ELECTROENCEPHALOGRAPHIC AND ELECTROMYOGRAPHIC BIOFEEDBACK TRAINING OF OCCIPITAL LOBE THETA RHYTHM

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ELECTROENCEPHALOGRAPHIC AND ELECTROMYOGRAPHIC BIOFEEDBACK TRAINING OF OCCIPITAL LOBE THETA RHYTHM

by

DAVID WILLIAM LAWSON

B.A., American International College, 1968
M.A., University of New Hampshire, 1975

A DISSERTATION

Submitted to the University of New Hampshire
in Partial Fulfillment of the Requirements of the Degree of Doctor of Philosophy
in Psychology

May, 1986
This dissertation has been examined and approved.

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Dec 17, 1985
Date
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David W. Lawson
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ABSTRACT

Electroencephalographic and Electromyographic Biofeedback Training of Occipital Lobe Theta Rhythm.

by

David William Lawson
University of New Hampshire, May, 1986

Three biofeedback training methods were used to train human subjects to enhance levels of occipital lobe theta electroencephalographic rhythms. It was hypothesized that the subject's level of physiological arousal would differentially influence the effectiveness of the training methods. The independent variables were: (1) baseline arousal, (2) training methods, and (3) pre/posttraining recording sessions. The dependent variables included: (1) theta EEG, (2) frontalis EMG, and (3) alpha EEG levels. For the baseline arousal variable, subjects were divided into low and high arousal groups based on a median-split of their pretraining frontalis EMG levels. The training methods included: (1) one-phase EMG/EMG feedback training that involved eight sessions of frontalis EMG training, (2) two-phase EMG/EEG feedback training with four sessions of frontalis EMG feedback followed by four sessions of theta EEG training, and (3) one-phase EEG/EEG training that
utilized eight sessions of theta EEG training. All three training methods were administered via random assignment to subgroups of the low and high arousal groups. Changes in the levels of the dependent variables were recorded across the two levels of the pre/posttraining recording sessions variable. It was predicted that low arousal subjects would significantly increase theta EEG levels when they received the one-phase EEG training; and, that the high arousal subjects would significantly increase theta EEG levels when trained with the one-phase EMG/EMG or the two-phase EMG/EEG training methods. The results indicated that although there was a statistically significant overall increase in theta EEG activity from the pretraining to the posttraining recording sessions, the research hypotheses of differential training effects were not supported. A posttraining inverse relationship between theta EEG and frontalis EMG that other researchers (Sittenfeld, Budzynski and Stoyva, 1976) had observed was not replicated. The statistically significant decreases in frontalis EMG levels were interpreted to be the result of the differential effectiveness of the training methods, but were unrelated to increases in theta EEG activity. The results did not support the hypothesis that the subject's level of physiological arousal, as indicated by frontalis EMG activity, was related to the effectiveness of the biofeedback training methods for the enhancement of theta EEG activity.
I. INTRODUCTION

Electrencephalographic theta rhythms are electrical signals recorded from the human cerebral cortex. These electroencephalographic (EEG) rhythms have been associated with distinctive alterations of human conscious experience. Theta rhythms have been most commonly identified with a state of low physiological arousal and subjective feelings of drowsiness. These feelings of drowsiness have been reported to be accompanied by a shift away from symbolic language oriented cognitive processes to a hypnagogic state dominated by visual imagery and non-verbal thought processes (Bertini, Lewis, and Witkin, 1969; Paivio, 1973; and Rechtschaffen, 1973). Theta rhythms, electrophysiologically, are defined as cortical waveforms falling within the frequency bandwidth of 3.50 to 7.50 hertz. The term "theta state" has been frequently used in the research literature to refer to an electroencephalographic condition characterized by a predominance of theta waves which may be accompanied by some faster frequencies. In an attempt to maintain continuity between the sleep and the psychophysiological literature, Elson, Hauri and Cunes (1977) have defined the "theta state" as being the equivalent of "Stage 1 sleep" as defined by Rechtschaffen and Kales (1968). Sleep researchers have consistently observed that there are systematic changes in electroencephalographic activity as subjects progressed from
full arousal to transitional states to unambiguous sleep (Elson et al, 1977). Full arousal is characterized by low voltage unsynchronized fast beta electroenchalographic rhythms. These beta rhythms typically range between 13 to 28 hertz. The transitional states that occur between full arousal and unambiguous sleep are characterized by either more than fifty percent alpha rhythms (8.00 to 12.00 hertz) or by a predominance of theta activity on a low voltage background (Elson et al, 1977). Sleep onset is, in turn, characterized by sleep spindles and k-complexes (Rechtschaffen and Kales, 1968). The transitional alpha-theta state has been observed to persist for approximately one-half to five minutes prior to the onset of unambiguous sleep (Vogel, Foulkes and Trosman, 1966). From this electrophysiological/behavioral context, two distinct forms of the "theta state" have been defined. The first, which is most commonly observed by sleep researchers, conforms to that described by Vogel and his colleagues (1966). This theta state, which leads to sleep onset within five minutes, may be labeled as "descending theta rhythm", "descending alpha-theta" or "hypnagogic alpha-theta state" (Elson et al, 1977). The second, which does not lead to sleep onset, may be labeled as "non-descending theta rhythm". There are no known electrophysiological distinctions between these two theta states. The principle distinctions are the duration of the period during which the theta rhythm is the dominant electroencephalographic rhythm, and the behavioral outcome.
The non-descending theta rhythm has been observed to persist for as long as 25 to 30 minutes, and it does not terminate in sleep onset. The biofeedback training of theta rhythm, in the present experiment, focused on training subjects to enhance and to maintain a non-descending theta rhythm state.

Until the development of biofeedback training in the late 1960's, it was not possible to train subjects via instrumentation, with appropriate controls, to volitionally control electroencephalographic activity. The early research of Kamiya (1968, 1969), Kamiya and Nowlis (1970), Miller, DiCara, Solomon, Weiss, and Dworkin (1970), and of Mulholland (1969), as well as many other investigators demonstrated that human subjects could be trained to regulate electroencephalographic alpha rhythms via direct electroencephalographic biofeedback training. In more recent years, a small number of researchers began to apply direct electroencephalographic biofeedback training methods to teach human subjects to regulate theta electroencephalographic activity. Interest in teaching humans to regulate theta rhythms was stimulated by possible applications to vigilance tasks (Beatty, 1972; Beatty, 1975; Beatty and O'Hanlon, 1975; Williams, 1975), relaxation training and stress management (Budzynski, 1971; Budzynski and Stoyva, 1969; Budzynski and Stoyva, 1973; Stoyva, 1973; Stoyva, 1976; Stoyva and Kamiya, 1968; and Stoyva and Budzynski, 1974), and the treatment of insomnia (Elson, Hauri and
I initially became interested in the biofeedback training of theta electroencephalographic rhythms in humans because of its putative value as a physiological correlate of human vigilance performance. Prior to the development of the current experiment on the training of theta enhancement, I had been conducting pilot research on the relationship between theta electroencephalographic activity and the vigilance performance of human subjects during a monotonous monitoring task. It had been well established by earlier researchers that the detection efficiency of human observers deteriorates rapidly in a wide variety of monotonous monitoring tasks (Sidowski, 1966; Worden, 1966; Mackworth, 1969; Mackworth, 1970; and Brazier, 1972). This effect has been labeled the "vigilance decrement"; and had been investigated extensively on a behavioral level. However, little research had been conducted on the neurological basis of this phenomena. It had been hypothesized that the vigilance decrement was related to the central nervous system processes which produce a decline in the organism's level of neurological arousal. Tests of this hypothesis had revealed that, of all of the available electroencephalographic indicators of central nervous system arousal, only occipital lobe theta
rhythms were reliably associated with vigilance responses (O'Hanlon, 1970; Beatty, 1972). This relationship between occipital lobe theta rhythm and human vigilance responding was examined experimentally by Beatty, Greenberg, Deibler and O'Hanlon in 1974, Beatty (1975), and Beatty and O'Hanlon (1975). They conducted an investigation, during which, subjects were taught via direct electroencephalographic biofeedback training to regulate their occipital lobe theta rhythms. One group of subjects was taught to enhance the level of theta rhythm, while a second group of subjects was taught to suppress the level of theta rhythm. When the ability to volitionally regulate the theta rhythm had been established, the effects of theta rhythm regulation on vigilance performance was then measured. It was found by these investigators that the suppression of occipital lobe theta rhythms improved monitoring performance on the vigilance task; and that the enhancement of occipital lobe theta rhythms was associated with a greater than normal decrement of monitoring performance on the vigilance task (Beatty, Greenberg, Diebler and O'Hanlon, 1974).

The results of this investigation were of significance, in that, for the first time a relationship was established experimentally between a physiological response and human vigilance performance. This was a finding of potentially the same magnitude as the discovery of REM sleep as a physiological correlate of dreaming. The finding that
vigilance performance could be influenced by the subjects' enhancement or suppression of occipital lobe theta rhythm activity was of interest to me because of its potential importance for basic research issues concerning information processing in the nervous system and for applied research in wide variety of human vigilance related problems. The applied research problems include the vigilance decrements encountered by air traffic controllers, radar and sonar operators ("radar blindness"), transportation safety problems related to "highway hypnosis" (Shor and Thackray, 1970), industrial monitoring situations requiring high levels of accurate performance during prolonged monitoring, and problems associated with sustaining attention in normal, perceptually handicapped or mentally impaired individuals in educational and work settings.

At that time, and still in the present, very little research had been conducted on the biofeedback training of theta rhythm; and, even less on the relationship between theta EEG activity and vigilance performance. Accordingly, I designed a research program, based on the available information, to further explore the relationship between the biofeedback trained regulation of occipital lobe theta EEG rhythms and human vigilance performance. In order to manipulate theta rhythms (enhancement and/or suppression) as an independent variable, it was necessary to have a standardized method of training the subjects to reliably regulate theta rhythm
activity. If theta rhythm regulation could be as easily taught as alpha rhythm regulation, then the relationship between theta rhythm activity and vigilance, information processing, and the areas of application could have been explored systematically. However, repeated attempts, over a four to five year period, to use the existing biofeedback method for the direct training of occipital lobe theta rhythms produced very few instances of regulation. When subjects were able to demonstrate some level of control over their theta EEG activity, it was neither strong enough nor reliable enough to utilize it as an independent variable in a vigilance experiment. This difficulty in teaching subjects to regulate theta rhythm was quite unexpected. The research evidence, that was then available, indicated that theta rhythm did not present any unusual obstacles to direct biofeedback training. However, it became obvious that the ease of training subjects to regulate theta rhythms reported by Beatty, Greenberg, Deibler and O'Hanlon (1974) was the exception rather than the rule. The persistent difficulties encountered, initially by me and later by others (Lutzenberger, Birbaumer, Wildgruber, 1975; Williams, 1975; and Lutzenberger, Birbaumer and Steinmetz, 1976), in replicating Beatty's ease of teaching subjects to regulate theta rhythm precluded any further attempts to systematically explore the relationship between theta electroencephalographic activity and vigilance performance. As a consequence, the orientation of the research program was shifted to an investigation
of the effectiveness of various biofeedback methods of training subjects to regulate theta rhythm activity. The first question to be addressed was whether biofeedback methods could be used to teach enhancement of theta EEG activity. The second question, if regulation was possible, was whether there were differences in the effectiveness of different biofeedback training methods.

At the time that this research program was designed to study the relationship between theta EEG regulation and vigilance, the vast bulk of research on the biofeedback training of electroencephalographic activity was devoted to the study of alpha rhythm and to sensorimotor rhythms. Alpha rhythm was so extensively researched, in part, because of its ease of training and its association with altered states of consciousness -- which was very much in harmony with the Zeitgeist of the 1960's and early 1970's. Interest was starting to develop in sensorimotor rhythm (SMR) because of its potential as a non-pharmaceutical alternative to the treatment of epileptic disorders (Sterman, 1974; and Sterman, 1977). Theta electroencephalographic biofeedback training was not and still has not been studied extensively.

During the period that I was attempting to study the relationship between theta electroencephalographic activity and vigilance performance, a few other researchers began to become interested in theta rhythm for other reasons. Most
notable among this small number were Johann Stoyva, Thomas Budzynski, and one of their students Pola Sittenfeld. Whereas, I was initially interested in utilizing the biofeedback training of theta electroencephalographic activity to study vigilance performance; they were interested in the relationship between the regulation of theta rhythms and the development of an anti-stress response that they labeled as "cultivated low arousal". It is to be emphasized that their research on theta EEG regulation was contemporaneous with mine; and that their interest focused on applied research for stress management, while mine focused on basic research concerning electrophysiological correlates of vigilance performance. The information from their investigations was not available at the onset of my studies on theta EEG activity and vigilance; and, became known to me as it became apparent that methodological issues concerning the biofeedback training of theta rhythm had to be addressed prior to the investigation of its relation to vigilance performance. The findings of a study by Sittenfeld, Budzynski and Stoyva (1976) provided a methodological and theoretical context for the current experiment on biofeedback methods of training human subjects to enhance occipital lobe theta rhythm activity.
Since 1938, when Edmund Jacobson published his now classic volume on deep muscle relaxation training, considerable attention has been devoted to the relationship between physiological relaxation and stress responses. Until recently the focus of this line of investigation was on the relationship between the state of the musculoskeletal system and the level of emotional arousal. A commonly encountered example of the application of the findings of this research may be seen in the use of deep muscle relaxation techniques to combat anxiety during Wolpe's systematic desensitization training (Wolpe, 1958; and Wolpe, 1973). Some biofeedback researchers (most notably Budzynski and Stoyva), who have been interested in the development of stress management techniques, have focused attention on non-descending theta rhythms. The primary reason for this interest had been the observation that subjects who are capable of producing and maintaining a non-descending theta rhythm state also experience deep muscle relaxation and a profound sense of emotional relaxation and calmness. It should also be noted that the physiological and emotional relaxation had been observed to accompany descending theta rhythm states as well (Foulkes and Vogel, 1965; Stoyva and Kamiya, 1968; Bertini, Lewis and Witkin, 1969; and Stoyva, 1970; and Rechtshaffen, 1973). However, a distinction was made in that with the non-descending theta rhythm state, the physiological and emotional
relaxation tends to persist in the waking state after the subject is no longer in the theta state; whereas, with the descending theta rhythm state, the subject directly enters stage 2 sleep and receives no residual benefits of the physiological or emotional relaxation in the waking state.

Consequently, researchers began to examine the possible application of the enhancement of non-descending theta rhythm activity as a means of developing anti-stress responses (Budzynski, 1977; Sittenfeld, Budzynski and Stoyva, 1976; Stoyva and Budzynski, 1972). They labeled the target anti-stress response that resulted from the ability to produce and maintain non-descending theta rhythm as a state of "cultivated low arousal" (Stoyva and Budzynski, 1972).

The working hypothesis underlying Stoyva and Budzynski's research was that individuals who are frequently stressed will demonstrate physiological hyperarousal in one of several bodily systems. A complimentary hypothesis of these investigators has been that frequently stressed or over-reactive individuals tend to lose the ability to relax well: i.e., to shift into a low arousal condition (Stoyva and Budzynski, 1972). They believed that these individuals develop, as a consequence of repeatedly having to mobilize their physical and mental resources, a response-set of high sympathetic arousal under conditions of stress. Due to this acquired tendency to mobilize themselves to meet stressors,
they proposed that these individuals are likely to lose their ability to execute the opposite response, i.e., to shift into the parasympathetic mode in which bodily recuperation normally occurs (Stoyva and Budzynski, 1972). They assumed that, since the physiological and behavioral patterns of hyperarousal under conditions of stress are acquired, these patterns could be modified to some degree. They proposed that biofeedback training procedures that had been designed to produce conditions of low physiological and behavioral arousal were appropriate for the modification of the patterns of hyperarousal. These biofeedback procedures were observed to produce effects indicative of parasympathetic dominance -- a condition assumed by Stoyva and Budzynski (1972) to be the opposite of the effects produced by stress. Other researchers had also explored this line of reasoning. For instance Rice and Blanchard (1982) indicated that the rationale for the utilization of biofeedback in the treatment of stress related disorders was based on the idea that if the autonomically mediated physiological arousal associated with anxiety could be reduced or controlled, the motor and behavioral manifestations as well as subjective reports might subsequently decrease. Hauri (1977) proposed that human subjects were capable of producing a generalized relaxation response that is apparently mediated via hypothalamic mechanics; and that clinical use of biofeedback seemed to be able to induce the "relaxation response" popularized and described by Benson and Klipper (1975), and
Benson, Kotch and Grassweller (1977). These views are all consistent with the concept of "cultivated low arousal" and the rationale of Stoyva and Budzynski for the development of methods to train an "anti-stress" response.

The research from their laboratory focused on the development of training methods to teach human subjects to acquire control over this state of cultivated low arousal. They observed that the acquisition of control over this state seemed to involve several stages (Stoyva and Budzynski, 1972). During the first stage, the subjects were able to relax only with deliberate effort. In the next stage, the relaxation response was easier to produce, even when the subjects were under some pressure. The final stage, only observed with some subjects, was characterized by the relaxation response having become virtually an automatic reaction, no longer requiring conscious effort. It is to be noted that these observations of the stages of training were made on patients in a clinical setting. Consequently, the description of these stages was in qualitative rather than quantitative terms. Their most recent orientation was experimental in nature and focused on examining methods of teaching subjects to lower their level of arousal by enhancing the level of theta electroencephalographic rhythms (Sittenfeld, Budzynski and Stoyva, 1976). Their experiments concentrated specifically on comparing the effectiveness of direct theta EEG biofeedback training with that of frontalis
electromyographic (EMG) biofeedback training on the acquisition of control over a state of cultivated low arousal. They predicted useful applications of these training methods in the treatment of tension headaches, essential hypertension, sleep onset insomnia, anxiety disorders, diabetes, muscular re-education, and as a preventative approach to stress alleviation (Stoyva and Budzynski, 1972; Budzynski, 1977).
Methods of Biofeedback Training of Theta Rhythms

At the time that this experiment was designed, and at the present time, there had been little research examining the biofeedback methods of training theta electroencephalographic activity. Beatty, Greenberg, Deibler and O'Hanlon (1974) reported successful acquisition of enhancement and suppression of occipital theta rhythms using direct theta biofeedback training in as little as two hours. Sittenfeld, Budzynski and Stoyva (1976) reported success in training subjects to enhance occipital theta rhythm activity with a two-phase biofeedback procedure, which coupled biofeedback pretraining of frontalis electromyogram activity (decrease) with follow-up direct biofeedback training of theta EEG activity. Lutzenberger, Birbaumer and Steinmetz (1976) reported successful enhancement of theta EEG activity in frontal lobe areas. They utilized simultaneous biofeedback training of heart rate and frontalis EMG activity as a pretraining for the regulation of theta EEG activity. They observed that there was a weak increase in frontal theta EEG activity over sessions of direct theta training, but a decrease within sessions. They concluded that pretraining with frontalis EMG and heart rate biofeedback had no influence on performance during the theta biofeedback training. With the exception of Beatty, Greenberg, Deibler and O'Hanlon (1974), most researchers have found that the production and maintenance of a non-descending theta state
was a difficult and demanding task for subjects to master. It is apparent that considerable work remains to be done on the development of reliable and efficient methods of training subjects to regulate theta electroencephalographic activity.

Thus far, two major obstacles to the biofeedback training of theta rhythm have been delineated. The first concerns the paradoxical nature of the subjects' subjective experiences when they finally manage to shift into a theta state. These experiences typically involve a high level of hypnagogic imagery and a low level of reactivity to exteroceptive stimuli. Consequently, the subjects' ability to perceive the external feedback signal becomes increasingly impaired as the density of the theta rhythm increases in the subjects' raw EEG.

The second involves the low baseline levels of theta rhythms in the raw electroencephalogram of the awake subject. Period analysis of the resting EEG in awake subjects indicated that the theta rhythms comprise less than twenty percent (Mean = 19.88%; Standard Error = 0.75%) of the EEG in the occipital lobes and slightly less in the temporal lobes (Cohen, Bravo-Fernandez, Hose and Sances, 1976). Preliminary measurements made by me indicated that the baseline of occipital lobe theta EEG rhythms in the awake resting subject may range from 0 to 21 percent of the raw
EEG. These low baseline levels present a particularly difficult obstacle to the researcher or clinical practitioner whose attempts to implement direct theta EEG biofeedback training depends on having the subject receive consistent and meaningful feedback information from the recording instruments. Typically, direct theta EEG training methods only provide feedback signals when theta rhythms are present in a pre-set minimal amount -- frequency, voltage and duration values are set on the biofeedback instrument. When theta EEG activity conforming to these values is detected, a feedback signal is triggered that informs the subject that the desired EEG pattern has occurred. When theta rhythms are absent or present at levels below the criteria set on the instruments, there is a corresponding absence of the feedback signals. As Stoyva (1973) has emphasized, learning to produce theta rhythms is a subtle task; and the baseline level is generally low -- often too low to generate a usable feedback signal. This creates a situation in which the experimental subject realizes little, if any, improvement in theta EEG regulation over the training sessions. Typically, this lack of progress causes frustration; which, in turn, leads to an increase in the subjects' level of physiological and behavioral arousal. The increase in physiological arousal has been detected through the measurement of increased levels of alpha and beta EEG activity. These increases in the proportion of alpha and beta rhythms in the raw EEG further decrease the probability of occurrence of
theta EEG activity. In essence a condition has been observed to develop, as a consequence of too little feedback, that ensures that the subject would experience progressively less feedback as the training session continued.

A possible solution to this problem was suggested by Stoyva (1973) and was tested by Sittenfeld, Budzynski and Stoyva (1976). As an alternative to the conventional direct theta EEG biofeedback training method, Stoyva (1973) suggested that it might be better to conduct the training in two phases. The initial phase involved direct frontalis EMG biofeedback training. This phase was followed by direct theta EEG biofeedback training. The initial frontalis EMG training was selected by Stoyva because prior work had indicated that it was an easy and superior method of teaching subjects to reach a low level of arousal (Budzynski and Stoyva, 1969; Budzynski and Stoyva, 1973; Stoyva and Budzynski, 1974). Budzynski and Stoyva (1969) also indicated that the reductions in frontalis EMG activity produced by biofeedback training generalized to other muscle groups and were associated with reductions in autonomic and subjective arousal. Thus, the frontalis muscle group seemed to possess unique properties that made it the logical choice for a pretraining procedure designed to reduce levels of arousal. Early work had also revealed that when frontalis EMG levels became very low that increases in theta EEG activity were likely to be observed (Budzynski and Stoyva, 1969). Thus,
they considered the pretraining sessions with frontalis EMG biofeedback to be analogous to an operant shaping procedure that served to increase the probability of occurrence of theta EEG activity in the awake subject. It was predicted that if it was used as a pretraining procedure for the later direct biofeedback training of theta EEG activity, it would increase the probability that the subject would receive high levels of meaningful feedback information at the onset of the direct theta training.

Sittenfeld, Budzynski and Stoyva (1976) were interested in developing biofeedback training methods to teach subjects to produce and maintain the state of cultivated low arousal that was observed to be associated with non-descending theta rhythms. Specifically, they were interested in determining whether biofeedback training methods could be matched to the physiological characteristics of the subjects. The purpose was to provide a means of screening subjects prior to training, so that the optimal training method could be applied to teach the acquisition of cultivated low arousal skills. The rationale of their experiment was firmly based on the earlier qualitative observation of Stoyva (1970) and Stoyva and Budzynski (1974) that an inverse relationship existed between the frontalis EMG level and the proportion of theta rhythms present in the raw EEG. This inverse relationship was of interest because of its potential to account for their observation that high arousal subjects
typically experienced difficulty in the acquisition of theta EEG control and the associated state of cultivated low arousal. The conventional method of training subjects to enhance theta rhythms, as indicated earlier, involves direct theta EEG biofeedback. This method only delivers feedback information when the theta EEG is actually detected in minimum quantities in the raw EEG. If the theta rhythm is not present in sufficient amounts, then the subject would not receive enough feedback information to produce increases in the theta rhythm levels.

Sittenfeld, Budzynski and Stoyva (1976) reasoned that the high level of arousal, as indicated by the frontalis EMG level, represented a unique physiological state which made the conventional direct theta EEG biofeedback training methods ineffective. On the other hand, the subjects characterized by a low state of arousal, as indicated by low frontalis EMG levels, were expected to exhibit high levels of theta EEG activity prior to training. These high pre-training theta EEG levels were expected to enable the low arousal subjects to readily acquire control of theta EEG activity with the conventional direct theta EEG biofeedback training. Thus, the low EMG subjects' physiological state of low arousal was expected to allow this method of training to be differentially effective for the enhancement of theta EEG activity. Conversely, the high EMG subjects' physiological state of high arousal was expected to interfere with
their ability to benefit from the same conventional direct theta EEG biofeedback training method.

From the context of the inverse relationship between frontalis EMG levels and theta EEG levels, Sittenfeld, Budzynski and Stoyva (1976) designed an experiment to test the hypothesis that the subjects' physiological arousal, as indicated by low or high frontalis EMG levels, would influence the effectiveness of the two selected biofeedback training procedures. The first method, labeled binary feedback (Kimmel, 1981), was the conventional direct theta EEG training procedure, in which, subjects over eight sessions received analogue feedback for the presence of theta EEG activity and no feedback for the absence of theta EEG activity. This method was hypothesized to be best suited to the physiological state of the low EMG subjects. The second training method was a two-phase procedure that involved four sessions of analogue biofeedback for the reduction of frontalis EMG levels. This was followed by four sessions of the conventional direct theta EEG training, during which subjects received analogue feedback for the presence of theta EEG activity, and no feedback for its absence. This method was hypothesized to be best suited to the physiological state of the high EMG subjects. As described earlier, the two-phase EMG/EEG training was considered to be analogous to an operant shaping procedure, where the level of arousal of the high EMG subjects would be
lowered by training subjects to reduce the frontalis EMG levels. It was anticipated, on the basis of prior EMG research, that the first four sessions of frontalis EMG training would lower frontalis EMG levels sufficiently to allow for a naturally occurring increase of the proportion of theta EEG activity present in the raw EEG. It was predicted that the high EMG subjects would then be better able to activate the direct theta EEG biofeedback instrument and receive enough feedback information in the final four sessions to bring theta EEG rhythms under control. Both of the training methods (one-phase EEG/EEG and two-phase EMG/EEG) were to be administered to different groups of low and high frontalis EMG subjects.

Sittenfeld, Budzynski and Stoyva (1976) equated the subjects' low frontalis EMG with a low level of physiological arousal and the high frontalis EMG with a high level of physiological arousal. In order to minimize confusion with the labels for the training methods that involved frontalis EMG training, I used the terms low arousal and high arousal as synonyms for Sittenfeld, Budzynski and Stoyva's labels -- "low EMG" and "high EMG", respectively.
Results of Sittenfeld, Budzynski and Stoyva's 1976 Experiment

The results of this experiment supported their research hypotheses and were interpreted from the context of the inverse relationship between frontalis EMG and theta EEG activity; the appropriateness of matching biofeedback training methods to the physiological characteristics of the subjects; and the concept of the state of cultivated low arousal as an anti-anxiety response.

Inverse Relationship Between Frontalis EMG and Theta Rhythm:
The first specific finding of note, was the confirmation of the inverse relationship between frontalis EMG and theta EEG levels. It had been proposed that the frontalis EMG level was an indicator of physiological arousal, and as such that it was inversely related to theta EEG activity -- an indicator of a low degree of central nervous system arousal. Sittenfeld and her colleagues reported that for the subjects as a whole, there was a clear inverse relationship between frontalis EMG levels and theta output. The negative correlation for all subjects, based on posttraining baseline data, was a -.53, p<.05.
**Training Procedures**

It was concluded by Sittenfeld, Budzynski and Stoyva (1976) that biofeedback training could be used to teach subjects to significantly increase their theta EEG levels to a point above their initial pretraining baseline levels. As may be observed in the summary table (Refer to Table I), they detected a significant three-way interaction for both the theta EEG and frontalis EMG measurements (Baseline Arousal Level X Training Method X Pre/Posttraining Recording Sessions). Their analysis of this interaction revealed that the overall significant increase in theta production from the pretraining to the posttraining recording sessions was mainly attributed to the large increases in the high arousal two-phase EMG/EEG group and the low arousal one-phase EEG/EEG group. It was, also, noted that the high arousal one phase EEG/EEG group actually demonstrated a slight decrease in theta output, although they had received theta biofeedback training for all eight sessions. It was observed that the low arousal two-phase EMG/EEG group did not produce any change from the pretraining to the posttraining baseline sessions. Thus, they concluded that it was necessary to take the subjects' physiological state, level of arousal -- as indicated by the frontalis EMG, into consideration before implementing any training procedure for the enhancement of theta EEG activity.
<table>
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<tr>
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<th>Theta EEG</th>
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<tbody>
<tr>
<td>Arousal</td>
<td></td>
</tr>
<tr>
<td>Training</td>
<td>* p &lt; .01 (increase)</td>
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<tr>
<td>Pre/Post</td>
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<tr>
<td>Arousal X Training</td>
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<tr>
<td>Arousal X Pre/Post</td>
<td>* p &lt; .001 (decrease)</td>
</tr>
<tr>
<td>Training X Pre/Post</td>
<td>* p &lt; .01</td>
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<tr>
<td>Arousal X Training X Pre/Post</td>
<td>* p &gt; .05</td>
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<table>
<thead>
<tr>
<th>Variable</th>
<th>Frontalis EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arousal</td>
<td>* p &lt; .01</td>
</tr>
<tr>
<td>Training</td>
<td>* p &lt; .001 (decrease)</td>
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<tr>
<td>Pre/Post</td>
<td>* p &lt; .01</td>
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<tr>
<td>Arousal X Training</td>
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<td>Arousal X Pre/Post</td>
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<tr>
<td>Training X Pre/Post</td>
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<td>Arousal X Training X Pre/Post</td>
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<table>
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<tr>
<th>Variable</th>
<th>Alpha EEG</th>
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<tbody>
<tr>
<td>Arousal</td>
<td>* p &lt; .01</td>
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<td>Training</td>
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<td>Pre/Post</td>
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<tr>
<td>Arousal X Training</td>
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<td>Arousal X Pre/Post</td>
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<td>Training X Pre/Post</td>
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<tr>
<td>Arousal X Training X Pre/Post</td>
<td>* p &gt; .05</td>
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They also interpreted the differential effectiveness of the two-phase EMG/EEG biofeedback procedure on the high arousal group as confirmation of the operant shaping properties of this procedure. In support of this interpretation that high arousal subjects had been shaped to produce higher levels of theta EEG rhythms during the first four EMG feedback sessions, it was noted that theta EEG levels had increased during these sessions -- prior to the receipt of theta biofeedback training.

As previously indicated, it was found that the subjects with the low arousal levels showed increases in theta EEG activity only if they were given the direct one-phase EEG/EEG theta feedback training for all eight of the training sessions. Thus, the one-phase EEG/EEG training was associated with an enhancement of theta rhythms for the low arousal subjects, while the two-phase EMG/EEG training did not produce any enhancement for the low arousal subjects. This differential training effect was interpreted also as supporting the hypothesis that the physiological state of the subject must be considered when selecting training methods. It was reasoned that low arousal subjects, by virtue of their low state of physiological arousal, would have been expected to have greater levels of theta rhythm present in their raw EEG prior to training. Consequently, they should have been able to receive enough feedback information with the one-phase EEG/EEG training procedure to
acquire learned control of their theta rhythm activity. The two-phase EMG/EEG training procedure, on the other hand, did not produce increases in theta EEG activity for these low arousal subjects. This finding was interpreted as being due to a combination of scaling and motivational factors. Because the frontalis EMG activity was already at a low level prior to training, scaling effects probably limited the degree of decrease possible during the four EMG training sessions. This would then produce a motivational effect. For these subjects whose frontalis EMG level was already quite low, the four sessions of EMG feedback training probably did not produce much expectation of success. Their feedback information would have indicated to them that their EMG levels were not changing greatly; and, this could have led to a "sense of incompetence and frustration". This negative motivational state could then have had the paradoxical effect of actually increasing the state of arousal in the very subjects who began the experiment with the lowest levels of arousal.

A Constructive Replication of the Sittenfeld, Budzynski and Stoyva (1976) Study

Consideration of the difficulties encountered in utilizing the "direct" biofeedback training methods for theta rhythm regulation by me and by those reported in the vigilance research, and the need to develop reliable and effective
methods of training theta rhythm regulation skills for research on the relationship of the "theta state" to physiological and psychological variables, made the Sittenfeld, Budzynski and Stoyva (1976) experiment a logical starting point. They had reported: (a) success in teaching subjects to enhance theta EEG activity in eight training sessions; (b) that a statistically significant inverse relationship existed between frontalis EMG levels and the amount of theta rhythm present in the EEG; (c) the finding that training techniques should be adapted to the physiological characteristics of the individual subject -- the pretraining baseline levels of frontalis EMG activity; and (d) that frontalis EMG is an indicator of physiological arousal.

I was interested in determining: (1) whether subjects could learn to enhance theta EEG levels; (2) whether some training methods produced differential effects in teaching subjects to regulate theta EEG activity; and (3) if subjects could be pre-selected according to frontalis EMG levels for assignment to "appropriate" training methods. It was anticipated that the inverse relationship between frontalis EMG and theta EEG activity would be easily replicated.

Sittenfeld and her associates (1976) observed that, in the course of training, the theta rhythm levels for the high arousal two-phase EMG/EEG group began to rise during the initial EMG feedback phase. This was prior to the
administration of any direct theta EEG feedback training. This observation, coupled with the significant inverse relationship between frontalis EMG and theta EEG activity, suggested that direct theta EEG feedback training may have been unnecessary for the high arousal subjects. However, the design of their experiment did not enable them to determine if a one-phase frontalis EMG/EMG biofeedback training procedure would have worked as well as the two-phase EMG/EEG feedback training procedure did for the high arousal subjects. If a one-phase EMG/EMG feedback procedure could be demonstrated to be differentially effective in teaching high arousal subjects to enhance theta rhythm activity, it would be a finding of great value.

Biofeedback training of frontalis EMG levels is an easily mastered task. It involves an analogue feedback signal that delivers consistent, easy to comprehend feedback information. This method, if proved successful as a means of teaching high arousal subjects to enhance theta EEG activity, would by-pass the difficulties inherent in the direct biofeedback training of theta rhythms. The direct feedback training, as described earlier, is of a "binary" type (Kimmel, 1981). If theta rhythm is detected in the raw EEG, then an analogue feedback signal is presented. If the theta rhythm drops below the criterion value, then the feedback signal is terminated. If theta rhythm is absent or below the criterion level, the feedback signal is not presented.

If the inverse relationship between frontalis EMG and theta
EEG levels is valid, then high arousal subjects would be unlikely to benefit from the direct EEG feedback training of theta rhythm. It would, obviously, be extremely useful to have a simple, easy to master EMG biofeedback task to employ with the high arousal subjects to train them to enhance theta rhythm activity. This "indirect" training of theta rhythm activity would sidestep all of the difficulties inherent in the direct EEG feedback procedure for these high arousal subjects. If a one-phase EMG/EMG procedure was found to be differentially effective with high arousal subjects, it would save vast amounts of time in both basic and applied research settings. If, in a basic research investigation, a researcher was interested in teaching theta enhancement skills in order to study the relationship between sustained theta EEG activity and some physiological or behavioral variable, then considerable savings of time and effort could be realized by assigning subjects to differentially effective training methods based on knowledge of an easily measured physiological variable -- frontalis EMG levels. The same would also hold true in an applied research or clinical setting, where the objective would be to teach the client anti-stress skills associated with the state of cultivated low arousal. Thus, research results concerning the differential effectiveness of a one-phase EMG/EMG and/or a two-phase EMG/EEG training method for high arousal subjects, and of a one-phase EEG/EEG training method for low arousal subjects would be of considerable practical
value. The basic intent of the present experiment was to replicate the findings of Sittenfeld, Budzynski and Stoyva's 1976 experiment on the differential effects of the one-phase EEG/EEG and the two-phase EMG/EEG training procedures for the low and high arousal subjects, respectively; and, to extend those findings by testing the effectiveness of an additional biofeedback procedure -- the one-phase EMG/EMG training of frontalis activity -- as a means of enhancing theta rhythm levels.

The methodology of Sittenfeld, Budzynski and Stoyva (1976) was followed in the present experiment to facilitate comparisons of the effectiveness of the training methods. The addition of an additional biofeedback training method -- one-phase EMG/EMG -- represents the only major deviation from their procedure. However, since different laboratories, recording and feedback instruments, and populations of subjects were employed, the present experiment must be considered a constructive rather than a literal or operational replication (Lykken, 1968). Thus, the intent of the replication should be construed as a validation and extension of the empirical relationships reported by Sittenfeld, Budzynski and Stoyva in their 1976 experiment, and not an exhaustive test of the "experimental recipe" presented in their methods section (Lykken, 1968).
Basic Experimental Design and Hypotheses

Independent Variables

There were three independent variables in the present experiment: (a) baseline arousal levels, (b) training methods, and (c) pre/posttraining recording sessions.

The baseline arousal variable involved a classification of subjects into two categories based on a median split of the frontalis EMG measurements made during two pretraining baseline recording sessions: (1) low arousal and (2) high arousal.

There were three training methods employed: (1) one-phase frontalis EMG biofeedback training (EMG/EMG) for all eight sessions; (2) two-phase frontalis EMG and theta EEG biofeedback training (EMG/EEG) — frontalis EMG training for the first four sessions and direct theta EEG training for the second four sessions; and (3) one-phase direct theta EEG biofeedback training (EEG/EEG) for all eight sessions.

The pre/posttraining recording sessions variable was divided into two levels: (1) pretraining baseline recording sessions; and (2) posttraining baseline recording sessions. The pretraining and posttraining data were derived from two separate baseline recording sessions prior to training and
two separate baseline recording sessions following training, respectively.

Dependent Variables

The dependent variables included: (1) percentage of theta rhythms in the raw electroencephalograph; (2) mean aptitude (microvolts) of the frontalis electromyograph; and (3) percentage of alpha rhythms in the raw electroencephalograph.

Hypotheses

The research hypotheses in the present experiment were as follows: (1) the one-phase EEG/EEG training procedure would be associated with a statistically significant increase in theta rhythm levels and a decrease in frontalis EMG activity for the low arousal subjects; and (2) the one-phase EMG/EMG training and the two-phase EMG/EEG training methods would be associated with a statistically significant increase in theta rhythm activity and a decrease in frontalis EMG levels for the high arousal subjects. It was also assumed that the correlation coefficient for all subjects would be statistically significant for the inverse relationship between frontalis EMG and theta rhythm levels at the posttraining baseline recording sessions.
**Theta EEG Predictions**

It was predicted that the differential effects on theta enhancement of the training methods for the low and high arousal subjects would be detected by a statistically significant Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions interaction.

**Frontalis EMG Predictions**

It was predicted that the differential effectiveness of the training methods for low and high arousal subjects on the reduction of frontalis EMG level would be indicated by a significant Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions interaction.
II. METHOD

Subjects

Thirty-six adult human subjects (thirty females and six males) were recruited through advertisements and the direct solicitation of classes from the student and staff population of the University of New Hampshire. The ages of the subjects ranged from 17 to 45 years. Subjects underwent a pre-experimental screening interview to detect those who may have been suffering from respiratory ailments, musculo-skeletal disorders, diabetes (Fowler, Budzynski and Vanden-Bergh, 1976 and Turkat, 1982), neurological disorders; or who may have been taking medications that have central nervous system or musculo-skeletal effects. If they had been suffering from any of the above disorders or taking medications, they would not have been allowed to participate in the experiment. However, none of the subjects were found to be suffering from any of these disorders or taking medications. Consequently, none of the subjects were excluded. During this interview the purpose of the experiment, procedures, and payment arrangements were explained to the subjects. The payments were drawn from an account set up from a Central University Research Fund Grant. The subjects were allowed to ask questions concerning procedures, scheduling and the nature of the experimental
tasks. A single blind control was employed. The subjects were allowed to ask questions so that informed consent could be meaningfully given. However, the subjects were not given information concerning the research hypothesis; the nature of the independent, dependent or control variables; levels of arousal; or information concerning relaxation skills. After the consent form was signed, an appointment for the first session -- the adaptation session was made. All subjects were requested to abstain from the use of alcohol, marijuana, caffeine containing products -- including coffee, teas, and soft drinks --, tobacco products, and medications for at least four hours prior to their participation in the experimental sessions. If this was not possible or if the subjects forgot and failed to abstain, they were told to inform the experimenter at the start of the session. As a backup, the experimenter questioned each subject prior to the start of the session. Four sessions were subsequently cancelled and rescheduled because of illness or drug usage. Each subject received a single twenty dollar payment upon completion of the final experimental session. The specific information given to the subjects concerning the experiment may be found in the consent form and questionnaire that are included in the Appendix.
Instrumentation

During all sessions, electroencephalographic measurements were recorded with an Autogen 120 Encephalograph Analyzer. The electromyographic measurements were recorded with an Autogen 1700 Myograph Analyzer. These instruments also supplied the biofeedback signals via a speaker placed 20 inches from the subjects.

The Autogen 120 Encephalograph Analyzer has two separate active band-pass filters that were pre-set to the theta rhythm (3.5 - 7.5 hertz) and the alpha rhythm (7.5 - 12.5 hertz) frequency ranges. The Autogen 120 also has a sleep alarm that was pre-set during all recording sessions to detect subjects who might slip into a sleep stage. The sleep alarm was not activated by any of the subjects during the course of the experiment. All EEG data were recorded directly from the Autogen 120 meter displays and a Data Technology digital multimeter.

The Autogen 1700 Myograph Analyzer was set to a band-width of 100 to 1 KHz. The sensitivity at this setting was 0.05 microvolts. All EMG data were recorded directly from the Autogen 1700 meter display and the Data Technology digital multimeter.
Electrode Placement

The EEG electrodes were positioned in a bipolar configuration at the occipital and central locations -- O2 and C4, as specified by the International 10 - 20 system (Jasper, 1958). The EMG electrodes were positioned in the standard frontalis sites (Lippold, 1967). The EEG electrodes were standard Grass gold cup electrodes. The EMG electrodes were standard Beckman silver skin electrodes. The EEG electrodes were attached to the scalp, after alcohol cleansing of the scalp, by tape. The EMG electrodes were attached, after alcohol cleansing of the skin, by double-sided adhesive electrode collars. Beckman Electrode Paste was used as the electrolyte with the EEG electrodes. Parker Signa Gel was used as the electrolyte for the EMG electrodes.

Feedback Signals

Binary feedback was provided for the theta EEG training. The feedback signal was a frequency and amplitude modulated tone that was presented only when theta rhythms were detected in the raw EEG. If theta rhythms were not detected, the feedback signal was not presented. The pitch of the tone was modulated by the frequency of the theta rhythms -- low pitch with lower theta rhythm frequencies and high pitch with higher theta frequencies. The volume of the tone was modulated by the amplitude of the theta rhythms -- low
volumes with low amplitude and high volume with high amplitude. The subjects were instructed to think of the feedback signal as being under the control of a volume switch and a bass-treble switch like that of a stereo system. They were not told of the relationship to EEG frequency and amplitude. They were instructed to try to get the signal to produce as much "bass" and "volume" -- low pitch and loud -- as possible, and to maintain the signal at that level. This meant that the theta rhythms should have been of low frequency and high amplitude, if the subjects were able to enhance the theta rhythm levels over the training sessions. A non-contingent feedback signal was briefly presented to the subjects prior to the start of the first theta feedback session to allow them to become familiar with its characteristics.

The EMG feedback signal was an audible click, which was linked to the frequency of the motor unit discharges for the frontalis muscle groups. The click signal gave the subjects continuous analogue feedback information about the degree and direction of change in the EMG amplitude. Subjects were instructed to produce and maintain the lowest possible rate of the clicks, which indicated a decrease in muscular activity. They were not told that this was associated with "relaxation" or "low arousal". No instructions for relaxation or relaxation training were given. Prior to the first training session, a non-contingent feedback signal was
briefly presented to the subjects to familiarize them with the nature of the signal.

Procedure

The procedure closely followed that of the 1976 Sittenfeld, Budzynski and Stoyva experiment. Each subject participated in thirteen separate experimental sessions, which included: one adaptation session, two pre-training baseline recording sessions, eight biofeedback training sessions, and two posttraining baseline recording sessions. Each of the sessions involved a 25 minute period of electroencephalographic and electromyographic recording. During the two pretraining and the two posttraining baseline recording sessions, the recordings were made without the presentation of feedback signals. The eight training sessions involved the same electroencephalographic and electromyographic recording procedures with the addition of an appropriate response contingent feedback signal. The number and the length of the sessions were selected as a replication of the conditions in the Sittenfeld, Budzynski and Stoyva (1976) experiment. All electrophysiological recordings were made in an electrically shielded, sound and light attenuated room. The recording instruments and the experimenter were in a separate adjoining instrumentation room while the recordings were being made. The subjects had the electrodes applied in an adjacent room; and were then led to the
experimental room, where they assumed a supine position on a bed. The application of the electrodes required approximately fifteen to twenty minutes. After the comfort of the subject was established, the experimenter left the experimental room and went into the instrumentation room to check the electrode impedance and to calibrate the recording instruments. A final check was made on the subject, then the lights were shut off and both the doors to the experimental room were closed. All recordings were made in a dark, silent room while the subject was in a supine position. Although the subject was physically isolated from the experimenter, who was in the adjacent instrumentation room, two-way communication was always possible via an intercom. The recording then commenced and continued without interruption for the next 25 minutes. Data were recorded for all dependent variables every 50 seconds. At the conclusion of each recording session, the electrodes were removed and the time and date of the next session was scheduled.

The adaptation and baseline sessions were identical for all subjects. Only the eight training sessions varied between the groups. The adaptation session was included to allow the subjects to adapt to the laboratory setting and the recording procedures prior to the two pretraining baseline recording sessions. In the adaptation session, the recording procedures and laboratory instruments were explained to the subject. The subject was then given another opportunity
to ask questions concerning procedures, possible risks and anything else relevant to informed consent. No information was given to the subject concerning the hypotheses being tested or the expected effects of the subject's training method on the dependent variables. Although enough relevant information was given to the subject concerning the recording procedures and possible risks to enable the subject to grant informed consent, the subject was kept "blind" to the nature of the independent variables, dependent variables and the hypotheses being tested. They were told that the experiment was examining the effects of various methods of training people to control brain wave patterns. No information concerning arousal, relaxation or the theta state was given to the subject. When the subject's questions, if any, had been answered, the electrodes were applied; and the subject entered the experimental room and recordings were made for 25 minutes. In the baseline sessions, the subjects were instructed to lie quietly while the recordings were being made. In the feedback sessions, the feedback signal was explained to the subject during the first session. In the first and in subsequent feedback sessions, the electrodes were connected and checked, the instruments were calibrated and the recordings were made for twenty-five minutes. At the start of the first feedback training session, the two-phase EMG/EEG and the one-phase EMG/EMG training groups were given instructions concerning the frontalis EMG feedback signal. They were told that the
recording instrument was monitoring the level of activity of the frontalis muscles directly above the eyes on the forehead; and that the feedback signal -- the clicking sound -- would tell them about this level of activity. A high rate of clicking indicated a high level of activity and a low rate of clicking indicated a low level of activity. These subjects were instructed to monitor the clicks during the training sessions, and to try to make the clicks occur at a slow steady rate. They were not told of the relationship of frontalis muscular activity to arousal, relaxation or theta EEG activity. They were not given any information concerning relaxation, training in relaxation skills, or suggestions for strategies that might help to control the feedback signal. At the start of the fifth feedback training session, the two-phase EMG/EEG training groups were told that the feedback signal would be changed and that it would give them information concerning the presence or absence of a brain wave pattern. The frontalis EMG signal was discontinued with the commencement of the theta EEG training. No mention was made of theta rhythm or any other specific EEG rhythms. The subjects were told that when the brain wave pattern was present that they would hear a tone, and that the tone would vary in pitch (treble to bass) and volume (faint to loud). They were instructed to monitor this signal and to try to maintain it with as much volume and as much bass as possible (as loud and low in pitch as possible). The subjects were then informed that the absence of
the feedback signal indicated the absence of the brain wave pattern. No instructions or strategies for the control of the feedback signal were given to the subjects. The preceding information, regarding the EEG biofeedback training, was also given to the one-phase EEG/EEG training groups at the start of the first training session.

After the conclusion of the final training session, the subjects were scheduled for the two posttraining baseline recording sessions. The procedures followed in the posttraining recording sessions were identical to that followed in the training sessions, except that the feedback signal was absent. The subjects were informed that the feedback signal was not going to be present during these two sessions; and that they should try to regulate the frontalis muscle activity level/or brain wave pattern without the feedback signal. The only instructions given to the subjects were to try to recreate the conditions -- internal sensations and/or subjective experiences -- that were associated with the successful regulation of the feedback signals during the training sessions. The nature of these sensations or subjective experiences was not specified.

Assignment of Subjects

As indicated above, the subjects were solicited via advertisements and classroom visits. After the screening
interview, the subjects were randomly assigned to the training method conditions. At the conclusion of the experiment, the subjects were classified into the low and high arousal groups based on a median-split of the average frontalis EMG values of the two pretraining recording sessions.

**Measurement of Alpha EEG**

Alpha rhythm measurements were included as a control for the detection of habituation effects and global relaxation effects that were not specific to effects of the training methods or the classification of subjects into Low and High Arousal groups.

It is emphasized that in the Sittenfeld, Budzynski and Stoyva 1976 experiment and the present experiment, specific patterns of responses were predicted as a function of the interaction of the training methods with the subject's baseline level of frontalis EMG. That is, the subject's pretraining level of physiological arousal would influence the effectiveness of the biofeedback training methods. In the present experiment, the high arousal groups were expected to demonstrate significant enhancement of theta EEG activity over the pretraining to the posttraining baseline sessions only when they received the one-phase EMG/EMG or the two-phase EMG/EEG training methods. The one-phase
EEG/EEG training method was not expected to be associated with an enhancement of theta rhythm activity for the high arousal subjects. On the other hand, the low arousal subjects were expected to demonstrate an enhancement of theta rhythm levels over the pretraining to the posttraining baseline sessions only when they received the one-phase EEG/EEG training methods. The one-phase EMG/EMG and the two-phase EMG/EEG training methods were not expected to be associated with an increase in theta EEG levels for the low arousal subjects.

A "no-feedback" or "no treatment" control was not utilized in the present experiment because specific patterns of results were being predicted by the research hypotheses. The no-feedback procedure has not been demonstrated to be an adequate control procedure in biofeedback research for non-specific or placebo effects when the purpose of the research was to compare the effectiveness of various treatment or training methods on the acquisition of control over some physiological response. In such a situation the absence of feedback is not equivalent to the absence of treatment. The absence of a feedback signal creates a markedly different motivational state than is found in any of the groups receiving a feedback signal. The feedback signal provides reinforcement for correct performance, information concerning current performance and progress over training sessions, and as previously described influences
expectations for success and motivation in later training sessions. If the purpose of the present research had been to examine the issues of "how biofeedback works" and the relationship between the characteristics of the feedback signal and the responses under study, then no-treatment controls, attention placebo groups and altered contingency groups (Hatch, 1982) would have been employed as needed. However, these controls were not considered necessary or appropriate to the design or interpretation of the current experiment or that of Sittenfeld, Budzynski and Stoyva (1976).

The question could be legitimately raised, since there was not a "no-treatment" control group, that increases in theta activity could be due to central nervous system habituation to the experimental environment or to non-specific variables in the experimental setting that would move all of the subjects in the direction of increased relaxation. Could simply letting subjects spend an equal number of sessions in the laboratory, but without feedback, have produced the same results? After all, the laboratory setting was designed to be conducive to comfort and relaxation. Subjects were tested while lying down on a bed in a sound-proof, light-proof room. Obviously, demand characteristics would be present that could influence subjects in the direction of increased relaxation -- decreased arousal over sessions. Thus, physiological indications of low arousal could be due
to a combination of habituation effects and non-specific variables associated with the experimental setting that could influence arousal over time and the various treatments and groups. However, the effects of habituation and the non-specific variables would be global and not systematically related to any of the training methods or baseline arousal groups. However, these factors would not account for the systematic differential changes in theta EEG activity that were predicted in the present investigation or the Sittenfeld, Budzynski and Stoyva (1976) experiment. It was determined by Sittenfeld and her associates that although habituation could account for a portion of their results, i.e., the general shift to low arousal, a simple habituation hypothesis could not account for the pattern of results observed in their four groups. This pattern was linked to both the training procedures used and to the subjects' baseline frontalis EMG levels. The pattern associated with the differential effects of the training methods on the low and high arousal groups was inconsistent, in terms of the direction and magnitude of change, with an habituation effect or the effects of non-specific conditions. If there had been a general increase in alpha rhythm over the pre-training to the posttraining sessions and greater levels of alpha EEG than theta EEG activity at the posttraining session, this would have been an indication of a global relaxation response due to the non-specific conditions. However, this type of systematic variation of alpha rhythm
levels did not occur between the pretraining and post-
training baseline sessions. Sittenfeld, Budzynski and
Stoyva (1976) interpreted this as indicating,

\[
\text{... that subjects were not simply producing a global relaxation response, but rather a physiological condition reflecting the feedback reinforcement contingencies, i.e., changes mainly in frontal EMG and theta outputs. (p. 43)}
\]

Groups that had acquired the ability to enhance theta rhythm levels through their training would be expected to produce high posttraining levels of theta EEG activity and comparatively lower levels of alpha rhythm activity. Groups that had not acquired the ability to enhance theta rhythm levels over the training sessions, could be expected to show increases in alpha activity or possibly even decreases at the posttraining recording sessions. Increases in alpha rhythm activity would be indicative of habituation (Lynch and Paskewitz, 1971) and/or a global relaxation response. Decreases in alpha rhythm levels, without a corresponding increase in theta rhythm levels, would suggest that the subjects' level of arousal had increased over sessions -- perhaps due to frustration or negative expectations about the success of the training. A recent discussion of the issues related to control groups in biofeedback research may be found in Hatch (1982).
III. RESULTS

The following statistical tests were performed for the theta EEG, frontalis EMG, and alpha EEG measurements:

(a) a 2 X 3 X 2 analysis of the variance with repeated measures on the third factor utilizing an unweighted means analysis (Winer, 1971, p. 337);
(b) simple-simple main effects analyses;
(c) Scheffe tests for the six groups at the post-training baseline condition;
(d) Pearson product-moment correlation coefficients for the theta EEG and frontalis EMG, the theta EEG and alpha EEG, and the frontalis EMG and alpha EEG measurements; and
(e) Pearson product-moment correlation coefficients for the pretraining and posttraining relationship for theta EEG, frontalis EMG and alpha EEG variables.

Statistical tests were utilized for two complimentary purposes: the first was to test the hypotheses concerning the relationship of physiological arousal to the effectiveness of the training methods on teaching subjects to enhance theta rhythm levels; and, the second was a post hoc analysis of the data to clarify the above relationship and to generate hypotheses to be tested in future investigations. The analysis of variance and simple-simple main effects analyses were employed to test the research hypotheses. The post hoc analyses involved the use of the Pearson product moment correlation and the Scheffe tests.
Analysis of Theta EEG Activity

Analysis of Variance

The analysis of variance yielded statistically significant main effects for the baseline arousal variable \( (F = 4.78, \ p = .03475) \) and for the pre/postrecording sessions variable \( (F = 4.87, \ p = .03317) \). The main effect for the training methods variable and the interaction effects were not statistically significant. (Refer to Table II., Table III., and Figure I.)

Simple-Simple Main Effects Analysis

The analysis of variance indicated that the three factor interaction of Baseline Arousal X Training Methods X Pre/Posttraining Recording Sessions yielded an \( F = 1.41, \ p = .260 \). Simple-simple main effects analyses were performed to clarify the nature of the significant main effects, which indicated that there were differences between the low and high arousal groups across the pre/posttraining recording sessions, and differences between the pretraining and posttraining levels of theta rhythm activity. The simple-simple main effects analysis of the interaction of the baseline arousal level, training methods and the pre/postrecording sessions variables revealed that the low arousal one-phase EMG/EMG groups was associated with an
<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arousal</td>
<td>1</td>
<td>782.35</td>
<td>782.35</td>
<td>4.78</td>
<td>.035</td>
<td>5.62 *</td>
</tr>
<tr>
<td>Training</td>
<td>2</td>
<td>405.37</td>
<td>202.68</td>
<td>1.24</td>
<td>.305</td>
<td>0.71</td>
</tr>
<tr>
<td>Arousal X Training</td>
<td>2</td>
<td>245.419</td>
<td>122.71</td>
<td>0.75</td>
<td>.514</td>
<td>0.00</td>
</tr>
<tr>
<td>Error/Between</td>
<td>30</td>
<td>4914.860</td>
<td>163.829</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre/Post</td>
<td>1</td>
<td>578.858</td>
<td>578.858</td>
<td>4.87</td>
<td>.033</td>
<td>4.20 *</td>
</tr>
<tr>
<td>Arousal X Pre/Post</td>
<td>1</td>
<td>1.530</td>
<td>1.530</td>
<td>0.01</td>
<td>.907</td>
<td>0.00</td>
</tr>
<tr>
<td>Training X Pre/Post</td>
<td>2</td>
<td>4.472</td>
<td>2.236</td>
<td>0.02</td>
<td>.982</td>
<td>0.00</td>
</tr>
<tr>
<td>Arousal X Training X Pre/Post</td>
<td>2</td>
<td>334.378</td>
<td>167.189</td>
<td>1.41</td>
<td>.260</td>
<td>0.01</td>
</tr>
<tr>
<td>Error/Within</td>
<td>30</td>
<td>3567.200</td>
<td>118.907</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>10834.400</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = statistically significant
### TABLE III

**Theta EEG (Mean Percent): Summary Table of Baseline Arousal Level X Pre/Posttraining Recording Sessions**

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Arousal</td>
<td>18.16</td>
<td>23.24</td>
</tr>
<tr>
<td>High Arousal</td>
<td>10.91</td>
<td>16.54</td>
</tr>
</tbody>
</table>
TABLE IV

SIMPLE-SIMPLE MAIN EFFECTS ANALYSIS.

Theta EEG (Mean Percent)

Baseline Arousal X Training Methods X Pre/Posttraining Recording (A X B X C) Interaction

<table>
<thead>
<tr>
<th>Level</th>
<th>Obtained F</th>
<th>Critical Value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>C @ ab1</td>
<td>2.233</td>
<td>2.18</td>
<td>.16</td>
</tr>
<tr>
<td>C @ ab2</td>
<td>1.429</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ ab3</td>
<td>0.015</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a2b1</td>
<td>0.262</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a2b2</td>
<td>0.178</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a2b3</td>
<td>3.075</td>
<td>3.07</td>
<td>.08</td>
</tr>
</tbody>
</table>
FIGURE I. Theta EEG Measurements for the Low Arousal and High Arousal Groups at the Pretraining and Post-training Recording Sessions
**TABLE V**

**Theta EEG (Mean Percent): Summary Table of Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions**

<table>
<thead>
<tr>
<th></th>
<th>EMG/EMG</th>
<th></th>
<th>EMG/EEG</th>
<th></th>
<th>EEG/EEG</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low Arousal</strong></td>
<td>15.81</td>
<td>25.22</td>
<td>15.39</td>
<td>22.92</td>
<td>25.54</td>
<td>21.82</td>
</tr>
<tr>
<td><strong>High Arousal</strong></td>
<td>7.06</td>
<td>10.28</td>
<td>13.37</td>
<td>16.02</td>
<td>12.56</td>
<td>24.66</td>
</tr>
</tbody>
</table>
FIGURE II. Theta EEG Measurements for Each of the Training Methods Used with the Low Arousal and High Arousal Groups at the Pretraining and Posttraining Recording Sessions
increase in theta EEG activity \((F = 2.233, p = .16)\); and that the high arousal one phase EEG/EEG group was also associated with an increase in theta EEG activity \((F = 3.075, p = .08)\). (Refer to Table IV., Table V., and Figure II.) These increases were not considered to be statistically significant.

**Scheffe Test**

The Scheffe tests revealed that at the posttraining session that: (a) the high arousal one-phase EEG/EEG training group differed significantly from the high arousal one phase EMG/EMG training group \((F = 3.439, p = .070)\); (b) the low arousal one-phase EMG/EMG group differed significantly from the high arousal one-phase EMG/EMG training group \((F = 4.09, p = .049)\); (c) the low arousal two-phase EMG/EEG group differed significantly from the high arousal one-phase EMG/EMG training group \((F = 2.92, p = .094)\); and (d) the low arousal training groups did not differ significantly from each other. In the absence of statistically significant simple-simple main effects, these posttraining differences cannot be attributed to the influence of the interaction of the subjects level of arousal and the training methods. It is likely that these differences reflected differences that were present prior to training. (Refer to Table V., Table VI. and Figure II.)
### TABLE VI

**Theta EEG Scheffe Test for Low and High Arousal Training Groups at the Posttraining Recording Sessions**

<table>
<thead>
<tr>
<th></th>
<th>Low</th>
<th>Low</th>
<th>Low</th>
<th>High</th>
<th>High</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG/EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- **Low EMG/EMG**: *p=.049
- **Low EMG/EEG**: *p=.094
- **Low EEG/EEG**: 
- **High EMG/EMG**: *p=.070
- **High EMG/EEG**: 
- **High EEG/EEG**: 

---

*Note: The table shows the comparison of EMG, EEG, and their combinations between low and high arousal training groups at posttraining recording sessions.*
Scheffe tests were also made on the comparison of the pooled low arousal training groups with the individual high arousal training groups. There were no statistically significant differences.

**Analysis of Frontalis EMG Activity**

**Analysis of Variance**

The analysis of variance yielded statistically significant main effects for the Baseline Arousal variable \( F = 39.46, \ p = .00001 \) and for the Pre/Posttraining Recording session variable \( F = 31.96, \ p = .00003 \). Statistically significant interaction effects were obtained for the baseline arousal level and pre/postrecording session variable interaction \( F = 21.64, \ p = .00018 \) and the training method and pre/posttraining recording sessions interaction \( F = 3.61, \ p = .03844 \). (Refer to Table VII., Table VIII., and Figure III.)

The remaining main effects and interactions were not statistically significant. The three factor interaction of the Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Session variables yielded a non-significant F-ratio of 1.588 \( (p = .21971) \). (Refer to Table VII. and Table IX.) This interaction was examined with a simple-
### TABLE VII

**Frontalis EMG: Analysis of Variance Summary Table.**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arousal</td>
<td>1</td>
<td>37.553</td>
<td>37.553</td>
<td>39.46</td>
<td>.00001</td>
<td>39.75 *</td>
</tr>
<tr>
<td>Training</td>
<td>2</td>
<td>1.719</td>
<td>0.859</td>
<td>0.90</td>
<td>.58128</td>
<td>0.00</td>
</tr>
<tr>
<td>Arousal X Training</td>
<td>2</td>
<td>2.269</td>
<td>1.134</td>
<td>1.19</td>
<td>.31782</td>
<td>0.40</td>
</tr>
<tr>
<td>Error/Between</td>
<td>30</td>
<td>28.551</td>
<td>0.952</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre/Post</td>
<td>1</td>
<td>7.158</td>
<td>7.158</td>
<td>31.96</td>
<td>.00003</td>
<td>7.58 *</td>
</tr>
<tr>
<td>Arousal X Pre/Post</td>
<td>1</td>
<td>4.842</td>
<td>4.842</td>
<td>21.64</td>
<td>.00018</td>
<td>5.06 *</td>
</tr>
<tr>
<td>Training X Pre/Post</td>
<td>2</td>
<td>1.613</td>
<td>0.806</td>
<td>3.61</td>
<td>.03844</td>
<td>1.28 *</td>
</tr>
<tr>
<td>Arousal X Training X Pre/Post</td>
<td>2</td>
<td>0.711</td>
<td>.0355</td>
<td>1.59</td>
<td>.21971</td>
<td>0.29</td>
</tr>
<tr>
<td>Error/Within</td>
<td>30</td>
<td>6.712</td>
<td>.0224</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>91.124</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = statistically significant
### TABLE VIII

Frontalis EMG (Mean Microvolts): Summary Table of Baseline Arousal Level X Pre/Posttraining Recording Sessions

<table>
<thead>
<tr>
<th>Arousal Level</th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Arousal</td>
<td>0.897</td>
<td>0.847</td>
</tr>
<tr>
<td>High Arousal</td>
<td>2.92</td>
<td>1.73</td>
</tr>
</tbody>
</table>
FIGURE III. Frontalis EMG Measurements for the Low Arousal and High Arousal Groups at the Pretraining and Posttraining Recording Sessions
## TABLE IX

Frontalis EMG (Mean Microvolts): Summary Table of Baseline Arousal Level X Training Methods X Pre/Post-training Recording Sessions

<table>
<thead>
<tr>
<th></th>
<th>EMG/EMG</th>
<th></th>
<th>EMG/EEG</th>
<th></th>
<th>EEG/EEG</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low Arousal</strong></td>
<td>0.92</td>
<td>0.73</td>
<td>1.11</td>
<td>0.90</td>
<td>0.84</td>
<td>0.90</td>
</tr>
<tr>
<td><strong>High Arousal</strong></td>
<td>3.01</td>
<td>1.25</td>
<td>2.69</td>
<td>1.52</td>
<td>3.08</td>
<td>2.54</td>
</tr>
</tbody>
</table>
simple main effects analysis in order to clarify the nature of the main effects and two way interactions.

**Simple-Simple Main Effects Analysis:**

The simple-simple main effects analysis of the three way interaction of the Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Session variables revealed that the high arousal one-phase EMG/EMG training group (F = 41.250, p < .01) and the high arousal two-phase group (F = 18.302, p < .01) were associated with statistically significant decreases in frontalis EMG activity. None of the low arousal training groups approached significant levels of change, and the change of the high arousal one-phase EEG/EEG training group was also non-significant. (Refer to Table IX., Table X. and Figure IV.)

**Scheffe Tests**

Scheffe tests were performed to compare the distribution of the training groups at the posttraining baseline recording session. As may be seen in Table IX., Table XI., and Figure IV., the high arousal groups were quite similar in their pretraining baseline levels of frontalis EMG. However, an examination of the distribution of these groups at the posttraining baseline sessions (Table XI.) indicated that there were differences in their frontalis EMG levels. The
TABLE X

SIMPLE-SIMPLE MAIN EFFECTS ANALYSES.

EMG (Mean Microvolts)

Baseline Arousal X Training Methods X Pre/Post-training Recording (A X B X C) Interaction

<table>
<thead>
<tr>
<th>Level</th>
<th>Obtained F</th>
<th>Critical Value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>C @ a1b1</td>
<td>0.487</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a1b2</td>
<td>0.570</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a1b3</td>
<td>0.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C @ a2b1</td>
<td>41.250</td>
<td>7.56</td>
<td>.01</td>
</tr>
<tr>
<td>C @ a2b2</td>
<td>18.302</td>
<td>7.56</td>
<td>.01</td>
</tr>
<tr>
<td>C @ a2b3</td>
<td>0.991</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
FIGURE IV. Frontalis EMG Measurements for Each of the Training Methods used with the Low Arousal and High Arousal Groups at the Pretraining and Posttraining Recording Sessions
**TABLE XI**

Frontalis EMG Scheffe Test for Low and High Arousal Training Groups at the Posttraining Recording Sessions

<table>
<thead>
<tr>
<th></th>
<th>Low EMG/EMG</th>
<th>Low EMG/EEG</th>
<th>Low EEG/EEG</th>
<th>High EMG/EMG</th>
<th>High EMG/EEG</th>
<th>High EEG/EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low EMG/EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*p = .005</td>
</tr>
<tr>
<td>Low EMG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*p = .009</td>
</tr>
<tr>
<td>Low EEG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*p = .070</td>
</tr>
<tr>
<td>High EMG/EMG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*p = .036</td>
</tr>
<tr>
<td>High EMG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*p = .091</td>
</tr>
<tr>
<td>High EEG/EEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
high arousal subjects that received the one-phase EEG/EEG training group had the highest posttraining frontalis EMG scores. The group that received the two-phase EMG/EEG training produced the next highest frontalis EMG level. The group that received the one-phase EMG/EMG training yielded the lowest posttraining frontalis EMG level of the high arousal groups.

The Scheffe tests, as may be seen in Table XI. and Figure IV., indicated that the high arousal one-phase EEG/EEG group was significantly different from both the high arousal one-phase EMG/EMG training group (F = 4.72, p = .0359) and the high arousal two-phase EMG/EEG group (F = 2.974, p = .0913). The high arousal one-phase EMG/EMG group was not significantly different from the high arousal two-phase EMG/EEG group.

The low arousal groups were quite similar to each other at the pretraining and the posttraining baseline sessions, as may be observed in Table XI. and Figure IV. The Scheffe tests did not reveal any significant differences between the low arousal groups at the posttraining baseline session.

The Scheffe tests were used to compare the low arousal and the high arousal training groups at the posttraining recording sessions. The low arousal training groups were pooled for this comparison. The pooled low arousal training groups
differed significantly from only the high arousal one-phase EEG/EEG training group ($F = 3.991$, $p = .0166$).

The simple-simple main effects analyses and the Scheffe tests indicate that only the high arousal subjects who received some form of frontalis EMG training were able to significantly decrease the frontalis EMG levels from the pretraining to the posttraining sessions. The subjects who received the greatest amount of frontalis EMG training -- the one-phase EMG/EMG group -- exhibited the greatest decrease. The low arousal subjects did not produce any significant decreases with any of the training methods. They maintained their already low pretraining level during the posttraining sessions.

**Analysis of Alpha EEG Activity**

**Analysis of Variance**

The analysis of variance of the alpha EEG data revealed only one statistically significant main effect. (Refer to Table XII.) The pre/posttraining recording sessions condition yielded a significant main effect ($F = 5.064$, $p = .0301$). As may be seen in Table XIII. and Figure V., this indicated that the level of the alpha EEG activity decreased from the pretraining to the posttraining baseline sessions. Table
### TABLE XII

**Alpha EEG: Analysis of Variance Summary Table**

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arousal</td>
<td>1</td>
<td>14.969</td>
<td>14.969</td>
<td>0.08</td>
<td>.775</td>
<td>0.00</td>
</tr>
<tr>
<td>Training</td>
<td>2</td>
<td>618.827</td>
<td>309.414</td>
<td>1.66</td>
<td>.205</td>
<td>2.53</td>
</tr>
<tr>
<td>Arousal X Training</td>
<td>2</td>
<td>85.848</td>
<td>42.924</td>
<td>0.23</td>
<td>.798</td>
<td>0.00</td>
</tr>
<tr>
<td>Error/Between</td>
<td>30</td>
<td>5580.610</td>
<td>186.020</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre/Post</td>
<td>1</td>
<td>402.613</td>
<td>402.613</td>
<td>3.06</td>
<td>.030</td>
<td>3.36*</td>
</tr>
<tr>
<td>Arousal X Pre/Post</td>
<td>1</td>
<td>195.691</td>
<td>195.691</td>
<td>2.46</td>
<td>.124</td>
<td>1.20</td>
</tr>
<tr>
<td>Training X Pre/Post</td>
<td>2</td>
<td>105.455</td>
<td>52.727</td>
<td>0.66</td>
<td>.527</td>
<td>0.00</td>
</tr>
<tr>
<td>Arousal X Training X Pre/Post</td>
<td>2</td>
<td>143.521</td>
<td>71.760</td>
<td>0.90</td>
<td>.581</td>
<td>0.00</td>
</tr>
<tr>
<td>Error/Within</td>
<td>30</td>
<td>2385.060</td>
<td>79.502</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>9532.59</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = statistically significant
### TABLE XIII

**Alpha EEG (Mean Percent): Summary Table of Baseline Arousal Level X Pre/Posttraining Recording Sessions**

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low Arousal</strong></td>
<td>77.29</td>
<td>69.39</td>
</tr>
<tr>
<td><strong>High Arousal</strong></td>
<td>74.76</td>
<td>73.53</td>
</tr>
</tbody>
</table>
FIGURE V. Alpha EEG Measurements for the Low Arousal and High Arousal Groups at the Pretraining and Posttraining Recording Sessions
XIV. and Figure VI. illustrate the changes associated with the specific low and high arousal training groups.

Correlational Analyses

Pearson product moment correlation coefficients were calculated for the relationship between theta EEG and frontalis EMG measurements. As may be observed in Table XV., the overall correlation coefficient for all subjects collapsed across all independent variables was -.275, p < .01. The complete correlation matrix may be found in Table XVI. The correlation coefficients for all subjects at the pretraining recording condition, as may be seen in Table XVI., was a -.418, p < .005; and at the posttraining baseline session was a -.115. The latter coefficient was not significant.

Pearson product moment correlation coefficients were calculated for the relationship between theta EEG and alpha EEG measurements. These coefficients may be found in Table XVII. The correlation coefficient for all subjects at the pretraining baseline was a -.297, p < .05; and at the posttraining baseline sessions was a -.485, p < .005.

The Pearson product moment correlation coefficients for the relationship between frontalis EMG and alpha EEG may be found in Table VIII. The correlation coefficient for all subjects at the pretraining recording sessions was a -.135;
TABLE XIV

Alpha EEG (Mean Percent): Summary Table of Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions

<table>
<thead>
<tr>
<th></th>
<th>EMG/EMG</th>
<th></th>
<th>EMG/EEG</th>
<th></th>
<th>EEG/EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Arousal</td>
<td>80.07</td>
<td>69.44</td>
<td>74.63</td>
<td>66.04</td>
<td>77.20</td>
</tr>
<tr>
<td>High Arousal</td>
<td>80.99</td>
<td>76.39</td>
<td>66.48</td>
<td>71.74</td>
<td>77.24</td>
</tr>
</tbody>
</table>
FIGURE VI. Alpha EEG Measurements for Each of the Training Methods Used with the Low Arousal and High Arousal Groups at the Pretraining and Posttraining Recording Sessions
<table>
<thead>
<tr>
<th>Correlation</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Theta - EMG</td>
<td>-0.275</td>
</tr>
<tr>
<td>Theta - Alpha</td>
<td>-0.439</td>
</tr>
<tr>
<td>EMG - Alpha</td>
<td>-0.013</td>
</tr>
</tbody>
</table>
**TABLE XVI**

Pearson Product Moment Correlation Matrix

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th></th>
<th></th>
<th>Posttraining</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EMG</td>
<td>Theta</td>
<td>Alpha</td>
<td>EMG</td>
<td>Theta</td>
<td>Alpha</td>
</tr>
<tr>
<td>EMG</td>
<td>+1.00</td>
<td>-.418</td>
<td>-.135</td>
<td>+.717</td>
<td>-.119</td>
<td>-.136</td>
</tr>
<tr>
<td>Pretrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theta</td>
<td></td>
<td>+1.00</td>
<td>-.297</td>
<td>-.156</td>
<td>+.278</td>
<td>-.134</td>
</tr>
<tr>
<td>Pretrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha</td>
<td>+1.00</td>
<td></td>
<td>-.073</td>
<td>-.019</td>
<td>+.383</td>
<td></td>
</tr>
<tr>
<td>Pretrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMG</td>
<td>+1.00</td>
<td></td>
<td>-.115</td>
<td>+.081</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posttrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theta</td>
<td>+1.00</td>
<td></td>
<td>-.485</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posttrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha</td>
<td>+1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posttrng.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
TABLE XVII

Pearson Product Moment Correlations for Theta EEG-Frontalis EMG, Theta EEG-Alpha EEG, and Frontalis EMG-Alpha EEG at the Pretraining and the Posttraining Recording Sessions

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Theta - EMG</td>
<td>-.418</td>
<td>-.115</td>
</tr>
<tr>
<td>Theta - Alpha</td>
<td>-.297</td>
<td>-.485</td>
</tr>
<tr>
<td>EMG - Alpha</td>
<td>-.135</td>
<td>+.081</td>
</tr>
</tbody>
</table>
and at the posttraining recording session was a -.081.
Neither of these coefficients was statistically significant.

Pearson product moment correlation coefficients were calculated for the relationship between the pretraining and posttraining theta EEG, frontalis EMG, and alpha EEG levels. As may be seen in Table XVIII., the coefficient for theta EEG was a +.278, the frontalis EMG was a +.717, the alpha EEG measurement was a +.383, p < .01.
**TABLE XVIII**

Pearson Product Moment Correlation of Theta EEG, Frontalis EMG, and Alpha EEG for the Pretraining and Posttraining Recording Sessions

<table>
<thead>
<tr>
<th>Theta</th>
<th>EMG</th>
<th>Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>.278</td>
<td>.717</td>
<td>.383</td>
</tr>
</tbody>
</table>
The primary objectives of the present experiment were to:

(1) determine whether subjects could learn to enhance their theta EEG levels through biofeedback training methods; (2) replicate the findings of Sittenfeld, Budzynski and Stoyva (1976) that the subject's state of physiological arousal -- as indicated by frontalis EMG levels -- must be taken into account when selecting a method of training subjects to enhance theta EEG levels; and, (3) to extend their findings by testing the effectiveness of an additional training method -- not included in their investigation. A secondary post hoc consideration, concerning the reported inverse relationship between frontalis EMG and theta EEG activity, arose when the correlational analyses revealed discrepancies between the results of the present experiment and those of Sittenfeld, Budzynski and Stoyva (1976).

From the context of the results of the Sittenfeld, Budzynski and Stoyva experiment (1976), I had hypothesized that the subjects' level of physiological arousal -- as indicated by the pretraining baseline level of the frontalis EMG measurements -- would influence the effectiveness of the three training methods for teaching the subjects to enhance theta EEG activity. Specifically, it had been predicted that the low arousal subjects would significantly increase their
theta rhythm activity only when trained with the one-phase EEG/EEG biofeedback method. It was also predicted that the high arousal subjects would demonstrate significant increases in theta rhythm level only when presented with the one-phase EMG/EMG or the two-phase EMG/EEG biofeedback training methods. None of these hypotheses were supported by the results of the present experiment. The analysis of variance, the simple-simple main effects analyses, the Scheffe tests and the correlational analyses -- to be discussed shortly -- revealed a lack of concordance with the results of the Sittenfeld, Budzynski and Stoyva experiment (1976). (Refer to the comparison table in the Appendix.)

It had also been assumed that the significant inverse correlation between theta EEG and frontalis EMG levels would be replicated at the posttraining recording sessions. It was not. This lack of verification was of concern on three counts: (1) the existence of this inverse relationship represented the logical core of Sittenfeld, Budzynski and Stoyva's (1976) interpretation that their results indicated differential effectiveness of the one-phase EEG/EEG procedure with the low arousal subjects and of the two-phase EMG/EEG procedure with the high arousal subjects; (2) it was the rationale for the inclusion of the one-phase EMG/EMG training method and its' predicted effectiveness with the high arousal subjects in the present experiment; and (3) the recent contradictions of Sittenfeld, Budzynski and Stoyva's
assertation that frontalis EMG levels are linked to arousal and that the effects of frontalis EMG relaxation training generalizes to other muscle groups (Budzynski and Stoyva, 1969).

Interpretation of Theta EEG Results

It had been hypothesized that the analysis of variance would yield a statistically significant Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions interaction. However, the interaction was not significant. The simple-simple main effects analyses of this three factor interaction were performed to ascertain whether the low arousal and high arousal groups responded differentially to the three biofeedback training methods. The simple-simple main effects analysis detected only two groups that were associated with increases in theta rhythm activity. As may be observed in Table IV. and Figure II., the low arousal one-phase EMG/EMG group did not approach the critical value of F until p = .16. The high arousal one-phase EEG/EEG group was associated with an increase in theta rhythm levels when the critical value reached p = .08. Since these simple-simple main effects could not be considered as statistically significant, they must be attributed to chance. The simple-simple main effects analysis reflected the degree of change in theta EEG levels from the pre-training to the posttraining recording sessions, but did not
indicate anything about the differences between the groups at the posttraining recording sessions. The Scheffe tests were used to detect these differences. As may be seen in Table V., Table VI. and Figure II., the high arousal one-phase EEG/EEG group started at the pretraining sessions at the second lowest theta level and finished at the post-training sessions at the second highest theta level. The Scheffe tests demonstrated that this high arousal one-phase EEG/EEG group differed significantly at the posttraining baseline from the high arousal one-phase EMG/EMG group, and that it did not differ significantly from the low arousal groups. It is of note that this group did not differ significantly at the posttraining sessions from the low arousal groups, which were expected to have had naturally occurring higher levels of theta rhythms by virtue of the inverse relationship between theta rhythm and frontalis EMG levels. If the simple-simple main effect had been significant at \( p < .05 \), this would have suggested that the high arousal groups were able to enhance theta only when they received the one-phase EEG/EEG training method. However, since this was not the case, the posttraining differences cannot be attributed to the differential effect of the one-phase EEG/EEG training on the high arousal subjects. The Scheffe tests also found that there were no significant differences between the low arousal groups at the post-training recording sessions. This result, coupled with the simple-simple main effects analysis, suggests that there
were no differential training effects associated with the low arousal group. These results for the low and high arousal groups are not in conformity with the predictions made from the context of the Sittenfeld, Budzynski and Stoyva (1976) findings. Since the three way interaction was non-significant \( p = .26 \) and the simple-simple main effects were not found to be significant at low alpha probabilities \( p < .05 \), it must be considered that the present experiment may have lacked the power to detect the differential treatment effects; or that there were no differential treatment effects to be detected. However, when considered in conjunction with the frontalis EMG and the alpha EEG results, the correlational analyses, and the recent research on the relationship of frontalis EMG to physiological arousal, some specific alternative interpretations may be reasonably considered.

**Interpretation of Frontalis EMG Results**

It was predicted that the Baseline Arousal Level X Training Methods X Pre/Posttraining Recording Sessions interaction would be significant. This interaction was not significant.

As may be seen in Table VII., Table VIII, and Figure III., there were significant main effects for the baseline arousal level and the pre/posttraining recording sessions factors. The following interactions were found to be statistically
significant: (1) Baseline Arousal Level X Pre/Posttraining Recording Session, and (2) Training Methods X Pre/Posttraining Recording Sessions. The simple-simple main effects analysis of the three factor interaction disclosed that only two of the training groups demonstrated significant effects. As may be seen in Table X., Table IX., and Figure IV., the high arousal one-phase EMG/EMG method and the high arousal two-phase EMG/EEG method each produced significant decreases in frontalis EMG levels. The high arousal one-phase EEG/EEG group produced a non-significant decrease. None of the low arousal groups produced a significant decrease in frontalis EMG levels. This was most probably due to a scaling effect that made it unlikely that the low arousal subjects, who started the pretraining recording sessions with extremely low frontalis levels, could realize further progress with training. The Scheffe tests indicated that the high arousal one-phase EEG/EEG group differed significantly from all of the other groups at the posttraining recording sessions. Its' posttraining frontalis EMG levels was significantly higher than the others. The other groups were not significantly different from each other at the posttraining baseline sessions. Thus, it may be inferred that the high arousal groups that received frontalis EMG relaxation training were able to reduce their posttraining levels of frontalis EMG to a level comparable to that of the low arousal groups. This is hardly a surprising finding, and confirms the prediction about differential effects of EMG
training on the reduction of frontalis EMG levels with the high arousal subjects.

Thus, the high arousal subjects that received the one-phase EMG/EMG or the two-phase EMG/EEG feedback training were able to demonstrate statistically significant levels of control over the reduction of frontalis EMG activity. However, these same high arousal groups did not demonstrate statistically significant increases in theta rhythm activity. Conversely, the groups that exhibited increases in theta rhythm activity did not produce statistically significant decreases in frontalis EMG levels. (Compare Figure II. with Figure IV.) The question, then, is whether frontalis EMG levels are related to theta EEG levels; and, whether the frontalis EMG level is indicative of the state of low arousal that has been associated with high levels of theta rhythms.

Frontalis EMG -- Arousal and Generalization

A central contention of the work of Budzynski and Stoyva has been that the frontalis EMG levels are linked to states of physiological and emotional arousal (Stoyva, 1970). They also have stated that the frontalis muscles represent "key" muscles, in that, when frontalis EMG relaxation training is administered -- the relaxation generalizes to other muscle groups and thereby reduces the subject's state of
physiological and emotional arousal (Sittenfeld, Budzynski and Stoyva, 1976). A review of the biofeedback literature in 1981 by Thompson, Haber and Tearnan concluded that frontalis EMG relaxation does not generalize to other muscle groups. They also challenged the findings that frontalis EMG relaxation is linked to a lowering of general arousal. At present, the research literature allows one to safely conclude that frontalis EMG biofeedback relaxation training does reduce tension in the frontalis muscles. However, the current literature does not support the earlier findings concerning the ability of frontalis EMG feedback training to induce a state of lowered physiological arousal (Alexander, 1975; Alexander, White and Wallace, 1977; Alexander and Smith, 1979). In fact, in a study by Shirley, Burish and Rowe (1982) where frontalis EMG biofeedback training was compared with multiple site EMG biofeedback and a no feedback control in the reduction of arousal, it was concluded that neither feedback procedure was effective in reducing anxiety. However, multiple site EMG feedback was effective in reducing several indices of autonomic arousal while frontalis EMG feedback was ineffective. They concluded that,

frontalis EMG feedback was not an effective procedure for controlling stress, but that EMG feedback can be effective in reducing self-reported anxiety and autonomic arousal if a multiple-site feedback procedure is employed (p. 167).
They also reported that recent research indicates that the three main assumptions about the stress-reducing effectiveness of frontalis EMG biofeedback are largely unfounded (Surwit and Keefe, 1978). In summarizing the research, they indicated that the first assumption -- that frontalis EMG reductions generalize to other muscle groups -- has not been supported (Davis, 1980; Glaus, 1979; Fridlund, Fowler and Pritchard, 1980). Shirley and her colleagues (1982) reported that although frontalis EMG biofeedback training produced reliable decreases in the EMG levels of the target muscle group, these reductions were not reliably associated with muscular decreases in the EMG levels of other muscle groups, even those proximal to the frontalis area. The second assumption, based on Budzynski and Stoyva's (1969) finding that frontalis EMG reductions were associated with reductions in autonomic arousal, also was not supported by the research literature (Burish and Horn, 1981). Shirley and her associates (1982) concluded that decreases in frontalis EMG activity do not reliably produce reductions in autonomic indices -- a position empirically supported by their own experiments. The third assumption, that changes in frontalis EMG were associated with changes in the subjects' subjective reports of anxiety or tension, was not supported by the research literature (Kuhlman and Kaplan, 1979). In their critique of Budzynski and Stoyva's early theorizing, they indicated that the concept of a "key" muscle that becomes very tense under stress and whose relaxation
produces a general relaxation effect may not be an inaccurate concept. However, they asserted that,

the key muscle site is not always or even usually the frontal site but instead varies from person to person. If the frontal muscles are not the key muscles in most individuals, and since decreases in frontal EMG do not produce EMG decreases in other muscle groups, frontal EMG biofeedback will generally not result in EMG decreases in the 'key' muscles (Shirley, Burish and Rowe, 1982, p. 169).

Their recommendation was to utilize multiple EMG feedback sites. This would increase the likelihood that the "key" stress-sensitive muscle groups would reduce their levels of activity. This reduction in EMG levels would then be more likely to be associated with a decrease in autonomic and subjective arousal. This hypothesis was partially confirmed by their experiment that demonstrated that multiple site EMG feedback decreased signs of autonomic arousal but not reports of situational anxiety. Weinman, Semchuk, Gaebe and Matthew (1983) used frontalis EMG relaxation training as a non-specific method to reduce overall stress and to facilitate coping in a study that evaluated the effect of frontalis EMG biofeedback and relaxation training in the treatment of anxiety. The "non-specific" factors that may contribute to the effectiveness of biofeedback involve cognitive processes that enable subjects to focus their attention on the relaxation of the muscles and away from the stress associated with tension (Meichenbaum, 1976; and Goldberg, Weller and Blittner, 1982). Weinman and her
colleagues (1983) summarized this view. They stated that the cognitive control may alter emotional arousal and facilitate coping. It was felt that successful EMG training could help a subject to call upon existing relaxation skills to help counteract the arousal and physiological reactions to stress and tension while enhancing an individual's belief of self, mastery, and competence (p. 193).

From their orientation they interpreted the effects of frontalis EMG training as being due to cognitive factors, such as the perception of control, rather than to direct effects of frontalis EMG relaxation on a state of physiological arousal. Thus, in light of these reports that were published after the present investigation was completed, it is very likely that the selection of frontalis EMG biofeedback training as a means of reducing the subjects' levels of arousal and thereby increasing theta rhythm activity may have been inappropriate.

**Interpretation of Theta Rhythm and Frontalis EMG Relationship:**

It had been hypothesized by Sittenfeld and her colleagues (1976) that an inverse relationship existed between theta EEG levels and frontalis EMG activity. This, as indicated earlier, was based on "qualitative" observations of subjects by Budzynski and Stoyva (1969). The confirmation of this
hypothesis by Sittenfeld, Budzynski and Stoyva (1976) represented the first quantitative assessment of this relationship. They reported a significant correlation of -.53, p < .05. This coefficient was based on the theta rhythm and frontalis EMG measurements made during the posttraining recording sessions. No other correlations were presented. As may be found in Table XV. and Table XVII., correlations were calculated in the present experiment for the theta rhythm and frontalis EMG measurements at the pretraining baseline recording sessions, the posttraining baseline recording sessions, and for the combined data of the pretraining and posttraining baseline recording sessions. The present experimenter was unable to replicate the significant correlation reported by Sittenfeld, Budzynski and Stoyva (1976) at the posttraining recording sessions. The overall correlation coefficient for the combined pretraining and posttraining recording sessions was a -.275, p < .01. The pretraining recording correlation coefficient was a -.418, p < .005. The posttraining recording correlation coefficient was a non-significant -.115. Thus, in the present experiment, theta EEG and frontalis EMG levels were significantly related prior to the biofeedback training sessions. After the biofeedback training, the relationship deteriorated. The significant overall correlation was due to the contributions of the pretraining measurements. Additional correlations were calculated for each of the dependent variables to assess the relationship of the
pretraining recording and posttraining recording sessions. In this case, a high significant coefficient would indicate a high degree of covariation of these measurements. As may be observed in Table XVIII., the theta EEG data yielded a coefficient of +.278, \( p < .05 \). The frontalis EMG measurements produced a coefficient of +.717, \( p < .001 \); and the alpha EEG measurements demonstrated a coefficient of +.383, \( p < .025 \). The theta EEG coefficient indicates that there was little consistency demonstrated in the changes in theta rhythm activity over the pretraining to the posttraining recording sessions. This was in accord with the analysis of variance and the simple-simple main effects analysis results. The analysis of variance demonstrated that there was a significant increase in theta EEG levels over the pretraining and the posttraining recording sessions. However, the predicted differential training effects for the low and high arousal subjects that would have been indicative of learned control of theta EEG were not detected. This, coupled with the correlation of the pretraining and posttraining recording session theta measurements, suggested that the increases in theta EEG activity were more likely due to non-specific decreases in arousal rather than learned control of theta rhythm levels. The change in the correlation of the theta EEG and alpha EEG measurements from the pretraining to the posttraining recording sessions also suggested a general decrease in arousal rather than specific training effects. The theta EEG and alpha EEG correlation
was a -.297, \( p < .05 \) at the pretraining recording, and a 
-.485, \( p < .005 \) at the posttraining recording sessions.
(Refer to Table XVII.) The only significant main effect for 
the analysis of variance of the alpha EEG data was on the 
baseline recording variable. This significant main effect 
indicates that there was a significant overall decrease of 
alpha EEG activity from the pretraining to the posttraining 
recording sessions. These coefficients indicate that as 
theta EEG activity increased the alpha EEG activity de­
creased. The higher posttraining theta and alpha EEG levels 
indicate that this inverse relationship was considerably 
stronger at the posttraining sessions than at the pre­
training recording sessions. It had been predicted that if 
differential training effects were significant for theta 
rhythms, then there should have been a corresponding de­
crease in alpha for those groups. However, since the theta 
EEG simple-simple main effects analysis did not reveal a 
strong pattern of differential training effects -- the high 
arousal one-phase EEG/EEG training group was the only one 
that approached an acceptable level of significance -- the 
decreases in alpha were not linked to specific groups. 
Thus, this indicates a general non-specific decrease in 
arousal rather than one linked to training effects. The 
distinction between differential decreases in arousal as 
 opposed to a non-specific generalized state of arousal would 
also relate to the breakdown of the correlational relation­
ship between theta EEG and frontalis EMG activity that
occurred over the pretraining to the posttraining recording sessions.

An alternative explanation for the decrement of the inverse relationship from the pretraining to the posttraining sessions could be posited on the basis of differential training effects on the high arousal subjects. The inverse relationship between theta EEG and frontalis EMG could also be expected to decrease in magnitude over the pretraining to the posttraining recording sessions, rather than increase if the training methods were differentially effective. As may be observed in Table IV., Table V., Table IX., and Table X. when the two-phase EMG/EEG or the one-phase EMG/EMG training methods were employed, the frontalis EMG levels decreased significantly for high arousal subjects while theta EEG activity changed only slightly. On the other hand when one-phase EEG/EEG training was used with the high arousal subjects, the theta rhythm levels increased markedly and frontalis EMG activity showed only a slight change. This pattern of results, accompanied by a significant decline in the absolute value of the correlation coefficient suggests that biofeedback specific influences may have disrupted the initial pretraining inverse relationship.

The earlier qualitative observations of the inverse relationship had indicated, specifically, that when frontalis EMG levels became very low that increases in theta EEG
activity were likely to be observed (Budzynski and Stoyva, 1969). Thus, it would be expected that as subjects within the high arousal two-phase EMG/EEG and the one-phase EMG/EMG training groups moved in the direction of decreased frontalis EMG levels, theta EEG levels would be expected to increase naturally. Indeed this was the rationale underlying the explanation posited by Sittenfeld and her associates (1976) for the differential effectiveness of the two-phase EMG/EEG training with the high arousal subjects. It was also the rationale for the prediction, in the present experiment, that the one-phase EMG/EMG training method would be differentially effective with the high arousal subjects. If the subjects were not gaining control of theta EEG activity or if, as has been recently demonstrated, that frontalis EMG activity is not directly related to reductions in autonomic or subjective arousal, then a disruption of an existing relationship between theta rhythm and frontalis EMG would be a reasonable outcome of the feedback training methods -- particularly if the training involved frontalis EMG feedback. Instead of training subjects to reduce their levels of physiological arousal, the recent EMG research suggests that the effects of frontalis EMG feedback training are localized to the target muscles and have no direct effect on autonomic arousal (Alexander, 1975; Alexander, White and Wallace, 1977; Alexander and Smith, 1979; Fridlund, Fowler, and Pritchard, 1980; Glaus and Kotses, 1979; Davis, 1980; and Burish and Horn, 1981; and Thompson,
Haber, and Tearnan, 1981). Thus, it is possible that there were two different processes influencing the measurements of theta EEG and frontalis EMG in the pretraining and post-training recording sessions. (Refer to Table VII. and Table X.) In the pretraining recording sessions, the subjects had not yet been trained to regulate the frontalis muscle activity. The large F-ratio for the pre/posttraining recording sessions (F = 31.96, p = .00003) and the significant simple-simple main effects indicate that subjects had acquired high levels of control over the frontalis EMG levels by the time they had completed the training and were measured at the posttraining recording sessions. In the pretraining recording sessions, the subjects were asked to lie quietly for the two twenty-five minute recording periods. In the posttraining recording sessions, the subjects were instructed to try to regulate the EMG or brain wave activity without the benefit of the feedback signal. If the subjects had learned to decrease their frontalis EMG levels, then it is likely that the underlying variable in the posttraining session was different than that of the pretraining session. It is very possible that the underlying variable in the pretraining session was a generalized state of low physiological arousal that would have, as naturally occurring components, low levels of muscular tension -- including frontalis EMG levels -- and increased levels of theta activity at the lower end of the scale. This is a normal phenomenon that has been repeatedly
associated with both the non-descending and descending theta state. However, in the posttraining recording sessions, the subjects who had learned to regulate their frontalis EMG levels were likely to be exhibiting a localized differentiated relaxation of the frontalis muscles, and not a generalized reduction of physiological arousal that has been associated with increased theta rhythm levels. With this in mind, it is of interest to note that the two groups that were observed to have enhanced their theta EEG levels to some degree, as indicated by the simple-simple main effects analysis, were different than the groups that exhibited significant learned reductions of frontalis EMG activity. (Refer to Table IV. and Table IX.) The two groups that demonstrated some enhancement of the theta EEG levels were the low arousal one-phase EMG/EMG and the high arousal one-phase EEG/EEG groups. The two groups that exhibited a high level of control over decreases in frontalis EMG activity, according to the simple-simple main effects analysis, were the high arousal one-phase EMG/EMG and the high arousal two-phase EMG/EEG groups. It is also noteworthy, that the low arousal groups did not decrease their frontalis EMG activity as a result of training -- but maintained low pretraining frontalis EMG levels -- failed to demonstrate significantly higher posttraining theta rhythm levels than the high arousal subjects. (Refer to Figure II. and Table VI.)
The results of the analysis of variance, correlational analyses, Scheffe tests, and simple-simple main effects analyses did not confirm the research hypotheses. The fundamental purposes of this experiment were to determine: (1) whether biofeedback training could be used to teach human subjects to enhance their theta EEG levels; (2) whether the low arousal subjects would achieve optimal control over their theta EEG levels with the one-phase EEG/EEG training; (3) whether the high arousal subjects would show the greatest enhancement of theta EEG activity with the one-phase EMG/EMG and/or the two-phase EMG/EEG training; and (4) if subjects could be pre-selected for assignment to effective methods of training theta enhancement by determining their pre-training levels of physiological arousal -- as indicated by the frontalis EMG levels. As stated earlier, a post hoc interest developed as a result of the data analysis in determining whether the inverse relationship between frontalis EMG and theta EEG activity, reported by Sittenfeld, Budzynski and Stoyva (1976), could be verified.

Theta Rhythm Enhancement

It had been hypothesized that biofeedback training methods could be used to teach subjects to enhance their theta EEG levels. Although the analysis of variance indicated that there was a significant increase in theta rhythm levels over
the pretraining and posttraining recording sessions, this hypothesis cannot be accepted. In the absence of clear differential training effects for any of the training methods, the lack of the predicted relationship between frontalis EMG and theta EEG measurements at the posttraining sessions, and the low correlation between the pretraining and posttraining theta EEG scores, it cannot be asserted that these increases in theta EEG activity were due to learning and not to a generalized state of decreased physiological arousal. This experiment cannot provide conclusive evidence that subjects are capable of learning to enhance theta rhythm levels through biofeedback training. The findings of this experiment are consistent with the observation of Rockstroh, Birbaumer, Elbert and Lutzenberger (1984) concerning the operant control of EEG activity. They have stated,

Results produced by conditioning of spontaneous EEG oscillations (alpha and theta) dampened the early enthusiasm: e.g., no increase above baseline levels could be achieved and no reliable behavioral effects became manifest (p. 139).

Low Arousal Differential Training Effects

The predicted differential effect of the one-phase EEG/EEG feedback training methods on the ability of the low arousal subjects to learn to enhance their theta EEG levels was not observed. The simple-simple main effects analysis revealed that the low arousal one-phase EEG/EEG group did not
demonstrate any significant changes in theta EEG level. In fact, the low arousal one-phase EEG/EEG group obtained the single lowest F-ratio in the theta EEG simple-simple main effects analysis. (Refer to Table IV.) In addition, the simple-simple main effects analysis indicated that the one-phase EMG/EMG training method was the only one to approach a significant level of theta enhancement for the low arousal subjects. This enhancement, although weak, was surprising because the low arousal one-phase EMG/EMG group did not demonstrate a significant decrease in their frontalis EMG levels and other low arousal groups with equivalent posttraining baseline EMG levels did not show any increase theta EEG activity. Also, according to the logic underlying the predicted pattern of training effects for the low arousal subjects, the one-phase EMG/EMG training method was expected to be the least effective feedback method.

**High Arousal Differential Training Effects**

The hypothesis that the high arousal subjects would demonstrate the greatest learned enhancement of theta EEG levels with the one-phase EMG/EMG and/or the two-phase EMG/EEG feedback training methods was not confirmed. The simple-simple main effects analysis of the theta EEG data demonstrated that the only high arousal group to markedly increase theta rhythm levels was the one that received the one-phase EEG/EEG feedback training. This low arousal
one-phase EEG/EEG training group demonstrated the highest posttraining frontalis EMG level and the least change in frontalis EMG levels from the pretraining to the post-training recording levels. (Refer to Tables IV. & X. and Figures II. & IV.) Thus, this effect was inconsistent with the predictions made on the basis of the Sittenfeld, Budzynski and Stoyva experiment (1976). However, it had been predicted that the high arousal subjects would exhibit the greatest reductions in frontalis EMG activity. As may be observed in Table X. and Figure IV., this prediction was confirmed. Therefore, it was surprising that these groups did not exhibit any significant increase in theta EEG activity.

**Physiological Arousal and Selection of Training Methods**

The hypothesis that the subject's level of physiological arousal -- as indicated by pre-training baseline frontalis EMG levels -- must be taken into consideration when selecting an appropriate method for training subjects to enhance theta EEG activity was not confirmed by the results of the present experiment. The results tend to be consistent with the findings of the most recent investigations that indicated that frontalis EMG feedback training results in a highly localized relaxation of the frontalis muscles that does not generalize to other muscle groups, or result in decreased
physiological or emotional arousal (Weinman, Semchuk, Gaebe and Matthew, 1983; Shirley, Burish and Rowe, 1982; Thompson, Haber and Tearnan, 1981; and Lutzenberger, Birbaumer and Steinmetz, 1976). From the context of the above and the results of the present experiment, it appears that the use of frontalis EMG activity as an indicator of the subject's level of physiological arousal, and as a means of decreasing the subject's level of arousal to increase theta rhythm activity was ineffective. However, as suggested by Shirley, Burish and Rowe (1982), the concept of a "key" muscle group that becomes very tense under stress and whose relaxation is instrumental in moving the subject towards a state of general relaxation may be an accurate concept, in spite of the refutation of Sittenfeld, Budzynski, and Stoyva's (1976) claim that the frontalis was such a "key" muscle group. Future research on the relationship of muscle relaxation training and its relationship to the enhancement of theta EEG levels might benefit from the utilization of the multiple feedback sites that Shirley and her colleagues (1982) used to reduce signs of autonomic arousal. Possible muscle groups to be included in multiple site feedback would include the frontalis, masseter, sternomastoid, and forearm flexor. These muscle groups have all been reported to reflect increased emotional and physiological arousal during periods of stress, and are likely to include the "key" muscle groups proposed by Budzynski and Stoyva (1969).
Inverse Relationship Between Theta EEG and Frontalis EMG

As discussed earlier, the inverse relationship between theta EEG and frontalis EMG levels could not be verified by the results of this experiment. If the more recent research indicating that frontalis EMG activity is unrelated to the subject's level of physiological arousal is correct, then it is understandable why the posttraining correlation of theta EEG and frontalis EMG was non-significant. However, it is not clear why Sittenfeld, Budzynski and Stoyva (1976) obtained a significant coefficient.
V. CONCLUSION

The hypotheses that theta rhythm could be enhanced by biofeedback training and that the subject's physiological level of arousal must be taken into consideration for the selection of an appropriate training method were not supported. The inverse relationship between frontalis EMG levels and theta EEG activity at the posttraining recording sessions was not replicated.

A review of the current research literature indicates that recent publications concerning the biofeedback regulation of theta rhythm activity have not been forthcoming. The research on the biofeedback training of relaxation skills has focused on EMG training and not on the relationship of EEG activity to states of low arousal. The EMG literature, as previously described, indicated that the level of frontalis activity was unrelated to the subject's state of physiological arousal. However, although the activity of the frontalis muscles does not seem to be either an indicator of the subject's state of arousal or a means of manipulating the subject's level of arousal, the relationship of other muscle groups and overall EMG activity has been reported to bear some relationship to physiological and emotional arousal. The research on the effects of theta EEG
regulation on arousal and vigilance is no longer being actively pursued.

Rather than totally abandoning this line of research, I recommend that an additional experiment be considered. For the moment, let it be assumed that the concept of "key" muscle groups as indicators of and a means of altering levels of physiological and emotional arousal is not erroneous; and that an inverse relationship may exist between the activity of muscle groups in addition to the frontalis, and theta EEG activity. Also, unless a Type I. error was committed by Sittenfeld, Budzynski, and Stoyva (1976), they did demonstrate that the effectiveness of the training methods interacted significantly with the subjects' baseline frontalis EMG levels. They used frontalis EMG baseline levels as an indicator of the subjects' level of physiological arousal. The use of frontalis EMG activity, in view of the more recent research literature cited earlier, was not appropriate for these purposes. Thus the results of their experiment now seem paradoxical; and their interpretations are no longer tenable in view of the more recent research findings that frontalis EMG is not an indicator of physiological arousal and that frontalis EMG relaxation training does not generalize to other muscle groups to lower the subject's overall level of arousal. However, I believe that the hypothesis that the subject's level of physiological arousal must be taken into consideration for the selection
of an optimal method of training subjects to enhance theta rhythm activity should not be discarded yet. Sittenfeld, Budzynski and Stoyva's (1976) results cannot be adequately explained by their interpretation that the high EMG subjects were "shaped" into a state of low arousal by the two-phase EMG/EEG training method. Thus, alternative explanations must be considered. Unfortunately, their published report does not provide enough statistical information to allow me to indulge in detailed and systematic speculation. Therefore, rather than speculate extemporaneously, an additional experiment is proposed.

This experiment would differ from the present experiment and that of Sittenfeld, Budzynski and Stoyva (1976) in four important respects. First, the number of subjects would be increased to enhance the power of the statistical analysis. It is possible that there may have been some weak three-way interaction effects that were not statistically significant in the present experiment due to small number of subjects per group. Second, frontalis EMG activity would not be used as a principal indicator of the subject's level of arousal. Instead, multiple measures of muscular, autonomic and central nervous system arousal would be utilized. These would include multiple site EMG recordings of frontal, masseter, sternomastoid, and forearm flexor muscles; heart rate, blood pressure, electrodermal activity and respiration measurements; as well as EEG recordings (Davidson and
Schwartz, 1976; Gatchel, Korman, Weis, Smith and Clark, 1978; and Shirley, Burish and Rowe, 1982). Third, the concept of "key" muscle groups would be preserved. However, frontalis EMG biofeedback training would not be used as a means of manipulating the subject's level of physiological arousal. Instead, multiple site muscle feedback would be used as a means of reducing the subject's level of arousal. This would initially follow the methodology outlined by Shirley, Burish and Rowe (1982). Frontal, masseter, sternomastoid and forearm flexor EMG recordings would be made from each subject. In subjects not receiving EMG feedback training, these measures would be only an additional index or arousal. The subjects receiving EMG biofeedback training would receive auditory feedback that would reflect an average integrated EMG level of all four of the combined muscle sites. This would increase the likelihood of altering the activity of the "key" muscle group(s), that may vary from subject to subject. It would also maximize the likelihood that the EMG training would move the subjects in the direction of a generalized level of low arousal that has been repeatedly associated with high levels of theta EEG activity. Fourth, the effectiveness of the multiple site feedback as a means of lowering arousal would be checked by comparison to a non-biofeedback method of relaxation training, such as deep muscle relaxation. This experiment would also allow for a more careful examination of the relationship of EMG levels and theta EEG levels; and the
relationship of the measures of autonomic arousal to both the EMG indices and the levels of theta rhythm. The design of the experiment would be essentially the same as that of the present experiment. Subjects would be divided into low and high arousal groups contingent upon their integrated average multiple site EMG level and the pattern of the autonomic indices at the pretraining recording sessions. Three biofeedback training methods would be used. They would include the one-phase EMG/EMG, the two-phase EMG/EEG and the one-phase EEG/EEG methods. The non-feedback muscle relaxation training would be used as the basis for a one-phase and a two-phase group. The one-phase group would receive eight sessions of deep muscle relaxation training, and the two-phase group would receive four sessions of deep muscle relaxation training followed by four sessions of direct theta EEG feedback training. The results of this experiment would establish what peripheral indicators of arousal (multiple site EMG or the autonomic measures) were related to the enhancement of theta EMG levels. It would also reveal whether more suitable forms of EMG feedback training or non-feedback relaxation training were effective as indirect means of enhancing or "shaping" theta EEG activity. It would also offer a more sensitive test of whether theta EEG levels could indeed be enhanced by biofeedback training.
Experiment Consent Form

Purpose:

It has been demonstrated that it is possible to train people to voluntarily control certain physiological processes, such as brain wave activity, blood pressure, muscle tension, skin temperature, heart rate, and gastrointestinal activity. The purpose of this experiment is to investigate the procedures used to train people to regulate the activity of certain brain waves. It is hypothesized that certain training procedures may be more effective than others in teaching the control of brain rhythms.

Procedure:

The procedure to be employed in this experiment will not subject the participants to physical, emotional or mental risks. The participants will not be subjected to any procedures which violate their rights to personal privacy, cause embarrassment, or inflict any type of pain. It is also emphasized that the level of performance on the experimental tasks does not reflect upon the individual's intelligence of any other personality trait.

The procedure requires a total of thirteen hours participation from each participant. The total time will be subdivided into thirteen separate one hour sessions. The participants will each receive training in the regulation of their frontalis muscles and/or brain rhythms. The training procedure requires that electrical activity be recorded from the brain and from the frontalis muscles (forehead). This procedure involves the fastening of electrodes via a head band and hair clips to the scalp, and via adhesive collars to the forehead. This recording procedure will not cause any discomfort. In addition, participants will receive information about the state of their brain wave activity/or frontalis muscles through a stereo speaker.

During all phases of the experiment, each participant will be seated in a quiet and dimly lit room. The experimenter will be in an adjacent room during the experimental sessions. Two way conversation will be possible, between the participant and the experimenter, at all times via an intercom. The participant will not be restrained, and the door to the experimental room will always be unlocked.

Payment:

Each participant will be paid at the completion of the final experimental session.

Questions:

As a necessary control procedure, certain information about the experiment cannot be disclosed, until the completion of
the final experimental session. However, questions concerning the procedures, scheduling, and the nature of the experimental tasks may be asked prior to the signing of the consent form and prior to the start of any of the experimental sessions. Upon completion of the experiment, each participant will be formally briefed about the purposes of the experiment and specific variables which were manipulated. All questions will be answered at that time.

Consent:

It is emphasized that any participant is free to withdraw consent and to discontinue participation in the experiment at any time.

I certify that I have read the above description of the experiment, and have been given the opportunity to inquire about the procedures to be employed. In addition, I certify that I have never been diagnosed as an epileptic or diabetic, and that I have never been treated for epilepsy, convulsions or any other neurological disorders. I hereby give my informed consent to be used as a participant (experimental subject) in the above described experiment, which will be conducted by David Lawson of the University of New Hampshire's Department of Psychology.

_________________________________________  ________________
Signature                                     Date
The following information will be kept completely confidential and will be used only for the interpretation of the experimental results.

**Biographical Data**

PLEASE PRINT

Name _________________________________ Age ___ Sex ___

Local Address _________________________________

_________________________________________

Phone # _________________________________

Are you currently taking any medically prescribed medications? Yes _____ No ____

If yes, please list and indicate how frequently.

Have you taken any tranquilizing, sedative, analgesic (pain killers), anti-depressant, or stimulant medications during the past five days? Yes _____ No ____.

If yes, please list and indicate them.

Have you consumed any coffee, tea (or other substances containing caffeine), tobacco products (cigarettes, cigars, pipe, tobacco, etc.) or alcohol within the past five hours? Yes _____ No ____.

If yes, please indicate what and when (how long ago)?

Are you now, or have you ever, been diagnosed or treated for diabetes, epilepsy, convulsions, stroke, loss of consciousness, or any neurological disorders? Yes _____ No ____.

If yes, please elaborate.
Comparison of the Sittenfeld, Budzynski and Stoyva (1976) Experiment with the Present Experiment

Statistically significant main effects and interaction effects are indicated with an (*). The alpha level, and the direction of change on the Pre/Postrecording Variable are also indicated.

 Theta EEG

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