

Obsessive Compulsive Disorder and the Efficacy of qEEG-Guided Neurofeedback Treatment: A Case Series

Tanju Siirmeli and Ayben Ertem

Key Words

Neurofeedback

Obsessive Compulsive Disorder

Quantitative Electroencephalography

ABSTRACT

While neurofeedback (NF) has been extensively studied in the treatment of many disorders, there have been only three published reports, by D.C. Hammond, on its clinical effects in the treatment of obsessive compulsive disorder (OCD). In this paper the efficacy of qEEG-guided NE for subjects with OCD was studied as a case series. The goal was to examine the clinical course of the OCD symptoms and assess the efficacy of qEEG guided NE training on clinical outcome measures.

Thirty-six drug resistant subjects with OCD were assigned to 9-84 sessions of qEEG-guided NE treatment. Daily sessions lasted 60 minutes where 2 sessions with half-hour applications with a 30 minute rest given between sessions were conducted per day.

Thirty-three out of 36 subjects who received NF training showed clinical improvement according to the Yale-Brown obsessive-compulsive scale (Y-BOCS). The Minnesota multiphasic inventory (MMPI) was administered before and after treatment to 17 of the subjects. The MMPI results showed significant improvements not only in OCD measures, but all of the MMPI scores showed a general decrease. Finally, according to the physicians' evaluation of the subjects using the clinical global impression scale (CGI), 33 of the 36 subjects were rated as improved.

Thirty-six of the subjects were followed for an average of 26 months after completing the study. According to follow-up interviews conducted with them and/or their family members 19 of the subjects maintained the improvements in their OCD symptoms. This study provides good evidence for the efficacy of NF treatment in OCD. The results of this study encourage further controlled research in this area.

INTRODUCTION AND BACKGROUND

OCD is a debilitating psychiatric disorder. It is characterized by recurrent and persistent thoughts, impulses, images (obsessions) and/or repetitive behaviors, or mental acts that the person is driven to perform, that are intrusive and inappropriate and cause marked anxiety or distress.¹ It is the fourth most common mental disorder and the tenth leading cause of disability in the world.

There are only 3 published reports^{2,3} on NF in the treatment of OCD. Currently, the most widespread treatment modalities for this disorder are pharmacological treatment with serotonin reuptake inhibitors (SRIs) with cognitive behavior therapy (CBT). Despite the proven efficacy of both SRIs and CBT, a substantial percentage of patients receive little benefit from these standard approaches.⁴

As effective as these treatments are, a response is usually contingent on an amelioration of the symptoms and not the remission of symptoms. After treatment persons suffering from this disorder may

not all patients show a response to these treatments. While controlled trials with SRIs have demonstrated a selective efficacy in OCD, up to 40-60% of patients do not have a satisfactory outcome.⁵ The fact remains that a large fraction of patients without substantial response to standard treatment experience significant morbidity.⁶

When investigating SRIs Ackerman and Greenland⁷ found that a meta-analysis of 25 drug studies with OCD patients had modest improvement with domipramme. The average treatment effect on the Y-BOCS was 10.64 (uncorrected for placebo effects), which is a 1.33 standard deviation improvement. For fluvoxamine, which is the most effective SSRI treatment for OCD, the mean Y-BOCS improvement was only 5.4 points. If the 10.64 average change of Ackerman and Greenland is used, patients scoring high on the Y-BOCS (20-30 points) will still have a mild to moderate range of symptoms (20-30 points) after drug treatment.

The efficacy and response to COT treatment is quite variable, and also may not be sustained in the long term, It is claimed that 76%-86% of patients who complete CBT treatment make improvements.⁸ On the other hand, intensive COT has been found to have a 75% remission rate.⁹ O'Connor et al.¹⁰ found that either cognitive behavioral therapy or medication alone can help the patients to a certain level.

It is evident that other novel treatment methodologies may be needed. As an alternative treatment Rucklidge¹¹ introduced micronutrients to a patient who did not respond to medications and subsequently underwent CBT with a modest response. Micronutrients worked well with this patient. Rucklidge concluded that many OCD patients are resistant to conventional treatments so alternative treatments should be introduced to patients and further research is needed on the mechanisms of micronutrients.

qEEG Findings and NF

Currently there is little evidence on the psychopathology of OCD. However, in order to apply NF treatment one needs to know which band to train and on which brain area to place the electrode. For this the qEEG method is quite successful in helping guide the practitioner in placement and band selection since some studies have been done in assessing the qEEG findings in OCD. One of the first qEEG studies conducted on OCD was done by Simpson et al.¹² In this study, patients' qEEG was recorded under symptom provocation (both live and imaginary). The results indicated that significant EEG changes were elicited by live contaminants, but not imaginary ones, and that an increase in OCD symptoms showed an increase in posterior relative alpha activity (in comparison to anterior areas).

Drs. T. Siirmeli and A. Ertem are from the Healthy Living Center for Research and Education, IstanOul, Turkey.

Address correspondence and requests for reprints to Tanis Siirmeli, MO, Healthy Living Center for Research and Education, Kore Sehtlen Caddesi No 49, Zincirlikuyu, Istanbul 34387 Turkey

neuropsychiatry@yahoo.com

Received, May 28, 2010; accepted: November 23, 2010,

Patients were classified as OCD, who shared the same characteristics, were divided into 2 subgroups by QEEG. One group was characterized as having diffuse excessive alpha and excessive beta in frontal central and mid-temporal areas, whereas the other group was characterized by excessive theta activity, especially in the frontal and posterior-temporal areas. Theta abnormalities have also been reported by Insel et al., Jenike and Bnotman, Palla et al., and Rockwell and Simons. Furthermore, Pritchard et al. and Hansen et al.²² have been able to identify pathophysiological subgroups within the OCD population who exhibit differences with regard to their response to serotonergic medications (responders vs. non-responders). 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 SRIs.

In a study conducted by Pogarell et al., authors found that patients who had high levels of obsessions had higher absolute EEG power measures, especially in the faster frequencies (alpha2, beta), whereas patients with high compulsion scores had lower absolute EEG Power. This may be related to increased mental activity in obsessions as opposed to compulsions.

In a study conducted by Bum et al., decrease of the slow alpha-band power in OCD as compared to healthy subjects was observed. A significant negative correlation between the slow alpha-band power and the time to complete a neuropsychological test exploring executive functions was found: the more reduced the slow alpha-band power, the slower the performance on this test. Bolwig et al. found an excess in the alpha range with sources in the corpus striatum, in the orbito-frontal and temporo-frontal regions in untreated OCD patients. This abnormality was seen to decrease following successful treatment with paroxetine. Finally, Tot et al. found OCD patients to be characterized by increased slower frequencies and slow alpha frequencies, especially in the left fronto-temporal areas, when compared against age matched norms.

As can be seen, since QEEG findings tend to see OCD as a heterogeneous group who share the same symptoms, this may explain why current treatments are not effective in all patients, and the duration of positive effects are not long lasting. This may indicate that different modalities of treatment may be needed to efficiently treat these subgroups.

NF is an intervention aimed at training individuals to better regulate the biological functioning of their own brain. This has generally involved the self-regulation of EEG rhythmic activity, traditionally referred to as EEG biofeedback, NF or neurotherapy. In NF training the subject is placed in front of a computer screen which displays the subjects digitized and analyzed brain electrical activity. The display can be either in the form of a complex video game type of displays, or in the form of simple bar graphs. The thresholds of the activities which are to be increased and/or decreased are set on the display. When the undesired activity decreases below the threshold and when the desired activity increases above the threshold a pleasant tone is heard through the attached headphones, and the display will change. In some systems, the subject can also earn points based on his/her performance providing additional feedback. As the sessions are repeated, the thresholds are gradually modified inhibiting the undesired activities and reinforcing the desired activities thereby conditioning to endure these activities."

NF has been used successfully with ADHD/Learning epilepsy,^{3,4,6} anxiety,^{3,4,6} mild head injuries^{3,4,6} and even in autism.^{4,7}

Therefore, seeing a need for more information in this area we decided to investigate the efficacy of QEEG-guided NF in subjects with OCD as case series

MATERIALS AND METHODS

We studied 36 subjects ranging in age 18-59 years old. Inclusion criteria: Subjects were included from patients visiting the center who met DSM-IV diagnostic criteria for OCD. Subjects should have had received at least one treatment modality which was ineffective. Additionally, the subjects should not have any history of physical illness; the baseline laboratory tests (Hemogram, BUN, Creatinine, TSH and urine drug screening for illicit drugs) had to be normal. Exclusion Criteria: The presence of any other psychiatric disorder, history of past or present drug abuse, head trauma with loss of consciousness, suicide risk and/or abnormal blood test results. All the subjects in the study used medication prior to the treatment. The mean total number of medications used in the past was 3.6 (± 2.2). The mean duration of illness was 8.0 years (± 4.7y.). On inclusion all medications were discontinued and 34 patients were medication free at baseline and for the entire NF treatment duration. Only 1 patient received medication (clomipramine) during NF treatment since it was necessary to manage her symptoms. However, she was taking 2 medications at the time of admission. It was discovered that another patient was self-medicating with bupropion during the treatment phase. Evaluation measures included family history, QEEG data which was processed with the NLink database, and the following ratings scales

The Yale-Brown obsessive compulsive scale (Y-BOCS) was designed to remedy the problems of existing rating scales by providing a specific measure on the severity of the symptoms of OCD that is not influenced by the type of obsessions or compulsions present. The scale is a clinician-rated, 10 item scale, each item rated 0 (no symptoms) to 4 (extreme symptoms) (total range 0 to 40). The scale rates obsession and compulsive components and provides subscores for each. A cut-off score of 16 is usually used for inclusion into OCD medication trials. In this study subjects were rated before treatment, and after completion of treatment,

The clinical global impression (CGI) rating scale is a commonly used scale that measures symptom severity, treatment response and the efficacy of treatments in treatment studies of patients with mental disorders. In this study changes in the severity scale, pre- and post-treatment were assessed. The clinical global impression-severity scale (CGI-S) is a 7-point scale that requires the clinician to rate the severity of the subject's illness at the time of assessment, relative to the clinician's past experience with subjects who have the same diagnosis. Considering the total clinical experience, a subject is assessed on the severity of the mental illness at the time of rating where: 1 = normal, not at all ill; 2 = borderline mentally ill; 3 = mildly ill; 4 = moderately ill; 5 = markedly ill; 6 = severely ill; and 7 = extremely ill.

An MMPI was administered to all subjects before treatment and after completion of treatment, however, results were only available for 17 out of the 36 subjects. The MMPI, although developed as a tool to assess personality, lends itself to measuring changes in psychopathology. It is appropriate for assessing treatment outcomes in patient samples where Psychopathology is being evaluated, particularly if the emphasis is being placed on DSM-IV Axis I disorders. Since it is sensitive to psychopathology, as the illness recedes the pathological scores decrease (e.g., normalize). The MMPI is very difficult to fool, whereas the patient can more easily manage the doctor interview and the Y-BOCS rating thus affecting the CGI and Y-BOCS results. Since this is not a double blind

study the MMPI may provide a counterpoint to the bias of the doctor, All patients were interviewed by the center staff. Informed consent was obtained from all subjects, and independent investigations were conducted.

To determine the locations and bands to be used in NF treatment, gEEGs were recorded with a Lexicor Neurosearch-24 qEEG system (software version 3.10). All EEGs were recorded drug free. In order to ensure that all subjects were drug free, all medications were discontinued at screening, and the recordings were performed after a washout period equal to 7 half-lives of the medication they were taking prior to admission. For example, the half-life of clomipramine is 35 hours so 7 half-lives (in days) would be equivalent to $7 \times 35/24 = 10.21$ days, therefore, the EEG would be recorded on the 12th day after cessation of medication. EEG signals were sampled at 128 samples per second per channel, Samples were analyzed with a normative neurometric approach using the Nx-Link database software (version 2,40), The NxLink database software is based on the work of E. Roy John and is a method of quantitative EEG that provides a precise, reproducible estimate of the deviation of an individual record from normal. QEEGs were recorded and compared against the Nxlink database both before and after treatment as well as every 40 sessions, in order to reveal the divergence of the brain electrical activity from norms, in the form of Z- scores, and to guide the NF treatment protocols by training the areas that show deviations from normal, as determined by the comparisons to the Nxlink database. In Neurometric QEEG analysis, all QEEG variables are calculated as Z-scores which is a score equal to the distance (deviation) from the norm in standard deviation units. The rationale behind this approach is that the subjects who normalize their QEEG Z- scores will benefit the most from NF.

When the baseline gEEG of this population was analyzed, excessive theta and/or alpha with a generalized distribution, especially in relative power was observed. In addition, the following findings were observed (Table 1). As can be seen, a little over half of the subjects showed increased relative alpha activity (in comparison to norms) and half of those with increased relative alpha power also showed increased coherence (hypercoherence) in the alpha band. Hypercoherence is said to be present in the EEG when two brain sites or areas are overly connected, as indicated by the two waveforms at these different sites being more similar in terms of morphology than an age-matched normal subject. Hypercoherence can be regarded as a kind of immaturity wherein cortical areas do not specialize and take on specific abilities

and thus appear overly similar to each other. **NF Treatment**

All the NF training was performed using Lexicor Biolex software (version 2.40). Each session was of 60 minutes duration, with 1 session per day. Electrodes were placed according to the International 10-20 System. Between 9 and 84 NF training sessions were completed, depending on the case. Treatment termination was based on the changes (a decrease) of symptoms in comparison to the pre-treatment complaints. The mean number of sessions was 50.2 (t 22,4 STD),

Electrode sites for training were selected based on the QEEG analysis (using the (Nx-Link database). The location of the deviant Z-scores is most important no matter what the EEG measure. A general rule is to link the patient's symptoms to deviant Z-scores located in regions of the scalp related to functional specialization in the brain and the patient's symptoms." The importance of proper area and band selection was also shown by Moore⁹ in a review of 2 OCD studies he conducted, where he found that pure alpha training did not produce any benefits. Moore concluded that the reason was that there were 2 OCD

Table I

Screening QEEG deviations from norms			
Primary Finding		Secondary Finding	
Increased relative alpha power	20	Increased alpha coherence	10
Increased relative theta power	10	Increased theta coherence	3
Increased relative beta power	4		
Increased relative theta and beta power			
Increased relative alpha and beta power			

subgroups both of which would not have benefited from alpha training. The frontal and frontotemporal electrode sites were selected according to the subject's QEEG and also according to previous studies based on the frontal, prefrontal and fronto-temporal deviations from norm based on the QEEG recording of OCD patients. The most commonly used electrode sites were as follows (both bipolar and monopolar). In NF inhibit means keeping the activity below a set threshold whereas reward means keeping the activity above a set threshold:

The frontal and centro-parietal-temporal electrode sites below were selected according to subjects QEEG and Brodmann Areas (BA) The criteria to shift from one site to another is the z-score values or based on the first author's clinical experience.

- FP1-FP2: Theta or p-inhibit, a-inhibit, p(21-32)-inhibit
- F3: Theta or (x-inhibit, a-inhibit, p(21-32)-inhibit or (1 3-32)-inhibit
- FL: Theta or a-inhibit, p(21-32)-inhibit, or N13-32)-inhibit
- F4: Theta-inhibit, a-inhibit, 3(21-32)-inhibit or ((13-32)-inhibit
- Fp1-14; Theta-inhibit, a-inhibit, p(21-32)-inhibit or p(13-32)-inhibit

The Fp0₂ site was helpful for the fear and anxiety problems. Fp^o stands for Frontal Pole Orbital (pre-frontal) and 7' signifies the right side of the brain This site is outside the standard 10-20 system at the juncture of the right brow bone and top of the nose, in the inner corner of the eye socket.^{6,2}

- Fp0₂: Theta-reward, a-inhibit, p(21-32)-inhibit or a-reward. Theta-inhibit. p(21-32)-inhibit

Central-parietal area electrode sites were selected for procedural memory and brain area 24, the Anterior Cingulate for being the hub of the affective limbic system. Brain Area 40 representing cognitive reasoning, imagination was also used.

- C4-P4: Theta-inhibit, a-inhibit, p(21-32)-inhibit or p(13-32)-inhibit or SMR-reward, Theta-nhibit, p(21-32)-inhibit
- P4; Theta-inhibit, a-inhibit, p(21-32)-inhibit or (1(13-32)-inhibit

The sensory area was selected for sleep regulation. BA 24 Anterior Cingulate: Hub of affective limbic system.

- Cz-G4: Delta-inhibit, Theta-inhibit, p(21-32)-inhibit

Coherence training was performed according to z-scores. Hyper coherence can be considered as a lack of differentiation of brain functions or as a decrease in "flexibility" of functioning.

- FP1-FP2.
- F3-F4, P3-P4: is coherence-inhibit, a-inhibit, p(21-32)-inhibit or p(13-32)-inhibit

Table 2

YBOCS results. Changes in the severity of illness based on the Yale Brown Obsessive Compulsive Scale

A) Patients	Total Score		Obsession Subscale		Compulsion Subscale	
	Pre	Post	Pre	Post	Pre	Post
Mean	27,58	6,06	17.08	3.64	10.50	2.42
Standard Deviaton	9,65	10,36	5.73	5.66	9.37	5.26
Mean Difference (Post-Pre)		-21,53	.13.44		-8.08	
F(1,35)		134.77	129.63		31.06	
r(1,35)		079	0.79		0.47	
<u>Significance</u>		P < 0.01	P < 0.01		P < 0.01	
Pure Subgroups			Obsessional Group (reponAg only obsessions will no compulsion)		Compulsion Group' (reporting only compulsions with no obsessions)	
			Pre	Post	Pre	Post
Mean			18.64	3,97	20.00	1.67
Standard Deviation			2.97	3.68	0	2.89
Mean Difference (Post-Pre)			-15.80		-18.33	
F(1,35)			193.9		226.63	
r(1,35)			0.93		0.99	
<u>Significance</u>			P < 0.01		P < 0.01	
Mixed Subgroup reporting both obsessions and compulsions)	Total Score		Obsession Subscale		Compulsion Subscale	
			Pre	Post	Pre	Post
Mean	36.44	9,61	18.78	5.06	17.67	4.56
Standard Deviation	4,20	13.50	2.05	7.03	3.65	6.78
Mean Difference (Post-Pre)	-26.83		-13.72		-13.11	
F(1,35)	56.48		50,05		47.67	
r(1,35)	0.64		0.61		0.60	
<u>Significance</u>	P < 0.01		P < 0,01		P < 0.01	

For illustration purposes only, due to small N (3)

RESULTS

The study included 12 males and 24 females. The mean age for the group was 30.1y (±9.0y). For males the mean age was 25.8y (±5.2y) and for females was 32.3y (±9.8y). Twenty-six out of 36 had a family history of some sort of psychiatric illness. Since the inclusion was based on patients that came for treatment to the clinic, without any a priori selection criteria, more females than males were included.

The pre- and post-study results are shown in Table 2. As can be seen NF treatment reduced Y-BOCS total score from 27.58 (±9.65 std) (which is above the cut-off score of 16) to 6,06 (±1036), which corresponds to a reduction of 21,53 points A repeated measures ANOVA, where intra-subject effects were accounted for, was performed on the data and the overall change was found to be significant at the p<0.001 level (F(1,35) =134.77, 01,35) = 0.79). Tests were performed on the subscales separately and all were found to be significant at a p < 0.001 level of significance. One group reported only obsessive symptoms (N=15), one group only compulsion symptoms (N=3) and a third group reported both symptoms (N=18). These were analyzed separately and all 3 groups reduced their scores significantly (p < 0.01).

The results of the CGI pre- and post-treatment assessment along with the statistical analysis of the results (based on repeated measures ANOVA corrected for intra-subject variance) are given in Table 3,

According to the CGI results the decrease of the score of -4 points was found to be statistically significant at the p < 0.01 level (F(1,35) = 205.94. 11²(1,35) = 0.85 The group (as a whole) was rated as being severely ill, whereas at the end of treatment they were rated as being borderline mentally ill showing a 4 point decrease in the severity of their illness.

Table 3
CGI Results. Changes in the clinical global impressions severity score

<u>Severity</u>	Pre	Post
Mean	6.22	2.03
Standard Devation	0.76	1.75
Mean Difference (Post-Pre)	-4.19	
F(1,35)	205.94	
11 ² (1,35)	0.85	
<u>Significance</u>	P < 0,01	

The MMPI was admwstered to all sub,jects before and after treatment, however, results were only available for 17 out of the 36 subjects. Two scores were analyzed, the psychasthenia score and the depression score. The Pt scale was originally developed to measure the general symptomatic pattern labeled by Marks et al." as psychasthena not commonly used today, which is characterized by excessive doubts, compulsions, obsessions, and a rigid and perfectionist personality with unreasonable fear. Psychasthenia can be considered very close to modern OCD. The depression score was analyzed because it showed the highest value at 75 indicating a score greater than 2 standard deviations from the norm. The results of the changes before and after treatment are given in Figure 1.

As this figure demonstrates, there is a general decrease in all the scores after NF treatment. The analyzed scores show a statistically significant decrease (Table 4). The depression score change of -17,88 was found to be significant at a p < 0,01 level as assessed by a repeated measures ANOVA taking into account the intra-subject

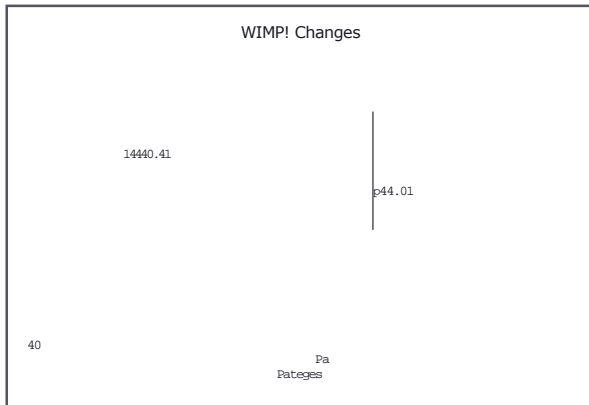


Figure 1. Changes in Pi category scores before and after NF treatment.

variance ($F(1,16) = 27.07, 01,16) = .64$). The same was true for the psychasthenia score which showed a change of -15,41 was also significant at the $p < 001$ level ($F(1,16)=19,42. r^2(1.16)=0.55$). Also, as can be seen in Figure 1, there is a trend towards normalization of all scores from 1-2 standard deviations above the norm, (50 = normal, 60 = 1std, 70 = 2std) to within the norm (under 60).

Long-Term Follow-Up

Two years after the subjects completed their treatment they were followed-up by telephone and queried as to their disposition. The average time of contact after termination of treatment was 26 months. Of the 36 original patients, all were reached. Of these 19 remained symptom free or improved, 9 had developed mild symptoms which did not interfere with their daily functioning, and they did not feel the need to seek treatment, and 5 had relapsed. Of the 2 patients who received medication during treatment, one was in the group that did not respond to treatment. The patient also did not respond to medication and was in the relapse group. The other patient who received medication responded well to NF treatment and remained improved and medication free

CONCLUSION AND DISCUSSION

The goal of this case series was to explore the effect of gEEG guided NF treatment in OCD, since very little information on this topic has been published.

The main NF treatment strategy was to decrease hypercoherence brain areas where the subjects coherence value is higher than the corresponding norm) first, and then decrease individual activities that showed deviations from norm. Overall, the most common training sites were F3, Fz, F4 and the C4-P4 bipolar site.

The case study group assessed in this study showed improvement in all of the scales measured. The subjects reported improvement as measured by the Y-BOCS. The magnitude of the improvement was 21.53 points which was almost double the 10.64 point improvement seen in the average improvement with drug treatment by Ackerman and Greenland.¹ These changes were observed by the physician, in that the group was rated at being severely ill at the start of treatment and were rated as being borderline ill at the end of treatment. This change was also observed by psychological testing as seen in the MMPI results. Not only was there a significant improvement of the scales that were clinically relevant (above 70), but all scores showed a general normalization (e.g., the group's values, after treatment are closer to the normal range than before treatment). Finally, when the subjects were followed-up 2 years

Table 4
MMPI results. Changes in the severity of illness based on the Minnesota multiphasic inventory (MMPI)

	Depression Score		Psychasthenia Score	
	Pre	Post	Pre	Post
Count	17	17	17	17
Mean	74.76	56.88	72.06	56.65
Standard Deviation	11.78	10.37	8.17	10.85
Minimum	50	37	55	37
Median	78	55	73	60
Maximum	90	78	85	71
Mean Difference (Post-Pre)	-17,88		-15.41	
F(1,32)	27,07		19.42	
101,32)	0.64		0.55	
Significance	P < 0,01		P < 0,01	

after treatment, of those from the original group that were contacted (36 out of the 36). 19 remained symptom free or improved, 9 had developed mild symptoms, and 5 had relapsed. Therefore, for the majority of this group of patients, NF treatment was not only effective, its effects lasted up to 26 months after cessation of treatment. These results are congruent with the results of long term follow-ups that have been done in other NF studies.¹¹ The same long-term effect of NF is also seen in this study of OCD.

Only 1 subject received medication (chlomipramine) during the course of the study. This subject did not respond to either NF or medication treatment. A second subject who responded to NF was later found out to have been self-medicating with biperidine. At this juncture it cannot be determined whether the improvement is due to NF treatment, the medication or the combination of the two.

Another important factor that NF may be able to address is learned helplessness, their inability to control their obsessions and compulsions, and the inability of their previous treatments in alleviating their condition reinforced their helplessness in overcoming this disorder. Learned helplessness is seen in people with pessimistic explanatory style — which sees negative events as permanent ("it will never change"), personal ("It's my fault"), and pervasive ("I can't do anything correctly") — are most likely to suffer from learned helplessness and depression.⁵ A common complaint verbalized by all of the subjects in this study was "Am I ever going to get better?", or "Do I have to live with this illness the rest of my life and I should get used to it?" In some cases the fact that they had to get used to living with this illness was conveyed by the physician that they sought treatment from, before coming to our center. Their inability to control their obsessions and compulsions, and the inability of their previous treatments in alleviating their condition reinforced their helplessness in overcoming this disorder. With NF treatment all subjects were actively engaged in their treatment since all of them complied with their schedule and training regimen. In this way the subject's own control systems most probably came into play without any recommendations and/or promoting from the center staff, and they learned how to work to overcome their disorder themselves.

The anatomical basis for OCD is complex and still under investigation although anterior cingulate cortex (ACC) abnormalities are being seen consistently in the pathophysiology of OCD.⁶⁵ The ACC can be divided into cognitive (dorsal) and emotional (ventral) components. The dorsal part of the ACC is connected with the prefrontal cortex and

parietal cortex as well as the motor systems and frontal eye fields." The ventral part has connections to the amygdala, the nucleus accumbens, the hypothalamus, and the anterior insula. It is involved in assessing the importance and relevance of emotional and motivational information. A number of SPECT studies report hyperfrontality (increased right and left anterior prefrontal cortex activity and increased anterior cingulate gyrus activity) and increased basal ganglia activity in obsessive compulsive disorder (OCD).% NF may be involved in helping in the proper self-regulation of these pathways.

The average length of treatment in our study was 1-2 months. This duration is less than seen with pharmacological treatment in OCD. According to the "Practice Guideline For The Treatment of Patients With Obsessive-Compulsive Disorder", prepared by the Work Group On Obsessive-Compulsive Disorder:

Most patients will not experience substantial improvement until 4-6 weeks after starting medication, and some who will ultimately respond will experience little improvement for as many as 10-12 weeks. Successful medication treatment should be continued for 1-2 years before considering a gradual taper by decrements of 10%•25% every 1-2 months while observing for symptom return or exacerbation.

When compared to 2 years of treatment, 1-2 months is favorable. Also when the previous treatment history of this group is taken into account we see that the mean of the total number of medications used in the past is 16 (± 2.2), and the mean of the duration of illness was 8,0 years ($\pm 4.7y.$). This group was able to be medication free and functioning within 2 months whereas they were suffering with their disorder for years, and taking numerous medications with little or no effect A

The goal of this study was to investigate the utility of NF as a treatment for OCD. Although the results were positive there are obvious limitations to this study. The male/female ratio was unbalanced; the treatment duration was not controlled showing variability in the number of sessions necessary for treatment, and the investigator and the patient were not blinded as to the treatment. It would be appropriate and useful to investigate whether these results are replicable with better, more controlled study designs, since in this group of patients we were able to see results comparable to those seen after medication treatment.

DISCLOSURE AND CONFLICT OF INTEREST

T. Surmeli and A. Ertem have no conflicts of interest in relation to this article.

REFERENCES

- Hammond DC. QEEG-guided neurofeedback in the treatment of obsessive compulsive disorder. *J Neurotherapy* 2003; 7: 25-52.
- Hammond DC. Treatment of the obsessional subtype of obsessive compulsive disorder with neurofeedback. *Biofeedback* 2004; 32: 9-12.
- Hammond DC, Neurofeedback with anxiety and affective disorders. *Child Adolesc Psychi Am* 2005; 14: 105-123,
- Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington DC: American Psychiatric Association; 1994: 285-286.
- Pallanti S, Quercioli L. Treatment-refractory obsessive-compulsive disorder. methodological issues, operational definitions and therapeutic lines, *Prog Neuropsychopharmacol Biol Psychiatry* 2006; 30:400-412.
- Pallanti S, Hollander E, Bienstock C, Koran L, Leckman J, Marazziti D, et al. Treatment-refractory OCD: clinical response to treatment with neurofeedback. *Neuropsychopharmacol* 2002; 5:181-191.
- Pallanti S, Quercioli L, Koran L M. Citalopram intravenous infusion in resistant obsessive-compulsive disorder: an open trial. *J Clin Psychiatry* 2002; 63: 796-801.
- Hollander E, Kwon JH, Stein DJ, Broatch J, Rowland CT, Filirnein CA. Obsessive-compulsive and spectrum disorders: overview and quality of life issues, *J Clin Psychiatry* 1996; 57(suppl 8): 3-6.
- Ackerman DL, Greenland S. Multivariate meta-analysis of controlled drug studies for obsessive-compulsive disorder. *J Clinical Psychopharmacology* 2002;22: 309-317.
- Foa ES, Franklin ME. Obsessive-compulsive disorder. In: Barlow DH (ed), *Clinical Handbook of Psychological Disorders*. 3rd ed. New York: Guilford; 2001: 209-263.
- O'Connor K, Todorov C, Robillard S, Borgeat E, Brault M. Cognitive behavior therapy and medication in the treatment of obsessive-compulsive disorder: a controlled study. *Can J Psychiatry* 1999; 44: 64-71.
- Rucklidge JJ. Successful treatment of OCD with a micronutrient formula following partial response to Cognitive Behavioral Therapy (CBI): a case study, *J Anxiety Disorders* 2009; 23: 836-840.
- Simpson HB, Tenke CE, Towey JB, Liebowetz MR, Bruder GE. Symptom provocation alters behavioral ratings and brain electrical activity in obsessive-compulsive disorder: a preliminary study, *Psychiatry Research* 2000. 95:149-155.
- Prichep LS, Mas F, Hollander E, Liebowitz M, John ER, Aknas M. et al. Quantitative electroencephalography (QEEG) subtyping of obsessive compulsive disorder. *Psychiatr Res* 1993: 25-32.
- Insel TR, Donnelly ER, Lalakea ML, Alteman IS, Murphy DL. Neurological and neuropsychological studies of patients with obsessive-compulsive disorder. *Biol Psychiatry* 1983; 18: 741-751,
- Jenike MA, Brotman AA+, The EEG in obsessive-compulsive disorder. *J Clin Psychiatry* 1984; 45: 122-124,
- Pacella BL, Polatin P, Nagler SH. Clinical and EEG studies in obsessive-compulsive disorder. *Am J Psychiatry* 1944; 100: 830-838.
- Rockwell FV, Simons DJ, The electroencephalogram and personality organization in the obsessive compulsive reactions. *Arch Neurol Psychiatry* 1947; 57: 71-80.
- Prichep LS, Mas F, Hollander E, Lebowitz M, John ER, Alman M, et al. Quantitative electroencephalography (QEEG) subtyping of obsessive compulsive disorder. *Psychiatry Research* 1993: 50:25-32.
- Hansen ES, Prichep LS, Bolwig TG, et al. Quantitative electroencephalography in OCD patients treated with paroxetine. *J Clin Electroencephalography* 2003; 34: 70-74.
- Pagarell O, Juckel G, Mavrogiorgou P, Mulert C, Folkerts KJ, Hauke W. et al. Symptom-specific EEG power correlations in patients with obsessive-compulsive disorder. *Int J Psychophysiology* 2006: 62: 87-92.
- Bucci P, Mucci A, Volpe U, Medal E, Gaiderisi S, Ma) M. Executive hypercontrol in obsessive-compulsive disorder: electrophysiological and neuropsychological indices. *Clin Neurophysiology* 2004;115: 1340-1349.
- Bolwig TB, Hansen ES, Hansen A Merkin H, Prichep LS. Toward a better understanding of the pathophysiology of OCD SSRI responders: QEEG source localization, *Acta Psychiatr Scand* 2007: 115: 237-242.
- Tot S, Cage A, Comelekoglu U, Yazici K, Bat, N. Association of QEEG findings with clinical characteristics of OCD: evidence of left frontotemporal dysfunction. *Can J Psychiatry* 2002; 47: 538-545.
- Darting M. School-Based Neurofeedback for Autistic Spectrum Disorder, Neurofeedback for ASD 2007: 1-7. Retrieved from <http://liwaishfamily>

- ceiro.com/clients/2774/documents/School%20Base%20Neurofeedback%20for%20Autistic%20Spectrum%20Disorder.pdf
26. Lubar JO, Luber JF. Electroencephalographic biofeedback of SMR and Beta for treatment of Attention Deficit Disorders in a clinical setting. *Appl Psychophysiol Biofeedback* 1984; 9:1-23.
 27. Luber JE Changing EEG activity through biofeedback applications for the diagnosis and treatment of learning disabled children, *Theory Into Practice*. Ohio State University 1985; 24: 106-111.
 28. Luber JF, Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback Self Regulation* 1991; 16: 201-225.
 29. Luber JF, Lubar JO. Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders (ADD/HD). In: Evans JR, Abarbanel A.(eds). *Introduction to Quantitative EEG and Neurofeedback*. San Diego, CA: Academic Press; 1999.
 30. Luber JF. Neurofeedback for the management of attention deficit disorders. In: Schwartz MS, Andrasik F, (eds). *Biofeedback: A Practitioner's Guide*. 3rd ed. New York; Guilford Press. 2003; 409-437.
 31. Thompson L, Thompson M. Neurofeedback combined with training in meta-cognitive strategies: effectiveness in students with ADD. *Appl Psychophysiol Biofeedback* 1998; 23: 243-263.
 32. Othmer S, Othmer SF, Marks CS, EEG biofeedback training for attention deficit disorder, specific learning disabilities, and associated conduct problems. *J Biofeedback Soc California* 1992; 7: 24-27.
 33. Breteler MHM, Arns M, Peters S, Giepmans I, Verhoeven L. Improvements in spelling after QEEG-based neurofeedback in dyslexia: a randomized controlled treatment study. *Appl Psychophysiol Biofeedback* 2010; 35: 5-11.
 34. Othmer S, Othmer SF, Kaiser DA. EEG biofeedback: training for AD/HD and related disruptive behavior disorders. In: Inoué JA, Mark-Golestein BS, Tessler D. (eds). *Understanding, Diagnosing, and Treating AD/HD in Children and Adolescents*. New York: Jason Aronson; 1999: 235-296.
 35. Othmer S, Othmer SF, Marks CS. EEG biofeedback training for attention deficit disorder, specific learning disabilities, and associated conduct problems. *J Biofeedback Soc California* 1992; 7: 24-27.
 36. Monastra VJ. Electroencephalographic biofeedback (neurotherapy) as a treatment for attention deficit hyperactivity disorder: rationale and empirical foundation. *Child Adolesc Psychiatric Clin N Am* 2005; 14: 55-82.
 37. Arns M, de Ridder S, Stiehl U, Breteler N, Coven A. Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. *EEG Clin Neurosci* 2009; 40: 180-189.
 38. Gani C, Birbaumer N, Strehl U. Long term effects after feedback of slow cortical potentials and of Meta-beta-amplitude as in children with attention-deficit hyperactivity disorder (ADHD). *Int J Bioelectromagn* 2008; 10: 209-232.
 39. Sterman MB. Sensorimotor EEG operant conditioning: experimental and clinical effects. *Pavlov J Biol Science* 1977; 12: 63-92.
 40. Lantz DL, Sterman MB. Neuropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG feedback training. *Epilepsia* 1988; 29:163-171.
 41. Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Olin Electroencephalogr* 2000; 31; 45-55.
 42. Egner T, Sterman MB. Neurofeedback treatment of epilepsy: from basic rationale to practical application. *Expert Rev Neurother* 2006; 6: 247-257.
 43. Sterman MB, Egner T. Foundation and practice of neurofeedback for the treatment of epilepsy. *Appl Psychophysiol Biofeedback* 2006; 31: 21-35.
 44. Tan G, Thomby J, Hammond DC, Strehl U, Canady B, Arneemann K, Kaiser DA. Meta-analysis of EEG biofeedback in treating epilepsy. *Clin EEG Neurosci* 2009; 40: 173-179.
 45. Moore NC. A review of EEG biofeedback treatment of anxiety disorders. *Clin Electroencephalogr* 2000; 31: 1-6.
 46. Thomas JE, Sattlberger E. Treatment of chronic anxiety disorder with neurotherapy: a case study. *J Neurotherapy* 1997; 2: 1-8
 47. Duff J. The usefulness of OEEG and neurotherapy in the assessment and treatment of post-concussion syndrome. *Olin EEG Neurosci* 2004; 35: 198-209.
 48. Pineda JA, Brang D, Hecht E, Edwards L, Carey S, Bacon M, et al. Positive behavioral and electrophysiological changes following neurofeedback training in children with autism. *Res Autism Spect Dis* 2008; 2: 557-581.
 49. Sehmel AG, Fehmi LG, Goldstein DM. Positive outcome with neurofeedback treatment in a case of mild autism. *J Neurotherapy* 1995; 1: 60-64.
 50. Jarusiewicz B. Efficacy of neurofeedback for children in the autistic spectrum: a pilot study. *J Neurotherapy* 2002; 6: 39-49.
 51. Scolnick B. Effects of electroencephalogram biofeedback with Asperger's syndrome. In: *J Rehab Res* 2005; 28: 159-163.
 52. Coben R, Padolsky I. Assessment-guided neurofeedback for autistic spectrum disorder. *J Neurotherapy* 2007; 11: 5-22.
 53. Kouzler MEJ, de Moor JMH, Gernts BJJ, Buitelaar JK, van Schie HT. Long-term effects of neurofeedback treatment in autism. *Res Autism Spect Dis* 2009; 3: 496-501.
 54. Kouzler MEJ, de Moor JMH, Gerrits BJJ, Congedo M, van Schie HT. Neurofeedback improves executive functioning in children with autism spectrum disorders. *Res Autism Spect Dis* 2009; 3:145-162.
 55. Kouzler MEJ, van Schie HT, de Moor JMH, Gerrits BJJ, Buitelaar JK. Neurofeedback treatment in autism: preliminary findings in behavioral, cognitive, and neurophysiological functioning. *Res Autism Spect Dis* 2009; 4: 386-399.
 56. Guy W, (ed). *Clinical Global Impression (CGI)*. ECDEU Assessment Manual for Psychopharmacology. Rockville, MD: U.S. Department of Health Education, and Welfare: 1976.
 57. Maruish ME, (ed). *The Use of Psychological Testing for Treatment Planning and Outcomes Assessment*. 3rd ed. London: Lawrence Erlbaum Associates Publishers; 2004.
 58. John ER. Principles of neurometrics. *Am J EEG Tech* 1990; 30: 251-266.
 59. Schummer G. The disconnection syndrome. *Biofeedback* 2008; 36: 157-162.
 60. Thatcher RW, North D, Corea R, Walker RA, Biver C, IF Gomez hi, Salazar A. An EEG severity index of traumatic brain injury. *J Neuropsychia: Clin Neurosci* 2001;13: 77-87.
 61. Thatcher RW, Biver CL, Gomez-Molina IF, North D, Curtin R, Walker RW, Salazar A. Estimation of the EEG power spectrum by MRI T2 relaxation time in traumatic brain injury. *Olin Neurophysiology* 2001;112: 729-745.
 62. Fisher S. FP02 and the regulation of fear. *ISNR J Newsletter* 2006; 1:117.
 63. Marks PA, Seeman W, Haller DL. *The actuarial use of the MMPI with adolescents and adults*. Baltimore MD: Williams & Wilkins; 1974.
 64. Yiicel M, Wood SJ, Wellard RN, Harrison BJ, Fomito A, Pujol J, Vela-Kouls O, Pantelis C. Anterior cingulate glutamate-glutamine levels predict symptom severity in women with obsessive-compulsive disorder. *Australian New Zealand J Psychiatry* 2008; 42: 467-477.
 65. Posner MI, Di Girolamo GJ. Executive attention: conflict, target detection, and cognitive control. In: Parasuraman R. (ed). *The Attentive Brain*. Cambridge, Mass: MIT Press; 1998
 66. Bush C, Luu P, Posner MI. Cognitive and emotional influences in anterior cingulate cortex. *Trends Cogn So* 2000; 4: 215-222.
 67. Gevenstaben H, Holl B, Albrecht B, Schlamp O, Kratz O, Studer P, et al. Neurofeedback training in children with ADHD: 6-month follow-up of a randomized controlled trial. *Eur Child Adolesc Psychiatry* 2010; 19: 715-724.
 68. Can C, Birbaumer N, Strehl U. Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attention-deficit/hyperactivity disorder. *Int J Bioelectromagn* 2008; 10: 209-232.
 69. Practice Guideline For The Treatment of Patients With Obsessive-Compulsive Disorder. Work Group on Obsessive-Compulsive Disorder. Arlington, VA: American Psychiatric Association: 2007.