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The Effects of Stimulant Therapy, EEG Biofeedback, and Parenting Style on the Primary Symptoms of Attention-Deficit/Hyperactivity Disorder[1]

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One hundred children, ages 6–19, who were diagnosed with attention-deficit/hyperactivity disorder (ADHD), either inattentive or combined types, participated in a study examining the effects of Ritalin, EEG biofeedback, and parenting style on the primary symptoms of ADHD. All of the patients participated in a 1-year, multimodal, outpatient program that included Ritalin, parent counseling, and academic support at school (either a 504 Plan or an IEP). Fifty-one of the participants also received EEG biofeedback therapy. Posttreatment assessments were conducted both with and without stimulant therapy. Significant improvement was noted on the Test of Variables of Attention (TOVA; L. M. Greenberg, 1996) and the Attention Deficit Disorders Evaluation Scale (ADDES; S. B. McCarney, 1995) when participants were tested while using Ritalin. However, only those who had received EEG biofeedback sustained these gains when tested without Ritalin. The results of a Quantitative Electroencephalographic Scanning Process (QEEG-Scan; V. J. Monastra et al., 1999) revealed significant reduction in cortical slowing only in patients who had received EEG biofeedback. Behavioral measures indicated that parenting style exerted a significant moderating effect on the expression of behavioral symptoms at home but not at school.

KEY WORDS: EEG biofeedback; ADHD; Ritalin; parenting style; outcome studies.

Attention Deficit-Hyperactivity Disorder (ADHD) is a psychiatric disorder, characterized by the primary symptoms of inattention and/or impulsivity and hyperactivity, that is evident in approximately 3–5% of school-aged children (American Psychiatric Association, 1994). Although currently defined in terms of behavioral symptoms, there is evidence that the core symptoms of ADHD can be associated with metabolic (Zametkin et al., 1990; Zametkin & Rapoport, 1987), circulatory (Amen, Paldi, & Thisted, 1993), neuroanatomical (Casey et al., 1997; Hynd et al., 1993), and electrophysiological abnormalities (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Chabot & Serfontein, 1996; Mann, Lubar,

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Zimmerman, Milller, & Muenchen, 1992; Monastra et al., 1999; Monastra, Lubar, & Linden, 2001). In addition to the primary characteristics of ADHD, there are multiple secondary symptoms that are frequently noted, including learning disorders, anxiety, depression and other mood disorders, tic disorders, and conduct disorders (Spencer, Biederman, & Wilens, 1999). Estimates of the incidence of these secondary or comorbid symptoms range from 50 to 90% (Barkley, 1998; Spencer et al., 1999).

Because of the significant impairment of academic, social, family, and vocational functioning that is caused by ADHD and these comorbid conditions (summarized by Barkley, 1996; Hinshaw, 1992), considerable scientific effort has been directed at developing effective pharmacological and psychological treatments. As reviewed by Spencer et al. (1996) and Swanson et al. (1993), the vast majority of these studies have indicated that pharmacological treatments can exert a positive effect on the core symptoms of inattention, impulsivity, and hyperactivity. However, as noted by Barkley (1998), approximately 35–45% of patients diagnosed with an "Inattentive" Type of ADHD and 10–30% of those with a "Combined" Type of ADHD fail to respond to medications. In addition, systematic review of the effects of pharmacological treatment on cognition, academic achievement, and social skills (Bennett, Brown, Craver, & Anderson, 1999; Brown & Sawyer, 1988; National Institute of Health, 1998) fails to support a conceptualization that the wide range of clinical problems presented by ADHD patients can be effectively treated by medication alone.

Based on a clinical perspective that many of the functional impairments associated with ADHD are not responsive to medication treatments, researchers have examined the role of various behavioral therapies in the development of academic, social, and attentional abilities. Such studies have examined the effects of "reinforced" instruction in psychosocial skills in school and "camp" settings (e.g., MTA Cooperative Group, 1999; Pelham, Wheeler, & Chronis, 1998), at home via parent training (Anastopoulos, Shelton, DuPaul, & Guevremont, 1993), or through a combination of these approaches (MTA Cooperative Group, 1999). The outcome of these studies suggests that although pharmacological treatments for ADHD are effective in treating core ADHD symptoms, a combination of such treatments with social skills and parent training yielded additional improvements in secondary areas of psychosocial functioning (e.g. learning, behavioral, emotional, social, and family problems). However, there is no evidence that these clinical improvements continue in the absence of sustained, long-term treatment with stimulant medication.

Because of concerns about the risks of long-term treatment with stimulants (Breggin, 1998; Jensen et al., 1999) examination of the effects of "nonpharmacological" treatments for ADHD has been encouraged (Breggin, 1998). Among these treatments, EEG biofeedback, a type of behavioral therapy developed to target the core ADHD symptoms of inattention, impulsivity, and hyperactivity, has "generated considerable interest" (National Institute of Health, 1998).

The initial description of the use of EEG biofeedback in the treatment of ADHD was reported in a pair of case studies (Lubar & Shouse, 1976; Shouse & Lubar, 1979). In their first study, Lubar and Shouse (1976) presented the application of operant conditioning techniques to reinforce specific types of electrophysiological activity for the purpose of treating the core symptoms of ADHD. Similar to other operant conditioning paradigms, this treatment involved providing patients with visual and auditory "feedback" for certain "neuronal behaviors." Based on earlier studies by Sterman and his colleagues (summarized in Sterman, 1996), Lubar and Shouse (1976) hypothesized that reinforcing increased production of electrophysiological activity within either the 12–15 Hz (SMR) or 16–20 Hz (beta)

ranges, while attempting to decrease "slower" cortical activity (4–8 Hz; theta), would result in reduction of impulsivity/hyperactivity and improvement of attention when recordings were obtained over either the sensorimotor region or the central frontal region. Their initial findings were consistent with this hypothsis as reduced hyperactive behavior and improved attention were reported in these early case studies.

Despite the positive clinical outcome of Lubar and Shouse's application of operant conditioning principles to treat ADHD by reinforcing electrophysiological activity within specific frequency bands, there have been few published reports of controlled group studies examining the efficacy of EEG biofeedback. As reviewed by Nash (2000), the majority of reports assess efficacy via analysis of multiple case studies, in which patient performance on certain measures (e.g., intelligence, academic skills, behavioral rating scales, continuous performance tests) is compared pre- and posttreatment with EEG biofeedback. Although these published case studies (e.g., Alhambra, Fowler, & Alhambra, 1995; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Lubar, Swartwood, Swartwood, & Timmermann, 1996; Thompson & Thompson, 1998) have yielded generally positive results, additional controlled clinical research was needed in order to address issues of treatment efficacy.

To date, only two controlled group studies have been published. The first (Linden, Habib, & Radojevic, 1996) utilized a randomized design and compared the effects of 40 sessions of EEG biofeedback (theta suppression/beta enhancement) with a "waiting list" control. A total of 18 patients (aged 5–15) participated in the study. Treatment sessions were conducted over a 6-month period. Medication therapy was not provided for members of either group. Results indicated improvement on a measure of intelligence, and reduced ADHD symptoms on a behavior rating scale in the biofeedback group.

The second "controlled" study was conducted by Rossiter and LaVaque (1995). In their design, 46 participants (aged 8–21) were given the opportunity to select participation in an EEG biofeedback group or a stimulant therapy group (titrated Ritalin). Twenty sessions of biofeedback were provided over a 3-month period. Pre- and posttreatment assessment for both groups consisted of behavioral rating scales and the Test of Variables of Attention (TOVA). Both groups showed significant improvement on dependent measures. There was no significant differences between the Ritalin and the biofeedback groups.

Although these two prior investigations of EEG biofeedback utilized controlled group designs and reported positive response on multiple dependent measures, examination of the methodology revealed several limitations, including small sample size and absence of follow-up data. In addition, although published EEG biofeedback protocols were utilized, examination of electrophysiological variables in response to treatment was not conducted and the designs did not provide a basis for comparing the unique contributions of EEG biofeedback and other "active" (e.g., Ritalin) or "placebo" treatments. Finally, although an attempt was made to limit other types of psychological interventions, it was evident that at least informal "parent counseling" and other nonspecific forms of counseling were provided to some of the participants.

The purpose of the present study was to examine the effects of EEG biofeedback and Ritalin on the primary symptoms of ADHD, as well as, on neuropsychological and electrophysiological measures, while controlling for other commonly provided types of clinical interventions (stimulant therapy, parent counseling, school consultation). Because previous controlled studies of EEG biofeedback had not provided extensive follow-up data, examination of treatment effects was conducted 1 year after initial evaluation. Because Ritalin has been shown to yield only short-term clinical effects (see review by Barkley, 1998) and

there is case study evidence that suggests the effects of EEG biofeedback may be more enduring (Lubar, 1995; Tansey, 1993), the use of a dismantling design was considered appropriate for examining transitory versus sustained clinical effects. As a result, examination of patients, both while being treated with Ritalin and following a medication "wash out" period, seemed required. In addition, because there is evidence that systematic use of reinforcement principles by parents/caretakers can contribute to improved social functioning of children diagnosed with ADHD (Pisterman, McGrath, Firestone, & Goodman, 1988; Pollard, Ward, & Barkley, 1983), evaluation of the effects of parenting style was considered necessary in order to clarify the effects of EEG biofeedback and Ritalin on behavioral characteristics of ADHD.

Our hypotheses were as follows. First, given prior reports suggesting the efficacy of both stimulant therapy and EEG biofeedback, we predicted that participants being treated with Ritalin alone or in combination with EEG biofeedback would show improvements on behavioral and neuropsychological tests of attention and impulse control during posttreatment evaluations conducted while using medication. Second, given the absence of long-term clinical effect of Ritalin, as well as, the lack of measurable change on QEEG indicators of cortical arousal over frontal and central cortical regions following administration of methylphenidate (Lubar et al., 1996), and the case reports of reduced cortical slowing following EEG biofeedback (Thompson & Thompson, 1998), we anticipated that only patients who received EEG biofeedback would demonstrate improvement on QEEG measures. Furthermore, we hypothesized that only patients who had received EEG biofeedback as part of treatment would show sustained improvement on behavioral, neuropsychological, and QEEG measures when tested after a 1-week medication "wash-out" period. Finally, because systematic use of reinforcement strategies by parents/caretakers has been associated with improved social functioning in patients diagnosed with ADHD, we predicted that parenting style would emerge as a moderating variable on behavioral measures, regardless of the inclusion of EEG biofeedback.

METHODS

Participants

One hundred children, ages 6–19 (83 males; 17 females), and their parents participated in this study. Based on parental preference, patients participated in either a Comprehensive Clinical Care (CCC) program, which included medication management, parent counseling, and school consultation, or a CCC plus EEG Biofeedback program (CCC+B). All were diagnosed with ADHD (24: ADHD, inattentive; 76: ADHD, combined) by a licensed clinical psychologist, based on DSM-IV criteria. None had a history of prior treatment for ADHD. As reflected in Table I, the composition of the two groups was comparable with respect to participant age, gender, diagnosis, intelligence, parental education, marital status, and median family income.

All participants were screened by The Family Psychology Institute, 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, schools, and mental health professionals located near the Institute referred the individuals who participated in the study. Individuals who had previously been diagnosed and treated for ADHD or other

Table I. Characteristics of Clinic Samples

	CCC	CCC+B
Gender		
Male/female (*N*)	40/9	43/8
Age (years)		
Mean	10.0	10.0
SD	3.7	3.1
Diagnosis		
ADHD, Inattentive (*N*)	14	10
ADHD, Combined (*N*)	35	41
Intelligence quotient		
Mean	105.9	105.2
SD	8.6	11.2
Parents		
Highest grade		
Mean	16.2	15.5
SD	2.5	2.3
Median income	$50,000–60,000	$50,000–60,000
Marital status		
Married (*N*)	49	50
Separated/divorced (*N*)	0	1

Note. CCC = comprehensive clinical care group; CCC+B = comprehensive clinical care plus biofeedback group.

psychiatric or medical disorders that could affect attentional functions were excluded from this study.

Procedure

Pretreatment Screening

Following physician evaluation of each participant for medical conditions (other than ADHD), which could cause symptoms of inattention and hyperactivity (e.g., anemia, hypoglycemia, thyroid disorders), parents were interviewed by a licensed clinical psychologist using Barkley and Murphy's structured format (Barkley & Murphy, 1998). This parental interview format provides extensive information regarding medical, developmental, academic, and social history, and serves as a foundation for comparing patient clinical history with DSM-IV criteria for ADHD and other psychiatric disorders. In addition, this interview provided a format for examining the behavioral management methods used by parents, and the degree of parental consistency. In order to be accepted for participation in the study, participants were required to meet all DSM-IV criteria for ADHD.

Subsequently, the parents completed the Home Version of the Attention Deficit Disorders Evaluation Scale (ADDES; McCarney, 1995). The ADDES is a behavioral rating scale that provides an indication of the frequency of ADHD symptoms and a basis for comparison with "nonimpaired" age peers. A School Version of the ADDES was completed by the child's teachers. In order to be included in this study, each participant needed to be rated as displaying a significantly greater frequency of ADHD symptoms than same age peers on both the Home and School Version of the ADDES. Specifically, a standard score below 7 was required on the Inattentive and/or Hyperactive/Impulsive subscales of both the Home and School Versions of the ADDES.

Children meeting the behavioral criteria for inclusion in the study were then evaluated with the Test of Variables of Attention (TOVA; Greenberg, 1996). Because of the potential rater bias associated with behavioral rating scales like the ADDES, the use of a computer-administered/scored test of attentional abilities was considered desirable in order to obtain an "objective" measure of attention and capacity for impulse control. Continuous Performance Tests, like the TOVA, provide an assessment of an individual's performance on a task that requires tracking of visual stimuli with differential response/nonresponse to target and nontarget stimuli. In this study, errors of inattention (i.e., failure to respond to a target stimulus) and impulsivity (i.e., response to a nontarget stimuli), as well as, response rate and the consistency of response rate (variability) were obtained.

The TOVA was selected because it has been utilized as one of the outcome measures in previous biofeedback studies (e.g., Lubar et al., 1995; Rossiter & LaVaque, 1995; Thompson & Thompson, 1998) and because it has been shown to demonstrate adequate criterion related validity when compared with physician diagnosis of ADHD (Monastra et al., 2001). In order to be included as a participant in the study, a standard score below 80 was required on at least one of the TOVA subscales (i.e., Omissions, Commissions, Response Rate, or Variability).

Finally, a Quantitative Electroencephalographic (QEEG) Scanning Process (Monastra et al., 1999) was conducted using the Autogenics A-620 Electroencephalograph (Wood Dale, IL) with associated Assessment Software (Wood Dale, IL). Because prior QEEG research (Chabot et al., 1996; Chabot & Serfontein, 1996; Mann et al., 1992; Monastra et al., 1999, 2001) indicated that patients with ADHD typically exhibit excessive "slow wave" activity (4–8 Hz), relative to "fast" EEG activity (13–21 Hz) over central-midline and frontal locations, Monastra et al.'s QEEG assessment was conducted in order to insure that only ADHD patients showing this type of QEEG profile were included in the study (Monastra et al., 1999).

Monastra et al.'s protocol involves a comparison of electrophysiological power recorded at 4–8 Hz ("theta") and 13–21 Hz ("beta") (Monastra et al., 1999). QEEG recordings are obtained from the vertex (Cz) with ear references. In the Monastra et al. (1999) process, a ratio comparing the power recorded within the "theta" and "beta" frequency bands is calculated based on QEEG data obtained during four, 90-s tasks (Baseline, Silent Reading, Listening, and Drawing). The overall average of these "power ratios" is then determined in order to obtain the electrophysiologically-based Attention Index. Participants needed to exhibit an Attention Index that was at least 1.5 *SD* greater than age peers based on the database provided by Monastra et al. (1999) in order to be included in this study.

All evaluations were completed between 9:00 a.m. and 3:00 p.m. None of the children were tested within 48 hr of using any type of medication. Participants were invited to participate in this study provided that the results of clinical interview, behavioral rating scales (both Home and School), the TOVA, and the QEEG Scan were all positive for ADHD. Subtype differentiation was made on the basis of interview and rating scales.

Treatment Phase

Following the pretreatment screening, those participants who met inclusion criteria were interviewed with their parents. A review of the treatments that would be provided to participants in this study was given at that time. These interventions included stimulant

therapy, parent counseling (individual and group), school consultation to establish/monitor a program of academic support, and EEG biofeedback. All participants received stimulant therapy, parent counseling, and school consultation. In addition, EEG biofeedback was offered and included in the treatment program of 51 of the participants. A brief description of each treatment follows.

Stimulant Therapy. As noted previously, all participants in the study were treated with Ritalin. Dosage was titrated as follows. Initially, all participants were prescribed a 5 mg dose, t.i.d. for 1 week. After a week, parents and teachers completed the Side Effects Rating Scale (Barkley & Murphy, 1998) and the child was tested with the TOVA. If the standard scores for all TOVA subtests were within 1.0 *SD* of age peers and IQ, and parent and teacher ratings for adverse side effects indicated that the medication was well-tolerated, no change in dose was made. If at least one of the TOVA subtest scores remained within the "clinical range" (i.e.,>1.5 *SD* below age peers and IQ) dose was increased by 2.5 mg per dose and the child was retested after 1 week. This process continued until all TOVA subtests were within the nonclinical range. The average daily dose of Ritalin (following titration) was 25 mg, t.i.d. (10–10–5) for the CCC group (range: 15–45 mg/day). This was the same as the average dosage administered to members of the CCC+B group (range: 15–45 mg/day).

Parent Counseling. Using the model presented by Anastopoulos, Smith, and Wien (1998) as a foundation, all parents participated in a 10 session parenting class, followed by individual consultation on an "as needed" basis. The program described by Anastopoulos et al. (1998) consists of a series of educational "steps" designed to increase parental understanding of the causes of ADHD, as well as, the role of positive parental attention and systematic use of reinforcement strategies in reducing the functional impairments associated with ADHD. Our parenting class also included presentations on "Problem solving with preteens/teens" (Robin, 1998), "Nutrition," and "The educational rights of children with ADHD." At the conclusion of our parenting classes, each parent had developed and was attempting to implement a program of systematic reinforcement to address either primary or secondary ADHD symptoms. The mean number of clinical contact hours for parenting classes/individual consultations was 27 for the CCC group; 25 for the CCC+B group.

School Consultation. At the conclusion of the pretreatment screening, those participants, who met inclusion criteria, were referred by their parents to the Committee for Special Educational Services in their home school district. Federal regulations under the Individuals with Disabilities Act (IDEA) and the Rehabilitation Act of 1973 specify that individuals diagnosed with ADHD are to be evaluated by their school district in order to determine the presence/degree of learning and functional disabilities. This evaluation is to be completed within 45 days of receipt of a letter requesting such an evaluation by a parent (or other caregiver). Based on this school evaluation, a program of academic remediation and/or accommodation is to be developed and monitored on an ongoing basis.

In accordance with these laws, each of our participants was evaluated by their school districts, and either an individualized educational program (IEP) or a plan of academic support/accommodation ("504 Plan") was developed, implemented, and revised with our assistance. The mean number of on-site school consultations was three for both the CCC and the CCC+B groups (range: 1–7). Weekly "progress" reports, listing any incomplete assignments, upcoming projects and tests, and any behavioral incidents, were also reviewed with the parents in order to insure parental reinforcement of "on-task" behavior at school.

EEG Biofeedback. For those patients whose parents selected EEG biofeedback, individual, weekly "attention training" sessions, lasting 30–40 min, were also provided using the Lubar Protocol (Lubar et al., 1995). In addition to the visual and auditory feedback that was provided by the Autogenics A-620 Neurofeedback System (Wood Dale, IL) each time that the child produced 0.5 s of improved arousal over the frontal cortex, participants were also reinforced for their efforts using a "point system." When the patient accumulated a total of 20 points (representing improved EEG performance on 20 "training" tasks) they could exchange these points for a cash "reward" of $15. Participants typically accumulated a sufficient number of points to earn such a "reward" every three to four sessions. EEG biofeedback sessions were conducted until the patient exhibited a degree of cortical slowing on the QEEG scan that was within 1.0 *SD* of age peers, based on the Monastra et al. (1999) database, and were able to maintain this level of arousal for 40 min in each of three consecutive treatment sessions. All of the participants in the CCC+B group achieved this criterion. The average number of sessions needed to reach this goal was 43 (range: 34–50).

Posttreatment Assessment

One year after the intake evaluation, each patient was reevaluated using the ADDES (Home and School), the TOVA, and the QEEG Scan. The first posttreatment assessment was conducted while the patient was being treated with Ritalin. A second posttreatment assessment was conducted after a 1-week medication "washout" period. During the "washout" period, no stimulant medications were administered.

In addition to evaluating patient progress, parenting style was evaluated at the conclusion of treatment based on interview. Parenting style was rated as "systematic" if parents reported use of time out, removal of privileges, and use of earned privileges "most of the time." Parenting style was rated as "nonsystematic" if the parents failed to report use of a combination of "reward" and "response cost" techniques, "most of the time," or if they reported use of physical punishment, acquiescence to child, or avoidance of the child, "most of the time."

Statistical analysis consisted of ANOVA to examine the main and interactional effects of Ritalin, EEG biofeedback, and parenting style on behavioral, neuropsychological, and QEEG measures. Post hoc analysis of significant main and interactional effects was conducted using Tukey's Honest Significant Difference Test. All statistical analyses were conducted using the Statistica Software Program (StatSoft, 1995). An alpha level of at least .05 was used for all statistical tests.

RESULTS

Pretreatment Assessment

Prior to conducting a statistical analysis of the main and interactional treatment effects of EEG biofeedback and parenting style, analysis of pretreatment scores on behavioral, neuropsychological, and electrophysiological measures was conducted in order to insure that the CCC and CCC+B groups were comparable in terms of initial severity of impairment. The mean pretreatment scores on the ADDES, TOVA, and QEEG Scan are provided in

Table II. Pretreatment Assessment: Without Ritalin

	CCC		CCC+B			
Dependent measure	Mean	*SD*	Mean	*SD*	$F(1, 98)$	p
Attention Deficit Disorders Evaluation Scales: Standard scores						
ADDES: Home						
Inattentive	3.92	2.02	4.22	2.23	0.48	.50
Hyperactive	6.02	3.53	5.09	3.48	1.72	.20
ADDES: School						
Inattentive	4.61	1.22	4.69	1.12	0.11	.70
Hyperactive	5.88	3.79	5.14	3.03	1.17	.30
Test of Variables of Attention (TOVA): Standard scores						
Inattention	77.00	27.08	69.57	27.46	1.86	.18
Impulsivity	74.96	25.96	68.98	24.89	2.07	.23
Response time	85.35	20.39	87.94	18.85	0.44	.51
Variability	64.57	17.33	62.45	18.71	0.34	.56
Quantitative EEG scanning process						
QEEG: Attention Index[a]	5.85	2.30	5.77	1.80	0.04	.85

[a] Attention Index = mean theta/beta power ratio, averaged for four tasks.

Table II. The results of this initial analysis revealed no significant group differences on any of the behavioral, neuropsychological, or QEEG measures.

Posttreatment Assessment

Behavioral Measures

One of the primary goals of this study was to examine whether EEG biofeedback exerted any effect on behavioral, neuropsychological, or electrophysiological measures beyond that associated with stimulant therapy. As a result, parent and teacher observations on the ADDES were obtained on two occasions, 1 year following initial assessment. These observations were first recorded while the participants were still being treated with Ritalin. A second posttreatment assessment was conducted after a 1-week period in which no stimulant therapy was provided. A summary of the mean standard scores derived from the ADDES during these two evaluations is provided in Table III. Standard scores below 7 on the ADDES are considered to be within the impaired range.

Initial inspection of the mean standard scores for inattentive and hyperactive/impulsive behaviors (presented in Table III), reveals a pattern of continued impairment in the CCC group, both at home and school. When tested 1 year after beginning stimulant therapy (Ritalin), no indication of sustained improvement was suggested by group data, regardless of the inclusion of medication or consideration of parenting style. All of the group means remained under 7, an indication of the need for continued intervention.

In contrast, mean standard scores for the group that had received EEG biofeedback (CCC+B) suggested sustained improvement, regardless of the use of Ritalin, when tested 1 year after the initial evaluation. These treatment gains were reported by parents and teachers. Inspection of the data contained in Table III revealed group means above the clinical cut-off score of 7 regardless of the use of Ritalin. In addition, the moderating influence of parenting style was also suggested in Table III, as those patients whose parents systematically employed reinforcement strategies demonstrated improved attention and

Table III. Posttreatment Assessment: Behavioral Measure

	Attention Deficit Disorders Evaluation Scales: Standard scores[a]							
	Home				School			
	Inattentive		Hyperactive		Inattentive		Hyperactive	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
Comprehensive clinical care group								
With Ritalin	4.63	0.95	6.06	3.14	4.96	0.82	5.96	3.44
Without Ritalin	3.10	0.91	4.51	3.79	3.29	1.06	4.53	3.76
Ritalin and systematic parenting	4.67	0.99	5.91	3.27	4.97	0.92	5.76	3.56
Systematic parenting without Ritalin	3.12	0.96	4.45	3.89	3.24	1.20	4.27	3.87
Ritalin without systematic parenting	4.56	0.89	6.38	2.92	4.94	0.57	6.38	3.24
No Ritalin and nonsystematic parenting	3.06	0.85	4.63	3.70	3.38	0.72	5.06	3.60
Comprehensive clinical care plus biofeedback group								
With Ritalin	8.59	1.86	8.65	2.16	9.35	0.72	9.63	1.09
Without Ritalin	8.16	2.10	8.37	2.35	9.53	0.61	9.69	0.84
Ritalin and systematic parenting	9.22	1.36	9.49	1.56	9.38	0.72	9.73	1.10
Systematic parenting without Ritalin	9.19	1.05	9.51	1.39	9.68	0.53	9.84	0.80
Ritalin without systematic parenting	6.93	2.02	6.43	1.99	9.29	0.73	9.36	1.08
No Ritalin and nonsystematic parenting	5.43	1.70	5.36	1.55	9.14	0.66	9.29	0.83

[a] Standard scores below 7 are considered indicative of impaired functioning.

reduced hyperactivity and impulsivity at home. Illustrations of the moderating effects of parenting style are presented in Figs. 1 and 2.

Statistical analysis of the posttreatment data, obtained while participants were being treated with Ritalin, revealed a significant main effect associated with EEG biofeedback. Analysis of variance results indicated that the group whose treatment included EEG biofeedback (CCC+B Group) showed significant greater attention, $F(1, 98) = 177.62$; $p < .001$, and less hyperactive/impulsive behaviors, $F(1, 98) = 23.18$; $p < .001$, at home compared to participants whose treatment did not include EEG biofeedback (CCC group). A similar degree of sustained improvement was also evident in the ratings of teachers, who rated the children in the CCC+B group as more attentive, $F(1, 98) = 821.30$; $p < .001$, and less hyperactive/impulsive, $F(1, 98) = 52.49$; $p < .001$, than those in the CCC group.

Further analysis of the main effect of EEG biofeedback was conducted on data obtained after a 1-week medication "washout." This data is also included in Table III. ANOVA results revealed that sustained improvement was reported by parents and teachers only in the CCC+B group, who continued to display significantly fewer inattentive behaviors at home, $F(1, 98) = 239.54$; $p < .001$, and at school, $F(1, 98) = 1313.13$; $p < .001$, than the members of the CCC group. Similarly, the CCC+B group exhibited signficantly fewer hyperactive and impulsive behaviors than the members of the CCC group both at home, $F(1, 98) = 37.81$; $p < .001$, and at school, $F(1, 98) = 91.02$; $p < .001$.

Another primary goal of this study was to examine the effect of parenting style on the manifestation of inattentive and hyperactive/impulsive behaviors. Particular interest was placed on determining whether participants whose parents/guardians were using a "systematic" type of parenting (i.e., consistent use of rewards and response cost strategies) would exhibit fewer behavioral symptoms than participants whose parents were "nonsystematic" in their parenting style.

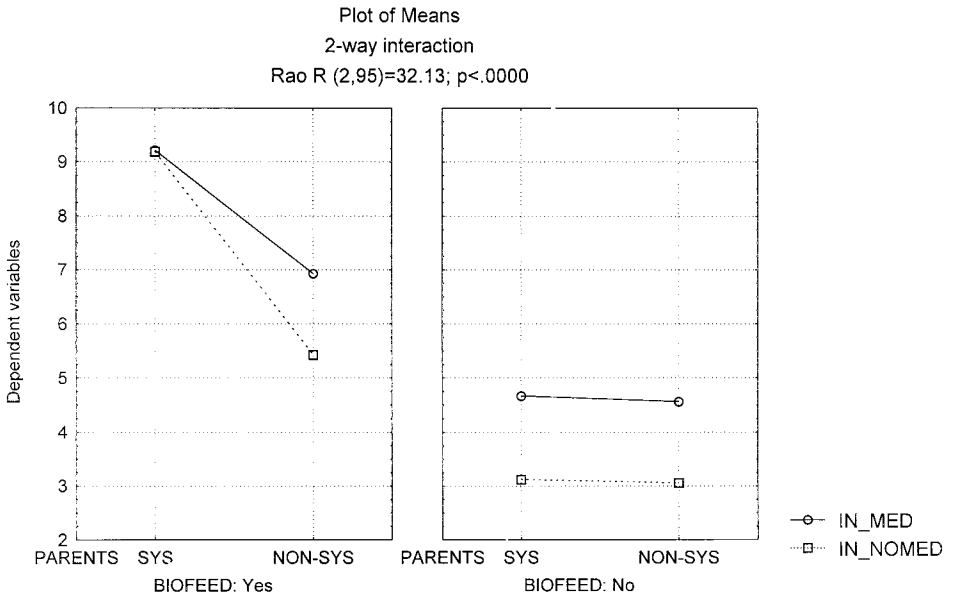

Fig. 1. Plot of the mean standard scores for the Inattentive Scale of the ADDES, 1 year after beginning treatment. Scores of 6 or less indicate continued impairment. Ratings were obtained from parents while their child was being treated with medication (IN_MED) and after a 1-week medication "wash-out" (IN_NOMED). Graph depicts results for two-way interaction, Parenting style (SYS: Systematic vs. NON-SYS: Nonsystematic) × EEG biofeedback (Yes vs. No.)

In order to clarify the contributions of EEG biofeedback and parenting style on behavioral measures obtained from parents and teachers, interactional effects were analyzed. The results of a statistical analysis of parental ratings indicated a significant interaction between parenting style and EEG biofeedback. These interactional effects were evident when participants were rated while being treated with Ritalin [Inattention: $F(1, 96) = 14.73$; $p < .001$; Hyperactive/Impulsive: $F(1, 96) = 10.23$; $p < .001$], and following a 1-week medication washout [Inattentive: $F(1, 96) = 15.45$; $p < .001$; Hyperactive/Impulsive: $F(1, 96) = 5.72$; $p < .02$].

As clarified by post hoc analyses (Tukey HSD), no significant effect of parenting style was noted in the CCC group, regardless of the use of medication. However, in the CCC+B group, participants whose parents consistently used effective reinforcement strategies showed significant reduction in symptoms. This pattern was noted at 1-year follow-up when patients were tested while using Ritalin ($p < .001$), as well as, after a 1-week medication washout ($p < .001$).

No evidence of an interactional effect was evident in statistical analyses of teacher ratings. This finding was noted both when participants were being treated with Ritalin [Inattentive: $F(1, 96) = 0.03$; $p = .86$; Hyperactive/Impulsive: $F(1, 96) = 0.79$; $p = .38$], as well as, after a 1-week medication washout [Inattentive: $F(1, 96) = 0.33$; $p = .56$; Hyperactive/Impulsive: $F(1, 96) = 1.77$; $p = .19$]. Post hoc analyses (Tukey HSD) revealed that significant improvements in attention and behavioral control at school was noted in the CCC+B group, regardless of parental style or use of medication ($p < .001$). In contrast, no statistically significant improvements on behavioral measures were noted in the CCC group, whose treatment did not include EEG biofeedback.

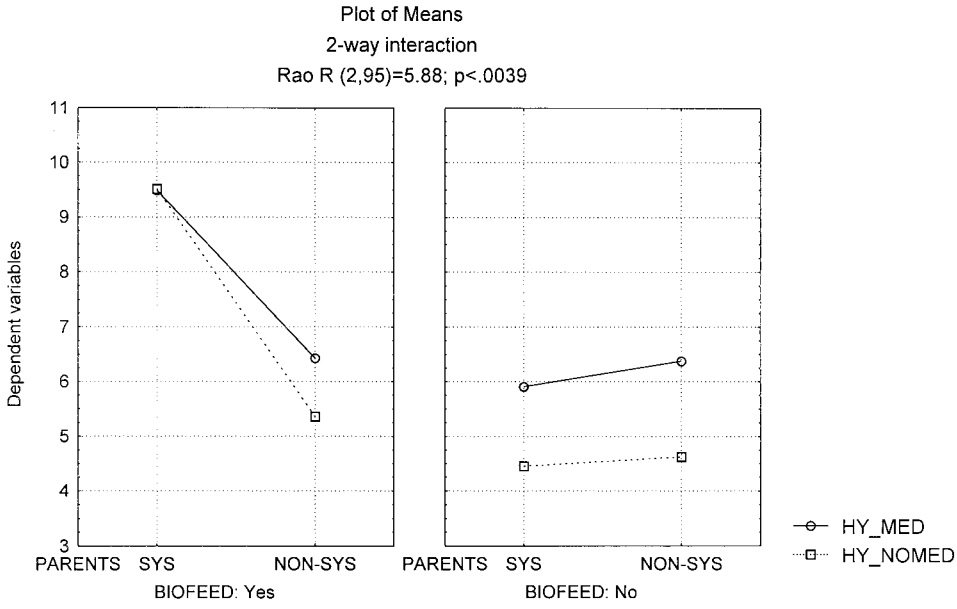

Fig. 2. Plot of the mean standard scores for the Hyperactive/Impulsive Scale of the ADDES, 1 year after beginning treatment. Scores of 6 or less indicate continued impairment. Ratings were obtained from parents while their child was being treatment with medication (HY_MED) and after a 1-week medication "wash-out" (HY_NOMED). Graph depicts results for the two-way interaction, Parenting style (SYS: Systematic vs. NON-SYS: Nonsystematic) × EEG biofeedback (Yes vs. No).

Neuropsychological Measure

In order to assess whether EEG biofeedback contributed to sustained improvement on a computerized test of attention and impulse control, participants in this study were retested with the TOVA, 1 year after their initial evaluation. During this posttreatment period, the TOVA was administered on two occasions, once while being treated with Ritalin and again after a 1-week medication washout period. As described previously, errors of inattention (i.e., failure to respond to a target stimulus) and impulsivity (i.e., response to a nontarget stimuli), as well as, response rate and the consistency of response rate (variability) were obtained in order to assess the sustained effects of EEG biofeedback. Standard scores below 80 on any of the TOVA subscales are considered to be significantly less than anticipated in individuals with average intelligence (such as our sample).

Table IV presents the standard scores and results of ANOVAs for both the CCC and CCC+B groups when participants were tested 1 year after their initial assessment. The upper half of this table reflects the positive effects of stimulant therapy in the CCC and the CCC+B groups. The mean scores for both groups were well within the unimpaired range. Comparison between both groups showed there was no significant difference between the performance of the two groups on any of the four TOVA subscales.

Following a 1-week discontinuation of Ritalin, participants from both groups were reevaluated with the TOVA. The results of this subsequent assessment are summarized in the lower half of Table IV. Significant differences in performance were noted between the CCC and CCC+B groups on all TOVA subscales. The CCC group exhibited an anticipated

Table IV. Posttreatment Assessment: Neuropsychological Measure

	Test of Variables of Attention (TOVA)					
	Comprehensive clinical care		Comprehensive clinical care + biofeedback			
Dependent measure	Mean	*SD*	Mean	*SD*	$F(1, 98)$	p
With Ritalin						
Inattention	102.24	5.89	101.45	7.21	0.36	.55
Impulsivity	103.96	7.60	101.10	10.78	2.34	.13
Response time	100.65	9.16	102.20	10.67	0.59	.44
Variability	98.98	10.53	100.10	9.56	0.31	.58
Without Ritalin						
Inattention	76.24	22.71	98.92	7.65	45.35	<.001
Impulsivity	79.82	23.48	95.16	14.67	15.48	<.001
Response time	88.24	17.05	97.02	8.19	10.89	<.001
Variability	64.04	12.44	94.39	9.49	189.11	<.001

return to baseline level of performance once Ritalin was discontinued. However, the group that had received EEG biofeedback (CCC+B) sustained a level of performance that was well within the unimpaired range. The TOVA scores in the CCC+B group revealed a level of performance that was significantly higher than the CCC group and the CCC+B baseline measures.

QEEG Scanning Process

In order to evaluate whether the effects of EEG biofeedback were simply a placebo effect, inclusion of a "biological" measure was considered essential. Because QEEG studies published by three research teams (Chabot & Serfontein, 1996; Mann et al., 1992; Monastra et al., 1999) had revealed significant differences in the degree of cortical "arousal" measured electrophysiologically over central and frontal locations, the use of QEEG assessment was considered appropriate as a "biological" measure of attention. Monastra et al.'s QEEG Scanning Process (Monastra et al., 1999) was selected for this study due to the availability of a published normative database, as well as, published reliability and cross validation studies (Monastra et al., 2001).

The Monastra et al. (1999) protocol requires quantitative analysis of EEG recordings obtained while participants are involved in the performance of tasks (reading, listening, drawing). An eyes fixed baseline is also obtained. Ninety-second recordings are analyzed for each task, and the electrophysiological power recorded between 4 and 8 Hz is divided by that recorded between 13 and 21 Hz for each condition. An Attention Index is then calculated by averaging the power ratios over the four conditions. The present study utilized this protocol for obtaining and analyzing QEEG data. The results of the Pretreatment Assessment (participants tested without Ritalin) and the Posttreatment results (participants tested without Ritalin) are presented in Table V. Attentional Indices greater than 5.03 (ages 6–11), 3.31 (ages 12–15), and 2.36 (ages 16–20) represent levels of electrophysiological slowing that are 1.5 *SD* greater than age peers without attentional deficits (source: Monastra et al., 1999).

Examination of the mean Attentional Indices for the CCC and the CCC+B groups revealed that both groups showed significantly greater degree of electrophysiological slowing

Table V. Electrophysiological Measure: Without Ritalin

	Attention Index					
	Comprehensive clinical care		Comprehensive clinical care + biofeedback			
Assessment	Mean	*SD*	Mean	*SD*	$F(1, 98)$	p
Pretreatment	5.85	2.30	5.77	1.80	0.04	.85
Posttreatment	5.88	2.22	2.99	0.82	75.48	<.001

than individuals without attentional deficits, hyperactivity, or impulsivity who served as the control group in the Monastra et al. (1999) study. In addition, as noted in Table V, there was no difference in the degree of slowing between the CCC and CCC+B groups during pretreatment assessment, conducted without the use of medication. These findings indicated that prior to initiating EEG biofeedback, the two treatment groups were comparable not only on behavioral and neuropsychological measures, but on a biological measure as well.

The results of posttreatment QEEG assessment, conducted without use of Ritalin, are summarized in Table V. Two primary findings are illustrated in this table. First, the CCC+B group exhibited significantly less cortical slowing than the CCC group. Second, the average Attention Index for the CCC+B group was consistent with the mean for the 6–11-year-old control group in the Monastra et al. (1999) study (the average age of participants in the CCC and CCC+B groups was 10). In contrast, the mean Attention Index for the CCC group remained approximately 1.5 *SD* greater than the mean for the control group in the Monastra et al. normative study. Consequently, the results of the present study indicated that the effects of EEG biofeedback included evidence of "normalization" on an electrophysiological measure. The improvement of cortical arousal was sustained in the absence of stimulant therapy.

DISCUSSION

The results of this study illustrated certain of the differential treatment effects of Ritalin, EEG biofeedback, and parenting style in patients diagnosed with ADHD. Consistent with prior research, our findings demonstrated certain short-term, beneficial effects of stimulant therapy. However, the use of a dismantling design (which incorporated assessment of treatment efficacy independent of stimulant use), permitted clarification of the clinical gains associated with parenting style and EEG biofeedback as well. Overall, the findings of this study are supportive of multimodal treatment models that include parent counseling and EEG biofeedback, in addition to stimulant therapy.

Our examination of the effects of a year-long treatment with a titrated dose of Ritalin revealed that stimulant medication yielded significant improvement on a neuropsychological test of attention and impulse control, independent of parenting style and EEG biofeedback. However, the effect of Ritalin on parent and teacher ratings of inattention, hyperactivity, and impulsivity was not robust, when measured 1 year after the initiation of stimulant therapy. Similarly, the use of Ritalin was not associated with an increase in the degree of electrophysiological arousal on the QEEG Scan.

Given the lack of any indication of improvement on the "biological" measure, rapid deterioration of performance was anticipated once Ritalin was discontinued. The results of this

study were consistent with this hypothesis. Despite a year-long pharmacological treatment, the neuropsychological effects of Ritalin were eliminated when patients were tested without medication at 1-year follow-up. These findings are consistent with the extensive research literature examining the effects of stimulant therapy for ADHD patients (summarized by Barkley, 1998), as well as, QEEG research (e.g., Lubar et al., 1996) and PET studies (Ernst et al., 1994; Matochik et al., 1994), which fail to demonstrate neurophysiological effects of Ritalin at the cortical level. In essence, stimulant therapy would appear to constitute a type of prophylactic intervention, reducing or preventing the expression of symptoms without causing an enduring change in the underlying neuropathy of ADHD.

However, in order to determine whether EEG biofeedback provided any benefits beyond those associated with stimulant therapy, examination of the effects of this treatment was conducted both with and without Ritalin use. Consistent with the results of prior published controlled studies examining EEG biofeedback (Linden et al., 1996; Rossiter & LaVaque, 1995), participants in our study who received EEG biofeedback showed significant improvement on behavioral, and neuropsychological measures. In addition, as was anticipated based on the case studies reported by Lubar et al. (1995) and Thompson and Thompson (1998), participants in the present study who received EEG biofeedback as part of their treatment program exhibited increased cortical arousal on the QEEG. In contrast with the CCC group, the behavioral, neuropsychological, and electrophysiological improvements in the CCC+B group were maintained following a medication "washout," suggesting that the use of EEG biofeedback impacted on the underlying neuropathy of ADHD.

Parenting style exerted a significant effect on the expression of inattentive, hyperactive, and impulsive behaviors. Our initial data analysis indicated that participants whose parents were systematically using reinforcement principles demonstrated a reduction in the frequency of core ADHD symptoms. However, subsequent analysis of the interaction between EEG biofeedback and parenting style revealed that the moderating effects of parenting were noted primarily in our CCC+B group. Patients treated with Ritalin alone or in combination with EEG biofeedback displayed no significant improvement on ratings of behavior at home if the parents were "nonsystematic" in their parenting style. When "systematic" approaches to parenting were used with members of the CCC+B group, improved behavioral ratings were maintained at home, even when medication was discontinued.

Teacher reports did not reflect an association between parenting style and manifestations of ADHD symptoms at school. The sustained improvement in the CCC+B group was evident regardless of parenting style. However, this was anticipated because one of the interventions provided to participants in both groups was the establishment and monitoring of an individualized education plan (IEP) or accommodation plan to facilitate functioning at school. Such programs routinely include the systematic use of reinforcement in the classroom.

The absence of significant improvement of behavioral symptoms in the members of the CCC group when rated 1 year after initiating stimulant medication was unexpected, given reports of the short-term efficacy of stimulant therapy (Swanson et al., 1993). The ratings of both parents and teachers on the ADDES indicated that numerous characteristics of ADHD continued to be observed "at least one to several times per day" in the members of our CCC group 1 year after a titrated dose of Ritalin was initiated. Although interviews with parents and teachers revealed an impression that the use of Ritalin was beneficial, the results of the ADDES revealed a persistence of ADHD symptoms in the CCC group.

Because our study used a stimulant with a 3–4-hr range of clinical efficacy (typically administered t.i.d.), it seems plausible that our findings reflect the behavioral rebound effects that commonly occur with short-acting stimulants. In addition, our findings may reflect a decline in the efficacy of a titrated dose of Ritalin within 1 year of initiation, supporting the necessity for reevaluation of medication dosage within that time frame. Finally, it may be the case that our use of a rating scale (the ADDES) that requires observers to provide a numerical estimate of the incidence of ADHD behaviors (e.g., one or several times per month, week, day, or hour) rather than more nonspecific ratings (e.g., "most of the time," "often," "some of the time," "a little," "rarely") permitted identification of ongoing ADHD symptoms that are not revealed by other questionnaires. Overall, our finding of a lack of sustained response to short-acting stimulants such as Ritalin seems consistent with recent efforts to develop stimulants with more enduring clinical effects (e.g., Adderall and Concerta).

Overall, the results of this study are consistent with an emerging neurological model of ADHD. Although ADHD is considered to be a psychiatric disorder, diagnosed on the basis of behavioral symptoms, our findings support the hypothesis that there are neurophysiological factors that contribute to the maintenance of this disorder. Specifically, the design of this study permitted demonstration of symptom relapse in those ADHD patients who continued to exhibit electrophysiological slowing, despite year-long treatment with Ritalin. Essentially, those patients who exhibited no increase in the degree of cortical arousal showed no sustained improvement when medication was discontinued. In contrast, no such relapse was noted in those patient's who were able to reduce the degree of electrophysiological slowing. In those participants, reduction in the degree of cortical slowing was associated with maintenance of clinical gains when medication was withdrawn.

Consequently, our findings of the short-term effects of Ritalin, the sustained behavioral, neuropsychological, and electrophysiological, improvements with the addition of EEG biofeedback, and the moderating role of parenting style support the examination of a multimodal treatment model in clinical research studies with ADHD patients. However, in contrast with the approach utilized by the MTA Cooperative Group (1999), our research supports the systematic examination of treatments designed to promote sustained neurophysiological and/or neurochemical change, in addition to the prophylactic use of stimulant medications like Ritalin and the inclusion of reinforcement-based parent counseling and social-skills training. At present, the only type of behavior therapy that has been associated with sustained improvement of core ADHD symptoms in the absence of stimulant therapy has been EEG biofeedback. Therefore, systematic examination of this type of behavioral treatment seems warranted.

To date, there has been considerable interest and debate regarding the use of EEG biofeedback (National Institute of Health, 1998). Our findings are consistent with prior studies and expand scientific understanding of the unique and combined effects of this type of therapy. However, although the present findings demonstrate specific effects of this type of treatment, numerous empirical questions remain.

First, although sustained modification in the degree of cortical arousal was noted in this study, the duration of the study precludes conclusions regarding the long-term effects of EEG biofeedback beyond 1 year. In addition, clarification of the underlying neuroanatomy and/or neurochemistry of this change process is needed. For example, if the primary functional deficits of ADHD are due to hypoperfusion of blood flow in frontal cortical regions, as suggested by SPECT studies (Amen et al., 1993), pharmacological and neuropsychological interventions directly targeting this process may prove more efficacious. Consequently,

follow-up studies, examining the long-term effects of EEG biofeedback on the primary symptoms of ADHD, as well as, on underlying neurophysiological processes are needed.

Second, our study does not address issues related to the attention training process. It remains unclear whether the protocol developed by Lubar and his colleagues represents the "optimal" methodology for training ADHD patients or whether other EEG training protocols (e.g., training output within other EEG frequency bands at multiple cortical sites as reported by Kaiser & Othmer, 2000) may prove more efficacious. This question becomes more salient as QEEG studies have reported a neurological "subtype" of ADHD that is not characterized by cortical slowing (Chabot et al., 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001; Monastra et al., 2001). As clarification of neurophysiological subtypes of ADHD emerges, training protocols specifically targeting these subtypes can be developed and evaluated.

Third, systematic evaluation of computer-based "cognitive training" activities (e.g., Captain's Log Cognitive Training System; Sanford, Brown, & Turner, 1996) may result in the identification of training procedures that effect the neurological foundation of ADHD without the need for EEG feedback. Although the Captain's Log and other "training" programs are commercially available, little systematic research has been conducted in this area. Given the costs associated with long-term pharmacological treatment or intensive training programs (e.g., EEG biofeedback; Social Skills Training Programs), examination of the role of computer-based "attention training programs" warrants consideration.

Finally, because the inclusion of EEG biofeedback in a multimodal treatment program for ADHD resulted in improvement in the level of cortical arousal, it is not clear whether the dose of stimulant needed to sustain clinical gains requires adjustment during and following the biofeedback treatment process. The present study does not directly address the question of whether patients will be able to sustain clinical improvements following EEG biofeedback, while eliminating or reducing Ritalin dose. Consequently, systematic, long-term follow-up studies examining the relationship between EEG biofeedback and stimulant dosing patterns also seem required.

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