Biofeedback and Self-Regulation, Vol. 12, No. 2, 1987

Task-Related Stress and EEG Alpha Biofeedback[1]

Paul D. Tyson[2]
Brock University

Establishing a contingency between stress and a physiological response is essential in biofeedback. The sensitivity of high alpha to contingent stress was investigated by manipulating conditions known to influence stress, such as the distribution, predictability, and controllability of stressful stimuli, and number of tasks performed. Forty subjects were divided into stress and nonstress groups. Within each group, one-half had the dual-task of anticipating and increasing alpha activity. The other half was initially instructed to only anticipate alpha and, later, had the dual task of anticipating and controlling alpha. No feedback training was included to distribute the task-related stressor and allowed the assessment of self-control. All of the stress manipulations significantly influenced the effects of stress on alpha production. The dual-task subjects produced less alpha and less self-control than did training with control phased in after subjects learned to anticipate alpha. Without stress, phased-in control produced highly significant increases in alpha production and self-control without feedback. The use of an alpha-contingent feedback paradigm and anticipation training was related to the therapeutic applications of alpha feedback to stress and anxiety.

Descriptor Key Words: biofeedback; EEG alpha; task-related stress; noise stress; anticipation training; self-control.

The application of alpha biofeedback to stress and anxiety (Hardt & Kamiya, 1978) became controversial when research began to question whether

[1]The author is grateful to Dr. Robert Ogilvie, Dr. Peter Ramm, and the journal reviewers for their helpful comments on an earlier draft of this paper.
[2]Address all correspondence to Dr. Paul D. Tyson, Department of Psychology, Brock University, St. Catharines, Ontario, Canada L2S 3A1.

0363-3586/87/0600-0105$05.00/0

alpha activity is a functional physiological correlate of perceived stress and anxiety (Plotkin, 1979). The present hypothesis is that high alpha activity has a clear relationship to stress when stress and alpha are contingent. Establishing a contingency between stress and a physiological response is essential for biofeedback, counterconditioning, and relaxation techniques (Burchfield, 1985). Task-related stress occurs when stress is contingent on your performance and is unique because the stress is self-produced. In a sense, all perceived stress is self-produced. Similar to the alpha-contingent paradigm (Mulholland & Eberlin, 1977) used to measure the effects of visual stimulation on alpha, Tyson (1982) introduced a contingency between a stressful noise and the alpha response. The task-related stress was made contingent on alpha activity by using a mildly stressful sound for feedback, with the volume of the stressor increasing as the subject increased alpha activity. Subjects given biofeedback, with massed trials of task-related stress, showed a significant reduction in their ability to increase alpha compared to subjects given feedback without stress.

Many researchers have found that below-baseline levels of alpha are relatively insensitive to noncontingent stress (Plotkin & Rice, 1981), and Tyson's (1982) research confirmed their results. To ensure that the stress effects were not caused by the background, noncontingent effects of stressful noise (Kryter, 1985), two stimulus control groups listened to the same modulated sounds as the feedback groups, and neither control group changed from baseline alpha over the session (Tyson, 1982). Baseline levels of alpha do not respond to stress, but the ability to increase alpha is influenced by contingent stress. The present question is how sensitive high alpha activity is to the manipulation of stress.

The present experiment was designed to manipulate alpha production using conditions known to have effects on stress. It has been reported that the detrimental effects of stress on task performance are reduced when stress is distributed, predictable, and controllable, and the tasks are simple (Lazarus & Folkman, 1984). However, the specific effects of unpleasant or threatening noise, as a task-related stressor, have been difficult to identify because researchers have not distinguished between background and task-related stress (Broadbent, 1978; Poulton, 1979; Kryter, 1985). This is an important distinction because many occupations, such as using a jackhammer, may have task-related stress. Task-related stress produced a U-shaped performance curve with massed alpha feedback trials. On the early trials, subjects produced more alpha and then performance decreased rapidly and continued until alpha was below baseline (Tyson, 1982). Hypothetically, declining alpha production was produced by the accumulation of mild stress (Cohen, 1980). In the present experiment, stress was distributed by interspacing no-stress trials, which should flatten the U-shaped curve by reducing the accumulation of stress and increasing processing capacity (London & Schwartz, 1984).

The final phase of the U-shaped performance curve, the recovery of alpha production, may have resulted from the stressful stimulus becoming more predictable and controllable (Glass & Singer, 1972). To evaluate this hypothesis, subjects in the present experiment were asked to predict or anticipate fluctuations in alpha activity. Anticipation training has not been incorporated into biofeedback applications, which is surprising considering its importance in biofeedback self-control theories (Epstein & Blanchard, 1977; Mulholland, 1984). In addition, Lazarus's (1977) analysis of stress reverses the usual wisdom that coping always follows stress, or is caused by it, and suggests that coping can precede stress when anticipated and can influence its form and intensity. The effects of anticipation training on alpha production should, initially, contribute to declining performance because the process of learning to predict can inhibit alpha activity (Tyson & Audette, 1979). Once a strategy is well practiced and requires less effort or capacity, predicting alpha activity should facilitate alpha production and reduce stress by increasing its predictability.

Control and predictability are closely interrelated; events cannot be controlled that are not predictable, but events can be predicted that are not controllable (Weinberg & Levine, 1980). In the present experiment, one group had the dual task of anticipating and increasing alpha activity, whereas another group was initially instructed to anticipate fluctuations in alpha without attempting to control or manipulate alpha activity and later had the dual task of anticipating and controlling alpha. Logically, it was impossible to have a group instructed to increase alpha without attempting to predict or anticipate alpha activity.

Background noise can have a positive effect, no effect, or a negative effect on dual-task performance, depending on the complexity of the tasks, auditory masking, and perceived stress attributed to the sound (Kryter, 1985). Under conditions perceived as stressful, complex simultaneous tasks usually reduce performance (Baum, Singer, & Baum, 1981). Therefore a single task, with control phased in after anticipation training, should facilitate alpha production compared with having a dual task from the beginning of the session.

With biofeedback, the assessment of self-control involves several methodological considerations, such as measuring control in the absence of feedback (Epstein & Blanchard, 1977). Peper and Mulholland (1970) demonstrated that alpha control can be maintained after training without external feedback, and it was found that alpha control diminished only slightly from feedback levels, 10 minutes after (Travis, Kondo, & Knott, 1974) and 25 minutes after training (Kondo, Travis, & Knott, 1975). In addition, subjects' ability to alter their brain waves before training must be differentiated from their ability after feedback training. Two studies included a pretraining no-feedback task condition but did not make the appropriate comparison and

omitted the pretraining data (Hord & Barber, 1971; Black, Cott, & Pavloski, 1977). More recent research has demonstrated that subjects can decrease alpha activity below pretraining levels without feedback but cannot significantly increase alpha activity during training or self-control trials (Cott, Pavloski, & Black, 1981; Cott, Pavloski, & Goldman, 1981). To assess self-control, the present experiment included identical pre and post no-feedback task conditions and no-feedback self-control trials interspaced during training. The no-feedback trials also distributed the task-related stressor. Alpha production should be manipulated by conditions known to influence stress, such as the distribution, predictability, and controllability of the stressful stimuli and number of tasks to be performed. The task-related stress feedback paradigm is consistent with behavioral approaches that emphasize the extinction of the stress response by learning an incompatible contingent physiological response (Burchfield, 1985). Alpha biofeedback as a rational therapy for stress requires, first, the establishment of a contingency and, second, a clear relationship between the manipulation of stress and the ability to increase alpha activity.

METHOD

Subjects

Forty undergraduates from Brock University received credit to satisfy partially the requirements of an introductory psychology course. The 24 female and 16 male subjects, all of whom indicated no prior biofeedback experience, were randomly assigned to a 2 × 2 factorial design. One additional subject replaced a person who fell asleep during the session.

Apparatus

The EEG, recorded from the right parietal lobe (P4) and right mastoid (A2), was filtered for alpha (8–13 Hz) by a Narco biofeedback system. The parietal derivation was selected because the degree of sensory-motor and intermodality interaction at this site is greater than at the occipital site (Graybiel, 1974; Critchley, 1953). The Narco audio module was modified to accept a taped auditory stimulus, which was intensity-modulated; the feedback threshold was set at 10 uV alpha, and the loudness was increased up to 65 dB as the filtered alpha increased up to 75 uV. The two auditory stimuli used for feedback were produced by synchronizing three frequencies using a Moog synthesizer, and the power spectra of the two auditory stimuli have been previously reported (Tyson, 1982). The raw and filtered EEG channels were recorded on a Nihon-Kohden (ME-175E) EEG machine and 8-channel Vetter FM

tape recorder, with flutter compensator, for the computer analysis. The raw P4-A2 EEG channel was first passed through active band-pass filters between 1 and 40 Hz and decomposed into frequency components by power spectral analysis (FFT). The raw EEG was transformed using a modified Bruker TI/II program that allowed magnitude integration within two alpha frequency bands (8–10.5 Hz and 10.5–13 Hz) and calculation of the mean integrated amplitude in microvolts (Tyson, 1982; Tyson, Ogilvie, & Hunt, 1984). The absolute spectral power was transformed at a sample rate twice the highest frequency (34 Hz), in consecutive 30-sec epochs, and was calibrated into microvolts with a 50-uV, 10-Hz sine wave recorded at the beginning of each session.

Design

Subjects were divided randomly into four groups of a 2 × 2 factorial design between groups. Half of the subjects received contingent task-related stress. The other half received a nonstressful auditory stimulus for feedback. The second factor was the number of tasks; subjects began training either with a single task (task 1) of anticipating alpha or with a dual task (task 2) of anticipating and increasing alpha. For the last three trials of the session, all subjects had the dual task to measure any transfer of training. In addition, the design included two repeated-measures variables, the presence or absence of feedback, and trials.

The dependent variables were the integrated alpha amplitude and the within-subject standardized (W-SS) alpha amplitude in two frequency bands (8–10.5 Hz and 10.5–13 Hz). W-SS procedures were used to transform alpha amplitude into standard deviation units relative to the individual's mean and estimate of normal response variability (Tyson, 1987). Instead of using one baseline, such as eyes open, the normal variability of alpha was estimated by using five conditions known to influence alpha activity. Research has shown that W-SS scores, calculated using the *z*-score formula, are more sensitive than alpha amplitude measures to individual differences and subjective correlates of alpha activity (Tyson et al., 1984). The subject's mean and standard deviation, in this experiment, were estimated from 28 30-sec samples of EEG alpha across five conditions designed to maximize the variability of alpha activity. There were two conditions to measure the effects of visual stimulation (eyes open and closed) on alpha activity, two auditory conditions (since sound was used for feedback and one sound was a mild stressor), and, finally, a task condition where the person was attempting to increase alpha without feedback. On each trial, alpha activity was expressed as an average alpha amplitude, W-SS alpha amplitude, and, finally, the difference in al-

pha amplitude from the pretraining no-feedback task condition used to estimate self-control.

Procedure

While the subjects were being prepared for recording, the experimenter explained each step in the placement of electrodes and experimental procedures to alleviate any anxiety about the situation. After the electrodes were attached, the subjects were seated in the experimental room and reminded that the experimenter could see them and could hear them when they spoke through the intercom. The instructions, baselines, and trials for each group were prerecorded to ensure consistency between subjects. The subjects had their eyes open (EO) for the complete session, with the exception of the baseline eyes-closed (EC) condition. Each subject, tested individually, had a session that consisted of (1) instructions, (2) two counterbalanced 3-min EC and EO conditions, (3) two counterbalanced 3-min auditory stimulus conditions, (4) a 5-min no-feedback task condition, (5) five 5-min feedback trials each followed by a 3-min no-feedback trial, (6) a questionnaire and instructions, (7) three more 5-min feedback trials each followed by a 3-min no-feedback trial, except the last, which was 5 min, and (8) a questionnaire. Half of the subjects were given eyes-open feedback using an intensity modulated auditory stimulus composed of sine waves that had been previously judged as pleasant and not stressful. The other half listened to an auditory feedback stimulus composed of sawtooth waves that had been judged mildly stressful and interfered with alpha training (Tyson, 1982). Within each group, one-half of the subjects had the single task of anticipating alpha increases and decreases by moving a lever to the right and left without trying to control, manipulate, or interfere with alpha activity. The other half was asked to anticipate alpha and, at the same time, increase the loudness of the feedback sound by increasing alpha activity. During the last three feedback and three no-feedback trials, all groups had the dual task of anticipating and producing more alpha activity.

After five feedback and no-feedback trials, the subjects were asked to fill out a questionnaire that asked them how they anticipated when the tone was going to get louder, describe how they anticipated when the tone was going to get softer, when they noticed how to anticipate the changes, and if they noticed any changes in their muscles, eyes, breathing, feelings, or thoughts that influenced the tone. After the last three trials, when all subjects were trying to increase alpha production, subjects were asked how they increased the loudness of the tone, whether they tried to control the increases and decreases of the tone by manipulating anything such as their muscles, eyes,

breathing, thoughts, etc., during the five trials before the break, and the same question for after the break. Most of these questions were designed to find out how well subjects, given the task of anticipating alpha without attempting to control or manipulate, in fact complied with the instructions. My present research assistant (M.L.V.), unfamiliar with the design or hypotheses in this experiment, coded the questionnaires, which were rated for control on a 6-point scale by the experimenter, who was blind to group membership.

RESULTS

In general, task-related stress reduced the amount of alpha production. All of the manipulations, known to have effects on stress, also had significant effects on alpha production. As predicted, the manipulations attenuated the U-shapped performance curve previously found when stress trials were massed. Subjects with the dual task produced less alpha and learned less self-control than the subjects with control phased in after anticipation training. However, the manipulations did not completely alleviate the effects of stress when compared with the groups without stress.

Although anticipation training and distributing trials flattened the U-shaped effects of massed task-related stress, the effects of contingent stress significantly decreased alpha production compared with the groups given a nonstressful feedback sound. Figure 1 shows the significant main effect of stress on the higher frequencies of alpha (HFA: $F(1, 36) = 8.27$, $p < .01$),

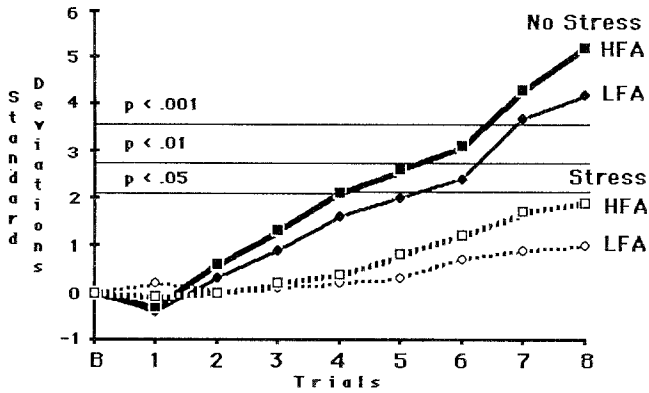

Fig. 1. Degree of alpha production standardized (W-SS) relative to the variability across five baselines for the higher frequencies of alpha (HFA) and lower frequencies of alpha (LFA) in groups with and without task-related stress.

lower frequencies of alpha (LFA: $F(1, 36) = 7.91, p < .01$), and interaction of stress over trials (HFA: $F(7, 252) = 4.69, p < .001$; LFA: $F(7, 252) = 8.01, p < .001$) using within-subject standardized (W-SS) alpha. The stress groups increased alpha production, but it approached the .05 level only after 8 feedback and 8 no-feedback trials. The groups without stress reached the .05 HFA level after 4 feedback and no-feedback trials, and continued to improve ($p < .001$) by the end of training. The slopes of the learning curves in Figures 1 and 2 were exaggerated by plotting feedback and no-feedback trials together; the subjects actually received 16 trials by the end of training. Some of the effects of stress can also be seen using average alpha amplitude as a dependent variable. There was a significant stress by trials interaction in both frequencies using average alpha amplitude (HFA: $F(7, 252) = 2.11$, $p < .05$; LFA: $F(7, 252) = 10.17, p < .001$) or the difference in amplitude from the no-feedback task baseline. The difference scores have the same significant interactions once all of the main effects have been removed by analysis of variance. On the other hand, the main effect of stress, using alpha amplitude or difference scores, was not significantly different in HFA, and the main effect in LFA was significant only for the difference score (LFA: $F(1, 36) = 5.55, p < .025$). Even with the manipulations, the effects of task-related stress were not completely alleviated by interspacing no stress trials and anticipation training when compared with no-stress conditions.

All of the experimental manipulations, known to have effects on stress, also influenced alpha amplitude, as shown by significant four-way interactions involving stress, tasks, feedback, and trials (HFA: $F(7, 252) = 2.05$, $p < .05$; LFA: $F(7, 252) = 5.37, p < .001$) and the same significant interactions using the difference from the no-feedback task baseline. The difference scores were used as the ordinate in Figure 2. The W-SS scores were less sensitive to higher-order interactions, had smaller F ratios for the four-way interactions, and were significant only in the LFA range (LFA: $F(7, 252) = 2.98, p < .001$). There also were significant interactions between single and dual tasks over trials (tasks $\times$ trials) in both frequencies of alpha and, within the lower alpha frequencies, significant interactions between feedback and no feedback over trials (feedback $\times$ trials) and stress with feedback over trials (stress $\times$ feedback $\times$ trials). These significant interactions will be described and illustrated with the four-way interactions in Figure 2.

Beginning with the group closest in task to Tyson's (1982) U-shaped performance curve, Figure 2A and B shows the effects of stress on the group given the dual task (FB task 2) of anticipating and increasing alpha production. This stress group, in general, had the lowest levels of alpha production in both alpha frequencies, but, as predicted, the U-shaped curve was attenuated. On the first trial (Figure 2A), the LFA increased more than that of any other group to 3.5 uV above baseline, then dropped to about 1.8 uV

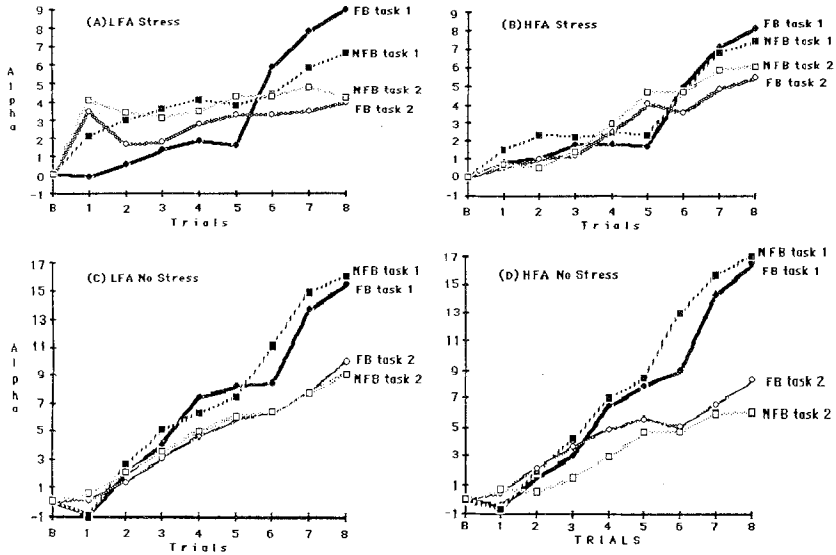

Fig. 2. Changes in the lower frequencies of alpha (LFA) and the higher frequencies of alpha (HFA) amplitude from the no-feedback task baseline for groups with (A and B) and without (C and D) task-related stress. One group had control phased in after anticipation training (task 1) and another group had the dual task (task 2) from the beginning. Within each session, subjects had eight feedback (FB) trials each followed by a no-feedback (NFB) trial.

for two trials, and finally gradually increased the LFA until it reached 4 uV above baseline after eight feedback and no-feedback trials. The gradual increase in HFA (Figure 2B), within the dual-task (FB task 2) group, showed no remnant of the U-shaped curve. Tyson (1982), using the same stress and EEG analysis, also found that the major U-shaped stress effects were within the lower alpha frequencies. For comparison, in the previous study the stress group, on the first trial, increased LFA about 4 uV above baseline. Performance then dropped for the next three trials until LFA was below baseline and finally returned to the first trial level of alpha production for the last two trials. Distributing trials and anticipation training flattened the U-shaped curve, but the dual task interfered with learning to produce more alpha activity and with contingent stress the dual task 2 produced the lowest levels of LFA and HFA by the end of training.

The other stress group (FB task 1) had alpha production or control phased in after anticipation training. The single-task 1 subjects were initially instructed not to attempt to influence, manipulate, or control alpha, and it was expected that alpha production would stabilize after a few trials. In Figure 2A the stress group began below the LFA baseline and then stabi-

lized around 1.6 uV on trials 3, 4, and 5. Between trials 5 and 6, the single-task 1 group was instructed to produce more alpha as well as anticipating alpha, the same dual task the task 2 stress group had from the beginning of training. After phasing in control, this group increased LFA production up to 9 uV above baseline by the end of training, compared with 4 uV with the dual task. The results were very similar when the higher frequencies of alpha were examined (Figure 2B). Alpha amplitude had significant tasks × trials interactions (HFA: $F(7, 252) = 2.64, p < .01$; LFA: $F(7, 252) = 6.13$, $p < .001$) and the W-SS transform had smaller F ratios, which reached significance in the LFA range (LFA: $F(7, 252) = 2.57, p < .025$). Anticipation training with phased-in control (FB task 1) definitely increased the average alpha amplitude produced with task-related stress and also had the highest level of performance without stress (Figure 2C and D).

It was expected that if stress was absent during the no-feedback trials, alpha production would be greater than the feedback trials, with the same task and contingent stress. In general, trials without the stressful feedback stimulus had higher levels of both the LFA and HFA amplitude, but the feedback × trials interaction was significant only in the lower alpha frequencies (LFA: $F(7, 252) = 3.16, p < .01$). The very noticeable exceptions in Figure 2A and B were in the task 1 groups during the last three dual-task trials. When control was phased in, after anticipating the stressor, alpha production was greater with the stressful feedback stimulus present and helped to make the stress × feedback × trials interaction significant (LFA: $F(7, 252) = 3.83, p < .001$).

Alpha production in the no-feedback self-control trials paralleled the feedback trials in all groups (Figure 2). Comparing only the baseline to the end of training, there was a significant increase in alpha amplitude from the no-feedback task baseline to no-feedback trial 8 (LFA: $F(1, 36) = 46.38$, $p < .001$; HFA: $F(1, 36) = 28.48, p < .001$). Figure 3 also shows a significant interaction in the LFA between stress and the two measures of self-control at the beginning and end of the session (LFA: $F(1, 36) = 7.33$, $p < .025$). Subjects were instructed to use their internal cues to anticipate alpha changes, particularly during no-feedback training. Consistent with self-control models, making alpha predictable before trying to control alpha resulted in the highest level of alpha production and the greatest degree of self-control using internal cues to produce more alpha activity. Examining the group with the best performance, was of interest in this experiment, because alpha production was generally higher without the feedback sound, which may suggest that the choice of feedback sound in this experiment was not the best possible for facilitating alpha production.

By the end of training, as expected, the groups with tasks phased in were superior to the dual-task groups. But surprisingly, in the early trials the

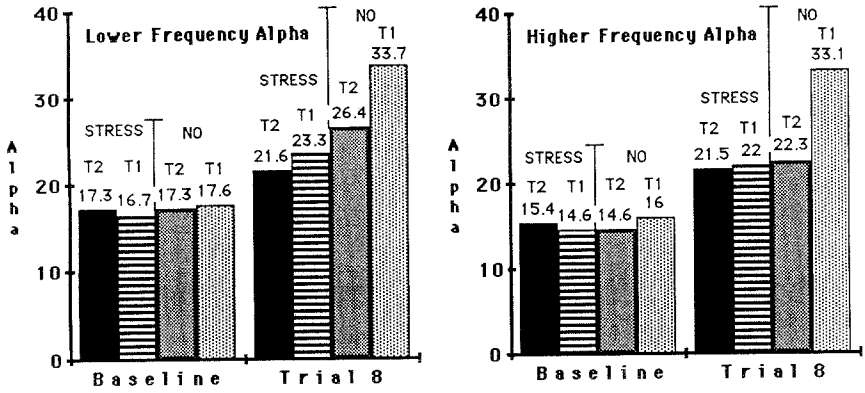

Fig. 3. Average alpha amplitude in microvolts for the no-feedback task baseline and the last no-feedback self-control trial (trial 8) with the task-related stress group and no-stress group. One-half of the subjects within each group had control phased in after anticipation training (T1) and the other half had the dual task (T2) from the beginning. The black bars have stress with the dual task, and the adjacent stripped bars have stress with control phased in. No-stress groups have the last two bars on the right for baseline and trial 8 with the dual task (T2) and phased-in task (T1).

nonstress group (FB task 1), instructed not to increase alpha, actually increased alpha production more than the FB task 2 group instructed to produce more alpha (Figure 2C and D). Either the subjects ignored the instructions not to control alpha during the first five trials or, without stress, learning to anticipate alpha actually increased alpha production, even when the subjects were not actively attempting to produce more alpha. After the first five feedback and no-feedback trials, and before the production instructions, all groups were given a questionnaire that asked how they anticipated alpha increases and decreases, when they discovered how to do the task, and whether they noticed any changes in their muscles, eyes, breathing, feelings, or thoughts that influenced the tone. At the end of the session subjects were specifically asked whether they tried to control anything during the first five trials. The ratings of the questionnaires for control, by the experimenter blind to group membership, found a highly significant main effect of task instructions ($F(1, 36) = 129.5, p < .001$), a significant difference between the stress and no-stress groups ($F(1, 36) = 13.37, p < .001$), and a nonsignificant interaction ($F(1, 36) = 2.64, p = .113$).

Although instructed not to control alpha, the single-task 1 subjects reported infrequent attempts to control alpha production, but the ratings of attempted control were considerably less than the groups instructed to increase alpha activity. The groups with task-related stress attempted more control strategies than the groups without stress. On a 6-point rating scale,

ranging from no attempted control (0) to continuous attempts to control, beginning during trials 1 or 2, and using multiple strategies (5), the single-task 1 group, without stress and with no manipulation instructions, averaged .9 on the scale. Over half of the nonstress-task 1 subjects attempted to control at least once, and one subject attempted two strategies on trials 4 and 5. The task 1 stress group attempted control more frequently (M = 2.2), although they were also instructed not to manipulate alpha. The groups instructed to produce more alpha, of course, attempted more control strategies earlier and more frequently, and the dual-task 2 stress group had higher control ratings (M = 4.6) than the group without stress (M = 4.1). The amount of attempted control in the task 1 nonstress group was too small to account for the rapid increases in alpha production during the first five trials. In addition, if the groups instructed not to control had totally ignored the instructions, then they should have been the same as the dual-task groups; somehow, anticipating alpha with minimal control increased alpha production.

In summary, the results of these manipulations suggest a functional relationship between task-related stress and EEG alpha production. Clearly, contingent stress reduced the amount of alpha production compared with groups without stress. Biofeedback and the manipulations, although important, were not sufficient to completely alleviate the effects of stress on performance. The nonstress group with phased-in control, after anticipation training, produced alpha increases 6 standard deviations above W-SS HFA and 5.5 above the W-SS LFA average amplitude. With or without stress, anticipation training, with phased-in control, produced higher levels of alpha production and greater self-control when compared with having the dual task from the beginning of training.

DISCUSSION

This experiment supports the hypothesis that high-amplitude alpha is sensitive to the manipulation of contingent stress and emphasizes the importance of establishing a clear contingency when applying biofeedback to stress. Coping with task-related noise is a major problem in many occupations, especially when people cope with contingent stress by becoming less productive or by taking frequent breaks from the stressful noise. The present experiment demonstrates the importance of distribution, predictability, controllability, and phased-in tasks for increasing alpha activity and reducing stress. Distributing the stressful stimulus reduces the accumulation of stress found when trials are massed and facilitates alpha production (Tyson, 1982). During the interspaced no-stress trials, subjects are predicting alpha fluctu-

ations using internal cues as training in self-control. Anticipation training with phased-in control facilitates alpha production, even with contingent stress. Without stress, learning to anticipate alpha and then phasing in control produced highly significant increases in alpha production and self-control.

A number of researchers have found that baseline levels of alpha are relatively insensitive to perceived stress (Plotkin & Rice, 1981) and have questioned applications of alpha biofeedback to anxiety and stress (Hardt & Kamiya, 1978). The present experiment shows how these apparently contradictory positions, in fact, complement each other. Any therapeutic benefits of below-baseline alpha feedback are mainly due to subjects' expectations, demand characteristics, and many other placebo effects. However, Plotkin (1979) asserts that it is impossible to significantly increase alpha above eyes-closed baselines. This experiment and previous research (Hardt & Kamiya, 1978; Tyson & Audette, 1979; Tyson, 1982), using different conditions, found that subjects are able to increase alpha significantly above eyes-closed baselines, above no-feedback task baselines, and above within-subject standardized (W-SS) average alpha amplitudes. The ability to increase alpha production above baseline, particularly in the lower frequencies, is reduced by contingent stress. However, baseline levels of alpha activity are not influenced by the presence of background stress (Tyson, 1982) and, in this experiment, are not significantly different from task baselines or W-SS alpha levels. The effects of these stress manipulations on alpha production can only be seen when compared with groups significantly increasing alpha activity. Plotkin's (1979) placebo manipulations would probably influence both high alpha production and stress if they were compared with groups significantly increasing alpha activity.

In addition to training procedures, the effects of the stress manipulations on alpha production are more apparent in particular EEG frequencies and response measures. The lower frequencies of alpha, recorded over the right parietal lobe, are more sensitive to the manipulations of stress than the higher frequencies of alpha. In addition, although higher-order interactions are significant using average alpha amplitude measures, the main effects of stress and lower-order interactions, typically found in most experiments, are more sensitive to within-subject standardized (W-SS) alpha amplitude (Tyson, 1987). A 10-microvolt increase in alpha amplitude may be extreme for some and normal for other subjects. Extreme physiological responses are defined relative to the individual's normal response variability after the W-SS transformation.

Researchers unable to significantly increase alpha activity and demonstrate self-control are using inadequate training procedures, alpha frequencies, electrode sites, and standardization, and in some cases may be inadvertently introducing task-related stress in their feedback stimulus (Cott,

Pavloski, & Black, 1981; Cott, Pavloski, & Goldman, 1981; Plotkin, 1979). Tyson and Audette (1977) reported that subjects found frequency- and amplitude-modulated feedback sounds particularly unpleasant as the frequency became higher, possibly a task-related stressor when used with biofeedback. However, future research will find that the present experiment also provides subjects with less than optimal conditions for alleviating the effects of stress on alpha production.

The utilization, in this experiment, of an alpha-contingent feedback paradigm (Mulholland & Eberlin, 1977) in conjunction with noise stress is consistent with both behavioral and relaxation approaches to stress (Burchfield, 1985; Meichenbaum, 1976). Alpha production is a good barometer of subjects' success in coping with a particular contingent stressful stimulus. In this case the stressor was noise, but the alpha-contingent paradigm should be equally useful for a variety of other stressful stimuli, such as the loudness of a baby crying, contingent on increasing alpha production or lowering EMG. Consistent with behavioral approaches, learning an incompatible physiological response, contingent with a stressful stimulus, should extinguish the stress response. Anticipation training with phased-in control and interspacing self-control training are more consistent with the cognitive approaches that emphasize the importance of predictability and controllability when coping with stress (Lazarus & Folkman, 1984). For particular types of stress and anxiety, considerable biofeedback research is needed to define the most appropriate stressful stimuli, contingent EEG responses, and task conditions, such as phasing in both control and contingent stress. As Mulholland (1984) explicitly illustrates, the gaps in our knowledge justifying the use of EEG feedback as a rational therapy for stress and anxiety are much wider than the treatment of paresis with integrated EMG or Raynaud's disease with skin temperature, and these gaps will not be filled in the near future if the declining interest in EEG alpha biofeedback persists.

REFERENCES

Baum, A., Singer, J. E., & Baum, C. S. (1981). Stress and the environment. *Journal of Social Issues, 37,* 4-35.

Black, A. H., Cott, A., & Pavloski, R. P. (1977). The operant learning theory approach to biofeedback training. In G. Schwartz & H. Beatty (Eds.), *Biofeedback: Theory and research.* New York: Academic Press.

Broadbent, D. E. (1978). The current status of noise research: A reply to Poulton. *Psychological Bulletin, 85,* 1052-1067.

Burchfield, S. R. (1985). *Stress: Psychological and physiological interactions.* New York: Hemisphere.

Cohen, S. (1980). Aftereffects of stress on human performance and social behavior. A review of research and theory. *Psychological Bulletin, 88,* 82-108.

Cott, A., Pavloski, R., & Black, A. H. (1981). An example of methological limitations inherent in response-discrimination experiments. *Journal of Experimental Psychology: General, 110,* 398-414.

Cott, A., Pavloski, R., & Goldman, J. A. (1981). Cortical alpha rhythm, biofeedback, and the determinants of subjective state. *Journal of Experimental Psychology: General, 110,* 381-397.

Critchley, M. (1953). *The parietal labes.* New York: Hafner Press.

Epstein, L. H., & Blanchard, E. B. (1977). Biofeedback, self-control, and self-management. *Biofeedback and Self-Regulation, 2,* 201-211.

Glass, D. C., & Singer, J. E. (1972). *Urban stress: Experiments on noise and social stressors.* New York: Academic Press.

Graybiel, A. M. (1974). Studies on the anatomical organization of posterior association cortex. In F. O. Schmitt & F. G. Worden (Eds.), *The neurosciences. Third study program.* Cambridge: M.I.T. Press.

Hardt, J., & Kamiya, J. (1978). Anxiety change through electroencephalographic alpha feedback seen only in high anxiety subjects. *Science, 201,* 79-81.

Hord, D., & Barber, J. (1971). Alpha control: Effectiveness of two kinds of feedback. *Psychonomic Science, 25,* 151-168.

Kondo, C. Y., Travis, T. A., & Knott, J. R. (1975). The effects of changes in motivation on alpha enhancement. *Psychophysiology, 12,* 388-389.

Kryter, K. D. (1985). *The effects of noise on man.* New York: Academic Press.

Lazarus, R. S. (1977). Cognitive and coping processes in emotion. In A. Monat & R. S. Lazarus (Eds.), *Stress and coping.* New York: Columbia University Press.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping.* New York: Springer.

London, M. D., & Schwartz, G. E. (1984). The effects of activation versus inhibition of feedback on perceived control of EEG activity. *Biofeedback and Self-Regulation, 9,* 265-278.

Meichenbaum, D. (1976). Cognitive factors in biofeedback therapy. *Biofeedback and Self-Regulation, 1,* 201-216.

Mulholland, T. B. (1984). Concepts of control in biofeedback. In Th. Elbert, B. Rockstroh, W. Lutzenberger, & N. Birbaumer (Eds.), *Self-regulation of the brain and behavior.* New York: Springer-Verlag.

Mulholland, T. B., & Eberlin, P. (1977). Effects of feedback contingencies on the control of occipital alpha. *Biofeedback and Self-Regulation, 2,* 43-57.

Peper, E., & Mulholland, T. B. (1970). Methological and theoretical problems in the voluntary control of the electroencephalographic occipital alpha by the subject. *Kybernetic, 1,* 10-13.

Plotkin, W. B. (1979). The alpha experience revisited: Biofeedback in the transformation of psychological state. *Psychological Bulletin, 86,* 1132-1148.

Plotkin, W. B., & Rice, K. M. (1981). Biofeedback as a placebo: Anxiety reduction facilitated by training in either suppression or enhancement of alpha brainwaves. *Journal of Consulting and Clinical Psychology, 49,* 590-596.

Poulton, E. C. (1979). Composite model for human performance in continuous noise. *Psychological Review, 86,* 361-375.

Travis, T. A., Kondo, C. Y., & Knott, J. R. (1974). Parameters of eyes-closed alpha enhancement. *Psychophysiology, 11,* 674-681.

Tyson, P. D. (1982). The choice of feedback stimulus can determine the success of alpha feedback training. *Psychophysiology, 19,* 218-230.

Tyson, P. D. (1987). Within-subject standardization and repeated measures applied to biofeedback. *Psychophysiology.*

Tyson, P. D., & Audette, R. (1977). *The controversy over the relationship between alpha waves and experience during feedback.* Paper presented at the annual meeting of the Biofeedback Society of America, Orlando, Florida.

Tyson, P. D., & Audette, R. (1979). A multivariate approach to the relationship between alpha waves and experience during feedback. *Biofeedback and Self-Regulation, 4,* 63-79.

Tyson, P. D., Ogilvie, R. D., & Hunt, H. T. (1984). Lucid, prelucid, and nonlucid dreams related to the amount of EEG alpha activity during REM sleep. *Psychophysiology, 21,* 442-451.

Weinberg, J., & Levine, S. (1980). Psychobiology of coping in animals: The effects of predictability. In S. Levine & H. Ursin (Eds.), *Coping and health.* New York: Plenum.

(Revision received April 17, 1987)