QEEG-Guided Neurofeedback in the Treatment of Obsessive Compulsive Disorder

D. Corydon Hammond, PhD

ABSTRACT. *Introduction*. Blinded, placebo-controlled research (e.g., Sterman, 2000) has documented the ability of brainwave biofeedback to recondition brain wave patterns. Neurofeedback has been used successfully with uncontrolled epilepsy, ADD/ADHD, learning disabilities, anxiety, and head injuries. However, nothing has been published on the treatment of obsessive-compulsive disorder (OCD) with neurofeedback.

Method. Quantitative EEGs were gathered on two consecutive OCD patients who sought treatment. This assessment guided protocol selection for subsequent neurofeedback training.

Results. Scores on the Yale-Brown Obsessive-Compulsive Scale and the Padua Inventory normalized following treatment. An MMPI was administered pre-post to one patient, and she showed dramatic improvements not only in OCD symptoms, but also in depression, anxiety, somatic symptoms, and in becoming extroverted rather than introverted and withdrawn.

Discussion. In follow-ups of the two cases at 15 and 13 months after completion of treatment, both patients were maintaining improvements in OCD symptoms as measured by the Padua Inventory and as externally validated through contacts with family members. Since research has

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found that pharmacologic treatment of OCD produces only very modest improvements and behavior therapy utilizing exposure with response prevention is experienced as quite unpleasant and results in treatment dropouts, neurofeedback appears to have potential as a new treatment modality.

KEYWORDS. Neurofeedback, EEG, biofeedback, quantitative EEG, OCD, obsessive-compulsive disorder

INTRODUCTION

Obsessive compulsive disorder (OCD) has a lifetime incidence in the range of 1% to 3% (Karno, Golding, Sorenson, & Burnam, 1988; Jenike & Brotman, 1984) and is widely regarded as having a strong biological basis. In a qEEG study, Kuskowski et al. (1993) discovered lower absolute power in delta, beta 1 and beta 2 bandwidths frontally and in the right hemisphere in OCD patients. They further discovered increased alpha relative power across temporo-parietal, central and occipital regions, along with decreased relative power in beta bands in the left frontal region. Their research additionally revealed severe right hemisphere hypoactivity, particularly in beta relative power. This is an interesting finding since treatment with clomipramine has been found to result in increased right hemisphere activity (MacCrimmon & Arato, 1991), which may help normalize the electroencephalogram (EEG) in OCD patients with this pattern.

On the surface these findings seem somewhat at odds with most PET and SPECT studies of OCD which have reported increased frontal blood flow and metabolism in mediofrontal, anterior cingulate, right frontal, and/or orbitofrontal areas (Baxter et al., 1987; Baxter, Phelps, & Mazziotta, 1988; Benkelfat et al., 1990; Harris, Pearlson, & Hoehn-Saric, 1993; Machlin, Harris, & Pearlson, 1991; Nordahl et al., 1989; Perani et al., 1995; Piacentini & Bergman, 2000; Rauch, Whalen, Dougherty, & Jenike, 1998; Rubin, Villaneuva-Meyer, & Anath, 1992; Sawle, Hymas, & Lees, 1991; Saxena, Brody, Schwartz, & Baxter, 1998; Swedo, Schapiro, & Grady, 1989; Szeszko et al., 1999). The neuroimaging findings converge in implicating a cortico-striato-thalamo-

cortical network. Resting studies of OCD seem to indicate hyperactivity in the orbitofrontal and anterior cingulate cortex and caudate nucleus, with this being attenuated under conditions of symptom provocation, and which attenuate following successful treatment (Rauch, 2000).

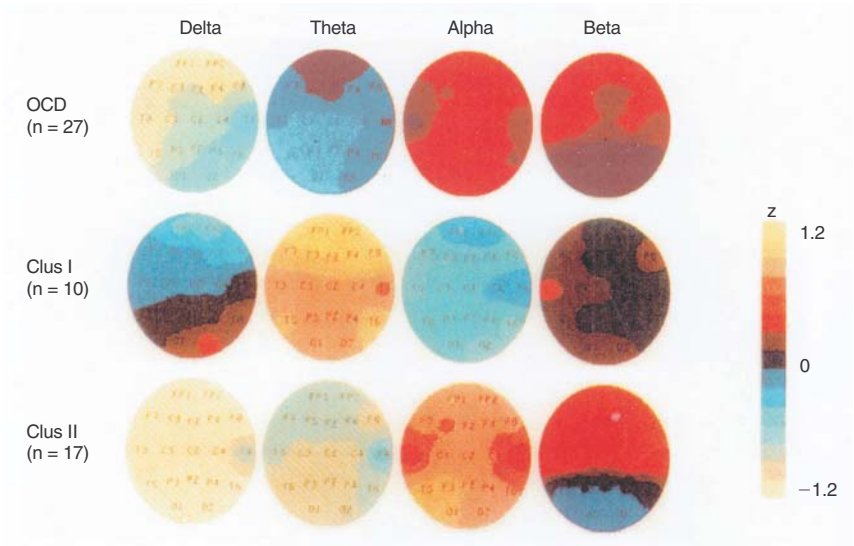
There are some evoked potential studies that are more congruent with neuroimaging findings. The anterior cingulate cortex may be involved with monitoring of behavior (Posner & Rothbart, 1998). An event related potential called error-related negativity (ERN) is a wave form that is associated with making mistakes or errors (Gehring, Coles, Meyer, & Donchin, 1990). It reflects the activity of a general error-processing system and one of the symptoms of OCD consists of excessive checking, rumination, and doubt–which amount to excessive response monitoring. The size of an ERN is sensitive to the size of an error. ERN has been localized as being generated from a single source in the medial frontal cortex (Dehaene, Posner, & Tucker, 1994; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Holroyd, Dien, & Coles, 1998; Luu, Collins, & Tucker, 2000). Gehring, Himle, and Nisenson (2000) found that the ERN was increased in OCD patients compared with matched controls, and the magnitude of the ERN was related with symptom severity. An fMRI study (Ursu et al., 2001) documented increased error-related activity in the anterior cingulate cortex in OCD patients, and this degree of activity was likewise correlated with severity of symptoms. Supporting Gehring et al. (2000), Hajcak and Simons (2002) also found a significantly larger Fz maximal negativity was associated with error responses in college undergraduates with OCD characteristics compared with students without such characteristics.

Some of the discrepancy between neuroimaging and qEEG studies may also stem from a bias in neuroimaging studies (Rauch, 2000); they often concentrate on only a limited number of brain areas, producing a potential confirmatory bias. Simpson, Tenke, Towey, Liebowitz, and Bruder (2000) conducted the first qEEG study under conditions of symptom provocation. Importantly, they found that only live exposure (and not imaginal exposure) to contaminants produced significant EEG changes. This is of importance since some OCD neuroimaging studies only used imaginal exposure. Simpson et al. (2000), using only vertex electrode sites, found a significant shift in the anterior-to-posterior topography of alpha power during live exposure compared with a control condition. Live exposure was associated with an increase in OCD symptoms and an increase in posterior relative to anterior alpha. No significant shifts occurred in the theta or beta bands. The observed changes were interpreted as reflecting a relative shift in brain activation from

posterior to anterior, which would be consistent with neuroimaging studies finding enhanced frontal activation during symptom provocation in OCD.

Other qEEG research has identified two subtypes of OCD patients (Mas, Prichep, John, & Levine, 1993; Perros, Young, Ritson, Price, & Mann, 1992; Prichep, Mas, & John, 1989; Prichep et al., 1993; Silverman & Loychik, 1990). Prichep et al. (1993) found one subgroup with excess alpha throughout most of the brain, but most excessive at T5, P3, O1 (which would coincide with findings by Kuskowski et al., 1993), and the frontal poles, along with a mild excess of beta in frontal, central and mid-temporal areas. Their other subgroup had a theta excess, most extreme throughout frontal areas and at posterior temporal electrodes. Figure 1 displays these subgroups. Theta abnormalities have also been reported by others (Insel, Donnelly, Lalakea, Alterman, & Murphy, 1983; Jenike & Brotman, 1984; Pacella, Polatin, & Nagler, 1944; Rockwell & Simons, 1947).

FIGURE 1. Group Average Topographic Maps (nose up) for Z Relative Power in the Delta, Theta, Alpha, and Beta Frequency Bands for the Two Neurometric Clusters of Patients with Obsessive-Compulsive Disorders



Reprinted from Prichep, L. S., Mas, F., Hollander, E., Liebowitz, M., John, E. R., Almas, M., DeCaria, C. M., & Levine, R. H. (1993). Quantitative electroencephalography (QEEG) subtyping of obsessive compulsive disorder. *Psychiatry Research*, *50*(1), 25-32, with permission from Elsevier Science.

Delayed onset of mu event-related desynchronization with preparation for movement and less post-movement beta (20 Hz) synchronization was reported by Leocani et al. (2001), a finding also found in Parkinson's disease (Pfurtscheller, Pichler-Zalaudek, Ortmayr, Kiez, & Reisecker, 1998). Leocani et al. (2001) suggested that a lower level of beta synchronization in OCD after a simple, self-paced movement raises a question about whether this may reflect the inability of these patients to inhibit themselves from compulsive actions. In this regard, lower P300 amplitudes in orbitofrontal areas in OCD patients (Malloy, Rasmussen, Braden, & Haier, 1989) likewise suggest impaired inhibitory mechanisms. Reduced motor cortical inhibition has also been found in this population with transcrancial magnetic stimulation (Greenberg et al., 2000). Perhaps related to these findings, Flor-Henry, Yeudall, Koles, and Howarth (1979) noted reduced left temporal (T3) variability in beta frequencies, but they did not examine frequencies lower than alpha.

Similar to Prichep, Mas, and John (1989), Prichep et al. (1993) and Mas, Prichep, John, and Levine (1993), Perros, Young, Ritson, Price, and Mann (1992) noted theta excess in 10 of 13 OCD patients in the 6.0-7.5 Hz range, predominantly in the left fronto-temporal area. They pointed out that such activity is frequently attributed to disturbances in deep midline structures (Gloor, 1976), which would be supported by some of the neuroimaging studies cited earlier. We might speculate that this theta subtype of OCD may be the group which has often produced some of the neuroimaging findings noted above. Relevant to one of the case reports which will be presented, another qEEG study has also pointed to abnormalities in the left posterior temporal area. Silverman and Loychik (1990) examined three siblings ranging from 23 to 29 years of age, all of whom had OCD. The left posterior temporal area was identified as abnormal in all three, both on qEEG evaluation and on auditory and visual evoked potentials, while the asymptomatic parents were entirely normal on all measures.

Treatments for OCD

Behavior therapy commonly uses exposure and response prevention techniques to treat OCD (Foa & Franklin, 2001), with claims that 76% to 86% of patients *who complete treatment* make improvements. In an earlier review by Foa, Steketee, and Ozarow (1985), they reported that in over 200 patients, 51% reduced their symptoms at least 70%. In Greist's (1990) review, however, he notes what I have experienced in

years past using a behavior therapy approach with OCD patients, which is that the greatest problem is that many patients dislike this treatment and fully one-quarter decline treatment or sabotage it with overt or covert avoidance. He also notes that behavior therapy has proven less successful with pure obsessional disorder (without rituals) and estimates the percent improvement in symptoms experienced following behavior therapy as 50%. Nonetheless, this psychiatrist's review estimates the degree of symptomatic improvement with serotonin drugs as only being 30%. Goodman, McDougle, and Price (1992) similarly found that symptom amelioration in OCD treatment with serotonin uptake inhibitors is about 35% on average, and that 50% of patients experience only partial symptomatic improvement.

The mean from four separate samples (Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989; Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989) of OCD patients on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989) is 24.7 (standard deviation = 6). A very recent (Ackerman & Greenland, 2002) meta-analysis of 25 drug studies found that with the most effective pharmacologic treatment for OCD (clomipramine) that the average drug treatment effect on the Y-BOCS was 10.64 (uncorrected for placebo effects), which is a 1.33 standard deviation improvement. In fluvoxamine (Prozac) studies, the mean Y-BOCS improvement was only 5.4 points. Interestingly, they discovered that the longer the clomipramine drug trial went on, the less improvement they found. Thus, 12-week trials had 5.78 less points of improvement than 10-week trials. Older patients also had less improvement on clomipramine. They found that the longer the pre-randomization period (during which some placebo responders are often dropped from inclusion in drug studies), the less improvement in drug response and in placebo response. The reviewers concluded that "the numerous side effects of clomipramine may have contributed to its greater effect size in placebo comparisons" (p. 315). This same conclusion was reached previously by the same group (Ackerman et al., 1996), as well as by Abramowitz's (1997) review. This is particularly relevant because recent reviews of antidepressant drugs studies (Antonuccio, Danton, DeNelsky, Greenberg, & Gordon, 1999; Kirsch & Sapperstein, 1998; Moncrieff, 2001) have identified that these studies commonly use inactive placebos which have no side effects, resulting in many patients and raters correctly discerning to which group they have been assigned, essentially unblinding the study. However, one review (Thomson, 1982) where an active placebo (e.g., atropine, which causes anticholinergic

side effects) was used found that only one in seven studies identified the antidepressant as superior to placebo. Another similar review (Moncrieff, Wessely, & Hardy, 1998) discovered that in only two of nine studies was antidepressant medication superior to an active placebo.

Despite the very modest effects of medication, there is evidence that qEEG has potential to assist in predicting medication response in treating OCD (Prichep et al., 1993). Those patients with excess alpha relative power (with some frontal and central beta excess) were found to respond positively 82% of the time to serotonin mediated antidepressants, whereas, the second subtype with increased theta relative power (with some alpha minima) failed to improve 80% of the time with SSRIs. Nonetheless, it must be emphasized that medication treated patients remain dependent on the medication, and in one study (Pato, Zohar-Kadouch, & Zohar, 1988), 89% of patients treated with clomipramine (Anafranil) relapsed after discontinuation of medication. Naturally, medications such as clomipramine also have numerous side effects which must be tolerated such as dry mouth, blurred vision, constipation, sweating, sedation, dizziness, and retarded ejaculation.

Psychiatry has also resorted to neurosurgical treatment for OCD, performing cingulotomies in cases that have proven resistant to both medication and a trial of behavior therapy. However, using a somewhat liberal criteria of having produced at least 35% improvement on the Yale-Brown Obsessive Compulsive Scale, such psychosurgery has only benefited from one-quarter to one-third of patients (Dougherty et al., 2002; Jenike et al., 1991), even with the confound that most of the patients continued receiving pharmacotherapy following cingulotomy. Rauch (2000) summarized, "For neurosurgical treatment of OCD, the overall rate of efficacy is quite modest, the costs are high, and the risks are considerable" (p. 169). It is thus apparent that current psychiatric treatment of OCD has significant limitations.

There also exists interesting neuroimaging research on pre-treatment brain characteristics that predict successful outcome. What is particularly interesting about these studies is that they show biological brain changes occurring following successful behavior therapy (exposure and response prevention), with such changes not occurring in persons who did not change in treatment or in normal controls. Schwartz, Stoessel, Baxter, Martin, and Phelps (1996) and Baxter et al. (1992) found significant changes in glucose metabolism in the right caudate following successful behavior therapy. They also found a significant correlation between change in left orbital frontal cortex with change in Y-BOCS scores, which has also been found by Swedo et al. (1992) in successful

medication treatment. Brody et al. (1998) reported normalization of left orbitofrontal cortex metabolism predicted positive treatment response to behavior therapy. They found that higher metabolism in the left orbitofrontal cortex predicted greater improvement with behavior therapy, but a worse outcome from fluoxetine treatment.

METHOD

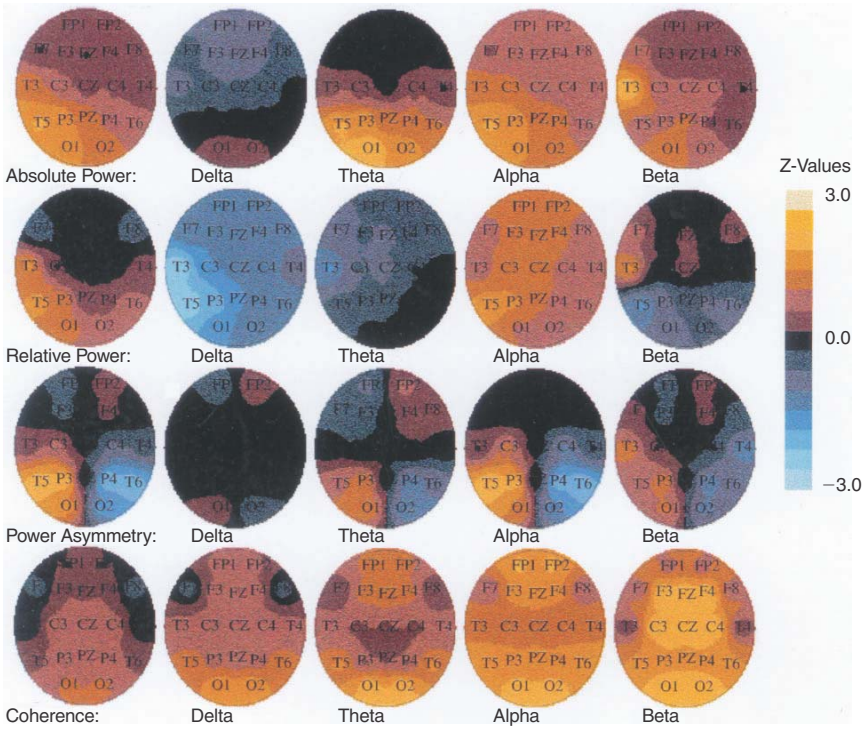
This paper will report on the treatment of two cases of OCD with neurofeedback. In both cases following the initial history taking, a quantitative EEG (qEEG) was done to evaluate brain function. Vigilance-controlled EEG was digitally recorded from the patients with Lexicor NRS-24 equipment with recording electrodes placed according to the 19 standard regions defined by the International 10/20 System of electrode placement, referenced to linked ears. All electrode impedance levels were below 3 Kohms, with no interelectrode differences of more than 500 ohms and ear references which were perfectly balanced. The vigilance level was controlled by noting signs of drowsiness appearing in the EEG, and then pausing the recording and verbally interacting with the patients, while they moved their arms and legs in the chair. A bipolar recording channel was used to monitor eye movement artifact. In each case, approximately 20 minutes of eyes-closed resting EEG were recorded and edited to reduce artifact. The recordings were of good quality. From the digitally stored EEG, 132 seconds and 120 seconds of EEG in the two cases were subjected to quantitative spectral analysis. In the first case, a second sample of 62 seconds was also gathered for purposes of establishing test-retest reliability. The R squared value for alpha was 96.5%. The R squared values for single hertz topographies may be seen in Figure 3. The results of spectral analysis from 1-32 Hz were displayed as computed color-graduated topographic maps and compared via a Z-score transformation to age-regressed data bases of normal subjects using the Nx Link database and the Thatcher Lifespan database with the NeuroRep Analysis and Report System, the latter of which was used to generate one hertz topographic maps. The female patient's eyes-closed EEG was also analyzed utilizing low resolution electromagnetic tomography (LORETA) to provide an estimation of the localization of underlying generators of the patient's alpha activity. Both patients engaged in an informed consent process and signed informed consent forms. The patients were tested pre-treatment and post-treatment with the Yale-Brown Obsessive Compulsive Scale

(Goodman, Price, Rasmussen, Mazure, Fleischmann et al., 1989; Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989) and the Padua Inventory (Burns, Keortge, Formea, & Sternberger, 1996), both of which demonstrate good reliability and validity. In the first case, the Minnesota Multiphasic Personality Inventory (MMPI) was also administered pre- and post-treatment.

Case 1

This patient was a single, 25 year-old woman who was employed as an elementary school teacher. She had been diagnosed as having OCD at age 17 by a psychiatrist, and her previous treatment had consisted of pharmacologic therapy. Her medications had included Prozac, Klonepin, Zoloft, Anafranil, Haldol, Amitriptaline, Effexor, Serzone, and Xanax. None of the medications had been very effective. She was on .25 mg of Klonepin per day at the time of the intake interview, but had been off all medication for over two weeks prior to her qEEG. No other family member had been formally diagnosed with OCD, but she indicated that there were many individuals in her father's family who exhibited OCD-like behavior. The MMPI was administered because she also described significant depression. It confirmed severe depression and anxiety, very low ego-strength, introversion and being withdrawn, and intense over-emotionality with an extreme, classic pattern for developing somatic complaints. She previously had two suicide attempts, both at the age of 17. She also suffered with insomnia, requiring two hours to fall asleep. She engaged in bruxism and experienced considerable anxiety. She had been a straight A student in high school. She started dating at age 16, but had not done much dating since going to college. Two years previous to our intake interview she was so overwhelmed by her OCD that she was asked to resign at the end of the year. The year previous to seeing me she had not taught because she felt too incapacitated by her OCD symptoms. These symptoms particularly focused on contamination obsessions and washing compulsions, as well as obsessional rumination about harming herself. She also engaged in a lot of mental counting and excessive blinking. She had currently been teaching again for two months, but she was wondering if she would have to resign before long because she was becoming so overwhelmed by the OCD. Her qEEG results from the Nx Link and Lifespan databases may be seen in Figures 2 and 3. In Figure 4 you can study her LORETA analysis using a Poisson Maximal Frequency Test for alpha frequency. It localized the left temporal alpha to the superior temporal gyrus in the

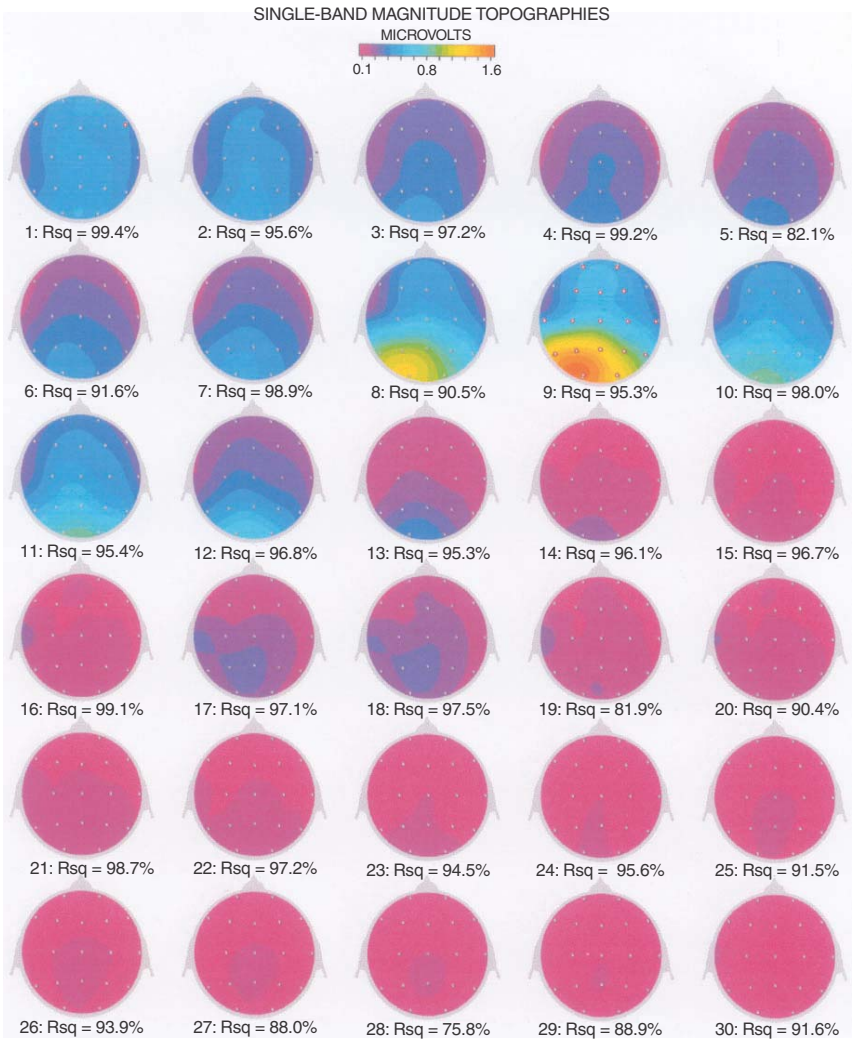
FIGURE 2. Case 1: Quantitative EEG Results from the Nx Link Database



vicinity of Brodmann areas 22 and 42, as well as in the middle temporal gyrus, Brodmann area 39, which is in the area of the angular gyrus.

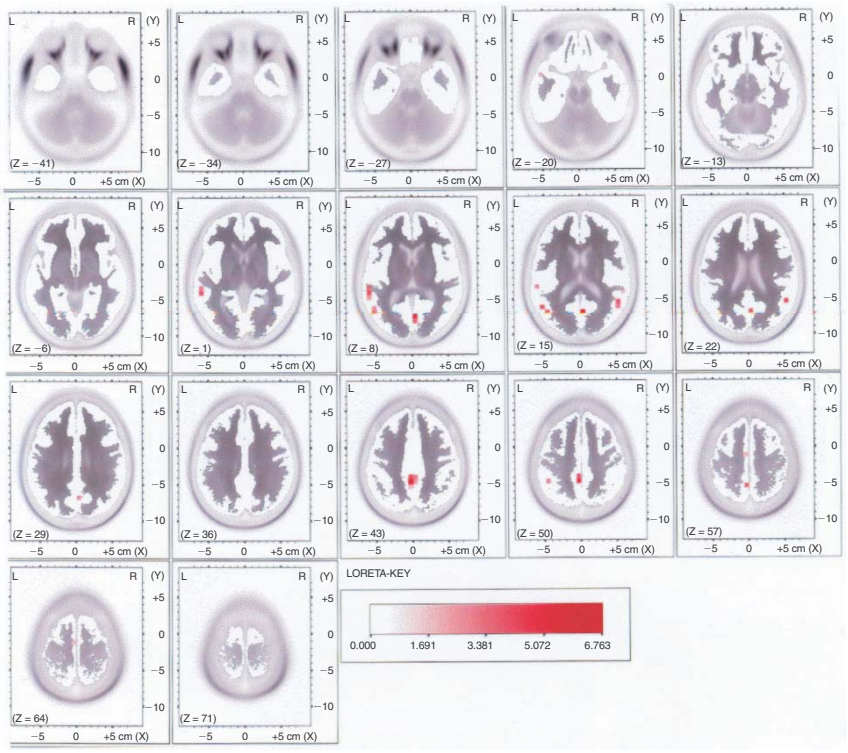
Following informed consent, we decided that due to the severity of her depression, our initial focus would be on reducing depression. Therefore, we utilized my depression protocol (Hammond, 2001a). This protocol was designed in response to the extensive qEEG and neuroimaging literature (summarized in Davidson, 1998a, 1998b) documenting a robust biological marker for depression which consists of greater left frontal alpha activity (inactivation) compared with right frontal activity. This protocol is also responsive to parallel research (Davidson, 1992; Heller, Etienne, & Miller, 1995; Heller, Nitschke, Etienne, & Miller, 1997; Isotani et al., 2001; Pizzagalli et al., 2002) confirming that a frontal asymmetry with greater right frontal beta activation (e.g., Fp2, F4) is associated with anxiety, and with panic disorder

FIGURE 3. Case 1: Single Hertz Magnitude Topographic Maps



(Wiedemann et al., 1999). A large proportion of depressed patients also experience anxiety. These latter qEEG studies are also congruent with PET and MRI studies (Abercrombie et al., 1996; Canli, Desmond, Zhao, Glover, & Gabrieli, 1998; Chua, Krams, Toni, Passingham, & Dolan, 1999; Dolan et al., 1996; Dolski et al., 1996; George et al., 1995;

FIGURE 4. Case 1: LORETA Analysis for Alpha Frequency Band



Naveteur, Roy, Ovelac, & Steinling, 1992; Reivich, Alavi, & Gur, 1984; Stapleton et al., 1997; Stewart, Devous, Rush, Lane, & Bonte, 1988) and transcrancial Doppler ultrasound studies (Troisi et al., 1999) of anxiety which find more right than left fronto-temporal activity, suggesting that the frontal cortex is involved in regulating and restraining subcortical limbic structures associated with affect. As a bridge facilitating understanding between neuroimaging and EEG research, a recent neuroimaging study (Nakamura et al., 1999) demonstrated a positive correlation between beta activity (13-30 Hz) and cerebral blood flow.

The patient's treatment began with twenty-one sessions of neurofeedback using the Roshi system (Hammond, 2001a) with two referential training sites at Fp1 and F3. The Roshi uses photic stimulation, wherein LED lights embedded in glasses vary in their pulsation, from moment to moment, pulsing on the peak frequency within the frequency

band being reinforced. The Roshi also has very low feedback latency. The 30 to 35 minute sessions at Fp1 and F3 used a program called Beta Max for the first half of the session, which reinforced 15-18 Hz while simultaneously inhibiting theta and alpha frequency bands, and finished the last half of the session with a program called SMR Max (inhibiting theta and alpha, reinforcing 12-15 Hz). Because it was observed that her dominant frequency not infrequently dropped into the delta range during this training, in the last 12 of these sessions we spent the first 10 minutes strictly inhibiting delta (1-4 Hz), followed by the Beta Max program and SMR Max program for 10 to 13 minutes each.

After two sessions of this training she reported feeling more energy and alertness, and being more social, "which is abnormal for me." She indicated that her depression level (0-10) had dropped from a 9 or 10 to a level 7. After 9 sessions the patient noted that her depression was "a lot better," her anxiety was less, and her sister had noted that her OCD symptom of excessive blinking had decreased. After 11 sessions she reported that a co-worker had also commented that she was blinking less and seeming calmer. After twelve sessions she said she had been feeling "pretty up, and I've been having more energy than normal and sleeping better too. The anxiety is really controllable now." After 13 sessions she commented, "I actually haven't felt this good in my life."

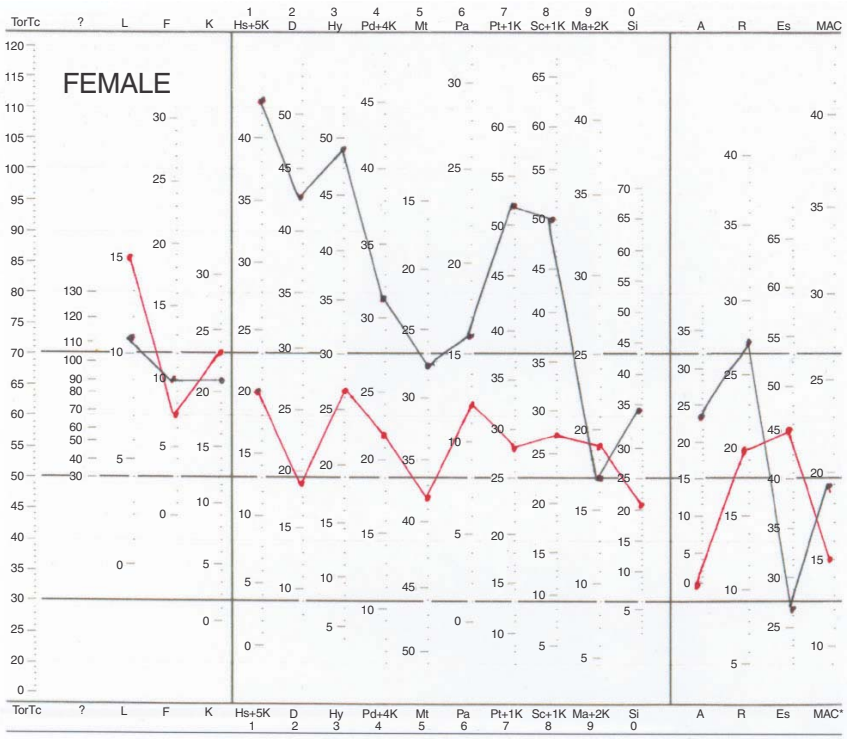
Based on her qEEG, our training now shifted and we now began using Neuropathways EEG equipment. This is a unit that samples at 250,000 samples per second and digitally filters the EEG signal, with a common mode rejection ratio >110 db wideband and >120 db at 60 Hz. I switched to Neuropathways to focus primarily on inhibiting inappropriate activity. Two sessions were done inhibiting 19.5-25 Hz at Pz-Cz with a sequential (bipolar) montage, while setting the threshold liberally to only mildly reinforce 12-14 Hz. This was followed by another session inhibiting 18-25 Hz and mildly reinforcing 12-15 Hz at Cz-Fz for 20 minutes and then inhibiting 19-25 Hz at Cz-T3 for 15 minutes. After these sessions she said she was mellower, able to concentrate easier, and "my mind didn't go as fast." We then did four sessions at T3-Cz, inhibiting 19-25 Hz while mildly reinforcing 12-15 Hz. After the first two of these four sessions she reported feeling less preoccupation with food contamination, that her OCD was improving, and she felt minimal depression. Due to some difficulty sleeping, we then did one Roshi session of SMR Max at C3 and C4. The goal of this session was to have a calming effect and to also encourage beta spindles and enhanced ability to fall asleep.

Next, we began focusing on the excess alpha activity in the left posterior area. Comparing her qEEG relative power alpha (Figure 2) with the alpha subtype from Prichep et al.'s (1993) research (see Figure 1), you will note that they are almost identical. It was my hope, therefore, that training at this site would begin to seriously impact her OCD symptoms. For the next eight consecutive sessions we inhibited 6.5-11 Hz while mildly reinforcing 15-18 Hz at T5-P3 with a sequential montage, using the Neuropathways unit. As hoped, with each session she reported that her OCD symptoms and brooding seemed to be improving. After seven sessions with this placement she estimated that her obsessions had improved by 75%. We then returned for two sessions to the left frontal area (Fp1-F3), using the Roshi depression protocol to reinforce changes in depression. A couple of days following these sessions, I spoke with the father of the patient on the telephone. He told me that a few days earlier his daughter had told him, "Dad, for the first time in my life I feel normal." Twelve more sessions focused on T5-P3, and in one of those sessions the time was split between T5-P3 and T5-O1. Two further sessions to reinforce changes in depression were also done at Fp1-F3. During this time she was reporting no problems with either depression or OCD symptoms.

After 50 sessions of neurofeedback, another MMPI was administered. The striking changes in her two MMPIs may be seen in Figure 5. Her depression normalized from a severe level and she became much less withdrawn, going from being introverted to extroverted. These latter changes have been commonly observed by me in pre-post testing using my depression protocol. Increasing left frontal activation would be anticipated to produce such changes, since the left frontal area is associated with not only happy emotions, but also with approach motivation (Davidson, 1998a). Her extreme levels of both over-emotionality and somatic symptoms decreased to within normal limits. Her anxiety and OCD symptoms, as measured in scales A and 7, dramatically decreased and her ego-strength and resilience increased. She was defensive in her test taking set at both administrations of the MMPI. Nonetheless, in her pre-treatment testing she still showed extreme levels of psychological disturbance, and in her post-treatment testing her defensiveness was only mildly greater.

At this same time the Padua Inventory was readministered, and I had a colleague administer the Y-BOCS to alleviate contamination effects. The last eight sessions of neurofeedback had placements at T5-Pz, inhibiting 4-8 Hz while mildly reinforcing 15-18 Hz. The first three of these maintenance sessions were held at two- to three-week intervals.

FIGURE 5. Case 1: Pre- and Post-Treatment MMPI



The final five sessions were spaced out over a five-month period for follow-up reinforcement and to check for maintenance of changes. Seven and a half months after termination of the five-month maintenance phase of treatment (and 15 months after the main treatment phase), I spoke with the patient and the Padua Inventory was readministered to the patient. I had a telephone interview with her mother at about the same time, obtaining external confirmation of the maintenance of changes.

Table 1 shows the changes in this patient on the Y-BOCS and the Padua Inventory from pre- to post-treatment and on follow-up. The Y-BOCS is the most respected measure of OCD and generally patients must score over 16 to be included in medication trials. The patient scored 26 initially, slightly above the averaged mean for four samples of OCD patients (Goodman, Price, Rasmussen, Mazure, Fleischmann et

TABLE 1. Case 1: Pre-Post-Follow-Up OCD Outcome Measures

TEST	Y-BOCS	Padua Inventory
OCD MEAN & S.D.	24.7 (S.D. = 6)	54.93 (S.D. = 16.72)
PRE-TREATMENT SCORE	26	72
POST-TREATMENT	4	8
FOLLOW-UP SCORE		12
S.D.'S IMPROVED	3.7	3.8 & 3.6
PERCENT IMPROVED	84.6%	88.9% & 83.3%

al., 1989; Goodman, Price, Rasmussen, Mazure, Delgado et al., 1989) on the Y-BOCS total score. The Y-BOCS (Goodman, Price, Rasmussen, Mazure, Fleischmann et al. 1989) mean for the Obsessions subscale is 10.7 and her pre-treatment score was 12.5. The Y-BOCS mean for the compulsions subscale is 11.2, and she scored 13.5. An independent examiner interviewed her with the Y-BOCS after 50 neurofeedback sessions. Instead of 26, she now scored 4–twice the average reduction in score that is usually found in drug studies with the most effective pharmacologic treatment (Ackerman & Greenland, 2002). This is a 3.67 standard deviation improvement, compared with the 1.33 standard deviation average improvement that results from the most effective pharmacologic treatment. Her score was now 3 on the Obsessions subscale, and 1 on the Compulsions subscale. The reliability on both administrations was rated as excellent. The Y-BOCS was not administered in a follow-up interview because the patient had moved away to another geographic area. However, in addition to a telephone interview with the patient and her mother, she did take the self-administered Padua Inventory 15 months following the second (post-treatment) administration of the YBOCS and the Padua Inventory.

On the Padua Inventory, her pre-treatment score of 72 significantly exceeded the mean for an OCD population (54.93; SD = 16.72; Burns et al., 1996), placing her one standard deviation above the mean for OCD patients. After 50 neurofeedback sessions her total score was 8, and on follow-up 15 months later, her score was 12. These improvements were comparable to those seen on the Y-BOCS (see Table 1). The mean total score on the Padua Inventory for a normal sample is 21.78 (SD = 16.33). Thus, at the completion of 50 sessions she was almost one standard de-

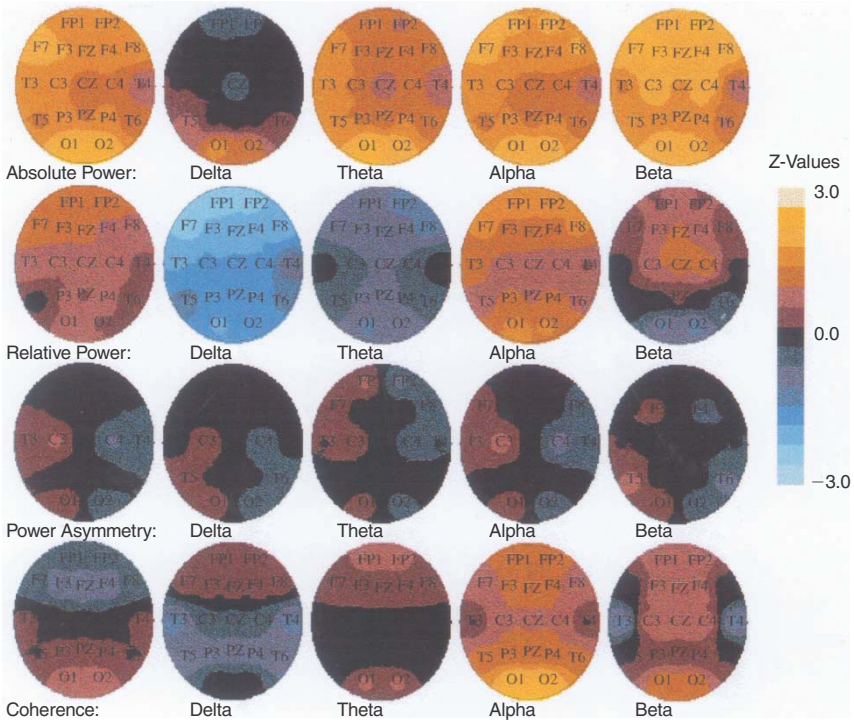
viation below the mean for a normative sample. On subscale 1 (Obsessive Thoughts about Harm to Self or Others; OCD Mean = 10.0), her pre-treatment score was 18, her post-treatment score was 4, and her follow-up score was 3. On subscale 2 (Obsessive Impulses to Harm Self or Others; OCD Mean = 6.0), her pre-treatment score was 6, her post-treatment score was 0, and her score on follow-up was 1. On subscale 3 (Contamination Obsessions and Washing Compulsions; OCD Mean = 13.87), her pre-treatment score was 25, her post-treatment score was 3, and her follow-up score was 2. On subscale 4 (Checking Compulsions; OCD Mean = 19.87), her pre-treatment score was 15, her post-treatment score was 1, and her score on follow-up was 4. Finally, on subscale 5 (Dressing/Grooming Compulsions; OCD Mean = 5.2), her pre-treatment score was 8, her post-treatment score was 0, and her score on follow-up was 2. Thus, in her 15 month follow-up she scored at or below the mean for normal, non-OCD individuals on all Padua Inventory subscales. This is even more significant because she was feeling under extra stress, having just begun teaching after a summer vacation. She was on no medication.

Case 2

The second case was a 25-year-old male who initially presented as having problems with attention deficit disorder. He had been "on heavy doses of Ritalin" for years. He indicated that when he began taking it, "it changed everything" and he felt he could think and function normally. "I looked forward to using Ritalin," he said, but at the same time it made him feel stigmatized. He had recently read about neurofeedback and wanted to experiment with its potential. His history included a significant previous problem with marijuana abuse and a problem with alcohol abuse. He said, "I can't stay with anything for more than five minutes. I want instant gratification." He had also had "breakdowns" in which he became depressed and would cry to his parents on the telephone. He had previously been on Effexor and Paxil, but was not currently depressed and was only taking Ritalin. He met the diagnostic criteria for ADD and ADHD. After an intake interview, we gathered qEEG data after he had been off Ritalin for three days.

In giving him feedback on the analysis, I commented on the mild excess of beta over the general area of the anterior cingulate gyrus (see Figure 6, electrode sites Fz and Cz), and inquired about OCD symptomatology. It quickly became apparent that he had many OCD symptoms (e.g., obsessions with contamination and washing rituals, and checking

FIGURE 6. Case 2: Quantitative EEG Results from the Nx Link Database



compulsions) and he admitted to feeling he "must be absolutely perfect," whether it was in playing music or making hamburgers. Therefore, I administered the Y-BOCS and gave him the Padua Inventory to take home. His Y-BOCS score of 25 and Padua Inventory score of 62 confirmed the dual diagnosis of OCD.

Forty-four 30-minute long sessions were spent inhibiting 19-25 Hz beta, while mildly reinforcing 12-15 Hz, typically for 15 to 20 minutes at Fz-Cz (using a sequential montage with a Neuropathways neurofeedback unit), followed by 15 minutes with the same protocol at Cz-C4. Interestingly, after two sessions he indicated: "A lot has changed. For two days I have felt I was no longer a prisoner to any situation. I feel turned-on, in a non-sexual way." He further volunteered that he had tended to compulsively masturbate and had also used masturbation as a soporific. However, he had been feeling no compulsion to masturbate. He also felt that he tracked words more smoothly on a page in reading.

Since there had been only two sessions, this could be the result of positive expectancy, but on the other hand, medication responses had not been overly positive with antidepressants. After four sessions he reported sleeping much better. After six sessions, he indicated that two friends, his father, and a sister had all spontaneously indicated that he seemed calmer. He said, "There's an absence of rudimentary fear and paranoia." After another session, he described himself as "mellow," and said, "It's much easier for people to be around me. Women actually want to get to know me!" After still another session, he indicated he was "in a good mood all the time." Following nine sessions he said that the Ritalin he was taking had been feeling more and more potent, and, therefore, he had been cutting it down and was now taking only between one-quarter and one-eighth of the prescribed dose daily.

At this time we began occasionally spending the first half of the session inhibiting 20-25 Hz beta at Cz-C4 or Fz-Cz, and then moving the electrodes to F7 and F8 and inhibiting 7-11 Hz while mildly reinforcing 13-16 Hz. This was done to begin addressing his ADD problems further, since F7 and F8 were both areas of alpha excess on his qEEG, as may be seen in Figure 6. After twelve sessions he said that his visual tracking of words felt "improved immensely." He also indicated, "I can't even drink coffee anymore" because rather than helping his concentration as it did before, it caused him to feel over stimulated. After another session he commented that his concentration was better when he was reading, and he was having fewer intrusive thoughts. Following his fifteenth session he reported being able to read for long periods of time. He continued to steadily report feeling better. After 26 sessions he indicated that his memory with reading was improved, he had less social anxiety, felt more energetic, and found he could do things musically that he could not do before due to anxiety.

As treatment progressed, we also did some prefrontal training at Fp1 and Fp2, inhibiting 7-11 Hz and mildly reinforcing 12.5-15 Hz. The patient felt that this frontal training improved his concentration "a lot," helped him to feel more confident and less afraid socially, and he believed that it felt as if it also assisted with the OCD symptoms. By the time we had reached 50 total sessions, he was feeling excellent. Women he dated found him much more mature and easier to relate to, and his family found him to be much easier to get along with. OCD symptoms were minimal. After 56 thirty-minute sessions, 35 of which had focused on inhibiting beta in the Fz-Cz-C4 area, an independent colleague readministered the Y-BOCS. His score had now dropped from 25 to 10–a decrease of 2.5 standard deviations.

The patient was feeling a desire to enhance his academic abilities further in anticipation of returning to college, and he had the financial resources to do so. Therefore, we continued neurofeedback. At the conclusion of treatment (93 total sessions), he had gone through 44 sessions inhibiting beta over the Fz-Cz-C4 area, 22 sessions inhibiting alpha and reinforcing low beta frequencies at F7-F8, 21 sessions at Fp1-Fp2, three and a half sessions inhibiting 2-9 Hz at O1-O2, and two and a half sessions inhibiting alpha in the parietal area.

A summary of the pre-, post-, and follow-up testing on this case is found in Table 2. At the end of treatment, after the 44 thirty-minute sessions of inhibiting beta along the vertex (and 93 total sessions), the Y-BOCS was again administered and his score had decreased further from 10 to 7. This translates to an improvement of 3 standard deviations in his Y-BOCS score from the beginning of treatment. His score on the Padua Inventory at that time had dropped from 62 to 7, representing a 3.4 standard deviation improvement from his pre-treatment level. At the conclusion of treatment, the patient moved out of state. However, 13 months later I was able to speak with his sister who had just returned from an extended visit with him. She reported that he remained dramatically changed from his pre-treatment adjustment. I also interviewed him on the telephone and had him complete the Padua Inventory. He had always had anxiety about flying, and since the September 11, 2001 terrorist attack in New York City, his fear had been exacerbated, but he no longer experienced any OCD symptoms. He was not on any medication for OCD and was not taking Ritalin. Nonetheless, he said, "My concentration is still a million times better." The improvement in concentration and OCD had given him the confidence to return to college. His 13-month follow-up score on the Padua Inventory was now 5, a 3.4

TABLE 2. Case 2: Pre-Post-Follow-Up OCD Outcome Measures

TEST	Y-BOCS	Padua Inventory
OCD MEAN & S.D.	24.7 (S.D. = 6)	54.93 (S.D. = 16.72)
PRE-TREATMENT SCORE	25	62
POST-TREATMENT	7	7
FOLLOW-UP SCORE		5
S.D.'S IMPROVED	3.0	3.3 & 3.4
PERCENT IMPROVED	72%	88.7% & 92%

standard deviation improvement from his pre-treatment score. His Padua Inventory subscale 1 (Obsessive Thoughts about Harm to Self or Others; OCD Mean = 10.0) pre-treatment score was 15, his post-treatment score was 2, and his follow-up score was 2. On subscale 2 (Obsessive Impulses to Harm Self or Others; OCD Mean = 6.0), his pre-treatment score was 2, his post-treatment score was 1, and his score on follow-up was 0. On subscale 3 (Contamination Obsessions and Washing Compulsions; OCD Mean = 13.87), his pre-treatment score was 20, his post-treatment score was 2, and his follow-up score was 2. On subscale 4 (Checking Compulsions; OCD Mean = 19.87), his pre-treatment score was 19, his post-treatment score was 2, and his score on follow-up was 1. Finally, on subscale 5 (Dressing/Grooming Compulsions; OCD Mean = 5.2), his pre-treatment score was 6, his post-treatment score was 0, and his score on follow-up was 0. Thus, in his 13 month follow-up he scored at or below the mean for normal, non-OCD individuals on all Padua Inventory subscales.

SUMMARY AND CONCLUSIONS

In research with uncontrolled epilepsy (summarized in Sterman, 2000, which has included placebo-controlled, blinded studies) neurofeedback has proven capable of reconditioning brain wave patterns. Outcome research has also been done on neurofeedback with ADD/ADHD, learning disabilities, depression, anxiety, brain injury, fibromyalgia, and posttraumatic stress disorder (Hammond, 2001b). This is the first publication, however, on the treatment of obsessive-compulsive disorder with neurofeedback. Quantitative EEGs were gathered on two consecutive OCD patients seeking treatment. This assessment then guided individualized protocol selection for subsequent neurofeedback training. The qEEG findings in the first case almost identically matched the average profile of an alpha subtype of OCD, leading to treatment focused in the left posterior area after we alleviated her depression. The author had never heard of anyone using this neurofeedback protocol and would not have considered using it without being guided by a qEEG assessment. The patient's more significant OCD symptoms primarily changed following treatment focused in the left posterior area. Scores on the Yale-Brown Obsessive-Compulsive Scale and the Padua Inventory normalized following neurofeedback. An MMPI was administered pre-post to the first patient, who showed dramatic improvements in not only OCD symptoms, but also in depression, anxiety, somatic symp-

toms, and in becoming extroverted rather than introverted and withdrawn. In follow-ups of the two cases at 15 and 13 months after completion of treatment, both patients were maintaining improvements in OCD symptoms as measured by the Padua Inventory and as externally validated through contacts with family members.

Since research has found that pharmacologic treatment of OCD produces only very modest improvements, and behavior therapy utilizing exposure with response prevention is experienced as quite unpleasant and results in treatment dropouts, neurofeedback appears to have potential as a new treatment modality for OCD. Further controlled research should be pursued in this area.

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