Journal of Adult Development, Vol. 12, Nos. 2/3, August 2005 (© 2005)
DOI: 10.1007/s10804-005-7029-5

Neurofeedback Treatment of Depression and Anxiety

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A robust body of research documents that there are biological predispositions that often exist for depression, anxiety, and obsessive–compulsive disorder. However, new research has shown that medication is only mildly more effective than placebo in the treatment of these problems. In treating these conditions, neurofeedback (EEG biofeedback) may offer an alternative to invasive treatments such as medication, ECT, and intense levels of transcrancial magnetic stimulation. This paper reviews the neurofeedback literature with these problems, finding particularly positive research support for the treatment of anxiety disorders. New findings on the neurofeedback treatment of depression are presented.

KEY WORDS: Neurofeedback; EEG biofeedback; QEEG; depression; anxiety; OCD.

INTRODUCTION

Biological Substrates of Depression, OCD, and Anxiety

Speaking as a psychologist, I think that it is not uncommon for us to minimize and, therefore, neglect the biological aspects of mental health disorders, with the exception of schizophrenia and bipolar disorder. Our training is primarily in psychological interventions rather than in directly modifying how the brain functions. However, as I have reviewed elsewhere (Hammond, 2003), there is strong evidence that obsessive–compulsive disorder has a significant biological component. There can also be strong biological predispositions to anxiety and panic disorder (e.g., Heller, Etienne, & Miller, 1995, 1997; Wiedemann et al., 1999).

A robust body of research has been summarized by Davidson (1998a) documenting that depression is associated with an activation difference between the right and left prefrontal cortex. A large number of EEG studies, summarized by Davidson (1992, 1995, 1998a), have demonstrated that the left frontal area is associated with more positive affect and memories, and the right hemisphere is more involved in negative emotion. When there is a biological predisposition to depression, there is a frontal asymmetry with more left frontal alpha activity, meaning that the left frontal area is less activated. This means that such individuals may be anticipated to 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. It has also been demonstrated (Henriques & Davidson, 1991) that the left hemisphere is associated with approach motivation and behavior whereas 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. Even the infants of depressed mothers have been found to display this same reduced left frontal EEG activation (Dawson, Grofer Klinger, Panagiotides, Hill, & Spieker, 1992; Dawson, Grofer Klinger, Panagiotides, Spieker, & Frey, 1992), even as young as 3–6 months (Field, Fox, Pickens, & Nawrocki, 1995) and 1 month of age (Jones, Field, Fox, Lundy, & Davalos, 1997).

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Baehr, Rosenfeld, and Baehr (1997) and Askew (2001) have expressed the belief that this frontal asymmetry may represent a state marker of depression, as well as reflecting a biological or trait marker of a vulnerability (Henriques & Davidson, 1990, 1991) to depression. Askew (2001) found a strong correlation between alpha asymmetry scores and the Beck depression Inventory ($p < 0.0001$) and on the MMPI-II Depression Scale ($p < 0.0001$). Davidson (1998b) expressed his belief that such an asymmetry is not necessary or sufficient for the production of a specific type of affective style or psychopathology, but that differences in prefrontal asymmetry may be most appropriately viewed as diatheses that bias a person's affective style, and then in turn modulate someone's vulnerability to develop depression. Davidson (1998b) does not subscribe to a strictly biological model of depression, but he believes that the asymmetry does predict 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. Not all persons with this frontal alpha asymmetry will be depressed, and someone can experience negative life events and still become depressed in the absence of this asymmetry. This EEG asymmetry is best seen when the EEG is examined with an average reference or a reference on the vertex at Cz (Baehr et al., 1997; Davidson, 1998a,b; Rosenfeld, Cha, Blair, & Gotlib, 1995).

Neurofeedback

EEG biofeedback (neurofeedback) has been found to be effective in modifying brain function and producing significant improvements in clinical symptoms in several clinical areas, including epilepsy, ADD/ADHD, learning disabilities, and head injuries. For example, Sterman (2000) comprehensively reviewed the literature on the neurofeedback treatment of uncontrolled epilepsy. Overall, this literature documented that 82% of the most severe, uncontrolled epileptics demonstrated a significant reduction in seizure frequency, with an average of a 70% reduction in seizures. Two studies even measured sleep EEG pre- and post-training and documented significant normalization of brain activity even when patients were asleep. Another new controlled study (Kotchoubey et al., 2001) validated the effectiveness of neurofeedback compared to medication and placebo. These neurofeedback studies meet the criteria for being both an efficacious and specific treatment, as established by the American Psychological Association Clinical Psychology Division (Chambless & Hollon, 1998; Chambless et al., 1998).

Monastra's (2002) recent research found neurofeedback to be significantly more effective than ritalin in changing ADD/ADHD, without having to remain on drugs. Other studies (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003) have found comparable improvements with 20 h of neurofeedback training (forty 30-min sessions) to those produced by ritalin, even after only twenty 30-min sessions of neurofeedback (Rossiter & LaVaque, 1995).

NEUROFEEDBACK TREATMENT OF ANXIETY AND DEPRESSION

Neurofeedback for Anxiety

Moore (2000) reviewed the literature on neurofeedback treatment for anxiety disorders. He reviewed eight studies of generalized anxiety disorder (GAD), three with phobic anxiety disorder, two studies of obsessive–compulsive disorder, and one published report with post-traumatic stress disorder (PTSD). There were several problems with this literature. One problem in the literature is that most studies only utilized very brief training. For instance, in the GAD studies that listed length of training, it only averaged 3.2 h! As a clinician, I will most commonly utilize 7–12 h of neurofeedback training with anxiety problems. Nonetheless, seven of the eight studies produced positive changes in clinical outcome.

The finest studies were the three studies of phobic (test) anxiety (Garrett & Silver, 1976), that included random assignment, alternative treatment control groups, and a wait-list control group. In one experiment, the group receiving alpha EEG enhancement training produced 33% more alpha post-treatment, and all three feedback groups demonstrated significant reductions in test anxiety, while the untreated control group and the relaxation training group experienced no significant reduction. In another experiment, participants received phases of alpha enhancement training and EMG biofeedback training. The alpha training increased alpha production from 64 to 78%, and anxiety scores dropped significantly ($p < 0.001$) for this combined treatment

group compared to a non-treatment group. Thus, according to APA Clinical Psychology Division criteria for efficacious treatments, neurofeedback for phobic anxiety qualifies for the status of possibly efficacious. Moore's review (2000) also concluded that a placebo effect was certainly present in these neurofeedback studies, but that alpha and theta enhancement training provided additional effects beyond placebo and are effective treatments of anxiety disorders.

There were two studies that was not reviewed by Moore (2000). Passini, Watson, Dehnel, Herder, and Watkins (1977) compared 25 anxious alcoholics with a matched control group before and after 10 h (over a 3 week period) of alpha neurofeedback training. Alpha neurofeedback training produced significant ($p < 0.001$) changes in state and trait anxiety compared with controls. Patients receiving neurofeedback training increased their eyes-closed alpha production from 38 to 55%, while controls dropped slightly. In an 18-month follow-up (Watson, Herder, & Passini, 1978), essentially identical results were still found, indicating that the anxiety changes from alpha neurofeedback were enduring. A new randomized, blinded, controlled study (Egner & Gruzelier, 2003) at London's Royal College of Music evaluated the ability of alpha/theta neurofeedback to enhance musical performance in very high level musicians when they were performing under stressful conditions. When compared with alternative conditions (physical exercise, mental skills training, Alexander Technique training, beta1 neurofeedback, and SMR neurofeedback), only the alpha/theta neurofeedback group resulted in enhancement of real-life musical performance under stress.

Two neurofeedback studies focused on chronic PTSD. In a randomized, control group study, Peniston and Kulkosky (1991) added thirty 30-min sessions of alpha/theta EEG biofeedback training to the traditional VA hospital treatment provided to a group of 15 PTSD Vietnam combat veterans, and compared them at follow-up with a contrast group of 14 veterans who only received traditional treatment. On 30-month follow-up, all 14 traditional treatment patients had relapsed and been rehospitalized, while only 3 of 15 neurofeedback training patients had relapsed. Although all 14 patients treated with neurofeedback had decreased their medication requirements by follow-up, among traditionally treated patients, only one patient decreased medication needs, two reported no change, and 10 required more psychiatric medications. On the MMPI, neurofeedback training patients improved significantly on all 10 clinical scales—dramatically on many of them—while there were no significant improvements on any scales in the traditional treatment group. An additional study, not originally reviewed by Moore (2000), was done by Peniston, Marrinan, Deming, and Kulkosky (1993). They randomly selected 20 chronic PTSD Vietnam veterans, who also had alcohol abuse, from a VA hospital population. They were treated with thirty 30-min sessions of alpha/theta neurofeedback training. On 26-month follow-up, only 4 of the 20 patients reported a few (1–3) instances of recurrence of nightmares/flashbacks, and the other 16 patients had no recurrence of PTSD symptoms.

Moore (2000) reviewed two published studies of OCD that used alpha enhancement training, without positive results. However, these studies utilized a naive treatment approach of only doing alpha enhancement training, and literature since that time has shown that there are at least three subtypes of EEG patterns that are found in OCD. More recently, I have reported on successful treatment with lengthy follow-ups of three consecutive cases of OCD, utilizing protocols that were individualized through using a quantitative EEG assessment. In the first publication, (Hammond, 2003) scores on the Yale–Brown Obsessive–Compulsive Scale (YBOCS) and the Padua Inventory normalized following treatment. The patients showed 3.7 and 3.0 standard deviation improvements on the YBOCS. This is particularly significant because a meta-analysis (Ackerman & Greenland, 2002) of 25 drug studies found that even the most effective pharmacologic treatment for OCD (clomipramine) only produced an average treatment effect on the Y-BOCS of a 1.33 standard deviations improvement (uncorrected for placebo effects), and about one-half that much improvement across studies with Prozac. Improvements were also documented with an MMPI, with follow-ups of the two cases at 15 and 13 months after treatment. Figure 1 shows the pre–post improvements in one of these cases. Maintenance of change was also externally validated through contacts with family members. I have now followed-up the third case (Hammond, 2004) for 10 months. Figure 2 displays his MMPI pre-treatment, mid-treatment, and at the conclusion of treatment. It may be seen that his Pt scale decreased from 115 *t*-scores to 60 *t*-scores. His Y-BOCS improved from his original score of 16 to a score of 3, representing a 2.2 standard deviation improvement. He had originally scored 6 on the compulsions subscale, and now scored zero, and his score had improved from 10 to 3 on the obsessions

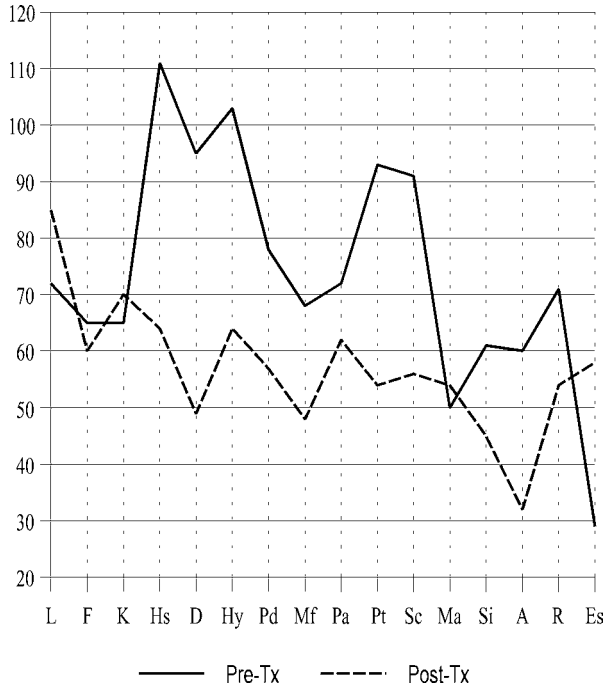

Fig. 1. MMPI Changes in an OCD case after 25 h of neurofeedback.

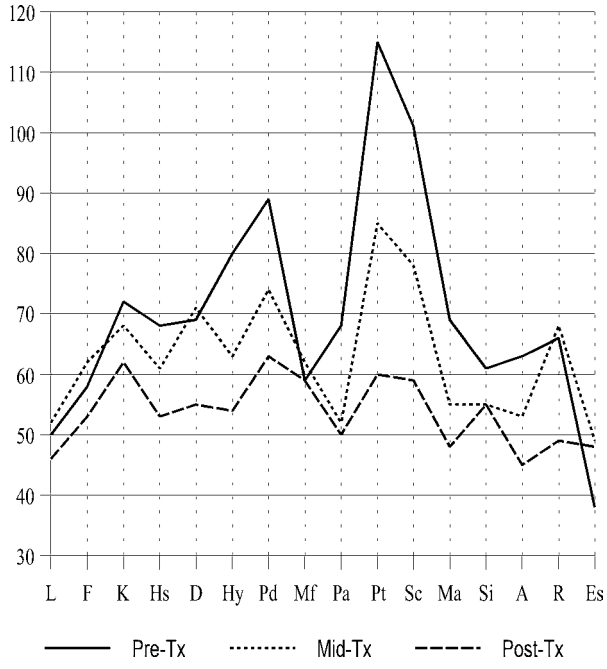

Fig. 2. Pre–Post MMPI changes in a case of obsessional OCD.

subscale. Once again, external validation of improvements and their maintenance was obtained by talking with his family.

Neurofeedback for Depression

Based on the large volume of research reviewed earlier that validates the role of the frontal alpha asymmetry in depression, Rosenfeld (1997) developed a neurofeedback protocol for modifying this asymmetry. His ALAY (standing for alpha asymmetry; F4−F3/F3+F4, with a reference electrode at Cz) protocol rests on very firm theoretical ground and the preliminary results from case studies (Rosenfeld et al., 1995; Baehr, Rosenfeld, & Baehr, 2001, 1997) are encouraging, although no controlled research has yet been completed. There have been long-term follow-ups, however. Baehr et al. (2001) reported on 1–5 year follow-ups on patients treated with the Rosenfeld protocol, documenting that the substantial changes were not only enduring, but also that the frontal alpha asymmetry had not only changed, but remained eliminated on long-term follow-ups. This is particularly significant because a variety of studies (Allen, Iacono, Depue, & Arbisi, 1993; Gotlib, Ranganath, & Rosenfeld, 1999; Henriques & Davidson, 1990; Kwon, Youn, & Jung, 1996) have found that following drug treatment that produced remission of the depression, the frontal alpha asymmetry remained, indicating a continued vulnerability to future depression.

Several years ago (Hammond, 2000) I likewise reported a case study with an eight and a half month follow-up of the effective alleviation of severe depression using my own neurofeedback protocol for modifying frontal alpha asymmetry. This protocol utilizes electrode sites Fp1 (on the left forehead) and F3, which is approximately 2.5–3 inch. straight above Fp1. In this protocol, we inhibit slow alpha and theta activity, while reinforcing 15–18 Hz beta for the first 20–22 min of each training session, after which the frequency band being reinforced is decreased to 12–15 Hz during the final 8–10 min of each session. Since the publication of the original report, I have continued to use this same protocol for the treatment of depression.

A new sample reported in this paper consists of nine consecutive, white, middle class (mean age 43.5; range 34–50 years) patients. Informed consent was obtained from all patients, all of whom presented with a primary complaint of depression, which was

confirmed through administration of the Minnesota Multiphasic Personality Inventory. The only other selection criterion was that they were each screened with the Rosenfeld protocol for the presence of the frontal alpha asymmetry associated with a predisposition to depression. Rosenfeld (Baehr et al., 2001) has found that percentage scores greater than 60 indicate that there is not a predisposition to depression, while scores of 58 or less indicate the presence of a predisposition. The mean percentage score for this sample was 40.05, and the mean of this sample on the MMPI Depression scale was 93.75 *t*-scores. Whereas patients in drug studies are often more moderately depressed, 7 of the 8 patients in this series were judged to be seriously to severely depressed, with only one that was moderately depressed. In contrast, the case reports cited earlier (Baehr et al., 1997, 2001) that used the Rosenfeld neurofeedback protocol involved relatively mild depression in the 62–64 *t*-score range on the MMPI, with an percentage score of only 51.3.

Eight patients completed training, requiring an average of 20.75 thirty-minute sessions (10.4 h) of neurofeedback, with no other psychotherapy provided. Seven of eight patients made very substantial improvements, and one dropped out after five sessions because he was too busy. The drop-out showed signs of questionable motivation from the beginning, seeming to be in treatment primarily to please his wife and daughter. Many of the patients were on medication at the time of initial testing, but were no longer on medication at the completion of treatment.

Pre–post changes on the MMPI may be seen in Fig. 3, with a mean decrease in the depression scale of 28.75 *t*-scores. One patient improved from severely depressed to normal and two progressed from being seriously depressed to normal. Three improved from severe to mild depression, and one improved from moderately depressed to mildly depressed. One case who was severely depressed only showed mild improvement. This was an individual who had lost his wife to cancer a year earlier and issues surrounding this loss seemed likely to need to be addressed, and he was referred for psychotherapy for these issues. Categorizing this last case and including the drop-out as failures, this represents 77.8% of cases who made significant improvements. The average length of follow-up for these cases was about 1 year, with a range from 2 years in two cases, to 4 months in the case of the individual who only mildly improved.

Fig. 3. Neurofeedback for depression: Average MMPI Pre-post changes for eight cases.

SUMMARY AND CONCLUSIONS

Through the years I found it irritating that psychiatrists tell patients that they have a "biological depression" without any objective validation, seemingly as a justification to then simply write a prescription. Yet, startlingly, pharmacologic treatment for depression (Antonuccio, Danton, DeNelsky, Greenberg, & Gordon, 1999; Greenberg, Bornstein, Greenberg, & Fisher, 1992; Kirsch, Moore, Scoboria, & Nicholls, 2002; Kirsch & Sapperstein, 1998; Krisch, Scoboria, & Moore, 2002; Moncrieff, 2001; Walach & Maidhof, 1999), anxiety (Khan, Khan, & Brown, 2002), and obsessive–compulsive disorder (Ackerman & Greenland, 2002; Goodman, McDougle, & Price, 1992) has been found to be only mildly effective over and above placebo effects. Nonetheless, there is a robust literature validating that in fact there are biological predispositions that exist to depression, OCD, and anxiety.

Neurofeedback is an encouraging development that holds promise as a method for modifying biological brain patterns associated with a variety of

mental health and medical (e.g., stroke, head injury, effects of aging) disorders–particularly because unlike drugs, electroconvulsive therapy, and intense transcranial magnetic stimulation, it is non-invasive and seldom associated with even mild side effects. Echoing similar sentiments, Frank H. Duffy (2000), a Professor and Pediatric Neurologist at Harvard Medical School, recently stated that scholarly literature now suggests that neurofeedback "should play a major therapeutic role in many difficult areas. In my opinion, if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used" (p. v). "It is a field to be taken seriously by all" (p. vii). Despite the promise of neurofeedback, however, Duffy also noted the need for improved and higher quality research. This is particularly true in the application of neurofeedback to the treatment of anxiety and affective disorders.

Since the completion of the successive cases reported in this paper, I have personally treated approximately 15 additional patients suffering with depression, but sometimes without post-treatment testing and lengthy follow-up. It is my impression from both this case series and from my subsequent clinical experience that the use of this neurofeedback protocol results in significant, enduring improvements approximately 80% of the time when patients have the frontal alpha asymmetry that reflects a biological predisposition to depression. Most patients perceive a difference after between three to six 30-min sessions, feel a very significant improvement after 10–12 sessions, and usually complete treatment within 20–22 sessions. It has been impressive to me that this treatment not only improves depression that has commonly been medication resistant, but it also commonly reduces anxiety and rumination, increases ego-strength, and as one would expect from activating an approach motivation area of the brain, decreases withdrawal and introversion. However, this report and the other literature just reviewed on neurofeedback with depression only represent uncontrolled case series reports. Thus, though encouraging, these preliminary results now require controlled trials. Similarly, the preliminary reports on the neurofeedback treatment of OCD are intriguing and encouraging, but likewise require controlled research. The research that we have reviewed on the neurofeedback treatment of generalized and phobic anxiety, as well as PTSD, is more rigorous, warranting at least the status of being considered a probably efficacious treatment.

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