. Wolraich et al. (1996) reported that only 25% of all elementary, school-aged children who met DSM-IV criteria for AD/HD in one Tennessee county had received medication. Jensen et al. (1999a) examined prescription practices in four United States communities and found that only 12% of children aged 9–17, who met DSM-IV criteria for AD/HD, were treated with stimulants during their 1 year study. At the national level, Jensen et al. (1999b) examined the total number of prescriptions written for stimulant medications in 1995 and concluded that only 25–50% of children with AD/HD received pharmacological treatment. Among those children, Jensen (2000) estimated that each child received

only four to eight prescriptions per year. Finally, even in a study involving participants who were carefully diagnosed and highly motivated (MTA Cooperative Group, 1999), only two-thirds of the patients were treated with any medication during the 14-month study. The average number of months in which medications were actually used by the patients in the MTA Cooperative Group Study was only eight (i.e. approximately 50% of the duration of the study).

As noted by Jensen (2000), “although research findings show that AD/HD can indeed be rigorously and reliably diagnosed under optimal conditions, and that carefully delivered treatments can yield substantial benefits, such best practices do not appear to be taking place in the real world” (p. 102). Studies directly examining the barriers to effective care have revealed patient, caregiver, and health-care network factors. Costello et al. (1996) and Angold et al. (2000) reported that children were resistant to treatment due to lack of information about AD/HD and medication, fear of being placed out of their home (or losing contact with their parents), fear of a negative response to treatment, or fear/dislike/distrust of professionals. Parents were concerned about the cost of treatment, lacked information about AD/HD, had previous negative experiences with professionals, did not have time to pursue treatment or were unable to locate a qualified provider. Primary care physicians cited lack of pediatric specialists in psychiatry, issues of insurance reimbursement, and burdensome paperwork as impediments. Other researchers have cited media exaggerations or misunderstandings about the overdiagnosis of AD/HD and overuse of stimulants (Jensen, 2000) and the lack of availability of stimulants in various geographic regions (Hoagwood et al., 2000) as impediments.

Because of the evidence of inconsistent and insufficient levels of care for children and adolescents with AD/HD, studies clarifying the nature of impediments to treatment and exploring methods for overcoming these barriers seemed necessary in order to promote the health and well-being of these patients. The purpose of this paper is to describe the results of two studies conducted in an outpatient clinic that specializes in the assessment and treatment of patients with AD/HD. The first study utilized a semi-structured, clinical interview format and a questionnaire to identify the factors that precluded parents from initiating/maintaining treatment for their child with AD/HD. The second study examined the effects of a neuroeducational intervention on initiation and retention in treatment over a 2-year period.

2. Study 1: identification of the barriers to effective care

2.1. Method

2.1.1. Participants

Eight hundred fifty-six families that included a child who had been diagnosed by their primary care physician with

Table 1
Distribution of participants by age, sex and diagnosis

Diagnosis	Sex	Age Group (years)			Total
		6–11	12–15	16–20	
AD/HDin	M	97	70	35	202
AD/HDin	F	25	24	13	62
AD/HDcom	M	235	159	90	484
AD/HDcom	F	55	42	11	108
Total	M	332	229	125	686
Total	F	80	66	24	170
Total participants					856

AD/HDin=Attention-deficit/hyperactivity disorder, predominately inattentive type; AD/HDcom=Attention-deficit/hyperactivity disorder, combined type; M=Male; F=Female.

AD/HD (based on DSM-IV criteria) participated in this study. Inclusion criteria were as follows:

- (a) the patient was between 6 and 20 years of age, had been evaluated by a board certified physician and determined not to have any other medical condition that could cause symptoms of inattention, hyperactivity, and impulsivity (e.g. anemia, allergies, hypoglycemia, diabetes, thyroid disorder, traumatic brain injury, seizure disorder, deficiencies of zinc, magnesium, and Vitamin-B, visual or hearing impairment, and use of illegal psychoactive substances);
- b) the patient was of at least average intelligence;
- c) following diagnosis with AD/HD by the child’s physician, the patient’s parents had either never followed medical advice to begin pharmacological treatment for AD/HD or discontinued such treatment within 3 months.

Parents were interviewed at the FPI Attention Disorders Clinic, a private outpatient psychological clinic located in a region of upstate New York with a population of approximately 500,000 within a 50-mile radius of the clinic. Physicians, teachers, health care providers, and the parents of current and former patients of our clinic referred participants for this study. Informed consent was obtained from each parent/guardian prior to participation in the study.

To confirm the diagnosis of AD/HD, parents (legal guardians) were interviewed by a licensed psychologist using a semi-structured format (AD/HD Clinic Parent Interview; Barkley and Murphy, 1998). Consistent with current practice guidelines, this assessment included direct examination of the child’s school records (e.g. report cards; tests of academic achievement) and completion of behavioral rating scales specific for AD/HD (Home and School Versions of the Attention Deficit Disorders Evaluation Scales, ADDES, McCamey, 1995). Participants who did not meet DSM-IV criteria (based on this evaluation by a licensed psychologist) were excluded from this study.

The study sample of 856 patients with AD/HD and their parents was derived from total of 1000 families that were evaluated during this study. One hundred forty-four patients

(14.4%) were excluded from the study: four percent because of a confounding medical condition, 5% because of disagreement in diagnosis between the physician and psychologist, and 5.4% because of a positive screening for illegal psychoactive substances. All participants were citizens of the United States. Ninety percent of the participants were of European ancestry. Ten percent were of African, Asian, Caribbean, or mixed origin. The distribution of participants by age, sex and diagnosis is presented in Table 1. The sample surveyed in this study was comprised of 416 participants who had never been treated for AD/HD with medication and 440 whose parents discontinued medication treatment within 3 months. Those participants who had been treated with medication received either methylphenidate or amphetamine-based medications.

2.1.2. Materials

Based on the research of Costello et al. (1996) and Angold et al. (2000), as well as informal interviews conducted by our staff during completion of two prior studies (Monastra et al., 1999, 2001), a questionnaire designed to elicit information from parents regarding the barriers to treatment was developed. The specific questions included in this questionnaire are provided in Table 2.

This survey was completed by the parent/guardian of each patient. Responses were tabulated by research assistants and manually entered into the Statistical software program (StatSoft, 1995) for subsequent statistical analysis.

Table 2
Barriers to treatment survey (BTS)

Child’s Name:	_____		Date:	_____
Person Completing Survey:	_____			
Instructions: Which of the following problems or concerns led you to postpone or discontinue use of medication to treat your child’s AD/HD? Circle “True” or “False” to indicate your answer.				
I did not have enough information about AD/HD	True		False	
I did not have enough information about medication benefits	True		False	
I did not have enough information about the risks of medications	True		False	
I was afraid of medication side effects	True		False	
I was afraid of, disliked, or distrusted my child’s doctor(s)	True		False	
I was not willing to give medication without testing of attention	True		False	
I could not afford the cost of treatment	True		False	
I did not have the time to obtain treatment for my child	True		False	
I could not find a doctor to prescribe medication for my child	True		False	
My child was unable to swallow pills	True		False	
My child developed side effects (weight loss, moody, insomnia)	True		False	
My child continued to have problems despite the use of medication	True		False	
Other problems interfered (please list)	_____			

2.1.3. Procedure

In order to clarify the factors contributing to a parental decision to postpone or discontinue medication for their child’s AD/HD, each parent/guardian participated in a 90-min session conducted at our clinic by a licensed psychologist or social worker. During this meeting, the Barriers to Treatment Survey (BTS) and the ADDES were completed by parents/guardians. The results of these questionnaires were reviewed with the parent/guardian. The School Version of the ADDES was mailed to each child’s primary teacher. A stamped envelope, addressed to our clinic was provided to facilitate return of the completed questionnaire.

2.2. Results

2.2.1. Attention deficit disorders evaluation scale

Evaluation of the results of these behavioral rating scales was initially conducted in order to determine if the frequency of AD/HD symptoms differed between the previously treated and untreated patients. The results of the ADDES are presented in Table 3. No significant differences were evident between the previously treated and untreated groups based on statistical analysis of means and standard deviation (Tukey Honest *t*-test). Significant differences were noted between the AD/HD, Predominately Inattentive and AD/HD, Combined groups on both parent and teacher ratings of hyperactivity/impulsivity ($p < 0.05$).

2.2.2. Barriers to treatment survey

The percentage of parents/guardians responding positively to items on the BTS is listed in Table 4. Multiple problems were cited by all of the respondents. Because of the equivalence of symptom severity between the previously

Table 4

Percentage of parents reporting problems on the barriers to treatment survey (BTS)

BTS item	Percentage reporting problems
1. Lack of information about AD/HD	22
2. Lack of information about medication benefits	15
3. Lack of information about medication risks	30
4. Fear of medication side effects	92
5. Fear, dislike, or distrust of doctor(s)	12
6. Discomfort using medication without direct testing of attention	90
7. Cost of treatment	7
8. Lack of time to obtain treatment	2
9. Lack of availability of a physician to prescribe medicine	8
10. Child’s inability to swallow pills	10
11. Development of side effects (e.g. insomnia or loss of appetite)	48
12. Continued problems at home or school despite medication	49
13. Other problems (e.g. loss of job or insurance benefits)	3

treated and untreated children (reported on the ADDES), the responses of parents from both groups were combined for data analysis.

Examination of survey results revealed that fear of medication side effects and a lack of confidence in an assessment process limited to interview, review of patient history, and rating scales were the primary impediments to treatment in both previously treated and untreated patients. In addition, the development of side effects and the persistence of significant functional impairment were cited as a common reason for discontinuation of treatment by those parents whose children had begun pharmacotherapy. Reports of adverse side effects (48% of respondents) and the persistence of significant functional problems (49% of respondents) were noted in the surveys completed by over 90% of parents whose children had begun pharmacological treatment.

Table 3

Means and standard deviations of standard scores derived from the attention deficit disorders evaluation scale (ADDES)

	Prior stimulant treatment				No prior stimulant treatment			
	Diagnosis		Diagnosis		Diagnosis		Diagnosis	
	AD/HDin Mean	AD/HDcom S.D.	AD/HDin Mean	AD/HDcom S.D.	AD/HDin Mean	AD/HDcom S.D.	AD/HDin Mean	AD/HDcom S.D.
Inattention (H)	4.6	2.1	4.2	2.6	4.8	1.7	4.1	2.8
Hyp/Imp (H)	10.4	1.8	4.8	2.4	9.6	2.3	3.9	3.1
Inattention (S)	5.1	2.4	2.9	3.6	4.8	2.7	2.5	2.8
Hyp/Imp (S)	11.2	3.3	2.5	2.9	10.4	1.4	3.0	3.2

AD/HDin=Attention-deficit/hyperactivity disorder, predominately inattentive type; AD/HDcom=Attention-deficit/hyperactivity disorder, combined type; Inattention=the Inattentive Subscale of the ADDES; Hyp/Imp=the Hyperactive/Impulsive Subscale of the ADDES; H=Home Version of the ADDES; S=School Version of the ADDES.

Norms for the ADDES were developed such that a standard score of 10 represents the mean frequency of occurrence for 50% of age peers without AD/HD. The standard deviation of the ADDES is 3.0. Standard scores below 7 are considered atypical.

3. Study 2: overcoming the barriers to effective care

The findings of the initial study indicated that parental fears, lack of information about AD/HD, discomfort with the brevity of the assessment process, and the development of adverse side effects either prevented treatment or resulted in the discontinuation of care. The second study examined the effects of a neuro-educational intervention on treatment retention rates.

3.1. Method

3.1.1. Participants

Six hundred fifty-eight families that included a child who had been diagnosed by their primary care physician with AD/HD (based on DSM-IV criteria) participated in this

study. None of these individuals participated in the initial study. As in the first study, each parent had declined or discontinued medical treatment of their child within 3 months, despite a diagnosis of AD/HD made by a physician. Inclusion criteria for this study were the same as in Study 1. Parents were interviewed at the FPI Attention Disorders Clinic. In addition, their child with AD/HD was also evaluated at our clinic. Physicians, teachers, health care providers, and parents of current and former patients referred participants for this study. Parental consent was obtained from parents/guardians prior to participation in the study. Assent was obtained from each child prior to initiating testing procedures.

As in Study 1, each parent/guardian was initially interviewed by a licensed psychologist to confirm diagnosis of AD/HD. The duration of the session was 90 min. A semi-structured format (ADHD Clinic Parent Interview; Barkley and Murphy, 1998) was utilized and direct examination of the child's school records was conducted. Rating scales specific for AD/HD were provided for completion by parents and teachers (ADDES: Home and School Version).

In order to obtain the study's sample of 658 participating families, a total of 800 families were evaluated. One hundred forty-two participants (18%) were excluded due to the presence of a confounding medical condition (5%), disagreement in diagnosis between physician and psychologist (7%), or positive screening for illegal psychoactive substances (6%). Racial composition of the sample was 90% Caucasian, 10% non-Caucasian. All participants were United States citizens. Distribution of participants by age, sex and diagnosis is presented in Table 5.

3.1.2. Materials

In order to address parental lack of confidence in an assessment process that did not include direct evaluation of attention, a computerized test of attention (Test of Variables of Attention: T.O.V.A., Greenberg, 1994) and a quantitative electroencephalographic (QEEG) test (The QEEG Scanning Process, Monastra et al., 1999) were administered to each child. The decision to include the T.O.V.A. was based on previously reported research (Monastra et al., 2001) which indicated that the T.O.V.A. demonstrated moderate test

sensitivity for AD/HD (72%). In addition, the results of Study 1 revealed a strong parental desire to obtain an "objective" measure of their child's attention.

During completion of the T.O.V.A., children were required to sustain attention while visual or auditory stimuli are presented. Each child was instructed to respond by pressing a microswitch when a target stimulus was presented; not respond when a "non-target" stimulus was presented. The Auditory and Visual Forms of the T.O.V.A. were administered to each child. In the Visual Form of the T.O.V.A., two designs were presented. The target stimulus was a white square with a small, embedded black square located in the upper-middle portion of the white square. The "non-target" stimulus was a white square with the small, embedded black square appearing on the bottom of the white square. On the Auditory Form of the T.O.V.A., two tones serve as the stimuli. Each stimulus was presented for 0.2 seconds. The inter-stimulus interval was 2.0 s. A total of 648 stimuli were presented. Test duration was approximately 20 min.

The decision to include a QEEG measure was derived from research supporting the sensitivity of data derived from QEEG in differentiating patients with AD/HD from healthy peers. At this time, several research teams have reported significant QEEG differences in the degree of cortical activation between patients diagnosed with AD/HD and healthy peers (e.g., Mann et al., 1992; Chabot and Serfontein, 1996; Monastra et al., 1999; Clarke et al., 2001; see Barry et al., 2003 for a review). In these QEEG studies, excessive slow wave (i.e. "theta": 3.5–8.0 Hz) activity and decreased "fast" or "beta" activity (13–21 Hz) have been exhibited over frontal and central, midline regions in patients diagnosed with AD/HD. The sensitivity of these QEEG indicators for AD/HD has ranged from 86% to 94% in previous studies.

Another reason supporting use of a QEEG measure was derived from parental concerns about adverse effects associated with the use of stimulant medication. Prior QEEG research (Chabot et al., 1999; Clarke et al., 2002) has suggested that patients with AD/HD who respond to stimulants tend to demonstrate excessive cortical slowing over frontal and central midline regions, whereas "non-responders" do not exhibit such slowing. Given the high rate of adverse side effects reported by parents who decided to discontinue medical treatment for their children, the results of the QEEG were used to recommend type of medication. Stimulant medication was recommended for those children exhibiting cortical slowing (e.g. sustained release (SR) forms of methylphenidate; extended release (XR) forms of mixed amphetamine salts). Non-stimulant medications (atomoxetine) were recommended for those not displaying cortical slowing.

In the present study, QEEG analysis was performed on EEG recordings obtained with an Autogenics A-620 EEG (Wood Dale, IL). Quantitative analysis of EEG was conducted using software specifically developed for quanti-

Table 5
Distribution of participants by age, sex, and diagnosis

Diagnosis	Sex	Age group (years)			Total
		6–11	12–15	16–20	
AD/HDin	M	102	50	31	183
AD/HDin	F	40	27	20	87
AD/HDcom	M	197	56	41	294
AD/HDcom	F	52	30	12	94
Total	M	299	106	72	477
Total	F	92	57	32	181
Total participants					658

AD/HDin=Attention-deficit/hyperactivity disorder, predominately inattentive type; AD/HDcom=Attention-deficit/hyperactivity disorder, combined type; M=male; F=female.

tative analysis by Autogenics. The system used in this study has been field tested in prior published research with AD/HD patients (Monastra et al., 1999, 2001).

To further address parental concerns, a parent education manual was developed for use in this study (Monastra, 2004). The manual was organized as a series of “lessons” for parents, presenting information about the causes of AD/HD, the rationale for using medication, strategies for reducing medication side effects, tips for improving diet, sleep and exercise habits, and the importance of adequate support and accommodation at school. The manual also presented practical strategies for addressing common behavioral, emotional, and social problems that were likely to persist during the early phases of treatment despite the use of medication.

3.1.3. Procedure

Following an interview with each patient’s parent(s) (to determine appropriateness for the study and obtain informed consent), each child/adolescent participated in three 90-min assessment sessions, conducted by a licensed psychologist trained in the use of the T.O.V.A. and QEEG Scanning Process. During the first session, the Visual Form of the T.O.V.A. was administered, scored and interpreted to the child and his/her parent(s). This process was repeated during the third session, using the Auditory Form of the T.O.V.A.

A simplified QEEG evaluation was conducted during the second session. The procedure was identical to that described by Monastra et al. (1999). In this QEEG Scanning procedure, the vertex (Cz) was located using the International 10–20 System of electrode placement (Andreassi, 1989). Next, the area was cleaned using Omni Prep and isopropyl alcohol. Conductive paste (Ten 20) was then applied to the scalp and to a Grass Gold Disc Electrode with hole (E5GH) and the sensor was attached to the scalp. A similar procedure was used for cleaning and preparing the ear lobes and one pair of Gold Disc Electrodes in Ear Clips (Grass E34D) was attached to each ear lobe. To assure quality of preparation, impedance and offset potentials of the sensors were assessed via an Autogenics Electrode Tester. Impedance readings were below 10 k Ω . Offset potential was below 10 μ V before recordings were obtained.

After sensors were attached and quality of preparation was determined to be adequate for recording, EEG frequency bands were defined on the Autogenics Assessment Software as follows: theta: 4–8 Hz; beta: 13–21 Hz. Sampling rate was set at 256 Hz with the Fourier transformation based on 2.0 s epochs. Subsequently, four 90 s EEG recordings were obtained. During the first 90 s recording, the patient was instructed to focus his or her gaze on a light displayed on a computer monitor. After 90 s, the EEG record was visually inspected in two second intervals (epochs) in order to eliminate periods containing eye blinks/rolls. In addition, an electromyograph (EMG) channel on the A-620 monitored muscular activity and background electrical “noise” and was used to identify periods of

excessive movement artifact and prevent contamination of the EEG analysis. A minimum of 15 low artifact epochs (i.e. no evidence of eye blinks or rolls and overall EMG output below 15 microvolts) was required for completion of this task. The electrophysiological power (picowatts) recorded at 4–8 Hz was divided by power at 13–21 Hz. This “theta/beta power ratio” was manually recorded for later data analysis.

After completing this “Eyes Open Baseline” task, the patient was asked to silently read passages from the Kaufman Test of Educational Achievement (KTEA: Kaufman and Kaufman, 1985). Recordings were obtained for 90 s and reviewed for artifact, as described above. A minimum of 15 low-artifact epochs was required. The theta/beta ratio was again calculated, based on selected epochs.

Next, the patient listened to passages read by the examiner for 90 s. The source of these readings was the KTEA. EEG review was conducted as described for baseline and reading tasks and the theta/beta ratio was calculated.

Finally, a stable drawing surface was placed in front of the patient who was instructed to copy geometric figures from the Benton Visual Retention Test (Benton, 1955). Brain activity was monitored for 90 s and reviewed for artifact. The theta/beta power ratio was calculated, as for prior tasks. An overall “Attention Index” (representing the average of theta/beta ratios across four tasks) was calculated as well.

Following completion of the EEG recordings, results were reviewed with the patient and his or her parent(s). Epochs clearly demonstrating periods of “cortical slowing” (i.e. evidence of repeated bursts of high amplitude, low frequency waves) were discussed and translated into everyday life examples in order to promote a better understanding of what “inattention looks like” on the EEG. These discussions and illustrations were used to help parents and children visualize one of the physical characteristics of AD/HD.

During the third session, all clinical findings were reviewed with the patient and his/her parent or guardian. This included a summary of patient history, the results of rating scales, the T.O.V.A., and the QEEG Scanning Process. Illustrations of the brain and brain cells were used to depict cortical regions involved in AD/HD, cellular structures that appear related to symptom manifestation (e.g. dopamine receptors and reuptake transporters; norepinephrine receptors and reuptake transporters) and how stimulants (and non-stimulants) “work”. Stimulant medications were discussed for use in patients exhibiting cortical slowing on QEEG; non-stimulants, for patients who did not exhibit cortical slowing. A review of the relationship between nutrition, sleep, exercise and attention was also provided.

Following completion of this session, the child’s parents, physician, and the school administrator responsible for developing and implementing a program of academic support received a detailed report that described test results and outlined a comprehensive care plan. Among the

recommendations was a specific request that each child undergo medical testing to “rule out” the following conditions: anemia, allergies, thyroid disorder, hypoglycemia, diabetes, sleep apnea, deficiencies of zinc, magnesium and Vitamin B, as well as visual and hearing impairments. Follow-up sessions at our clinic were conducted 6 weeks, 6 months, 1 year and 2 years following initial assessment in order to monitor patient progress and medication usage.

3.2. Results

Two dependent measures were of primary interest in this study. The first was the percentage of parents who initiated medical treatment for AD/HD after they had completed our assessment and education program. The second dependent measure was the rate of discontinuation of treatment due to medication side effects.

The percentage of parents who initiated and maintained medical treatment for their child was monitored through a series of sessions conducted over a 2-year period. At these sessions, type and dose of medication was determined and side effects were reviewed. For the purpose of statistical analysis, patients were grouped on the basis of their score on the Attention Index. Participants whose average theta/beta power ratio over the four tasks (Attention Index) was 1.5 Standard Deviations greater than the mean for healthy age peers (Monastra et al., 1999) were considered to have cortical slowing (CS Group). Participants with an Attention Index within 1.5 S.D. of healthy peers were not considered to have cortical slowing (NCS Group). A summary table describing medication utilization patterns in the two groups is provided in Table 6. The mean and standard deviation of the Attention Index and the percentage of patients demonstrating impairment on the ADDES and T.O.V.A. are also reported in Table 6.

Inspection of Table 6 revealed that 22% of the CS and 30% of the NCS groups were not being treated pharmacologically for AD/HD at the 6-week follow-up session. The primary reasons for failure to begin treatment were inability to schedule and complete recommended medical evaluation

(85%), residual fears about using medication (22%), and a desire to try alternative treatments for AD/HD (e.g. EEG biofeedback; homeopathic remedies; nutritional supplements: 3%). After 6 months, only 4% of the CS and 2% of the NCS group were not being treated with medication. Two percent of the CS group (1% of the NCS group) had been diagnosed and were being treated for another medical condition (e.g. anemia, hypoglycemia, or allergies). Over the course of this study, 6% of the CS group (5% of the NCS group) had also been treated for AD/HD using EEG biofeedback.

Further examination of Table 6 indicates that after 2 years, 98% of patients in the NCS group continued to be primarily treated with non-stimulant medication alone or in combination with stimulants. Initial daily doses of non-stimulants were as follows: guanfacine (2 mg/day), clonidine (0.1 mg/day), atomoxetine (18 mg/day). Conversely, after 2 years, 96% of patients in the CS group were being primarily treated with stimulants (although approximately one-third of these patients were also using non-stimulant medications).

Initial daily doses of stimulant in both groups were as follows: methylphenidate-SR: 20 mg/day; mixed amphetamine salts XR: 5 mg/day. After 2 years, the median daily dose of stimulant in the CS group was 40 mg of methylphenidate-SR (range 20–60 mg) or 10 mg of mixed amphetamine salts (range 5–40 mg). Among this group, the median, non-stimulant medication daily use after 2 years was as follows: atomoxetine: 60 mg (range 18–100 mg), clonidine: 0.15 mg (range 0.1–0.3 mg) and guanfacine: 4 mg (range 3–6 mg).

At 2-year follow-up, the members of the NCS group continued to be primarily treated with non-stimulants, although stimulants were also being used by nearly one-third of these patients. The median daily doses of non-stimulant medications were as follows: 40 mg of atomoxetine (range 18–60 mg), 0.2 mg of clonidine (range 0.1–0.3 mg) and 4.0 mg of guanfacine (range 3–6 mg). Among those patients being treated with stimulants, the median doses were as follows: methylphenidate-SR (20 mg, range 20–40 mg); mixed amphetamine salts-XR (5 mg, range 5–15 mg).

Because reduction of medication side effects was a secondary goal of this study, patients and their parents/guardians were interviewed at each follow-up session to determine side effects and adjust medication. A summary of side effects is reported in Table 7. Side effects are reported as the percentage of patients experiencing specific side effects in the CS and the NCS groups. The percentages are reported only for those patients who were receiving pharmacological treatment.

Review of side effects profiles reveals an initially higher incidence of all side effects (except nausea and sedation) in the CS group. The members of this group were treated primarily with stimulants. The vast majority of the CS group experienced appetite suppression, weight loss, and insomnia within the first 6 weeks of treatment. In order to treat these

Table 6
Percentage of patients being treated with stimulant and/or non-stimulant medications 6 weeks, 6 months, 1 year, and 2 years following diagnosis

QEEG	Percentage of patients using medication											
	6 weeks			6 months			12 months			24 months		
	NM	SM	NSM	NM	SM	NSM	NM	SM	NSM	NM	SM	NSM
C.S.	22	78	10	4	95	25	4	95	27	4	96	34
N.C.S.	30	25	45	2	52	96	2	42	96	4	30	98

C.S.=Cortical Slowing. The Attention Index on QEEG Scan was >1.5 S.D. above the mean for healthy age peers.
 N.C.S.=No Cortical Slowing. The Attention Index on QEEG Scan was within 1.25 S.D. of the mean for healthy age peers.
 NM=Percentage of patients not being treated with medication; SM=Percentage of patients being treated with a stimulant medication; NSM=Percentage of patients being treated with a non-stimulant medication.

Table 7

Percentage of patients reporting medication side effects 6 weeks, 6 months, 1 year and 2 years following diagnosis

Side Effect	Post diagnosis interval							
	6 weeks		6 months		12 months		24 months	
	Group		Group		Group		Group	
	C.S.	N.C.S.	C.S.	N.C.S.	C.S.	N.C.S.	C.S.	N.C.S.
Appetite Loss	92	15	56	25	18	10	15	8
Weight Loss (>5 lb)	85	8	12	14	3	<1	1	<1
Nausea	12	22	15	8	1	2	1	2
Insomnia	96	6	40	15	15	5	10	3
Increased Irritability	52	17	10	7	9	5	10	4
Increased Anxiety	15	4	6	5	4	6	5	6
Increased Depression	25	12	12	4	3	4	2	4
Sedation	6	36	7	9	3	5	2	4
Vocal/Motor Tics	3	<1	<1	<1	<1	<1	<1	<1

C.S.=Patient exhibited cortical slowing on the Attention Index.

N.C.S.=Patient did not exhibit cortical slowing on the Attention Index.

side effects, improvement of dietary habits and combination with non-stimulant medication was encouraged. Approximately 25% of the CS group members began treatment with non-stimulants in combination with the stimulant and were able to reduce most adverse effects. After 2 years, only 15% of the CS group continued to report/display appetite suppression and only 10% demonstrated irritability or insomnia. All other side effects were present in fewer than 10%.

Members of the NCS group demonstrated fewer side effects throughout treatment. These members were typically treated with non-stimulants although stimulants were used by 25% of these patients. Side effects of primary concern at 6 weeks included nausea and sedation. To reduce sedation, a combination with stimulant medication was recommended at 6 weeks. Examination of side effects profiles for the remainder of the study generally revealed a low rate of side effects (<10%) throughout the remainder of the study.

4. Discussion

Prior research has indicated that the majority of parents of children diagnosed with AD/HD will either not seek or will discontinue treatment within 6 months (Wolraich et al., 1996; Jensen et al., 1999a;b). As reviewed by Barkley (1998) without effective treatment, these patients are at substantially higher risk to develop substance abuse problems, discontinue their education, engage in criminal activities, and contract health problems due to impulsive behavior. Consequently, efforts to understand and overcome barriers to treatment seemed essential from a prevention standpoint.

Studies seeking to clarify these barriers to treatment (e.g. Costello et al., 1996; Angold et al., 2000) suggested that insufficient information about AD/HD and medications and a fear of adverse medication effects were the primary

barriers to effective care from the parents' perspective. In order to further clarify the nature of impediments to care, two studies were conducted in a private outpatient clinic. Because our primary interest was to understand and remediate problems that interfered with treatment, only families that had declined or discontinued treatment within 3 months participated in this study. The primary findings were as follows.

First, consistent with the findings of Costello et al. (1996) and Angold et al. (2000), a fear of medication usage/side effects was a significant impediment to treatment. This fear was expressed by approximately 90% of our parents/guardians, who noted both a discomfort with the use of medication and a distrust of an evaluation process limit to interview, records review, and completion of rating scales. Additionally, our findings indicated that nearly all of the patients who had discontinued medication within 3 months did so because of side effects or the persistence of emotional, behavioral or social problems.

Based on this understanding of the barriers to treatment, a neuro-educational intervention program was developed and implemented. Due to parental distrust of practitioners who prescribed medication for AD/HD without directly measuring attention, our intervention program included neuropsychological tests of attention. Similarly, because parents in our initial study desired information about the physical causes of AD/HD, a QEEG examination was conducted. This procedure provided parents/guardians with an opportunity to view the electrophysiological activity of their child's brain during completion of school-related tasks, thereby illustrating the relationship between cortical slowing, AD/HD, and the use of medications that "stimulate" the brain. Finally, our intervention program provided parents with a ninety minute consultation session to review test findings and learn about the causes and treatments for AD/HD. A detailed report describing test results and treatment recommendations was prepared for each patient. In addition, a manual for parents was also developed (Monastra, 2004). This manual was provided to each parent/guardian to be used as an additional resource to help parents understand and overcome common problems that were likely to arise during the first 3 months following diagnosis.

The impact of this neuro-educational intervention was evident both in the percentage of parents who began pharmacological treatment for their children following evaluation (over 70%) and in the high percentage of patients who were receiving such treatment 2 years after their evaluation (95%). Such a treatment retention rate represents a substantial improvement over rates reported by the MTA Cooperative Group (1999), Jensen (2000), and Wolraich et al. (1996).

A secondary goal of this research program was to reduce medication side effects by matching patient to type of medication on the basis of QEEG findings. In the present study, stimulant medications were recommended

for patients who exhibited cortical slowing over central, midline brain regions; non-stimulants were recommended for those patients who did not exhibit cortical slowing on QEEG examination. The results of this study indicated that patients with cortical slowing who were treated with stimulants had a higher rate of side effects (e.g. appetite suppression, weight loss, insomnia, and irritability) than patients without cortical slowing who were treated with non-stimulants. However, because our research design did not control prescription practices of physicians, the matching of patient to medication type based on QEEG indicators of cortical slowing was confounded. Patients with or without cortical slowing received both types of medication, although patients without cortical slowing were far less likely to continue stimulant medication after 2 years and showed fewer side effects than patients with cortical slowing who were being treated with stimulants.

Overall, the present study served to underscore the importance of addressing parental fears as part of the treatment process. The implementation of an assessment process that incorporated the use of neuropsychological and neurophysiological tests and provided parent education during a three session consultation process substantially improved rate of retention in treatment. By reducing parental fears, over 95% of the participants in this study continued to receive effective treatment for AD/HD 2 years post diagnosis. Based on the results of this study, several research initiatives merit consideration.

First, although the present study indicated certain benefits associated with a “neuro-educational” intervention with children and parents, systematic analysis of the types of functional problems that persisted 2 years after diagnosis was not conducted. Such an evaluation seems essential in order to design problem-focused interventions throughout the developmental course of AD/HD. The foundation of this study was based on an effort to conceptualize and remediate barriers to treatment occurring within the first 3 months after diagnosis. Similar designed studies to assess treatment needs throughout the lifespan of patients with AD/HD appear needed as well.

Secondly, the concept of matching patient to type of pharmacological treatment based on the presence of cortical slowing over frontal and central, midline regions merits further examination. Both Chabot et al. (1999) and Clarke et al. (2002) have provided evidence that cortical slowing appears to be present in patients with AD/HD who are “stimulant responders” and the present study suggests that such matching may reduce adverse side effects and promote retention in treatment. However, controlled studies comparing treatment response and side effect profiles of AD/HD patients who are prescribed stimulants uniformly (on the basis of DSM-IV criteria) or selectively (based on the presence of cortical slowing on QEEG in addition to meeting DSM-IV criteria) are needed in order to develop empirically-based guidelines that can enhance efficacy of pharmacological interventions.

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