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The Neurotherapy of Anxiety Disorders

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Only five studies of patients met criteria for this review. Four of the five had a good clinical outcome. The only one without efficacy reported no enhancement of the target waveform. Of the other four, two reported some enhancement, whereas two gave no details about changes in the target waveform. There were 10 studies of nonpatients with generalized anxiety, of which eight had a good outcome. The decrease of anxiety in these eight studies was correlated with enhancement of the target waveform in five. Three of the eight had no increase of alpha, showing placebo plays a part in neurotherapy. There were nine studies of nonpatients with phobic anxiety, of which six had enhancement of the target waveform accompanied by good outcome. The remaining three studies had poor outcome in spite of enhancement of the target waveform.

KEY WORDS: neurotherapy; anxiety; alpha-enhancement; theta-enhancement; alpha–theta-enhancement.

INTRODUCTION

All studies reported in this paper, were published in peer-reviewed journals, patients had a clinical diagnosis of one of the five anxiety disorders (American Psychiatric Association, 1994), and volunteers had objective evidence of anxiety. Four years ago, in a similar review (Moore, 2000), four studies of Generalized Anxiety Disorder, two of Phobic Disorder, two of Obsessive-Compulsive Disorder, and one of Posttraumatic Stress Disorder met these criteria. No Panic Disorder papers did so. For this review, a further six papers on Generalized Anxiety and four on Phobic Anxiety were found that met criteria for inclusion. Most of the literature is about nonpatients undergoing a single session of neurotherapy. Studies of real patients who had several sessions of neurotherapy, as in clinical practice, are more relevant and will be discussed first. Throughout the review, unless otherwise specified, the neurotherapy was with eyes closed and recording of electroencephalography (EEG) was from the occipital area.

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STUDIES OF PATIENTS

Generalized Anxiety Disorder

Watson, Woolley-Hart, and Timmons (1979) carried out alpha-enhancement in 32 anxious patients using eyes-open visual feedback. There were six sessions at weekly intervals, plus home practice. Because this was a pilot study, there were no controls. The authors preferred eyes-open training, so that patients could maintain alpha enhancement in the state in which they experienced most of their anxiety. Because anxious patients do not habituate, but continue to react to stimuli, the visual feedback was not given until the alpha had been sustained. More than half improved clinically. The authors believed the outcome was more than a placebo effect, because the increased alpha and decreased anxiety were correlated, alpha increased before anxiety decreased, patients were not aware of the level of alpha success or failure, and all had a long history of anxiety without improvement. Hare, Timmons, Roberts, and Burman (1982) provided further information about

this study. The weekly sessions included alpha enhancement, suppression, and equilibration (achieving the same alpha as last session). Threshold amplitude for feedback was the level at which pretraining alpha was present 30% of the time. There were 14 learners (defined as having alpha 50% of the time during enhancement trials) and 18 nonlearners. By session 3, learners had significantly increased alpha, and their clinical response (self assessment by linear analogue scale) had significantly improved by session 4. Nonlearners remained unchanged mentally.

Watson and Herder (1980) gave alpha-enhancement contingent feedback to 22 patients, and noncontingent to the same number. Another 22 control patients got no treatment. Although all had anxiety or depression, almost half were schizophrenic. There were ten 1-hr sessions, and baseline alpha was the threshold. None of the three groups increased alpha. The results were re-analyzed after removal of the schizophrenic patients, leaving nine participants per group. Anxiety and tension, as measured by the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), improved in both biofeedback groups, whereas the placebo group worsened. It would be of interest to know whether these changes were associated with changes in alpha, but this information was not provided for the non-schizophrenic patients. The authors thought the greater efficacy they found for alpha enhancement in a previous study (Passini, Watson, Dehnel, Herder, & Watkins, 1977) was probably due to different diagnoses, a finger-temperature control exercise, and more emphasis on relaxation in the instructions. Their final conclusion was that alpha feedback success was in part due in part to coincident training.

Obsessive Compulsive Disorder

Mills and Solyom (1974) gave from 7 to 20 1-hr sessions of α-enhancement training to five ruminating obsessive patients. Medicines were discontinued for 2 weeks before training began. Alpha abundance was calculated as % of total time that alpha was at least 20 μV. The first five sessions had minimal instructions. Subsequent sessions included information and verbal encouragement, which did not improve alpha production. One participant increased alpha after the first session, with continued increase until he dropped out after session 7 when the total increase was 22%. Another increased alpha after five sessions, but dropped out after session 9 when the total increase was 51%. The other three each had 20 sessions without any significant increase in alpha. The authors suggested that 5-hr training were sufficient to distinguish between learners and nonlearners. During feedback, one patient had less rumination and four had none. Reduction of ruminations did not generalize to outside the laboratory. Because three improved without any increase in alpha, the benefits were probably partly due to factors other than alpha-enhancement.

Glueck and Stroebel (1975) studied 225 inpatients with a variety of psychiatric illnesses. Of 26 patients assigned to alpha-enhancement training, four had obsessive-compulsive disorder (OCD). EEGs were recorded from right and left frontal, parietal, temporal, and occipital leads. Patients had a total of twenty 1-hr alpha-enhancement training sessions, but were able to control their alpha after 15 sessions. The 4 OCD patients had great difficulty enhancing alpha, and only one of them improved clinically. A total of 187 patients were assigned to transcendental meditation (TM). As in practitioners of yoga and Zen, TM patients had an increase in alpha in association with a state of restful alertness. The alpha appeared in the dominant hemisphere, spread to the other side, and then theta (4–7 Hz) and high frequency beta (20–35 Hz) appeared. Twelve patients had autogenic training, which consisted of voluntary muscle relaxation, starting with the toes and moving upwards to involve the whole body. All in autogenic training dropped out by the fourth week because of boredom. Clinical response to TM was significantly better than either alpha-enhancement or autogenic training.

Posttraumatic Stress Disorder

Peniston and Kulkosky (1991) gave alpha-theta (8–13 and 4–8 Hz)—enhancement training to 15 Vietnam veterans with a 12 to 15 year history of chronic combat-related posttraumatic stress disorder. A matched control group of 14 patients underwent traditional medical treatment. Alpha–theta-enhancement sessions were held 5 days a week. Alpha threshold was based on calibration of the feedback monitor, and the theta threshold was set 10 mV lower. Patients received eight 30-min temperature feedback sessions to achieve a temperature of 95°F

for 1 session, followed by thirty 30-min sessions of alpha–theta-enhancement training. Information about levels of alpha and theta before and after treatment was not provided. The feedback patients improved on all 10 clinical MMPI (Dahlstrom & Welsh, 1960) scales: Hypochondriasis; Depression; Conversion Hysteria; Psychopathic Deviate; Masculinity–Femininity; Paranoia; Psychasthenia; Schizophrenia; Hypomania; Social Introversion. Traditional treatment improved only the Schizophrenia scale. All 14 medicated feedback patients required less medicine, compared with only one of 13 medicated traditional treatment patients. At 30 months, all 14 traditional treatment patients had relapsed, compared with only three feedback patients.

Discussion

From Table I it can be seen that in four of five studies of anxious patients, neurotherapy resulted in a good clinical outcome. The one study with a poor outcome was the only one to report no enhancement of the target waveform, although two good-outcome studies did not provide data about changes in the target waveform. Most practitioners use eyes-closed procedures, but Watson et al. (1979) showed that eyes-open neurotherapy is also successful. It may even be preferable, if benefits are to continue outside the clinic. Future studies could clarify whether eyes-open or closed neurotherapy is best in the long term. All the successful treatments used 5 hr or more of neurotherapy, as recommended by Mills and Solyom (1974). One of these (Watson et al., 1979) did not give details of time, but the likelihood is that their six sessions lasted more than 5 hr.

Table I. Studies of Patients

Author	*N*	$\alpha\Uparrow$	$\alpha\theta\Uparrow$	EO	5 hr	Good outcome
Generalized anxiety						
Watson (1979)	32	+		+	6 sessions	+
Watson (1980)	9	NA		−	+	+
Obsessive compulsive						
Mills (1974)	5	2+, 3−		−	+	+
Glueck (1975)	4	−		−	+	−
Posttraumatic stress						
Peniston (1991)	15		NA	−	+	+

Note. N: number of participants; $\alpha\Uparrow$: alpha increase; $\alpha\theta\Uparrow$: alpha and theta increase; EO: eyes open; NA: not available; +: present; −: absent.

STUDIES OF NONPATIENTS

Generalized Anxiety Disorder

Sittenfeld, Budzynski, and Stoyva (1976) used theta (3.5–7 Hz) enhancement to lower arousal, the rationale being that theta is associated with the onset of sleep. Twenty volunteers were assessed for frontal EMG levels, and 10 high-EMG participants were compared with 10 low-EMG. Five from each group received eight sessions of theta-enhancement. The other five from each group received four sessions of EMG feedback, followed by four sessions of theta-enhancement. This second group opened its eyes occasionally for EMG visual feedback, but the feedback was primarily auditory theta. To ensure that theta feedback was given only when participants were experiencing theta-like experiences such as drowsiness, theta feedback was inhibited if alpha was present. High-EMG participants increased theta only if they first received EMG biofeedback, whereas low-EMG participants did better with theta feedback only. During the EMG only feedback, theta increased. Theta-enhancement EEG changes were specific, with no change in alpha, proving that they were not simply reflecting a general relaxation. Theta-enhancement training is very difficult in the awake participant, and is possible only in the presence of low EMG. As theta increased, EMG and heart rate decreased significantly.

Pressner and Savitsky (1977) studied four groups of 20 students matched for baseline alpha (7–12.5 Hz). They measured anxiety with the Multiple Affect Adjective Check List (Zuckerman & Lubin, 1965). The students underwent twenty 100-s trials of alpha-enhancement. Auditory feedback was contingent in half of the students, and noncontingent in the other half. Half were told the experience would be positive, and half that it would be negative. Amplitude threshold for measuring alpha, and eyes-open or closed status were not provided. Contingent feedback produced significantly more alpha than noncontingent, but alpha increase did not reduce anxiety. Those expecting a positive experience reported more reduction in anxiety. Unlike clinical practice, the training period was brief, which may explain the poor outcome.

Valle and DeGood (1977) trained 20 students in alpha (8.5–13.5 Hz) enhancement, and an equal number in alpha suppression. Audio feedback occured when alpha exceeded 10 μV. There were four

40-min weekly sessions of eyes-closed alpha feedback. Participants were able to suppress alpha, but not to enhance it. The State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970) was used to divide the participants into 20 low and 20 high in self-reported anxiety. Those with low initial anxiety were more able to suppress alpha, but there was no difference in their ability to enhance it. All participants experienced a decrease in anxiety, regardless of success or direction of alpha. The final state of anxiety was not related to alpha control. The investigators recommended that future studies should incorporate: within-participant enhancement and suppression of alpha; eyes-open training; and measurement of habituation, because it is slower in anxious patients.

Lehrer (1978) gave alpha feedback training to 11 healthy volunteers, relaxation to another 11, and no treatment to 12. Ten anxious patients received relaxation, and 10 received no treatment. Anxiety was assessed with the STAI. Those in active treatment had four or five 40-min sessions over a 3-week period. Feedback was a flashing light through closed eyes for alpha over 18 μV. In patients, relaxation increased alpha significantly. In volunteers, alpha feedback compared with relaxation produced greater increases in alpha, but smaller changes in heart rate and anxiety. Patients' alpha blocking did not habituate after loud noises, but nonpatients did.

Hardt and Kamiya (1978) assessed 100 male college students with the MMPI Welsh A anxiety scale, and compared the eight who scored highest on trait anxiety with the eight who scored lowest. Participants underwent seven alpha feedback sessions in 7 days. Each session included 32 min of alpha-enhancement and 16 min of alpha-suppression. The alpha threshold was 10 μV. Alpha-enhancement reduced both state and trait anxiety in high-trait anxiety participants, but had no effect in low-trait anxiety participants. Alpha-suppression increased state, but not trait, anxiety in high-trait anxiety participants. As training progressed for these participants, alpha-enhancement and state anxiety reduction became more strongly associated. These findings suggest that the reduction of anxiety was due to alpha-enhancement, and not to other variables such as feelings of success, which was also present in alpha-suppression where no clinical improvement occurred. Alpha-enhancement at central sites resulted in reduction of anxiety, whereas alpha-suppression at occipital sites resulted in increased anxiety, suggesting that the location of the alpha-measurement is important. Anxiety reduction was most marked after 2 hr of alpha-enhancement, and the authors suggest that at least 5 hr are needed. Negative findings in other studies may be due to the baseline anxiety level being too low or the training being too brief.

Plotkin and Rice (1981) studied 10 undergraduate volunteers who scored high on the Welsh A anxiety scale, the STAI and the Taylor Manifest Anxiety Scale (Dahlstrom & Welsh, 1960). Five participants had five alpha-enhancement sessions, five had an equal number of alpha-suppression sessions, whereas three on the waiting list served as controls. On the first 2 days they had 32 min of alpha-enhancement (or alpha-suppression) and 8 min of alpha-suppression (or alpha-enhancement). On the other 3 days they had 32 min of alpha-enhancement (or alpha-suppression). After 2 min the true feedback score was given to the participants, and then after every 2-min trial their score was increased by 1% of that day's baseline score. Alpha-enhancement feedback did not increase alpha, whereas alpha-suppression reduced it. Both feedback groups improved in state and trait anxiety, whereas the control group did not change. The authors concluded that perceived success caused the reduction in anxiety.

Rice, Blanchard, and Purcell (1993) studied 38 volunteers who met *DSM-III* (American Psychiatric Association, 1980) criteria for GAD. They had suffered from three of four symptom categories for at least 1 month. They also studied seven who were subclinical, having suffered from two of the four categories. The 45 participants had been anxious for an average of 3.8 years. Five groups of nine randomly assigned participants underwent frontal EMG feedback, alpha-increase feedback, alpha-decrease feedback, pseudo-meditation or waiting list control. Each of 8 feedback sessions lasted 20 min. Both alpha groups were given verbal feedback of success. Their scores were incremented by 2% every 2 min, leading them to believe they were being successful. The enhancement group did not change alpha, whereas the suppression group decreased it significantly. All four active treatments reduced anxiety, and improvements were maintained for 6 weeks after treatment. Alpha-increase reduced heart rate reactivity to stress, whereas alpha-suppression increased it.

Discussion

From Table II it can be seen that the target waveform was enhanced in 7 of 10 studies of nonpatients with symptoms of anxiety. The type of

Table II. Studies of Nonpatients Generalized Anxiety Disorder

Author	*N*	$\alpha\Uparrow$	$\theta\Uparrow$	EO	5 hr	Good outcome
Sittenfeld (1976)						
Low-EMG	10		+	−	+	+
Hi-EMG	10		+	−	+	+
Pressner (1977)						
Positive experience	5	+		NA	−	+
Negative experience	5	+		NA	−	−
Valle and DeGood (1977)	20	−		−	−	+
Lehrer (1978)	11	+		−	−	+
Hardt (1978)						
Hi-Anxiety	8	+		−	+	+
Lo-Anxiety	8	+		−	+	−
Plotkin (1981)	5	−		−	−	+
Rice (1993)	9	−		−	−	+

Note. N: number of participants; $\alpha\Uparrow$: alpha increase; $\theta\Uparrow$: theta increase; EO: eyes open; NA: not available; +: present; −: absent.

biofeedback training was critical (Sittenfeld et al. 1976). High-EMG participants only increased alpha if first trained in EMG feedback, whereas low-EMG participants only increased alpha if EMG feedback was omitted. Five of the seven with waveform enhancement had a good clinical outcome, suggesting that alpha or theta-enhancement was important. Alpha-enhancement was superior to suppression as shown by stress-induced heart rate decreasing in the former and increasing in the latter (Rice et al., 1993), and clinical symptoms worsening after alpha-suppression (Hardt & Kamiya, 1978). These findings suggest that the biofeedback-induced enhancement of alpha and theta provide additional benefits to the presumed placebo effects of suppression. Three studies (Valle & DeGood, 1977; Plotkin & Rice, 1981; Rice et al., 1993) showed that clinical improvement could be obtained without increased alpha, proving that there is a placebo effect in neurotherapy. This is not surprising, because all successful therapy is known to have a placebo effect. For example, in three published studies of six-month long pharmacotherapy of Generalized Anxiety Disorder, the response rates for placebo were 42% (Gelenberg et al., 2000), 46% (Allgulander et al., 2001) and 48% (Lenox-Smith et al., 2003). The respective response rates for venlafaxine were 69%, 76% and 52%. One group of participants (Pressner & Savitsky, 1977), which had alpha increase without clinical benefit, had been told to expect a negative experience, showing that the placebo effect is absent when the ambience is negative. Perceived success in carrying out the task plays an important role in clinical improvement (Plotkin & Rice, 1981; Rice et al., 1993). Another group (Hardt & Kamiya, 1978), which increased alpha without clinical benefit, was chosen for low levels of anxiety. Perhaps there was too little room for improvement in these participants. In nonpatients it does not seem to matter that treatments were mostly less than 5 hr. Using too few electrodes may result in vital information being missed. A correlation between alpha-enhancement and reduced anxiety was seen only centrally, whereas a correlation between alpha-suppression and increased anxiety was seen only occipitally (Hardt & Kamiya, 1978).

Phobic Anxiety Disorder

Garrett and Silver (1976) carried out two studies of students suffering from test anxiety. In the first study they tested 163 students with the Debilitating Anxiety Scale (Alpert & Haber, 1960) and six other questions about illness and muscle tension resulting from doing tests. Of those who scored in the upper two thirds, 36 entered the study: 18 to alpha enhancement training and 18 to an untreated control group. The threshold for alpha feedback was 21 μV, except for two participants whose voltage setting was reduced to 19 μV and one other whose setting was increased to 23 μV. Half of the feedback group started with alpha-enhancement training and half started with EMG feedback. Each participant received three 40-min sessions of each type of feedback training. The feedback increased alpha from 64 to 78%, and reduced EMG voltage from 5.84 to 3.49 μV. The majority, 83% were more relaxed in and out of the laboratory. Nine thought that alpha was best for relaxation, 4 preferred EMG, and 5 thought they were equally effective. The feedback groups' test anxiety scores improved (from 50 to 32), while the untrained group's were essentially unchanged (48–47), a statistically significant difference ($p < .001$).

Garrett and Silver (1976) used the same methodology in their second study. Groups of 10 students, who scored above the median on the same anxiety questionnaire, were randomly assigned to one of five treatments, each involving 10 sessions. The first, alpha-enhancement increased alpha by 33%. The second, EMG reduction decreased muscle tension by 50%. The third, combined alpha-enhancement and EMG reduction feedback alternated between the two types of feedback, half starting with each type. This group. increased alpha by 45%, and reduced muscle tension by 41%. One participant dropped out of the combined group and could not be replaced. The fourth treatment, relaxation increased alpha by

18%, and reduced muscle tension by 41%. The fifth group had no training. All three feedback groups had a significant reduction in test anxiety, whereas the relaxation and the untreated groups had none, suggesting that the improvement in the feedback groups was not merely a placebo effect.

Chisholm, DeGood, and Hartz (1977) divided 18 male and 18 female students into three treatment groups, matched for gender. The students then received eight electric shocks, resulting in reduced alpha, increased tension and increased heart rate. One group had a 24-min eyes-open session of contingent alpha enhancement. Participants were instructed to keep the signal on as much as possible, with no mention of alpha or expectation. The biofeedback tone was activated for alpha of 20 μV or more. One control group received 24 min of noncontingent alpha enhancement, and the other listened to music for the same length of time. Contingent feedback and no treatment increased alpha, whereas the noncontingent feedback decreased alpha. All subjects then underwent eight more shocks with feedback tones or music on, followed by four shocks with sound off. Only the contingent feedback group maintained increased eyes-open alpha during the posttreatment aversion trial, and this ceased when the tone was switched off. In eyes-closed aversion, contingent feedback resulted in increased alpha whether the tone was on or off, whereas neither control group maintained increased alpha in any aversive condition. Disappointingly, pulse, anxiety and tension were not correlated with alpha. Reductions in anxiety and tension were significant in all three groups, but not different between them. There was a reduction of heart rate in all groups, but reached significance in the music group only.

Pettigrew and Dawson (1979) selected 40 female undergraduates free of severe psychopathology or recent severe trauma, such as the death of a loved one, for a study of death anxiety. Groups of 10 received one session of hypnosis, alpha contingent feedback, prestige suggestion, or silence to attenuate death anxiety. They eliminated those who did not increase alpha over the threshold at or more than 10 μV. After treatment they were shown a tape/slide presentation about personal death. There was no increase or decrease of death anxiety after any of the four treatments.

Holmes, Burish, and Frost (1980) gave 11 students eyes-open alpha enhancement with instructions and 11 with no instructions. Eleven got instructions only, and 11 got neither instructions nor alpha feedback. The session lasted 20 min, and for the feedback group a tone indicated eyes-open alpha at 10 μV or more. These 44 participants were threatened with electric shock, whereas 11 new volunteers were not threatened. Even though the shock was not delivered, the threat caused an increase in stress and arousal. Alpha increased with instructions alone, indicating that the increase with feedback was due to the instructions rather than the EEG alpha enhancement procedure. Under stress previous feedback did not increase alpha or reduce arousal.

Tyson (1996) assigned 15 female students to three groups for pretraining. The first group was assigned to alpha enhancement training with feedback by recorded baby crying. The second group's feedback was by a harmonious sound. The third group listend to crying without any feedback training. All 15 then received the same alpha enhancement relaxation with crying. The feedback for alpha at or over 10 μV was increased volume of crying or harmonious sound. Parietal, rather than the usual occipital, EEG recording was chosen because the degree of sensorymotor and intermodality interaction at this site is greater. Each participant had 30 min of pretraining alpha-enhancement, followed by 15 min desensitization training with apha-enhancement. Pretraining alpha increased in both feedback groups, but not in the control group. Alpha remained low in control group participants, showing that they did not habituate to the crying. Both feedback groups had reduced cortical arousal, perceived arousal and anxiety while listening to crying later. In those listening to crying without any training, alpha remained reduced showing that it had not habituated.

Discussion

Table III shows the results of neurotherapy studies of nonpatients who had phobic anxiety. Alpha increase was achieved in all studies. Two of the three studies with poor outcome had eyes-open neurotherapy, whereas the fourth did not supply information about eye condition. Future studies should include comparisons between open and closed eye conditions. This will clarify which is preferable in different subtypes of anxiety, and which maintains improvement in the long term. Interestingly, in three of six studies with good clinical outcome, neurotherapy lasted more than 4 hr. The minimum duration of EEG-biofeedback training required for efficacy is unclear. Five hours have been suggested (Hardt & Kamiya, 1978), yet shorter periods have been

Table III. Studies of Phobic Anxiety Disorder Nonpatients

Author	*N*	$\alpha\Uparrow$	EO	5 hr	Good outcome
Garrett (1976)	18	+	Some at end	4 hr	+
Garrett (1976)					
Alpha	10	+	−	+	+
Alpha + EMG	10	+	−	+	+
Chisholm (1977)	12	+	+	−	+
Pettigrew (1979)	10	+	NA	−	−
Holmes et al. (1980)					
Alpha with instructions	11	+	+	−	−
Alpha without instructions	11	+	+	−	−
Tyson (1996)					
Crying	5	+	−	−	+
Harmonious sound	5	+	−	−	+

Note. N: number of participants; $\alpha\Uparrow$: alpha increase; EO: eyes open; NA: not available; +: present; −: absent.

effective (Pressner & Savitsky, 1977; Valle & DeGood, 1977; Lehrer, 1978; Chisholm et al., 1977; Tyson, 1996; Plotkin & Rice, 1981; Rice et al., 1993; Garrett & Silver, 1976). Five hours have also been suggested as the minimum required to distinguish between learners and nonlearners (Glueck & Stroebel, 1975).

RECOMMENDATIONS

Agreement should be reached on the threshold for feedback to occur. Knox (1980) measured eyes-closed alpha at 15 μV or more in 94 psychology students, who were medicine-free and within normal for anxiety, impulsivity, and monotony avoidance. At this threshold the majority, 64 participants, had 24 or less % alpha. Only 20 students had 25–74%, whereas 10 had 75% or more. The authors recommended that all amplitudes of baseline alpha should be used as the threshold for neurotherapy, otherwise the majority of participants would receive little feedback. The guidelines, laid out by Rice and Blanchard (1982), remain the minimum standard for future studies. They recommend that future research should answer the following questions affirmatively: (1) Was there evidence that the EEG-biofeedback training led to reliable change in the target waveforms? (2) Did the EEG-biofeedback training lead to more of the desired change than control conditions? (3) Was there significant anxiety reduction associated with EEG-biofeedback training? (4) Did the EEG-biofeedback training lead to more anxiety reduction than control conditions? (5) Is there evidence that EEG-biofeedback-mediated physiological change per se accounts for the observed anxiety reduction? From this review, further guidelines can be added. Based on the findings of Hardt and Kamiya (1978), who found that alpha changes in the occipital and central areas had different effects on anxiety, multiple electrode sites should be used. Participants should be clinical patients, not volunteers, and they should have a minimum of 5-hr training. Variables needing study are duration of treatment, type and severity of anxiety, number and type of EEG waveforms used, pretreatment with other kinds of feedback, position and number of electrodes, and presence of concomitant medication.

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