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Parameters Associated with Rapid Neurotherapeutic Treatment of Common ADD (CADD)

Paul G. Swingle, PhD

ABSTRACT. Although there are many types of ADD/ADHD, a common form of ADD (CADD) in children (high theta/beta ratio at Cz) can be successfully treated in less than 15 sessions. The increased efficacy relative to the standard beta enhance/theta suppress protocol results from precise but brief diagnosis, home cognitive exercises with a theta suppression harmonic, disentraining and/or entraining visual stimulation, and

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clear treatment termination parameters. A single case example and data from 30 patients show the usual course of treatment.

KEYWORDS. EEG, ADD, CADD, children

INTRODUCTION

Several factors contribute to prolonging the neurotherapeutic treatment of attention deficiencies in children. Perhaps the most prevalent is that children arrive with the diagnosis of ADD or ADHD but without any diagnostic precision. The purpose of this paper is to describe the diagnosis, treatment and termination parameters for the rapid neurotherapeutic resolution of a prevalent form of ADD in children, which I call Common ADD (CADD). Only children with CADD as determined by QEEG assessment, described later, are included in this study. Children with all other forms of ADD, ADHD, and other learning difficulties are also excluded. Patients with conditions that mitigate against rapid resolution including those with comorbid conditions, pathogenic family dynamics or reinforced agonistic behavior of the child are excluded from this report. Also excluded from this report are patients who were over the age of 18 at the beginning of treatment. Inevitably, older patients, with unresolved ADD, present with considerable "excess baggage" such as addictions, indifference, non-committal, and assorted psychological defenses to mitigate feelings of self-loathing similar, in some respects, to the personality disordered patient. Clearly, patients with such comorbidities will require extended psychoneurotherapeutic treatment and are not the subject of the present discussion.

The context of the present study is Practice Oriented Research in which unselected clients presenting for psychoneurophysiological treatment of an attention problem are treated in a noncontrolled manner. The treatment of clients is driven solely by the data obtained from the neurotherapy session, parental reports and clinical judgment. Patients included in the analyses are those whose data (i.e., initial EEG assessment, neurotherapy session outcome, patient/parental reports and termination parameters) survive criteria for retention in the study. Therapeutic recommendations are guided by consistency between EEG data and patient/parent reports of behaviors clinically observed to be predictable from specific EEG architecture.

METHODS

The patient population included females between the ages of six and seventeen who had been diagnosed elsewhere as ADD or ADHD. Patients included those on stimulant medication, those who had discontinued stimulant medication and those who had never received medication for the attention deficiencies. The protocol includes a brief EEG assessment, home based cognitive tasks with subthreshold theta suppressing harmonics (Swingle, 1996), standard theta suppress/beta enhance neurofeedback (Lubar, 1991), EEG dependent (Ochs, 1993), and EEG independent (Swingle, 1995) visual stimulation.

Phase One: Diagnosis

Recognizing that the client has presented with complaints of attention deficits, the major focus of diagnostic procedures is not to validate the complaint nor to document EEG deviations from normative values but rather to isolate the neurophysiological specificity of the attention problem. In the case of CADD, questionnaires, continuous performance tests, and rating scales are largely irrelevant because data from these sources do not influence therapeutic decisions although one might use them to track therapeutic progress. Further, long intakes imply long treatment, so only therapeutically relevant data are collected. Parental and patient reports are accepted to identify comorbid or other compromising conditions. The diagnosis of CADD is based almost exclusively on the EEG at five measurement sites: Cz, O1, Fz, F3, and F4. The measurement bands are theta (3-7 Hz), alpha (8-12 Hz), beta (16-25 Hz) and hibeta (28-40 Hz). Assessment epochs are minimally 20 seconds each and include eyes open (EO), eyes closed (EC), reading (R), and harmonic stimulation (HS). The HS is subthreshold alpha frequency sound, which has been shown to suppress theta band amplitude (Swingle, 1996). It is used in the treatment of CADD if the assessment EEG confirms theta suppression. The assessment protocol is as follows: At Cz: EO, EO, EC, EO, R, R, EO, HS, EO; at O1: EO, EO, EC, EO; at F4, F3 and Fz: all EC. Data reported in this study are peak-to-peak amplitudes obtained with ear references and ground.

CADD has the following profile: Parental and client reports reveal no mitigating conditions. The assessment at Cz shows theta/beta ratios in excess of 2.2 with alpha enhancement during eyes closed of at least 20%. Reading EEG usually shows an increase of theta amplitude; however, lack of theta amplitude increase under cognitive challenge is not

exclusionary. Theta suppression with the HS is at least eight percent. HS suppression of theta that is less than eight percent does not exclude the client from CADD categorization but does exclude the client from the rapid treatment protocol which depends heavily on home treatment using HS, to be described later in this paper. The assessment at O1 shows at least a 40 percent increase of alpha EC/EO and a theta/beta ratio of at least two. The assessment at the F3 and F4 positions is to exclude depression and high frontal alpha forms of ADD/ADHD. The ratio of beta F4/F3 < 1.3 and the alpha/beta ratio at both sites is less than 2.5. The hibeta/beta ratio at Fz should be between .45 and .55. Values above .60 and below .40 exclude the client from the CADD category and, it is hypothesized, reflect hyper and hypo activity of the cingulate gyrus reflected behaviorally, in excesses in inflexibility/compulsivity and passivity/ductility, which would require treatment beyond that required for CADD. Finally, the total of the theta, alpha and beta band amplitudes at location Cz is less than 60.0. Total amplitude above 60.0 usually implies more severe cognitive deficits than those associated with CADD. In summary, for a CADD child to be included in this sample of patients, the EEG assessment must be characterized by: a theta/beta ratio > 2.2 at Cz, alpha enhancement of > 20% (EC/EO), theta suppression with HS > 8%; O1 theta/beta > 2, alpha enhancement > 40% (EC/EO); F4/F3 ratio of beta < 1.3, alpha/beta at both sites < 2.5; hibeta/beta ratio at Fz between .40 and .60. Although not the subject of this report, the author suggests the following clinical probes based on the EEG assessment: if alpha enhancements with eyes closed do not meet criteria values probe for abuse and other forms of traumatic stress; if O1 theta/beta is below 2 probe family addiction history; if F4/F3 beta ratio is greater than 1.3 probe for clinical depression and perhaps reassess using Cz as referential site for F3 and F4. If Fz hibeta/beta ratio exceeds .60, probe for compulsive/inflexible/perseverating behavior/thoughts. If the ratio is below .40, probe for excessive passivity/ductility.

Phase Two: Treatment

In Office

The core of the in office treatment protocol for CADD is theta suppress/beta enhance (T/B) at position Cz (Lubar, 1991). The factors that make this treatment protocol so much more effective than the 40 to 80 sessions previously required (Lubar, 1991) are precise diagnosis, short

intake session, and the use of theta suppressing harmonics, visual stimulation and precise termination criteria. Following the diagnostic EEG evaluation, the patient is exposed to EEG disentrainment (Ochs, 1993). This procedure involves measuring the Dominant Frequency (DF), averaged over one-second periods, at Cz and based on that frequency stimulating with light using standard LED goggles used in audiovisual stimulation (AVS) devices at five percent ahead and five percent behind the DF alternatively for four minutes each repeated twice. Following this 16-minute sequence the stimulation is changed to one percent ahead and one percent behind the DF alternating every minute for eight minutes. If a disentrainment device is not available an AVS device may be used. The stimulation should be either 10 Hz and 18 Hz alternating every two minutes (Russell & Carter, 1993) or 10 Hz to 18 Hz in 1 Hz intervals alternating randomly every two to five seconds (Swingle, 1995) for 24 minutes. The preferred stimulation program is the latter (Swingle, 1995) because fixed stimulation has been found to have different effects on clients with high versus low prestimulation alpha and beta amplitudes (Rosenfeld, Reinhart, & Srivastava, 1997). After one treatment of either disentrainment, 10 and 18 Hz AVS, or random 10 to 18 Hz, standard T/B neurofeedback at position Cz is administered. Each session of neurofeedback includes 10 to 15 minutes of T/B (starting with several two and one-half to five-minute sessions and working up to one 15-minute session), then five minutes of the 10 to 18 Hz random AVS while monitoring theta and beta. The session is completed with a final 10 to 15 minutes of standard T/B training.

It should be noted that AVS and EDF do present some minor risk of seizure. Harding and Jeavons (1994) report that for youth under the age of 19 the frequency of Photosensitive Epilepsy (PSE) is 5.7 per 100,000 of the population. Overall the frequency is reported to be 1.1 per 100,000. For epileptics, those who have PSE are about two percent for persons of all ages and about 10 percent for those under 19 years. All clients receive an information document describing the various forms of stimulation, including visual, that are used in the clinic and permission is obtained prior to exposure to any form of stimulation.

At Home

Two features of this protocol that are crucial to the markedly superior effectiveness are monitoring of the attention symptoms and systematic and diligent use of the theta-suppressing harmonic. At intake both the parent(s) and the client are instructed to start a daily recording system to

monitor the problem(s) for which treatment is being administered. Behaviors monitored usually include alertness while doing homework, teacher reprimands at school, time to complete homework, alertness at school, and of course, teacher reports, test results and spontaneous comments of teachers, special education instructors and coaches. This daily rating is an important behavioral intervention.

The second and critical treatment element is the use of the theta suppressing subthreshold alpha range harmonic. The harmonic is a blend of a 300 Hz and a 310 Hz tone imbedded in pink noise filtered with 50-decibel roll off at 1000 Hz. The two tones are imbedded at -15 and -25 decibels. Research described in Swingle (1996) found that the harmonic suppressed theta amplitude for adults and children significantly more than filtered pink noise alone (the control condition). Procedures for preparing these treatment tapes may be found in Swingle (1992). The client is instructed to have the harmonic play softly in the background while doing homework or other cognitive activity such as reading. A minimum of 15 minutes exposure to the harmonic, each day, while engaged in a cognitive task is required.

Phase Three: Termination of Treatment

The treatment frequency is once per week for about eight sessions and then once every second week for about four sessions. Termination is expected between 12 and 15 sessions. Follow-up sessions are scheduled for six weeks, four months and ten months post termination to confirm that changes are stable. Termination parameters are:

1. Parental and client reports of satisfactory attention skills.
2. Best epoch theta/beta ratio not significantly different from session average theta/beta ratio. The epoch unfortunately is defined very differently in the software of the various equipment manufacturers. However most software programs can provide an epoch estimate. The criteria presented in the present paper were based on the epoch being 10 percent of the total session. Thus, if the session were 10 minutes in length, the epoch would be 60 seconds. The comparison of best epoch versus total session reflects the child's ability to sustain his or her best effort over a prolonged period of time (e.g., the theta/beta ratio of an entire 15 minute session is not significantly different from the best 90 second epoch). This is, of course, exactly what we are attempting to teach the child to do in school: to sustain attention.

3. No significant difference between the theta/beta ratios of neurofeedback sessions and visual stimulation sessions. This indicates that the child can duplicate the theta/beta ratio they can produce with stimulation.

RESULTS

To illustrate the protocol, a single case will be presented in addition to group data for 30 Common ADD clients. The client, a 17-year-old female diagnosed with ADD (and “possible Asperger’s”), was previously treated with Cylert. The parent had discontinued Cylert seven months prior to neurotherapy because no benefits were observed. The client and parent reported that the client could not concentrate, had great difficulty reading and retaining information, was often reprimanded in school for inattentiveness, experienced a poor friendship network, hyperfocused on computer games, but had no discipline problems in school. EEG evaluation: At Cz, theta/beta ratio 2.71; theta increase when reading 12 percent; theta suppression with harmonic 11.6 percent; EC alpha increase 34.4 percent. At O1, theta/beta ratio 2.14; EC alpha increase 42.2 percent. At Fz, theta/beta ratio 2.89; alpha/beta ratio 2.0; F4/F3 beta ratio 1.04. Termination statistics: M theta/beta = 2.49 (SD = .14); M (AVS) theta/beta = 2.54; M (best epoch) theta/beta = 2.32 (SD = .18). The means are pretreatment baseline obtained at the final session. All comparisons were nonsignificant (all *t* values < 1.0). Parent and client report dramatically improved ability to concentrate, greater interest in academic activities, applied to university (she was subsequently accepted), markedly increased friendship network (mother’s comment: “. . . she had no real friends . . . now the phone never stops ringing”). The client had 14 sessions, including the initial evaluation session. Follow-up sessions at six weeks, four months and 10 months indicated that the EEG changes were stable. Parental and client reports indicated continuing improvement in school, general motivation, and in social contexts.

The following data are from 30 recent consecutive clients. All satisfied the criteria for CADD at intake. M intake theta/beta at Cz = 2.98, SD = .67; M theta suppression with harmonic = 17.2%, SD = 9.8; M termination theta/beta = 2.19, SD = .42; M (AVS) theta/beta = 2.24, SD = .48; M best epoch theta/beta = 2.03, SD = .42; M number of sessions = 12.51, SD = 2.86 (excluding follow-up sessions). Of the 30 clients, one client re-

turned for further treatment. The longest follow-up report to date is three years, five months, so it appears that the effects are permanent.

DISCUSSION

The results indicate that marked and stable improvement can be accomplished in the treatment of children with CADD in significantly fewer sessions than conventionally prescribed. This results, in my opinion, from more precise diagnosis of the exact locations of central nervous system inefficiencies, abandonment of lengthy intake protocols and non-EEG diagnostic testing, more dynamic and interactive neurotherapeutic sessions, and far greater emphasis on treatment administered outside of the context of the neurotherapist's office. The intake diagnostic session, including the five-site EEG analysis, can be accomplished in less than thirty minutes allowing time to briefly expose the child to AVS stimulation and instruction on the home trainer using the alertness-inducing harmonic. In short, treatment starts immediately and the child has something she or he can do immediately that they *know* can help because they have seen the effects on the EEG. The intake EEG diagnostic indicates when it is appropriate to use the theta suppression harmonic. First, does it reduce theta? If so, it is given for home use. If not, then it is not given for home use. Additionally, since the harmonic has stimulating effects, children with high *hibeta*/beta ratios *do not* receive the harmonic for home use until the amplitude of the high frequency activity is reduced.

An examination of the effects of the harmonic on midline sites was determined on 11 male and female patients who were scheduled for full 19 site QEEG for another purpose. The data indicate that the stimulating effect of the theta-suppressing harmonic extends along the entire midline (i.e., Fz, Cz, and Pz). The percent theta suppression of 30 seconds of harmonic exposure is 26.0 at Fz (SD = 15.9), 22.0 at Cz (SD = 13.5) and 22.7 at Pz (SD = 18.0). All means are significant ($p < .01$, 2t) but not significantly different from each other. Because the harmonic suppresses theta activity at Pz and Fz as well as Cz, the harmonic is usually not prescribed for use with autistic clients who show high amplitude beta activity over the frontal midline nor for clients who show a deficiency of theta activity in the occipital region.

The effects of the theta-suppressing harmonic were tested on 119 consecutive new patients with a wide range of diagnoses. The test was administered during the intake EEG assessment, similar to that used in

the CADD assessment described above. The mean theta suppression was 21.4 percent (suppression of theta relative to the immediately preceding 15 second epoch), $SD = 13.4$, $t = 17.8$, $p < .001$. To determine if theta suppression is related to the average theta/beta ratio, the data were submitted to product moment correlation. The correlation was $-.29$ ($p < .05$, 2t) indicating a very weak negative correlation but substantiating that theta suppression of the harmonic is not a positive function of the theta/beta ratio of the client, eyes open, unchallenged. The suppression data were also correlated with patient's age, revealing that age is positively correlated with magnitude of theta suppression ($r = .52$, $df = 66$, $p < .01$). The patient population was reduced to only those reporting a problem with attention that also satisfied the EEG criteria for CADD. This population was further divided into those who were under 18 years of age and those 18 and older. The average theta suppression for the older males was 31.1 percent ($SD = 16.0$) and for the younger males the average was 15.6 percent ($SD = 8.6$) ($t = 3.54$, $48df$, $p < .01$). The average theta suppression for older females was 28.5 percent ($SD = 12.6$) and for the younger females the average was 18.5 percent ($SD = 8.8$) ($t = 2.13$, $df = 18$, $p < .05$). Thus, although the theta-suppressing harmonic is effective for both groups and both genders, it appears as though the harmonic, at least initially, has greater effect on older patients who satisfy the criteria for CADD.

A second study of the effectiveness of the home use of the harmonic was conducted by Francois Dupont (1998) in my laboratories at Ottawa University. This study involved 11 children diagnosed elsewhere as having attention difficulties who used the harmonic daily, while doing homework or reading, for seven weeks. Each week the children were brought into the laboratory and the theta/beta ratio was determined at Cz. None of the children were receiving neurofeedback training. The results indicated a steady decline in the theta/beta ratio over five weeks of home use of the harmonic. At the end of the five-week period the theta/beta ratio had declined by 13.3 percent, on average, without any neurofeedback treatment ($t = 2.72$, $p < .05$).

It seems the harmonic home treatment serves three important functions in the treatment of CADD. First, it provides the child with an immediately applicable treatment that provides an experience of increased focus. In short, they are involved in a discrimination type protocol in which they gain experience in what it feels like to have increased focus. Second, it clearly enhances neurotherapy because it reduces the theta/beta ratio consistently with continued use. Finally, it provides a procedure

for sustaining the gains made in neurotherapy after termination of treatment.

It is interesting to note that occasionally we find children with CADD who respond well to treatment yet show no discernable change in the theta/beta ratio. We often encounter children who improve because their theta amplitudes drop, or their beta amplitudes increase or both but we are puzzled when we get glowing reports of improvement without theta/beta changes. There is an inclination to attribute these changes to the placebo effects of neurotherapy but I would propose that, when such anomalies occur, one have a close look at the entire brainwave spectrum because a change in brainwave activity may have occurred that was not apparent. One such case was a child with CADD with a preliminary theta/beta ratio of 2.53. After 20 sessions the theta/beta ratio remained essentially unchanged at 2.46. However, the parents were thrilled with the child's progress both at home and at school. To understand why we obtained such marked improvement in the absence of observable changes in theta, beta or theta/beta ratio, the various bands of the EEG were analyzed. It was then immediately obvious that we had decreased delta activity. The delta amplitude dropped precipitously (35.3 percent) after session 14 and remained stable through session 20.

Whenever a dramatically more effective treatment variation is proposed the most obvious concern is that the effects are primarily associated with patient expectations (placebo effect). There are several issues associated with the premise that effects are placebo. First, all treatments gain potency if the patient's positive expectations can be marshaled. Evans (1985) points out that about 50 percent of the beneficial effects of all interventions, including medications from morphine to aspirin, result from patient expectations (i.e., the placebo effect). Practice oriented research always has the advantage/limitation of all treatments being bona fide with the enthusiasm, intention and expectations of the therapist as well as the client being focused on positive change for the client.

Whenever a more efficacious variant of a traditional treatment is found, one must consider the possibility that the variant is better able to more efficiently marshal the client's expectations for positive change. In one sense, such enhanced expectations or focus is a meaningful contribution to treatment efficacy, given our understanding that such expectations do, in fact, contribute to treatment efficacy. However, when a new and more efficacious variant is thought to be effective only because of placebo effects, and by definition, unstable and transient in terms of beneficial change for the client, one must examine the details

of the procedures to determine if they are substantially different. It is true, I believe, if someone claimed that the traditional theta/beta training in the region of Cz to Pz, bipolar or unipolar, accomplished permanent and beneficial change in CADD clients in 10 to 15 sessions, that one would conclude that the therapist is either terminating the treatment at a propitious peak in client enthusiasm, or is uniquely talented at scamming the client. But, if the variant is, in fact, substantially different from the grandfather procedure, then the former must be judged independently from the latter. The treatment proposed in this paper bears little resemblance to standard theta/beta training and, thus, must be judged on its own merit. Twelve-session standard theta/beta training would, in my judgment, be clearly placebo. Twelve-session Swingle protocol treatment of conditions, strictly defined in terms of the criteria specified above, is stable, has a track record, is based on years of research on treatments that are self-administered by the client and thus appears to be a more efficient way to treat CADD.

A more important issue than that of potential placebo effects is, in my judgment, the treatment context. More generally, it speaks to the issue of equivalence of treatments offered by different therapists in different clinics. One normally assumes that an aspirin administered in one office will have an equivalent effect as the same medication administered by some other practitioner. In neurotherapy, the equivalence metaphor is inappropriate. Theta/beta training offered by one practitioner is not necessarily equivalent to that offered by a different therapist. And, in addition to the research pointing out the importance of therapist variables, we all know that some of us are simply better therapists because our therapeutic skills independent of our technical capabilities with neurofeedback are simply better, more seasoned, and potentiated by disciplined empathy, openness to competing therapeutic metaphors, and emotional grounding that facilitates aggressive treatment. Thus the important question is not if this is a placebo effect, but rather can other practitioners obtain similar results when strictly following the prescribed protocol for the strictly described patient population. In my experience with training many interns, some will and some will not. But the ones who will do so consistently and eventually blend the learned protocol into their own therapeutic style.

Thus I invite readers to strictly apply the above protocol which is very precisely defined in terms of both procedure and client population. It is, after all, very consistent with our stated neurotherapeutic philosophy of treating anomalies found in the QEEG. What is inappropriate, in my judgment, is to assume that conventional theta/beta training at Cz is

appropriate for all varieties of AD/HD without attention to the unique features of each. The above protocol for CADD is simply one protocol to efficiently handle one form of ADD, and do so with far fewer sessions than with the canned theta/beta protocol.

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