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

Tanju Siirmeli and Ayben Ertem

<u>Key Words</u> 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 dinical effects in the treatment of obsessive compulsive disorder (OCD).. In this paper the efficacy of ctEEG-guided NE for subjects with OCD was studied as a case series. The goal was to examine the dinical course of the OCD symptoms and assess the efficacy of gEEG 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 MMP1 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.' It is the fourth most common mental disorder and the tenth leading cause of disability in the world.

There are only 3 published reports!". on NF in the treatment of OCD. Currently, the most widespread treatment modalities for this disorder are pharmacological treatment with serotonin reuptake inhibitors (SR1s) with cognitive behavior therapy (CBT). Despite the proven efficacy of both SR1s and CBT, a substantial percentage of patients receive little benefit from these standard approaches.'

As effective as these treatments are, a response is usually con-%tiered 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." The fact remains that a large fraction of patients without substantial response to standard treatment experience significant morbidity!⁸

When investigating SRIs Ackerman and Greenland' 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," On the other hand, intensive COT has been found to have a 75% remission rate.' O'Connor et al." 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 Rucklidge¹² introduced micronutnents to a patient who did not respond to medications and subsequently underwent CBT with a modest response. Micronutrients worked well with this patient. Ruckfidge 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 eleetrode. For this the gEEG method is quite successful in helping guide the practitioner in placement and band selection since some studies have been done in assessing the gEEG findings in OCD. One of the first qEEG studies conducted on OCD was done by Simpson et al:' In this study, patients' qEEG was recorded under symptom provocation (both live and imaginary). The results indicated that significarit 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. Siimmeli and A. Erten are from the Healthy Living Center for Research and Education. IstanOul. Turkey.

Address correspondence and requests for reprints to Tanis Surmeli, MO, Healthy Living Center for Research and Education. Kore Sehttlen Caddest No 49, Zincirlikuyu, Istanbui 34387 Turkey neuropsychiatrogyahoo.ccor

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

PiircepdeVioured rig a groty classified as OC D. who shared the sews sairiploodalegy, cxiti ce staffed into 2 subgroups by qE.EG. One par teas characterized as ham) diffuse excessive alpha and emotive beta in frontal central and mid-teriporal areas, whereas the other glee!) was cnaractenzed by exar, sive theta activity, especially in the frontal anC posterior-temporal areas. Theta abnormalities have also been reported by Insel et al ." Jenike and Bnotman,.' Palla et al.." and Rockwell and Simons." Furthermore, Phchep et 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 authors found that patents who had high levels of obsessions had nigher absolute EEG power measures. especially in the faster frequencies (alpha2, betel), 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 Burn et al," decrease of the slow a-band power in OCD as compared to healthy subjects was observed. A significant negative correlation between the slow a-band power and me time to complete a neuropsychoogical test exploring executive functions was found: the more reduced the slow a-band power, the slower the performance on this test. Bolwig et found an excess in the alpha range with sources in the corpus striatum, in the orbito-frontal and temporo-frontal regions in untreated OCD patents. 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 frontotemporal areas, when compared against age matched norms.

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

NF is an intervention aimed at training individuals to better regulate tie 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 NE 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 andice 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 tie desired activities thereby conditioning to endure these activities."

NF has been used successfully with ADHD/Learning

epilepsy,""' anxiety.3.4% mild head injuries" and even in autism.4"

the treatment of OCD with NE. Therefore, seeing a need for more information in this area we decided to investigate tie efficacy of gEEGguided NE 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, 812, 86, Folic Acid, THS and urine drug screening for illicit drugs) had to be normal. Exclusion Criteria: The presence of any other psychiatric disorder, nistory 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 (chlomipramine) during NF treatment since it was necessary to manage her symptoms However, she was taking 2 medications at toe time of admission, It was discovered that another patient was self-medicating with bipehdine during the treatment phase. Evaluation measures included family history. QEEG data which was processed with the Nx-Link 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, eacn 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 r 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 = midly 4 = moderately ill; 5 = markedly ill: 6 = severely ill; and 7 = extremely

An MMH 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-fives of the medication they were taking prior to admission. For example, the half-life of chlornipramine 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 quantitiative 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 2- 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 OEEG 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 Zscores 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 Moore^o 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 <u>Finding</u>		Secondary Finding					
Increased relative	20	Increased alpha	10				
alpha power		coherence					
Increased relative	10	Increased theta	3				
theta power		coherence					
Increased relative	4						
beta power							
Increased relative							
theta and beta power Increased relative alpha <u>and beta power</u>							

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 moans 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 Broadmann 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 (i(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.⁶²

FpO₂: 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.

(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				
<u>sults.</u> Changes	in the severity of ill	ness based on the Yale E	Brown Obsessive Com	npulsive Scale		
			Obsession Subscale		Compulsion Subscale	
-					Post	
					2.42	
9,65			5.66		5.26	
	'					
		129.63		31.06		
				0.47		
	P < 0.01	P < 0.01		P < 0.01		
Pure Subgroups		Obsessional Group		Compulsion Group'		
			(reponAg only obsessions will no complsionsl		(reporting only compulsions	
					bsessions)	
					Post	
			3,97		1.67	
			5.00		2.89	
Total Sc	ore	Obsession Sub	oscale	Compulsion Sub	oscale	
		Pre	Post	Pre	Post	
36.44	9,61	18.78	5.06	17.67	4.56	
4,20	13.50	2.05	7.03	3.65	6.78	
-26.83		-13.72		-13.11		
56.48		50,05		47.67		
0.64		0.61		0.60		
P < 0.01		P<0,01		P<0.01		
	Total Sc Pre 27,58 9,65 Total Sc 36.44 4,20 -26.83 56.48 0.64	Total Score Pre Post 27,58 6.06 9,65 10,36 -21,53 134.77 079 P < 0.01	sults. Changes in the severity of illness based on the Yale B Total Score Obsession Su Pre Post Pre 27,58 6.06 17.08 9,65 10,36 5.73 -21,53 .13.44 134.77 129.63 079 0.79 Pre 0.01 P<<0.01	Sults. Changes in the severity of illness based on the Yale Brown Obsessive Com Total Score Obsession Subscale Pre Post Pre Post 27,58 6.06 17.08 3.64 9,65 10,36 5.73 5.66 -21,53 .13.44 134.77 129.63 079 0.79 0.79 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 $30.1y (\pm 9.0y)$. For males the mean age was $25.8y (\pm 5.2y)$ and for females was $32.3y (\pm 9.8y)$. 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 (\pm 9.65 std) (which is above the cut-off score of 16) to 6,06 (\pm 1036), which corresponds to a reduction of 21,53 points A repeated measures ANOVA, where intrasubject 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 th:rd 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.

Ta	ible 3	
	anges in the clinical ons severity score	
<u>Severity</u>	Pre	Post
Mean	6.22	2.03
Standard Devation	0.76	1.75
Mean Difference (Post-Pre)	-4.19	10/5
F(1,35)	205.94	
)1 ² (1,35)	0.85	
Significance	P < 0,01	

The MMPI was admiwstered to all subjects 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 psychastnena 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 modem 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

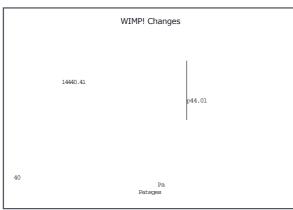
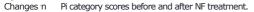


Figure 1.



variance (F(1,16) = 27.07, 01,16) = .64). The same was true for the psyr.tasthenia score which showed a change of -15,41 was also significant at the p < 001 level (F(1,16)=19,42. $rj^2(1.16)=0.55$). 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 intelere with their daily functioning, and they did not feel the need to seek treatment, and 5 had relapsed. Of tie 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 topc nas 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.' These changes were observed by the physician, in that the group was rated at being severely 11 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 dinically relevant (above 701, 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 fo.3owed-up 2 years

		1						
MMPI results. Changes in the severity of illness								
based on the Minnes	ota multipl	nasic inve	ntory (MMPI)				
	Depression		Psychasthenia					
	Score		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 tie original group that were contacted (36 out of the 36). 19 remained symptom free or improved, 9 had developed mild symptoms, and 5 nac relapsed. Therefore, for the malonty 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.""""""""""The same long-term effect of NF is also seen in this study of OCD.

Only 1 subject received medication (chlomipramine) dunng 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 nelplessness, 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 (Ifs 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 tie 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 NP 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 dorsa• 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 acumens, 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 (0CD).% 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 comparedlo 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 (\pm 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 usefui 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 oeurofeedback 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 Blot Psychiatry 2006: 30:400-412.
- 6. Pallanti S, Hollander E, Bienstock C, Koran L, Leckman J, Marazziti D, et
- virterabalitetmenRefactoyOCConsoftmyTitetment"ReponentOCDmetruttuides.esarchpeabaldefionsin Neu ropsychop ha rmacol 2002: 5:181-191.
- Pallanti S. Quercioli L, Koran L M. Citalopram intravenous infusion in resistant obsessive-compulsive disorder: an open trial. J Ctin Psychiatry 2002; 63: 796.801.
- Hollander E, Kwon JH, Stein DJ, Broatch J, Rowland CT, filimeein 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 led), 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 mitronutrient formula following partial response to Cognitive Behavioral Therapy (CBI): a case study, J Anxiety Disorders 2009; 23: 836-840.
- 13. Simpson HB, Tenke CE, Towey JB, Liebovetz MR, Bruder GE. Symptom provocation alters behavioral ratings and brain electrical activity in obses-

sive-compulsive eisorder: a preliminary study, Psychiatry Research 2000. 95:149-155.

- Prichep LS, Mas F, Hollancer E, Liebowitz M. John ER, Aknas M. at al Quantitative electroencephalography (QEEG) subtyping of obsessive compulsive disorder. Psychiatr Res 1993: 25-32.
- Inset TR, Donnelly ER, Lalakea ML, Alterman IS, Murphy DL. Neurological and neuropsychological studies of patients with obsessive-compulsive disorder. Biol Psychiatry 1983; 18: 741-751,
- 16, Jenike MA, Brotman AA+, The EEG in obsessive-compulsive disorder. J Clin Psychiatry 1984; 45: 122-124,
- 17. 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) sublyping of obsessive compulsive disorder. Psychiatry Research 1993: 50:25-32.
- Hansen ES, Pricnep LS, Bolwig TG, at al. Quantitative electroencephalography in OCD patients treated with paroxetine. J Clin Electroencephalogr 2003: 34: 70-74.
- Pagarell 0, Juckel G. Mavrogiorgou P. Mulert C, Folkerts kJ, Hauke W. et al. Symptom-specific EEG power correlations in patients with obsessivecompulsive 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 dinical characteristics of OCD: evidence of left frontolemporal dysfunction. Can J Psychatry 2002; 47: 538-545.
- Darting M. School-Based Neurofeedback for Autistic Spectrum Disorder, Neurofeedback for ASD 2007: 1-7. Retrieved from httpliwaishfamily

;

<u>ceiro.com/dientsf2774idocuments/School%20Baseark20Neu</u> rofeedback% 20forli20Auestic%20Spectrum%200isorder Pdf

- Lubar JO, Luber JF. Eleceoencephaiographic biofeedback of SMR and Beta for treatment of Attention Deficit Disorders in a clinical setting. Appl Psychophysiol Biofeedback 1984: 9:1-23.
- Luber JE Changing EEG activity through biofeedback applications for the diagnosis and treatment of learning disabled children, Theory Into Practice. Onio State University 1985; 24: 106-111.
- Luber JF, Discourse on the development of EEG diagnostics and biofeedback for attention-deficithyperactivity disorders. Biofeedback Self Regulation 1991; 16: 201-225.
- Luber JF, Luber JO. Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders (ADD/HD). In: Evans JR. Abarbanel A.(eds). Introduction to Quantative EEG and Neurofeedback. San Diego, CA: Academic Press; 1999.
- Luber IF. Neurofeeciback for the management of attention deficit disorders. In: Schwartz MS. Andrasik F, reds). Biofeedback: A Practitioner's Guide. 3rd ed. New York; Guilford Press. 2003; 409-437.
- Thompson I_O Thompson M. Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. Appl Psychophysid Biofeedback 1998; 23: 243-263.
- Othmer 5, Othmer SF, Marks CS, EEG biofeedback training for attention def c t disorder, specific learning disabilities, and associated conduct probems. J Biofeedback SOC California 1992; 7: 24-27.
- Breteler MHM, Arns M, Peters S. Giepmans I, Verhoeven L. Improvements in spelling after QEEG-based neurofeedback in dyslexia: a randomized controllec treatment study. Ape(Psychophysiol Biofeedback 2010; 35: 5-11.
- Othmer S. Othmer SF, Kaiser DA. EEG biofeedback: training for AD/HO and related disruptive behavior disorders. In: Incorvaia JA, Mark-Golestein BS, Tessmer D. (ads). Understanding, Diagnosing, and Treating AD/HO in Children and Adolescents, New York: Jason Aronson; 1999: 235-296.
- Othmer S, Othmer SF, Marks CS. EEG biofeedback training for attention deficit disorder, specific learning aisabifities. and associated conduct problems. J Biofeedback Soc California 1992; 7: 24-27.
- Monastra VJ. Electroencephalograpeic biofeedback (neurotherapy) as a treatment for attention oeficit hyperactivity disorder: rationale and empirical foundation. Child Adoles Psychiatric Olin No America 2005: 14: 55-82.
- 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.
- Gani C, Birbaurner N, Strehl U. Long term effects after feedback of slow cortical potentials and of Meta-beta-amplitude as in children with attentiondeficit 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.
- Lantz DL, Sterman MB. Nevropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG feedback training. Epilepsia 1988; 29:163-171.
- Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning, Olin Electroencephalogr 2000; 31; 45-55.
- Egner T. Sterman MB. Neurofeedback treatment of epilepsy: from basic rationale to practical application. Expert Rev Neurother 2006; 6: 247-257.
- Sterman MB, Egrier. Foundation and practice of neurofeedback for the treatment of epilepsy. Appl Psychophysol Biofeedback 2006: 31: 21-35,
- Tan G Thomby J. Hammond DC, Strehl U, Canady B Arnemann K. Kaiser DA. Meta-analysis of EEG biofeedback in treating epilepsy. Clon EEG Neuroso 2009; 40: 173-179.
- Moore NC. A review of EEG biofeedback treatment of aexery 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

- 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.
- S'ehel AG, Fehmi LG, Goldstein DM. Positive outcome with nelirofeedback treatment in a case of mild autism. J Neurotherapy 1995: 1: 60-64.
- Jarusiewicz B. Efficacy of neurofeedback for children in the autistic spectrum: a pilot study. I Neurotherapy 2002:6: 39-49.
- Scolniek B. Effects of electroencephalogram biofeedback with AspergerS syndrome. In: I Rehab Res 2005; 28: 159-163.
- Coben R, Padolsky I. Assessment-guided reurofeedback for autistic spectrum disorder, J Neurotherapy 2007; 11: 5-22.
- Kouilzer MEJ, de Moor JMH. Gernts BJL, Buitelaar JK, van Schie HT. Long-term effects of neurofeedback treatment in autism. Res Autism Spedt Dix 2009; 3: 496-501.
- Kou#er MEJ. de Moor INN, Gerrits BJL, Congedo M, van Schie HT. Neurafeedback improves executive functioning in children with autism spectrum disorders, Res Autism Spec', Dix 2009; 3:145-162.
- Kouitzer MEJ. van Schie HT, ae Moor JMH, Gerrits BJL, Buitelaar JK. Neurofeedback treatment in autism; preliminary findings in behavioral, cognitive, and neurophysickgical functioning. Res Autism Sped t Dis 2009. 4: 386-399.
- 56, Guy W, (ad). Clinical Global Impression (CGI). ECDEU Assessment Manual for Psychopharmacology. Rockville, MD: U.S. Department of Health Education, and Welfare: 1976.
- Maruish ME, (ad). The Use of Pasychological Testing for Treatment Planning and Outcomes Assessment. 3rd ad. London: Lawrence Ellbaum Associates Publishers; 2004.
- John ER. Principles of neurometrics. Am J EEG Tech 1990; 30: 251-266.
- Schummer G. The disconnection syrkircme. Biofeedback 2008: 36: 157-162.
- 60, Thatcher RW, North 0. 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.
- 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.
- Fisher S. FP02 and the regulation of fear. ISNR J Newsletter 2006; 1:117.
- Marks PA, Seeman W, Haller DL. The actuarial use of the MMPI with adolescents and adults.Baltimore MD: Williams 8 Wilkins; 197⁴.
- Yiicel M. Wood SJ, Wellard RN, Harrison BJ, Fomito A. Pujol J, VelaKouls 0, Pantelis C. Anterior cingurate glutamate-glutamine levels predict symptom severity in women with obsessive-compulsive disorder. Austrarian New Zealand J Psychiatry 2008; 42: 467-477.
- Posner MI, Di Girolamo GJ. Executive attention: conflict, target detection, and cognitive control. In: Parasuraman R. (ad). 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.
- Gevenstaben H, Holl B, Albrecht B, Schlamp 0, Kratz 0, 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,
- Can C. Birbaurner N. Strehl U. Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attentiondeficit/hyperactivity disorcter, Int J Bioelectromagn 2008; 10: 209-232.
- Practice Guideline For The Treatment of Patients With Obsessive-Compulsive Disorder. Work Group on Obsessive-Compulsive Disorder. Arlington, VA: American Psychiatric Association: 2007.