

Alpha-Theta Brainwave Neuro-Feedback for Vietnam Veterans with Combat- Related Post-Traumatic Stress Disorder

Eugene G. Peniston

V.A. Medical Center, Fort Lyon, Colorado

Paul J. Kulkosky

University of Southern Colorado, Pueblo, Colorado

The Minnesota Multiphasic Personality Inventory (MMPI) was used to assess personality changes in Vietnam combat veterans with post-traumatic stress disorder (PTSD), after either traditional medical treatment (TC) or alpha-theta brainwave neuro-feedback therapy (BWT). Application of brainwave training for thirty 30-minute sessions resulted in decreases in MMPI T-scores on clinical scales labelled hypochondriasis, depression, hysteria, psychopathic deviate, masculinity-femininity, paranoia, psychasthenia, schizophrenia, hypomania, and social introversion-extroversion. The traditional medical control group showed decreases in T-scores only on the scale labelled schizophrenia. All fourteen BWT patients initially receiving psychotropic medication reduced their dosages after treatment, but only one of thirteen TC patients reduced dosage. A thirty-month follow-up study showed that all fourteen TC patients had relapsed, in contrast to only three of fifteen BWT patients. These findings indicate that application of alpha-theta brainwave training is a more efficacious treatment modality in the treatment of PTSD and prevention of relapse.

Special thanks to Fort Lyon VA Medical Center for support of the study and to Sharon Pruett of University of Southern Colorado for assistance with manuscript preparation. Reprint requests may be sent to Eugene G. Peniston, Clinical Psychologist, Psychology Service (116B), VA Medical Center, Fort Lyon, CO 81038.

INTRODUCTION

The National Vietnam Veterans Readjustment Study was mandated by Congress in November 1983 under U.S. Public Law 98-160. A four-year study was conducted under a VA contract by the Research Triangle Institute (RTI) to examine the post-war readjustment problems among male and female Vietnam Era veterans of all branches of the U.S. military services on a nationwide basis. The RTI study disclosed: (1) an estimated 479,000 (15.2%) of some 3.1 million male Vietnam theater veterans currently have Post-Traumatic Stress Disorder (PTSD); (2) an estimated 610 (8.5%) of 7,000 female Vietnam theater veterans currently have PTSD; (3) 30 percent of some 3.1 million Vietnam theater veterans have had PTSD at some point in their lives; (4) likewise, almost half of Vietnam theater veterans have had at least one psychiatric disorder; alcohol abuse or dependence accounted for the greatest proportion; (5) one in 10 Vietnam theater veterans has been homeless or vagrant at some point during their lives; and (6) those veterans exposed to high levels of war stress had higher rates than other Vietnam veterans for divorce, marital problems and problems in parenting their children (Kulka, Schlenger, Fairbank, Hough, Kulkaph, Jordan, Marmar, & Lueiss, 1988).

Although PTSD is relatively new as a specific psychiatric diagnosis, reports in professional literature have described predictable intrusive thoughts, sleep disturbance, and avoidance behavior following many years of trauma exposure (Kardiner, 1941; Grinker & Spiegel, 1945; Trimble, 1981; Kolb, Burris, & Griffiths, 1984; Fay, Sipprelle, Rueger, & Carroll, 1984; Carroll, Rueger, Fay & Donahoe, 1985). In chronic combat-related PTSD, symptoms of anxiety, disgust, alcohol abuse, suicidal thoughts, hostility, marital distress, depression and irritability are likely, along with the core PTSD diagnostic symptoms described above (DSM-III, 1987). The implication of these clinical studies is that combat can have consistent and devastating after-effects. Figley (1978) concluded that veterans who had been exposed to the most extreme stress in combat showed a greater incidence of psychological difficulty. Maladaptive behavior problems (i.e., chief complaints) reported by these veterans were chronic states of anxiety, recurring nightmares, flashbacks, depression, panic attacks, and vivid reexperiencing of an extremely traumatic combat experience (Williams, 1980). These symptoms have been subsumed under the diagnosis of PTSD, which assumes the occurrence of a specific stressful event that results in long-continuing stress symptoms (DSM-III, 1987). As yet, only one controlled group study of biofeedback-induced desensitization for combat-related PTSD treatment has been reported (Peniston, 1986). However, research on clinical outcomes has recently been reported in case studies by Schindler (1980) and Keane and Kaloupek (1982), who each employed desensitization and/or imaginal flooding techniques, and hypnotic recall of traumatic events (Leahy & Martin, 1967; Balson & Dempster, 1983) in the treatment of post-traumatic stress in a Vietnam combat veteran. These recent findings on the effectiveness of relaxation therapies demonstrate promising techniques, but analyses were based on minimal samples and lacked comparison data from control samples. Although the clinical literature on war-related PTSD is rich in the discussions of diagnosis and assessment (Atkinson, Spar, Sheff, White & Fitzsimmons, 1984; Goodwin, 1980; Keane, Malloy, & Fairbank, 1985), common treatment themes (Engedorf, 1975), establishing therapeutic alliances (Haley, 1978; Williams, 1980), and intervention strategies (Figley, 1978; Horowitz & Solomon, 1975), systematically controlled treatment outcome data are almost entirely lacking.

Behavioral treatments such as systematic desensitization, imaginal flooding techniques and relaxation training were developed as effective means of reducing stress-related anxiety reactions and eliminating nightmares (Budzynski & Stoyva, 1969, 1973; Marshall & Segal,

alpha-theta neuro-feedback training is a biofeedback technique used to learn control of particular brainwaves in the treatment of a variety of disorders (Peniston & Kulkosky, 1989; Ayers, 1981, 1983; Lubar & Lubar, 1984; Lubar, Shabsin, Natelson, Holden, Whitsett, Pamplin and Krulidowski, 1981; Green & Green, 1977; Watson & Herder, 1978; Passini, Watson, Dehner, Herder, & Watkins, 1977). There is promising evidence that EEG alpha-theta brainwave neuro-feedback training is an effective treatment for stress disorders (Green & Green, 1977) and alcoholism (Peniston & Kulkosky, 1989). We hypothesized that Vietnam theater veterans with combat-related PTSD provided prolonged EEG alpha-theta brainwave neuro-feedback training will show significant reductions in their post-traumatic stress symptoms (fears, anxiety, stress, etc.), psychotropic medications, and nightmares and flashbacks. To test those hypotheses, an EEG alpha-theta brainwave neuro-feedback program was used as a treatment technique for Vietnam theater veterans with combat-related PTSD.

The purpose of this initial study was three-fold: (1) to test the effectiveness of EEG alpha-theta brainwave neuro-feedback therapy in the treatment of recurring combat-related nightmares/flashbacks of traumatic aversive situations; (2) to determine the effectiveness of EEG alpha-theta brainwave neuro-feedback therapy in the reduction of psychotropic medications for PTSD; and (3) to use MMPI profiles as evaluative measures of personality changes in PTSD patients after undergoing either an experimental 26-day EEG alpha-theta brainwave training program that has been described earlier (Peniston & Kulkosky, 1989; 1990), or a control treatment of traditional medical therapy.

METHOD

Subjects

The subjects in this initial study consisted of 29 Vietnam theater veterans with a twelve to fifteen year history of chronic combat-related post-traumatic stress disorder (PTSD). All subjects volunteered and gave their informed consent to participate in this study. The subjects were randomly selected from a population of Vietnam theater combat veterans evaluated for treatment of chronic combat-related PTSD at Fort Lyon VA Medical Center, and were selected based on the following criteria: (a) diagnosis of combat-related PTSD as defined by DSM-III manual; (b) no evidence of psychotic symptoms (i.e., hallucinations or delusions); (c) no known organic dysfunction; and (d) frequent recurring combat-related nightmares/flashbacks that were anxiety-evoking events. The subjects were randomly assigned to either the EEG alpha-theta brainwave neuro-feedback training (BWT) group (N=15) given a modified version of EEG alpha-theta brainwave training (Peniston & Kulkosky, 1989), or a Traditional Medical Control (TC) group (N=14) given psychotropic medications and combined individual and group therapy to treat combat-related PTSD.

The BWT group had a mean chronological age (CA) of 36.12 years (standard deviation (SD), 2.62); a mean number of prior hospitalizations (prior/H) of 5.40 (SD, 1.42); a mean Shipley Institute Scale Intelligence Quotient (SIS) IQ of 105.4 (SD, 6.45). The TC group had a mean CA of 37.25 years (SD, 2.82); a mean number of prior/H of 5.40 (SD, 1.42), a mean SIS IQ of 106.42 (SD, 9.34).

5/13

11

Minnesota Multiphasic Personality Inventory

Each participant was requested to complete the long form (R) of the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway and Meehl, 1951). The complete questionnaires were scored on all 10 clinical scales and the three major validity scales. The scores were K corrected, converted to T scores, and coded according to the procedures of Welsh (Dahlstrom, Welsh, & Dahlstrom, 1972). Fairbank, Keane, & Malloy (1983) found that MMPI profiles of Vietnam theater veterans with combat-related PTSD are characterized by prominent high elevations on the F scale and on clinical scales SC (schizophrenic) and D (depression), and the other clinical scales (except MF, MA, and SI) are elevated into the pathological range. Traditionally, elevated F scores are indicative of malingering or a "cry for help," however, when such scores are obtained by PTSD patients, it appears to be associated with genuinely high symptom severity (Fairbank, Keane, Malloy, 1983). A decision rule for identifying PTSD profiles and a PTSD special subscale were developed by Keane, Malloy, & Fairbank (1984) and have been repeatedly cross-validated by Cannon, Bell, Andrews, and Finkelstein (1986); Hyer, O'Leary, Saucer, Blount, Harrison, and Boudewyns (1986); and Keane, Malloy, and Fairbank (1984). An elevated PTSD scale raw score (e.g., of 30 and above) effectively separated the combat-related PTSD patients from the non-PTSD patients (Fairbank *et al.*, 1983; Keane, Malloy, & Fairbank, 1984; Foy, Sipprelle, Rueger, & Carroll, 1984; Merbaum, 1977). For the purpose of this study, the above-described Fairbank *et al.* (1983) decision rule for identifying PTSD profiles was used. In addition to this decision rule, an elevated PTSD special subscale also served as a diagnostic measure of PTSD syndrome of the Vietnam veteran with combat-related post-traumatic stress symptom.

Medication Consumption

After one week of daily practice of BWT, the drug dosage (tricyclic antidepressants, antipsychotics, anxiolytics) for BWT subjects (N=14) and TC subjects (N=13) was gradually reduced at their request. During BWT sessions, subjects were monitored by both the open psychiatric ward and the Outpatient Clinic throughout the withdrawal period. The physicians were aware of the treatment groups (BWT and TC) and a weekly record was maintained on each patient's medication reductions. If an attempt by the physician to withdraw the subject's initial psychotropic medication resulted in intense depression, or intense stress, or anxiety, etc., the subject was reintroduced to psychotropic medication.

Apparatus

An Autogen 2000 Feedback Thermometer (Autogenic Systems, Inc.) was used to measure the subjects' temperatures and to provide audio feedback. Audio feedback was in the form of a beep tone that rose in pitch as subjects' temperatures increased and that lowered in pitch to corresponding decreases in temperature. The thermometer data were collected in the form of degrees Fahrenheit (°F) using an Autogen 5600 Digital Integrator. The Integrator supplies a digital record of summated temperature activity, once every 30 seconds. An EEG Feedback Monitor (Model E430) and EEG Timer (ET 330) (RI Company, Topeka, KS) were used to measure the subjects' brainwave activity and to provide both audio and visual feedback. This EEG Monitor detects information in raw EEG by using three active band-pass filters. Alpha (8-13 Hz), beta (13-26 Hz), and theta (4-8 Hz) rhythms are detected by filters with 71dB per octave attenuation rates. The micro-processor-based timer accumulated time for an EEG

band whenever the signal exceeded the threshold for that band. The audio-visual feedback unit of the instrument contained an individually controlled tone generator. The microvolt levels for each of these band-pass filters were controlled independently, and different individual tones provided audio feedback for the alpha, beta, or theta frequencies. If the frequency (i.e., alpha, beta, or theta) remained above the threshold, a tone was presented. In addition to this audio feedback, a separate set of visual percentage counters was activated by the presence of alpha, or beta, or theta thresholds, and a calculation of accumulated time was available. At the conclusion of training, the EEG Timer calculated the percentage of time that each band exceeded the threshold levels.

Brainwave Training Procedures

Prior to the initiation of BWT, both groups (BWT and TC) were required to: (a) complete the MMPI Form R, and (b) read and sign an informed consent form allowing information from their files to be used for this research. The MMPI was scored in accordance with the manual directions.

All subjects were given a brief introduction to EEG brainwave biofeedback and were told how to interpret the audio feedback (i.e., beta, alpha, theta) sounds. During this initial session, and subsequently, the following procedures were implemented. Earlobes and theinion were cleaned with alcohol prior to attaching the monopolar electrode leads. Omni Prep was used as a conduction medium to fill the electrode cups and in the preparation of the electrode scalp site. An occipital (O1) electrode was attached approximately 1 cm above and 1 cm left of the inion and held in place by a stretching headband. Two ear clip electrodes were attached and the active electrode was referenced to the left earlobe (A1), with the ground on the right earlobe (A2). Before recording commenced, electrode impedance was checked and electrodes were reapplied if necessary. Beta, alpha and theta sensitivity threshold settings were adjusted on the feedback monitor for each subject. Prior to recording each individual's initial alpha and theta base-rate scores, the threshold dials of the feedback monitor were adjusted (aided by the use of a MFE Posi-Traci I-strip chart recorder) to a point at which the waves characteristic of beta, alpha, and theta registered on the feedback monitor and on the cumulative recording computer-based timer. Because theta was not produced uniformly during the calibration sessions, this procedure could not be used to set a theta threshold. Instead, theta thresholds were arbitrarily set at points 10 microvolts below those for alpha because the theta and alpha thresholds of previous patients who had produced theta during calibration tended to differ by this amount. Beta, alpha and theta rhythms were defined in terms of time that the input signals exceeded the machine-set thresholds. Each subject was seated in a comfortable reclining chair in a sound-proof room and was instructed to sit quietly and relax with eyes closed for five minutes while a base-rate recording was obtained.

Only the BWT subjects received eight 30-min sessions of pre-training in temperature biofeedback-assisted autogenic training and thirty 30-min BWT sessions. During the pretraining sessions, the medical psychotherapist (MP) attached a temperature thermistor to the tip of the middle finger and middle toe of the subject's dominant hand and foot with the micropore tape. The BWT subjects were instructed to sit in a comfortable reclining chair and relax and close their eyes. Then the MP introduced the subjects to autogenic training exercises and rhythmic breathing techniques in an effort to induce relaxation of the body and quieting of the mind. In the following next six or seven sessions, the subjects practiced temperature feedback until the hand and/or foot could be warmed to more than 95° F and held there over one session. It is believed that temperature training simulates the production of the "theta state" (Hall, 1977).

Following the temperature biofeedback pre-training session, the experimental subjects completed a total of thirty 5-min baseline intervals and thirty 30-minute BWT sessions. Subjects were seen five times a week (5 days) for a duration of twenty-eight days. During BWT, subjects were instructed to close their eyes and construct visualized scenes of their nightmares and flashbacks. The patient received the following instructions from the MP: "Now, go back to Vietnam where these traumatic combat events occurred." Then, they were instructed to visualize imageries of increased alpha rhythm amplitude and scenes of the normalization of their personalities. Then, the MP instructed the subjects to "sink-down" into theta state keeping the mind quiet and alert (but not active), and the body calm. Finally, subjects were instructed by the MP to initiate the session with a quiet command: "Do it." Prior to the MP's exit from the room, the beta feedback volume control band was turned off; alpha and theta feedback volume control bands were adjusted for a comfortable listening level for each subject; and the overhead light was turned off. The MP returned to the room 30 minutes later and pressed the "stop" button of the computer-based timer and gently returned the subjects to a state of awareness. The aforementioned procedures were employed throughout the thirty 30-minute sessions.

Data collection for this initial study was terminated at the completion of the thirty 30-minute sessions. All subjects were administered the MMPI measure and this post-MMPI was evaluated by another psychologist, who was unfamiliar with the design (treatment blind). These data (pre- and post-treatment MMPI test scores) were analyzed with split-plot analyses of variance, followed by Duncan's test, at an alpha significance level of $p < 0.05$. As described previously, a record was maintained also on each patient's weekly medications. The changes in total amount of psychotropic drug dosage in BWT and TC patients were analyzed with a X^2 test, also at $p < 0.05$.

The TC group was given only the pre- and post-MMPI, and base-rate EEG brain-wave measures. Also, the TC group were instructed not to use any biofeedback relaxation training procedure during the study. The rationale for the subjects' participation in the experiment included statements on the informed consent form that the purpose of the study is to determine if EEG alpha-theta brainwave training will eliminate posttraumatic stress symptoms (i.e., recurring nightmares and flashbacks, chronic states of anxiety, depression, vivid reexperiencing of traumatic combat events, etc.) and significantly change combat-related veterans' personality characteristics.

Follow-Up Study

All 29 (BWT and TC) Vietnam theater veterans and their informers (wives, family members) were contacted by telephone at monthly intervals for 30 months after completion of BWT. To determine the long-term effects of EEG alpha-theta brainwave training, subjects and informers were asked to report instances of posttraumatic stress symptoms such as flashbacks, nightmares, anxiety attacks, depression, etc. These data were analyzed with a X^2 test after application of Yates' correction for continuity, at $p < 0.05$.

RESULTS

Minnesota Multiphasic Personality Inventory

Mean (+ standard deviation, SD) MMPI T-scores on three validity scales (L, F, K) and ten clinical scales (HS, D, HY, PD, MF, PA, PT, SC, MA, SI) of the traditional medical control

group (TC) and the brainwave training experimental group (BWT), before (PRE) and after (POST) treatment, are shown in Figures 1 and 2, respectively. On scale L (lie) analysis revealed a significant interaction of treatment group (BWT vs. TC) and testing time (pre- vs. post-treatment), ($F(1,27)=5.58, p<.05$), as the BWT group showed a slight increase in scores on the post-test.

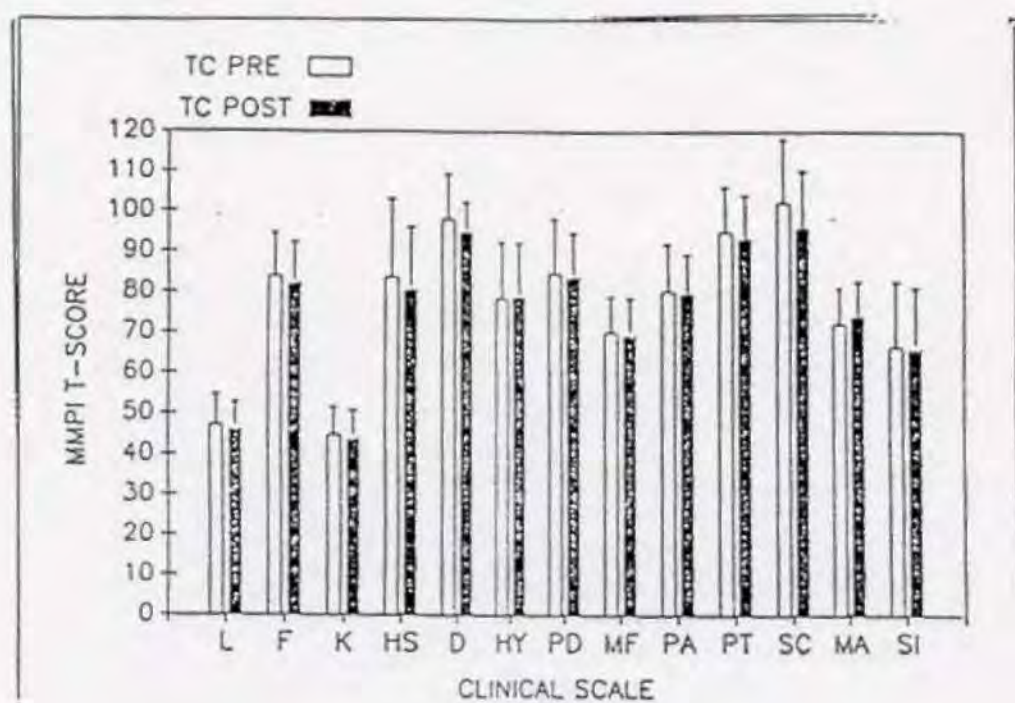
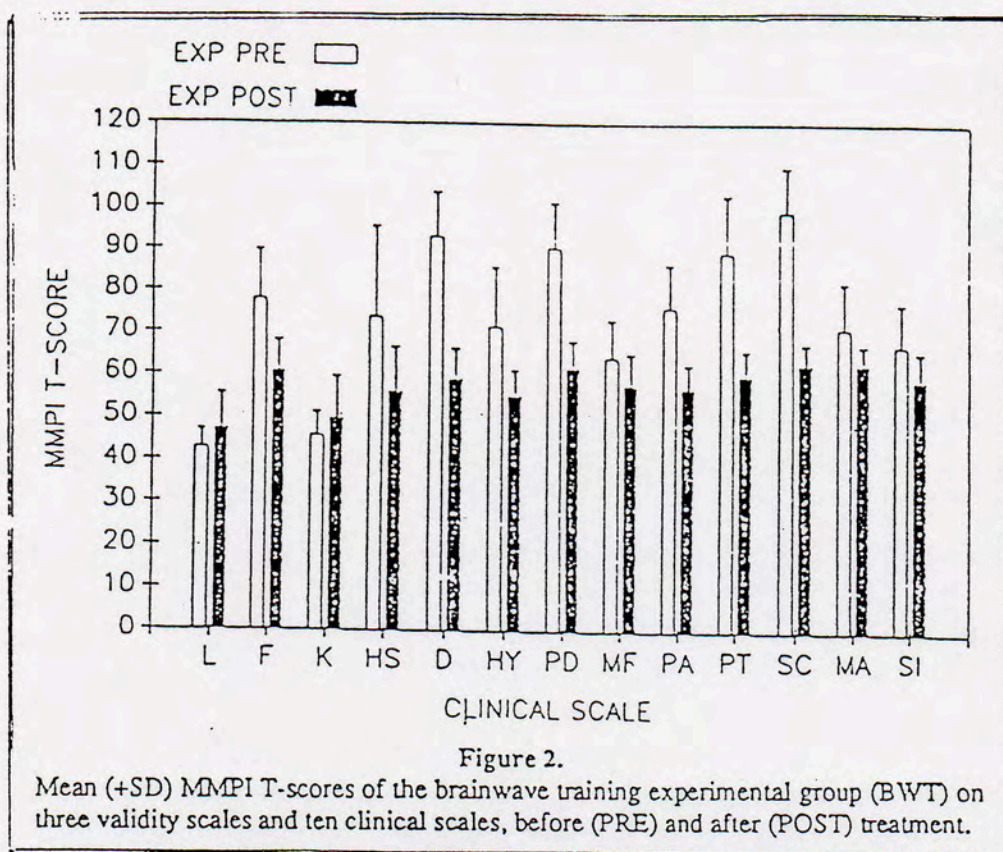


Figure 1.

Mean (+SD) MMPI T-scores of the traditional medical control group (TC) on three validity scales and ten clinical scales, before (PRE) and after (POST) treatment.

On scale F (frequency), there were significant main effects of treatment group, ($F(1,27)=16.36, p<.05$), and testing time, ($F(1,27)=32.78, p<.05$), and a significant interaction of group and time, ($F(1,27)=22.24, p<.05$). Only the BWT group showed a significant decrease in scores across testings, and the mean score of the BWT group was lower than that of TC group on the post-test. On scale K (correction), there were no statistically significant main effects or interaction.

On scale HS (hypochondriasis), main effects of group and time and their interaction were statistically significant, (respective $F(1,27)s=8.36, 16.32$, and $8.18, ps<.05$). Post-hoc Duncan's tests revealed a significant ($p<.05$) decrease in T-scores in the BWT group, which also had a significantly lower mean than the TC group on the post-test. On scale D (depression), both main effects (group and time) and the interaction were significant, (respective $F(1,27)s=40.95, 170.44$ and $119.81, ps<.05$). Post-hoc analyses indicated a significant decrease in mean score in the BWT group, and a significant difference between the groups on the post-test. On scale HY (hysterical), effects of group and time and their interaction were statistically significant,

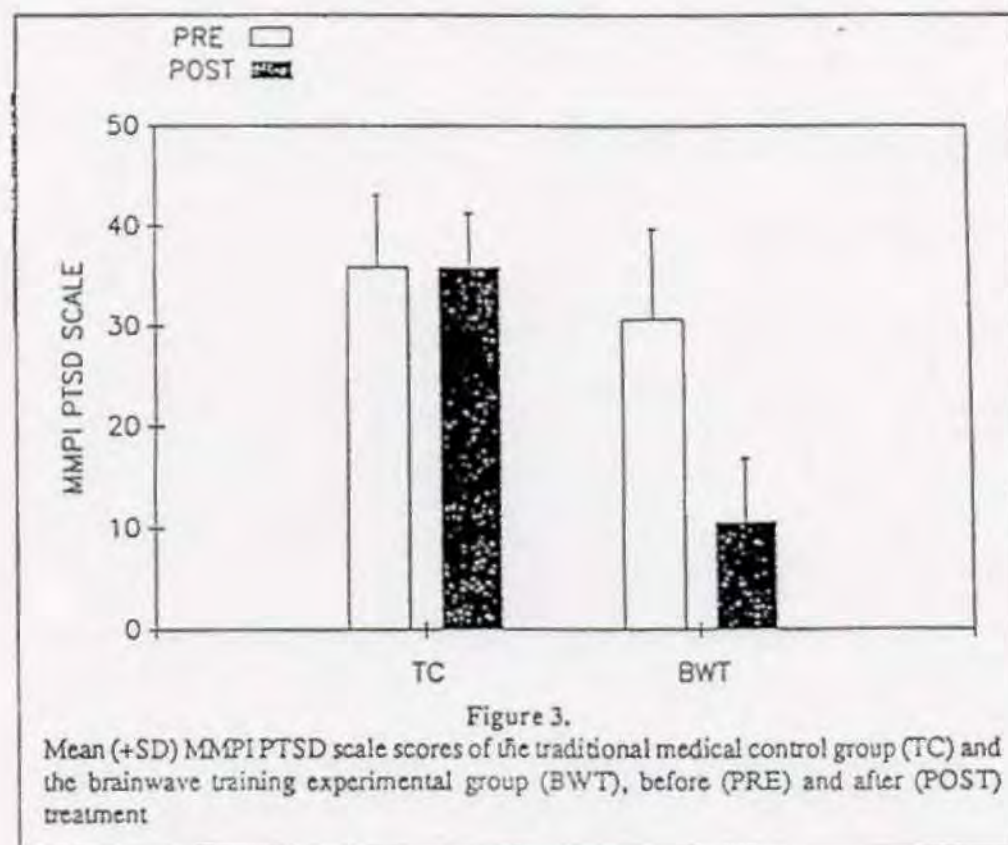


(respective $F(1,27)s=12.71, 18.42, \text{ and } 20.48, ps<.05$). The BWT group showed both a significant decrease in scores across testings, and significantly lower scores than the TC group on the post-test ($ps<.05$). On scale PD (psychopathic deviate), main effects of group and time and the interaction were significant, (respective $F(1,27)s=4.75, 81.13, \text{ and } 73.45, p<.05$). Duncan's test revealed a significant decrease in T-scores in group BWT, and a significant difference in mean scores between the groups on the post-test. On scale MF (masculinity-femininity), main effects of group, time and the interaction were statistically significant, (respective $F(1,27)s=7.66, 24.87, \text{ and } 16.55, ps<.05$). Analysis of means revealed a decrease in group BWT across testings, and a lower mean score, compared to group TC on the post-test. On scale PA (paranoia), effects of group and time and the group \times time interaction were statistically significant, (respective $F(1,27)s=15.94, 52.74, \text{ and } 48.19, ps<.05$). Scale PA T-scores decreased in group BWT and were lower than those of group TC on the post-test ($ps<.05$). On scale PT (psychasthenia), effects of group, time, and the interaction were significant, (respective $F(1,27)s=29.4, 68.17, \text{ and } 55.04, ps<.05$). T-scores in group BWT decreased significantly, and were lower than those of group TC on the post-test. Analysis of scale SC (schizophrenia) revealed that effects of group, time and their interaction were significant, (respective $F(1,27)s=18.75, 147.29, \text{ and } 74.0, ps<.05$). Post-hoc analysis of means showed that scores of both groups BWT and TC decreased significantly across testings, and group BWT had lower T-scores than group TC on the post-test ($ps<.05$). On scale MA (hypomania), only

both decreased significantly

the main effect of treatment group and the group \times testing time interaction were significant, (respective $F(1,27)s=4.73$ and 9.01 , $ps<.05$). Group BWT showed a significant decline in scale MA T-scores across testings, and the group BWT mean was lower than that of group TC on the post-test. Finally, on scale SI (social introversion-extroversion), the main effect of testing time and the group \times time interaction were statistically significant, (respective $F(1,27)s=6.18$ and 5.36 , $ps<.05$). Post-hoc tests revealed only a significant decrease in mean scores in group BWT across testings.

Figure 3 depicts mean (+SD) MMPI PTSD scale scores of groups BWT and TC before and after treatment. Split-plot analysis of variance revealed significant main effects of treatment group (BWT vs. TC), ($F(1,27)=47.81$, $p<.05$), and testing time (pre- vs. post-treatment),



($F(1,27)=49.61$, $p<.05$), and a significant interaction of those factors, ($F(1,27)=49.61$, $p<.05$). Duncan's multiple range test revealed a significant decrease in mean score in group BWT, and significantly lower scores in group BWT, than in group TC on the post-test.

Medication Consumption

Table 1 displays counts of increases, decreases, and absences of change in total psychotropic drug dosage of groups BWT and TC.

A chi-square test indicated a significant difference in changes between the two groups, $\chi^2(2)=23.26$, $p<.05$. Whereas all fourteen BWT patients initially receiving psychotropic medications decreased total dosage at the end of the experiment, only one of thirteen TC patients so reduced their psychotropic medications.

Table 1
Changes in Psychotropic Drug Abuse

		Increase	Decrease	No Change
$N=15$	Group BWT	0	14	0
$N=14$	Group TC	10	1	2

Number of patients in Group BWT and Group TC who increased, decreased, or did not change their total psychotropic drug dosage, by the end of the experiment.

Follow-Up Study

Table 2 displays counts of relapse or continued absence of PTSD symptoms of patients in groups BWT and TC, thirty months following treatment. A chi-square test showed a significant difference between the groups in relapse, $\chi^2(1)=15.8$ $p<.05$.

Table 2
Thirty-month follow-up study

	Relapse	No relapse
Group BWT	3	12
Group TC	14	0

Number of patients in Group BWT and Group TC who showed relapse of PTSD symptoms or continued absence of symptoms, thirty months following treatment.

All fourteen control group patients had relapsed by thirty months after treatment, but only three of fifteen experimental group patients had relapsed by then.

DISCUSSION

In the present study, alpha-theta brainwave neuro-feedback therapy (BWNT) produced significant MMPI-indexed personality changes in Vietnam theater veterans with chronic combat-related PTSD symptoms. A follow-up study indicated that BWNT significantly reduced anxiety-provoking traumatic recurring nightmares/flashbacks. Also, this initial study indicated that BWNT patients had significant reductions in their psychotropic medications (antidepressant and anxiolytic) for PTSD. This study provided clinical observations to support the idea that Vietnam combat veterans' recurring anxiety-provoking nightmares/flashbacks are symbolic expressions of survival guilt feelings reflective of those combat traumatic events that had been repressed and displaced by guilt ridden emotions. BWNT appeared to allow those repressed Vietnam combat-related anxiety-provoking events to

become conscious, by reliving them emotionally through hypnagogic imageries. This unexpected development had been referred to in the past decade as 'Breuer and Freud's Abreaction Concept (1950).' In accord with these clinical explanations are the following three examples of Vietnam theater veterans' repressed combat anxiety-provoking events: (1) one patient's repressed combat anxiety-provoking traumatic event involved frequent nightmares about the fear of someone torturing him to death. While the patient was undergoing BWNT, he re-experienced (relived), through hypnagogic imageries, a repressed combat anxiety-provoking event where he observed his platoon leader and two other enlisted soldiers torture to death a captured Viet Cong soldier who was tied to a tree to obtain military information. The patient, a jeep driver for the platoon leader, has felt displaced guilt-ridden emotions for not interceding/preventing the Vietnamese's death; (2) another episode of a repressed combat anxiety-provoking event concerned a patient's nightmares about his fear of being mutilated. While undergoing BWNT, the patient relived a repressed combat anxiety-provoking event that occurred when he was with Security Forces in Vietnam and his buddy was killed by two captured Viet Cong soldiers during an interrogation session. The patient blew one Viet Cong soldier's head off with a shot gun and the other Viet Cong soldier was beaten to death and his body dismembered; and (3) a third episode of a repressed combat anxiety-evoking event concerned a patient who had nightmares of survival guilt feelings. When the patient had a flashback at work, he became violent, destroying property, and he often had to be apprehended or sedated. While undergoing BWNT, the patient re-experienced a repressed combat anxiety-evoking event when he was out in the bush with his buddy on patrol duty. His buddy was wounded and he hid him in the brush along the trail and returned to the helicopter for assistance. It was getting dark and the helicopter crewmen were apprehensive about remaining in the Viet Cong area after dark. The patient was told that his buddy would be all right until morning and they would return to get him early in the morning hours. The following morning the helicopter crew and patient returned to the area where his buddy was hidden. The patient saw his buddy nude, hanging by his feet from a tree, his body mutilated. The patient has felt survival guilt feelings for not going back to get his wounded buddy that evening and/or staying with him.

These clinical observations lend some support to the hypothesis that Vietnam theater combat veterans are unable, in most incidences, to identify their combat-related flashbacks/nightmares with specific repressed combat anxiety-evoking traumatic events. BWNT tends to induce the vivid re-experiencing (reliving) of those extremely traumatic combat events that were repressed in Vietnam. In contrast, the Traditional Medical Control (TC) group did not show any significant changes in MMPI-indexed PTSD scale or a significant reduction in anxiety-provoking traumatic recurring nightmares/flashbacks. Nor were there any significant reductions in the TC patients' psychotropic medications for their combat-related PTSD. Only the patients in the BWNT group revealed accumulating evidence that improved functioning was being maintained over a two-year, six-month follow-up period. Some of the patients who underwent the BWNT program reported a few (1 to 2) instances of recurrence of the nightmares/flashbacks, as essentially anxiety-free episodes. Only three of fifteen BWNT-treated Vietnam veterans reported disturbing flashbacks/nightmares after a 30-month follow-up period. Of these three, all elected to undergo six booster BWNT sessions; one required rehospitalization during treatment. In contrast, the VA medical records indicated that all 14 TC patients have been readmitted to VA medical centers two or more times for PTSD during the 30-month follow-up period. These results provided supportive evidence that the BWNT patients' response to EEG BWNT resulted in moderately long-term prevention of PTSD relapse.

Some authors have proposed a link between PTSD and the activity of endogenous opioid peptides such as the endorphins and enkephalins (Copolov, 1985; Hoffman, Watson, Wilson & Montgomery, 1989; Rose, 1985; van der Kolk, 1987; van der Kolk, Greenberg, Boyd, & Krystal, 1985; Watson, Hoffman & Wilson, 1988). For example, Hoffman, *et al.* (1989) reported that plasma beta-endorphins were significantly lower in PTSD patients, in comparison to controls, and they suggested that chronic depletion of endogenous opioids may cause or maintain PTSD symptoms. In contrast, we (Peniston & Kulkosky, 1989) found that absence of a significant increase in circulating beta-endorphin levels accompanied the prevention of relapse in alcoholics who had received brainwave relaxation therapy. Further study of the relations of opioids to stress-related diseases is needed to clarify the roles of neuropeptides in the etiology, maintenance, relapse and therapy of disorders such as PTSD and alcoholism.

In summary, alpha-theta BWNT produced significant personality changes, reductions in combat-related PTSD symptomatology, and relapse, and reductions in psychotropic medications in Vietnam theater combat veterans. Follow-up data indicate that BWNT is clinically superior for long-term prevention of PTSD relapse in contrast to the control treatment. BWNT is a promising treatment alternative to traditional treatment modalities (i.e., rap groups, group therapy, individual therapy, flooding and desensitization therapies) for extinguishing PTSD and expanding the therapeutic knowledge presently available about treating posttraumatic stress symptoms. Also, it is suggested that traditional medical treatment such as rap groups, group therapy and individual psychotherapy, only reinforces and ventilates those combat-related posttraumatic stress behaviors of Vietnam theater veterans. These therapeutic procedures do not get at the cause and effect of Vietnam theater veterans' combat-related PTSD. Present results encourage further study of the mechanism of the therapeutic effect of alpha-theta brainwave neuro-feedback therapy and the contribution of demand characteristics, placebo or Hawthorne effects.

REFERENCES

- AMERICAN PSYCHIATRIC ASSOCIATION (1987). *Diagnostic and Statistical Manual of Mental Disorders* (revised 3 Ed.). Washington, DC: APA.
- ATKINSON, R.M., SPARR, L.F., SHEFF, A.G., WHITE, R., & FITZSIMMONS, J.T. (1984). Diagnosis of posttraumatic stress disorder in Vietnam veterans: Preliminary findings. *American Journal of Psychiatry*, 141 (5), 694-696.
- AYERS, M.E. (1983). Electroencephalographic feedback and head trauma. In *Head and neck trauma: The latest information and perspectives on patients with less-than-optimal recovery*, (pp. 244-257). Los Angeles: UCLA Neuropsychiatric Institute.
- AYERS, M.E. (1981). A report on a study of the utilization of electroencephalography (Neuroanalyzer) for the treatment of cerebrovascular lesion syndromes. In L.P. Taylor, M.E. Ayers & Tom (Eds.), *Electromyometric biofeedback therapy*, (pp. 9-12). Biofeedback and Advanced Therapy Institute.
- BALSON, P., & DEMPSTER, C. (1980). Treatment of war neuroses from Vietnam. *Comprehensive Psychiatry*, 21, 167-176.
- BREUER, J. & FREUD, S. (1936). *Studies in hysteria*. New York: Nervous and Mental Disease Publishing Co.
- BUDZYNSKI, T.H., & STOVVA, J.M. (1969). An instrument for producing deep muscle relaxation by means of analog information feedback. *Journal of Applied Behavior Analysis*, 2, 231-237.
- BUDZYNSKI, T.H. & STOVVA, J.M. (1973). Biofeedback techniques in behavior therapy. In D.

- veterans. In C.R. Figley (Ed.) *Trauma and its wake: The study and treatment of posttraumatic stress disorder*. New York: Brunner-Mazel.
- KOLB, L.C., BURRIS, B.C., & GRIFFITHS, S. (1984). Propranolol and clonidine in the treatment of the chronic posttraumatic stress disorders of war. In B.A. Van der Kolk (Ed.), *Post-traumatic stress disorder: Psychological and biological sequelae*, (pp. 98-105). Washington, D.C.: American Psychiatric Press.
- KULKA, R.A., SCHLENGER, W.E., FAIRBANK, J.A., HOGUE, R.L., KULKAMI, B., JORDAN, K., MARMAR, C.R., LUESS, D.S. (1988). *The national Vietnam veterans readjustment study (NVVRS): Description, current status, and PTSD prevalence estimates*. Research Triangle Park, North Carolina: Research Triangle Institute.
- LEAFY, M.R., & MARTIN, I.C. (1967). Successful abreaction after twenty years. *British Journal of Psychiatry*, 101, 141-148.
- LUBAR, J.O., & LUBAR, J.F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Journal of Biofeedback and Self-Regulation*, 9(1), 1-23.
- LUBAR, J.F., SHAHSEN, H., NATELSON, S.E., HOLDER, G., WITSETT, S.F., PAMPLIN, W.E., & KRULIDOWSKI, D. (1981). EEG operant conditioning in intractable epileptics. *Archives of Neurology*, 38, 700-704.
- MARSHALL, W.L., & SEGAL, Z. (1988). Behavior therapy. In C.G. Last and M. Hersen (Eds.), *Handbook of anxiety disorders*. New York: Pergamon Press.
- MERBAUM, M. (1977). Some personality characteristics of soldiers exposed to extreme war stress: A follow-up study of post-hospital adjustment. *Journal of Clinical Psychology*, 33, 558-562.
- PASSINI, F.T., WATSON, C.B., DEBEL, L., HERDER, J., WATKINS, B. (1977). Alpha wave biofeedback training therapy in alcoholics. *Journal of Clinical Psychology*, 33, 292-299.
- PENISTON, E.G. (1986). EMG biofeedback-assisted desensitization treatment for Vietnam combat veterans post-traumatic stress disorder. *Clinical Biofeedback and Health*, 9(1), 35-41.
- PENISTON, E.G. & KULKOSKY, P.J. (1989). Alpha-theta brainwave training and β -endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*, 13(2), 271-279.
- PENISTON, E.G. & KULKOSKY, P.J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*, 3, 37-55.
- ROSE, R.M. (1985). Psychoendocrinology. In J.D. Wilson and D.W. Foster (Eds.), *Williams' Textbook of Endocrinology*. Philadelphia: W.B. Saunders Co.
- SCHINDLER, F.E. (1980). Treatment by systematic desensitization of a recurring nightmare of a real life trauma. *Journal of Behavior Therapy and Experimental Psychiatry*, 11, 53-54.
- TRIMBLE, M.R. (1981). *Post-traumatic neurosis: From railway spine to the whiplash*. New York: John Wiley and Sons, Inc.
- VAN DER KOLK, B.A. (1983). Psychopharmacological issues in posttraumatic stress disorder. *Hospital and community psychiatry*, 34(8), 683-684, 691.
- VAN DER KOLK, B.A. (1987). *Psychological trauma*. Washington, D.C.: American Psychiatric Press.
- VAN DER KOLK, B.A., GREENBERG, M., BOYD, H., & KRISTAL, J. (1985). Inescapable shock, neurotransmitters, and addiction to trauma: Toward a psychobiology of post-traumatic stress. *Biological Psychiatry*, 20, 314-325.
- WATSON, C.G., & HERDER, J. (1978). Alpha biofeedback therapy in alcoholics: An 18-month follow-up. *Journal of Clinical Psychology*, 34, 765-769.
- WATSON, I.P.B., HOFFMAN, L., & WILSON, G.V. (1988). The neuropsychiatry of post-traumatic stress disorder. *British Journal of Psychiatry*, 153, 164-173.
- WILLIAMS, T. (Ed.) (1980). *Post-traumatic stress disorder of the Vietnam veteran*. Cincinnati: Disabled American Veterans.

- Shapiro, T.X., Barber, L.V., DiCara, J., Kamiya, N.E., Miller and J. Stoyva (Eds.), *Biofeedback and Self-Control 1972*, (pp. 437-459). Chicago: Aldine.
- CARROLL, E.M., RUEGER, D.B., FOY, D.W., & DONAHOE, C.P. (1985). Vietnam combat veterans with post-traumatic stress disorder: An analysis of marital and cohabitating adjustment. *Journal of Abnormal Psychology*, 94, 329-337.
- CANNON, D.S., BELL, W.E., ANDREWS, R.H., & FINKELSTEIN, A.S. (1987). Correspondence between MMPI/PTSD measures and clinical diagnosis. *Journal of Personality Assessment*, 51(4), 517-521.
- COPOLOV, D. (1985). Opioid biology: The next set of questions. *Australian and New Zealand Journal of Medicine*, 15, 98-106.
- DAILSTROM, W.G., WELSH, G.S., & DAILSTROM, L.E. (1972). *An MMPI Handbook (Vol.1): Clinical Interpretation*. Minneapolis: University of Minnesota Press.
- EGENDORF, A. (1975). Vietnam veterans' rap groups and themes of post war life. *Journal of Social Issues*, 31(4), 111-124.
- FAIRBANK, J.A., KEANE, T.M., & MALLOY, P.F. (1983). Some preliminary data on the psychological characteristics of Vietnam veterans with posttraumatic stress disorders. *Journal of Consulting and Clinical Psychology*, 51, 912-919.
- FIGLEY, C.R. (Ed.) (1978). *Stress disorder among Vietnam veterans: Theory, research and treatment*. New York: Brunner-Mazel.
- FOY, D.W., SIPPPELL, R.C., RUEGER, D.B., & CARROLL, E.M. (1984). Etiology of posttraumatic stress disorder in Vietnam veterans: Analysis of premilitary, military, and combat exposure influences. *Journal of Consulting and Clinical Psychology*, 52, 79-87.
- GOODWIN, J. (1980). The etiology of combat-related posttraumatic stress disorder. In T. Williams (Ed.), *Posttraumatic stress disorder of the Vietnam veteran*. Cincinnati, OH: Disabled American Veterans.
- GREEN, E.E., GREEN, A.M., & WALTERS, E.D. (1973). Biofeedback training for anxiety tension reduction. *Annals of New York Academy of Science*, 233, 157-161.
- GREEN, E.E., & GREEN, A.M. (1977). *Beyond biofeedback*. San Francisco: Delacorte.
- GRINKER, R.R., & SPIEGEL, J.P. (1945). *Men under stress*. New York: Blakiston.
- HALEY, S. (1978). Treatment implications of post-combat stress response syndromes for mental health professionals. In C. Figley (Ed.), *Stress disorders among Vietnam veterans*. New York: Brunner-Mazel.
- HALL, M.P. (1977). Theta training: Imagery and creativity. In E.E. Green and A.M. Green (Eds.), *Beyond biofeedback*. San Francisco: Delacorte.
- HOFFMAN, L., WATSON, P.B., WILSON, G., & MONTGOMERY, J. (1989). Low plasma β -endorphin in post-traumatic stress disorder. *Australian and New Zealand Journal of Psychiatry*, 23, 269-273.
- HYER, L., O'LEARY, W.C., SAUCER, R.I., BLOUNT, J., HARRISON, W.R., & BOUDEWYNS, P.A. (1986). Inpatient diagnosis of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 54(5), 698-702.
- HOROWITZ, M., & SOLOMON, G. (1975). A prediction of delayed stress response syndromes in Vietnam veterans. *Journal of Social Issues*, 31(4), 67-80.
- KARDNER, A. (1941). *The traumatic neuroses of war*. New York: Paul Hoeber.
- KEANE, T.M., MALLOY, P.F., & FAIRBANK, J.A. (1984). Empirical development of an MMPI subscale for the assessment of combat-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 52, 888-891.
- KEANE, T.M., & KALOUPEN, D.G. (1982). Imaginal flooding in the treatment of a post-traumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 50, 138-140.
- KEANE, T.M., FAIRBANK, J.A., CADDELL, J.M., ZIMMER, R.T., & BENDER, M.E. (1984). A behavioral approach to assessing and treating post-traumatic stress disorder in Vietnam

**EEG Alpha-Theta Brainwave Synchronization
in Vietnam Theater Veterans
With Combat-Related
Post-Traumatic Stress Disorder
and Alcohol Abuse**

Eugene G. Peniston
Sam Rayburn Memorial Veterans Center
Bonham, TX 75418

Dale A. Marrinan, Wendy A. Deming, Paul J. Kulkosky
Department of Psychology
University of Southern Colorado
Pueblo, CO 81001

Special thanks to Fort Lyon VA Medical Center for support of this study and to Lynwood Hoppel, M.D., EEG consultant to Psychiatry Service (116-A) for his assistance.

Inquiries or reprint requests should be directed to: Eugene G. Peniston, Chief, Psychology Service, Sam Rayburn Memorial Veterans Center, Bonham, TX 75418.

An experimental group of 20 male Vietnam combat veterans with a diagnosis of Post Traumatic Stress Disorder (PTSD) and alcohol abuse were treated with alpha-theta Brainwave Neuro-feedback Therapy (BWNT). A four channel EEG, video screen and printer were used to determine the efficacy of BWNT in developing synchronization and altering amplitudes of intrasubject brainwaves. Each patient was measured electrophysiologically before the start of treatment and immediately following the last BWNT session in which anxiety-provoking abreactive imagery was reported. Alpha-theta brainwave therapy produced significant increases in the percentage of synchrony in brain channel pairs in the frontal and parieto occipital lobes of the cerebral cortex in these patients. Also, the mean percentage of theta waves above preset amplitude threshold increased gradually across the 20 trials of the last abreactive session, while the mean percentage of alpha waves decreased. These changes in alpha and theta waves during the abreactive session resulted in a "cross-over" pattern as indicated by a significant interaction of wave type and trials. Further, it was found that the mean amplitudes of the alpha and theta brainwaves across the 20 trials of the abreactive imagery BWNT session displayed a corresponding cross-over pattern. This pattern identifies a state of consciousness referred to as the "EEG window of opportunity of the reverie state" which is believed to optimize the surfacing of hypnogogic and/or abreactive imageries.

INTRODUCTION

There is evidence to suggest that the war in Vietnam is not over for all of the veterans. Many Vietnam veterans suffer from the delayed after-effects of the war. This condition has been labeled by Keltner, Doggett, and Johnson (1983) as the "Vietnam Post-Traumatic Stress Disorder" (PTSD) or the "Post-Vietnam Syndrome." Included in the delayed after-effects are nightmares, flashbacks to previously experienced combat, and severe stress, among others. Keltner et al. (1983), estimated the number of veterans suffering from the effects of PTSD at 1.5 million.

There are numerous other events, in addition to war-time trauma, which elicit similar symptoms. Natural disasters, head injuries, severe burns, rape, concentration camp internment, and others can also result in similar symptomatology. This disorder, PTSD, has been recognized by the American Psychiatric Association and has been included in the *Diagnostic and Statistical Manual* (APA, 1987) under Anxiety Disorders (classification 309.89).

Reactions to stressful events have been thought to be biologically based as early as 1941 by A. Kardiner, who proposed that the PTSD-type symptomatology was a "physioneurosis." Everly (1989) postulated that the structures which mediate PTSD symptomatology reside in the hippocampal region of the brain, and effective treatment of PTSD must include a combination of psychotherapeutic and physiotherapeutic technologies. EEG alpha-theta brainwave neuro-feedback therapy is designed to train patients to change their brain function, and from this perspective, it appears to be a promising biobehavioral modality for intervention into PTSD (Peniston and Kulkosky, 1989, 1990, 1991).

Levine, Herbert, Haynes, and Strobel (1978) found that meditators are able to produce high amplitude, synchronous beta, alpha, and theta frequencies. The literature has indicated that during deep meditation, alpha and theta frequencies become synchronous. Synchrony in this study is defined as phase and frequency synchrony between the dominant frequency components from two channels, in a one second epoch. The in-phase criterion is that the phase angles of the dominant frequency components lie in the same quadrant. The various types of electroencephalographic (EEG) biofeedback are sensorimotor rhythm (Lubar and Lubar, 1984), alpha (Kamiya, 1961), alpha-theta (Green, Green, and Walters, 1980; Peniston and Kulkosky, 1989, 1990, 1991). Hypnagogic (i.e., dreamlike) images have been found to occur during the theta and/or "reverie" state (Foulkes and Vogel, 1965; Budzynski and Stoyva, 1969; Green, Green, and Walters, 1970). The combination of sensory and cognitive perceptions comprise the experiences of hypnagogic imagery. Green, Green, and Walters, (1974), associated this type imagery with creativity and integrative experiences and recently, Peniston and Kulkosky (1991) associated it with abreactive (i.e., traumatic anxiety-provoking) experiences. In order to experience vivid imaging, the subject should be in an alpha-theta mode for a sustained period of time. These images, or the memories, can then be retrieved while in a normal beta-alpha state or conscious mode. Fehmi (1978; Fehmi and Selzer 1980) found that training of brainwave synchrony tended to relieve stress-related symptoms, reduce sensations of chronic pain, and enhance selective attention with middle-management executives.

The purpose of this initial study was to: (1) examine the effects of a four channel EEG alpha-theta brainwave neuro-feedback training on EEG synchronization and wave form abundance and amplitude; (2) to bring forth the abreactive imagery that provides the opportunity for patients to deal with the causes associated with PTSD; and (3) to evaluate those clinical changes (i.e., flashbacks and nightmares) in PTSD patients after undergoing BWNT that has been described earlier.

METHOD

Subjects: The subjects in this study were 20 Vietnam theater veterans with twelve to fifteen year histories of chronic combat-related PTSD and coexisting alcohol abuse. Peniston and Kulkosky's (1989, 1990, 1991) earlier studies included alcoholics with PTSD syndrome. The present study will focus primarily on changes in dominant EEG rhythms (synchrony) and abreactive imageries in chronic combat-related PTSD patients. All subjects volunteered and gave their informed consent to participate in this study. The rationale for the subjects' participation in the experiment included statements on the informed consent form that the purpose of the study is to determine whether electrophysiological variables are factors that contribute significantly to the personality changes of veterans with combat-related PTSD (Peniston and Kulkosky, 1989, 1990, 1991). The subjects were randomly selected from a population of Vietnam veterans evaluated for treatment of chronic combat-related PTSD at Ft. Lyon V.A. Medical Center. They were selected based on the following criteria: (a) diagnosis of combat-related PTSD as defined by the DSM-III-R (APA, 1987); (b) no evidence of

psychotic symptomatology (i.e., hallucinations or delusions); (c) no known organic dysfunction; and (d) frequent (i.e., two to three episodes per week), anxiety-evoking, combat-related nightmares/flashbacks. All subjects were given a previously described EEG alpha-theta brainwave neuro-feedback treatment (Peniston and Kulkosky, 1989, 1990, 1991) for combat-related PTSD.

These alpha/theta brainwave therapy (BWNT) subjects had a mean chronological age of 37.25 years with a standard deviation (SD) of 2.82; a mean number of prior hospitalizations of 5.0, SD, .79; and a mean Shipley Institute Scale Intelligence Quotient (IQ) of 105.4, SD, 6.45.

DEPENDENT MEASURES

Percentage of Synchrony Per Quadrant Pair: CapScan Prism Five[®] displays percentage of synchrony per quadrant pair as follows, in accord with international 10-20 system for electrode placement (Jasper, 1958); (Phase 1 [F₇, F₈], Phase 2 [F₈, O₂], Phase 3 [O₁, O₂], Phase 4 [F₇, O₁]). Synchrony is calculated for each trial in each session and printed out to enhance pattern recognition. Signal analysis in CapScan Prism Five[®] is based on data derived from Fast Fourier Transforms (FFTs) on the signals from all 4 channels in successive 1 second intervals. Hamming weighting is applied to 128 time samples in each 1 second interval. The resulting 64 frequencies, bins, are one cycle apart and are centered on integral frequencies. Alpha (9-to-13 Hz), beta (14-to-29 Hz) and theta (4-to-8 Hz) band measures are attained by selecting and stating the amplitude of the largest component in the defined frequency range. Adjacent signals (Phase 1, 2, 3, or 4) are considered synchronous in a given 1-second interval when the largest amplitude component in the spectrum of the first signal occurs at the same frequency as its counterpart for the second signal, and (2) the FFT-derived phase angles of the two dominant amplitude components lie in the same vector quadrant (i.e., 0-90, 90-180, 180-270, 270-360). For the purpose of this study, the above described synchrony is defined as the predominance of a single brainwave frequency throughout several lobes, both hemispheres, or the entire cortex of the brain (Banquet, 1973) and served as the electro-physiological measure of synchrony.

Percentage of Time Above Threshold: Alpha (9-to-13 Hz), beta (14-to-29 Hz), and theta (4-to-8 Hz) sensitivity threshold settings were adjusted in the following manner for each subject: The audio-feedback screen (monitor) was observed for four or five trials (length of each trial = 90 sec) to calculate the highest amplitude of alpha, beta, and theta brainwave rhythms (in microvolts) which were exhibited during this initial phase. Then, the sensitivity threshold level was calculated by multiplying the peak amplitude by 40 percent which determined the alpha and/or beta threshold levels, while the critical theta sensitivity level was arbitrarily set 5-10 microvolts below the alpha threshold throughout the brainwave therapy session. The feedback tone occurs anytime the EEG signal exceeds the threshold level within the frequency band. As the percentage of feedback for alpha and theta rise above 25 percent during a 30-minute training session the threshold level was readjusted/increased to reduce the average amount of alpha and/or theta feedback to enhance learning of psychophysiological

skills. The audio-feedback unit of the instrument contained an individually controlled tone generator, and different tones provided audio-feedback for alpha, beta, or theta frequencies. If the frequency of alpha or theta remained above the threshold, a tone was presented, while the beta tone during training is turned off, although, beta frequency data continues to be collected. For computational purposes, each session was divided into twenty trials of 90 seconds each. A summary of the percentage of synchrony and the alpha/theta amplitudes was calculated for each trial and the mean calculation for the session was printed out at the completion of the training session.

Mean Amplitudes: The CapScan Prism Five® is calibrated with single frequency integral value sine waves. CapScan returns the peak to peak amplitudes in microvolts. Spectral analysis on mixed frequency signals is performed by the FFT with spectral lines 1 Hz apart at integral frequencies and using a Hamming time window.

Apparatus: The CapScan Prism Five® (American Biotech Corp., Ossining, NY) is a 4 channel electroencephalograph (EEG) system that is fully computerized to provide a brain mapping visual display system as well as a conventional EEG recording system. The system is a low noise, 4 channel, balanced differential system with two active commons, including a one channel, electrically isolated AC amplifier and differential input. The CapScan Prism Five® was used to provide audio-feedback, and to measure the subjects': (1) percentage of synchrony by quadrant pair for each 90-second trial across the 30-minute session; (2) frequency and amplitude per 1 second epoch; and (3) percentage of time above threshold by band width. An adjustable artifact inhibit detection circuitry, stops all feedback tones when the artifact (i.e., EMG from eye movement or other muscle signals) exceeds the selected artifact inhibit amplitude threshold. The audio-feedback threshold for each frequency band (alpha, beta, and theta) was displayed as a color coded horizontal line and was adjusted before each training session. Only alpha and theta band widths were fed back simultaneously to the subject using distinctly different notes which make up a musical chord.

Brainwave Training Procedure: Each subject was given a brief introduction to EEG brainwave biofeedback and was told how to interpret the audio-feedback monitor (i.e., alpha, beta, and theta) sounds in accord with Brainwave Neuro-feedback Therapy. During this initial session, and subsequently, the following procedures were implemented: the temporal regions (F_7 , F_8), a one (1) inch square on the forehead area, and an area approximately one (1) inch square on the two occipital positions, (O_1 , O_2), were cleaned with alcohol prior to attaching the electrodes. Omni Prep® was used as a conduction medium to fill the electrode cups and in preparation of the electrode scalp sites. The surface electrodes were applied, employing the international 10-20 system for electrode placement (Jasper, 1958). Both frontal (F_7 , F_8) electrodes were attached to the left and right temporal areas and one (1) ground electrode was attached to the forehead. Both occipital (O_1 , O_2) electrodes were attached approximately 1 cm above and 1 cm left/right of theinion and held in place by a velcro head band around the head. Two ear clip electrodes were attached and the active electrode was referenced to the left earlobe (A_1), with the ground on the right earlobe (A_2). Before recording commenced, electrode impedance was checked by connecting the electrode cable to an impedance meter. When all of the readings were low enough ($<10,000$ ohms), the electrode assembly was connected to the appropriate input on the back of the CapScan unit.

Electrophysiological measures were obtained during a 30-minute pretest session and a 30-minute posttest abreactive anxiety-provoking imagery BWNT session. Pretest electrophysiological measures were obtained prior to brainwave therapy, and consisted of: (1) percentage of synchrony per quadrant pair; and (2) mean alpha, beta, and theta amplitudes by band-width for twenty trials of 90 seconds each. Posttest electrophysiological measures were obtained during the last abreactive anxiety-provoking imagery BWNT session.

All subjects received five or six 30-minute session of pretraining in temperature biofeedback-assisted autogenic training and thirty 30-minute BWNT sessions (Peniston and Kulkosky, 1989, 1990, 1991). During the pretraining sessions, the medical psychotherapist (MP) attached a temperature thermistor to the tip of the middle finger of the subject's hand with micropore tape. The BWNT subjects were instructed to sit in a comfortable reclining chair, close their eyes, and relax. Then the MP introduced the subjects to autogenic training exercises and rhythmic breathing techniques (Green and Green, 1978) in an effort to induce relaxation of the body and quieting of the mind. In the following next five or six sessions the subjects practiced temperature biofeedback until the hand could be warmed to more than 94°F and held at that point for one entire session. It is believed that temperature training stimulates the production of the "theta state" (Hall, 1977). Following the temperature biofeedback pretraining session, the experimental subjects completed a total of thirty 30-minute BWNT sessions. Subjects were seen five times a week (5 days); twice daily for a total of twenty-one days. During BWNT, subjects received the following instructions from the MP: "close your eyes and construct a visualized abstinence/alcohol rejection scene... Now, tell your subconscious to go back to Vietnam where these traumatic combat events (nightmares and flashbacks) occurred and resolve these conflicts." Then the subjects were instructed to visualize imageries of increased alpha rhythm amplitude and scenes of the normalization of their personalities. Further, the MP instructed the subjects to "sink down" into a theta (reverie) state keeping the mind quiet and alert (but not active), and the body calm.

Finally, the subjects were instructed to initiate the session with a quiet command, "do it." Prior to the MP's exit from the room, the beta audio-feedback volume control was turned off while alpha and theta audio-feedback volume control bands were adjusted for a comfortable listening level for each subject and the overhead light was turned off. The MP returned to the room 30 minutes later and pressed the program termination key which printed out EEG data analyses, and gently returned the subjects to a state of awareness. At the end of each BWNT session the MP conducted a clinical interview reviewing the subject's verbal report on any visual or auditory images that were experienced during the BWNT session. The above mentioned procedures were employed throughout each of the 30-minute BWNT sessions.

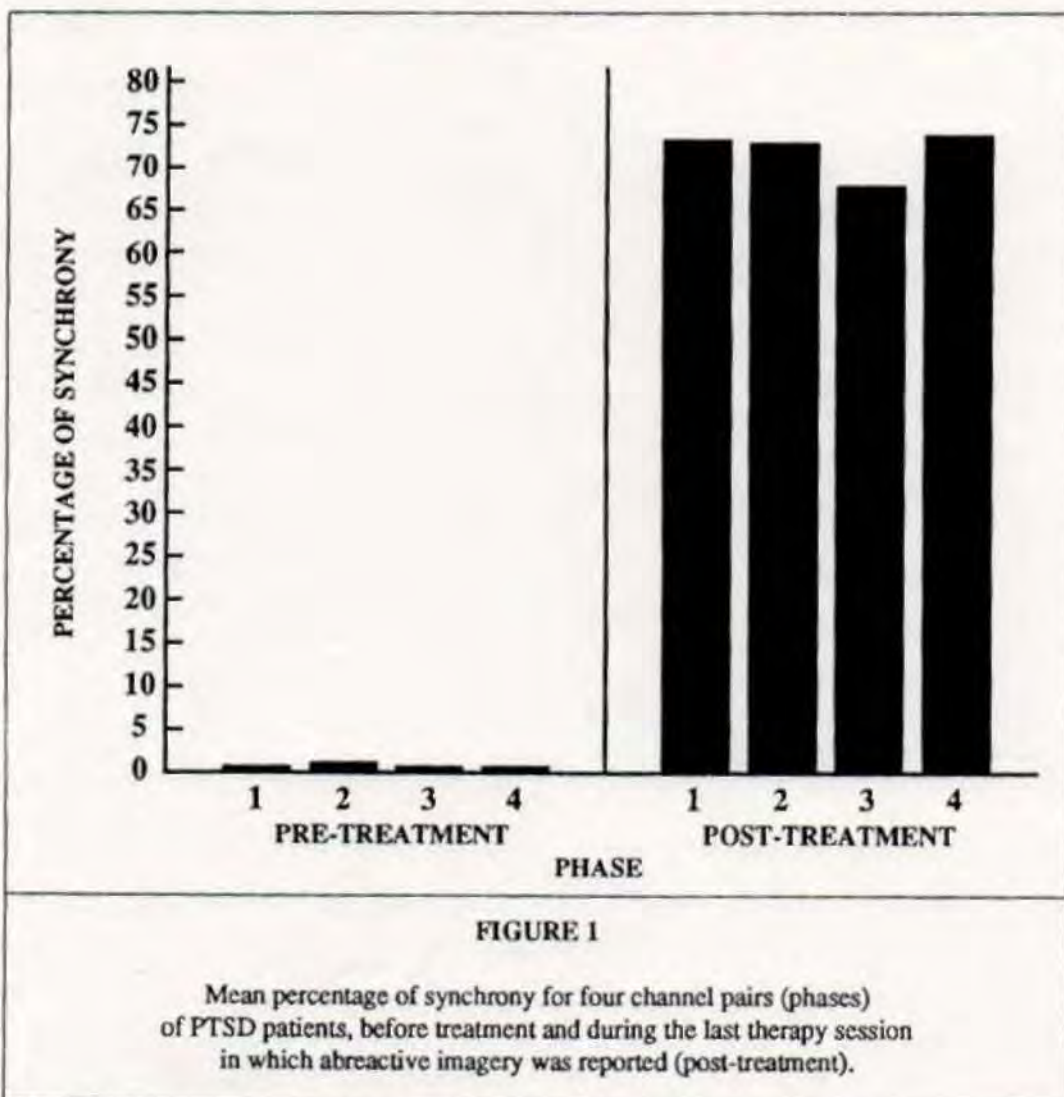
Data collection for this initial study was terminated at the completion of the last post-test abreactive brainwave therapy session. These data (pre and post percentages of synchrony per quadrant pair (phase) across session trials and mean amplitudes) were analyzed with repeated measures analyses of variance followed by Duncan's Multiple Range Test, at an alpha significance level of $p < 0.05$.

Follow-up Study: All twenty BWNT Vietnam theater veterans and their informers (wives, family members) were contacted by telephone at monthly intervals for 26 months after completion of BWNT. To determine the long-term effects of EEG alpha-theta brainwave

training, subjects and informers were asked to report instances of flashbacks and nightmares. In previous studies (Peniston & Kulkosky, 1989, 1990, 1992) the relapse rates for patients receiving BWNT were much lower than those patients receiving traditional therapy.

RESULTS

Percentage of Synchrony Per Quadrant Pair: Figure 1 displays the mean percentage of brain channel synchrony, before and after brainwave therapy, at the two pairs of electrode placements (phases 1-4) described above. A repeated measures analysis of variance revealed



significant main effects on synchrony of testing time (pre-treatment vs. post-treatment), $F(1, 3040) = 14,932.3, p < 0.05$, and phase (1-4), $F(3, 3040) = 5.76, p < 0.05$, but the effect of trials within sessions (1-20) was not significant, $F(19, 3040) = 0.48, p > 0.05$. The interaction of the factors of testing time and phase was significant $F(3, 3040) = 5.76$, but the other two-way interactions and the three-way interaction were not statistically significant, all $F_s < 1.0, p_s > 0.05$. Inspection of figure 1 reveals little synchrony at any electrode pair prior to treatment, and large increases in synchrony at each electrode pair (phase) after brainwave treatment. The increase in synchrony at phase 3 (electrode pair O_1, O_2) was slightly less than increases at the other phases on the post-treatment measurement, which accounts for the interaction of testing time and phase.

Mean Amplitudes: Figure 2 depicts mean amplitude (in microvolts) of alpha, beta, and theta brainwaves across the 20 trials of the initial pre-treatment measurement and the abreactive therapy session. Analysis revealed that the main effects on amplitude of brainwave frequency

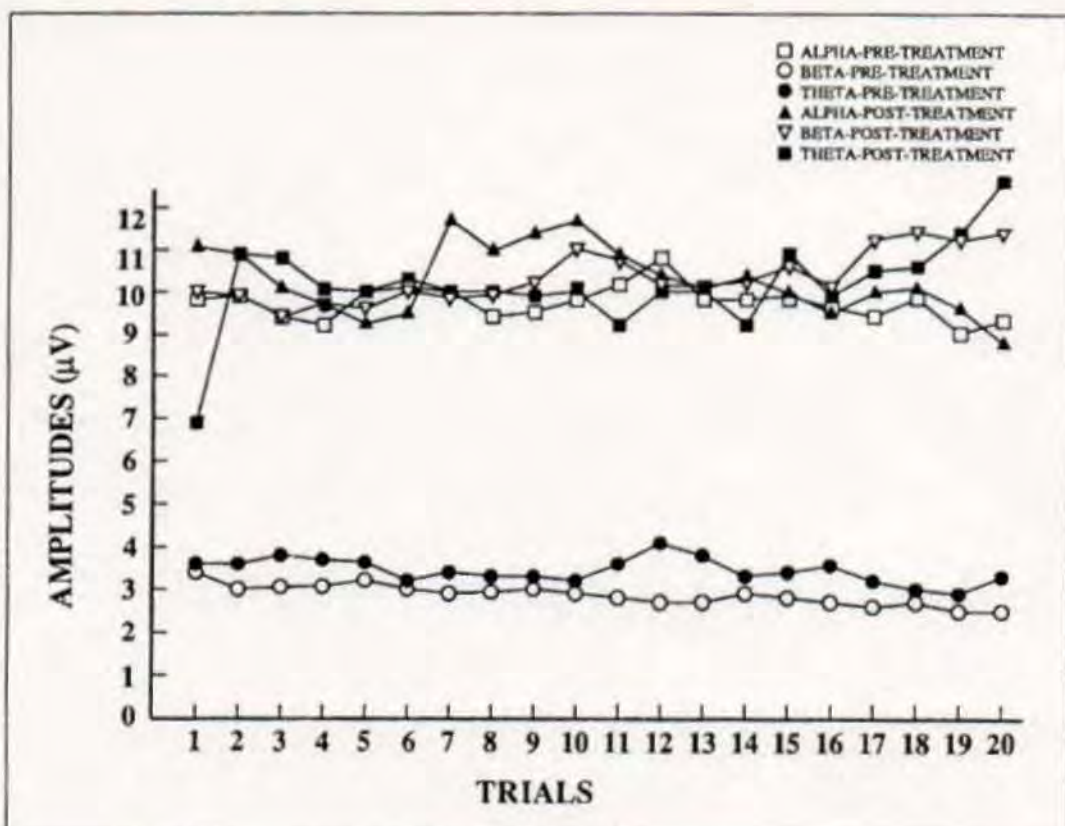


FIGURE 2

Mean amplitude of alpha, theta, and beta waves, before treatment and during the last abreactive therapy session (post-treatment).

band, $F(2, 2280) = 32.41$, $p < 0.05$, and of measurement period (pre- vs. post-), $F(1, 2280) = 56.98$, $p < 0.05$, were significant. The interaction of brainwave type and measurement period (pre- vs. post-) was statistically reliable, $F(2, 2280) = 30.61$, $p < 0.05$. This interaction may be seen in figure 2, wherein beta and theta waves increase greatly from pre- to post-treatment measurement periods, while alpha waves do not change substantially. The resulting alpha-theta "cross-over" pattern may be seen in figure 2, as theta waves gradually increase in amplitude across post-treatment trials, and alpha waves decrease across trials. In a separate analysis of alpha-theta amplitudes, this cross-over pattern is indicated by a significant interaction of wave type (alpha vs. theta) and post-treatment trials (1-20), $F(19, 361) = 2.00$, $p < 0.05$.

Follow-up Study: Table 1 displays counts of relapse or continued absence of PTSD symptoms of BWNT patients in the experimental group twenty-six months following training. Only four of the twenty experimental patients had relapsed by twenty-six months after training.

Table 1 Twenty-six month follow-up study		
	Relapse	No Relapse
N	4	16
Number of patients who showed relapse of PTSD symptoms or continued absence of symptoms, twenty-six months following BWNT training.		

DISCUSSION

In the present study, alpha-theta brainwave neuro-feedback therapy (BWNT) produced significant increases in the percentages of brain channel pair synchrony in the frontal and parieto occipital lobes of the cerebral cortex in Vietnam theater veterans with combat-related PTSD symptoms. It was further revealed that there were significant increases in the theta and beta, but not the alpha wave amplitudes in the abreactive session compared to pre-treatment measures. The mean amplitudes of alpha and theta brainwaves across the 20 trials of the abreactive imagery BWNT session displayed a statistically reliable interaction seen as a "cross-over" pattern, wherein theta waves gradually increase across trials and alpha waves decreased across trials. The aforementioned pattern is thought to identify a state of consciousness in which the patient is sensitive to hypnogogic imagery which relates symbolically to issues in the patient's own life (Davis, Davis, Loomis, Harvey, and Hobart, 1938; West, 1962; Stoyva, 1973; Dement and Kleitman, 1957; Foulkes and Vogel, 1965; Green, Green, and Walters, 1970; Kamiya, 1961; Budzynski and Stoyva, 1969; Kamiya and Nowlis, 1970). The above described state of consciousness has been referred to as the "EEG window of opportunity of the reverie state" where hypnogogic imageries surface (Davis et al., 1938; Foulkes and Vogel, 1965; Green, Green, and Walters, 1970). It is postulated that the increases in the theta

amplitude in conjunction with the decreases in the alpha amplitude during the abreactive session seem to be correlated with the strong affective experiences of childhood and/or adulthood—particularly, past traumatic anxiety-evoking events (i.e., abreactive imageries). These memories tend to surface during this BWNT session and this experience is targeted as a goal of brainwave neuro-feedback therapy. Alpha-theta brainwave neuro-feedback therapy gradually trains the patient to remain partly conscious as his EEG displays the pattern referred to as the "window of opportunity," wherein he can once again access these highly emotional (traumatic anxiety-provoking) images. This may reflect a newly learned state of consciousness which is close enough to a waking alpha state to facilitate transfer of these images.

In conclusion, it is hypothesized that the more the synchronicity and amplitude of theta waves increase the deeper the patient is able to descend into the reverie (theta) state which activates anxiety-evoking imageries. It is further postulated that during this state of consciousness, the limbic system and both hemispheres are more synchronized and the increased theta and beta rhythms reflect a brain process which enables the patient to remember and/or relive the traumatic anxiety-provoking event (Horowitz, 1970). Greater hemispheric synchrony appears to occur during the reverie state of the abreactive BWNT session (Surwillo, 1971; Busk and Galbraith, 1975). The aforementioned synchronization results seem to be consistent with Banquet's (1973) Levin, et al.'s (1978), and Venneman's (1991) findings. Winson (1972), 1990) thought that dreams (including nightmares/flashbacks) may reflect a memory-processing mechanism inherited from earlier species, in which information important for survival is reprocessed during the theta rhythm state. It is believed that dream content reflects the person's unconscious processes and is strongly correlated with the manner in which the subject is coping with crises in the real world (Cartwright, 1983; Trenholme, Cartwright, and Greenberg, 1984; Cartwright, Lloyd, Knight, and Trenholme, 1984). Following each traumatic imagery BWNT session, a clinical review was conducted, and each patient reported their abreactive imageries, which were recalled and then interpreted by the patients themselves without questions that might influence their interpretation. It was as though the patient was capable of integrating past traumatic experiences by coping with previously unresolved conflicts represented in the essentially anxiety-free imageries and memories generated during the theta state of consciousness.

Freud (1953), Kardiner and Spiegel (1947), and Kolb and Multalipassi (1982) postulated that traumatic anxiety-provoking imageries may be due to longstanding amnesias, shorter-term defenses against remembering, or the patient's inability to convey his internal experience. We theorize that whenever a person is blocking the memory of a traumatic anxiety-provoking event (i.e., he does not have any and/or very little conscious knowledge of the event) the frontal and parieto occipital areas of the brain are out of synchrony. When the person remembers/relives a traumatic anxiety-provoking event, the aforementioned areas of the brain are synchronous. The aforementioned contention is further supported by Hodgson and Rachman (1974) and Green, Romney, and Leboeuf (1989) who suggested that high arousal subjects will manifest a greater degree of synchrony between the physiological (skin conductance) and cognitive (subjective units of discomfort) measures of anxiety than will low arousal subjects. It is further postulated that the healing process (self-awareness) is manifested in high amplitude beta and theta waves in conjunction with the aforementioned cross-over

pattern of alpha and theta waves. This seems to enhance the patient's "flow-state" (i.e., all of the person's thought processes become focused) enabling the patient to understand his abreactive anxiety-provoking imageries. Only four of the twenty BWNT treated Vietnam Veterans reported a few (1-to-3) instances of recurrence of the nightmares/flashbacks, as essentially anxiety-free episodes after a 26-month follow-up period. Of these four, all elected to undergo seven booster BWNT sessions. These results provided supportive evidence that the BWNT patients' response to EEG BWNT resulted in moderately long-term prevention of PTSD relapse.

Present results encourage further study of the mechanism of the electrophysiological effects of alpha-theta brainwave neuro-feedback therapy and its underlying neurochemistry, as well as the contribution of placebo or Hawthorne effects.

REFERENCES

- American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders* (Third Edition-Revised). Washington, D.C.: Author.
- Banquet, J. P. Spectral analysis of the EEG in meditation. *Electroencephalography and Clinical Neurophysiology*. 35, 143-151.
- Budzynski, T. H. and Stoyva, G. M. (1969). An instrument for producing deep muscle relaxation by means of analog information feedback. *Journal of Applied Behavioral Analysis*. 2(4), 231-237.
- Busk, J. and Galbraith, G. C. (1975). Electroencephalography of visual-motor practice in man. *Electroencephalography and Clinical Neurophysiology*. 38, 415-422.
- Cartwright, R. D. (1983). Rapid eye-movement sleep characteristics during and after mood-disturbing events. *Archives of General Psychiatry*. February, 40(2), 197-201.
- Cartwright, R. D., Lloyd, S., Knight, S., and Trenholme, I. (1984). Broken dreams: a study of the effects of divorce and depression on dream content. *Psychiatry*. 47(3), 251-259.
- Davis, H., Davis, P. A., Loomis, A. L., Harvey, E. N., and Hobart, G. (1938). Human brain potentials during the onset of sleep. *Journal of Neurophysiology*. 1, 24-28.
- Dement, W. and Kleitman, N. (1957). Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalography and Clinical Neurophysiology*. 9, 673.
- Everly, G. S. Jr. (1989). *A Clinical Guide to the Treatment of the Human Stress Response*. New York: Plenum Press.
- Fehmi, L. G. (1978). EEG biofeedback, multichannel synchrony training, and attention. In A. A. Sugarman and R. E. Tarter (Eds.). *Expanding Dimensions of Consciousness*. New York, New York. Springer Publishing Company.
- Fehmi, L. G. and Selzer, F. A. (1980). Biofeedback and attention training. In Boorstein, MD, Seymour, (Ed.). *Transplanted Psychotherapy*. Palo Alto, Ca. Science and Behavior Books, Inc. 314-337.
- Foulkes, D. and Vogel, G. (1965). Mental activity at sleep onset. *Journal of Abnormal Psychology*. 70, 231-243.

- Freud, S. (1939). In *The Interpretation of the Complete Psychological Works of Sigmund Freud*, Dreams. Standard Edition, J. Strachey (Ed.). London: Hogarth Press, 1953.
- Gerew, A. B., Romney, D. M., and Leboeuf, A. (1989). Synchrony and desynchrony in high and low arousal subjects undergoing therapeutic exposure. *Journal of Behavior Therapy and Experimental Psychiatry*. 20(1), 41-48.
- Green, E. E., Green, A. M., and Walters, E. D. (1970). Voluntary control of internal states. *Journal of Transpersonal Psychology*. 1, 1-26.
- Green, E. E., & Green, A. M. (1977). *Beyond Biofeedback*. San Francisco, Delacarte.
- Hall, M. P. (1977). Theta training: imagery and creativity. In Green, E. E. and Green, A. M. (Eds.). *Beyond Biofeedback*. San Francisco, Delacarte. 118-152.
- Hodgson, R. and Rachman, S. (1974). Desynchrony in measures of fear (II). *Behavior Research and Therapy*. 12, 319-326.
- Horowitz, M. J. (1970). *Image Formation and Cognition*. New York: Appleton-Century-Crofts.
- Jasper, H. H. (1958). The 10-20 electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology*. 10, 371-375.
- Kamiya, J. (1961). Behavior, subjective, and physiological aspects of drowsiness and sleep. In Fiske, W. and Maddi, S. (Eds.). *Functions of Varied Experience*. Homewood, Ill. Dorsey Press.
- Kamiya, J. and Nowlis, D. (1970). The control of electroencephalographic alpha rhythms through auditory feedback and the associated mental activity. *Psychophysiology*. 6, 476.
- Kardiner, A. (1941). *The Traumatic Neuroses of War*. New York: P. Hoeber.
- Kardiner, A. and Spiegel, H. (1947). *War Stress and Neurotic Illness*. New York. Appleton-Century-Crofts.
- Keltner, N. L., Doggett, R., and Johnson, R. (1983). For the Vietnam veteran the war goes on. *Perspectives in Psychiatric Care*. 21, 108-113.
- Kolb, L. C., & Multalipassi, L. R. (1982). Conditioned emotional response: a sub-class of the chronic and delayed post-traumatic stress disorder. *Psychiatric Annals*. 12, 979-987.
- Levine, P. H., Herbert, R., Haynes, C. T., and Strobel, U. (1978). EEG coherence during the transcendental meditation technique. In D. W. Orme-Johnson, and J. T. Farrow (Eds.). *Scientific Research on the Transcendental Meditation Program: Collected Papers*. 1(20).
- Lubar, J. O. and Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation*. 9(4), 585-600.
- Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*. 13(2), 271-279.
- Peniston, E. G. (1990). EEG brainwave training as a bio-behavior intervention for Vietnam combat-related PTSD. *The Medical Psychotherapist*. 6(2), Spring.
- Peniston, E. G., and Kulkosky, P. J. (1990). Alcoholic Personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*. 3, 37-55.
- Peniston, E. G., & Kulkosky, P. J. (1991). Alpha-theta neurofeedback therapy for Vietnam veterans with combat related post-traumatic stress disorder. *Medical Psychotherapy: An International Journal*. 4, 47-60.

- Stoyva, G. M. (1973). Biofeedback techniques and the conditions for hallucinatory activity. In McGuigan, F. G. and Schoonover, R. A. (Eds.). *The Psychophysiology of Thinking*. New York and London. Academic Press. 387-414.
- Surwillo, W. W. (1971). Interhemispheric EEG differences in relation to short term memory. *Cortex*. 7, 246-253.
- Trenholme, I., Cartwright, R. D., and Greenberg, G. (1984). Dream dimension differences during a life change. *Psychiatry-Research*. May, 12(1), 35-45.
- Venneman, J. A. (1991). Effects of thermal and EEG biofeedback on brainwave synchrony. *Medical Psychotherapy*. 4, 61-70.
- Weil, A., and Rosen, W. (1983). *From Chocolates to Morphine: Understanding Mind-Active Drugs*. Boston: Houghton Mifflin.
- West, L. G. (1962). A general theory of hallucinations and dreams. In West, L. G. (Ed.). *Hallucinations*. New York: Grune & Stratton. 275-291.
- Winson, J. (1972). Interspecies differences in the occurrence of theta. *Behavioral Biology*. 7(4), 479-487.

α - θ Brainwave Training and β -Endorphin Levels in Alcoholics

Eugene G. Peniston and Paul J. Kulkosky

An α - θ brainwave biofeedback training program was applied as a novel treatment technique for chronic alcoholics. Following a temperature biofeedback pretraining phase, experimental subjects completed 15 30-min sessions of α - θ biofeedback training. Compared to a nonalcoholic control group and a traditionally treated alcoholic control group, alcoholics receiving brainwave training (BWT) showed significant increases in percentages of EEG record in α and θ rhythms, and increased α rhythm amplitudes. Alcoholics receiving BWT showed a gradual increase in α and θ brain rhythms across the 15 experimental sessions. These experimentally treated alcoholics showed sharp reductions in self-assessed depression (Beck's Depression Inventory) compared to the control groups. Alcoholics receiving standard medical treatment (abstinence, group psychotherapy, antidepressants) showed a significant elevation in serum β -endorphin levels at the conclusion of the experiment. This neuropeptide is an index of stress and a stimulant of caloric (e.g., ethanol) intake. Application of brainwave treatment, a relaxation therapy, appears to counteract the increase in circulating β -endorphin levels seen in the control group of alcoholics. 13-month follow-up data indicate sustained prevention of relapse in alcoholics that completed α - θ brainwave training.

RECENTLY, investigations have described the electroencephalographic (EEG) features of human alcoholism. Several groups that have studied the sons of alcoholics and chronic alcoholics have demonstrated that even after prolonged abstinence, alcoholics often have lower levels of α waves on background cortical EEGs before ethanol. Further, they are more likely to increase the amount of α waves after alcohol challenge.¹⁻³ Associated with the poor EEG synchrony exhibited by alcoholics is deficient α activity and decreased amplitudes and increased latencies in some subwaves of event-related potentials.⁴⁻⁸ These findings suggest that some persons with a predisposition to the development of alcoholism are characterized by deficient α activity compared to controls.^{5,9-11} If persons with predisposition to development of alcoholism exhibit deficient α activity while sober, they may be especially vulnerable to alcohol's effects, if drinking enables them to attain a reinforcing psychological state associated with increased α activity.

Major outcome studies that have used specific therapeutic

interventions such as controlled drinking, abstinence, compulsory AA attendance, and an active follow-up program yielded results after 2 and 8 years that were no better than those of the natural history of the disorder.¹²⁻¹⁴ Considering the overall lack of success of other treatment techniques (i.e., controlled drinking, abstinence, compulsory AA), there is an urgent need for a more efficacious, innovative treatment approach in dealing with alcoholism. Over the past two decades, a variety of techniques subsumable under the global label of relaxation training have been used to treat an exceedingly diverse array of clinical problems, including depression and alcohol abuse.¹⁵⁻¹⁸ The application of relaxation training in the treatment of substance abuse is effective in alleviating anxiety, assessed either in nonstress or stress situations that have been associated with increased drinking of alcohol.¹⁹⁻²⁴ Using such techniques as systematic desensitization and biofeedback, researchers and therapists have taught voluntary control of a variety of physiological changes including blood pressure, muscle tension, skin temperature, and particular brain-rhythm patterns. However, the empirical efficacy of these techniques, and their long term efficacies, remains equivocal.²⁵⁻²⁸

EEG brainwave α - θ training, a biofeedback technique used to learn control of particular brain-waves, is being increasingly applied for the treatment of a variety of disorders. Many researchers involved in the study of α brainwaves biofeedback training concur on one broad conclusion regarding its character, that a relaxed state is associated with the α rhythm, labeled "enjoyable," "tranquil," "calm," and "serene."²⁹⁻³¹ Much less has been published about the study of the θ rhythm, though it is known that it appears in the brainwaves record of deeper stages of meditation.³²⁻³⁴ Results of the application of relaxation training techniques to the treatment of alcohol abuse neither clearly support nor disconfirm the efficacy of such techniques.²⁵⁻²⁸ It is hypothesized that the EEGs of chronic alcoholics provided prolonged EEG brainwave α - θ training will show reliable elevations of EEG α amplitudes and an increase in the amount of EEG α activity.

Aside from the EEG α deficiencies exhibited by some alcoholics, alterations have been identified in steroid and peptide hormones and biogenic amines.^{35,36} However, there is relatively little known about the causes and effects of changes in the levels of humoral factors in alcoholism. It is speculated that changes in EEG may correlate with significant changes in circulating β -endorphin levels.³⁷ β -

From the Veterans Administration Medical Center, Fort Lyon, Colorado and The Department of Psychology, University of Southern Colorado, Pueblo, Colorado.

Received for publication June 20, 1988; revised manuscript received November 23, 1988; accepted November 23, 1988.

Reprint requests: Eugene G. Peniston, Clinical Psychologist, Psychology Service (116B), VA Medical Center, Fort Lyon, CO 81038.

Copyright © 1989 by The Research Society on Alcoholism.

Endorphin is an endogenous 31-amino acid opioid peptide that functions to regulate bodily responses to stress by controlling responsiveness to pain.³⁸⁻⁴² Since this neuro-peptide increases in blood under conditions of stress^{41, 43, 44} a relaxation therapy such as α - θ training may be expected to lower β -endorphin levels. Injection of the opioid β -endorphin potentiates the behavioral effects of ethanol,^{45, 46} and chronic ethanol treatment lowers plasma and pituitary β -endorphin in rats.⁴⁷⁻⁴⁹ However, acute ethanol treatment in vivo or in vitro stimulates β -endorphin release.^{50, 51} Many authors have proposed a link between alcoholism and the activity of endogenous opioids⁵²⁻⁵⁴ and other peptides,⁵⁵ but no conclusive evidence of such a link is yet available.

An EEG brainwave α - θ training program was used as an innovative treatment technique for chronic alcoholic patients. The purpose of this initial study was threefold: (a) to test the electroencephalographic effects of brainwave α - θ training on chronic alcoholics; (b) to determine whether significant changes occur in the blood levels of β -endorphin in fasting human alcoholics after admission or at termination of the EEG brainwave training program, in comparison with matched alcoholic and nonalcoholic controls; and (c) to determine the efficiency of EEG brainwave training as indexed by changes in assessed depression, in comparison with the nonalcoholic controls and subjects receiving the traditional medical program (abstinence, psychotherapy, and psychoactive drugs) for the treatment of alcoholism.

SUBJECTS AND METHODS

The sample was composed of 30 subjects who were randomly selected from three different socioeconomic statuses (lower, middle, and upper-middle) of a population from the alcohol treatment unit ($n = 20$), and the medical center ($n = 10$). The criteria for selection of the alcoholic group included: (a) subjects with alcoholism diagnoses based on DSM-III⁵⁶ and clinical records maintained in the medical center; (b) subjects' medical records indicate four or more prior hospitalizations for alcohol treatment at various hospitals; (c) subjects' medical records indicate 20 or more years of alcoholism; (d) subjects with low average and above intelligence quotients (IQs); and (e) subjects were not on psychotropic medications for psychiatric problems. These subjects were assigned at random to either the EEG brainwave α - θ training experimental alcoholic group (EXPALC, $n = 10$), given a modified version of EEG brainwave α - θ training, or to a traditional control alcoholic group (ALCONT, $n = 10$) that was given daily group therapeutic sessions and lectures. Subjects ($ns = 2$) in both alcoholic groups received antidepressant medications as determined by the attending physician. A third group ($n = 22$) was identified as nonalcoholics through records and interviews of a population within the medical center. Ten subjects were assigned randomly to the nonalcoholic control group (NONALC). These control subjects ($n = 10$) were also given the pre- and post-EEG, α - θ brainwave, depression inventory, and blood sample measures, and were matched with EXPALC and ALCONT subjects on age and social class.

The EXPALC group had a mean alcoholic history (ALC/H) of 22.50 years (standard deviation (SD), of 7.16); a mean number of prior hospitalizations (prior/H) of 5.4 (SD, 1.42); a mean Shipley Institute Scale intelligence quotient (SIS)IQ of 106.80 (SD, 19.60); a mean chronological age (CA) of 49.29 years (SD, 10.57); and a mean social-economic status (SES) of 4.19 (SD, 0.42). The ALCONT group had a mean ALC/H of

21.20 years (SD, 5.95); a mean number of prior/H of 5.40 (SD, 1.42); a mean SIS IQ of 107.09 (SD, 18.65); a mean CA of 49.00 (SD, 10.16); and a mean SES of 4.09 (SD, 0.56). The NONALC group had a mean SIS IQ of 126.44 (SD, 7.98); a mean CA of 44.09 (SD, 12.04); and a mean SES of 3.09 (SD, 0.87).

Social-Economic Status

Hollingshead's⁵⁷ Two Factor Index of Social Position was used in this study to assign subjects to one of five distinct social classes. In the aforementioned process the subjects were asked to report on their occupation and education. The two factors employed in the Two Factor Index of Social Position are "... (1) the precise occupational role the head of the household performs in the economy, and (2) the amount of formal schooling he has received."⁵⁷ Occupation responses were coded into seven categories: (a) executives and proprietors of large concerns and major professions; (b) managers and proprietors of medium sized concerns and minor professions; (c) administrative personnel of large concerns, owners of small businesses and semi-professionals; (d) owners of little businesses, clerical and sales workers and technicians; (e) skilled workers; (f) semiskilled workers; and (g) unskilled workers. Seven categories were used to measure educational participation: (a) graduate degree; (b) standard college or university graduate; (c) some college but no degree; (d) high school graduation (e) some high school, 10-11 years; (f) completion of 7-8-9 grade; or (g) less than the seventh grade. The individual's rank score on each dimension is multiplied by its respective weight (7 for occupation and 4 for education) and summed to achieve a total score from 11 to 77.

Five classes are created as follows: Class I (11-17); Class II (18-27); Class III (28-43); Class IV (44-60); and Class V (61-77).⁵⁸ In general, the lower the point total the higher the social class or status of the subject's family. This schema has been used extensively in other studies.^{58, 59}

Beck Depression Inventory (BDI)

Each participating subject was asked to respond to a self-report measure designed to assess depression prior to and after completion of treatment. The BDI⁶⁰ is designed to assess the severity of a variety of symptoms of depression. Each of 21 items consists of four sentences, and the subject is instructed to choose the one that best describes himself at the present time. Each set of sentences describes symptoms of depression, ranging from normalcy to severe clinically significant symptoms. Each item is scored from 1 to 4, resulting in a range of scores from 21 to 84. Limits of severity are based on mean scores (i.e., normal range below 50, mild to minimal depression 50-59, moderate to marked depression 60-69, severe to extreme depression 70 and over). BDI has been widely used in research studies investigating individual differences in severity of levels of depression.⁶¹⁻⁶⁴ Subjects also completed Millon Clinical Multiaxial Inventory (MCMI) and Sixteen Personality Factor (16PF) personality inventories (Peniston and Kulkosky, unpublished observations).

β -Endorphin Levels

Two 5.0-ml blood samples were drawn by venipuncture from members of each group (EXPALC, ALCONT, and NONALC) of patients before and after the brainwave training (BWT) experiment. Each sample was taken after a 14-h fast, between the hours of 9:00 and 10:00 a.m., when the circadian rhythm of endorphin concentration in plasma was at the medium level in men.^{43, 44, 65} The first blood sample was collected prior to the initial BWT session 1 week after admission and abstinence. For 1 hr prior to each sampling subjects were given a systematic desensitization^{21, 66} session, i.e. while the patient remained deeply relaxed, he was instructed to imagine various situations or stimuli which normally produced a mild anxiety reaction. The final blood sample was obtained at the end of the BWT experiment, and the patients were again given a systematic desensitization session 1 hr prior to sampling.

Other important variables that may influence the metabolism and secretion of endorphins such as stress^{39,41,65} and motoric behavior^{67,68} were stabilized by providing a desensitization session 1 hr prior to sampling.

5-ml whole blood samples were collected in 5- or 10-ml vacutainer glass tubes with EDTA 7.2 mg/5 ml as an anticoagulant, and spun in a refrigerated centrifuge for 15 min at $750 \times g$. The plasma samples were then placed into storage tubes and immediately frozen and stored at -20°C . The specimens were then immersed in liquid nitrogen, packed in dry ice, and shipped by air to Edward Hines VA Medical Center, Endocrinology Laboratory (Hines, IL) to be analyzed. β -Endorphin concentrations were determined from extracted serum samples of blood by radioimmunoassay (Inctstar, 46065), at an assay sensitivity of 4.7 pmol/liter.

Medication Consumption

After 1 week of daily practice of α - θ BWT, the drug dosage (tricyclic antidepressants) of BWT subjects ($n = 2$) and ALCONT subjects ($n = 2$) was gradually reduced at their request. During BWT sessions, subjects were monitored by the physicians on the Alcoholic Treatment Unit (ATU) and in the Outpatient Clinic throughout the withdrawal period. The physicians were aware of the treatment groups (EXPALC and ALCONT) and a weekly record was maintained on each patient's medication reductions. If an attempt by the physician to withdraw the subject's initial antidepressant medication resulted in intense depression, the subject was reintroduced to medication.

Electroencephalogram Recording

The Beckman Accutrace TM 200 Autoencephalograph, 16 channel recorder was used to register 60-min EEG recordings before and after the BWT experiment. Prior to the initiation of BWT, all three (EXPALC, ALCONT, and NONALC) groups were administered a pre-EEG assessment, BDI, blood sample extraction and base-rate EEG brainwave measure. Pre-post base-rate measures of α and θ production (percentage of session time in which α and θ was produced) in 5-min intervals were obtained from each subject prior to initial training session and after the last training session. No feedback signals were presented to the subjects during the pre-post base-rate brainwave measurements. The EEG assessments were administered to the subjects during the same hours of the day over a 5-day period. The International 10-20 system for electrode placement was used.⁶⁹ Subjects' EEG assessment included the bilateral control (C_3 , C_4), parietal (P_3 , P_4), and occipital (O_1 , O_2), scalp derivations which were referenced against linked ears. All EEG records were visually inspected by two independent raters for artifact (i.e., muscle, EKG, blinking, etc.) detection and EEG synchrony. The raters were unaware of the subject's assignment in the experimental groups. The EEG records from each group (EXPALC, ALCONT, and NONALC) were presented randomly and then rated independently by the two raters. Each rating was based on three levels (central, parietal, and occipital) of scalp region, and laterality (two levels: left and right) of scalp sites. For the purpose of this study, the EEG records were rated on the following distinct EEG markers. The first characteristic was the average α wave frequency (in Hz) and the average amplitude (measured in μV) of α rhythm generated in each EEG record. The second characteristic obtained was the percentage of α frequency waves exhibited. Percentage agreement was obtained by dividing the pre- and post-EEG evaluations between raters by agreements plus disagreements and multiplying by 100. Interraters agreement on the EEG markers ranged from 86 to 100% across all EEG evaluations in which reliability was assessed.

Apparatus

An Autogen 2000 Feedback Thermometer (Autogenic Systems, Inc.) was used to measure the subjects' temperatures and to provide audio feedback. Audio feedback was in the form of a beep tone that rose in pitch as subjects' temperatures increased and that lowered in pitch to

corresponding decreases in temperature. The thermometer data was collected in the form of degrees Fahrenheit using an Autogen 5600 Digital Integrator. The integrator supplied a digital record of summated temperature activity, once every 30 sec. The EEG Feedback Monitor (Model E 430) and EEG Timer (ET 330) (RI Company) were used to measure the subjects' brainwave activity and to provide both audio and visual feedback. This EEG Monitor detects information in raw EEG by using three active band-pass filters. α (8–13 Hz), β (13–26 Hz), and θ (4–8 Hz) rhythms are detected by filters with 71 dB per octave attenuation rates. The microprocessor-based Timer accumulated time for a EEG band whenever the signal exceeded the threshold for that band. The audiovisual feedback unit of the instrument contained an individually controlled tone generator. The microvolt levels for each of these band-pass filters were controlled independently, and different individual tones provided audio feedback for the α , β , or θ frequencies. If the brainwave frequency (α , β , or θ) remained above the threshold, a distinct tone was continuously presented. In addition to this audio feedback, a separate set of counters on the EEG Timer was activated by the presence of α , or β , or θ thresholds and thus a calculation of accumulated time was available. At the conclusion of training the EEG Timer also calculated the percentage of time that each band exceeded the threshold levels.

BWT

All subjects were given a brief introduction to EEG brainwave biofeedback and were told how to interpret the audio feedback (i.e., β , α , θ) sounds. During this initial session the following procedures were implemented. Each subject was seated in a comfortable reclining chair in a sound-proof room and was instructed to sit quietly and relax with eyes closed for 5 min while a base-rate recording was obtained. For biofeedback training purposes, monopolar electrode placements were used to provide a stable high amplitude signal to the instrument input. Earlobes and the area around theinion were cleaned with alcohol prior to attaching the electrode leads. Omni Prep was used as a conduction medium to fill the electrode cups and in the preparation of the electrode scalp site. An occipital (O_1) electrode was attached approximately 1 cm above and 1 cm left of the inion and held in place by a stretching headband. Two ear-clip electrodes were attached and the active electrode was referenced to the left earlobe (A_1), with the ground electrode on the right earlobe (A_2). Before recording commenced, electrode impedance was checked and electrodes were reapplied if necessary. β , α , and θ sensitivity threshold settings were adjusted on the feedback monitor for each subject. Prior to recording each individual's initial α and θ base-rate scores, the threshold dials of the feedback monitor were adjusted (aided by the use of a MFE Posi-Traci 1-strip chart recorder) to a point at which the waves characteristic of β , α , and θ registered on the feedback monitor and on the cumulative recording computer-based timer. Because θ was not produced uniformly during the calibration sessions, this procedure could not be used to set a θ threshold. Instead, θ thresholds were arbitrarily set at points $10 \mu\text{V}$ below those for α because the θ and α thresholds of previous patients who had produced θ during calibration tended to differ by this amount. β (13–26 Hz), α (8–13 Hz), and θ (4–8 Hz) rhythms were defined in terms of time that the input signals exceeded the machine-set thresholds.

Only the EXPALC subjects received eight 30-min sessions of pretraining in temperature biofeedback assisted autogenic training and 15 30-min BWT sessions. During the pretraining sessions, the experimenter (E) attached a temperature thermistor to the tip of the middle finger and middle toe of the subject's dominant hand and foot with micropore tape. The BWT subjects were instructed to sit in a comfortable reclining chair and relax and close their eyes. Then the E introduced the subjects to autogenic training exercises and rhythmic breathing techniques in an effort to induce relaxation of the body and quiet the mind. On the following six or seven sessions, the subjects practiced temperature feedback until the hand and foot could be warmed to more than 95°F and held there over one session. It is speculated that temperature training stimulates the production of the "theta state."³⁴ Following the tempera-

ture biofeedback pretraining sessions, the experimental subjects completed a total of 15 5-min baseline intervals and 15 30-min BWT sessions. Subjects were seen five times a week (5 days) for a duration of 28 days. Subjects were instructed to close their eyes and construct visualized abstinence/alcohol rejection scenes, and imageries of increased α rhythm amplitude and scenes of the normalization of their personalities. The E instructed the subjects to "sink-down" into θ (reverie) state keeping the mind quiet and alert (but not active), and the body calm. Then, subjects were instructed by the E, to initiate the session with a quiet command, "do it." Prior to the E's exit from the room, the β feedback volume control band was turned off; α and θ feedback volume control bands were adjusted for a comfortable listening level for each subject; and the overhead light was turned off. The E returned to the room 30 min later, E pressed the "stop" button of the computer based timer and gently returned the subjects to a state of awareness. The aforementioned procedures were employed throughout the 15 30-min sessions.

Data collection for this initial study was terminated at the end of the 15th 30-min session. The subjects were readministered the EEG assessment, BDI, blood sample extraction, and base-rate EEG brainwave measures. These data (pre- and postmeasures), in conjunction with the 15 5-min baseline intervals and 30-min BWT sessions, were analyzed with split-plot analyses of variance, followed by Duncan's test, at an α significance level of $p < 0.05$.

The ALCONT and NONALC groups were given only the pre- and post-EEG assessments, BDI, repeated blood sample extractions, and base-rate EEG brainwave measures. Both control groups were instructed not to use any biofeedback relaxation training procedure during the study. The rationale for the subjects' participation in the experiment included statements on the informed consent form that the purpose of the study is to determine if EEG α - θ brainwave training will significantly change β -endorphin levels associated with human alcoholism, and if brainwave training is associated with changes in the α rhythms.

Follow-Up Study

All 20 (EXPALC and ALCONT) alcoholic subjects and their informers (wives, family members, halfway house superiors) were contacted by telephone at monthly intervals for 13 months after completion of treatment. To determine the long-term effects of EEG α - θ brainwave training, subjects and informers were asked to report instances of alcoholic relapse, defined as drinking constantly for a 1-week duration. Data were analyzed with a χ^2 test after application of Yates' correction for continuity, at an α significance level of $p < 0.05$.

RESULTS

β -Endorphin Levels

Table 1 presents mean and standard error (SE) radioimmunoactive β -endorphin levels of extracted serum samples from alcoholic controls (ALCONT, $n = 8$, two samples not obtained), nonalcoholic controls (NONALC, $n = 9$, one sample not obtained), and experimentally treated alcoholics (EXPALC, $n = 9$, one sample not obtained), before and after α - θ brainwave training of the EXPALC group. Analysis revealed a significant main effect of sampling time, $F(1,23) = 6.68$, $p < 0.05$. Duncan's posthoc test indicated a significant increase in β -endorphin level only in the ALCONT group ($p < 0.05$). The mean peptide level in the ALCONT group also was significantly greater than that of the NONALC group at the posttreatment sample ($p < 0.05$). No other significant differences were detected between or within groups ($p > 0.05$).

Table 1. β -Endorphin Levels (pmol/liter) in Alcoholics and Controls before and after α - θ Brainwave Training

Individual values and mean (+ SE) radioimmunoactive β endorphin levels (pmol/liter) in extracted serum samples of alcoholic controls (ALCONT group), nonalcoholic controls (NONALC group), and experimental alcoholics (EXPALC group). Blood samples were taken before initiation of treatment (pre) and at the conclusion of the experiment (post).

Alcoholic controls			Nonalcoholics			Experimental alcoholics		
S	Pre	Post	S	Pre	Post	S	Pre	Post
1	6.3	4.8	11	7.3	7.9	21	4.0	2.8
2	4.0	4.3	12	3.5	3.6	22	5.1	5.9
3	6.9	12.6	13	4.7	5.3	23	8.2	10.9
4			14	7.6	8.5	24	4.9	5.3
5	3.8	5.6	15	5.6	7.1	25	5.0	5.3
6	8.2	11.8	16	3.7	2.7	26	6.0	5.5
7	4.3	5.7	17			27		
8	11.6	12.5	18	4.8	4.6	28	5.0	5.5
9	5.9	6.5	19	2.2	4.3	29	5.6	3.5
10			20	4.3	4.6	30	6.8	7.7
\bar{X}	6.375	7.975		4.844	5.400		5.622	5.822
SE	0.923	1.289		0.588	0.664		0.415	0.787

Electroencephalographic Scores

Fig. 1 shows mean (+SE) percentage of brainwaves in the α frequency range, for the ALCONT, NONALC, and EXPALC groups before (pre) and after (pos) treatment. There were significant main effects of group $F(2,27) = 39.40$, $p < 0.05$, and testing time, $F(1,27) = 110.66$, $p < 0.05$, and a significant interaction of group and time, $F(2,27) = 88.61$, $p < 0.05$. There was a significant, nearly 12-fold increase in α waves in the EXPALC group ($p < 0.05$). The mean posttreatment value of the EXPALC group differed from all other means ($p < 0.05$). The latter means did not differ from each other ($p > 0.05$).

Figure 2 shows mean (+SE) percentage of brainwaves in the θ frequency range, for the ALCONT, NONALC, and EXPALC groups before and after treatment. There were significant main effects of group, $F(2,27) = 16.39$, $p < 0.05$, and testing time $F(1,27) = 23.91$, $p < 0.05$, and a significant interaction of group and time, $F(2,27) = 33.96$, $p < 0.05$. There was a significant, nearly 7-fold increase in theta waves in the EXPALC group ($p < 0.05$). Duncan's test revealed significant differences only between the post-treatment mean of the EXPALC group and all other means ($p < 0.05$).

Figure 3 depicts mean (+SE) amplitude (in μV) of α brainwaves for the ALCONT, NONALC, and EXPALC groups at recording coordinates P301 and P402, before and after treatment. Analysis revealed significant main effects of group, $F(2,24) = 10.25$, $p < 0.05$, and testing time, $F(1,24) = 10.31$, $p < 0.05$, and a significant interaction of group and time, $F(2,24) = 6.00$, $p < 0.05$. At both P301 and P402, only the EXPALC group showed significant increases in α wave amplitude ($p < 0.05$). α amplitudes of the EXPALC group nearly doubled across testings, and differed significantly from that of the ALCONT and NONALC groups at the second testing ($p < 0.05$). No other between- or within-groups comparisons were statistically significant ($p > 0.05$).

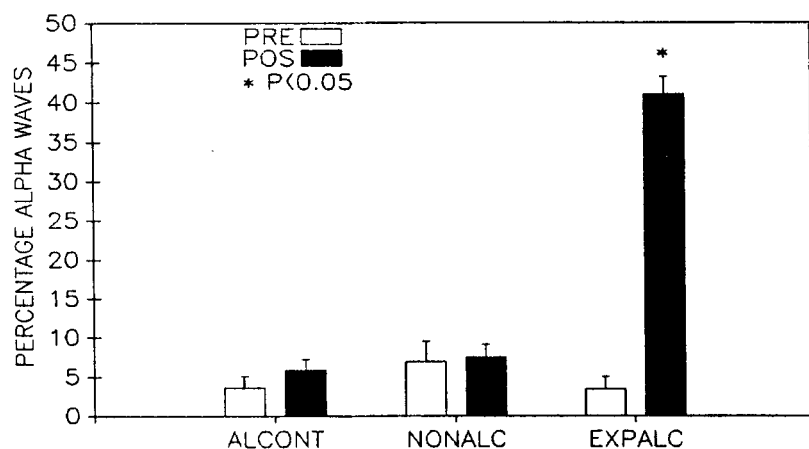


Fig. 1. Mean (+SE) percentage of EEG record in α rhythm frequency range, for the ALCONT, NONALC, and EXPALC groups, before (pre) and after (pos) BWT of the EXPALC group.

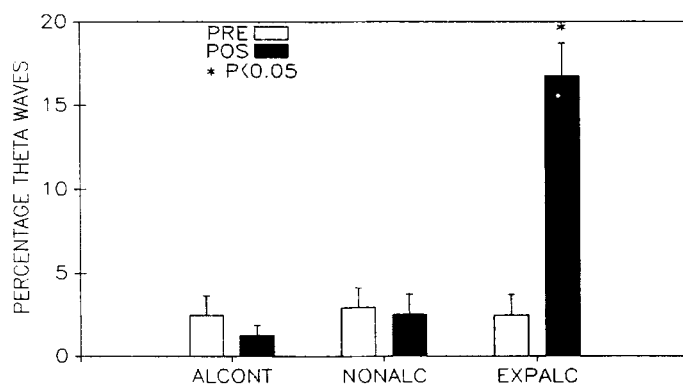


Fig. 2. Mean (+SE) percentage of EEG record in θ rhythm frequency range, for the ALCONT, NONALC, and EXPALC groups before and after BWT of the EXPALC group.

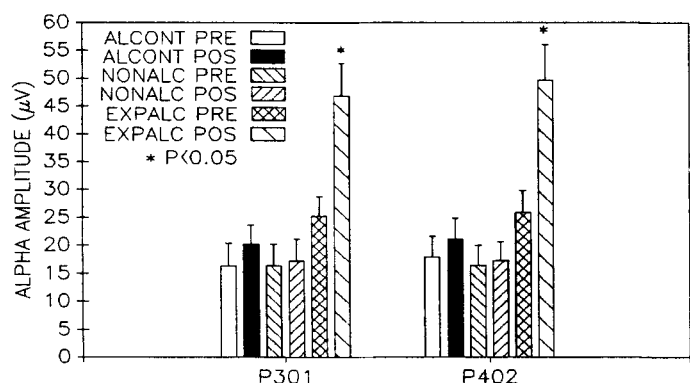


Fig. 3. Mean (+SE) α rhythm amplitude (in μ V) of the ALCONT, NONALC, and EXPALC groups, before and after BWT of the EXPALC group, at recording coordinates P301 and P402.

Figure 4 shows mean (+SE) percentage of brainwaves in the α frequency range, for the EXPALC group during a 5-min baseline period and during a 30-min biofeedback training period, across 15 sessions. There were significant main effects of testing time, $F(1,9) = 606.05$, $p < 0.05$, and session, $F(14,126) = 12.06$, $p < 0.05$, and a significant interaction of time (pre-post) and session, $F(14,126) = 2.25$, $p < 0.05$. There were significant ($p < 0.05$) increases in percentage of brainwave in α after training in all sessions except the seventh. Baseline (pre) scores increased significantly from the first session on sessions 5–15 ($p < 0.05$).

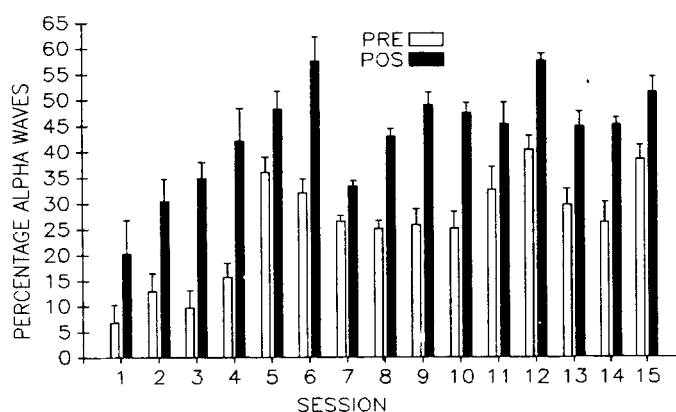


Fig. 4. Mean (+SE) percentage of EEG record in α rhythm frequency range, for the EXPALC group before and after 15 daily BWT sessions.

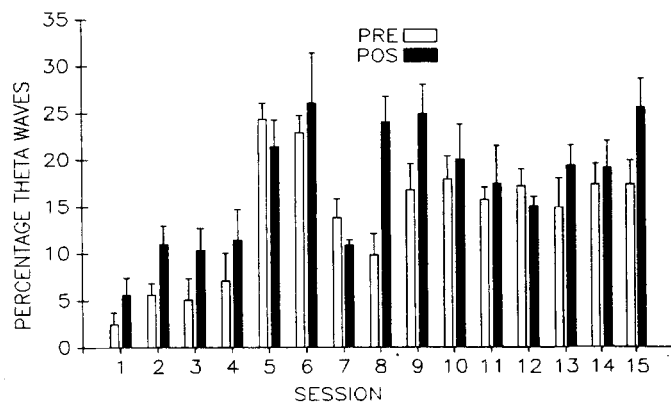


Fig. 5. Mean (+SE) percentage of EEG record in θ rhythm frequency range, for the EXPALC group before and after 15 daily BWT sessions.

Training (post) scores increased significantly from the first session on sessions 2–15 ($p < 0.05$). Baseline α percentage increased nearly 6-fold across sessions 1–15 and training α percentage increased by a factor of about 2.5 across sessions 1–15.

Figure 5 displays mean (+SE) percentage of brainwaves in the θ frequency range, for the EXPALC group at baseline (pre) and after biofeedback training (post), across 15 sessions. There were significant effects of testing time (pre-post), $F(1,9) = 13.94$, $p < 0.05$, and session, $F(14,126) = 8.66$, $p < 0.05$, and a significant interaction of testing

and sessions, $F(14,126) = 2.14$, $p < 0.05$. There were significant increases in θ waves after training only in sessions 8, 9, and 15 ($p < 0.05$). Baseline scores increased significantly from the first session on sessions 5, 6, 7, 9–15 ($p < 0.05$). Training scores increased significantly from the first session on sessions 5, 6, 8–15 ($p < 0.05$). Baseline θ percentage increased 7-fold across sessions 1–15, and training θ percentage increased by a factor of about 4.6 across sessions 1–15.

Depression Scores

Figure 6 depicts mean (+SE) score on Beck's Depression Inventory (BDI) before and after treatment, for the ALCONT, NONALC, and EXPALC groups. Analysis of variance revealed significant effects of group, $F(2,26) = 12.45$, $p < 0.05$, and testing time, $F(1,26) = 205.0$, $p < 0.05$, and a significant interaction of group and time, $F(2,26) = 222.31$, $p < 0.05$. On the pretest, both groups of alcoholic subjects had higher BDI scores than nonalcoholic controls ($p < 0.05$). However, on the post-test, only the alcoholic controls had higher BDI scores than the NONALC group ($p < 0.05$). Only the EXPALC group showed a significant decrease in BDI scores after treatment ($p < 0.05$). In this group, BDI scores were reduced by half, and did not differ from the NONALC group on the posttest ($p > 0.05$).

Follow-up Reports

Table 2 displays the number of reported instances of alcoholic relapse or maintenance of abstinence in the EXPALC and ALCONT subjects across a 13-month follow-up period. χ^2 analysis of these data indicated a significant difference in incidence of alcoholic relapse in the two groups, $\chi^2 = 5.0$, $df = 1$, $p < 0.05$. Of the 10 chronic alcoholics that participated in the EEG α - θ brainwave

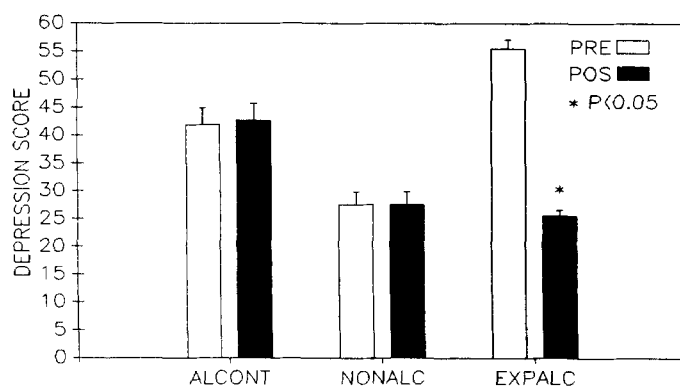


Fig. 6. Mean (+SE) score on Beck's Depressive Inventory, for the ALCONT, NONALC, and EXPALC groups, before and after BWT of the EXPALC group.

Table 2. Reported Instances of Alcoholic Relapse or Continued Abstinence in EXPALC and ALCONT Groups during a 13-Month Follow-up Period

Group	Abstinence	Relapse	Total
EXPALC	8	2	10
ALCONT	2	8	10

training project, only two patients experienced alcoholic relapse after being discharged into their respective communities. In contrast, the alcoholic control patients' displayed eight instances of alcoholic relapse and readmission to VA Medical Centers for alcohol dependence treatment.

DISCUSSION

The results provide clear evidence of the effectiveness of α - θ brainwave training in changing electroencephalographic scores and self-rated depression in alcoholics. Substantial, significant increases of percentages of α and θ brain rhythms and α rhythm amplitude were observed in experimentally treated alcoholics. Depression, as indexed by Beck's Depression Inventory, was significantly reduced to control (nonalcoholic) level after BWT. Time-course analysis of the EEG effects of BWT revealed that increases in α and θ rhythms occurred gradually across the 15 treatment sessions. Baseline α rhythms increased reliably only after 5 sessions, indicating the importance of substantially repeated BWT sessions for the production of durable changes in EEG scores.

In accord with previous studies,^{70,71} there were no significant differences in radioimmunoactive β -endorphin levels between abstinent alcoholic subjects and controls at the beginning of treatment. However, the control group of alcoholics that received traditional medical treatment showed a significant increase in β -endorphin levels at the completion of treatment, relative to their initial levels or nonalcoholic control levels. This unexpected result suggests that the standard medical treatment of alcoholism may induce a state of unrelieved stress. Several reviews have summarized the extensive evidence that an increase in β -endorphin levels is a reliable index of severity of stress in the environment.³⁸⁻⁴² The lack of a corresponding increase in β -endorphin in alcoholics receiving BWT further suggests that the deliberate application of relaxation therapies counteracts the stress of abstinence. For example, BWT may relieve the tension and negative self-efficacy reported to be associated with early stages of abstinence.⁷²⁻⁷⁵

The radioimmunoassay results may provide a neuropeptide-based explanation of the extremely high rate of relapse in alcoholics that receive traditional treatment. Administration of β -endorphin and other opioid peptides has been clearly shown to increase caloric intake in animals and humans.⁷⁶⁻⁷⁸ An extensive literature confirms that alcohol intake is regulated by the same factors that control food intake.⁵⁵ Thus, a neuropeptide that excites energy intake will stimulate ethanol intake, if the caloric consequences of ethanol are familiar. If β -endorphin is elevated in alcoholics, a return to consumption of ethanol calories would be inevitable. Some feature or interaction of relaxation therapies such as autogenic and temperature training, desensitization, and BWT appeared to prevent the relapse to ethanol consumption that may be motivated by elevated endorphinemia.

α - θ brainwave training is also associated with a sharp reduction in self-assessed depression. Results of the BDI are validated by corresponding changes in MCMI and 16 PF scales (Peniston & Kulkosky, unpublished observations). It is not clear whether initial group differences in IQ and/or SES may have contributed to observed depression score differences. No such changes in depression were detected in the traditional alcoholic control group. Relapse of this control group is understandable, in view of the increased blood-indexed stress that is unaccompanied by significant changes in the alcoholic personality. Fundamental changes in blood neurochemistry, brain electrical activity, and personality are produced by BWT, in comparison to the control treatment of alcoholics. However, the mechanism of these therapeutic effects was not addressed in the present experiment. For example, it is possible that some interaction between BWT and the autogenic and temperature training and desensitization sessions contributes to the observed effects. Some feature of the substantially longer exposure of the EXPALC subjects to the relaxation therapies and personnel produced greater relaxation and α and θ brain rhythms than the control alcoholic subjects.

The results of the follow-up study showed that most of the EXPALC patients were maintaining abstinence and preventing alcoholic relapse during this period. Of the two EXPALC subjects that experienced alcoholic relapse over the 13-month follow-up phase, one elected to return to the VA Medical Center for EEG α - θ brainwave "booster" sessions. The informers reported these two EXPALC patients' tolerance for alcohol was significantly reduced, resulting in a psychophysiological reaction of rejection of ethanol exposure. In addition, most (7) of the EXPALC patients have either successfully completed a practical nursing program, or are attending junior college in their communities, or are attending a state training program for certification as alcoholic counselors. These clinical observations lend some support to the hypothesis of these patients undergoing a personality change, as confirmed by BDI results during the experiment. In contrast, the VA medical records indicated that of the 10 ALCONT patients, eight have been readmitted to VAMCS for alcohol dependence treatment and the other two patients were experiencing some uncontrolled drinking episodes during the 13-month follow-up period. These results provided supportive evidence that the EXPALC patients' response to EEG α - θ brainwave training resulted in moderately long-term prevention of alcoholic relapse.

In summary, α - θ brainwave training produces profound increases in α and θ brain rhythms, decreases self-assessed depression, and appears to prevent an elevation of serum β -endorphin levels during the course of treatment of alcoholism. The experimental results and follow-up evidence indicate that this biobehavioral approach to the treatment of chronic alcoholism is a promising alternative to traditional medical treatment of alcoholism. Further

study of the mechanism of the therapeutic effect, such as the contribution of placebo or Hawthorne effects, is clearly warranted.

ACKNOWLEDGMENTS

Special thanks to Fort Lyon VA Medical Center for support of the study and to Carol Hunter, MD, Chief, Psychiatry Service, Lynwood Hoppel, MD, EEG Consultant to Psychiatry Service (116A), and Robert G. Sisneros, EEG Technician for their assistance. Also, special thanks to A. M. Lawrence, MD, PhD, Associate Chief of Staff for Education and Program Director, Endocrinology, and Lidia Kirsteins of Edward Hines, Jr. VA Medical Center, for performing the radioimmunoassay of β -endorphin.

REFERENCES

1. Johansson G, Berglund MJ, Ingvar, DH: EEG abnormalities in chronic alcoholism related to age. *Acta Psychiatr Scand* 65:148-157, 1982
2. Volavka J, Pollock V, Gabrielli WF, Jr, Mednick SA: The EEG in persons at risk for alcoholism, in Galanter M (ed): *Recent Developments in Alcoholism*, vol 3. New York, Plenum, 1985, pp 21-36
3. Pollock VE, Volavka J, Goodwin DW, Mednick SA, Gabrielli WF, Knop J, Schulsinger F: The EEG after alcohol in men at risk for alcoholism. *Arch Gen Psychiatry* 40:857-864, 1983
4. Elmasian R, Neville H, Woods D, Schuckit MA, Bloom F: Event-related brain potentials are different in individuals at high and low risk for developing alcoholism. *Proc Natl Acad Sci USA* 79:7900-7903, 1982
5. Gabrielli WF, Mednick SA, Volavka J, Pollock VE, Schulsinger F, Itil TM: Electroencephalograms in children of alcoholic fathers. *Psychophysiology* 19:404-407, 1982
6. Porjesz B, Begleiter H: Brain dysfunction and alcohol, in Kissin B, Begleiter H (eds): *The Pathogenesis of Alcoholism*. Plenum, 1983, pp 415-483
7. Propping P, Kruger J, Janah, A: Effect of alcohol on genetically determined variants of the normal electroencephalogram. *Psychiatry Res* 2:85-98, 1980
8. Propping P, Kruger J, Mark N: Genetic disposition to alcoholism. An EEG study in alcoholics and their relatives. *Hum Genet* 59:51-59, 1981
9. Funderburk WH: Electroencephalographic studies in chronic alcoholics. *Electroencephal Clin Neurophysiol* 1:369-370, 1949
10. Funkhauser JB, Nagler B, Walker DN: The electroencephalogram of chronic alcoholism. *South Med J* 46:423-428, 1953
11. Vogel F, Schalt E, Kruger J, Propping P, Lihner KF: The electroencephalogram (EEG) as a research tool in human behavior genetics: Psychological examinations in healthy males with various inherited EEG variants. I. Rationale of the study; material; methods; heritability of test parameters. *Hum Genet* 47:1-45, 1979
12. Marlatt, GA: The controlled-drinking controversy: A commentary. *Am Psychol* 38:1097-1110, 1983
13. Moos RH, Finney JW: The expanding scope of alcoholism treatment evaluation. *Am Psychol* 38:1036-1044, 1983
14. Vaillant GE: *The Natural History of Alcoholism: Cause, Patterns, and Paths to Recovery*. Cambridge, MA, Harvard University Press, 1983
15. Hillenberg JB, Collins FL: A procedural analysis and review of relaxation training research. *Behav Res Ther* 20:251-260, 1982
16. King NJ: The therapeutic utility of abbreviated progressive relaxation: A critical review with implications for clinical practice, in Hersen M, Eister RM, Miller PM (eds): *Progress in Behavior Modification*, vol 10. New York, Academic, 1980
17. Passini FT, Watson CB, Dehnel L, Herdee J, Watkins B: Alpha wave biofeedback training therapy in alcoholics. *J Clin Psychol* 33:292-299, 1977
18. Peniston EG, Hughes RB, Kulkosky PJ: EMG biofeedback-as-

- sisted relaxation training in the treatment of reactive depression in chronic pain patients. *Psychol Rec* 36:471-482, 1986
19. Budzynski TH, Stoyva JM: Biofeedback techniques in behavior therapy, in Shapiro D, Barber TX, DiCara LV, Kamiya J, Miller NE, Stoyva J (eds): *Biofeedback and Self-Control*. Chicago, Aldine, 1972, pp 437-459
20. Peniston EG: EMG biofeedback-assisted desensitization treatment for Vietnam combat veterans post-traumatic stress disorders. *Clin Biofeedback health* 9:35-41, 1986
21. Wolpe J: *Psychotherapy by Reciprocal Inhibition*. Stanford, CA, Stanford University, 1958
22. Averill JR: Personal control over aversive stimuli and its relationship to stress. *Psychol Bull* 80:286-303, 1973
23. Higgins RL, Marlatt GA: Fear of interpersonal evaluation as a determinant of alcohol consumption in male social drinkers. *J Abnorm Psychol* 84:644-651, 1975
24. Miller PM, Hersen M, Eisler PM, Hilsman G: Effects of social stress on operant drinking of alcoholics and social drinkers. *Behav Res Ther* 12:67-72, 1974
25. Klajner F, Hartman LM, Sobell MB: Treatment of substance abuse by relaxation training: A review of its rationale, efficacy and mechanisms. *Addict Behav* 9:41-55, 1984
26. Wadden TA, Penrod HJ: Hypnosis in the treatment of alcoholism: A review and appraisal. *Am J Clin Hypnosis* 24:41-47, 1981
27. Watson CG, Herder J: Alpha biofeedback therapy in alcoholics: An 18-month follow-up. *J Clin Psychol* 34:765-769, 1978
28. Wong MR, Brochin NE, Gendron KL: Effects of meditation on anxiety and chemical dependency. *J Drug Educ* 11:91-105, 1981
29. Brown BB: Recognition of aspects of consciousness through association with EEG alpha activity represented by a light signal. *Psychophysiology* 6:442-452, 1970
30. Green EE, Green A: *Beyond Biofeedback*. San Francisco, Delacorte, 1977
31. Kamija J: Operant control of the EEG alpha rhythm and some of its reported effects on consciousness, in Tart CT (ed): *Altered States of Consciousness*. New York, Wiley, 1969, pp 519-529
32. Anand BK, Chhina GS, Singh B: Some aspects of electroencephalographic studies in yogis. *Electroenceph Clin Neurophysiol* 13:452-456, 1961
33. Oliver G: Imagery reported during theta brain rhythm state. Doctoral Dissertation, California School of Professional Psychology, 1974
34. Hall MP: Theta training: Imagery and creativity, in Green EE, Green AM (eds): *Beyond Biofeedback*. San Francisco, Delacorte, 1977, pp 118-152
35. Cicero TJ: Alcohol effects on the endocrine system. *NIAAA Alcohol and Health Monograph* 2:53-91, 1982
36. Pohorecky LA, Brick J: Pharmacology of ethanol. *Pharmac Ther* 36:335-427, 1988
37. Triana E, Frances RJ, Stokes PE: The relationship between endorphins and alcohol-induced subcortical activity. *Am J Psychiatry* 137:491-493, 1980
38. Akil H, Watson CG, Young E, Lewis ME, Khachaturian H, Walter JM: Endogenous opioids: Biology and function. *Ann Rev Neurosci* 7:223-255, 1984
39. Amir S, Brown ZW, Amit Z: The role of endorphins in stress: Evidence and speculations. *Neurosci Biobehav Rev* 4:77-91, 1980
40. Bolles RC, Fanselow MS: Endorphins and behavior. *Ann Rev Psychol* 33:87-101, 1982
41. Naber D, Bullinger M, Zahn T: Stress effects of beta-endorphin in human plasma: Relationships to psychophysiological and psychological variables. *Psychopharmacol Bull* 17:187-189, 1981
42. Riley AL, Zellner DA, Duncan HJ: The role of endorphins in animal learning and behavior. *Neurosci Biobehav Rev* 4:69-76, 1980
43. Dent RRM, Guilleminault C, Albert LH, Posner BI, Cox BM, Goldstein A: Diurnal rhythm of plasma immunoreactive β -endorphin and its relationship to sleep stages and plasma rhythms of cortisol and prolactin. *J Clin Endocrinol Metab* 52:942-947, 1981
44. Kalin NH, Loevinger BL: The central and peripheral opioid peptides. *Psychiatric Clin North Am* 6:415-428, 1983
45. Luttinger D, Frye GD, Nemeroff CB, Prange AJ, Jr: The effects of neurotensin, β -endorphin, and bombesin on ethanol-induced behaviors in mice. *Psychopharmacology* 79:357-363, 1983
46. Morrow EL, Erwin VG: Calcium influence on neurotensin and β -endorphin enhancement of ethanol sensitivity in selectively bred mouse lines. *Alcohol Drug Res* 7:225-232, 1987
47. Dave JR, Karanian JW, Eskay RL: Chronic ethanol treatment decreases specific nonopioid β -endorphin binding to hepatic and kidney membranes and lowers plasma β -endorphin in the rat. *Alcohol Clin Exp Res* 10:161-166, 1986
48. Gambert SR, Pontzer CH, Barboriak JJ: Effect of ethanol consumption on central nervous system (CNS) β -endorphin and ACTH. *Horm Metab Res* 13:242-243, 1981
49. Seizinger BR, Bovermann K, Maysinger D, Holtt V, Herz A: Differential effects of acute and chronic ethanol treatment on particular opioid peptide systems in discrete regions of rat brain and pituitary. *Pharmacol Biochem Behav* 13 (S1):361-369, 1983
50. Gianoulakis C, Barcomb A: Effect of acute ethanol in vivo and in vitro on the β -endorphin in the rat. *Life Sci* 40:19-28, 1987
51. Keith LD, Crabbe JC, Robertson LM, Kendall JW: Ethanol-stimulated endorphin and corticotropin secretion in vitro. *Brain Res* 367:222-229, 1986
52. Blum K, Topel H: Opioid peptides and alcoholism: Genetic deficiency and chemical management. *Funct Neurol* 1:71-83, 1986
53. Genazzani AR, Nappi G, Facchinetti F, Mazzella GL, Parrini D, Sinforiani E, Petraglia F, Savoldi F: Central deficiency of β -endorphin in alcohol addicts. *J Clin Endo Metab* 55:583-586, 1982
54. Topel H: β -Endorphin genetics in the etiology of alcoholism. *Alcohol* 5:159-165, 1988
55. Kulkosky PJ: Brain-gut neuropeptides and the limitation of ethanol consumption. *Neurosci Biobehav Rev* 9:179-190, 1985
56. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*. Washington, DC, 1987
57. Hollingshead BA: *Two Factor Index of Social Position*. New Haven: Wiley, 1957
58. Hollingshead BA, Redlich FC: *Social Class and Mental Illness*. New York: Wiley, 1958
59. Myers J, Bean B: *A Decade Later, A Follow-up of Social Class and Mental Illness*. New York: Wiley, 1968
60. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J: An inventory for measuring depression. *Arch Gen Psychiat* 4:561-571, 1961
61. Dorus W, Kennedy J, Gibbons RD, Ravi SD: Symptoms and diagnosis of depression in alcoholics. *Alcohol Clin Exp Res* 11:150-154, 1987
62. Hengeveld MW, Ancion FA, Rooifmans HG: Prevalence and recognition of depressive disorders in general medical inpatients. *Int J Psychiatry Med* 17:341-349, 1987
63. Steer RA, Beck AT, Shaw BF: Depressive symptoms differentiating between heroin addicts and alcoholics. *Drug Alcohol Depend* 15:145-150, 1985
64. Steer RA, McElroy MG, Beck AT: Correlates of self-reported and clinically assessed depression in outpatient alcoholics. *J Clin Psychol* 39:144-149, 1983
65. Naber D, Cohen RM, Pickar D, Kalin NH, Davis G, Pert CB, Bunney WE, Jr: Episodic secretion of opioid activity in human plasma and monkey CSF: evidence for a diurnal rhythm. *Life Sci* 28:931-935, 1981
66. Boeringa JA: Rapid treatment of a blood phobia. *VA Practitioner* 3:66-71, 1986
67. Farrell PA, Gates WK, Maksud MG, Morgan WP: Increase in plasma β -endorphin/ β -lipotropin immunoreactivity after treadmill running in humans. *J Appl Physiol* 52:1245-1249, 1982

68. Fraioli F, Moretti C, Paolucci D, Alicicco E, Crescenzi F, Fortunio G: Physical exercise stimulates marked concomitant release of β -endorphin and adrenocorticotrophic hormone (ACTH) in peripheral blood in man. *Experientia* 36:987-989, 1980
69. Jasper HH: The 10-20 electrode system of the International Federation. *EEG Clin Neurophysiol* 10:371-375, 1958
70. Barret L, Bourhis F, Buffet H, Danel V, Debru JL: Determination of β -endorphin in alcoholic patients in the acute stage of intoxication: Relation with naloxone therapy. *Drug Alcohol Depend* 19:71-78, 1987
71. Brambilla F, Zarattini F, Gianelli A, Bianchi M, Panerai A: Plasma opioids in alcoholics after acute alcohol consumption and withdrawal. *Acta Psychiatr Scand* 77:63-66, 1988
72. Marlatt GA, Gordon JR: Determinants of relapse: Implications for the maintenance of behavior change, in Davidson P (ed): *Behavioral Medicine: Changing Health Lifestyles*. New York, Brunner/Mazel, 1979, pp 410-452
73. Rollnick S, Heather N: The application of Bandura's self-efficacy theory to abstinence-oriented alcoholism treatment. *Addict Behav* 7:243-250, 1982
74. Hoy RM: The personality of inpatient alcoholics in relation to group psychotherapy, as measured by the 16-PF. *Q J Stud Alcohol* 30:401-407, 1969
75. White WF: Personality and cognitive learning among alcoholics with different intervals of sobriety. *Psychol Rep* 16:1125-1140, 1965
76. Baile CA, McLaughlin CL, Della-Fera MA: Role of cholecystokinin and opioid peptides in control of food intake. *Physiol Rev* 66:172-233, 1986
77. Morley JE, Levine AS, Yim GK, Lowy MT: Opioid modulation of appetite. *Neurosci Biobehav Rev* 7:281-305, 1983
78. Reid LD: Endogenous opioid peptides and regulation of drinking and feeding. *Am J Clin Nutr* 42:1099-1132, 1985