

## **A Review of Research on Anxiety Disorders and Neurofeedback Training**

The following entry on anxiety disorders and neurofeedback training is excerpted from an article published in the peer reviewed journal **Child and Adolescent Psychiatric Clinics of North America** 14 (2005) pp 105-123. The article is written for an audience of child and adolescent clinicians, but reviews the general research literature, usually conducted on adults, for neurofeedback and anxiety.

### **Neurofeedback with Anxiety and Affective Disorders**

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#### **(Excerpted section on Anxiety Disorders specifically)**

A review of the literature on the neurofeedback treatment of anxiety disorders was conducted by Moore [58]. He was able to identify eight studies of generalized anxiety disorder, three studies with phobic anxiety disorder, two studies of OCD, and one report of using neurofeedback with PTSD. He noted several problems with this literature. One problem was that most of the research studies only used brief neurofeedback training in comparison with what clinicians tend to do. For example, in the generalized anxiety disorder studies, treatment only averaged 3.2 hours, whereas clinicians often anticipate needing 7 to 12 hours of neurofeedback training with anxiety problems. The eight studies of generalized anxiety disorder also only averaged 6.25 subjects per study, but seven of the eight studies that he reviewed produced positive changes in clinical outcome. The best studies of neurofeedback with anxiety were three outcome studies [59] with phobic (test) anxiety. These studies included random assignment, four alternative treatment control groups, and a wait-list control group. In one study, the group that received alpha EEG enhancement training produced 33% more alpha after treatment, and all three feedback groups (who received alpha enhancement biofeedback, electromyography [EMG] [muscle] biofeedback, and alpha plus EMG biofeedback) demonstrated significant reductions in test anxiety. In comparison, the untreated control group and the relaxation training group experienced no significant reduction. In another study, subjects received phases of alpha enhancement training and EMG biofeedback training. The alpha training was found to increase alpha production from 64% to 78%, and anxiety scores dropped significantly ( $P < 0.001$ ) for this combined treatment group compared with a nontreatment group. Moore [58] concluded in his review that a placebo effect was present in these neurofeedback studies but that alpha and theta enhancement training provided additional effects beyond placebo and are effective treatments for anxiety disorders. When these results are compared with the American Psychological Association Clinical Psychology Division criteria [60,61] and comparable biofeedback specialty criteria [62] for evaluating the status of efficacious treatments, neurofeedback for phobic anxiety qualifies for the status of a probably efficacious treatment.

Before proceeding further, an outline of these guidelines for evidence-based support is reviewed. According to the biofeedback efficacy guidelines [62], the status of “possibly efficacious” is accorded for treatments that have been investigated in at least one study and had sufficient statistical power and well identified outcome measures but lacked randomized assignment to a control condition internal to the study. For the last two decades, randomized, controlled trials have been emphasized as the scientific gold standard by the pharmaceutical industry, in medicine, and in the recent clinical psychology guidelines for defining empirically supported therapies. Recently, however, this academic “gold standard” has been challenged by two research reports in the scientifically prestigious *New England Journal of Medicine* [63,64] and another study [65]. The three studies discovered that results from nonrandomized observational studies were similar to randomized, controlled trials. To attain the lower evidence-based status of “possibly efficacious,” a randomized, controlled trial was deemed unnecessary.

The biofeedback efficacy guidelines define a treatment as meriting the status of “probably efficacious” when multiple observational studies, clinical studies, wait-list controlled studies, and intrasubject or within-subject replication studies demonstrate efficacy. A biofeedback treatment is considered to have reached the higher “efficacious” status when research by at least two independent research groups (which has included comparison with a no-treatment control group, alternative treatment group, or sham/placebo control group with randomized assignment) has found that the experimental treatment is significantly superior statistically to control conditions or equivalent to a treatment of established efficacy. Finally, a biofeedback treatment is considered as having reached the status of “efficacious and specific” if, in addition to the previous criteria, the treatment has been demonstrated to be statistically superior to a credible sham therapy, pill, or bona fide treatment in at least two independent studies. With regard to requiring placebo-controlled studies to establish efficacy for psychological treatments, however, in which a known effective treatment is already available, this has been deemed unethical by medical ethicists [66,67] and by the Declaration of Helsinki of the World Medical Association [68]. Supporting the Declaration of Helsinki, a university Institutional Review Board (IRB) committee deemed that a study proposal to include a placebo control condition compared with neurofeedback to treat attention deficit disorder and ADHD would be considered unethical because a medication treatment with known effectiveness existed already for this condition [69].

Returning to the literature review, two relevant studies of neurofeedback for the treatment of anxiety were not reviewed by Moore [58]. Passini et al [70] used 10 hours of alpha neurofeedback training, comparing 25 anxious patients (23 of whom were alcoholics) with a control group of 25 anxious patients (22 of whom were also alcoholics), most of whom were seeking treatment at a Veterans Administration hospital brief treatment unit. While most subjects were assigned to one group or the other randomly, deliberate placement of younger patients in the control sample occurred toward the end of data collection and was implemented to offset an age difference that had developed earlier between the groups. Thus, this would be considered to be a matched control group study. Although they did not evaluate drinking status, the alpha neurofeedback training produced significant ( $P < 0.001$ ) changes in state and trait anxiety compared with controls. This was accompanied by an increase in eyes-closed alpha production from 38% to 55%, whereas controls dropped slightly. An 18-month follow-up of those patients was published, with virtually identical results of lower anxiety still found, which validated that the anxiety changes from alpha neurofeedback were enduring [71]. A recent randomized, blinded, controlled study was conducted at London’s Royal College of Music to evaluate the ability of alpha-theta neurofeedback to enhance musical performance in high talent level musicians when they were performing under stressful conditions in which their performance was being evaluated [72]. When compared with five alternative treatment groups, only the neurofeedback group that received training to increase alpha and theta resulted in enhancement of real-life musical performance under stress. These results qualify under the guidelines reviewed earlier as meeting probably efficacious status for neurofeedback treatment of anxiety.

Two neurofeedback outcome studies have focused on chronic PTSD, only the first of which was reviewed by Moore [58]. In a randomized, controlled group study [73], 30 30-minute sessions of alpha-theta EEG biofeedback training were added to the traditional Veterans Administration hospital treatment that was provided to a group of 15 Vietnam combat veterans with PTSD. The study compared them after treatment and at follow-up with a contrast group of 14 veterans who only received traditional treatment. One strength of this study is that in addition to the posttreatment testing, on a monthly basis, patients and informers were contacted for a full 30-month follow-up period to determine if there had been PTSD symptoms (eg, flashbacks, nightmares, anxiety attacks, depression). At follow-up, all 14 traditional treatment patients had experienced relapse, whereas only 3 of 15 neurofeedback training patients had experienced relapse. Another outcome measure involved psychotropic medication requirements. Medications were equivalent at the onset of treatment, with 14 of the

neurofeedback group receiving medication and 13 of the 14 standard Veterans Administration hospital treatment group on medication. All 14 patients who were treated with neurofeedback had decreased their medication requirements at follow-up, whereas in contrast, only 1 traditional treatment patient had decreased medication needs, 2 reported no change, and 10 required more medications. Changes on the MMPI may be seen in [Figs. 1 and 2](#). Neurofeedback training patients improved significantly on all ten MMPI clinical scales—in many instances dramatically—but there were no significant improvements on any scales in the traditional treatment group.

In examining the figures, T-scores may be seen down the left hand side of each figure. A T-score of 50 represents the mean average of a “normal” population, and only 2.5% of normals score higher than the heavy line that goes across the figures at T-score 70. For readers unfamiliar with the MMPI, a brief overview of what the clinical scales measure is helpful. The first three scales (L, F, and K) are validity scales. When the F scale is elevated, as it is in these two samples, it is associated with an endorsement of more problematic symptoms. Scale 1 measures somatic symptoms. Scale 2 is the depression scale, and both treatment groups showed a severe level of depression before treatment. Scale 3 is associated with over-emotionality and repression. Scoring high on scale 4 indicates tendencies to be nonconforming, resentful of rules and authority, manipulative, and self-centered. Scale 5 measures traditionally masculine versus more feminine or more passive interest patterns. Elevations on scale 6 suggest that a patient is more paranoid, suspicious, hostile, and prone to project blame and responsibility. Scale 7 is associated with obsessive-compulsive symptoms, anxiety, and feelings of inferiority or inadequacy. Higher scores on scale 8 tend to be associated with being withdrawn, having odd or peculiar (thought disorder) thinking patterns, and feeling alienated from self and others. Scale 9, when it is elevated, can be associated with impulsiveness, high energy level, or manic tendencies. Scale 0 is an introversion/extroversion scale, with elevations associated with being introverted and having a deficit in social skills.

In another Veterans Administration hospital uncontrolled study [\[74\]](#), 20 Vietnam veterans with chronic PTSD, all with comorbid alcohol abuse, were randomly selected. All patients showed frequent (eg, two to three times per week) episodes of PTSD symptomatology and had been hospitalized for PTSD an average of five times. They were treated with 30 30-minute sessions of alpha neurofeedback training. Follow-up interviews occurred with the patients and their wives or family members on a monthly basis for 26 months. In that time, only 4 of the 20 patients reported a few (one to three) instances of recurrence of nightmares or flashbacks, and the other 16 patients had no recurrence of PTSD symptoms. The status of alcohol symptoms was not reported. According to the biofeedback efficacy criteria [\[62\]](#), neurofeedback treatment of PTSD qualifies for the status of probably efficacious.

Two published studies of OCD were reviewed by Moore [\[58\]](#). Both studies used alpha enhancement training, without positive results. Viewed from a modern perspective, these studies, which were published in the mid-1970s, used a naïve and simplistic treatment approach of only doing alpha enhancement training. Literature since that time [\[17–21\]](#) has shown that there are at least two subtypes of EEG patterns that are found in OCD, neither of which would be anticipated to benefit from alpha enhancement training.

Recent reports are available on the successful treatment, with lengthy followups, of three consecutive cases of OCD. In each of these cases, neurofeedback protocols were individualized to the unique neurophysiologic characteristics of each patient through using a qEEG assessment. In the first report [\[75\]](#), scores on the Y-BOCS and the Padua Inventory normalized after treatment, with the two patients improving on the Y-BOCS from scores of 26 and 25 to scores of 4 and 7 (showing 3.7 and 3 standard deviation improvements, respectively). This should be considered particularly significant because a meta-analysis of 25 drug studies found that even the most effective pharmacologic treatment for OCD only produced an average treatment effect on the Y-BOCS of a 1.33 standard

deviations improvement (uncorrected for placebo effects) and approximately one half that much improvement across studies with fluoxetine (Prozac) [50]. Improvements also were documented with an MMPI, and follow-ups of the two cases at 15 and 13 months after treatment (which included interviews with relatives) found that changes were maintained.

A third case of neurofeedback treatment of OCD with a college student also has been reported [76]. The individual suffered with obsessional OCD, which is the type of OCD that has proven most resistant to cognitive-behavioral treatment [58]. He proved resistant to improvement with trials of eight previous medications. On his pretreatment MMPI he scored 115 T-scores on the Pt (7) scale. After treatment, his Pt scale decreased to 60 T-scores. Before treatment he scored 16 on the Y-BOCS, which is the cut-off score generally used for inclusion in OCD medication trials. On the obsessions subscale he scored 10; the mean for patients with OCD is 10.7. At the completion of neurofeedback treatment, his Y-BOCS score had improved to 3 (a 2.2 standard deviation improvement) and his obsessions subscale score decreased to 0. Changes were maintained at 10 months, with external validation of improvements with his family.

All three of these cases had been treated unsuccessfully with various medications. In addition to these published cases, there are many clinical reports of comorbid OCD and ADHD improving with neurofeedback. Although these are uncontrolled case reports and do not yet even meet criteria for the status of a possibly efficacious treatment, the outcomes from treatment with neurofeedback in these preliminary reports are encouraging. The father of one of these patients, after having completed 21.5 hours of neurofeedback, said, "This week my daughter told me, Dad, for the first time in my life, I feel normal." The patient has been followed for more than 2 years and she has maintained her improvements.