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SPECIAL ISSUE

Anxiety Associated With Post Traumatic Stress Disorder—The Role of Quantitative Electroencephalograph in Diagnosis and in Guiding Neurofeedback Training to Remediate the Anxiety

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The literature regarding neurofeedback treatment of anxiety associated with post traumatic stress disorder (PTSD) is reviewed. The results of quantitative electroencephalograph (QEEG) guided neurofeedback training for anxiety in nineteen PTSD patients is analyzed, along with the change in anxiety in four control patients who did not do neurofeedback. Those who did neurofeedback training experienced clinically significant reductions in anxiety, whereas there was no significant change in anxiety in the control group. QEEG-guided neurofeedback appears to be effective in a higher percentage of patients than non–QEEG-guided training in increasing alpha and theta, based on results in the published literature.

Introduction

A high incidence of electroencephalograph (EEG) and quantitative EEG abnormalities are found in anxious individuals (Small, 1993; Jenike & Boatman, 1984; Abraham, 1989; Louie, Lamson, & Ketter 1998; Abraham & Duffy, 1991). A decline in alpha activity has been reported in anxiety disorder using quantitative electroencephalograph (QEEG) (Buchsbaum, Hazlett & Sicotte, 1985; Siciliani, Schiavon, & Tavella, 1975; Heller, Nitschke, Etienne, & Miller, 1997). Neurofeedback has been used to reduce anxiety in several studies (not based on QEEG findings) (Moore, 2003). Typically, biofeedback is used to reward an increase in alpha activity, and theta increases may also be rewarded. In a randomized controlled study of veterans with post traumatic stress disorder (PTSD) (Peniston & Kulkosky, 1991), occipital alpha/theta training (along with traditional psychotherapy) resulted in improvement in all 10 MMPI clinical scales, whereas psychotherapy alone did not affect MMPI scores in the control subjects. Over a 26-month period, only four of the neurofeedback patients experienced reoccurrence of occasional flashbacks or nightmares. At follow-up (26 months), all 14 traditionally treated patients had experienced relapse whereas only 3 of 15 neurofeedback patients experienced relapse of symptoms sufficient to meet the criteria for PTSD.

In another V.A. hospital uncontrolled study (Peniston, Marrineau, Deming, & Kulkosky, 1993), 20 Vietnam veterans with chronic PTSD and comorbid alcohol abuse were treated with thirty 30-minute sessions of occipital alpha-theta neurofeedback training. Only 4 of the 20 patients reported an occasional reoccurrence of nightmares or flashbacks, and the other 16 patients had no reoccurrence of PTSD symptoms.

Hammond (2003) was the first therapist to use QEEG as a guide to neurofeedback for anxiety associated with obsessive compulsive disorder. Each individual had different QEEG abnormalities. Improvements were documented with the Yale Brown Obsessive Compulsive Scale (Y-BOCS) and Padua inventories, as well as Minnesota Multiphasic Personality Inventory (MMPI). The changes were maintained at 15 and 13 months. A similar improvement was noted in another patient who had QEEG guided neurofeedback training by Hammond (2004).

The QEEG abnormalities in 100 anxious patients were reported by Gurnee (2003). He described 6 QEEG subtypes—high beta, high alpha, low alpha, cingulate dysfunction, high mean frequency beta, and high mean frequency alpha. He reported that training designed to normalize the different QEEG patterns usually resulted in relief of the anxiety. His QEEG's were done using the NXLink Database (John, Prichep, Fridman, & Easton, 1988) which includes 13–25 Hz as beta, and does not separate out 21–30 Hz activity, which is done using the most recent Neuroguide data base, which we have used in evaluating and treating our patients (Thatcher, Walker, Biver, North, & Curtin, 2003).

Methods

This study included 23 patients seen in our clinic with a diagnosis of PTSD. Their ages ranged from 18 to 65, (mean) ±

10.2 (SD). There were 23 women and 10 men. They were asked to judge their average persistent anxiety on a scale of 1–10 over the last 5 years. Each of them had a QEEG examination, using the Thatcher Neuroguide database. Excessive high frequency beta (21–30 Hz) was then downtrained for 5–7 sessions for each site where there was excessive high frequency beta. Ten Hz activity was uptrained at the same sites (see Table 1). Following neurofeedback training, in 19 subjects, the patient was asked to again judge his level of anxiety (1–10) on a persistent basis. At 1 month posttraining they were asked again to judge their persistent anxiety on a scale of 1–10. Four patients who declined neurofeedback training were interviewed 3 months after their QEEG, and asked to judge their level of anxiety at the beginning and end of the 3-month period. These four subjects were felt to represent a no-treatment control group.

Results

Incidence of QEEG abnormalities in the total group (see Table 2). The abnormalities seen in the group as a whole, with a significance level of ≤.05, are seen in the table (Chi-squared analysis). The abnormalities investigated included the absolute and relative incidence of focal delta excesses (1–3 Hz), focal theta excesses (4–7 Hz), focal alpha excesses (8–12 Hz), focal beta excesses (13–20 Hz), and focal high frequency beta excesses (21–30 Hz). The only significant findings were in the excessive high frequency beta domain.

The effect of training high frequency beta abnormalities on the degree of anxiety pre- and posttraining and at 1 month after the completion of training. Table 1 shows the training for each subject, showing cortical sites trained, specific frequency ranges trained, and the anxiety self-ratings at each sampling time.

Table 1. The effect of training high frequency beta abnormalities on the degree of anxiety pre- and posttraining and at 1 month after the completion of training

Patient	Sites Trained	Frequencies Trained (Hz)	Number of Sessions	Pretreatment Anxiety	Posttreatment Anxiety	Anxiety 1 Month after Training
1	FP1, FZ, FP2, P3	↓21–30/↑10	7 ea.	6/10	2/10	2/10
2	CZ, FZ, PZ, C3	↓21–30/↑10	7 ea.	7/10	2/10	2/10
3	T5, F8, C3, CZ, FZ, FP2	↓21–30/↑10	7 ea.	7/10	2/10	2/10
4	T3, T4, T5, T6	↓21–30/↑10	5 ea.	7/10	2/10	2/10
5	C3, C4, F3, F4	↓21–30/↑10	6 ea.	7/10	2/10	2/10
6	CZ, FZ	↓23–30/↑10	7 ea.	6/10	1/10	1/10
7	FP2, F4, FP1	↓21–30/↑10	5 ea.	7/10	1/10	1/10
8	FP2, F8, FP1, Cz	↓21–30/↑10	7 ea.	6/10	1/10	1/10
9	P3, P4, F3, F4	↓23–30/↑10	7 ea.	6/10	1/10	1/10
10	FZ, C3, F3, P3	↓27–30/↑10	5 ea.	6/10	2/10	2/10
11	FZ, F2	↓21–30/↑10	5 ea.	6/10	1/10	1/10
12	P3, P4, O1, O2	↓22–30/↑10	5 ea.	6/10	1/10	1/10
13	O2, O1, P3, P4	↓22–30/↑10	5 ea.	6/10	1/10	1/10
14	FP1, FP2, FZ , F7	↓27–30/↑10	5 ea.	6/10	1/10	1/10
15	P3, P4, F1, T3	↓25–30/↑10	5 ea.	6/10	1/10	1/10
16	FP2, FP1	↓21–30/↑10	5 ea.	5/10	0/10	0/10
17	F3, F4, C3	↓21–30/↑10	5 ea.	6/10	1/10	1/10
18	C3, C4, P3, P4	↓21–30/↑10	5 ea.	5/10	0/10	0/10
19	P3, P4	↓21–30/↑10	5 ea.	6/10	1/10	1/10

Table 2. Localization of significant frequencies associated with moderate to severe anxiety		
Abnormality	**Frequency (%)**	**P (Chi-Squared)**
Excess relative power high frequency beta F4	58	<.05
Excess relative power high frequency beta P3	58	<.05
Excess relative power high frequency beta P4	58	<.05
Excess relative power high frequency beta T5	55	<.05
Excess relative power high frequency beta T7	55	<.05
Excess relative power high frequency beta Tz	50	<.05
Excess relative power high frequency beta Cz	50	<.05
Excess relative power high frequency beta FP1	50	<.05
Excess relative power high frequency beta F3	46	<.05
Excess relative power high frequency beta O2	46	<.05

Table 3. Result of no training in four subjects who had QEEGs, but chose not to do training (control group)			
Patient	**Sites with Excess Relative High Frequency Beta Activity**	**Initial Anxiety Level (1–10)**	**Anxiety Level 3 Months Later (1–10)**
1	C4	6/10	6/10
2	F3, F4, T5	7/10	7/10
3	F7, F8, T4	7/10	6/10
4	F3, F4	5/10	5/10

Result of a no-training condition in 4 subjects who had QEEG's, but chose not to do training (control group). Table 3 shows the anxiety levels in four subjects who declined treatment, over the same three month period.

The control subjects experienced little or no decrease in their chronic anxiety. In contrast, all subjects who did training to decrease excessive high frequency beta and increase 10 Hz activity experienced significant improvement in their chronic anxiety. These were patients with moderately severe anxiety (5/10–7/10) prior to training. At the end of training, all experienced a clinically significant reduction in anxiety (0/10–2/10). The reduction in anxiety was stable 1 month after the completion of training (still 0/10–2/10).

Discussion

Using QEEG to guide neurofeedback training to reduce anxiety appears to be quite useful, resulting in clinically significant reductions in anxiety, which appear to be stable. It should be noted that in earlier studies, non-QEEG based neurofeedback for anxiety probably included reduction of high frequency beta. Typically, when alpha and theta are rewarded, high frequency beta activity is inhibited. In most of these earlier studies an occipital placement was used. In our population, high frequency beta was usually not present occipitally, with more frequent frontal, central, and parietal localizations. I suspect that training the areas where the high frequency beta is localized will prove more effective than training non-affected areas (such as the occipital and temporal areas). On the other hand, occipital alpha/theta training might be found to reduce high frequency beta in parietal, central, and frontal areas. Pre- and post-QEEGs would need to be done to see if that is the case.

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