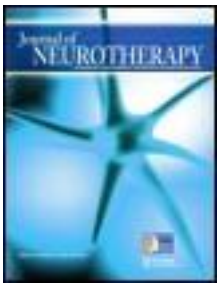

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Neurofeedback Treatment of Depression with the Roshi

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Neurofeedback Treatment of Depression with the Roshi

D. Corydon Hammond, PhD

ABSTRACT. *Introduction*. A patient with severe, medication resistant depression was found to have the frontal alpha asymmetry described in Davidson's (1998a) research as demonstrating a predisposition to depression.

Treatment. Initial sessions of EEG neurofeedback using Rosenfeld's (1997) protocol for correcting the alpha asymmetry were discouraging, actually producing slight negative change. Therefore, treatment shifted to using the Roshi, a two channel unit combining neurofeedback and photic stimulation, doing primarily left hemisphere beta training.

Results. The very first Roshi session produced positive changes, and within five sessions the patient reported feeling less depressed and more energetic. At the conclusion of thirty training sessions, objective testing documented dramatic reductions in depression, somatic symptoms, overemotionality, anxiety, rumination, and fatigue.

Discussion. In support of Henriques and Davidson's (1991) belief that hypoactivation of the left hemisphere results in an "approach deficit" and more withdrawal behavior, post-testing and interview data also documented that the patient had become less withdrawn, more active, sociable, and less distrustful. Eight and one-half month follow-up documented maintenance of changes. Continued exploration of left hemisphere beta protocols in treating depression, and of the combined use of neurofeedback with photic stimulation are encouraged.

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KEYWORDS. Neurofeedback, biofeedback, EEG, hypnosis, alternative medicine, complementary medicine, EEG biofeedback

INTRODUCTION

Considerable research, summarized by Davidson (1998a) has documented now that depression is associated with an activation difference between the right and left prefrontal cortex. More specifically, it has been shown that there is more right than left frontal activity in depression. Numerous EEG studies (e.g., Davidson, 1992, 1995, 1998b; Davidson & Fox, 1982, 1989; Fox & Davidson, 1986; Davidson, Chapman, Chapman & Henriques, 1990; Fox, 1991; Heilman, 1997; Reuter-Lorenz & Davidson, 1981; Schiff & McDonald, 1990; Tomarken, A.J., Davidson, R.J., & Henriquez, 1990) demonstrate that the left frontal area is associated with more positive affect and memories, and the right hemisphere is more involved in negative emotion. Therefore, the asymmetry wherein there is more left frontal alpha activity in depressed persons means that they may be less aware of positive emotions while at the same time being more in touch with the negative emotions that are associated with the right hemisphere. In addition, Henriques and Davidson (1991) believe that the left hemisphere is associated with approach behavior while the right hemisphere is involved in withdrawal behavior. Thus, when the left hemisphere is basically ''stuck'' in an alpha idling rhythm, there is more withdrawal behavior in addition to the deficit in positive affect. Although Baehr, Rosenfeld, and Baehr (1997) expressed the belief that to some degree this frontal asymmetry may represent a state marker of depression, it has primarily been discussed as reflecting a biological or trait marker of a vulnerability (Henriques & Davidson, 1990, 1991) to depression.

However, more recently Davidson (1998b) clarified his position, indicating that such an asymmetry is ''neither necessary nor sufficient for the production of a specific type of affective style or psychopathology,'' but that ''differences in prefrontal asymmetry are most appropriately viewed as diatheses that bias person's affective style, and then in turn modulate an individual's vulnerability to develop depression'' (p. 608). Rather than being a unitary model of depression, he believes the asymmetry simply predicts a vulnerability to depression so that when negative life events occur over a prolonged period of time to such a person, there is an increased probability of them becoming depressed. Davidson (1998b) stated:

> However, as a contributory cause, we would (1) not expect all subjects with relative right-sided anterior activation to be depressed; and (2) not expect all depressed subjects to show relative right-sided anterior activation, because we assume that there are multiple, complex routes to this disorder. (p. 608)

Based on this substantial research on frontal alpha asymmetry in depression, Rosenfeld (1997) developed a neurofeedback protocol for modifying this asymmetry. Given the research, this ALAY (standing for alpha asymmetry; F4 − F3/F3 + F4) protocol stands on very firm theoretical ground and the preliminary results from case studies (Rosenfeld, Cha, Blair, & Gotlib, 1995; Baehr et al., 1997) are encouraging. There may, however, be other neurofeedback protocols that may also correct or assist in overcoming the effects of a frontal alpha asymmetry, resulting in the successful treatment of depression.

Background

The patient was a 44-year-old man with a presenting problem of retarded ejaculation. Assessment revealed a severe level of depression (MMPI depression scale T score of 97). Thus, it was felt to be inappropriate to treat the patient's sexual problem until his depression was resolved. The patient had previously been on Prozac without significant positive results, and was on Wellbutrin at the time of testing.

He described himself as a type-A personality who found it hard to relax, was compulsive, perfectionistic, self-critical, and had low self-esteem. At times he struggled with insomnia. He suffered with somatic symptoms of diarrhea once weekly, headaches twice a week, and often had a general ''achy, lethargic feeling all over.'' He usually felt tired and fatigued. He also admitted that to a considerable degree he was a loner, who was distrustful of people and rather withdrawn. Despite the severe depression, he was not suicidal. These symptoms were compatible with his MMPI elevations on scales 1, 2, 6, 7, 8, 0, and his low score on the ego-strength scale.

Evaluation Instruments

Six different objective measures of change were utilized during treatment. These included: (1) a physiological measure of frontal alpha asymmetry at electrode sites F4-F3, referenced to Cz, using the ALAY protocol on the Lexicor NRS-24 EEG unit; (2) the Minnesota Multiphasic Personality Inventory; (3) the PANAS (Positive and Negative Affect Scale) (Watson, Clark & Tellegen, 1988), which provides ratings of both positive and negative affect; (4) the Beck Depression Inventory; (5) the Inventory to Diagnose Depression; and (6) a 10 centimeter visual analogue rating scale with the two ends defined with zero representing ''the worst you have ever felt,'' and ten representing ''the best you have ever felt.''

TREATMENT

The patient was hypnotized in two interviews and a self-hypnosis tape was made for him to use at home which was focused only on stress management. Although a formal hypnotizability scale was not utilized, he was estimated to be of mid-range hypnotic responsiveness based on the elicitation of ideomotor phenomena and his ability to develop a glove analgesia, but not complete anesthesia (Hammond, 1998). Since antidepressants were not producing beneficial results, the research described above on frontal alpha asymmetry as a biological marker for depression was discussed with him. We then performed an electrophysiological evaluation of his frontal alpha activity at F3-F4 referenced to Cz, using software from the ALAY protocol. A pilot study by Baehr, Rosenfeld, Baehr, and Earnest (1998) suggested that depressed patients score 55% or less, while non-depressed patients score 60% or higher. The mean score of this patient for three two-minute baselines was 36.1%. His Rose score (amplitude + or −) was −5.3.

Neurofeedback was presented to him as a possible experimental treatment option. The patient felt positive about this, and following informed consent, we began using the ALAY protocol (Rosenfeld, 1997; Rosenfeld et al. 1995; Baehr et al. 1997) in an effort to correct frontal alpha asymmetry using the Lexicor NRS-24 neurofeedback unit. All impedances were kept to 4000 ohms or less, and the threshold was set at zero so that his scores below zero represented greater left than right alpha magnitude, while his scores above zero demonstrated reverse asymmetry. Scores at the end of sessions represented the mean value for the entire session as a mean asymmetry score. A bell tone provided reinforcement in the session whenever the asymmetry score exceeded zero.

His first three neurofeedback sessions using the ALAY protocol were discouraging for him because he experienced considerable difficulty in changing his asymmetry score. In fact, his scores deteriorated in the second and third sessions. His percentage scores (the percent of the time that the asymmetry is above the zero threshold) at the beginning and end of these three sessions were, respectively: 41.7% and 44.8%; 30% and 28.4%; and 41.7% and 33.6%. Thus, at the end of three sessions of training, his asymmetry had actually changed in a negative direction an average of −2.2%. His "Rose" scores (the amplitude + or −) at the end of a two minute baseline and at the end of each of these three sessions were 2.7 and −1.3, −6.5 and −7.7, and −5.0 and −4.8. The movement of a Rose score in a negative direction is consistent with a worsening of symptoms, which appeared to be the case in his initial ALAY neurofeedback sessions. His baseline scores for the three sessions averaged −8.8, and his mean scores at the completion of the three neurofeedback sessions averaged −13.8. Davidson (1998b) recently emphasized the importance of finding test-retest stability over a period of

weeks in the frontal alpha asymmetry. Only when this has been found in two assessments separated by a few weeks, has it been discovered to predict reactivity to emotional elicitors. This patient certainly met Davidson's careful criteria.

As a consequence of his discouragement, and operating on the assumption that "many paths lead to Rome," we switched protocols. We began using a neurofeedback unit called the Roshi. It utilizes photic stimulation which can be set so that the frequency of photic stimulation varies depending on the patient's existing dominant brainwave. Another unique feature of the Roshi is that it also trains simultaneously at two electrode sites. The Roshi system samples at 128 samples/second and amplifies the raw EEG at a gain of 82000 in each channel. The filter bandwidth is on the order of 2 to 42 Hz, at -3 dB points. These signals are, then, subjected to a Fast Fourier Transform (FFT) that is performed at the 128 samples/second data rate. Although the use of a cosine-tapered window is implemented, it can be optionally selected by the user, at the output of the FFT, not on the raw EEG input. These techniques bolster the power of Roshi's unique audio-visual stimulation (AVS) system. These Fourier RMS magnitudes are, then, subjected the standard 1 to 4 Hz (Delta), 4 to 7 Hz (Theta), 8 to 13 (Alpha), 12 to 15 Hz (SMR or low beta) and 15 to 20 Hz (Beta) frequencies. An added uniqueness of the Roshi system is in its use of interhemispheric Fourier products in its training modalities, with very low feedback latency. The A/D conversion rate is 128 samples per second.

Electrodes continued to be placed at F3 and F4, and we were reinforcing 15-18 Hz while simultaneously inhibiting alpha and theta frequencies. For the initial eight to ten minutes of a session, we used Roshi's "complex (adaptive)" light stimulation. This consists of feeding back to the left half of each eye (through varying the frequencies of photic stimulation) information from the F3 (left) electrode site, and vice versa. This is, however, undoubtedly less than perfect in practice since slight lateral eye movements could cause the information projected to the hemiretinas to really be projected to both sides of the brain simultaneously. The theory of Chuck Davis, the designer of the Roshi, is that half of each eye sees separate information which is reflected in the EEG, which causes the brain to seek to correct for the apparent error, increasing brain flexibility. In clinical practice, it is usually found that the result of using complex (adaptive) stimulation is a flattening of the spectrum. Following this brief stimulation, for the majority of each training session a program was used called Beta Max. In this program, the photic stimulation is now different, varying from moment to moment, pulsing on the peak frequency within the 15-18 Hz range. Alpha and theta frequencies continued to be inhibited, while beta activity in the 15-18 Hz range was reinforced.

The results were rapid and fairly dramatic. After five sessions the patient

was reporting that he did not feel depressed any longer. Our initial training on Roshi consisted of twelve sessions of training at F3-F4 as specified above. All electrode impedances were always kept at 5000 ohms or less, and a left ear lobe ground and right mastoid reference were used. As the patient came in for this thirteenth session on Roshi, he reported that in the five days since his last session, he had felt agitated and jittery. In response, during this session we trained on a program called SMR Max at Cz and C4 to promote more calming. This program provides photic stimulation, moment to moment, that is focused on the patient's peak frequency in the 12-15 Hz range, reinforcing this SMR band while simultaneously inhibiting the ranges from 4 to 12 Hz.

Two days after this SMR session, as he came for another session, he reported that he had remained very calm since our last training. Subsequently, in an attempt to calm his rumination and protect against any further agitation, we switched the training procedure. For the first twenty-five minutes of sessions, we used the Beta Max program at Fp1 and C3, similar to what the Othmers have utilized for addressing rumination. Then, for the final ten minutes of training, we used the SMR Max program with training at C3-C4, to promote greater calming. This continued for the next thirteen sessions.

The final four sessions of training prior to post-testing occurred with only the Beta Max program at F3 and at F9. The following rationale was the basis for this change. Knowing that the orbitofrontal cortex seems to have involvement in depression (Davidson & Irwin, 1999) and other problems, the author had been wondering about the feasibility of using nasopharangeal electrodes for a few sessions of training. In visiting with the director of our EEG lab in the University of Utah School of Medicine, she indicated that they had abandoned nasopharangeal electrodes and had come to believe that another placement was just as effective in accessing orbitofrontal and temporal areas, and yet far less uncomfortable. They have begun placing electrodes just to the left and very slightly above the outside corner of the eye on the temple. Further investigation found that research by Gambardella et al. (1998) documented that the F9-F10 positioning of electrodes, which they referred to as a latero-orbital placement, provided comparable information to anterior temporal electrodes in detecting temporal epileptiform activity. They, therefore, recommended their use in day-to-day evaluation of patients, instead of basal electrodes (sphenoidal, nasopharyngeal, zygomatic, or anterior temporal). This was the rationale for placing an electrode beside the left eye (F9) along with the F3 placement, although it is possible that such a placement may provide more information about the inferior lateral prefrontal cortex than the orbitofrontal cortex and is susceptible to eye movement artifact.

RESULTS

In an effort to evaluate physiological data comparable to the first three sessions with the ALAY protocol, there were four sessions during which the patient was attached to both the Roshi for neurofeedback training, and to the Lexicor NRS-24 for purposes of physiological monitoring with the ALAY protocol (although the patient was not receiving feedback from the latter protocol). In contrast to the −2.2% Rose score change in asymmetry (which is the percent of time the asymmetry was above the zero threshold) in treatment in the first three sessions with the ALAY protocol, the mean change from the beginning to the end of these four sessions of training with Roshi was a +.6 (individual session Rose scores were −3.4, −8.3, 3.6, and 10.5). Whereas his percentage scores (the percent of the time that the asymmetry is above the zero threshold) at the end of the initial 3 ALAY protocol sessions averaged 35.6, the average post-session percentage score was 49% for the four Roshi sessions.

Table 1 summarizes the non-physiological outcome measures administered at varying periods during and at the conclusion of treatment. Pre-testing was completed by the time of the first training session with the ALAY protocol (2-23-99). Between the first and second (3-4-99) testing, treatment consisted of three sessions utilizing the Rosenfeld alpha asymmetry protocol, and one session of beta training with the Roshi. Some improvement was already apparent on all measures, which may represent treatment responses

TABLE 1. Psychometric Evaluations of Depression

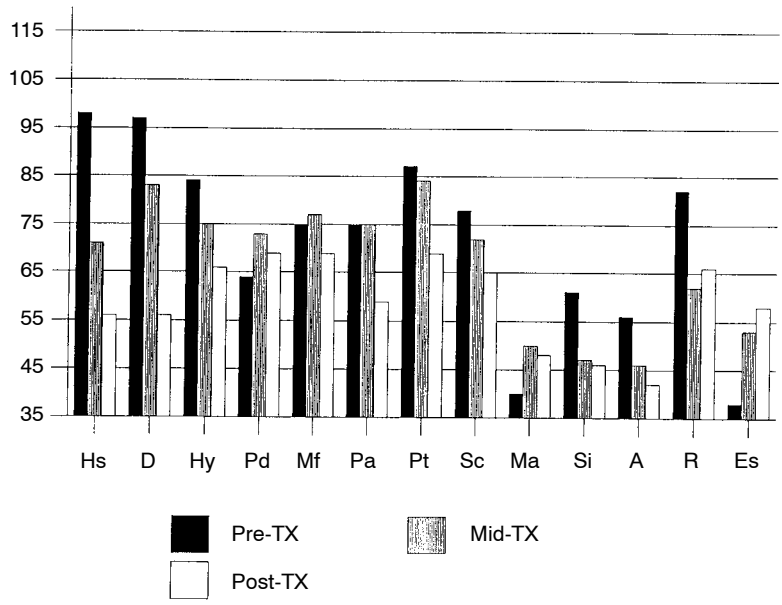
Date	VAS Rating	PANAS	Inventory to Dx Depression	BDI	MMPI 2 Scale
2-23-99	3.0	+ = 2.4 − = 2.9	28	21	97 t-scores
3-4-99	4.5	+ = .21 − = 1.5	20	15	
3-23-99	7.7	+ = 2.7 − = 1.3	8	6	83 t-scores
4-8-99	7.6	+ = 2.9 − = 1.2	5	7	
4-29-99	9.1	+ = 3.3 − = 1.2	1		
6-29-99	9.1	+ = 4.3 − = 1.1	0	2	56 t-scores

or placebo effects. It can be stated, however, that the patient was encouraged following his first Roshi session. Instead of the asymmetry increasing in a negative direction, it improved 3%, and he reported feeling positive.

The third testing time (3-23-99) occurred following five more sessions on Roshi, and substantive improvement was now apparent, although the MMPI (Figure 1) continued to remain significantly elevated. The fourth tests were administered after five more Roshi training sessions (4-8-99), representing a total of eleven Roshi sessions and three ALAY protocol sessions. Slightly more improvement was seen at that time. The fifth testing (4-29-99) occurred following another five training sessions using the Roshi, and still further improvement is seen across measures. The final testing (6-29-99) occurred after a total of thirty Roshi sessions (and 3 ALAY protocol sessions). By this time, his visual analogue scale had improved from 3.0 to 9.1. His ratings of positive feelings (on the PANAS) increased 79%, while his ratings of negative feelings decreased by 62%. Scores on the Inventory to Diagnose Depression, an instrument viewed very favorably by academic researchers, improved from 28 to 0, and the BDI improved from 21 to 2.

The depression scale of the MMPI improved from T scores of 97 to 56. This is particularly impressive since many of the case reports (Baehr et al.,

FIGURE 1. MMPI T-Scores Before, During, and After Neurofeedback



1997) using the ALAY protocol have involved relatively mild depression in the 62-64 T score range. Other improvements in addition to depression are also seen in the post-test MMPI, and were confirmed in interviews. Somatic symptoms (gastritis, headaches, achiness, and preoccupation with health) dramatically improved, as did his overemotionality, anxiety and rumination, and fatigue. The dramatic changes on the MMPI in areas other than depression mirror the kinds of psychological changes noted in previous case studies (Baehr et al. 1997) using the ALAY protocol to treat depression. Co-workers had spontaneously commented on how much more relaxed and less tense he seemed. The MMPI changes are interesting in terms of Henriques and Davidson's (1991) belief that hypoactivation of the left hemisphere results in an "approach deficit" and more withdrawal behavior. Following neurofeedback that was focused on activating the left hemisphere, MMPI and interview data demonstrated that the patient had become less withdrawn, more active, sociable, and less distrustful. There literally was more "approach" behavior. All of this was in spite of the fact that two weeks before his final testing, his primary relationship had broken up because of his partner's unwillingness to remain monogamous and committed. In the ten days following the last testing, the patient was seen for two more follow-up neurofeedback reinforcement sessions using Roshi. On telephone follow-ups nine weeks later and eight and one half months later, the patient was not excited about the possibilities of taking lengthy psychological tests again, but he reported that he was maintaining his changes and that depression was not and had not been a problem.

In his last neurofeedback session (#32), physiological measurements were still indicative of an alpha asymmetry, with greater alpha power in the left anterior region. However, this finding simply replicates several studies (e.g., Allen, Iacono, Depue, & Arbisi, 1993; Gotlib, Ranganath, & Rosenfeld, 1999; Henriquez & Davidson, 1990; Kwon, Young, & Jung, 1996) where follow-ups discovered that even after remission from depression, the frontal alpha asymmetry remained, suggesting that this asymmetry may be more trait than state-dependent. But, we should not assume that the original alpha asymmetry may not, in fact, change given longer term treatment or with the ALAY protocol, since neurofeedback treatment studies have previously reported enduring post-treatment changes in EEG patterns (e.g., Sterman, 2000). It must also be acknowledged that we do not know the role that enhanced cerebral blood flow from either the light stimulation and/or neurofeedback may have played in alleviating depression and other symptoms.

DISCUSSION AND CONCLUSIONS

This paper has added to the literature a case report of the successful treatment of severe depression and psychological disturbance using neuro-

feedback. However, in this case, equipment was used that combined photic stimulation with neurofeedback and the protocol relied primarily on left hemisphere beta training. Previous case reports (Rosenfeld et al. 1995; Baehr et al. 1997) using the Rosenfeld ALAY protocol to address the frontal alpha asymmetry have likewise reported successful treatment of depression, and in a similar although perhaps somewhat longer length of time. It is possible that this case might have also ended in a successful outcome had we continued to use the ALAY protocol. However, the author suspects that the patient might well have dropped out of neurofeedback treatment if encouraging improvements had not occurred within another two or three sessions. It is my belief, from carefully questioning the patient after neurofeedback sessions, that his difficulty in responding to the ALAY protocol stemmed from his excessive rumination. Just as he tended to do in sexual situations, during neurofeedback he was continually worrying about his performance. He was perfectionistic, self-critical, and a worrier. Incidentally, his problem with retarded ejaculation remains untreated since he is still not involved in a committed relationship.

My psychotherapeutic approach is one of eclecticism, emphasizing flexibility and the importance of having a variety of potential interventions to assist patients in overcoming problems. This philosophy seems to also have potential value in the use of neurofeedback protocols and equipment. The author has had considerable experience in using traditional neurofeedback equipment with patients and then having them work on the Roshi, which trains on two electrode sites simultaneously and uses photic stimulation to assist in guiding the brain toward the desired goal. The subjective experience of the large proportion of these patients is very similar to the response of this patient. The Roshi feels more powerful. A not uncommon expression following a session on the Roshi, after having experienced many training sessions on more traditional neurofeedback equipment, is that the first machine was good, ''But, this is great!'' It has been found that when patients have trained on traditional equipment and then on the Roshi, the great majority do not want to return to other equipment. In the case of this patient, he was encouraged by his very first Roshi session because he subjectively felt that more was occurring. Then, upon learning that during his first Roshi session that his asymmetry score had improved rather than getting worse, this provided even further reinforcement. Within five training sessions on Roshi, the patient was reporting that he was feeling less depressed and more energetic. Such subjective reports and objective testing from patients encourage continued exploration of the potentials of combining other technology with neurofeedback.

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