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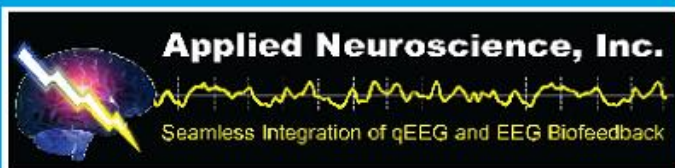Joseph E. Thomas Ph.D. & Elizabeth Sattlberger B.A.
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Treatment of Chronic Anxiety Disorder with Neurotherapy: A Case Study

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The objective of the present case study is to report the effects of alpha-decrease biofeedback training on a patient diagnosed with Anxiety Disorder. Three Minnesota Multiphasic Personality Inventories (MMPI and MMPI-2) were used as objective mea - sures of treatment efficacy. Following 15 sessions of slow wave inhibit/fast wave increase EEG feedback training, the patient reported a significant reduction in anxi - ety-related symptoms. At three year follow-up, results of an MMPI-2 showed all clini - cal scales within normal range. In addition, self-reports confirmed that the patient was symptom free. After treating the patient with several other clinical modalities, only the alpha-decrease feedback training produced effective, long-term improvement of symptoms.

Conventional wisdom suggests that an anxious person needs to be calmed down. In addition to traditional psychotherapy and drug therapy, treatments of anxiety disorders include low arousal training methods, such as progressive relaxation, autogenic training, meditation, and biofeedback. In biofeedback training, the modalities most commonly used are electromyogram (EMG) feedback to reduce muscle tension, thermal feedback to reduce vascular tension, and electroencephalogram (EEG) feedback to reduce cerebral tension.

The early history of EEG biofeedback in the treatment of psychological disorders was based on the observations of individuals in altered states of consciousness (Schwartz, 1987). Individuals in meditative states exhibited high-amplitude alpha activity and a deeply relaxed psychological and physiological state. Based on these observations, biofeedback training focused on increasing alpha activity for patients with anxiety disorders. Hardt and Kamiya (1978) reported that training to increase EEG alpha had a distinct axiolytic effect at least in subclinical anxious college students. However, Plotkin and Rice (1981) found that EEG alpha increase as well as EEG alpha decrease had an anxiety-reducing effect on a similar pop-

ulation. It was found that some people with high alpha amplitude also reported high lev-els of anxiety. In view of this inconsistency, EEG biofeedback in the treatment of anxiety disorders was largely given up by clini-cians in favor of EMG biofeedback. How-ever, EMG training also found conflicting results in reducing anxiety. Raskin, Johnson, and Rondestradt (1970) used EMG training on seven patients. Of the seven, six patients showed no improvement in anxiety symptoms.

In another study by Rice, Blanchard, and Purcell (1993), comparing frontal EMG biofeedback to EEG biofeedback (training either to increase or decrease alpha activi-ty), researchers found that, although all three training methods worked in reducing anxiety, "only alpha-increase biofeedback subjects showed significant reductions in heart rate reactivity to stressors at a sepa-rate psychophysiological testing session" (Rice et al., 1993, p. 93).

Hurley and Meminger (1992) suggest that these inconsistencies can be attributed to the way anxiety is assessed in individual patients (state versus trait anxiety) and the actual length of treatment, with longer peri-ods of treatment reducing anxiety more effectively.

We have successfully treated six patients suffering from Chronic Anxiety who exhibited abnormally high levels of slow wave activity by training them to inhibit theta/alpha and increase beta. The following case study was selected for two reasons: (a) The patient has a lifelong history of symptoms of moderate to severe anxiety, and (b) the patient was handled with a variety of treatment modalities in our office over a span of twelve years, before initiation of alpha-decrease EEG training. In addition, we were able to follow up with the patient to assure maintenance of recovery. The purpose of this case study is to show that alpha-decrease EEG training is effective in treating anxiety when the patient already exhibits abnormally high alpha wave activity and to suggest a differential treatment approach to treating anxiety, based on the individual patient.

Method

Subject

The patient was a self-referred, married, white female who sought treatment with us in April, 1981 and continued to see us for therapy until October, 1993. At the time of initial evaluation, she was 31-years-old. Presenting complaints included nervousness, low self-esteem, fears of eating and choking on food, numbness in the left arm, and a "bubbling" sensation in the throat. In addition, she complained of having difficulty breathing and a fear of getting dizzy whenever she felt nervous. The patient reported that she had always been a nervous person, but the current anxiety symptoms started two years ago during her last pregnancy, when she developed a fear of being "fat and ugly." This was followed by a panic attack suffered at a party. Soon she developed a fear of swallowing certain kinds of solid foods such as chicken, sausage, lettuce, and celery. A medical evaluation showed no physical basis for the presenting complaints.

Birth, infancy, and childhood were reported to be unremarkable. The patient's parents were both alive at the time of initial evaluation. The patient has an older brother and a younger sister. Her brother was previously diagnosed with Panic Disorder by his own psychiatrist. The patient reported that her parents as well as her siblings have "nervous" problems. The father is a perfectionist and experienced periods of panic whenever he was home alone. The mother suffered a nervous breakdown after her first pregnancy, similar to what the patient reported after her most recent pregnancy. The nervous breakdown suffered by the mother was reported to be a brief episode and she enjoyed good health until dying of cancer in 1984.

Measures

Three Minnesota Multiphasic Personality Inventories (MMPI and MMPI-2) were administered during the course of therapy: (a) the first at initiation of therapy (1981), (b) the second following alpha-decrease EEG training (1993), and (c) the third at three year follow-up (1996). This measure was selected as a means of evaluating the patient's presenting complaints and assessing the effectiveness of treatment.

In addition, self-reports and interviews with the patient's husband were used at the end of EEG training and at follow-up to further assess the efficacy of therapy.

Treatment

After an evaluation interview and two sessions of progressive relaxation training, the first MMPI (see Table 1), was administered. The patient's MMPI profile (Welsh Code 2" 7'036-814/95:F/K:L) showed significant elevations on Scale 2 (T-score 82) and Scale 7 (T-Score 73), and near significant score on Scale 0 (T-score 73). On the basis of her presenting complaints, her psychiatric history and her MMPI profile, we gave a diagnosis of Anxiety Disorder with Passive Dependent Personality. Although the patient scored high on Scale 2 (Depression), the depressive features seen on the MMPI could only be secondary to the chronic panic attacks, anxiety, and phobic conditions.

Previous treatments consisted of psychopharmacological interventions with no significant relief of symptoms. At the time the patient was referred to us, she was taking Xanax (5 mg daily). She continued her medication in consultation with her physician during part of her psychological therapies. However, she was not known to be using any medication during the time she underwent neurotherapy.

The initial treatment plan consisted of low arousal training followed by psychotherapy. Over the course of twelve years, various methods were attempted to relieve anxiety symptoms, including: (a) an adaptation of Jacobson's progressive relaxation technique, (b) EMG biofeedback, (c) thermal biofeedback (right and left digitalis), (d) Wolpe's systematic desensitization, (e) assertiveness training, (f) hypnotherapy, (g) visualization, and (h) cognitive behavioral therapy. Relaxation practice gave the patient only a transient feeling of calmness and there was no carry over into her daily life. The patient showed very little response to any insight-oriented psychodynamic therapy and attempts in that direction proved unproductive.

The patient's fear of eating subsided gradually in response to systematic desensitization and therapy was discontinued in September 1983. Six months later, the patient returned with complaints of anxiety and severe marital problems. The patient was currently going through a separation and divorce. She had started working again; however, she was experiencing episodes of anxiety, and every new situation was overwhelming to her.

The patient remained at her current job, although she was dissatisfied with her employment. Fear of new changes inhibited her from interviewing for a new job, transferring to another division, or accepting a promotion.

Anxiety surged to a new high when the patient started dating again. She was petrified of contracting AIDS and avoided all physical contact. During this time, she continued to seek psychotherapy treatments two to three times per month. The treatment approach was mostly cognitive behavioral. The patient responded well during therapy sessions, but could not translate this into everyday life. She experienced panic attacks and anxiety when the next situation presented itself, sometimes experiencing symptoms within hours after a therapy session.

In November, 1991 we introduced the patient to EEG feedback training, using the Lexicor Neurosearch-24, to relax and remove tension. The original protocol was to increase alpha (8-12 Hz) and decrease beta (16-20 Hz). Initial baseline readings showed that the patient's dominant brain wave frequency was already alpha (amplitude greater than 35 Hz). Based on this observation, it did not seem useful to increase alpha any further. Instead, we introduced a new EEG protocol, focusing on decreasing alpha and increasing beta.

The patient received weekly sessions of alpha-decrease/beta-increase training. Feedback was contingent upon the presence of beta and the suppression of alpha, EMG, and movement. The threshold was set at 40% reward for beta and 60% inhibit for alpha. The threshold was increased when the patient maintained 60% reward and 40% inhibit.

The Standard International (10-20) Electrode Placement System was used for determining electrode placement (Jasper, 1958). Based on this system, five Ag-AgCl electrodes were placed at the following locations: (a) one at Mid-Frontal (Fz), (b) one at Mid-Parietal (Pz), (c) one at Ground, and (d) one on each earlobe for reference. The two active electrodes used for feedback training were Fz and Pz; all other electrodes were neutral.

The patient received both audio and visual feedback. Aurally, the patient heard a tone when the protocol was successfully met; visually, the patient watched a computer screen which indicated when she met the defined threshold. After 15 weekly EEG

feedback sessions, treatment was cut back to bimonthly sessions.

Results

After ten sessions of alpha-decrease/beta-increase training, the patient reported significant decreases in anxiety. Four months after initiation of therapy, the patient experienced a minor panic attack at work. She reported that the attack was mild and that she was able to pull herself together without any help. After 15 EEG feedback sessions, the patient was able to accept and fulfill a new job assignment, which did not evoke panic as it had in the past.

After therapy was cut back to one to two sessions per month, the patient's progress continued to be steady. In January, 1993 she was able to go for new job interviews and reported feeling free of anxiety. A second MMPI-2 was administered at this time and showed a profile with all clinical scales within normal range (see Table 1; Welsh code 4-3276189/05: K-F/L). A follow-up MMPI-2, done three years after termination of therapy, also showed a normal profile (see Table 1; Welsh code 349-27/01685: K-F/L). In a recent telephone interview, the patient reported being symptom free as well as being able to handle her job, marriage, and family life. In addition, two separate interviews with the patient's husband confirmed the fact that the patient is functioning well. Both of these interviews were conducted during the follow-up period.

Discussion

In our experience, we found that EEG alpha-increase is beneficial only for patients who exhibit low amplitude alpha. Patients with anxiety who show abnormally high levels of alpha at baseline readings do not respond effectively to alpha-increase biofeedback. Instead, these patients report a continuation or even an increase in anxiety with low arousal training. Abatement of symptoms occurs only after these patients are trained to decrease alpha activity. It seems that the presence of abnormally high levels of slow wave activity on a day-to-day basis leads to an inability to organize one's cognitive, perceptual, and emotional world. The anxiety in these patients arises when they are confronted with a task or life event that they are unable to organize and handle effectively, leading to feelings of helplessness and of being overwhelmed. Furthermore, such patients do not respond well to

Table 1

Patient's Pre-treatment, Post-treatment, and Follow-up MMPI and MMPI-2 T-Scores.

Scales	L	F	K	1	2	3	4	5	6	7	8	9	0
				Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma	Si
Pre-treatment	46	58	48	54	82	63	53	43	62	73	55	43	69
Post-treatment	50	55	61	52	59	59	62	43	53	58	52	50	49
Follow-up	50	40	57	48	57	61	60	41	47	55	46	60	49

Note: Pretreatment T-scores from MMPI; Post-treatment and Follow-up T-scores from MMPI-2.

traditional psychotherapy, ruling out the possibility of unconscious conflicts or traumas as a cause of anxiety.

This case report attempts to demonstrate the application of EEG feedback to reduce anxiety by decreasing alpha- and increasing beta-wave activity. Although this study cannot resolve the inconsistent results reported in EEG alpha feedback to treat anxiety (Plotkin & Rice, 1981; Raskin et al., 1970), it may offer a basis of differentiation in therapeutic technique. These findings may lend further support to the contention that inconsistencies in the treatment of anxiety may be based upon the way anxiety is assessed in patients (Hurley & Meminger, 1992). Further studies should address the possible differences in brainwave activity in anxiety sufferers as a basis for differential treatment.

It can be argued that the other treatment modalities (Jacobson's Progressive Relaxation Technique, EMG biofeedback, Thermal biofeedback, etc.) ameliorated the patient's anxiety. However, it must be noted that the patient suffered a relapse in symptoms six months after termination of said treatment. Although her fear of eating subsided, other reported physical complaints and symptoms of anxiety persisted. With the introduction of the EEG alpha wave inhibit/beta wave increase biofeedback, the patient showed significant reductions in symptoms within a short period of time, suggesting a significant impact of therapy. In addition, at three-year follow-up, there was no relapse of detectable symptoms, suggesting a long-term effect of the therapy.

However, it should also be noted that an MMPI was not administered at the beginning of alpha-decrease EEG feedback. As a result, we do not have an objective measure of the patient's anxiety level and extent of symptoms prior to initiation of this modality of treatment. But clinically, prior to the introduction of the alpha wave inhibit/beta wave increase biofeedback, the patient continued to report recurrent panic reactions and pervasive anxiety. The new modality of

treatment reported here was introduced only after other attempts at addressing the problem proved unsuccessful. It is recommended that subsequent studies should control for pre- and post-measurements of psychophysiologic and psychological profiles to better assess the impact of therapy.

Furthermore, it should be noted that all treatments referred to in this paper were carried out by the same therapist. Therefore, the improvement in the patient's symptoms can be attributed to the slow wave inhibit/fast wave increase biofeedback, and not to a therapist effect.

In conclusion, it is our impression that neither increasing nor decreasing EEG alpha should be used as a uniform protocol for the treatment of anxiety disorders. Instead, each case should be planned individually, depending upon patient history and baseline EEG patterns. Finding a balance in the EEG pattern may be more important than increasing or decreasing EEG alpha. A controlled study on a larger population is strongly recommended and our observations are only suggestive of the need for a differential approach to neurotherapy.

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