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SADLER, Roberta Ray, 1950-
AROUSAL LEVEL AND VOLUNTARY ALPHA
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The University of North Carolina at
Greensboro, Ph.D., 1977
Psychology, physiological

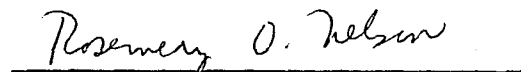AROUSAL LEVEL AND VOLUNTARY ALPHA CONTROL

by

Roberta Ray Sadler

A Dissertation Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
1977

Approved by

Dissertation Adviser   Dissertation Adviser

APPROVAL PAGE

This dissertation has been approved by the following committee of the Faculty of the Graduate School at the University of North Carolina at Greensboro.

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Committee Members _____

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February 4, 1977
Date of Acceptance by Committee

ii

SADLER, ROBERTA RAY. Arousal Level and Voluntary Alpha Control. (1977)
Directed by: Drs. Robert G. Eason and Rosemery O. Nelson. Pp. 63

To examine the influence of arousal level on subjects' ability
to voluntarily control alpha activity level, loud unpleasant noises
were presented at random intervals. The effect of this aversive stimu-
lus situation on alpha activity level relative to no noise was examined
while subjects attempted to enhance or suppress alpha activity in both
eyes-open and eyes-closed conditions. The eight subjects were pretrained
to a specified criterion of alpha control before participating in the
four experimental sessions. In addition to recordings of the EEG alpha
activity, concurrent measures of neck EMG activity and heart rate were
also obtained to provide information concerning general somatic arousal
during the various experimental conditions.

The results indicated that the strength and direction of the
interrelationships of the physiological variables—heart rate, EMG, and
alpha level—were dependent on whether the eyes were open or closed.
The failure to demonstrate a relationship between alpha level and a sub-
jectively aversive situation was discussed as possibly being dependent
on the nature of the biofeedback control of alpha.

ACKNOWLEDGEMENTS

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Special thanks is given to Dr. Robert G. Eason for his time and patience in the supervision of this research and his concern for my progress throughout my graduate career.

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CHAPTER I

INTRODUCTION

The recently popularized area of biofeedback research is perhaps

prematurely embarking on a therapeutic path. While it is evident that

human subjects can learn to gain voluntary control of many physiological

processes, the evidence for therapeutic benefits of such control has not

been sufficiently founded to allow for uncautious endorsement of the use

of biofeedback techniques in therapy. Perhaps the most tenuously based

therapeutic intervention involving biofeedback is the training of volun-

tary control of alpha brain wave activity (see review by Blanchard &

Young, 1974).

The EEG alpha rhythm has for some time been recognized as an electro-

physiological correlate of general arousal, desynchronization reflecting

increased cortical activation and dominant alpha activity reflecting a

low arousal state. Evidence for such a correlation is to be found in

the classical studies conducted by Moruzzi, Magoun, Lindsley, and asso-

ciates (e.g., Lindsley, 1952; Moruzzi & Magoun, 1949). Prior to this

research, the first reported characteristic of the alpha rhythm was the

fact that it was blocked when any of a variety of sensory or attentional

stimuli were presented. Following a few repetitions of these stimuli,

this rhythm would no longer block. Berger (see Brazier, 1958), who is

credited with discovering the alpha rhythm in humans, believed that

alpha blocking resulted from the focus of attention upon the specific

sensory system being stimulated. Adrian and Matthews (1934) further

substantiated this view. Their observations, along with those of

Berger, subsequently led to the concept of attention being included in

the official definition of alpha activity by the terminology committee

of the International Federation for Electroencephalography and Clinical

Neurophysiology. Thus, alpha rhythm, as defined by that organization,

is a "...rhythm, usually with a frequency 8-12 c/sec in adults, most

prominent in the posterior areas, present most markedly when eyes are

closed, and attenuated during attention, especially visual."

The traditional interpretation of prominent alpha activity as re-

flecting a low arousal level characterized by awake, nonattentive

states is presumably one of the bases for the assumption that the train-

ing of voluntary alpha control would be beneficial in therapy aimed at

reducing arousal or anxiety fundamental to a variety of behavioral prob-

lems. In fact there are few studies that have attempted to examine

whether psychophysiological indicants of arousal vary systematically

with voluntary alpha control. In one study Beatty and Kornfeld (1972)

found heart rate and respiration to vary independently of voluntary en-

hancement and suppression of alpha activity. However, subjects partici-

pated in only one session totaling eight trials. Sadler and Eason

(1976) conducted a more extensive study in which changes in physiological

indicants of cortical activation level and general bodily arousal were

systematically observed while subjects voluntarily enhanced and sup-

pressed alpha activity for four eight-trial sessions. The hypothesis

was that voluntary control of alpha is at least partly mediated through

self-induced changes in cortical activation level and bodily arousal.

It was thought that high states of arousal and visual attentiveness would be associated with low alpha levels, and that relaxation and de-focussed attention would be associated with high alpha levels. The findings based on four physiological indicants of arousal (cortical evoked potentials to probe light flashes, oculomotor activity, neck muscle tension, and skin conductance) generally supported the hypothesis, although the relationship was far from perfect. Apparently other factors in addition to self-induced changes in arousal level influence the voluntary control of alpha.

Perhaps a more dominant influence on alpha activity than general arousal factors is the modality specific influence of the visual system, either through direct visual stimulation, attention to visual stimuli, or through oculomotor activity. The oculomotor data from the Sadler and Eason study indicated that some of the subjects were differentially manipulating their eyes during the voluntary generation of high and low alpha levels. However, in that study it was not clear in what manner oculomotor activity was changing because the records only reflected general eye motor activity and not direction of movement.

In an earlier review of the possible factors influencing or mediating the learned control of alpha activity, Lynch and Paskewitz (1971) proposed that increases in alpha activity level in the feedback situation occur when that subject ceases to pay attention to any of a number of stimuli that normally block this activity. In two subsequent experiments, Paskewitz and Orne (1973) and Lynch, Paskewitz, and Orne (1974) found that feedback training could increase alpha activity level only

under conditions in which ambient stimuli were suppressing alpha activity. They ascribed the increase in alpha activity to a gradual process of disinhibition of visual stimulation, and an attenuation of visual attention and general arousal factors. While their analysis of alpha control was restricted to their experimental feedback conditions, the explanation proposed by them can incorporate positions that emphasize the influence of oculomotor change on alpha control and visual input factors as well. That is, increases in alpha control could be due to a gradual reduction of oculomotor activity and/or acquisition of skill in disregarding visual stimuli which would ordinarily inhibit such activity.

Though the question does not seem to have been too systematically studied, the evidence is that alpha activity is closely associated with the oculomotor responses of fixation, accommodation, and convergence (e.g., Mulholland & Peper, 1971). If such is the case, then perhaps voluntary control of alpha activity could be facilitated through the use of feedback training procedures directed toward control of oculomotor activity, rather than toward alpha activity per se. As a preliminary investigation into this possibility, Eason and Sadler (1977) designed a study to determine whether the manipulation of degree of eye convergence would be effective in controlling alpha level. Using an auditory feedback tone correlated either with alpha activity level or degree of eye convergence in various eyes-open and eyes-closed conditions, subjects attempted to control either alpha activity level or degree of eye convergence. The results indicated that degree of eye convergence was very closely linked to alpha activity level. Furthermore

there was strong support for the view that the voluntary control of alpha can be very effectively mediated through degree of eye convergence. The results were suggestive that alpha level may be more readily brought under a greater degree of control with eye than with alpha feedback, at least in the preliminary stages of training. Plotkin (1976) arrived at a similar conclusion from results of a study that suggested oculo-motor instructions together with alpha feedback provided the most effective method of controlling alpha activity. However, the efficacy of a therapeutic technique designed to enhance alpha activity level by manipulating oculomotor activity rather than alpha activity directly would only be of importance if in fact there are beneficial physiological and/or psychological changes associated with alpha enhancement.

Most researchers who assume that therapeutic benefits can be attributed to alpha control, rather than relying on physiological data investigating correlates of alpha control, choose to rely more heavily on the phenomenological verbal reports of subjects. The subjective feelings reported to be associated with a high alpha state are pleasantness, serenity, and meditativeness. In fact the relationship between alpha activity and subjective mood were reported prior to Kamiya's original work with alpha biofeedback (Kamiya, 1968, 1969; Nowlis & Kamiya, 1970). Observations of Zen and Yoga practitioners indicated that during meditation high and sustained levels of alpha activity were accompanied by mental and physical experiences similar to those reported by subjects in alpha biofeedback studies. The subjects experience relaxation, inner calm, and pleasant, "ego-free" perceptions.

However, the actual evidence for a particular subjective state accompanying alpha is inconsistent. Although Brown (1970, 1971) provided more (and stronger) support for the reported subjective feelings of the Kamiya studies, expectancy and demand characteristics of the experimental situations were not adequately controlled. The experimental setting and the subject's perception of his performance at the experimental task are known to frequently influence subjective reports. As Lynch and Paskewitz (1971) pointed out

> Ss enter the experiment expecting to experience
> alterations in mood, expecting the session to
> be pleasant, perhaps a "high" or if they don't
> feel this way initially, the experimenter may
> reinforce such feelings, both in the pre-experimental
> interview and in the actual instructions given
> during the experiment. (p. 205)

Paskewitz, Lynch, Orne, and Costello (1970) observed that subjects resting in total darkness failed to report any of the pleasant characteristics of the "alpha experience" though they were at the time producing large amounts of alpha. In a well-controlled study, Beatty (1972) found conflicting subjective reports when subjects were uninformed as to the affective states associated with alpha. Only subjects who were informed, "presumably because of their initial biases, reported the typical correlates of brain alpha rhythms--relaxation, calmness, inner awareness, etc." (p. 153). In a more recent study, Walsh (1974) found that alpha feedback and alpha instructions individually had no effect on reported subjective responses. Rather, the two variables interacted in such a way that for alpha experiences to be reported alpha activity had to be present in the EEG, and the appropriate cognitive set or

expectation had to be induced. "Either alone is a necessary but not a sufficient condition for the experience of the 'alpha state'" (Walsh, 1974, p. 433).

Walsh interprets his findings in terms of Schachter and Singer's (1962) theory of emotion. That is, the report of an emotion or affective state is dependent upon both the physiological change and the cognition or evaluation of that change. In Walsh's words,

> The so-called alpha experience may be associated directly with the occurrence of alpha rhythm activity, as is commonly believed, but the subjective changes involved may be so subtle as to be easily blocked by situational factors. As a result, these changes may only become apparent when the situation provides appropriate preparation for the experience, including some concepts to use in describing it. (Walsh, 1974, p. 433)

This study, then provides some support for the popular notions regarding the "alpha state," and helps to explain some of the inconsistencies previously mentioned. Though it is apparently true that alpha activity is accompanied by certain subjective experiences, without the appropriate instructional set the experiences may not be reported. Plotkin and his associates (Plotkin, 1976; Plotkin & Cohen, 1976; Plotkin, Mazer, & Loewy, 1976) take issue with Walsh's conclusion, and state in a definitive manner based on several studies, that an "alpha experience" is independent of the degree of alpha enhancement. They further state that alpha strength "is a direct function of only oculomotor processing" (Plotkin & Cohen, 1976, p. 16). In any case, the implication of these conclusions is that the effectiveness of clinical treatment in terms of alpha feedback is probably dependent primarily on

expectation, cognitive set, and general placebo factors. This evidence certainly constitutes a weak data base on which to develop a therapeutic technique.

Unless one is willing to proceed on the basis of something as ephemeral as the alpha experience, more research is needed to establish a firmer empirical foundation for the commonly held premise that therapeutic benefits may be derived from voluntary alpha control. More specifically the mechanisms underlying alpha control need to be further examined. If it is true that alpha activity reflects a state of relaxation, nonattention, and pleasantness then it should follow that inducing stress or anxiety should interfere with successful alpha enhancement (whether one chooses to emphasize the physiological or psychological components). One study in the literature has examined this assumption. Orne and Paskewitz (1974) conducted an experiment in which shock avoidance was contingent on successful alpha enhancement. They found that anticipation of electric shock did not interfere with voluntary alpha enhancement. Because this study contradicts a commonly held assumption, one would have to question whether Orne and Paskewitz were successful in manipulating anxiety and arousal with their shock avoidance technique. Furthermore, their failure to demonstrate interference of alpha enhancement cannot be interpreted as evidence against such interference. For stronger evidence as to the effect of inducing stress or anxiety on alpha control, conditions of both alpha enhancement and suppression should be studied. The various combinations of possible results would provide more information with which to interpret the findings.

The present study was designed to examine more systematically the effects of "induced stress" on voluntary alpha control. The procedure for inducing "stress" was the presentation of loud, unpleasant noises at random intervals. The effects of this aversive stimulus situation on alpha activity level were examined by comparing two conditions, one in which the aversive noise condition was in effect, and the other in which there were no aversive noises. The effect of the aversive noise condition relative to no noise was examined for conditions of enhancement (high alpha) and suppression (low alpha). In this way the effects on the bidirectional control of alpha could be assessed.

To manipulate the ambient level of alpha activity and thus gain further information related to the effects of the aversive noise vs. no noise condition on the bidirectional control of alpha, conditions in which the subjects' eyes were open and closed also were included. Normally, alpha activity level is maximal with eyes closed and greatly reduced with eyes open. In addition to EEG alpha activity, concurrent recordings of neck EMG activity and heart rate were obtained. These dependent variables provided information concerning general autonomic and somatic arousal.

CHAPTER II

METHOD

Subjects

Eight graduate students served as subjects. They were chosen on the basis of their ability to demonstrate reliable changes in alpha activity level. During pre-experimental practice sessions they produced at least a 20% μv difference between alpha enhancement and alpha suppression when provided with an auditory feedback tone. Participation in the pre-experimental sessions familiarized the subjects with the laboratory and data collection procedures. During this training period the subjects were informed as to the general characteristics of alpha activity (relaxed, awake, nonattentive state). Three of the subjects had previously participated in alpha biofeedback studies.

Experimental Design

After demonstrating reliable control of alpha, each subject ran in four experimental sessions. During all experimental trials subjects received continuous auditory feedback reflecting alpha level. For two of the sessions subjects participated in an eyes-open (EO) condition throughout the experimental trials; for the other two sessions, they were subjected to an eyes-closed (EC) condition throughout the experimental trials. The order of EO and EC sessions was counterbalanced across subjects.

For each session there were four blocks of six 2-minute trials. For two blocks of trials the aversive noise condition (condition A) was in effect; i.e., brief bursts (1-2 sec duration) of loud, unpleasant noises were presented randomly at an average interval of about 20 sec. For the other two blocks of trials no aversive noises were presented (condition NA). The order of the blocks of trials was counterbalanced across subjects and sessions with the restriction that within any given session one A and one NA condition had to occur before and after the midsession break.

Within each block of trials there were six alternating 2-minute trials in which the subject was instructed to enhance alpha (high alpha or H condition) or suppress it (low alpha or L condition). There was a brief 15-second break between trials, with a 1-minute break between blocks of trials, and a 10-minute (midsession) break between Blocks 2 and 3. During the midsession break the subject left the recording room and walked around.

A schematic representation of the experimental design is shown in Table 1.

Table 1

Experimental Design

	A		NA	
	H	L	H	L
EO				
EC				

A sample session is illustrated in Table 2 with each 2-minute trial indicated as either H (for high alpha) or L (for low alpha).

Table 2

Sample Session

H L H L H L	(A)	Block 1
L H L H L H	(NA)	Block 2
		Midsession Break
H L H L H L	(NA)	Block 3
L H L H L H	(A)	Block 4

Note. H,L: 2-minute trials of high and low
            alpha, respectively
        A: aversive noise condition
       NA: no aversive noise condition

The dependent variables were integrated alpha level, neck EMG, heart rate, and subjective report.

Apparatus

The subject was seated in an electrically shielded, semi-darkened room during the recording session. A Grass Model 7 polygraph equipped with appropriate preamplifiers was used to record the various physiological events, permanent records being obtained with the oscillographic unit of the polygraph.

A white linen sheet was suspended across the experimental room to provide a more uniform, nondistracting visual field for the eyes-open conditions. The subject rested his chin on a metal chin bar (padded) to

minimize head movements, to help control body posture, and to permit the electro-oculographic recording of eye movements.[1]

EEG and integrated alpha. EEG activity was recorded monopolarly with Grass gold disc electrodes placed 2½ cm above the inion on the mid-line (O_z) with the reference electrode clipped to the right earlobe. The electrodes were connected to the input of a 7P5 EEG preamplifier, with ½ amplitude low and high frequency filters set at 1 and 35 Hz, respectively. The EEG activity was then directed through a low frequency band pass filter set at 8-12 Hz to a Grass Model 7P3 preamplifier that amplified the filtered signal and integrated the EEG in the alpha frequency band. This integrated signal of d.c. voltage was used to operate a frequency modulated power driver connected to a speaker which provided continuous feedback to the subject in terms of the pitch of a tone. The integration procedure also provided an easily quantifiable measure of alpha activity.

EMG. Muscle action potentials were recorded from two gold disc electrodes attached 5 cm apart over the trapezius muscle of the neck. The ½ amplitude low and high frequency filters of the 7P3 preamplifier were set at 10 and 75 Hz. The potentials were integrated, with the time constant equal to .5 sec, to facilitate quantification.

Heart rate. Heart rate (HR) changes were monitored on a beat by beat basis with a Grass 7P4A Tachograph preamplifier. Gold disc

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[1]Eye position in terms of convergent and divergent eye movements was recorded, but the measurement procedure proved to be unreliable. These data were not analyzed.

electrodes attached to the left ankle and right forearm were used to record electrocardiographic (EKG) signals.

Auditory feedback system. Through the use of solid state logic modules, a continuous auditory tone was programmed to vary in pitch as a function of the voltage level of the integrated alpha measure. For four subjects an increase in pitch indicated an increase in alpha; for the other four subjects a decrease in pitch indicated an increase in alpha. The pitch of the tone varied within less than an octave's range, with slight changes in pitch easily discriminable by the subjects.

Aversive noises. The aversive noises consisted of the recording of brief bursts of noises obtained by modulating the frequency of a frequency modulator. (Pilot subjects had indicated that the variety of noises obtained in this manner were the most aversive noises of an original recording of sounds that also included sirens, alarms, buzzers, drills, screams, clashing pots and pans, and metal scraping tin.) During trials in which the aversive noise condition was in effect, the loud, unpleasant noises were presented randomly at an average interval of about 20 sec (range approximately 5 to 40 sec). The prerecorded noises were presented via a speaker located on the ceiling of the subject's room. The sound level during the aversive noises as measured with a 1551-C Sound-Level Meter (weighting = A) was approximately 106+ db. (The noises were not all of equal intensity.)

Procedure

Each subject acted as his/her own control, participating in all conditions of the experiment. She/he had demonstrated the ability to control alpha in pre-experimental practice sessions. At the beginning of each experimental session she/he was informed as to whether she/he should keep his/her eyes open (EO condition) or closed (EC condition) during recording periods. At the beginning of each block of six trials she/he was informed as to whether the aversive noise condition was in effect (A condition) or not in effect (NA condition). For the aversive condition the instructions were:

> This is the aversive condition. During the next block of trials you will be randomly presented with loud, unpleasant noises. While you concentrate on controlling alpha I want you at the same time to maintain an awareness that the noises will be nasty and unpleasant. Try to establish better control of alpha despite the noises. Remember, the noises are aversive, very unpleasant.

For the non-aversive condition the instructions were:

> This is the non-aversive condition. During the next block of trials, just relax and put all of your efforts into controlling the feedback tone. Please try as hard as you can to maintain the difference in your alpha level.

Each trial began with instructions to either try to produce alpha (H condition) or try not to produce alpha (L condition). The termination of a trial was signalled by the offset of all auditory stimuli including random (background) noise which served to mask extraneous sounds during each trial.

CHAPTER III

RESULTS

In synthesizing the results it is important to remember that each subject ran in four experimental sessions, two sessions with eyes open and two with eyes closed. Each session consisted of four blocks of six 2-minute trials. For two blocks of trials the aversive noise condition was in effect (condition A); for the other blocks of trials, the aversive noise condition was not in effect (condition NA). Three of the 2-minute trials within each block were the high alpha condition and three were the low alpha condition.

Each trial was divided into two 1-minute intervals to facilitate measurement of the analogue data. A "best fit" visual average for the HR, integrated EMG, and alpha polygraph records was obtained for each minute interval. This was accomplished by drawing a horizontal line through each 90-mm (1-min) segment of the analogue records such that the total area lying above and below the horizontal line was approximately equal. The mm measurements of the distance from the horizontal line to baseline were then converted to microvolts (µv) for the EMG and alpha measures and to beats per minute (BPM) for HR. For each physiological measure the resulting 12 data-units per block of trials were collapsed into 2 by averaging the 6 for the high alpha condition (two 1-minute intervals for three trials) and averaging the 6 for the low alpha condition. Thus the basic units for statistical analysis were the average

levels of each physiological measure within each block of trials for the high alpha condition and the low alpha condition.

The statistical analyses were of three types: univariate analyses of variance (ANOVAs), correlations and multiple regressions.[2] These analyses were performed on the composite data collected under all conditions of the experiment. In addition, correlational and multiple regression analyses were performed separately on the data for the eyes-open condition and the eyes-closed condition.

Composite Data

ANOVAs

Repeated measures univariate analyses of variance were performed on the group data for each physiological measure—alpha level, HR, and EMG. The treatment factors consisted of alpha condition (H-L), eye condition (Eye—EO or EC), and noise condition (N—no aversive noise or aversive noise). The temporal factors were session-half (H—first half of session or second half—or before or after midsession break) and repetition (Rep, there were two EO and EC sessions). Including subjects (S) with the treatment and temporal factors, there were a total of six variables which yielded a 2x2x2x2x2x8 factorial design. Table 3 summarizes the significant effects obtained from the analyses of alpha level, HR, and EMG.

---

[2]The two computer packages used for statistical analysis were SAS (Statistical Analysis System) for the correlations and multiple regressions, and BMD08V for the ANOVAs.

Table 3

Summary of the Significant Effects from the Analyses

of Variance for Alpha Level, HR, and EMG

Source	Alpha	HR	EMG
H-L	**		
Eye			
N			
H	*	*	
Rep			
H-L x Eye			
H-L x N			
H-L x H			
H-L x Rep			
Eye x N			
Eye x H			
Eye x Rep			
N x H			
N x Rep			
H x Rep			
H-L x Eye X N		*	
H-L x Eye x H			
H-L x Eye x Rep			
H-L x N x H			
H-L x N x Rep			
H-L x H x Rep			
Eye x N x H			
Eye x N x Rep			
Eye x H x Rep			
N x H x Rep			
H-L x Eye x N x H	*		
H-L x Eye x N x Rep			
H-L x Eye x H x Rep			
H-L x N x H x Rep			
Eye x N x H x Rep			
H-L x Eye x N x H x Rep		*	

Table 3 (Continued)

Source	Alpha	HR	EMG
S x H-L	**	**	**
S x Eye	**	**	**
S x N			
S x H	*	**	**
S x Rep	*	**	**
S x H-L x Eye	*		
S x H-L x N			
S x H-L x H			
S x H-L x Rep	*		
S x Eye x N		*	
S x Eye x H		**	*
S x Eye x Rep	*	**	**
S x N x H			
S x N x Rep		**	
S x H x Rep		**	
S x H-L x Eye x N			
S x H-L x Eye x H			
S x H-L x Eye x Rep			
S x H-L x N x H			
S x H-L x N x Rep			
S x H-L x H x Rep			
S x Eye x N x H			
S x Eye x N x Rep		**	
S x Eye x H x Rep		**	
S x N x H x Rep			
S x H-L x Eye x N x H			
S x H-L x Eye x N x Rep			
S x H-L x Eye x H x Rep			
S x H-L x N x H x Rep			
S x Eye x N x H x Rep		**	
S x H-L x Eye x N x H x Rep			

*p < .05
**p < .01

Alpha level. The significant effects averaged across the subjects
factor, are presented for the alpha data in Figures 1 and 2. Figure 1A
shows the consistent difference in alpha activity level between the
high and low alpha conditions ($p < .05$), with alpha activity level in-
creasing in the second half of the session following the midsession
break.

From Figure 2 the nature of the significant third-order interaction
involving alpha condition, eyes-open vs. eyes-closed, noise condition,
and session-half can be discerned ($p < .05$). The most noticeable change
is the sharper increase in alpha level between the first and second
half of the session for the high alpha condition during the nonaversive
noise condition for eyes-closed sessions. Except for this noticeably
steeper increase during the no noise, high alpha condition, the increase
between first and second session-halves appears to be generally less for
the eyes-closed condition than for eyes-open.

In addition to the effects just described there were several signif-
icant interactions involving subjects with the treatment factors of
alpha condition and eye condition and the temporal factors of session-
half and repetitions. Other than the effect shown in Figure 2 and de-
scribed above, the noise condition failed to produce a significant ef-
fect on alpha level, even in interaction with other factors. (See
Table 3).

Heart rate (HR). The only significant main effect for HR involved
the temporal factor of session-half as illustrated in Figure 3. HR de-
creased significantly following the midsession break ($p < .05$). Two
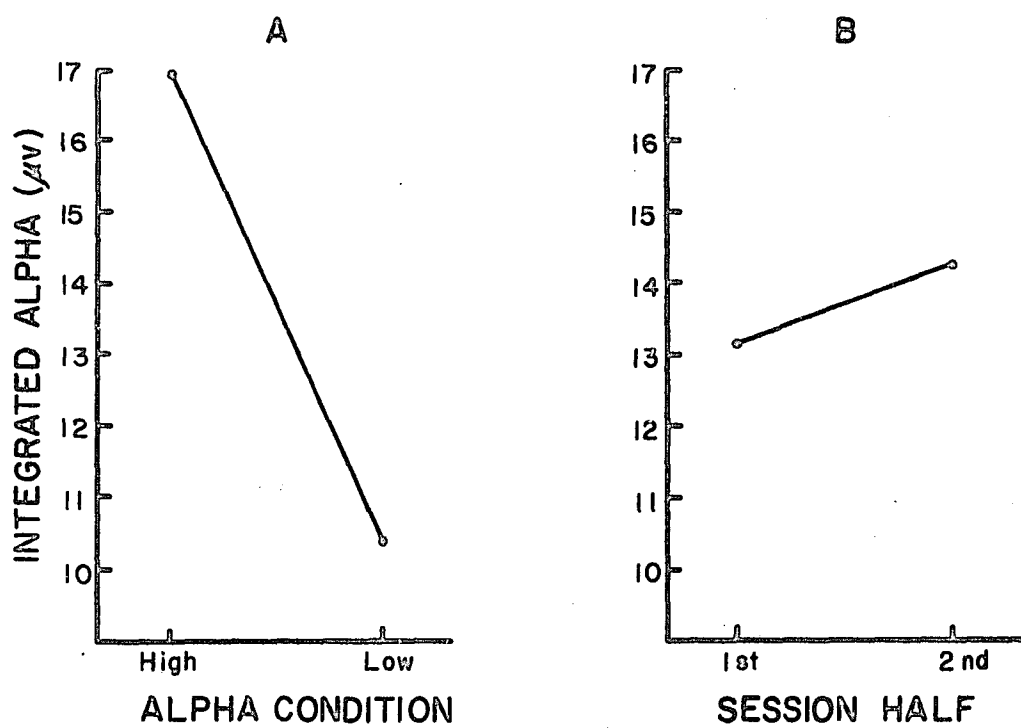
Figure 1. Integrated alpha level as a function of high or low alpha condition (A) and first or second session half (B).
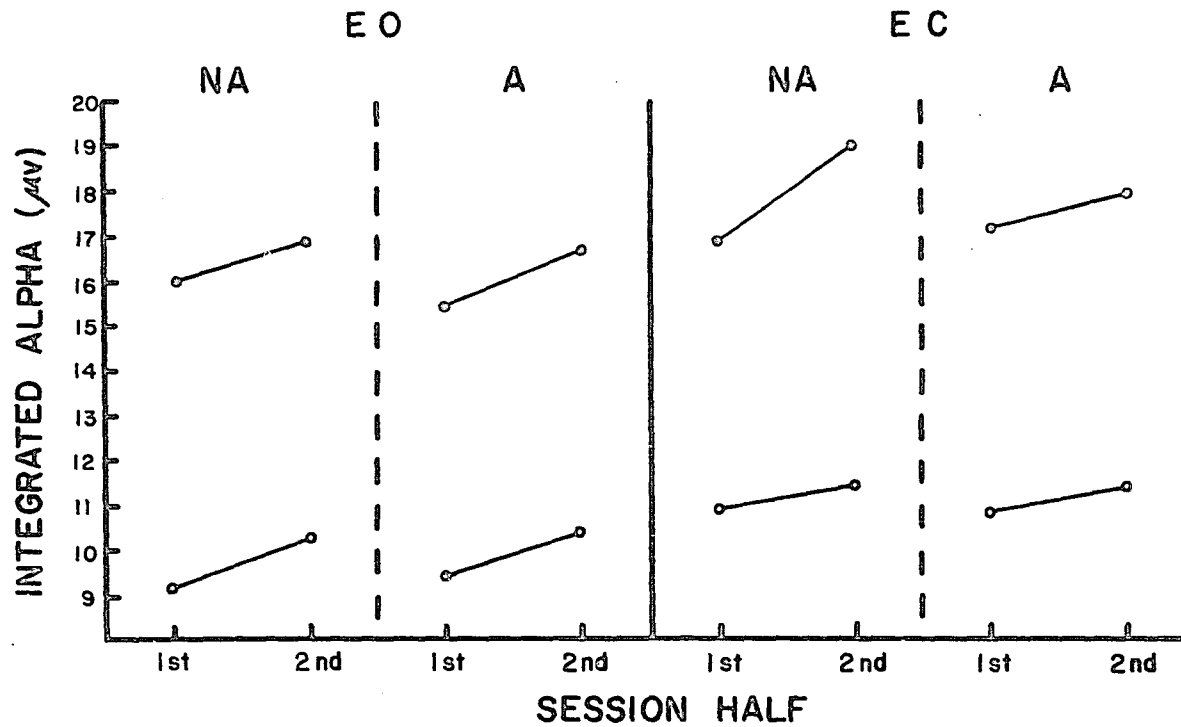
Figure 2. Integrated alpha level as a function of the alpha, eye, and noise
conditions, and session half. The closed circles represent the
high alpha condition (o—o); the open circles represent the low
alpha condition (o—o).

EO: eyes open   NA: no aversive noise
EC: eyes closed   A: aversive noise
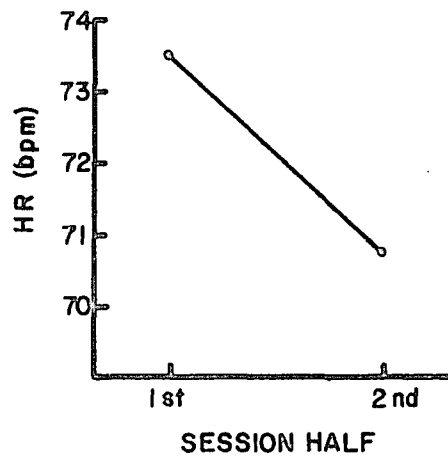
Figure 3.   Heart rate as a function of session half.

interactions not involving subjects were found to be significant. The three treatment factors--high or low alpha, eyes-open or closed, and no noise vs. aversive noise--formed a significant second-order interaction (p < .05), illustrated in Figure 4. As shown in this figure the degree of difference in HR between the high and low alpha conditions depended on whether the eyes were open or closed and, at the same time, whether aversive noise was present. (Although in Figure 4 HR appears to be consistently greater during the eyes-open than the eyes-closed condition, and greater during the low alpha than high alpha condition, these effects did not reach significance due to the amount of variance between subjects.)

The three treatment and two temporal factors produced a significant fourth-order interaction (p < .05) that is depicted in Figure 5. This complex picture indicates that differences in HR between the high and low alpha conditions depended on the eye and noise conditions as well as the session-half and replication.

The variability of HR between subjects is evidenced by the number of significant interactions involving subjects and both treatment and temporal factors. (See Table 3.)

EMG. There were no significant effects in the EMG data, except for interactions and differences involving subjects. (See Table 3.)

Interactions involving subjects. There were a number of interactions involving subjects for the three physiological measures. Of most interest are the first-order subject interactions. As can be
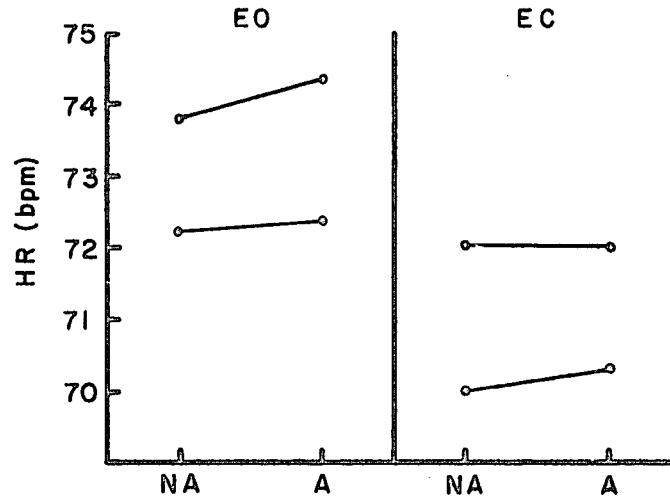
Figure 4. Heart rate as a function of the
alpha, eye, and noise conditions.
The closed circles represent the
high alpha condition (o——o); the
open circles represent the low
alpha condition (o——o).
EO: eyes open
EC: eyes closed
NA: no aversive noise
 A: aversive noise
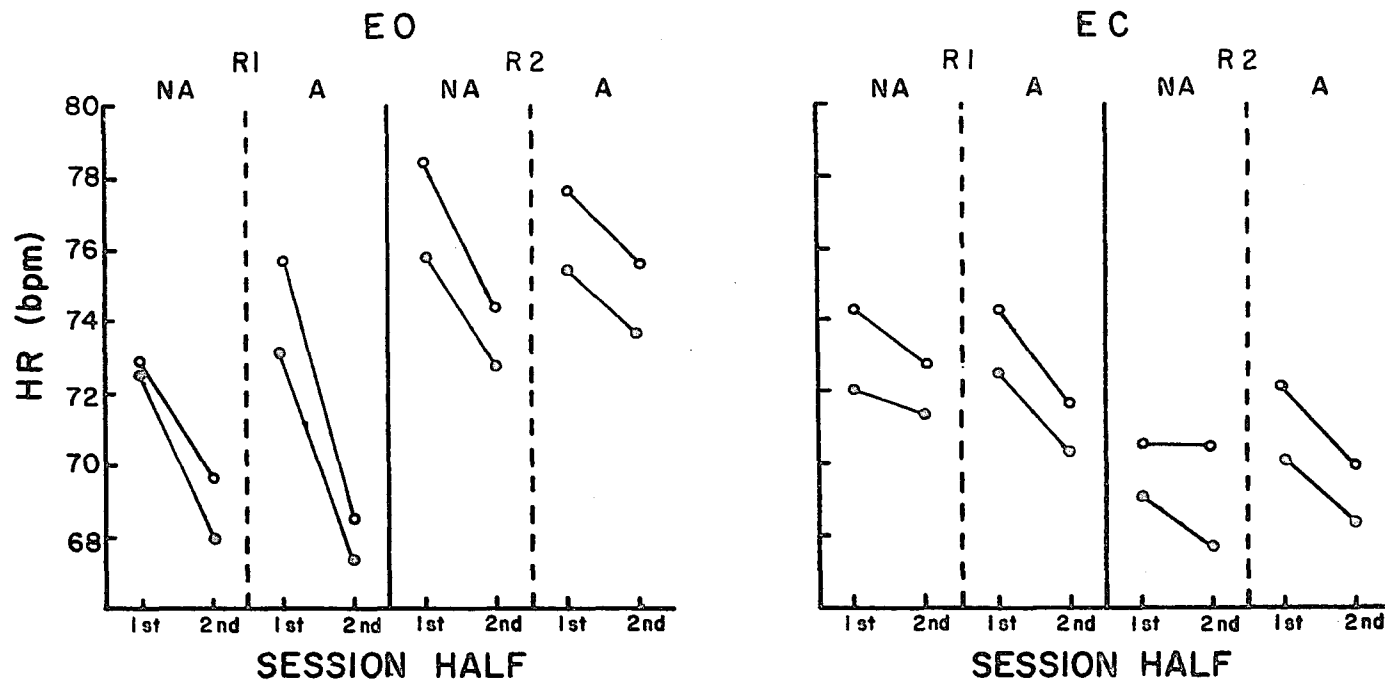
Figure 5. Heart rate as a function of alpha, eye, and noise conditions as well as
replication and session half. The closed circles (o—o) represent the high
alpha condition; the open circles (o—o) represent the low alpha condition.
EO: eyes open   NA: no aversive noise
EC: eyes closed   A: aversive noise
R1, R2: replication

observed in Table 3, the only first-order interaction involving subjects that did not attain significance across the three physiological variables was the subject X noise condition. Thus variations in alpha level, HR, and EMG were affected idiosyncratically by the treatment factors of alpha and eye condition and the temporal factors of session-half and repetition.

Correlations

Table 4 presents the correlation coefficients and p values obtained for the three physiological measures (alpha, HR, and EMG) as well as for a categorical variable, alpha condition (i.e., the high vs. low alpha condition). An α level of .001 was chosen as the criterion for significance of all linear correlation coefficients to allow for the probability inflation caused by multiple correlational analyses. (There were 18 computed linear correlations: $\alpha J = p$; $\alpha = .001$; $J = 18$; actual $p = .018$.)

Alpha level and HR. Alpha level and HR were not demonstrated to be significantly correlated ($r = -.15$, $p < .02$).

Alpha level and EMG. Alpha level and EMG were not found to be related significantly ($r = .04$).

EMG and HR. There was a significant, moderate correlation between EMG and HR ($r = .23$, $p < .001$). Variations in HR accounted for 5% of the variance in EMG ($r^2 = .051$), the standard error being 9.49 (EMG mean = 18.02).

Table 4

Correlation Coefficients/p Values

for the Composite Data

	Alpha Level	HR	EMG	Alpha Condition
Alpha Level	1.00	−.15 .0195	+.04 .4777	+.53 .0001*
HR		1.00	+.23 .0003*	−.09 .1418
EMG			1.00	−.05 .4286
Alpha Condition				1.00

*p < .001

Alpha condition correlations. The dichotomous variable of alpha condition (either high or low alpha) was of course significantly related to alpha level (r = .53; p < .001). Alpha condition accounted for 28% of the variance in alpha level (r^2 = .28). Neither HR nor EMG were significantly correlated with alpha condition (for HR, r = .09, p < .14; for EMG, r = .05, p < .43).

Multiple Regression

A significant multiple correlation coefficient of .17 (p < .028) was obtained when HR and EMG were used as combined predictors of alpha level. However the combination of EMG and HR accounted for only a slightly greater percentage of the variance in alpha level than did HR alone (r^2 = .028 for HR + EMG; r^2 = .021 for HR alone).

Table 5 presents the multiple regression statistics. The regression coefficients from this table yielded the following prediction equation:

$$\text{Alpha level} = 20.33 - 0.10\text{HR} + 0.05\text{EMG}$$

However the t test for the significance of the coefficient (as listed in the table) indicates that EMG should be eliminated as a variable in the prediction equation, because the value of the EMG coefficient is not significantly different from its standard error (t = 1.28; p < .20). EMG is not a significant source of variation in the multiple regression analysis.

Table 5

Multiple Regression Statistics for the Prediction of

Alpha Level by the Combination of HR and EMG

for the Composite Data

	Regression Coefficients	Standard Error of Coefficients	t for Significance of Coefficients	p
Intercept	20.3260	2.8730	7.0700	.0001**
HR	-0.1050	0.0400	-2.5800	.0103*
EMG	0.0520	0.0400	1.2800	.1999

*p < .05
**p < .01

Data Separated By Eye Condition

Correlations Within the EO and EC Conditions

Table 6 compares the correlation coefficients relating HR and EMG to alpha level for the EO condition and the EC condition, along with the composite data contained in Table 4. The correlation between HR and alpha level did not reach significance at the .001 level in any of these cases. Even though, as noted in the preceding section, the composite data yielded no significant relationship between EMG and alpha level, separation of the data by eye condition resulted in two significant correlations. During the EO condition, EMG and alpha level were positively related ($\underline{r}$ = .32; $\underline{p}$ < .0002). This positive correlation accounted for 10% of the variance in alpha level ($\underline{r}^2$ = .102) with a standard error of 6.04 (mean alpha level = 13.0). During the EC condition EMG varied inversely with alpha level ($\underline{r}$ = -.30; $\underline{p}$ < .0007). The negative correlation accounted for 9% of the variance in alpha level with the eyes closed ($\underline{r}^2$ = .087), the standard error being 5.72 (mean alpha level = 14.43). These two moderate correlations were directionally determined by the eye condition, and, being of approximately the same degree of strength, cancelled out in the composite data analysis.

Additional comparisons between the two eye conditions and the composite data with respect to the correlational analyses are presented in Table 7. The dichotomous variable of alpha condition (either high or low alpha) was significantly related to alpha level ($\underline{p}$ < .0001) for the composite data as well as the separate EO and EC conditions. The correlation coefficients were essentially the same, being .51 for the EO

Table 6

Correlations of HR and EMG with Alpha Level for the EO and EC

Conditions and the Composite Data; Correlation

Coefficients/p Values

	EO	EC	Composite
HR	−.21 .0174	−.05 .5829	−.15 .0195
EMG	+.32 .0002*	−.30 .0007*	+.04 .4777

*p < .001

Table 7

Correlation Coefficients/p Values for the EO and EC

Conditions and the Composite Data

	EO	EC	Composite
Alpha Level and Alpha Condition	+.51 .0001*	+.56 .0001*	+.53 .0001*
HR and EMG	+.18 .0424	+.30 .0006*	+.23 .0003*

*p < .001

condition, .56 for the EC condition, and .53 for the composite data. The correlations accounted for 25%, 31%, and 28% of the variance in alpha level, respectively. (Alpha condition was in no instance significantly correlated with HR or EMG, the smallest p value being .14.)

Also presented in Table 7 are the correlation coefficients for HR and EMG. As previously described, HR and EMG were significantly correlated, based on the composite data analysis (r = .23; p < .0003). While HR and EMG were not significantly correlated during the EO condition (r = .18, p > .001), there was a significant, moderate correlation during the EC condition (r = .30; p < .0006). In the latter case HR accounted for 9% of the variance in EMG (r^2 = .089), the standard error being 8.78 (mean EMG level = 18.49).

Multiple Regression for the EO Condition

A multiple correlation coefficient of .42 (p < .0001) was obtained when HR and EMG were used as combined predictors of alpha level during the EO condition. This moderate correlation accounted 18% of the variance in alpha level (r^2 = .176). The multiple regression statistics for the EO condition are presented in Table 8. The regression coefficients from this table yielded the following prediction equation:

$$\text{Alpha level} = 21.773 - 0.17\text{HR} + 0.23\text{EMG}$$

Both HR and EMG are significant variables in this equation (p < .0011 and .0001, respectively). Combining HR and EMG to predict alpha level accounted for less than 3% of the variance for the data as a whole. Separating the EO condition from the EC substantially increased prediction accuracy (to 18%).

Table 8

Multiple Regression Statistics for the Prediction of

Alpha Level by the Combination of HR and EMG

for the EO Condition

	Regression Coefficients	Standard Error of Coefficients	t for Signifi-cance of Coefficients	p
Intercept	21.7730	3.7970	5.7300	.0001**
HR	−0.1740	0.0520	−3.3500	.0011**
EMG	0.2280	0.0510	4.4800	.0001**

**p < .01

Multiple Regression for the EC Condition

Analysis of the data for the EC condition yielded a multiple corre-
lation coefficient of .30 when HR and EMG were combined to predict alpha
level. This regression analysis accounted for 9% of the variance in
alpha level ($\underline{r}^2$ = .087). In the EC condition, combining HR and EMG
offered no improvement in the prediction of alpha level over EMG alone
(see Table 6 and discussion above). This is further substantiated by
examination of the multiple regression statistics in Table 9. Although
the regression coefficients yielded the following prediction equation:

$$\text{Alpha level} = 16.17 + 0.03\text{HR} - 0.20\text{EMG}$$

The $\underline{t}$ tests for the significance of the coefficients indicated that HR
was not a significant variable ($\underline{p}$ < .6308) and should be eliminated from
the regression analysis.

Summary of the Correlational and Multiple Regression Analyses

To summarize briefly the results of the correlational and multiple
regression analyses with respect to the prediction of alpha level, with
the data combined for the eye conditions, HR alone was the best predic-
tor of alpha level with the direction of the relationship being negative.
For the EC condition, EMG alone was the best predictor of alpha level,
with the direction of the relationship being negative. For the EO condi-
tion, combining HR and EMG for the prediction of alpha level improved
the accuracy of the prediction, accounting for more variance than the
linear correlations of alpha level with either HR or EMG. In fact the
the greatest amount of variance in alpha level is accounted for by the

Table 9

Multiple Regression Statistics for the Prediction of

Alpha Level by the Combination of HR and EMG

for the EC Condition

	Regression Coefficients	Standard Error of Coefficients	t for Significance of Coefficients	p
Intercept	16.1710	3.9230	4.1200	.0001**
HR	0.0280	0.0570	0.4800	.6308
EMG	−0.2000	0.0580	−3.4800	.0008**

**p < .01

multiple regression with EO than by any other linear or multiple regressions examined. The coefficients of the EO multiple regression analysis indicated the relationship with alpha level was negative for HR and positive for EMG.

Subjective Report Data

In addition to the physiological data just described several types of subjective data were also obtained throughout the course of the experiment from seven of the eight subjects. One subject, the author, did not contribute self-report data because she served both as a subject and an experimenter, and thus could not offer unbiased information.

Subjective Ratings

At the beginning of the first experimental session the subject was presented an index card containing the following typed information:

> Subjective ratings of the difference between the aversive and non-aversive conditions.
>
> Two 5-point rating scales:
> 1. Physical feelings; how your whole body and muscles feel physically.
>
> 2. Changes in mental feelings of relaxation; feelings of pleasantness, calmness, and lack of mental tension.
>
> > +2 a large degree of perceived change in a more relaxed direction
> > +1
> > 0 no change
> > -1
> > -2 a large decrease in feelings of relaxation
>
> > (modified from Alexander, 1975)

The subject was then instructed to give two ratings at the end of each block of trials, ratings of "physical feelings" and "mental

feelings." The reader is reminded there were 16 blocks of trials in the four sessions, half of which contained aversive sounds. Table 10 presents the sum of the points for the seven subjects for the two scales-- mental and physical feelings--for the two noise conditions (NA and A). These subjective ratings as shown in Table 10 indicate that subjects experienced enhanced feelings of relaxation during the nonaversive condition and a considerable decrease in feelings of relaxation during the aversive condition.

Adjectives

At the end of the first and final sessions, subjects were requested to provide three adjectives to described the aversive noises as perceived during that session. These adjectives are listed in Table 11.

Table 10

Total Points for the Subjective Ratings of the Difference Between

the Aversive (A) and Nonaversive (NA) Conditions in Terms

of Physical and Mental Feelings of Relaxation

for 7 of the 8 Subjects

| | | Condition | |
		NA	A
	Mental	+38	-43
Relaxation			
	Physical	+26	-48

Table 11

Adjectives to Describe the Aversive Noises Obtained Following

the First and Final Sessions (Sessions 1 and 4)

from 7 of the 8 Subjects

Subject	Session 1	Session 4
1	funny loud abrupt	funny loud variable
2	abrasive startling frightening	horrible startling abrupt
3	obnoxious disruptive loud	disturbing annoying irritating
5	discomfitting disquieting irritating	annoying startling abrasive
6	startling raucous aggravating	raucous startling intense
7	agonizing hideous painful	disturbing painful arousing
8	sudden screechy prehistoric	sudden startling almost shrill

CHAPTER IV

DISCUSSION

As stated in the introduction, a primary purpose of this study as originally planned was to determine the effect of "stress" on voluntary control of alpha activity. However, this aspect of the study was not realized, because the presentation of loud, unpleasant noises apparently had little objective effect on the dependent variables examined in this experiment. The noises were subjectively disturbing for all of the subjects (except one who found them "funny") as evidenced by the physical and mental relaxation ratings (Table 10) and the adjectives given to describe the noises (Table 11). Nonetheless, the analyses of variance performed on the physiological variables uniformly evidenced no main effect of the aversive noise condition. In fact there were no first-order interaction effects involving the aversive noise (see Table 3). The aversive noise condition, although subjectively disturbing, apparently was not objectively stressful.

In general, the results of the analyses of variance revealed few main effects of the treatment and temporal factors. The only significant treatment effect was the effect of alpha condition on alpha activity level (Figure 1A). Though this supports the notion that subjects can gain bidirectional control of alpha, this fact is of little interest since the subjects were selected for the experiment on the basis of their having learned to exercise such control.

The temporal main effect of session-half achieved significance for two of the physiological variables. As noted in the results section and illustrated in Figure 1B for alpha level and Figure 3 for HR, alpha level increased during the course of a session while HR decreased. This inverse relationship between alpha level and HR is the type of covariance which supports the view that increases in alpha level may reflect general autonomic and/or somatic relaxation (e.g., HR decrease). The increase in alpha level within a session has been reported previously (e.g., Beatty & Kornfeld, 1972; Brolund & Schallow, 1976), and has led to the inference that increased alpha enhancement within the feedback situation is not due so much to the influence of the feedback stimulus on learned control, but more simply to the effects of becoming adapted to the experimental setting and becoming more relaxed.

Along this line, Plotkin and Cohen (1976) have suggested a possible explanation for the "alpha experience" or pleasant affective states said to accompany alpha enhancement. The characteristics of the experimental situation are implicated rather than alpha enhancement per se. During the typical biofeedback experiment, subjects are required to maintain a state of sustained attention while monitoring the feedback tone. In addition, prolonged sitting in a soundproof, darkened room may induce a state of mild sensory deprivation. These aspects of the biofeedback setting, along with expectations of having an "alpha experience" could lead to subjective attributes of tranquility and mental and physical relaxation which may be conducive to alpha enhancement but are independent of the subjects' voluntary attempts to produce it via auditory feedback.

Because alpha enhancement occurs within a session whether accurate feedback is provided or not, Prewett and Adams (1976) have termed voluntary alpha enhancement an "epiphenomenon." They found that, although voluntary alpha suppression appears to be acquired as a function of feedback, alpha enhancement, while coincidentally related to the instructions, was independent of feedback. In fact, alpha level was greatest during rest periods and when subjects did not know the significance of the feedback cue. Thus different processes may characterize voluntary alpha enhancement and suppression. Alpha enhancement and the shifty "alpha experience" that occasionally is said to accompany it may in fact represent epiphenomena more accurately ascribed to the influence of the experimental setting and subtle demand characteristics. On the other hand, alpha suppression may involve the more active deployment of oculomotor strategies.

Paskewitz and Orne (1973) have suggested that subjects will succeed in unidirectional enhancement of alpha activity above a baseline level only in situations that have suppressed the baseline level. They feel that any increase in alpha level is due to the subjects' acquiring skill at disregarding any stimuli that may suppress alpha level rather than actually learning to enhance alpha above an "optimal" resting level. Normally resting alpha activity level is found to be greater during eyes-closed than eyes-open conditions. Lewis and McLaughlin (1976) reported the standard finding that per cent time alpha was greater for eyes closed than for eyes open during resting baselines, and also that there was more variation in alpha activity level during baseline than

feedback conditions. Though this second finding is seldom mentioned in the literature it is not surprising that the biofeedback situation should reduce the naturally dynamic range, or inherent variability in alpha activity level. (In the present study, the eye condition effect on alpha level did not reach significance, indicating that EC alpha level was not consistently greater than EO alpha for all of the subjects in the feedback situation.)

As discussed above, some researchers feel that different processes underlie voluntary enhancement and suppression of alpha activity. Similarly, it is possible that EO and EC conditions can differentially influence the nature of voluntary alpha control. While such effects were not revealed in the variance analyses of the present study, the correlational analyses and multiple regressions offer more information.

Digressing for a moment, statements of individual researchers as to whether it is easier or more difficult to control alpha level in an eyes-open or eyes-closed condition (e.g., Plotkin & Cohen, 1976; Mulholland, 1976) are inherently biased by the individual's personal theory of alpha control--that is, at this time only one experimental study has addressed the issue (Travis, Kondo, & Knott, 1974; for enhancement only). Intuitively it would appear that the nature of the task involved in voluntary (bidirectional) control of alpha activity depends on whether the eyes are open or closed. In the resting eyes-closed condition it is generally accepted that alpha activity is maximal as compared to an eyes-open condition. Thus, in a bidirectional control situation, in which to be successful the subject must both enhance and suppress

alpha at specified periods, the main burden in the task would shift from trying to suppress alpha with eyes closed to trying to enhance it with eyes open.

While the present study was not designed primarily to assess this problem, the correlational and multiple regression results obtained for the EO, EC, and composite data are pertinent to the issue. The HR and EMG data were examined in relation to their ability to predict alpha level either singly or combined. For the composite data, which encompasses both the EO and EC conditions, HR alone, which was inversely related to alpha level, was the best predictor. However the negative correlational regression of HR only accounted for 2-3% of the variance in alpha level. The prediction of alpha level was markedly improved when the data were analyzed separately for the EO and EC conditions. In the EO condition, HR and EMG in combination accounted for 18% of the variance in alpha level. HR again was negatively related to alpha level, but EMG was positively related. In the eyes-closed condition, EMG alone was the best predictor of alpha level, accounting for approximately 9% of the variance in alpha level. However in this EC condition the direction of the relationship between EMG and alpha level was reversed, the direction of the correlation being negative. From the available data it is impossible to offer an interpretation of the nature of these results, e.g., why there are HR decreases and EMG increases in relation to alpha increases with the eyes open. For the purposes of the present argument the specific nature of the predictions is not as important as the difference in the covariance among these variables as a function of

eye conditon. That is, the strength and direction of the interrelation-
ships of the physiological variables HR, EMG, and alpha level were depen-
dent on whether the eyes were open or closed.

The fact that the correlation between alpha level and alpha condi-
tion (see Table 7) remained essentially the same when the data were
examined separately for the EO and EC conditions, as well as when the
data were combined, perhaps indicates that subjects were equally profi-
cient at controlling alpha level whether the eyes were open or closed.
The change in the nature of the covariance among the physiological vari-
ables perhaps reflects changes in the nature of the task of bidirectional
control of alpha level, depending on eye condition.

The purpose of the present study was to further examine mechanisms
underlying voluntary alpha control in order to evaluate the efficacy of
alpha biofeedback as a therapeutic technique. The original premise was
that if alpha level reflects a state of relaxation, then inducing stress
or anxiety should interfere with successful alpha enhancement. The rela-
tion between the subjective report data and alpha level found in this
study is consistent with that of Orne and Paskewitz (1974) in that sub-
jects reported a situation to be disturbing and unpleasant without any
associated change in the ability to control alpha level. Failure to
demonstrate a relationship between alpha level and a subjectively aver-
sive situation could be inherent to the biofeedback control of alpha.
As discussed above, there is some evidence that the natural variations
in alpha level observed in resting conditions is reduced during biofeed-
back control. It is possible that the voluntary control of alpha results

from processes unlike those that naturally regulate brain-wave activity. Gaining control of alpha level in the biofeedback situation may remove or mask the influence of other variables. To learn such control subjects may be manipulating some subset of all variables, physiological and environmental, that normally influence alpha. If this is the case, as seems likely, then it is not surprising that the present study and others (Beatty & Kornfeld, 1972; Sadler & Eason, 1976) have failed to find strong relationships between voluntarily controlled alpha levels and other physiological indicants of arousal.

Whether the validity of this conclusion can be supported or not, the lack of any substantial evidence supporting a relationship between voluntary alpha control and general arousal level strongly suggests that it is premature to engage in procedures involving alpha biofeedback for training relaxation and reducing anxiety. Future research should perhaps focus on direct monitoring of physiological indicants of relaxation (rather than relying on verbal reports) during various training procedures. At this time there is little, if any, evidence that alpha level increases during more traditional methods of training relaxation and anxiety reduction. Research in this direction might provide a firmer basis for equating alpha level with other measures of relaxation than is currently available.

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APPENDIX OF TABLES

Table 12

ANOVA for the Alpha Data

Source	MS	Error Term	df	F
H–L	2737.76	32	1	34.69**
Eye	130.27	33	1	4.57
N	1.95	34	1	2.13
H	71.61	35	1	9.29*
Rep	3.15	36	1	.31
H–L x Eye	.27	37	1	.03
H–L x N	3.97	38	1	3.22
H–L x H	3.04	39	1	.79
H–L x Rep	19.80	40	1	2.24
Eye x N	.17	41	1	.14
Eye x H	.03	42	1	.02
Eye x Rep	17.85	43	1	1.70
N x H	1.85	44	1	.50
N x Rep	3.34	45	1	2.73
H x Rep	3.64	46	1	2.42
H–L x Eye x N	.12	47	1	.14
H–L x Eye x H	4.70	48	1	3.30
H–L x Eye x Rep	2.71	49	1	1.35
H–L x N x H	1.17	50	1	2.29
H–L x N x Rep	8.18	51	1	2.74
H–L x H x Rep	.66	52	1	.16
Eye x N x H	2.67	53	1	2.37
Eye x N x Rep	.91	54	1	.37
Eye x H x Rep	.78	55	1	.86
N x H x Rep	2.15	56	1	1.62
H–L x Eye x N x H	3.49	57	1	6.15*
H–L x Eye x N x Rep	1.39	58	1	3.42
H–L x Eye x H x Rep	.38	59	1	.91
H–L x N x H x Rep	.86	60	1	.36
Eye x N x H x Rep	5.37	61	1	1.45
H–L x Eye x N x H x Rep	.06	62	1	.04

Source	MS	Error Term	df	F
S x H-L	78.92	62	7	47.46**
S x Eye	28.50	62	7	17.14**
S x N	.92	62	7	.55
S x H	7.71	62	7	4.64*
S x Rep	10.21	62	7	6.14*
S x H-L x Eye	9.88	62	7	5.94*
S x H-L x N	1.23	62	7	.74
S x H-L x H	3.84	62	7	2.31
S x H-L x Rep	8.82	62	7	5.30*
S x Eye x N	1.19	62	7	.72
S x Eye x H	1.48	62	7	.89
S x Eye x Rep	10.53	62	7	6.33*
S x N x H	3.70	62	7	2.22
S x N x Rep	1.22	62	7	.73
S x H x Rep	1.51	62	7	.91
S x H-L x Eye x N	.86	62	7	.52
S x H-L x Eye x H	1.27	62	7	.76
S x H-L x Eye x Rep	2.01	62	7	1.20
S x H-L x N x H	.51	62	7	.31
S x H-L x N x Rep	2.98	62	7	1.79
S x H-L x H x Rep	4.20	62	7	2.53
S x Eye x N x H	1.13	62	7	.68
S x Eye x N x Rep	2.44	62	7	1.47
S x Eye x H x Rep	.91	62	7	.55
S x N x H x Rep	4.40	62	7	2.64
S x H-L x Eye x N x H	.57	62	7	.34
S x H-L x Eye x N x Rep	.41	62	7	.24
S x H-L x Eye x H x Rep	.42	62	7	.25
S x H-L x N x H x Rep	2.38	62	7	1.43
S x Eye x N x H x Rep	3.71	62	7	2.23
S x H-L x Eye x N x H x Rep	1.66		7	

*p < .05
**p < .01

Table 13

ANOVA for the HR Data

Source	MS	Error Term	df	_F_
H–L	204.32	32	1	3.53
Eye	284.06	33	1	4.72
N	3.60	34	1	.96
H	483.92	35	1	7.88*
Rep	56.80	36	1	1.64
H–L x Eye	.02	37	1	.005
H–L x N	.06	38	1	.06
H–L x H	.39	39	1	.29
H–L x Rep	3.17	40	1	1.58
Eye x N	.64	41	1	.07
Eye x H	90.97	42	1	3.75
Eye x Rep	805.03	43	1	5.25
N x H	11.06	44	1	2.86
N x Rep	3.21	45	1	.22
H x Rep	34.23	46	1	1.93
H–L x Eye x N	1.92	47	1	10.76*
H–L x Eye x H	.31	48	1	.58
H–L x Eye x Rep	.71	49	1	.41
H–L x N x H	3.02	50	1	1.89
H–L x N x Rep	.29	51	1	.32
H–L x H x Rep	.22	52	1	.10
Eye x N x H	2.48	53	1	.86
Eye x N x Rep	6.53	54	1	.19
Eye x H x Rep	15.95	55	1	1.18
N x H x Rep	17.98	56	1	1.48
H–L x Eye x N x H	.00	57	1	.00
H–L x Eye x N x Rep	.65	58	1	.15
H–L x Eye x H x Rep	2.61	59	1	2.24
H–L x N x H x Rep	.21	60	1	.26
Eye x N x H x Rep	18.06	61	1	1.31
H–L x Eye x N x H x Rep	8.44	62	1	6.37*

Source	MS	Error Term	df	F
S x H-L	57.82	62	7	43.65**
S x Eye	60.23	62	7	45.47**
S x N	3.74	62	7	2.83
S x H	61.40	62	7	46.35**
S x Rep	34.67	62	7	26.17**
S x H-L x Eye	4.38	62	7	3.30
S x H-L x N	.88	62	7	.66
S x H-L x H	1.37	62	7	1.03
S x H-L x Rep	2.01	62	7	1.52
S x Eye x N	8.95	62	7	6.76*
S x Eye x H	24.28	62	7	18.33**
S x Eye x Rep	153.30	62	7	115.72**
S x N x H	3.86	62	7	2.92
S x N x Rep	14.51	62	7	10.96**
S x H x Rep	17.71	62	7	13.37**
S x H-L x Eye x N	.18	62	7	.13
S x H-L x Eye x H	.54	62	7	.40
S x H-L x Eye x Rep	1.72	62	7	1.30
S x H-L x N x H	1.60	62	7	1.21
S x H-L x N x Rep	.90	62	7	.68
S x H-L x H x Rep	2.13	62	7	1.61
S x Eye x N x H	2.88	62	7	2.18
S x Eye x N x Rep	33.71	62	7	25.45**
S x Eye x H x Rep	13.53	62	7	10.20**
S x N x H x Rep	12.14	62	7	9.17**
S x H-L x Eye x N x H	1.09	62	7	.82
S x H-L x Eye x N x Rep	4.30	62	7	3.24
S x H-L x Eye x H x Rep	1.16	62	7	.88
S x H-L x N x H x Rep	.82	62	7	.62
S x Eye x N x H x Rep	13.73	62	7	10.37**
S x H-L x Eye x N x H x Rep	1.32		7	

*$p < .05$
**$p < .01$

Table 14

ANOVA for the EMG Data

Source	MS	Error Term	df	$\underline{F}$
H-L	59.56	32	1	.28
Eye	57.36	33	1	.08
N	23.06	34	1	.37
H	364.90	35	1	2.44
Rep	384.16	36	1	1.71
H-L x Eye	17.61	37	1	2.66
H-L x N	43.36	38	1	.59
H-L x H	4.37	39	1	1.25
H-L x Rep	38.37	40	1	.08
Eye x N	4.26	41	1	.67
Eye x H	49.72	42	1	.01
Eye x Rep	2.74	43	1	2.08
N x H	116.53	44	1	2.28
N x Rep	81.85	45	1	.06
H x Rep	3.98	46	1	.31
H-L x Eye x N	3.74	47	1	.31
H-L x Eye x H	.64	48	1	.04
H-L x Eye x Rep	26.20	49	1	1.54
H-L x N x H	7.83	50	1	1.17
H-L x N x Rep	16.02	51	1	.88
H-L x H x Rep	23.87	52	1	3.76
Eye x N x H	.92	53	1	.02
Eye x N x Rep	30.21	54	1	1.14
Eye x H x Rep	21.26	55	1	.50
N x H x Rep	14.20	56	1	1.08
H-L x Eye x N x H	1.10	57	1	.10
H-L x Eye x N x Rep	34.70	58	1	1.22
H-L x Eye x H x Rep	1.75	59	1	.14
H-L x N x H x Rep	.72	60	1	.08
Eye x N x H x Rep	11.53	61	1	1.18
H-L x Eye x N x H x Rep	5.25	62	1	.28

Source	MS	Error Term	df	F
S x H-L	214.87	62	7	11.55**
S x Eye	758.71	62	7	40.77**
S x N	63.06	62	7	3.39
S x H	149.52	62	7	8.04**
S x Rep	224.24	62	7	12.05**
S x H-L x Eye	43.58	62	7	2.34
S x H-L x N	16.31	62	7	.88
S x H-L x H	7.42	62	7	.40
S x H-L x Rep	30.79	62	7	1.65
S x Eye x N	50.76	62	7	2.73
S x Eye x H	73.99	62	7	3.98*
S x Eye x Rep	252.55	62	7	13.57**
S x N x H	55.88	62	7	3.00
S x N x Rep	35.92	62	7	1.93
S x H x Rep	66.68	62	7	3.58
S x H-L x Eye x N	11.92	62	7	.64
S x H-L x Eye x H	14.36	62	7	.77
S x H-L x Eye x Rep	17.04	62	7	.92
S x H-L x N x H	6.70	62	7	.36
S x H-L x N x Rep	18.17	62	7	.98
S x H-L x H x Rep	6.35	62	7	.34
S x Eye x N x H	54.11	62	7	2.91
S x Eye x N x Rep	26.49	62	7	1.42
S x Eye x H x Rep	42.81	62	7	2.30
S x N x H x Rep	13.10	62	7	.70
S x H-L x Eye x N x H	10.89	62	7	.58
S x H-L x Eye x N x Rep	28.42	62	7	1.53
S x H-L x Eye x H x Rep	12.16	62	7	.65
S x H-L x N x H x Rep	9.15	62	7	.49
S x Eye x N x H x Rep	9.78	62	7	.53
S x H-L x Eye x N x H x Rep	18.61		7	

*p < .05
**p < .01

Table 15

Correlation Coefficients/$\underline{p}$ Values for the EO Data

	Alpha Level	HR	EMG	Alpha Condition
Alpha Level	1.00	−.21 .0174	+.32 .0002*	+.51 .0001*
HR		1.00	+.18 .0424	−.09 .3218
EMG			1.00	−.02 .8095
Alpha Condition				1.00

*$\underline{p}$ < .001

Table 16

Correlation Coefficients/$\underline{p}$ Values for the EC Data

	Alpha Level	HR	EMG	Alpha Condition
Alpha Level	1.00	-.05 .5829	-.30 .0007*	+.56 .0001*
HR		1.00	+.30 .0006*	-.10 .2737
EMG			1.00	-.08 .3603
Alpha Condition				1.00

*$\underline{p}$ < .001

Table 17

Multiple Regression ANOVA Table for the Composite Data

Source	df	SS	MS	F	p	r^2
Regression	2	269.87	134.94	3.00	0.028	0.0276
Error	253	9495.81	37.53			
Corrected Total	255					

	Standard Error	Alpha Mean
	6.126	13.713

Source	df	Sequential SS	F	p	Partial SS	F	p
HR	1	207.87	5.54	0.0194	250.48	6.67	.0103
EMG	1	62.01	1.65	0.1999	62.01	1.65	.1999

Table 18

Multiple Regression ANOVA Table for the EO Data

Source	df	SS	MS	F	p	r^2
Regression	2	902.29	451.15	13.36	0.0001	0.176
Error	125	4220.74	33.77			
Corrected Total	127	5123.04				

	Standard Error	Alpha Mean
	5.811	13.000

Source	df	Sequential SS	F	p	Partial SS	F	p
HR	1	225.65	6.68	0.0109	378.32	11.20	.0011
EMG	1	676.65	20.04	0.0001	676.65	20.04	.0001

Table 19

Multiple Regression ANOVA Table for the EC Data

Source	df	SS	MS	F	p	r^2
Regression	2	401.74	200.87	6.11	0.0033	0.089
Error	125	4110.63	32.88			
Corrected Total	127	4512.38				

	Standard Error	Alpha Mean
	5.734	14.423

Source	df	Sequential SS	F	p	Partial SS	F	p
HR	1	10.83	0.33	.5671	7.63	0.23	.6308
EMG	1	390.91	11.89	.0008	390.91	11.89	.0008