

An EEG Biofeedback Protocol for Affective Disorders

By

J. Peter Rosenfeld, Ph.D.
Department of Psychology
Northwestern University
Evanston, IL

REVISED FINAL DRAFT

For Special Issue on EEG Biofeedback; Clinical Electroencephalography
edited by Norman Moore, 8/18/99

Introduction

Despite our own older work which showed that biofeedback with somatosensory evoked EEG potentials can have profound effects on pain perception in rats and people (Dowman & Rosenfeld, 1985; Rosenfeld, Silvia, Weitkunat, & Dowman, 1985; Rosenfeld, 1990), it seemed to us quite a leap to think that an application of EEG biofeedback could be effective in treatment of depression and other affective disorders. Yet the logic of developing such an intervention would be no different than that which we used in our work in the pain modality: In that work, we were aware of a sizeable literature documenting evoked EEG potential (EP) correlates of pain (e.g., Rosenfeld, Diaz-Clark, & Olson, 1983; Buchsbaum, Davis, Coppola, & Naber, 1981a, 1981b; Carmon, Dotan, & Sarne, 1978; Chapman, Chen, & Harkins, 1979). We reasoned simply that if a large value of a particular EP component accompanied intense pain, whereas a smaller value accompanied no pain or analgesia, then if one could train individuals to reduce the particular EP, one ought to see reductions in perceived pain. This approach yielded very promising results (reviewed by Rosenfeld, 1990). Thus it seemed to us that if we could find a reliable EEG index of affect, then we would be in a position to develop an EEG biofeedback protocol for depression.

However, until the relatively recent publication of work from R. Davidson's laboratory (reviewed by Davidson, 1995), there were no documented reliable indices of affect in the waking EEG. Based on evidence from the neurology literature, Davidson and associates hypothesized that the right frontal cortex contained a neural system mediating negative emotion and avoidance behavior, whereas, in contrast, the left frontal cortex contained a neural system mediating positive affect and approach behavior. An active cortex is known to show higher (13-30 Hz) "Beta" frequencies in a low amplitude, desynchronized EEG, whereas an idling or inactive

cortex is known to show lower (8-12) “Alpha” frequencies of synchronous (sinusoidal) higher amplitude activity. Davidson and colleagues thus hypothesized that positive emotion should correlate with high beta and low alpha activity in the left frontal cortex and with low beta and high alpha activity in the right frontal cortex. Negative emotion would correlate with the reverse pattern of cortical activity: high left frontal alpha, low left frontal beta, high right frontal beta, and low right frontal alpha. Because there are harmonics of electromyographic activity reaching down to the beta range (which could be therefore mistaken as beta), many researchers have focused on the alpha (inverse) indices of emotion. (It is possible to utilize beta, but it requires added steps to correct for electromyographic artifact.)

In a series of ingenious, original experiments, Davidson and colleagues provided a strong set of evidence that cortical activation asymmetry (as inversely indexed by alpha power or magnitude) was a reliable correlate of positive and negative emotion. The asymmetry metric developed by the Davidson group will be referred to here as the asymmetry score = $A_1 = \log R - \log L$ where R is alpha power at cortical site F_4 and L is alpha power at cortical site F_3 . It is also possible to define an asymmetry score as $A_2 = (R-L/R+L)$. Although A_1 and A_2 are not mathematically equivalent, they correlate very highly ($\geq .98$; Baehr, Rosenfeld, Baehr, & Earnest, 1998). What has been generally found is that a higher A_1 or A_2 scores go with positive affect and lower A_1 or A_2 scores go with negative affect. (Hereafter, I will sometimes use the unsubscripted term “A-score” to refer to generic alpha asymmetry indexed by either A_1 or A_2). The former condition means relatively greater left frontal activation; the latter means relatively greater right frontal activation. I use the term “relatively” because in any individual case, one cannot say from an A-score whether the critical effects are in the left versus right cortex (or both) since A-scores combine R and L.

Davidson and colleagues (Davidson, 1995) did a variety of studies to support their hypothesis. For example, they showed that a person's resting frontal alpha asymmetry predicted their affective responses to emotionally positive and negative film clips (Tomarken, Davidson, & Henriques, 1990). They also showed that rewards and punishments led to differential asymmetry responses (Sobotka, Davidson, & Senulis, 1992). They also showed that facial expressions of emotion were systematically related to asymmetry scores (Ekman, Davidson, & Freisen, 1990. Many other examples are reviewed in Davidson, 1995.)

Most relevant to this chapter, (1) Henriques & Davidson (1990) showed that currently depressed persons have left frontal hypoactivation (lower A_1 scores) in comparison with never depressed persons; (2) Henriques & Davidson (1991) showed that previously depressed but now remitted persons show also a relative left frontal hypoactivation in comparison with never depressed persons. We (Gotlib, Ranganath, & Rosenfeld, 1998) replicated and extended this finding by comparing (in one study) three groups: currently depressed, formerly depressed, and never depressed persons. We found that both the currently depressed and remitted patients had comparably low levels of left frontal activation (reduced A_1 scores) in comparison with the never-depressed controls. From one perspective, the significance of these findings was that the activation asymmetry seen was apparently a trait marker, since the current state of depression (or remission) did not predict the A_1 -score. The implication was that a pathological activation asymmetry indicated the ubiquitous vulnerability to depressive reactions to stressful events. Remitted depressives may be currently not depressed, but are always vulnerable to depression as indexed by their putatively chronic left frontal hypoactivation.

Experimental EEG Biofeedback Studies in Normals: State-Trait Issues.

When we first learned of the bare outlines of Davidson's work, we were encouraged about a possible biofeedback application for depression because we now had (in the A-score) a possible reliable neural correlate of affect to train. However, the notion that the A-score indexed an innate vulnerability, a constitutional trait, suggested that the picture was less promising: It did not seem intuitively reasonable to contemplate modification of an innate physiological tendency. On the other hand, the reports of Henriques & Davidson (1990, 1991) and Gotlib et al (1998) were consistent with, but did not prove the trait hypothesis: The data were also consistent with the view that no one shows a pathological activation asymmetry until his/her first bout of depression, which then imposes the pattern on a more or less permanent basis. The pattern could thus be seen as a consequence of, rather than a necessary antecedent condition for depressive reactions.

In any case, it seemed for us that the best approach to seeing if activation asymmetry was modifiable would be to try to modify it. Thus, in two experiments, each using a different method of extracting alpha energy in the EEG, we trained 13 normal subjects to increase A-scores (Rosenfeld, Cha, Blair, & Gotlib, 1995) over a period of just three training days. (Details of the training protocol as used for patients are given below.) The results were that nine of the 13 subjects doubled their rates of A_1 -scores reaching an a priori hit criterion equal to the pretraining mean A_1 value plus .85 standard deviations. The other four subjects were unsuccessful. However, since most modern EEG biofeedback applications call for 40 or more training days, we were extremely encouraged by our first data set.

These results in no way suggested that the EEG biofeedback protocol utilized would affect emotion, even in the normal subjects utilized in the experiment. This is because no measures of

emotion were studied in this first exploratory experiment. However, these results certainly suggested that in the next study, emotion should be measured in conjunction with EEG. The results also suggested that those sources of variance in frontal cortical activation asymmetry which were operantly conditionable were state variables: Although the A-score might be a trait indicator in part, as indicated by earlier studies discussed above, it was also subject to the influence of phasic psychological states under a subject's self-control.

Another of our studies also suggested that the A-score was a state indicator as well as a possible trait indicator: Rosenfeld, Baehr, Baehr, Gotlib, & Ranganath (1996) utilized a clinical population of depressed out-patients in therapy sessions to track day-to-day fluctuations in A₂-scores, and their relationship to affect changes. We found that the A₂-score obtained in the beginning of a therapy session correlated significantly and as highly as Pearson $r > .5$ with the change in affect seen during the therapy session. Thus, day-to-day fluctuations in A₂-score predicted whether affect would improve or become negative in response to the therapy session. Here was further evidence of the lability of activation asymmetry in conjunction with affect.

There was yet one more study (Quinn, 1998) performed in my laboratory that provided strong support for the lability rather than or in addition to the trait-like fixedness of the A-score: It has been long-known that humans have a nasal cycle in which one or the other nostrils is dominant. That is, every few hours, the blood vessels in the walls of one of the two nostrils will become engorged (under autonomic control), and the other nostril then passes more air to the lungs. The consequence is that since the sensation of air passing through the nostril is relayed contralaterally (via the trigeminal nerve) to the cortex, the left and right cortices should show alternating activation in phase with the nasal cycle, and this effect should result in alternating positive and negative affect. To test this hypothesis, Quinn (1998) tested nostril dominance of

subjects as they entered our lab, and then immediately tested A_2 -scores and affect scores in both males and females. The result in males was exactly as expected (as it was in females, but not significantly so). Those entering the lab with dominant right nostrils showed left frontal cortical activation and higher positive affect than those with left nostril dominance. Clearly, the A-score was not simply a constant trait indicator since it varied with the nasal cycle. All these results strongly suggested that a biofeedback study be undertaken, with affect scores tracked along with A-scores.

Allen & Cavender (1996) were the first to replicate and extend our work (Rosenfeld et al., 1995) by utilizing affect measures along with biofeedback of A_1 -scores: In two groups of subjects, one trained to increase, the other to decrease A_1 -scores, it was seen that the uptrained subjects increased their A_1 -scores whereas the downtrainers decreased these scores. Moreover, the direction of training was related to subsequent affective responses to emotionally evocative film clips: Subjects trained to increase A_1 -scores showed greater positive affect to happy and neutral films than did subjects trained to decrease A_1 -scores. This was exactly what would have been predicted from Davidson's earlier results and formulations.

Clinical EEG Asymmetry Studies

While the results just described were exciting for us—it is always gratifying to be replicated and extended by an independent research team—they did not involve clinical effects of EEG biofeedback in a clinical population. Such results were eventually provided by Baehr, Rosenfeld & Baehr, (1997), and are extended and reviewed by Baehr, Rosenfeld, & Baehr (1999).

In all our clinical training sessions with patients, we use the following protocol: Prior to EEG biofeedback training sessions, patients are trained for 15-30 minutes to breath

diaphragmatically and warm their hands to a 95°F criterion. These relaxation procedures help minimize artifacts. In EEG training, patients sit in a recliner with elevated feet. The EEG biofeedback sessions, twice per week, consist of 50% biofeedback followed by 50% psychotherapy including discussion of feelings during and about biofeedback. For EEG biofeedback, F3 and F4, both referenced to Cz, are recorded. Impedances are maintained below 5kohms. EEG for both right and left sites are derived via FFT with Blackman-Harris windowed analog signals over one second epochs. The calculated index for each epoch is A_2 as defined earlier in this chapter, $(F4-F3)/(F4+F3)$. When this value exceeds zero, a clarinet tone signals the patient of a successful trial, and its pitch varies with the A_2 value. No sound is heard when $A_2 < 0$. Patients are told to try to keep the sound on and to try to continuously raise its pitch. Patients receive EEG biofeedback training for 30-60 sessions.

In Baehr, Rosenfeld & Baehr (1997), two case studies from the clinical out-patient practice of Elsa and Rufus Baehr were presented. In one of these cases, the A_2 -score averaged over the first nine sessions was about +4.3. For the last nine (of 36 total) sessions, the average A_2 -score approached +8.0, an almost 100% increase. The objective depression index used in that study was the D-scale of the MMPI, which changed from >60 to <40 from before to after training. As detailed in Baehr et al. (1997), there was also a correlated improvement in the clinical picture. In the second case, the A_2 -score improved from +4.7 to +7.2 in comparing the A_2 averages of the first eight and last 10 days of training. In this case, there was also a reduction of the D-scale of the MMPI from about 64 to about 47, and a clear clinical improvement as assessed by psychiatric evaluation. The rather remarkable feature of this case was that the person had been the patient of Elsa Baehr for 12 years, during which time a variety of other interventions were tried, including pharmacotherapy, and other EEG biofeedback protocols. It was only after the

EEG frontal alpha asymmetry protocol was applied that major, stable clinical improvements were documented, and the patient discontinued Paxil during sessions 25-34. This is the only dataset (based on the asymmetry protocol) we know of in which some control for non-specific effects is present. Of course it remains possible that simple passage of time mediated a spontaneous remission. It is clear that in all this kind of work (including work from other chapters in this issue), good control studies are essential, yet largely absent, in order to allow attribution of clinical benefits specifically to the EEG biofeedback protocol. Such systematic data are also lacking with respect to the asymmetry protocol; we are trying to collect them with various clinical collaborators from around the country, however it has been an unfortunately elusive goal to set up control conditions (described below) within the constraints imposed by private clinical practices. So far, the second case described above is the closest thing to a control study that we have.

The reason it has been difficult to run a solid control study in a clinical setting is quite easily appreciated upon consideration of what ideal experimental and control treatment groups should look like. What the groups look like, in turn, depends upon what inferences one wants to draw from the study. The most extreme inference would be that the EEG biofeedback component of the protocol is necessary and sufficient to effect change in mood of clinical significance. To make this statement, one would put one (experimental) group through EEG asymmetry training only; (i.e., no concomittant psychotherapy, pharmacotherapy, etc.). The control group would receive another form of EEG biofeedback which trains an EEG variable not associated with affect, e.g., increased sensorimotor rhythm. Control and experimental subjects would be randomly assigned to groups and drawn from the same population. A neutral technician would run the subjects who, along with the trainer, would be blind to which protocol

was being used. It would be necessary to show that both groups reach similar training levels in terms of hit rate, but that only the experimental group showed significant changes in EEG asymmetry and affect. (A more scientifically perfect but ethically impossible study would train one group in increased asymmetry and another in decreased asymmetry. The former group should improve clinically, the latter group should get worse!)

A more feasible control study would utilize randomly assigned patients, drawn from the same population, however these patients might all have other concomittant treatments. It would be necessary for medication levels to remain constant throughout the training. Again, experimental subjects would receive asymmetry training, control subjects would receive some other EEG biofeedback protocol unrelated to affect. Again it would be best for the study to be run in a double-blind fashion. If there were clinical differences between groups, one could infer that the EEG asymmetry component of the treatment package was a necessary component; one would conclude nothing about sufficiency. This would, nevertheless, be a significant addition to knowledge.

During the Baehr et al. (1997) study, it was also seen that the course of training was not always smooth. One patient in particular received some serious bad news during training, and her A_2 -scores promptly regressed, before ultimately recovering and progressing further in a positive direction. This observation has two important implications: 1) the protocol training effects may be influenced by life's vicissitudes, which should temper unrealistic enthusiasm about the protocol which, may have positive but not perfect effects, and 2) it is clear that frontal alpha asymmetry is more than a trait index, since it changes with life events.

More supporting clinical data are being collected by independent clinicians and by ourselves. These data will be shortly presented, but since they use an index of alpha asymmetry

not yet explained here, it will be introduced now: The average A-score (whether A_1 or A_2) as a summary statistic for a session is easily influenced by occasionally very high (or low) samples, and is thus quite variable. Baehr, Rosenfeld, Baehr, & Earnest (1998) reasoned that an index based on percent of time when the A-score exceeds some criterion (e.g., zero) might be a less variable, summary asymmetry index for a session since it is not influenced by occasional extreme values. For example, both small and large departures from threshold would count the same. We tested this notion by comparing depressed and normal patients on both session average A_2 -scores, as well as on PCT scores = percentage of time in a session during which the A_2 score was greater than zero (by any amount). It was found that indeed the PCT score was a significantly better diagnostic index than the A_2 -score. Obviously, the moment-to-moment A-score must still be used with a reinforcement criterion during a training session. However, the PCT-score is not only a better summary discriminator for the entire session, it is also easier to define for patients during review of their progress with them during the course of treatment. Its lower variability also allows the patient to more readily appreciate progress.

Therefore, we present in Fig. 1, the PCT scores, MMPI-T scores (on D-scale), and Beck Depression (BDI) scores, before and after training for four patients. (The details of these and two other cases are in Baehr et al., in press.) It is clear that as PCT increases, the BDI and MMPI-T scores decline. These kind of results have now also been replicated by an independent single case report from another clinician (Earnest, 1999). This report extended our results also by demonstrating the success of the protocol for the first time with an adolescent patient suffering with depression.

Conclusions and Remaining Problems:

The clinical story, to date, has been most promising, but we do need control data, as noted above. We should also note that our protocol has been unsuccessful with two bipolar patients. However, there have been recent problems regarding the original empirical foundation of the asymmetry protocol: For example, in comparing adolescent suicide attempters with normals in cortical asymmetry, Graae, Tenke, Bruder, et al. (1996) found differences, but the non-depressed attempters (vs. depressed attempters) accounted for the preponderance of asymmetry effects particularly in posterior (vs. frontal) regions. This is not what the Davidson group might have predicted, since their major effects are more often seen frontally. However, it is noted that the Graae et. al. (1996) group utilized a nose reference for EEG recording, which the Davidson group does not. That the choice of referencing montage can have a profound effect on EEG recording has long been known. Indeed, the effects cited above by Quinn (1998) regarding nasal cycle and asymmetry were obtained only when referencing F3 and F4 to Cz, as the Davidson group did in early studies. (More recently, they have used other montages, however the asymmetry effects reported were usually obtained with all montages.) Recently, Reid, Duke, & Allen (1998) also failed to replicate the typical early findings of the Davidson group, and likewise noted differential effects of montage. They too found key effects to occur parietally rather than frontally. Likewise, Hagemann, Naumann, Becker, Maier, & Bartussek (1998) reported that analysis procedure and referencing montage affected outcome, such that one could replicate or fail to replicate the early Davidson group findings depending on the reference used. However Hagemann et. al. did replicate the typical frontal asymmetry association with affect only when they used a Cz referencing montage along with appropriate other procedures. This agrees with findings in our lab (Gotlib et. al., 1998; Quinn, 1998), however as discussed by

Hagemann et al., the use of a Cz reference for lateral leads could imply that asymmetries discovered are in phase rather than amplitude of alpha. They could not replicate the typical findings using other referencing schemes which are theoretically and empirically better for demonstration of any EEG asymmetry.

The implications of these conundrums for our clinical program are minimal: we still have a viable EEG protocol in need of further support via controlled studies. However the interpretation (conceptual foundation) of these effects will have to be changed should it turn out that alpha phase asymmetry, rather than amplitude (activation) asymmetry underlies the correlation of the Cz referenced A-score and affect. (Davidson, 1998, has also addressed these concerns).

Table 1: Abbreviated Patient Descriptions:

<u>Case</u>	<u>Sex</u>	<u>Age</u>	<u>DSM-IV Diagnosis</u>	<u>Prior and Concomitant Other Treatments</u>
1	F.	65	296.32	Psychotherapy 12 yrs, Paxil, 20 mg/day at start of asymmetry training; discontinued 30 days later.
2	M.	37	300.4	Psychotherapy 1yr., Zoloft, 75 mg/day 5 mos. prior to asymmetry treatment, discontinued during treatment.
3	F.	34	296.21	Psychotherapy, Prozac, 20 mg/day 15 mos. prior to EEG training, discontinued after 6 weeks of training.
4	F.	40	300.4	Psychotherapy, Paxil, 20 mg/day 2 yrs. prior to treatment, continued during treatment.

References

- Allen, J.B. and Cavendar, J.H. (1996). Biofeedback alters EEG Asymmetry. *Psychophysiology*, 33 (Supplement), S17 (abstract).
- Baehr, E., Rosenfeld, J.P., Baehr, R. (1997). The clinical use of an alpha asymmetry biofeedback protocol in treatment of depressive disorders: two case studies. *J. Neurother.* 2, 12-27.
- Baehr, E., Rosenfeld, J.P., Baehr, R., & Earnest, C.(1998). Comparison of two EEG asymmetry indices in depressed patients vs. normal controls. *Int. Journal Psychophys.*, 31, 89-39.
- Baehr, E., Rosenfeld, J.P., & Baehr, R., (1999). Clinical use of an Alpha Asymmetry Protocol in the treatment of Mood Disorders. In J.R. Evans & A.Abarbanel,(eds.) *Introduction to Quantitative EEG and Neurofeedback*. San Diego: Academic Press
- Buchsbaum, M.S., Davis, G.C., Coppola, R., and Naber, R.(1981). Opiate pharmacology and individual differences. I. Psychophysical pain measurements. *Pain*, 10, 357-366.
- Buchsbaum, M.S., Davis, G.C., Coppola, R., and Naber, R.(1981). Correlation of subjective pain experience with cerebral evoked responses to noxious thermal stimulations. *Exp. Brain Res.*, 33:445-453.
- Carmon, A., Dotan, Y., and Sarne, Y. (1978). Correlation of subjective pain experience with cerebral evoked responses to noxious thermal stimulations. *Exp. Brain Res.*, 33, 445-453.
- Chapman, R.C., Chen, A.C.N., and Harkins, S.W.(1979). Brain evoked potentials as correlates of laboratory pain: a review and perspective. *Adv. Pain Res. Ther.*, 3, 791-803.
- Davidson, R.J. (1995). Cerebral Asymmetry, Emotion, and Affective style. In R.J. Davidson & K. Hugdahl (Eds.) *Brain Asymmetry*, Cambridge: M.I.T. Press, pp. 361-387.
- Davidson, R.J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology*, 35, 607-614..
- Dowman, R., & Rosenfeld, J.P. (1985). Operant conditioning of somatosensory evoked potential (SEP) in rats. 1. Specific changes in SEP amplitude and a naloxone-reversible, somatotypically specific change in facial nociception. *Brain Research*, 333, 201-212
- Earnest, C. (1999). Single case study of EEG Asymmetry Biofeedback for depression: An independent Replication in an Adolescent. *J. Neurotherapy*, 3, 28-35.
- Ekman, P., and Davidson, R.J., and Friesen, W.V. (1990). Duchenne's smile: Emotional expression and brain physiology, *Journal of Personality and Social Psychology*, 58, 342-353.

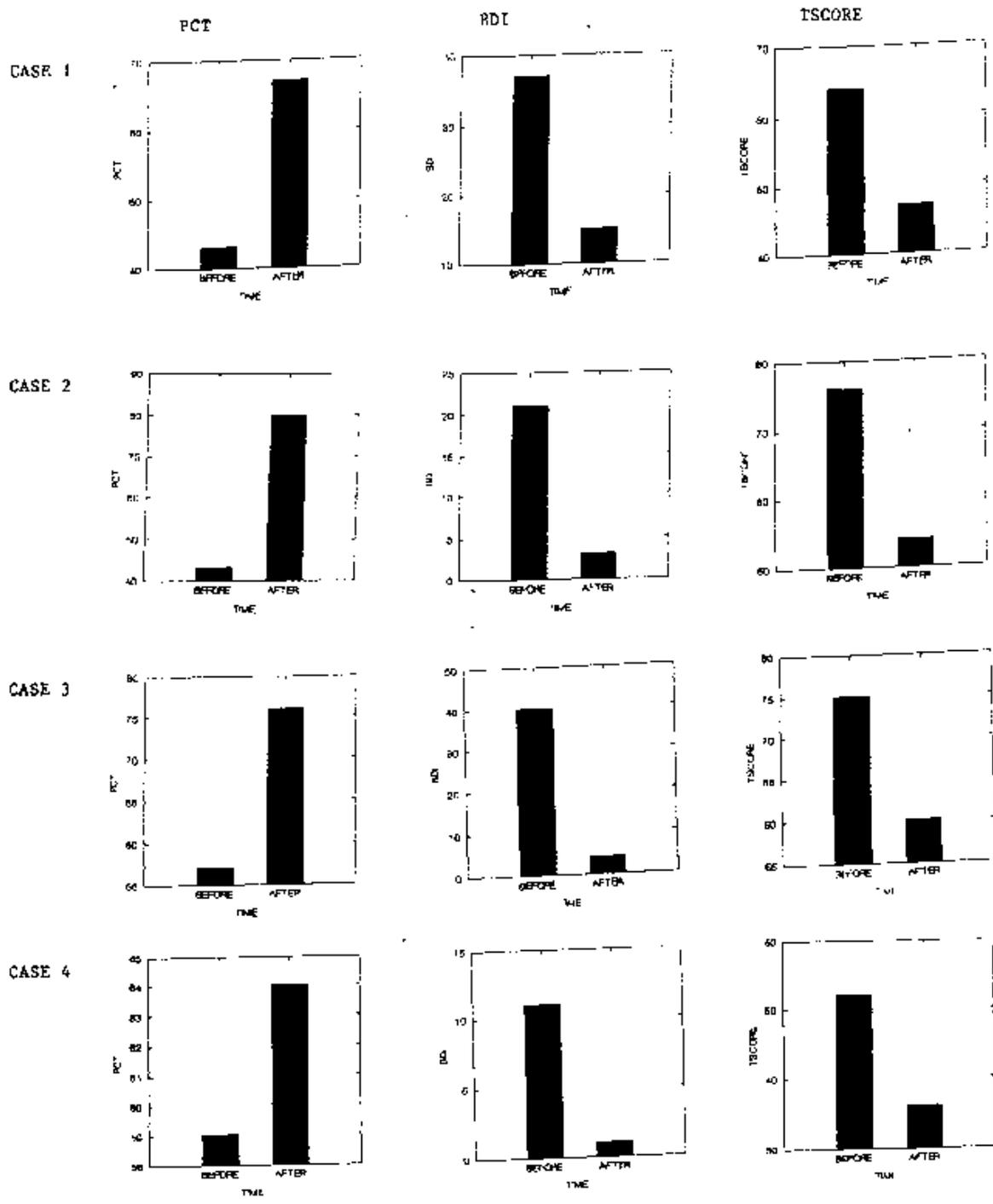
- Graae, F., Tenke, C., Brader, G., Rotheram, M., Piacentini, J., Castro-Blanco, D., Leite, P., & Towey, J. (1996). Abnormality of EEG Alpha Asymmetry in female adolescent suicide attempters. *Biol. Psychiatry*, 40, 706-713.
- Gotlib, I.H., Ranganath, C., Rosenfeld, J.P., (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, 12, 449-478.
- Hagemann, D., Naumann, E., Becker, G., Maiers, S. & Bartussek, D. (1998). Frontal Brain Asymmetry and Affective Style: A conceptual Replication. *Psychophysiology*, 35, 372-388.
- Henriques, J.B., Davidson, R.J. (1990). Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *J. Abnorm. Psychol.* 99, 22-31.
- Henriques, J.B., Davidson, R.J. (1991). Left frontal hypoactivation in depression. *J. Abnorm. Psychol.*, 100, 535-545.
- Quinn, L. (1998). Hemispheric EEG Asymmetry, emotion, and the nasal cycle. Unpublished Ph.D Thesis, California School of Professional Psychology, San Diego, California.
- Ranganath, C. (1996). Anterior EEG Asymmetry, Personality, and Depression. Unpublished Masters Thesis, Northwestern University, Evanston, IL.
- Reid, S., Duke, L., and Allen, J.B. (1998). Resting Frontal EEG Asymmetry in Depression: inconsistencies suggest the need to identify mediating factors. *Psychophysiology*, 35, 389-404.
- Rosenfeld, J.P. (1990). Applied Psychophysiology and biofeedback of event-related potentials (brain waves): Historical perspective, review, future directions. *Biofeedback and Self-Regulation*, 15, 99-120.
- Rosenfeld, J.P., Diaz-Clark, A., & Olsen, R.E. (1983). Response to painful electrical stimulation in MPD Syndrome Patients. *Journal of Dental Research*, 62, 259-260.
- Rosenfeld, J.P., Silvia, R., Weitkunat, R., & Dowman, R. (1985). Operant control of human somatosensory evoked response alters experimental pain perception. *Advances in Neurology*, 9, 343-348.
- Rosenfeld, J.P., Baehr, E., Baehr, R., Gotlib, I.H., Ranganath, C. (1996). Preliminary evidence that daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions. *Int. J. Psychophysiol.* 23, 137-141.
- Rosenfeld, J.P., Cha, G., Blair, T., Gotlib, I.H. (1995). Operant (Biofeedback) control of left-right frontal alpha power differences: potential neurotherapy for affective disorders. *Biofeedback Self Regul.* 20, 241-258.

Sobotka, S., Davidson, R.J., and Senulis, J. (1992). Anterior brain electrical asymmetries in response to reward and punishment. *Electroencephalography and Clinical Neurophysiology*, 83, 236-247.

Tomarken, A.J., Davidson, R.J., and Henriques, J.B. (1990). Resting frontal brain asymmetry predicts affective responses to films. *Journal of Personality and Social Psychology*, 59, 791-801.

Figure Legend

Fig 1. Each pair of black bars in a graph gives for 4 cases, one per row, the scores before(left bar) and after(right bar) EEG Biofeedback training. The first column(PCT) gives the percent of time the Activation Asymmetry or A-score>0. (See text regarding A-Scores.)The second column gives the Beck Depression score (BDI), and the third column gives the T-score on the Minnesota Multiphasic Personality Inventory (MMPI).



The American Journal of Drug and Alcohol Abuse, 31:455–469, 2005
Copyright © Taylor & Francis Inc.
ISSN: 0095-2990 print / 1097-9891 online
DOI: 10.1081/ADA-200056807

Effects of an EEG Biofeedback Protocol on a Mixed Substance Abusing Population

William C. Scott, B.S.W., C.C.D.P.,¹ David Kaiser, Ph.D.,² Siegfried Othmer, Ph.D.,³ and Stephen I. Sideroff, Ph.D.⁴

¹Neuropsychiatric Institute, University of California, Los Angeles, California, USA and Restorative Technologies, Santa Monica, California, USA

²Department of Psychology, Rochester Institute of Technology, Rochester, New York, USA

³EEG Spectrum International Inc., Encino, California, USA

⁴Department of Psychiatry and BioBehavioral Sciences, University of California, Los Angeles, California, USA and Moonview Sanctuary, Santa Monica, California, USA

Abstract: This study examined whether an EEG biofeedback protocol could improve outcome measures for a mixed substance abusing inpatient population. *Method.* One hundred twenty-one volunteers undergoing an inpatient substance abuse program were randomly assigned to the EEG biofeedback or control group. EEG biofeedback included training in Beta and SMR to address attentional variables, followed by an alpha-theta protocol. Subjects received a total of 40 to 50 biofeedback sessions. The control group received additional time in treatment equivalent to experimental procedure time. The Test of Variables of Attention (TOVA), and MMPI, were administered with both tester and subject blind as to group placement to obtain unbiased baseline data. Treatment retention and abstinence rates as well as psychometric and cognitive measures were compared. *Results.* Experimental subjects remained in treatment significantly longer than the control group

Address correspondence to Stephen I. Sideroff, Ph.D., Department of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, 1245 16th St., Suite 210, Santa Monica, CA 90404, USA; E-mail: Sideroff@ucla.edu

($p < 0.005$). Of the experimental subjects completing the protocol, 77% were abstinent at 12 months, compared to 44% for the controls. Experimental subjects demonstrated significant improvement on the TOVA ($p < .005$) after an average of 13 beta-SMR sessions. Following alpha-theta training, significant differences were noted on 5 of the 10 MMPI-2 scales at the $p < .005$ level. *Conclusions.* This protocol enhanced treatment retention, variables of attention, and abstinence rates one year following treatment.

Keywords: EEG, biofeedback, EEG biofeedback, addiction treatment, chemical dependency, alpha-theta, TOVA, MMPI

INTRODUCTION

Alcohol and drug abuse is an ongoing societal and treatment problem (1, 2). While major resources have been employed to study and treat addiction, there has been little significant improvement in the success rate of treatment. Relapse rates remain high, typically over 70% (3–5). Gossop et al. (6) reported 60% of heroine addicts relapsed one year following addiction treatment.

Peniston and associates have demonstrated significantly higher abstinence rates with alcoholics when they incorporated EEG biofeedback into the treatment protocol (7–10). Eighty percent of subjects in these experiments were abstinent one-year posttreatment.

EEG biofeedback training is an operant conditioning technique used to reinforce or inhibit specific forms of EEG activity. In the alpha-theta protocol employed by the Peniston studies, low frequency EEG activity was reinforced. The alpha theta protocol was first demonstrated to be effective with posttraumatic stress disorder (11).

The efficacy of alpha-theta EEG biofeedback may lie in its ability to allow participants to better tolerate stress, anxiety, and anxiety-eliciting situations, which are particularly evident during the initial phases of recovery. This protocol was shown to significantly lower 13 of the scales of the Millon Clinical Multiaxial Inventory (MCMI), including anxiety, whereas traditional treatment produced decreases in only two of these scales (7). There have been, however, questions raised in the literature regarding the sample size, sample independence, and methodology in the Peniston et al. studies (12). Furthermore, there have been no controlled studies reported that extend these findings to other substances of abuse.

In addition to the psychological problems that substance abusers face in remaining abstinent, they also experience comorbid conditions that affect cognitive and attentional deficits. These deficits may be acquired from prolonged substance abuse (13–17), but the evidence also points to

deficits that predate the abusing behavior (18). For example, in one study, approximately 35% of treatment-seeking cocaine abusers met the *DSM-IV* criteria for childhood attention deficit hyperactivity disorder (19). Also, adult alcoholics report more residual-type attention deficit disorder (ADD) than controls (20). Low cognitive ability also has been shown to predict relapse after treatment at an alcohol treatment facility (21).

EEG biofeedback also has been used successfully to improve attentional, cognitive, and psychosocial functioning, including reductions in impulsivity (22–24). These and other studies have employed a protocol in which beta and SMR frequencies (15–18 Hz and 13–15 Hz, respectively) were operantly conditioned, while inhibiting theta frequencies, in remediating attentional and cognitive deficits in children and adults with ADD (25–28, 35). Given the relationship between cognitive/attentional impairment and addiction it would strengthen a treatment model to address these deficits.

In the present study, a beta/SMR EEG biofeedback training regimen was combined with an alpha-theta protocol in the treatment of a mixed substance abusing population. One expected objective was the enhanced ability of the subjects to focus on the treatment program, reduce impulsivity, and, thereby, increase program retention.

In order to extend the positive EEG biofeedback findings in the alcoholic population, an addict population was selected that included patients addicted to the following primary drugs: heroin, crack/cocaine, and methamphetamine, as well as alcohol.

METHOD

Participants

One hundred twenty-one volunteers from the Cri-Help, Inc. residential treatment program in the Los Angeles area participated in this study. There were 49 females and 72 males. They were 19 to 53 years of age, with a mean age of 32.4. The primary drug of choice reported at admission was 31% heroin, 28% crack cocaine, 26% methamphetamine, 6% alcohol, and 9% other controlled substances; 94% were multiple-drug users.

Subjects determined to have a diagnosed psychotic or personality disorder (based on *DSM-IV* criteria), or a seizure disorder, were excluded. Subjects were randomly assigned to the EEG biofeedback plus conventional treatment group (60 experimental subjects) or the conventional treatment-only group (61 control subjects).

Subjects were provided informed consent before participating in this experiment, approved by the UCLA Human Subjects Protection Committee.

Procedures

All subjects received treatment based on the Minnesota Model 12-step oriented program described by Stinchfield and Owen (29) supported by group, family, and individual counseling. In addition, the experimental group received 40–50 EEG biofeedback sessions. The control group received additional treatment time equivalent to the biofeedback sessions.

Experimental subjects underwent two sessions of EEG biofeedback training (45 minutes per session) five days a week for four to five weeks. EEG biofeedback was performed on a Neurocybernetics 2-Channel EEG biofeedback system.

In Phase I, experimental subjects underwent 10–20 sessions of Beta-SMR EEG biofeedback in which operant conditioning was used to augment either 15–18 Hz (beta) or 12–15 Hz (SMR) EEG activity. At the same time, training attenuated elevated activity in the 2–7 Hz (theta) and 22–30 Hz (high beta) ranges. Active bipolar electrode placement was at C3-FPZ for beta and at C4-PZ for SMR, based on the international 10–20 system of electrode placement (30).

The starting protocol consisted of beta training 50% of the time and SMR training 50% of the time. These percentages would be altered based on changing symptomatology and TOVA results (31) with inattentive or impulsive profiles resulting in increased beta or SMR training, respectively.

After 10 Beta-SMR EEG biofeedback sessions, participants were reassessed with the TOVA. If a participant scored within the normal range (i.e., scores of 85 or above), he or she began alpha-theta training. If the TOVA remained abnormal after the initial 10 Beta-SMR sessions, 5 or 10 additional Phase 1 treatments were administered. It took a median of 10 Beta-SMR sessions with a mean of 13 sessions for the TOVA to normalize for the experimental subjects.

In Phase II, subjects underwent 30 sessions of alpha-theta training. The frequency range for alpha was 8–11 Hz and for theta it was 5–8 Hz. The initial sessions were used to train down alpha levels that were above 12 μ V (peak to peak), while augmenting theta, until there was “crossover.” This was defined as the point at which the alpha amplitude drops below the level of theta. Subsequent to the first achievement of crossover, both alpha and theta frequencies were augmented.

Before initial crossover was achieved, excess EEG activity in the range of 15–30 Hz was inhibited. This was intended to reduce muscle tension and to quiet the mind. After crossover was achieved, the 2–5 Hz frequency range

also was inhibited. This was intended to discourage the sleep transition during low-arousal states.

Each alpha-theta session began with the subject sitting in a chair with eyes closed. The active electrode was placed at Pz with a left-ear reference (A1). The right earlobe was connected to circuit ground. Two distinct tones were employed for alpha and theta reinforcement, with the higher pitched sound used to index the higher-frequency alpha band.

At the start of each session, the technician spent 3–5 minutes reading a script of guided imagery to the experimental subject that dealt with identified essential elements of maintaining abstinence. These included ongoing regular attendance at 12-step meetings; weekly meetings with a sponsor, expanding the individuals identified comfort zones, and mental exercises dealing with cue extinction and relapse rejection.

After the guided imagery, it was made clear to the subject that the objective of the training did not involve explicit rehearsal of the script during the EEG biofeedback. Subjects reporting previous meditative practices were asked not to use them during the training, since meditation has been observed to override alpha-theta reinforcement effects. Following the alpha-theta training, clients were given the opportunity to process their experience.

When it appeared that sleep might be occurring during training, subjects were told prior to their next session to move a limb if they heard the technician say either, “Right foot, left foot, right hand, or left hand.” At points where the subject’s delta activity (2–5 Hz EEG) started to elevate, as well as at their highest amplitudes (indications of sleep onset), the limb commands were given to determine responsiveness. The delta amplitude value at which the subject transitioned to nonresponsiveness was documented. Subsequently, during sessions where delta was elevating toward nonresponsiveness levels, the feedback sounds were inhibited in order to discourage the sleep transition.

Measurements

Tests were administered prior to training, after Beta-SMR training (Phase 1) and after alpha-theta training (Phase 2) for experimental subjects and at commensurate points in time for the control group (typically 1, 16, and 46 days into the research program). All subjects had acclimated to the institutional setting for a minimum of 7 days prior to testing. The initial testing was accomplished with subjects and experimenters blind to group placement.

The TOVA was administered to assess attentional and cognitive functions (31–33). The Minnesota Multiphasic Personality Inventory (MMPI-2) was administered at the start of the study and again at 46 days.

Patient abstinence was determined by collateral contacts in addition to self-report. Follow-up interviews for this purpose took place at 3-month intervals over a 12-month period. Research subjects gave permission to contact individuals who were intimately involved in their recovery. These individuals were their 12-step sponsors, family members, and those people referring the subject into the program. Subjects who used substances beyond one 4-week window were considered to have fully relapsed. Those whose relapse duration was within a single 4-week window were categorized as a brief relapse (Please see appendix for procedures flow chart.).

RESULTS

Days in Treatment

Length of stay in treatment averaged 138 days for experimental subjects and 101 days for controls. This difference was significant $t(119)=-3.07$, $p<0.005$. Median length of stay was 147 days for experimental and 103 days for control subjects. Figure 1 shows retention in the program over the first 12 weeks of the program. As can be seen, at the end of this period, 46% of control subjects had dropped out of treatment, compared to only 24% of those who received EEG biofeedback. A chi-square analysis demonstrated a significant difference in drop-out rate between experimental and control groups over the 12-week period [X^2 ($n=121$)=6.29, $p<.05$.] There was no

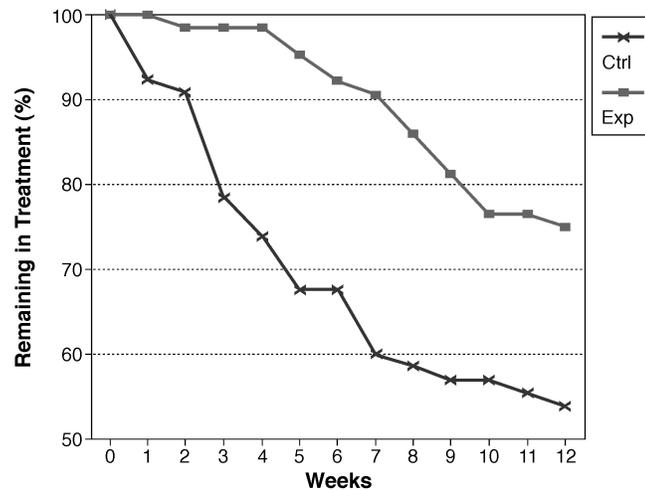


Figure 1. Effect of the EEG biofeedback protocol on patient retention for control ($n=61$) and experimental ($n=60$) subjects.

significant interaction between drug type used (stimulant vs. sedating drugs) and days remaining in treatment [$F(1,118) = .004, ns$].

Abstinence Rate

Figure 2 presents the data for the 103 subjects who had reached their 12-month poststudy status. This includes 55 experimental and 48 control subjects. Of these subjects, there were 7 experimental and 17 control subjects who dropped out of treatment prior to completing the study (the initial 45 days), while there were 4 control subjects and 1 experimental subject who could not be contacted at the 12-month interval.

Of the remaining experimental subjects who completed the study and were assessed at 12 months, 36 of 47 (77%) were abstinent. This included 8 subjects who had one brief relapse period of less than 30 days during the year. Of the control subjects who completed the study, there were 12 of 27 subjects (44%) who were abstinent. This included 1 subject who had one brief relapse period of less than 30 days. A chi-square analysis demonstrated a significant difference between one year abstinence rates of the experimental group versus the control group ($\chi^2 2(74) = 7.78 p < 0.01$). There was no significant interaction between drug type used (stimulant versus depressant) and abstinence rate [$F(1,113) = .844, p > .05$].

MMPI-2 Data

Figure 3 presents pre and posttraining MMPI data, including the 10 clinical scales and 3 validity scales, for the experimental and control groups. Subjects with Lie scores greater than 70 on either pre or posttraining tests were excluded from analysis ($n = 3, 2$ experimental and 1 control). A univariate

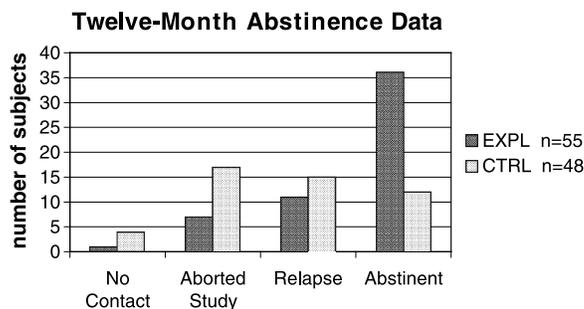


Figure 2. Twelve-month follow-up abstinence data for experimental ($n = 55$) and control ($n = 48$) groups.

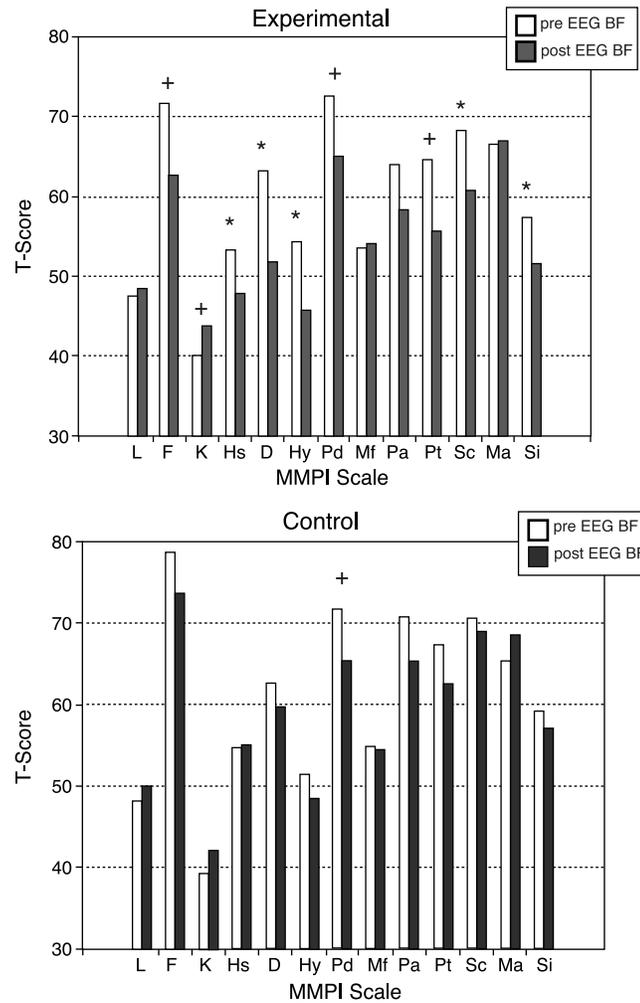


Figure 3. Change in 10 MMPI clinical scales and 3 validity scales for the experimental group ($n=50$) and the controls ($n=33$) (+ $p<.05$, * $p<.005$).

mixed-design analysis of variance (ANOVA) was used to evaluate the effects of the experimental protocol compared to controls on the 10 clinical scales.

As shown in Figure 3, the experimental group's changes exhibited significant improvement compared with the changes in the control subjects ($p < 0.005$), on the Hs (Hypochondriasis), $F(1, 81)=14.087$; D (Depression), $F(1, 81)=48.129$; Hy (Conversion Hysteria), $F(1, 81)=32.682$; Sc (Schizophrenia), $F(1, 81)=15.241$; and Si (Social Introversion) scales, $F(1, 81)=24.647$, $p<.005$. The experimental group also improved on the Pt

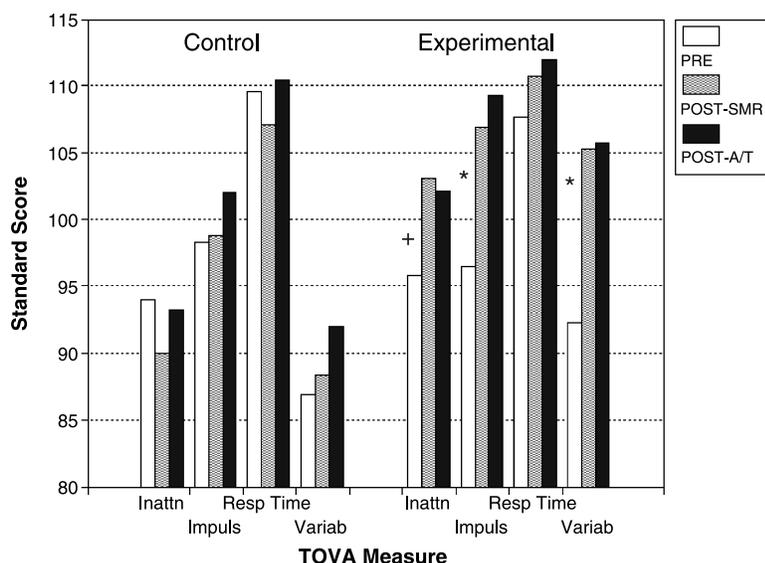


Figure 4. TOVA standard scores for experimental and control groups for pre-training, post-SMR, and post-alpha-theta assessments (+ $p < .05$, * $p < .005$).

(Psychasthenia) scale, although the difference between groups on this scale was not significant $F(1, 81) = 1.727$, $p > .05$. Both groups improved on the Pd (Psychopathic Deviate) scale, $F(1, 81) = 29.016$; $F(1, 81) = 12.832$, $p < .05$, respectively.

TOVA

Mean TOVA standard scores are presented for both groups in Figure 4 (42 experimental, 28 controls). More participants were tested but only those who provided scores from all three test periods (baseline, post-SMR, post-alpha-theta) were analyzed. There was no significant difference between groups in initial baseline TOVA scores [$F(1,303) = 1.333$, $p > .05$]. A univariate, mixed-design ANOVA was used to compare the two groups on four dependent measures of the TOVA: inattention (percent omission), impulsivity (percent commission), response time, and response variability. Low scores were truncated at four standard deviations below normal.

As can be seen in Figure 4, the experimental group exhibited significant improvement in impulsivity and variability measures in response to Beta-SMR training $F(1, 68) = 18.749$; $p < .005$ whereas no comparable change was found for the control group $F(1, 68) = 19.405$; $p > 0.05$. Experimental subjects also demonstrated significant improvement in inattention; however, the score

only marginally differed from that of the control group $F(1, 68)=5.549$ ($p < .05$). TOVA scores were not further enhanced by either the alpha-theta training nor 30 additional days of treatment.

DISCUSSION

The results of this study support the efficacy of EEG biofeedback training in an inpatient drug treatment program. Success was determined by length of time in treatment, or treatment retention, as well as by abstinence rates one year after termination of treatment. Results were further supported by positive changes in attentional variables, and positive changes on the MMPI 2. These findings extend the previous research findings employing alpha-theta EEG biofeedback with an alcoholic population, to other substances of abuse.

The present study employed a Beta-SMR protocol prior to the alpha-theta procedure previously used in addiction studies (7–10). Beta-SMR training previously had been shown to be effective in remediating attentional and cognitive deficits. Results of baseline performance testing using the TOVA did not demonstrate that this population had significantly below average attentional indices. However, testing following the Beta-SMR protocol showed that this procedure improved these test measures for the experimental subjects, particularly impulsivity and variability. This result may partly account for the improved treatment retention of this group.

It has been shown that time in treatment is one of the best predictors of remaining abstinent (34). In the present study, the experimental subjects averaged 136 days in treatment. This compared to 98 days for the control population. In addition, treating therapists reported that they noticed experimental subjects appearing more cooperative and more attentive as EEG biofeedback progressed. This subjective observation should be a focus in future studies with a more systematic observation of subjects' behavior.

There were 8 experimental subjects who used briefly (less than 30 days) but were abstinent at the 12-month follow-up, and there was 1 subject from the control group who had this experience. It has been noted in the previous alpha-theta treatment studies that patients report dysphoria when they used a substance following the EEG biofeedback protocol (8). Some of the experimental subjects in this study had similar experiences. This may indicate that a more fundamental neurophysiological change had taken place as a result of the treatment. Peniston and Kulkosky (7) for example, noted that experimental subjects receiving EEG biofeedback did not show increased circulating beta-endorphin levels, an index of stress, which was found in the control group.

It can be noted that once the EEG biofeedback was concluded, at week five, the subsequent attrition rates became indistinguishable between the two

groups. It may be useful in future studies to extend the length of the biofeedback training to see if it has further impact on experimental results.

One of the more striking findings of the present study and similar to the Peniston (8) results, is the positive change noted in the MMPI. The experimental subjects showed significant improvement in five of the clinical scales: Hypochondriasis, Depression, Hysteria, Schizophrenia, and Social Introversion. These changes indicate a lowered level of general distress or discomfort. More specifically there may be a reduced sense of alienation and depression, as well as defensiveness. These are vital factors in recovery.

The present study did not demonstrate differential effectiveness of the EEG biofeedback protocol for sedative or stimulant drug abusers. This should be a focus of future research in which larger numbers of subjects are employed. Both groups of subjects appeared to benefit from this protocol. If the lack of dependency on drug type is confirmed, the case can be made that alpha/theta training addresses core issues in addiction rather than drug-specific aspects of dependency.

In the present study, one-year abstinence was determined by collateral contacts in addition to self-report. These individuals were reliable sources who were intimately connected to the recovery process, including their 12-step sponsors, family members, and those people referring the subject into the program. Future research results should be supported by incorporating urine testing as a further corroboration of abstinence.

Since EEG-based reinforcement was such a prominent constituent of the experimental program, the question arises as to whether the benefits of training could also be documented through observable EEG changes. The present study was not designed to analyze the appropriate artifact free data. Future research should incorporate methodology to record and analyze the appropriate quantitative EEG data.

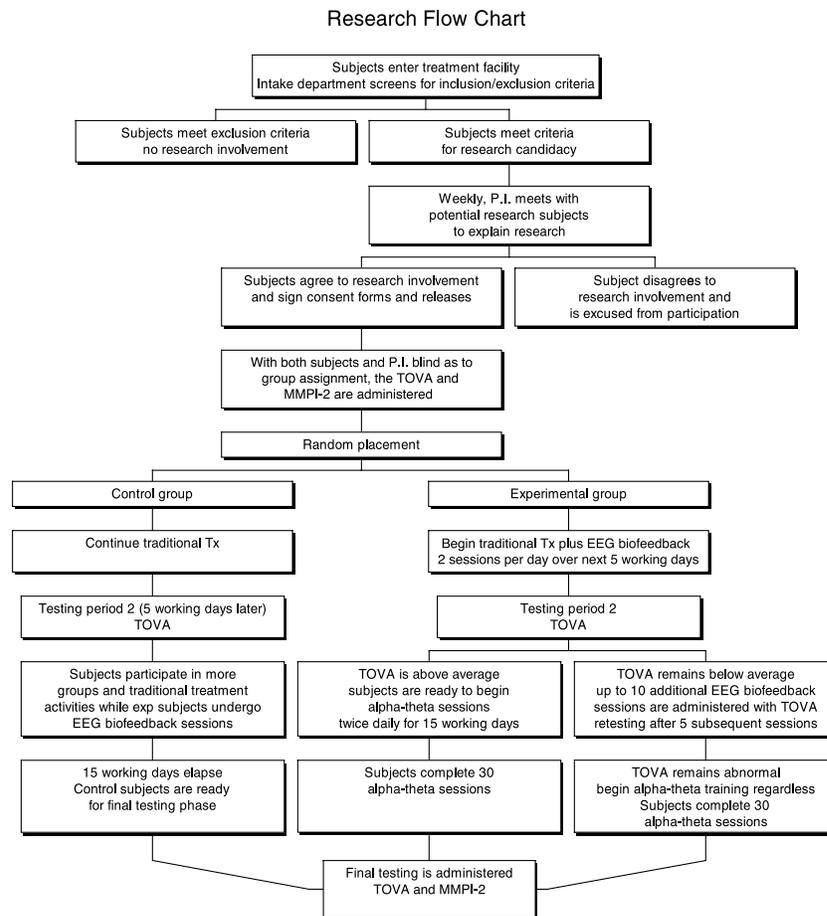
It is important to place the results of this study in the context of the long-standing difficulty in achieving successful abstinence with the drug-abusing population. EEG biofeedback appears to promote and support positive change in the level of neurophysiological and psychosocial functioning in the addict, as well as enhancing treatment retention. It therefore constitutes a promising approach that now requires additional study for further validation as well as to elucidate operative mechanisms to optimize the procedures, and to facilitate integration into standard treatment programs.

CONCLUSION

The present study supports the efficacy of an EEG biofeedback protocol as adjunctive therapy in an in-patient drug treatment program. This protocol appears to be beneficial for both sedative as well as stimulant substances of

abuse. Success was determined by length of time in treatment as well as by abstinence one year after termination of treatment. Supportive data were provided through attentional/cognitive and psychological assessments. These findings extend the research employing alpha-theta EEG biofeedback with an alcoholic population to other drugs of abuse.

APPENDIX



ACKNOWLEDGMENTS

We wish to thank Marcus Sola (CRI-Help, Chairman of the Board), Jack Bernstein (CRI-Help, CEO), and Marlene Nadel (CRI-Help, Clinical

Supervisor) for their participation and willingness to add an innovative approach to their existing treatment model. Thanks also to the CRI-Help Board of Directors for providing funding for this project. We also thank EEG Spectrum International for their donation of a Neurocybernetics EEG biofeedback system. We wish to thank Don Theodore, MA, MFT and Leslie Ruddock, BA (Research Technicians) who administered all EEG biofeedback protocols and coordinated subject sessions with the traditional treatment team. We also thank Susan Othmer who shared her knowledge of beta/SMR protocols and for case consultation. Thanks also to Meredith Sagan for her consultation and critical review of the manuscript. National Computer Systems (NCS Assessments) donated administrations of a self-scoring computerized version of the MMPI-2. Universal Attention Disorders, Inc. contributed administrations of the Test of Variables of Attention (TOVA).

REFERENCES

1. Fagan J. Women and drugs revisited: female participation in the cocaine economy. Special Issue: drugs and crime revisited. *J Drug Issues* 1994; 24:179–225.
2. Yu J, Williford WR. Alcohol, other drugs, and criminality: a structural analysis. *Am J Drug Alcohol Abuse* 1994; 20:373–393.
3. Higgins ST, Budney AJ, Bickel WK, Badger G, Foerg F, Ogden D. Outpatient behavioral treatment for cocaine dependence: one year outcome. *Exp Clin Psychopharmacol* 1995; 3:205–212.
4. Kang S, Kleinman PH, Woody GE, Millman RB, Todd TC, Kemp J, Lipton DS. Outcomes for cocaine abusers after once-a-week psychosocial therapy. *Am J Psychiatr* 1991; 148:630–635.
5. Marlatt GA, Gordon JR. Determinants of relapse: implications for the maintenance of behavior change. In: Davidson P, ed. *Behavioral Medicine: Changing Health Lifestyles*. New York: Brunner/Mazel, 1979:410–452.
6. Gossop M, Stewart D, Browne N, Marsden J. Factors associated with abstinence, lapse or relapse to heroin use after residential treatment: protective effect of coping responses. *Addiction* 2002; 97(10):1259–1267.
7. Peniston EG, Kulkosky PJ. Alpha theta brainwave training and beta-endorphin levels in alcoholics. *Alcoholism: Clin Exp Res* 1989; 13(2):271–279.
8. Peniston EG, Kulkosky PJ. Alcoholic personality and alpha-theta brainwave training. *Med Psychother: Int J* 1990; 3:37–55.
9. Peniston EG, Marrinan DA, Deming WA, Kulkosky PJ. EEG alpha-theta brainwave synchronization in Vietnam theater veterans with combat-related post-traumatic stress disorder and alcohol abuse. *Adv Med Psychother* 1993; 6:37–50.

10. Saxby E, Peniston EG. Alpha-theta brainwave EEG biofeedback training: an effective treatment for male and female alcoholics with depressive symptoms. *J Clin Psychol* 1995; 51:685–693.
11. Peniston EG, Kulkosky PJ. Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related posttraumatic stress disorder. *Med Psychother: Int J* 1991; 4:47–60.
12. Graap K, Freides D. Regarding the database for the Peniston alpha-theta EEG biofeedback protocol. *Appl Psychophysiol Biofeedback* 1998; 23(4):265–275.
13. Ardila A, Rosselli M, Strumwasser S. Neuropsychological deficits in chronic cocaine abusers. *Int J Neurosci* 1991; 5:73–79.
14. Arria AM, Tarter RE, VanThiel DH. Liver-brain relations in alcoholics. *Alcohol Health Res World* 1990; 14:112–117.
15. Hambidge DM. Intellectual impairment in male alcoholics. *Alcohol Alcohol* 1990; 25:555–559.
16. Marchesi C, de Risio C, Campanini G, Maggini C. TRH test in alcoholics: relationship of the endocrine results with neuroradiological and neuropsychological findings. *Alcohol Alcohol* 1992; 27:531–537.
17. O'Mahony JF, Doherty B. Intellectual impairment among recently abstinent alcohol abusers. *Br J Clin Psychol* 1996; 35:77–83.
18. Pogge DL, Stokes J, Harvey PD. Psychometric vs. attentional correlates of early onset alcohol and substance abuse. *J Abnorm Child Psychol* 1992; 20:151–162.
19. Carroll KM, Rounsaville BJ. History and significance and childhood attention deficit disorder in treatment-seeking cocaine abusers. *Compr Psychiatry* 1993; 34:75–82.
20. Nixon SJ, Tivis R, Parsons OA. Behavioral dysfunction and cognitive efficiency in male and female alcoholics. *Alcohol: Clin Exp Res* 1995; 19:577–581.
21. Miller L. Predicting relapse and recovery in alcoholism and addiction: neuropsychology, personality, and cognitive style. *J Cocaine Abuse Treat* 1991; 8:277–291.
22. Lubar JF, Swartwood MO, Swartwood JN, O'Donnell PH. Evaluation of the effectiveness of EEG biofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC–R performance. *Biofeedback Self-Regul* 1995; 20:83–99.
23. Lantz D, Serman MB. Neuropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG feedback training. *Epilepsia* 1988; 29(2):163–171.
24. Othmer S, Othmer SF, Kaiser DA. EEG Biofeedback: training for AD/HD and related disruptive behavior disorders. In: Incorvaia JA, Mark-Goldstein BS, Tessmer D, eds. *Understanding, Diagnosing, and*

- Treating AD/HD in Children and Adolescents, an Integrative Approach. New York: Aronson Press, 1999:235–318.
25. Cartozzo HA, Jacobs D, Gevirtz RN. EEG Biofeedback and the remediation of ADHD symptomatology: a controlled treatment outcome study. Presented at the Annual Conference of the Association for Applied Psychophysiology and Biofeedback, Cincinnati, Ohio, March 1995.
 26. Linden M, Habib T, Radojevic V. A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorders and learning disabilities. *Biofeedback Self-Regul* 1996; 21:35–50.
 27. Rossiter TR, La Vaque TJ. A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorder. *J Neurother* 1995; 1:48–59.
 28. Thompson L, Thompson M. Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. *Appl Psychophysiol Biofeedback* 1998; 23(4):243–263.
 29. Stinchfield R, Owen P. Hazelden's model of treatment and its outcome. *Addict Behav* 1998; 23(5):669–683.
 30. Jasper HH. The 10–20 system of the international federation. *Electroencephalogr Clin Neurophysiol* 1958; 10:371–375.
 31. Dupuy TR, Greenberg LM. TOVA Manual. Los Alamitos, CA: Universal Attention Disorders, 1989.
 32. Greenberg LM, Waldman ID. Developmental normative data on the Test of Variables of Attention (T.O.V.A.). *J Child Psychol Psychiatry* 1993; 34:1019–1030.
 33. Kane RL, Kay GG. Computerized assessment in neuropsychology: a review of tests and test batteries. *Neuropsychol Rev* 1992; 3:1–117.
 34. Ciraulo DA, Piechniczek-Buczek J, Iscan EN. Outcome predictors in substance use disorders. *Psychiatr Clin North Am* 2003; 26(2):381–409.
 35. Egner T, Gruzelier JH. Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *Neuroreport* 2001; 12:4155–4159.