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To test the hypothesis that voluntary alpha control is at least partly mediated through self-induced changes in cortical activation level and bodily arousal, changes in several physiological indicants of such activity were systematically examined while subjects voluntarily produced and suppressed alpha activity. Physiological indicants consisted of cortical evoked potentials, eye motor activity, neck EMG, and skin conductance.

Three male and three female subjects were pretrained to a specified criterion to produce and suppress alpha activity, using auditory feedback. Following pretraining, each subject participated in four experimental sessions under instructions to either produce or suppress alpha while keeping his eyes closed. Each session consisted of eight alternating three-minute trials of high and low alpha conditions with rest intervals between trials.

During each session EEG activity was recorded monopolarly from the occiput with the reference electrode on the right earlobe. Filtered, integrated EEG consisting of alpha activity only, drove a frequency-modulated power driver connected to a speaker which provided continuous feedback in terms of the pitch of a tone. Cortical evoked potentials based on the presentation of 100 irregularly presented light flahses per trial were recorded with a Computer of Average Transients. Using the appropriate Grass Model 7 preamplifiers, skin conductance was recorded from the first and third fingers of the left hand; EMG from the trapezius neck muscle, and eye motor activity with electrodes placed diagonally across the eyes.

The cortical evoked potentials reflecting visual attention and degree of cortical activation differed consistently between the high and low alpha conditions. The peripheral physiological measures of eye motor activity, EMG, and skin conductance provided further evidence that subjects were more highly activated during the low alpha condition. In general the results of the experiment support the notion that alpha control is partly mediated through self-induced changes in cortical activation and bodily arousal.

PSYCHOPHYSIOLOGICAL CORRELATES OF VOLUNTARY ALPHA CONTROL

by

Roberta Ray Sadler

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Approved by

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APPROVAL PAGE

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CHAPTER I

INTRODUCTION

Much research has been devoted to the alpha rhythm, usually defined as the frequency band of 8-13 c/sec in the EEG. Historically, investigators in the areas of neurophysiology, physiology, and physiological psychology have studied this neuro-electrical activity most intensively. However, in recent years psychologists in the areas of operant conditioning and personality have become interested in such activity in view of preliminary reports by Kamiya and his associates (Kamiya, 1968, 1969; Nowlis & Kamiya, 1970) that human subjects could learn to control these waveforms with operant techniques, and that certain subjective moods accompany this control. Studies by Kamiya indicated that human subjects could control their alpha activity when provided with visual or auditory feedback correlated with the amount of alpha activity in their EEG. The feedback mechanism can be considered either in terms of a cybernetic control system (e.g., Gaardner, 1971) or in terms of reinforcement in an operant conditioning paradigm. The operant model suggests control procedures that could determine the efficacy of the feedback technique in actually altering alpha activity.

Beatty (1971) has examined the question of whether feedback, *per se*, is the effective factor in altering alpha, by manipulating the contingency of such feedback on EEG output level. He employed a yoked control procedure wherein he trained his experimental subjects to increase differentially the abundance of alpha and beta (above 13 c/sec) frequency bands when auditory feedback was contingent on their EEG

activity. The yoked control group receiving feedback which was noncontingent in relation to their own EEG output levels (being the recorded feedback of their matched experimental subjects) failed to show any difference in their EEG activity between the alpha and beta conditions. These results suggest that some kind of contingent sensory feedback is necessary in order for subjects to gain control over their alpha activity.

Other studies have confirmed the fact that with contingent feedback subjects can learn to gain bidirectional control of alpha, either increasing or decreasing alpha at specified times (Beatty, 1972; Dewan, 1967; Hart, 1968; Kamiya, 1968, 1969; Mulholland & Evans, 1965; Nowlis & Kamiya, 1970). Having well established the fact that alpha level can be brought under voluntary control, investigators have been concerned with methodology and techniques to enhance the efficiency with which such control is attained. Thus, investigators have studied the effects of: (a) eyes open vs. eyes closed on alpha production with either continuous or binary (discrete) feedback (Travis, Kondo, & Knott, 1974); (b) auditory feedback by means of presentation of a tone vs. termination of an ongoing tone for criterion alpha (Hord & Barber, 1971); (c) instructional information vs. auditory feedback (Beatty, 1972); and (d) various control procedures such as irrelevant feedback, no feedback, and yoked controls. While the conditions under which individuals can gain some control of their alpha activity have been intensively studied, researchers in this area have virtually ignored the fundamental issue of exactly what producing alpha does for the individual, as well

as the nature and scope of any physiological and psychological changes which accompany alpha production.

Despite the lack of definitive evidence, individuals in therapeutic settings have proposed the use of alpha training in treating certain kinds of behavior problems, in view of reports of pleasure, relaxation, and increased awareness accompanying alpha production (Kamiya, 1968; Nowlis & Kamiya, 1970). If it can be reliably demonstrated that a dominant alpha state is associated with lower levels of affective arousal and a "relaxation of normal perceptual and cognitive control" (Nideffer, 1972, p. 179), then the use of training procedures to enhance voluntary alpha control might indeed be valuable in a variety of therapeutic procedures. Based perhaps more on hope than on empirical evidence of its potential effectiveness, Nideffer (1972) has suggested voluntary alpha control training for schizophrenics as a means to reduce hypothesized stimulus overload. Budzynski and Stoyva (1972) have employed alpha feedback training in behavior therapy in an attempt to help a patient regain a state of calm after an anxiety episode in a desensitization program. Alpha enhancement has been used (unsuccessfully) as a treatment for pain by Gannon and Sternbach (1971), with the rationale that a high alpha state and pain are incompatible. Judging from these and similar kinds of reports, the effectiveness of alpha control training as a therapeutic device has not yet been clearly demonstrated.

The actual evidence for a particular subjective state accompanying alpha is inconsistent. Although Brown (1970, 1971) provides 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 (see Lynch & Paskewitz, 1971). 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' " (p. 433).

Apparently the psychological effects of producing alpha activity depend on what the individual expects. If biofeedback of alpha activity is to have any direct practical applications research is now needed to determine whether particular physiological events are associated with alpha production. Without entering the controversy of whether feedback is theoretically a reinforcer or a source of

information apart from reinforcement, one can use the feedback technique as a means of investigating the nature of the physiological correlates of voluntarily induced changes in alpha level. Training subjects to produce and suppress alpha activity would permit systematic investigation of any physiological concomitants of alpha vs. non-alpha states.

Much of the psychophysiological research reported in the literature has dealt with factors affecting alpha blocking, those factors including level of arousal and/or visual activity. Special emphasis has been given to the effects of visual attention on alpha blocking, since alpha activity is most dominant at and probably originates in the occipital lobe of the brain. In relation to visual motor activity and acuity Oswald (1957, 1959) reported that a loss of ocular fixation and accommodation is accompanied by an increase in the dominance of the alpha rhythm (when the subject is engaged in a task that demands auditory alertness). Hord, Naitoh, and Johnson (1972) found slow frontal EEG activity, due primarily to eye movements, to be less intense during self-regulated high alpha activity than during baselines. The results of these studies suggest that ocular fixation, accommodation, and eye movements are associated with, if not actively involved in the production of, low- or non-alpha states. More direct evidence in support of this conclusion is provided by the following studies. Lehtonen and Lehtinen (1972) found ocular fixation or events related to fixation to interfere actively with the alpha rhythm. When subjects were instructed to fixate on a spot in an otherwise uniform visual field, enduring desynchronization was observed (eye-position or fixation was not

directly measured). Visual perception and attention (flash counting) did not interfere with the alpha rhythm. In an EEG study involving the concurrent electro-oculographic recording of eye movements (EOG), in conjunction with subjects' reports of subjective clarity or apparent clearness of the target, Mulholland and Peper (1971) reported that alpha attenuation or blocking was not due to "'visual attention' but to processes of fixation, lens accomodation, and pursuit tracking" (p. 556). The conclusion that alpha blocking occurred only with oculomotor change was based on the authors' inference that "visual attention" remained relatively constant between tasks that differed only in the presence or absence of oculomotor activity. However, they had no direct measure of visual attention, and such a measure would seem necessary before its influence on alpha activity can definitely be ruled out. The visually evoked potential would seem to be a good candidate for monitoring shifts in visual attention, given its established relationship to attentional processes (Donchin & Cohen, 1967; Eason, Harter, & White, 1969).

Long before the relationships between EPs and attentional processes were established, the alpha rhythm was recognized as an electrophysiological 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 associates (e.g., Lindsley, 1952; Moruzzi & Magoun, 1949). However, other electrophysiological measures of arousal, for example EMG

activity, have not shown a consistent relationship to alpha activity (Kreitman & Shaw, 1965, with forearm muscles; Lehtonen & Lehtinen, 1972, with extrinsic laryngeal muscles). Oswald (1959) has suggested that alpha blocking may represent an increase of "specific visual alertness which may be but one component of general arousal." He found that alpha rhythms occurred at times of intent auditory alertness (and were accompanied by loss of ocular fixation and accomodation). He proposed that specific auditory alertness may be associated with "reciprocal inhibition of visual functions." In line with this, Martinius and Hoovey (1972) reported that acoustic attention, required by a tonal discrimination task, resulted in a quantitative increase in occipital alpha waves over background (resting) alpha rhythms.

There is an obvious need for research more closely examining the physiological correlates of high and low alpha conditions. The biofeedback technique offers a convenient procedure for obtaining discrete time samples of relatively high and low alpha production. Since it has been established that alpha level can be brought under voluntary (bidirectional) control, concommitant changes in other physiological responses could be observed in direct relationship to specified periods of high and low alpha levels. The present study used a feedback technique to obtain experimental conditions of high and low alpha in order to investigate concurrent changes in other physiological events with specific interest directed toward arousal-attentional factors.

To expand on the evidence relating alpha activity to cortical arousal, two peripheral electrophysiological measures of arousal were

chosen, one controlled primarily by the somatic nervous system and one controlled primarily by the autonomic nervous system. These measures were neck EMG and skin conductance, respectively. Since there is much literature establishing a relationship between oculomotor activity and alpha, a measure of general eye motor activity was also obtained.

In order to clarify the relationship between visual attention and alpha activity, visually evoked potentials were obtained during high and low alpha conditions. The visually evoked potentials (EPs) furnished the dependent variable of primary interest, because it provided a direct measure of cortical responses to external stimuli during the experimental conditions of voluntary production of high and low alpha activity.

CHAPTER II

METHOD

Subjects

Five graduate students and one speech therapist (age range from 23 to 26 years) served as subjects. They were chosen from an initial group of 12 individuals for the main part of the study on the basis of their ability to demonstrate during preexperimental practice sessions reliable changes (at least a 20% difference) in alpha production when provided with an auditory feedback tone. The other six individuals were not included because they demonstrated no apparent progress in gaining control of alpha after several practice sessions. Participation in the preexperimental sessions familiarized each subject 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).

Experimental Design

The preexperimental screening sessions functioned as practice sessions for those subjects who were selected to participate in the experiment. These sessions consisted of alternating 2-minute trials of high and low alpha conditions with a 30-second rest period between each trial and the next and a 2-minute rest period following each block of five trials. After every three blocks the subject was asked to get up and walk around for 5 minutes. Practice sessions were continued until

a criterion performance level (at least a 20% difference in the integrated alpha measure between high and low alpha conditions) was reached.[1]

After demonstrating reliable control of alpha, each subject participated in four experimental sessions. Each session consisted of two blocks of four alternating 3-minute trials of high and low alpha conditions. There was a 10-minute rest following each block, during which the subject left the subject's room and walked around. After each of the trials there was a 1-minute rest during which the subject remained seated in the subject's room. The order of trials was counterbalanced for each subject. Each subject ran in no more than one session on a given day.

After at least two sessions each subject was asked to write down his strategy for controlling the feedback tone.

Apparatus

The subject was seated in an electrically shielded, semi-darkened room during the recording session. White noise was fed into the subject's room to mask extraneous sounds. A Grass Model 7 Polygraph equipped with appropriate preamplifiers was used to record the various physiological events, with permanent records obtained on an attached oscillograph.

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[1]Alpha level will be used to refer to the dependent variable of the integrated alpha measure; alpha condition will refer to the experimental manipulation of high vs. low alpha.

EEG and EPs. EEG activity was recorded monopolarly with a Grass gold disc electrode placed 2½ cm above the inion on the midline. The reference electrode was clipped to the right ear lobe. The electrodes were connected to the input of a 7P5 EEG preamplifier, with half-amplitude low and high frequency filters set a 1 and 35 Hz, respectively. The amplified EEG activity was fed to a Computer of Average Transients (CAT, Model 400B) which was triggered by the presentation of a light flash. Each averaged evoked potential (EP) was the sum of individual responses to 100 light flashes. A permanent record of each EP was obtained with a Moseley X-Y plotter (Model 2D-2). The CAT analysis time was 500 msec.

Integrated Alpha. EEG activity was directed through a low-frequency band-pass filter set at 8-13 Hz to a Grass Model 7P3 preamplifier which 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 half-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 seconds, to facilitate quantification.

Eye Motor Activity. Two gold disc electrodes, one placed above and to the right of the right eye, the other below and to the left of the left eye, were used to record eye motor activity. The potentials were integrated by means of a 7P3 preamplifier (time constant, .5 seconds) to obtain a more easily quantifiable measure of eye activity. (The electrode positioning did not yield information about the direction of eye movement. Only general information about the amount of eye movement and muscle tension around the eyes was obtained.) The half-amplitude low- and high-frequency filter settings were 3 and 35 Hz.

In attaching the electrodes to record EEG, EMG and eye motor activity, skin resistence was reduced to less than 10,000 ohms with Redux Electrode Paste. This procedure reduced the possibility of 60-cycle interference.

Skin Conductance. Skin resistance was recorded with silver silver-chloride electrodes attached to the volar surface of the first and third fingers of the left hand. (An exception was one subject who had a wart on the recording surface of the third finger. For this person the second finger was used.) A Grass 7P1 preamplifier was used to amplify resistance changes and to balance the resistance bridge. The resistance level (expressed in ohms) was derived from the calibrated polygraph records for the beginning and end of each trial with an intermediate measurement being the average of the resistance levels at the beginning and end of the second 1-minute interval. The resistance values were converted to conductance values (micromhos) before subjecting the data to statistical analysis.

Auditory Feedback System. Through the use of solid state logic modules, a continuous auditory tone was programmed to vary in frequency as a function of the voltage level of the integrated alpha measure. For three subjects an increase in pitch indicated an increase in alpha; for the other three 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.

Visual Stimulus. The EPs were evoked by the presentation of unpatterned light flashes generated by a Grass PS-2 photostimulator (with flash intensity set at 2). The distance from subjects' eyes to the stimulus display was approximately 122 cm. Because the subjects sat with eyes closed throughout the recording sessions the visual stimuli were further diffused by the eyelids, giving them a true Ganzfeld quality. The light flashed at irregular intervals throughout each trial, with a minimum interval of 1075 msec. The longest interstimulus intervals may have approached, but probably never exceeded, 2 seconds. The averaged EPs were based on the sum of the responses to the first 100 light flashes within each trial; more than the required 100 for averaging generally occurred during a 3-minute trial.

Procedure

Three of the subjects were informed that a high-pitched tone reflected low alpha activity and that a low-pitched tone reflected high alpha activity. Instructions for the low alpha condition were, "Try to keep the tone high; try not to produce alpha;" for the high alpha

condition, "Try to keep the tone low; try to produce alpha." The other three subjects were informed that a low-pitched tone reflected low alpha activity and that a high-pitched tone reflected high alpha activity. Instructions for the low alpha condition were, "Try to keep the tone low; try not to produce alpha;" for the high alpha condition, "Try to keep the tone high; try to produce alpha." All subjects were instructed to keep their eyes closed during recording periods.

CHAPTER III

RESULTS

The reader will recall that each subject ran in four experimental sessions, each consisting of four trials of high alpha and four trials of low alpha. Each trial was divided into three 1-minute intervals to facilitate visual average measurements and to gain information about temporal changes within a trial. A "best fit" visual average per 1-minute interval for the integrated eye motor activity, EMG, and alpha polygraph records was obtained, after it had been established that this could be done with a consistency of ±.5mm by the author. The mm measurements were then converted to microvolts (μv) for statistical analysis. For skin conductance (SC), measurements (in ohms) were made at the beginning and end of each trial and at the beginning of the second and third 1-minute intervals. The latter two were collapsed into a single measure by averaging across them. The resulting three measurements were then converted to conductance (micromhos).

Each of these physiological measures was analyzed both for the group and for individual subjects by means of univariate analyses of variance (ANOVAs). For individual subjects, the factors consisted of alpha condition (H-L), sessions, trials, and 1-minute intervals (2x4x4x3). The group analyses contained an additional (subjects) factor. The EP data for each subject were analyzed by visual inspection.

Alpha Level, Eye Motor Activity, EMG, and SC

GROUP RESULTS

Table 1 summarizes the significant effects obtained from the group analyses of the integrated alpha measure, eye motor activity, EMG, and SC. The significant effects not involving the subject factor are depicted in Figure 1 with integrated alpha and SC plotted as functions of 1-minute intervals and trials for the high and low alpha conditions. Since group analyses of the eye motor activity and EMG data revealed no significant effects except interactions involving subject factors, these data are not presented graphically.

The alpha activity presented in Figure 1 shows the consistent difference between the high and low alpha conditions that was required by the experimental task. The H-L effect was significant at $p < .009$. The nature of the trials effect can be seen in the figure by visualizing the average of the functions for the high and low alpha conditions for each trial. The only noticeable change is a slight increase in alpha on the third trial, which followed the mid-session break. The change in alpha across 1-minute intervals within trials depended upon the experimental conditions of high or low alpha; the H-L X 1-Minute Intervals interaction was significant at $p < .002$. Viewing the graphic data, it appears that during the low alpha condition alpha activity tended to increase, and during the high alpha condition alpha activity perhaps tended to decrease slightly.

From the graph of the SC data presented in Figure 1, the most obvious effect is the decrease in SC across 1-minute intervals; this

Table 1

Summary of the Significance Levels (p-values) From the Group Analyses of Variance of the Integrated Alpha Measure, Eye Motor Activity, EMG, and SC

Source	Alpha	Eye	EMG	SC
H-L	.009			
Ses				
Trials	.001			.003
Min Int				.001
H-L X Ses				
H-L X Tr				
Ses X Tr				
H-L X Min Int	.002			
Ses X Min Int				
Tr X Min Int				.004
H-L X Ses X Tr				
H-L X Ses X Min				
H-L X Tr X Min Int				
Ses X Tr X Min Int				
H-L X Ses X Tr X Min Int				
S X H-L	.001	.001	.001	.001
S X Ses	.001	.001	.001	.001
S X Tr	.001	.001	.001	.001
S X Min	.001		.001	.001
S X H-L X Ses	.001	.001	.001	.036
S X H-L X Tr	.001	.001	.001	.006
S X Ses X Tr	.001	.001	.001	.001
S X H-L X Min Int		.002	.001	
S X Ses X Min Int	.010			.001
S X Tr X Min Int				
S X H-L X Ses X Tr	.001	.001	.001	.001
S X H-L X Ses X Min Int				
S X H-L X Tr X Min Int			.047	
S X Ses X Tr X Min Int			.018	

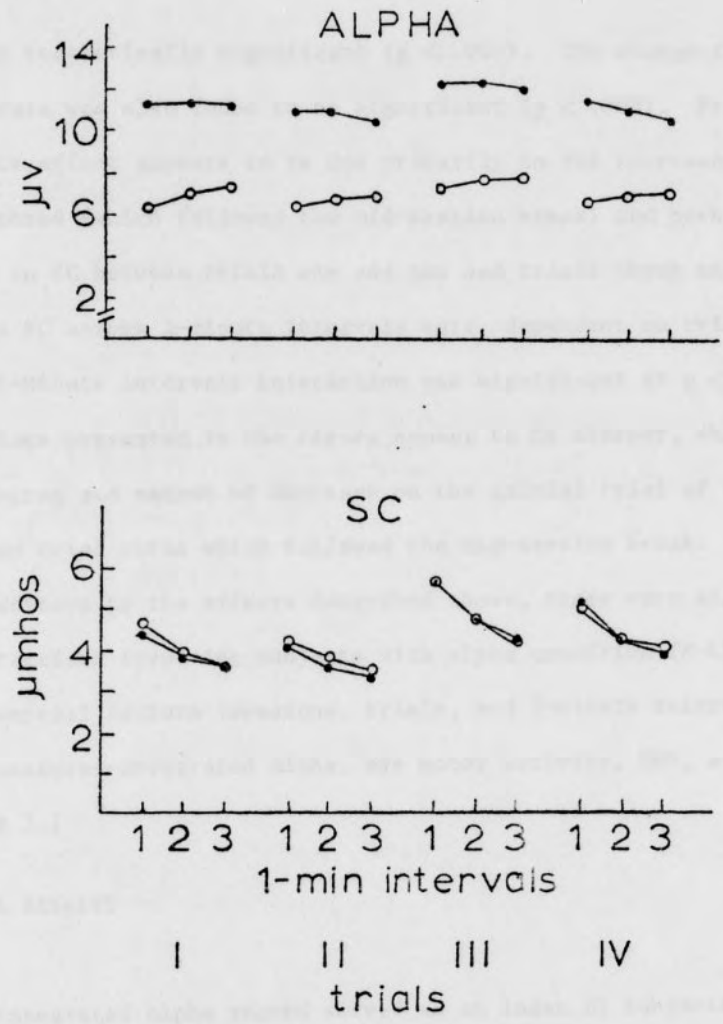


Figure 1. Group changes in alpha level and SC as a function of alpha conditions, 1-minute intervals, and trials. Open circles represent the low alpha condition (o—o); filled circles represent the high condition (•—•).

effect was statistically significant ($p < .001$). The change in SC across trials was also found to be significant ($p < .003$). From the figure this effect appears to be due primarily to the increase in SC on trial three (which followed the mid-session break) and perhaps to decreases in SC between trials one and two and trials three and four. Changes in SC across 1-minute intervals were dependent on trials--the Trials X 1-Minute Intervals interaction was significant at $p < .004$. The functions presented in the figure appear to be steeper, showing a greater degree and extent of decrease on the initial trial of the session and on trial three which followed the mid-session break.

In addition to the effects described above, there were significant interactions involving subjects with alpha condition (H-L) and/or various temporal factors (sessions, trials, and 1-minute intervals) for each measure--integrated alpha, eye motor activity, EMG, and SC. (See Table 1.)

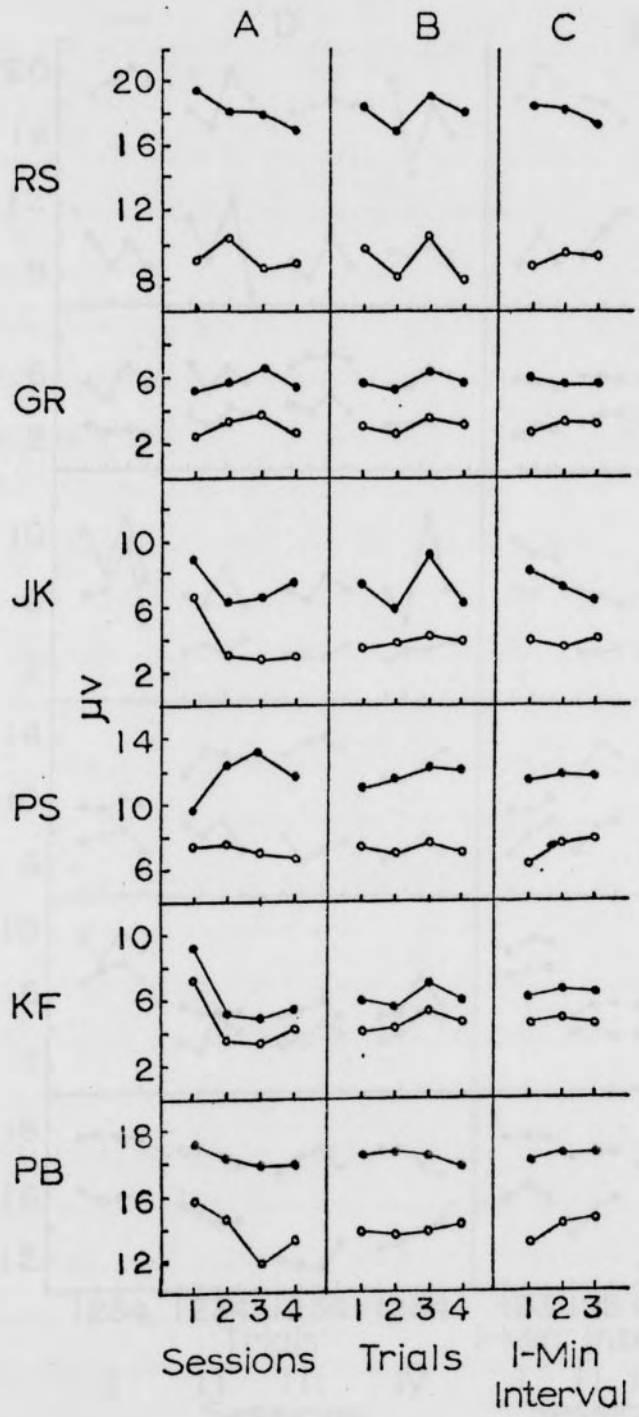
INDIVIDUAL RESULTS

Alpha

The integrated alpha record served as an index of subjects' ability to follow instructions to generate high or low alpha, and provided evidence for the effectiveness of the experimental manipulation. Figure 2 shows alpha level, with alpha conditions as a parameter and with the following time bases on the abscissa: (a) sessions in graphs A, (b) trials in graphs B, (c) 1-minute intervals in graphs C, (d) sessions and trials in graphs D, and (e) sessions and 1-minute intervals

A
B
C

Figure 2. Alpha level under high and low conditions for each S as a function of (A) sessions, (B) trials, (C) 1-minute intervals, (D) trials and sessions, and (E) 1-minute intervals and sessions. Open circles represent the low alpha condition (o—o); filled circles represent the high condition (•—•).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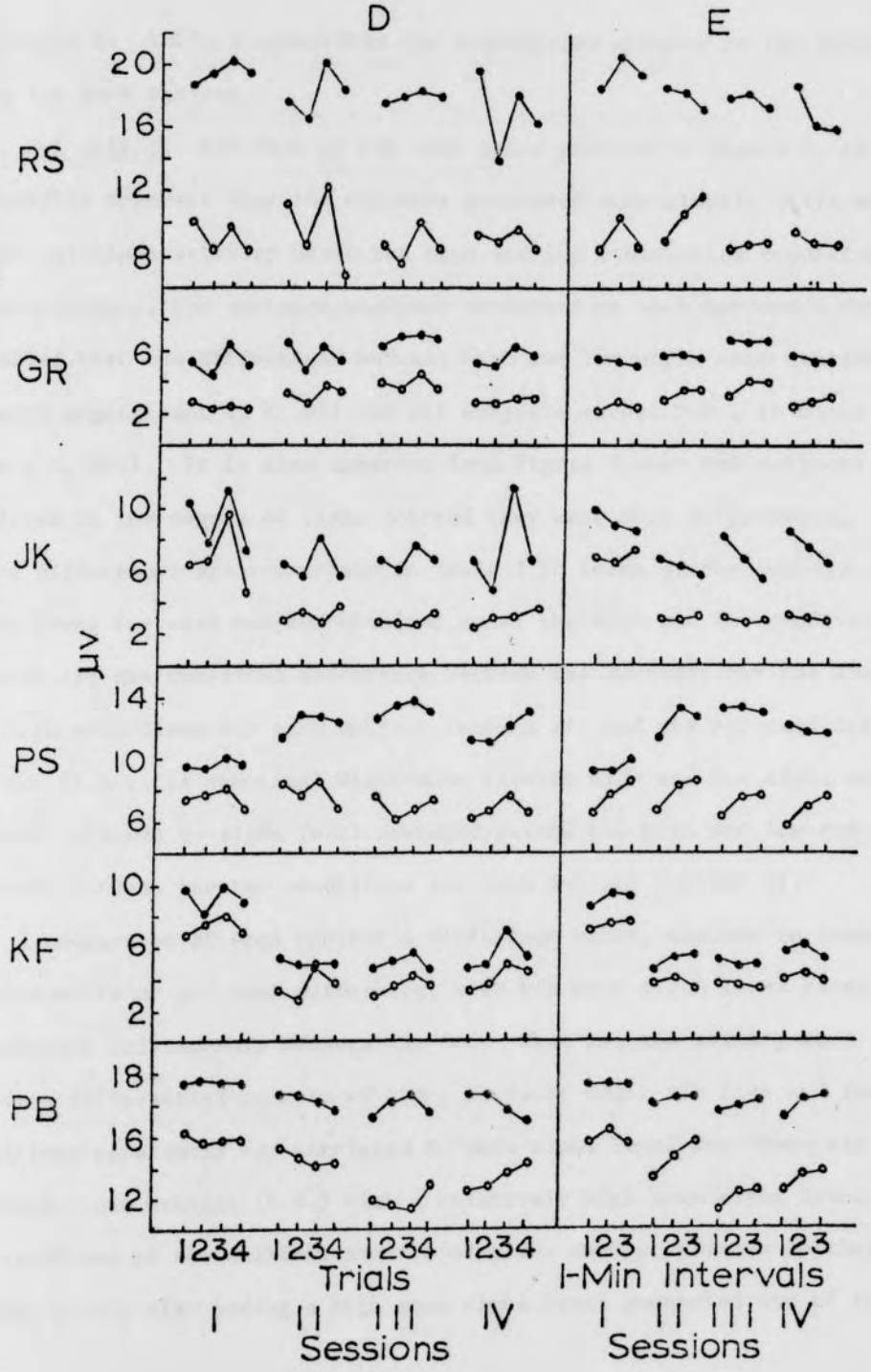
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1-Min Intervals
I II III IV
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in graphs E. Table 2 summarizes the significant effects in the alpha data for each subject.

H-L effect. For each of the time bases plotted in Figure 2, it is readily apparent that the subjects generated consistently different levels of alpha activity under the high and low instruction conditions. Unsurprisingly, the variance analyses performed on each subject's data revealed that the differences between high and low alpha were statistically significant ($p < .001$ for all subjects except P.S., in whose case $p < .002$). It is also apparent from Figure 2 that the subjects differed in the degree of alpha control they were able to generate. These differences are summarized in Table 3 in terms of the average alpha level for each subject obtained under the high and low conditions (column 1); the numerical difference between the averages for the low and high conditions for each subject (column 2); and the per cent difference (i.e., the numerical difference between high and low alpha expressed relative to alpha level averaged across the high and low conditions) between the two conditions for each subject (column 3).

A comparison of each subject's difference score, whether in terms of microvolts or per cent difference, with his mean alpha level reveals no apparent relationship between the two. That is, the ability to generate differential amounts of alpha activity under the high and low conditions apparently was unrelated to mean alpha level for these six subjects. One subject (P.B.) with a relatively high mean alpha level generated one of the smallest amounts of alpha change, whereas another subject (R.S.) also having a high mean alpha level generated one of the

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Table 2

Summary of the Significance Levels (p-values) Obtained From the Individual Analyses of Variance of the Integrated Alpha Measure for Each S

Source	R.S.	G.R.	J.K.	P.S.	K.F.	P.B.
H-L	.001	.001	.001	.002	.001	.001
Ses		.001	.001		.001	
Trials	.001	.002	(.069)		.005	
Min Int				.048		.003
H-L X Ses	.004			.001		.016
H-L X Tr			(.055)			
Ses X Tr						
H-L X Min Int	.040	.001	.001	.001		
Ses X Min Int	.040			.009		
Tr X Min Int					.014	
H-L X Ses X Tr		.001	.001	.015	.003	
H-L X Ses X Min Int					.043	
H-L X Tr X Min Int						
Ses X Tr X Min Int						

Table 3

Rank Order of the Ss in Terms of (1) the Mean Level in μv of Alpha (High + Low/2), (2) the Absolute Difference in μv Between High and Low Alpha, and (3) the Per Cent Difference Between High and Low Alpha (Absolute Difference/Mean)

S	Mean Alpha (μv) H+L/2	S	Absolute Difference Between H and L (μv)	S	Per Cent Difference (Abs Diff/Mean)
P.B.	16.16	R.S.	8.80	R.S.	.64
R.S.	13.77	P.B.	4.44	G.R.	.62
P.S.	9.49	P.S.	4.41	J.K.	.60
J.K.	5.70	J.K.	3.44	P.S.	.46
K.F.	5.46	G.R.	2.75	K.F.	.28
G.R.	4.46	K.F.	1.53	P.B.	.27

highest levels of alpha change. The same kind of dissociation exists for the two subjects having the lowest mean alpha levels, one of them (K.F.) having generated a relatively low degree of alpha change and the other (G.R.) a relatively high degree of change. Of the remaining two subjects, the one with a moderate alpha level (P.S.) generated an intermediate per cent change score while the one with a relatively low mean alpha level (J.K.) manifested a relatively high degree of alpha control.

Temporal main effects. Figure 2A shows the effects of sessions on alpha level for each subject. Although there are apparent changes across sessions for all subjects, the variance analyses revealed significant effects only for subjects G.R., J.K., and K.F. ($p < .001$). Even though significant effects were obtained for these three subjects, it is clear from the figure that the direction and degree of the changes are specific to each individual. Thus G.R. showed a progressive increase followed by a decrease whereas J.K. and K.F. both showed a marked decrease between the first and second sessions with little or no change throughout the remaining sessions.

The trials effect, shown in Figure 2B, was significant for R.S. ($p < .001$), G.R. ($p < .002$), and K.F. ($p < .005$), and approached significance for J.K. ($p < .07$). As the figure indicates, this difference is characterized by an S-shaped curve generated by a decrease in alpha level between the two prebreak and postbreak trials and a sharp increase between trials two and three, which were separated by the mid-session break.

The changes within trials (i.e., across 1-minute intervals) are shown in Figure 2C. The apparent changes depicted in these graphs were significant only for P.S. ($p < .048$) and P.B. ($p < .003$). Both of these subjects show a similar pattern of change. Visualizing the average of the high and low data, it is apparent from the graphs that the significant effect found for these two subjects reflects a general increase in alpha level during the 3-minute trial.

First-Order Interactions. A significant H-L X Sessions interaction was found for R.S. ($p < .004$), P.S. ($p < .001$), and P.B. ($p < .016$); however, as Figure 2A indicates, the nature of the interaction was not consistent for the three subjects. For R.S., the interaction effect appears to reflect a progressive convergence of the two curves over the four sessions, whereas the effect appears to be due primarily to a divergence of the two functions across sessions for the other two subjects.

Alpha level changed across the 1-minute intervals differentially for high and low alpha conditions. Figure 2C depicts this pattern of activity. The H-L X 1-Minute Intervals interaction was significant for four of the six subjects; R.S. at $p < .04$, and G.R., J.K., and P.S. at $p < .001$. Referring to the graphic data, one can surmise that the interaction effect for R.S. is due primarily to a drop in alpha during the course of the trial under the high alpha condition and to an increase over the 1-minute intervals for the low alpha condition. For G.R. and P.S., the interaction effect reflects the fact that alpha level remained essentially constant during the high alpha condition

but increased progressively across the 1-minute intervals during the low alpha condition. For J.K. the interaction effect reflects a convergence of the two functions across the 1-minute intervals due primarily to a decrease in alpha level during the high alpha condition, with no substantial change under the low condition.

Figure 2E depicts changes across 1-minute intervals and sessions. Subjects R.S. and P.S. showed significant Sessions X 1-Minute Intervals interactions for alpha at $p < .04$ and $p < .009$, respectively, though the nature of these effects were different for the two subjects. Again visualizing the average function for high and low conditions, one can observe from the graphs for R.S. an inverted-V pattern across 1-minute intervals during session one, essentially flat functions during sessions two and three, and a decreasing function during session four. For P.S., one can observe a general increase in alpha within trials for each of the sessions, but the degree of increase becomes progressively less from session to session.

Because a significant Trials X 1-Minute Intervals interaction was found for only one subject (K.F.; $p < .014$), this effect is not presented graphically. However, the effect may be described verbally as reflecting a tendency for alpha level to increase during the course of trial one, remain constant during trials two and three, and decrease during trial four.

Second-order interactions. The H-L X Sessions X Trials effect can be seen in Figure 2D. This interaction was significant for four of the six subjects; G.R. ($p < .001$), J.K. ($p < .001$), P.S. ($p < .015$), and

K.F. ($p < .003$). The complex patterns manifested in Figure 2D indicate that alpha level did not vary simply as a function of sessions, trials within sessions, or conditions of high and low alpha, but was specifically dependent on which condition existed within a particular trial of a particular session.

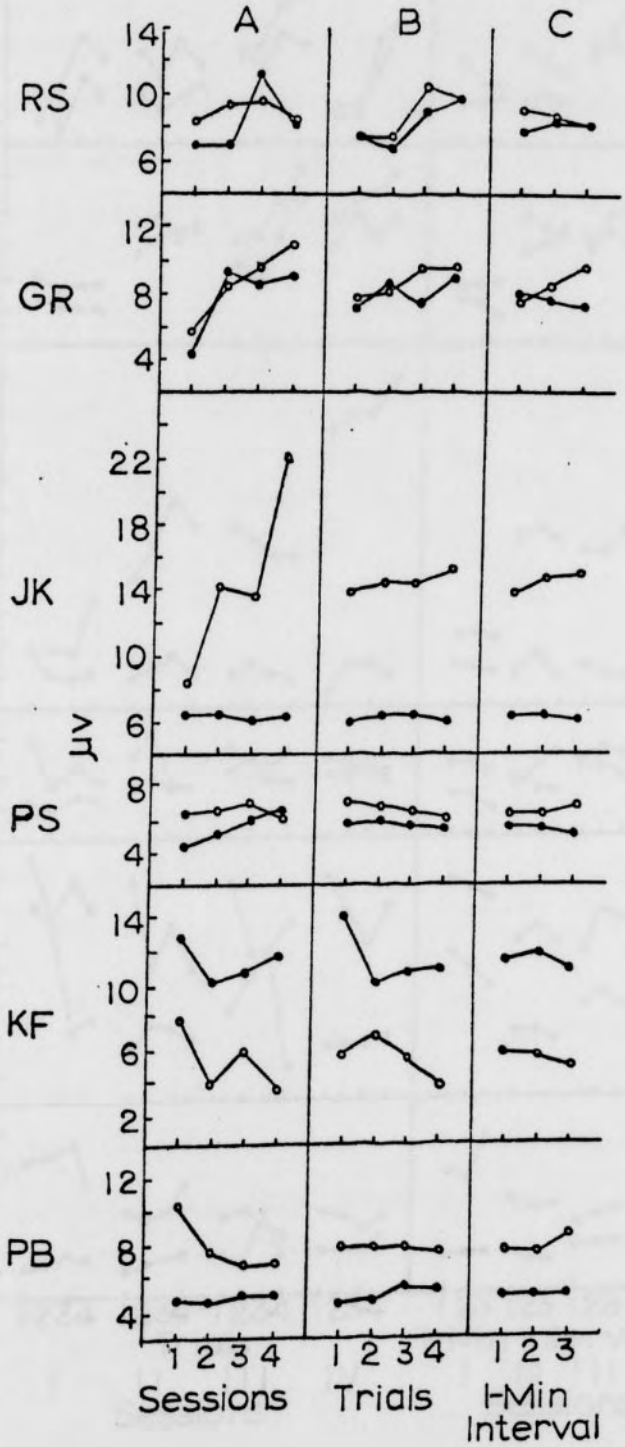
Another significant effect involved a second-order interaction between alpha level, sessions, and 1-minute intervals for K.F. ($p < .043$). This effect can be seen in Figure 2E. This complex picture also indicates that the difference in alpha for high and low conditions was dependent for this subject on sessions as well as 1-minute intervals within each session.

Eye Motor Activity

Figure 3 shows eye motor activity as a function of sessions, trials, 1-minute intervals, sessions and trials, and sessions and 1-minute intervals for each subject (in a fashion similar to the presentation of alpha in Figure 2). Table 4 summarizes the significant effects found from the analyses of the eye motor activity for each subject.

H-L Effect. Examining Figure 3, it is apparent that the subjects differed from each other with respect to both the direction and degree to which eye motor activity was affected by the high and low alpha conditions. Also, the subjects differed with respect to temporal changes in such activity for the high and low alpha conditions. Despite these temporal variations, the degree of eye motor activity under the high and low alpha conditions differed significantly for

Figure 3. Eye motor activity under high and low conditions for each S as a function of (A) sessions, (B) trials, (C) 1-minute intervals, (D) trials and sessions, and (E) 1-minute intervals and sessions. Open circles represent the low alpha condition (o—o); filled circles represent the high condition (•—•).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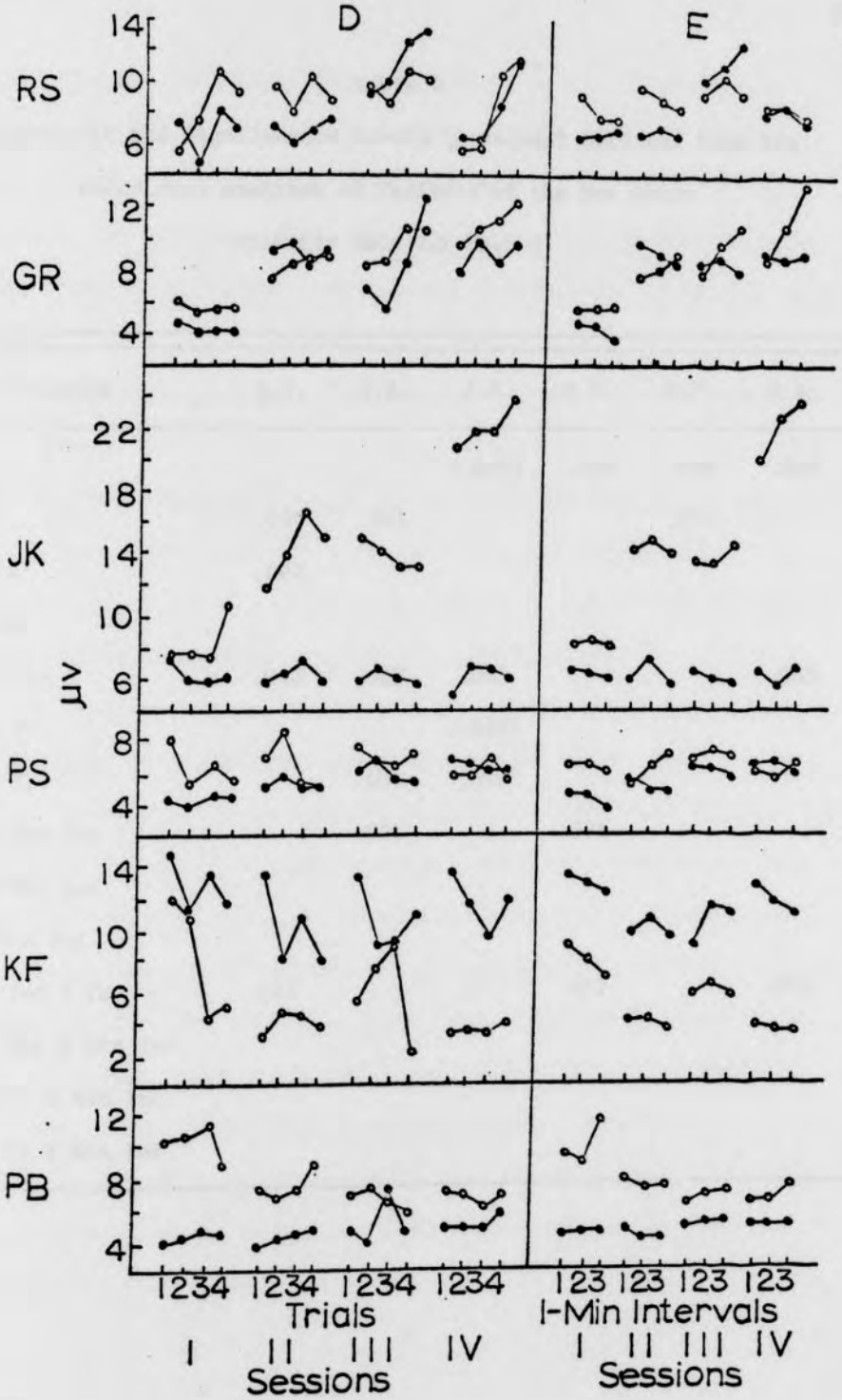
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Trials
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1-Min Intervals
I II III IV
Sessions

Table 4

Summary of the Significance Levels (p-values) Obtained From the Individual Analyses of Variance of the Eye Motor Activity Data for Each S

Source	R.S.	G.R.	J.K.	P.S.	K.F.	P.B.
H-L			(.070)	.050	.020	.020
Ses	.020	.001			.003	
Trials	.002					
Min Int						
H-L X Ses	.040	.005	.001			.002
H-L X Tr			(.060)			
Ses X Tr		.001	.007			
H-L X Min Int		.001		.003		
Ses X Min Int						
Tr X Min Int						
H-L X Ses X Tr	.023			.002		.001
H-L X Ses X Min Int						
H-L X Tr X Min Int						
Ses X Tr X Min Int						

three of the six subjects (K.F., $p < .02$; P.S., $p < .05$; and P.B., $p < .02$), and approached significance at the .05 level for another (J.K., $p < .07$). For three of these subjects (P.S., P.B., and J.K.) eye motor activity was greater during the low alpha condition, but for K.F. such activity was significantly greater during the high condition.

Temporal Main Effects. Changes in eye motor activity across sessions can be seen in Figure 3A by visualizing the average of the functions for high and low alpha conditions. The sessions effect was statistically significant for three subjects, R.S. ($p < .02$), G.R. ($p < .001$), and K.F. ($p < .003$). The nature of these changes were specific to the individual subject as shown in Figure 3A. For R.S. the significant effect appears to reflect primarily the relatively high degree of eye motor activity under the low alpha condition during the third session, whereas for G.R. it reflects a general increase in motor activity across the four sessions under both conditions. In contrast to both R.S. and G.R., the significant effect noted for K.F. may be characterized as reflecting a general decrease across the four sessions. In the case of J.K. a significant sessions effect was not found even though there was a marked increase in eye motor activity across sessions under the low alpha condition. A cursory inspection of Figure 3A for this subject suggests that any such main effect may have been masked by the marked H-L X Sessions interaction which clearly exists (to be described below).

The changes in eye motor activity with trials are shown in Figure 3B. The trials effect reached significance for only one subject R.S. ($p < .002$). In this case there is a noticeable increase in eye motor activity on the third trial following the mid-session break.

First-order interactions. Eye motor activity changed as a function of alpha level condition and sessions as depicted in Figure 3A. The H-L X Sessions interaction was significant for four of the subjects, R.S. ($p < .04$), G.R. ($p < .005$), J.K. ($p < .001$), and P.B. ($p < .002$). The pattern of changes reflected in Figure 3A was different for each of these subjects. For R.S., eye motor activity changed little across sessions during the low alpha condition, but during the high alpha condition there was a sharp increase in such activity on the third session, a circumstance generating the significant interaction. For G.R. eye motor activity increased progressively across sessions for both high and low alpha conditions, but the pattern of increase was markedly different for the two conditions, being approximately linear under the low condition and negatively accelerated under the high condition. For J.K. the interaction effect is characterized by an obvious progressive divergence of the two curves across sessions, due to the marked increase in eye motor activity across sessions under the low alpha condition, with little or no change under the high condition. For P.B. the effect reflects a progressive convergence of the curves over session, primarily due to a tendency for eye motor activity to decrease under the low alpha condition.

The changes in eye motor activity across trials interacted with sessions for two subjects, G.R. ($p < .001$) and J.K. ($p < .007$). The pattern of changes across sessions and trials can be seen in Figure 3D by visualizing the average of the functions for the high and low alpha conditions in each case. For G.R. there was a tendency for the change across trials to become more pronounced in later sessions. Thus, in session one, eye motor activity remained essentially constant across trials; in session two, there was a slight tendency for such activity to increase; and during sessions three and four, there was a marked increase across trials. For J.K. the effect may be characterized as reflecting changes across trials which differed from session to session, with no systematic pattern of change being manifested across the four sessions.

Figure 3C depicts eye motor activity for the high and low alpha conditions as a function of 1-minute intervals. The two subjects for whom the H-L X 1-Minute Intervals interaction was significant, G.R. ($p < .001$) and P.S. ($p < .003$), show a similar pattern of differential change. As depicted, there is a progressive increase in eye motor activity for the low alpha condition and a progressive decrease for the high alpha condition. (The H-L X 1-Minute Intervals interaction approached significance for R.S., $p < .06$, with the pattern of eye motor activity apparently the opposite of that described for G.R. and P.S.)

Second-order interactions. A H-L X Sessions X Trials interaction, manifested in Figure 3D, was significant for three of the six subjects

(R.S., $p < .023$; P.S., $p < .002$; and P.B., $p < .001$). As indicated by the graphs presented in this figure, the direction and degree of difference in eye motor activity between the high and low alpha conditions were dependent on the particular trial of a particular session for each of the subjects, the nature of these dependencies being specific to each subject.

EMG

The EMG data for each subject are plotted in Figure 4. The significant differences found from the statistical analyses of the EMG data for each subject are presented in Table 5.

H-L Effect. The subjects showed different patterns of EMG activity across the various time periods for the high and low alpha conditions, as depicted in Figure 4. For subjects R.S., P.S., and J.K., EMG activity, when averaged across all of the time bases plotted in the figure, was reliably greater during the low alpha condition ($p < .002$, .003, and .004, respectively) than during the high.

Temporal Main Effects. EMG changes over sessions are shown in Figure 4A. The main sessions effect may be visualized by perceptually averaging the functions for high and low alpha conditions. The sessions effect was significant for four subjects, R.S. ($p < .05$), G.R. ($p < .002$), P.S. ($p < .039$), and P.B. ($p < .001$). Viewing the graphic data, it is apparent that the pattern of change across sessions was idiosyncratic. For R.S. there was an S-shaped pattern reflecting a decrease in EMG between sessions one and two and between sessions three and four, with an increase on session three. For G.R. there was a


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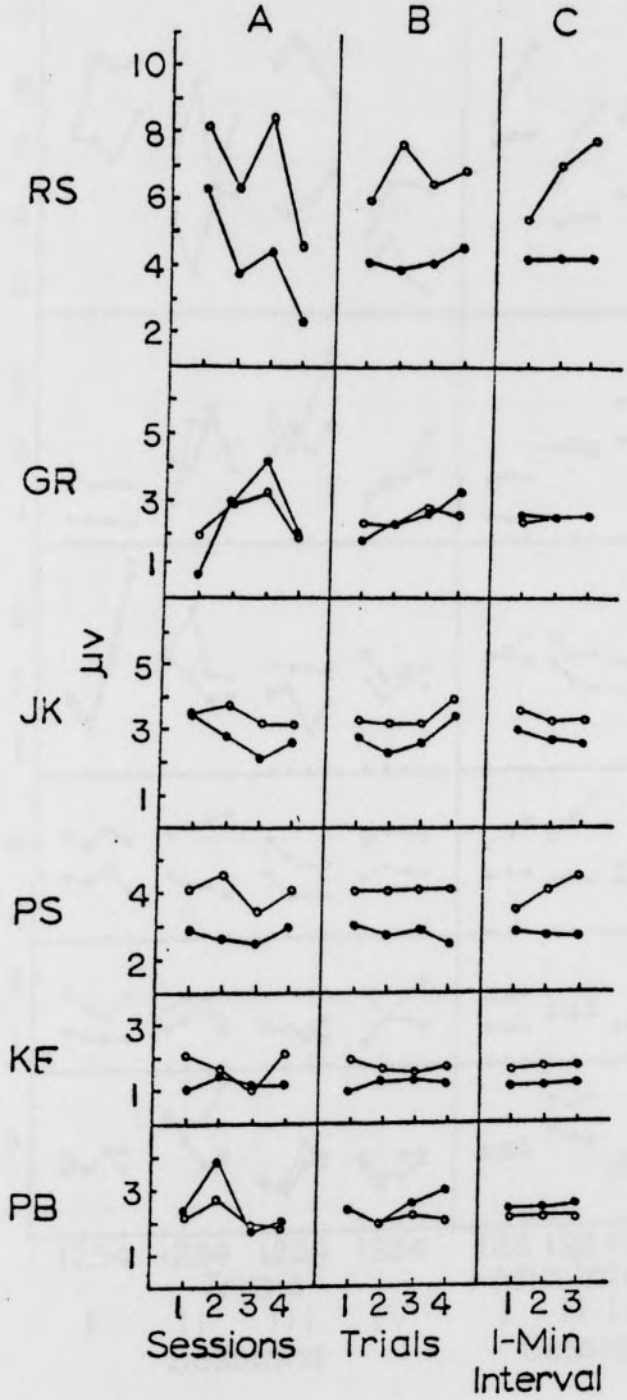
Figure 4. EMG under high and low conditions for each S as a function of (A) sessions, (B) trials, (C) 1-minute intervals, (D) trials and sessions, and (E) 1-minute intervals and sessions. Open circles represent the low alpha condition (o—o); filled circles represent the high condition (•—•).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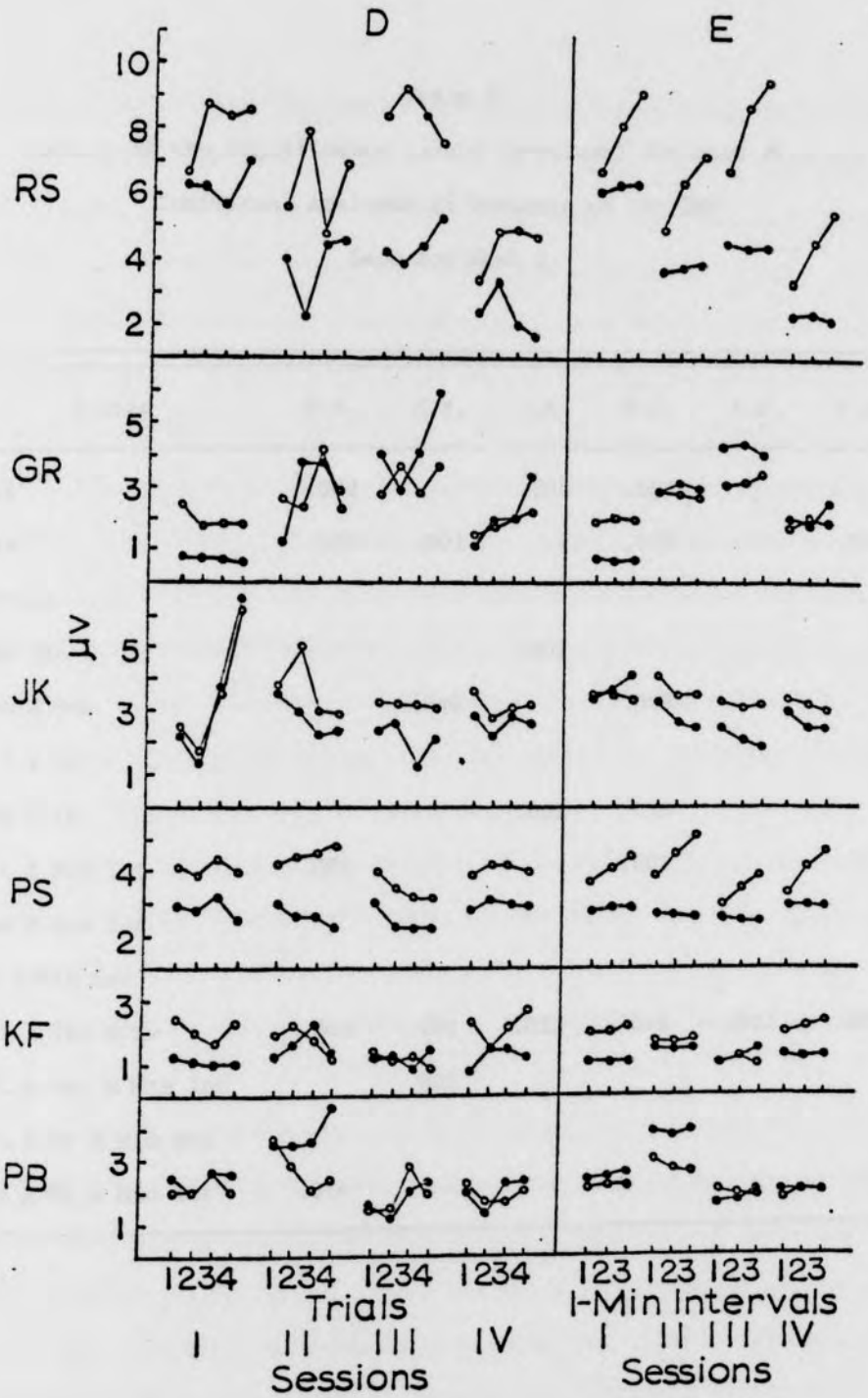
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I II III IV
Sessions

Table 5

Summary of the Significance Levels (p-values) Obtained From the Individual Analyses of Variance of the EMG Data for Each S

Source	R.S.	G.R.	J.K.	P.S.	K.F.	P.B.
H-L	.002		.004	.003		
Ses	.050	.002		.039		.001
Trials						
Min Int			.003			
H-L X Ses		.040		.003		
H-L X Tr						
Ses X Tr			.001	.025		
H-L X Min Int	.001			.001		
Ses X Min Int						
Tr X Min Int						
H-L X Ses X Tr	.001	.001	.023	.040	.001	.001
H-L X Ses X Min Int		.001				
H-L X Tr X Min Int						
Ses X Tr X Min Int	.016					

progressive increase in EMG across the first three sessions followed by a decrease in the final session. For P.S. there was a progressively mild decrease in EMG across the first three sessions followed by an increase in the fourth, generating a shallow U-shaped function. For P.B. an S-shaped function was generated which mirror-imaged that of R.S.

The simple 1-minute intervals effect for EMG was significant for only one subject, J.K. ($p < .003$). The data depicted in Figure 4C indicate that for this subject EMG tended to decrease during the course of a trial. Viewing the graphs in this figure for the other subjects there is an apparent change across 1-minute intervals for subjects R.S. and P.S. However, this change did not gain significance, due to the magnitude of the interaction with the H-L factor described below.

First-order interactions. The interaction of alpha level with sessions for the EMG data was significant for G.R. ($p < .04$) and P.S. ($p < .003$). The interaction as depicted in Figure 4A reflected different patterns of change for these two subjects. For G.R. there was a greater degree of change across sessions under the high than under the low alpha condition, whereas for P.S. just the opposite was the case.

A significant Sessions X Trials interaction was found for J.K. ($p < .001$) and P.S. ($p < .025$). One can see this interaction effect by visualizing the average of the functions for high and low alpha conditions in Figure 4D and noting the differential change across trials for each session. The graph for J.K. indicates a substantial increase in EMG across trials in session one, followed by little or

no change in the remaining three sessions. For P.S. the interaction appears to reflect the general decrease in EMG across trials in session three as compared to relatively little change within the other three sessions.

A statistically significant H-L X 1-Minute Intervals interaction in EMG was found for two subjects, R.S. and P.S. ($p < .001$). Figure 4C for these two subjects depicts this interaction. It can be seen upon inspection of this graph that EMG increased within trials during the low alpha condition but not during the high alpha condition.

Second-order interactions. The changes in EMG as a function of alpha level, as well as sessions and trials within each session, is depicted in Figure 4D for each subject. All six of the subjects showed significant H-L X Sessions X Trials interactions (for R.S., G.R., K.F., and P.B., $p < .001$; for J.K., $p < .023$; and for P.S., $p < .04$). As indicated by the figure, the direction and degree of difference in EMG between the high and low alpha conditions were dependent on the particular trial of a particular session.

For one subject, G.R., the direction and degree of difference in EMG between the high and low alpha conditions were also dependent on sessions and on 1-minute intervals within each session. This second-order interaction is depicted in Figure 4E for this subject. (The H-L X Sessions X 1-Minute Intervals interaction was significant for G.R. at $p < .001$.)

EMG was significantly affected by sessions, trials, and 1-minute intervals in only one case (R.S., $p < .016$). This interaction is not

depicted graphically but can be described verbally as reflecting progressive increases in EMG activity across 1-minute intervals with the degree of increase being dependent on sessions as well as trials within each session.

SC

The changes in SC during the high and low alpha conditions for sessions, trials, 1-minute intervals, sessions and trials, and trials and 1-minute intervals are presented in Figure 5. Significant effects revealed by the analyses of the SC data for each subject are summarized in Table 6.

H-L Effect. Viewing the various functions plotted in Figure 5, it is apparent that in general there were no consistent differences in SC between the high and low alpha conditions. Only one subject (G.R.) showed a significant difference in SC as a function of alpha condition. In this case SC was greater during the low alpha condition ($p < .008$).

Temporal Main Effects. Figure 5A illustrates the changes in SC across sessions for each subject, all of which were significant ($p < .001$ for G.R., J.K., K.F., and P.B.; and $p < .002$ for R.S. and P.S.). The nature of these changes as reflected in Figure 5A was specific to the individual subject. For R.S., SC was relatively high during session three, whereas for G.R. and P.B. it was at a relatively high level during session two. For P.S. and K.F. the graphic data reflect a progressive decrease in SC across early sessions, with an apparent increase during later sessions. For J.K., SC tended to increase over sessions.

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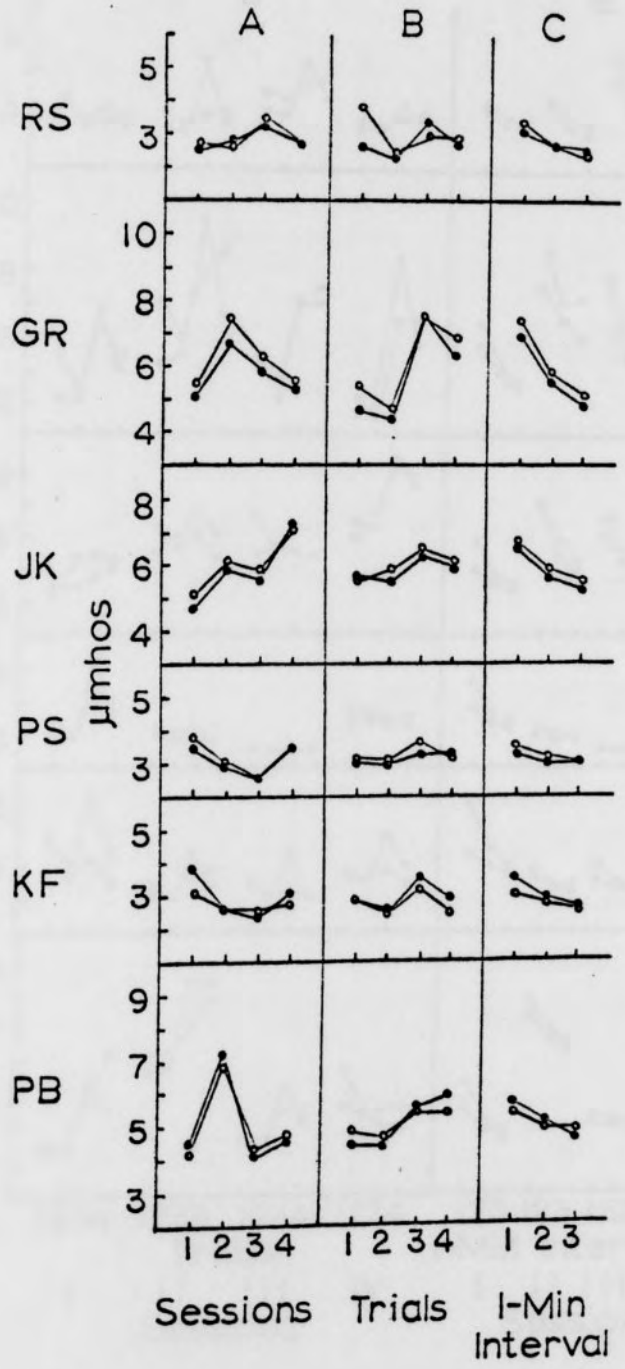
Sessions
Trials
Interval

Figure 5. SC under high and low conditions for each S as a function of (A) sessions, (B) trials, (C) 1-minute intervals, (D) trials and sessions, and (E) 1-minute intervals and sessions. Open circles represent the low alpha condition (○—○); filled circles represent the high condition (●—●).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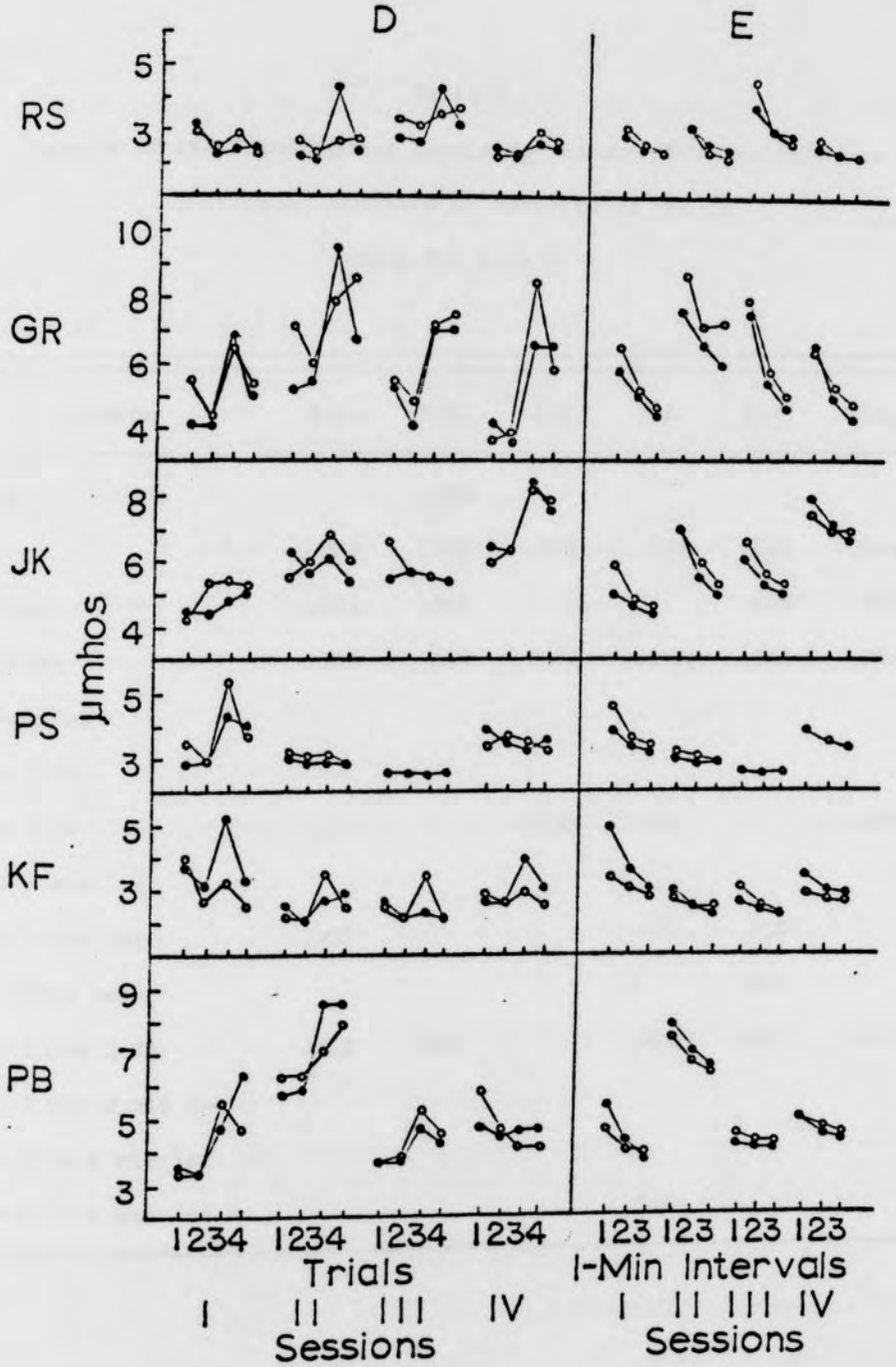
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Sessions
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1-Min Intervals
I II III IV
Sessions

Table 6

Summary of the Significance Levels (p-values) Obtained From the Individual Analyses of Variance of the SC Data for each S

Source	R.S.	G.R.	J.K.	P.S.	K.F.	P.B.
H-L		.008				
Ses	.002	.001	.001	.002	.001	.001
Trials	.004	.001			.001	.013
Min Int	.001	.001	.001	(.070)	.001	.004
H-L X Ses						
H-L X Tr						
Ses X Tr			.030	.003		.050
H-L X Min Int						
Ses X Min Int	.007				.050	
Tr X Min Int					.002	
H-L X Ses X Tr	.013	.001		.030	.001	.040
H-L X Ses X Min Int						
H-L X Tr X Min Int						
Ses X Tr X Min Int				.010		

The changes in SC with trials is shown in Figure 5B. The trials effect was significant for four subjects, R.S. ($p < .004$), G.R. ($p < .001$), K.F. ($p < .001$), and P.B. ($p < .013$). This effect appears to reflect a similar pattern of change for each of these subjects as depicted in the figure. In each case there is an S-shaped function indicating a decrease in SC between trials one and two and between trials three and four, with an increase on trial three following the mid-session break.

Figure 5C shows an apparent decrease in SC within each trial for each subject. The effect of 1-minute intervals as reflected in this figure, was significant for five of the six subjects, R.S., G.R., J.K., and K.F. at $p < .001$, and P.B. at $p < .004$; it approached significance for the sixth subject, P.S. ($p < .07$).

First-order interactions. Figure 5D illustrates the changes in SC across sessions and across trials within each session. The Sessions X Trials effect, reflected in this figure by visualizing the average of the functions for the high and low alpha conditions, was significant for three subjects, J.K. ($p < .03$), P.S. ($p < .003$), and P.B. ($p < .05$). The pattern of changes was different for each of these subjects. As reflected in the figure, skin conductance for J.K. appeared to remain relatively stable across trials for the first three sessions, showing a tendency to increase across trials in session four. In the case of P.S., SC showed a tendency to increase across trials in sessions one, and remained essentially stable across trials in the last three sessions. For P.B., the graphs reflect progressive increases in SC across

trials for the first three sessions and an apparent decrease across trials in session four.

Apparent changes across 1-minute intervals for each session can be seen in Figure 5E by visualizing the average functions for the high and low alpha conditions. A significant Sessions X 1-Minute Intervals interaction was found in two cases, R.S. ($p < .007$) and K.F. ($p < .05$). There was no systematic tendency for the degree of change within trials to either increase or decrease across sessions for either of these subjects. Rather, the decrement in SC across sessions was greatest during session three for R.S. and during session one for K.F. with no appreciable differences in the slopes of the curves during any of the remaining sessions.

A Trials X 1-Minute Intervals interaction was found to be significant for one subject, K.F. ($p < .002$). Though this interaction is not depicted graphically, it can be verbally characterized as reflecting differential decreases in SC across 1-minute intervals, the extent of decrease being dependent on the particular trial. The most pronounced decrease occurred on trial three, the initial 1-minute interval of this trial (which immediately followed the mid-session break) having the largest SC value.

Second-order interactions. Changes in SC as a function of alpha level, sessions, and trials within each session are presented in Figure 5D. The H-L X Sessions X Trials interaction was significant for five of the six subjects, R.S. ($p < .013$), G.R. ($p < .001$), P.S. ($p < .03$), K.F. ($p < .001$), and P.B. ($p < .04$). The pattern of

change reflected in the graphs for these subjects indicates that the degree and direction of difference in SC for the high and low alpha conditions were dependent on the particular session and the particular trial within each session, but the exact nature of these dependencies is specific to each subject.

A second-order interaction involving sessions, trials, and 1-minute intervals was found to be significant for one subject (P.S., $p < .01$). The appropriate graphs to reflect this interaction are not presented, but the data can be verbally described as indicating that the extent of any change in SC across 1-minute intervals for this subject was dependent on the particular session and the particular trial within each session.

Subjective Reports

The subjects were asked to report the strategies they used to control the tone (i.e., to increase or decrease alpha). According to their verbal reports, three subjects, R.S., P.S., and P.B., used similar strategies throughout most of each session. These subjects reported that for the low alpha condition they imagined a spot or image very close to the eye and concentrated on focusing their eyes on that spot or image. For the high alpha condition they stated they simply remained "relaxed" and tended to daydream. J.K. stated that he imagined listening to music during the high alpha condition (sometimes breathing out or saying "omm" to himself); during the low condition he "watched" the inside of his eyelids, sometimes visualizing images or working math problems. K.F. reported looking at "spots" on her

eyes, or concentrating on bodily sensations (e.g., a heavy, weighted-down feeling) during the low alpha condition. For the high condition she would, "get in a relaxed, lightheaded state and let my mind wander." During the low alpha condition, G.R. reported, he squinted his eyes, moved his eyes rapidly, gazed with concentration at an image produced on his eyelid by the light flashes, tensed his body, and thought about things in rapid succession. During the high condition he tried to avoid concentrating on any particular cognition, to relax his body, to roll his eyes back, and to open and close his Eustachian tubes.

Summary

Consistent differences in the physiological measures between the high and low alpha conditions were observed for particular subjects. As shown in Table 7, in 7 of the 18 opportunities, indicated by asterisks, these physiological measures were significantly greater during the low alpha condition. In three cases eye motor activity was greater during the low alpha condition, in three cases EMG was greater, and in one case SC was greater. In only one instance, indicated by an X and involving eye motor activity for K.F., was a statistically significant difference found in the opposite direction, i.e., greater during the high alpha condition. This is a likely chance occurrence at the 5% level, inasmuch as there were 18 opportunities for obtaining a statistical α of .05.

There were statistically significant temporal changes in one or more of the peripheral physiological variables for each subject. In

Table 7

Summary of the Significant H-L Effects Obtained From the Individual Analyses of Variance for Each S for Eye Motor Activity, EMG, and SC

	Physiological Measures		
Subjects	Eye Motor Activity	EMG	SC
R.S.	-	* (.002)	-
G.R.	-	-	* (.008)
J.K.	* (.07)	* (.004)	-
P.S.	* (.05)	* (.003)	-
K.F.	X (.02)	-	-
P.B.	* (.02)	-	-

*indicates the physiological measure was greater during the low alpha condition.

Xindicates the physiological measure was greater during the high alpha condition.

some instances such changes were within trials, some were across trials, and some were across sessions. A number of first and second-order interactions involving changes within and across trials and across sessions also were found for some of the variables. The temporal changes noted tended to be quite specific for each subject; also, such changes did not tend to covary in any consistent manner with concomitant temporal changes occurring in alpha level. Some of the more salient temporal changes, either in terms of their consistency or magnitude, were: (A) a progressive increase in eye motor activity under the low alpha condition across sessions for two subjects (G.R. and J.K.), with essentially no change occurring under the high alpha condition; (B) a differential change in EMG within trials for two subjects (R.S. and P.S.), increasing during the low alpha condition and remaining essentially constant during high alpha; (C) a drop in SC within trials for all subjects; and (D) a significant change across trials for four of the six subjects for SC and alpha. In addition to these changes, significant interactions involving alpha conditions, sessions, and trials were obtained in 18 of 24 opportunities for the various physiological variables.

Averaged Evoked Potentials

The EPs for each subject are presented in Figure 6. Each potential is the sum of individual responses to 100 light flashes per trial. Each tracing is the superimposition of 16 potentials for each subject from four trials of high alpha and four trials of low alpha, for each of the four sessions.

Figure 6. Averaged evoked potentials for each S for the high (H) and low (L) alpha conditions. Each potential is based on responses to 100 light flashes. Each tracing is the superimposition of 16 potentials (one for each of 4 trials for 4 sessions).

H
L
RS
GR
JK
PS
KF
PB
4 μv
msec x 100
0 1 2 3 4

It is clear upon inspection of Figure 6 that the EPs obtained under the high and low alpha conditions differed for each of the subjects. Generally speaking, there was less variability in the superimposed tracings under the low condition. While noticeable for all subjects, the more highly replicable response under the low condition is particularly apparent for subjects G.R. and P.B.

Although the EP patterns differ for each of the subjects under the two conditions, the nature of the difference is idiosyncratic. That is, there was no tendency for the major components of the EP patterns to be consistently greater under one condition than under the other for all the subjects. For any given subject, however, specific components of the EPs did differ consistently for the two conditions throughout all 16 replications.

The EPs of subject R.S. were highly variable across replications, there being somewhat less variability under the low than under the high conditions. There is an indication that the averaged potentials were loosely time locked to the probe stimuli under both conditions but with somewhat greater consistency under the low than under the high condition. Negative components with peak latencies of 80-100 msec. and 140-160 msec. and positive components with peak latencies of 110-120 and 170-190 msec. are readily discernible in the superimposed tracings obtained under the low condition. Although less readily perceptible, these same components can be visually abstracted from the tracings obtained under the high condition.

For G.R. the EPs obtained under the low condition, in addition to being more consistent than those obtained under the high, also are more highly articulated. There is a distinct, although small, positive component with a peak latency of about 90 msec. in the EP data obtained under the low condition which is followed by a positive (110 msec.) and second negative component (peak latency of 120 msec.). None of these components is discernible in the EPs obtained under the high condition. A late positive component (peak latency of 170-180 msec.) appears in both sets of EP data but is more sharply defined for the low alpha condition.

For J.K., the first negative component, occurring at 100-110 msec., was greater under the low than under the high condition; a positive component occurring at about 150 msec. was very pronounced under the low condition but barely discernible under the high; a negative component occurring at about 170 msec. is present under both conditions, as is a positive component with a peak latency of about 200 msec.; finally, a well-articulated third negative component with a peak latency of about 210-220 msec. is present under the low but not under the high condition. In other terms, the EP for this subject is more highly articulated under the low than under the high condition. There is a distinct "W" configuration composed of the three negative and two positive components just described in the EPs for the low condition whereas for the high condition there is a "V"-shaped pattern consisting of a single negative deflection and a slow-rising positive wave.

For subject P.S. highly similar EP patterns were obtained under the high and low conditions. The most salient difference between the two was that the major components of the EPs (a positive and negative wave with peak latencies of 180-190 and 210-220 msec. respectively) were consistently smaller under the low than under the high condition.

In contrast to the other five subjects, the EP patterns obtained for K.F. were more pronounced and more highly articulated under the high than under the low condition. In both sets of tracings a "W" configuration beginning and ending with two negative components with peak latencies of 90-100 and 220-240 msec. is readily apparent. The negative and positive components composing the inner legs of the "W" are considerably more pronounced under the high than under the low condition. The degree of between-replication variability was of approximately the same magnitude under both conditions. It should be recalled that with respect to eye motor activity, the effect of the high and low conditions for this subject was inconsistent with the statistically significant effect obtained for three other subjects (J.K., P.S., and P.B.). The eye motor effect is also inconsistent with the significant changes found in EMG and SC for four of the subjects.

The difference in EP patterns obtained under the high and low conditions for P.B. are dramatic. Under the high condition there is very little indication that the averaged potentials contain components which were time locked to the proble stimulus. Instead, the tracings appear to contain primarily sinusoidal oscillations which reflect

ongoing spontaneous EEG activity. While there is some hint from the superimposed tracings that this sinusoidal activity may be loosely time locked to the probe stimulus, the pronounced variability across replications strongly suggests that light flashes had a very weak effect, if any, on the cortical evoked response. In contrast to the highly variable and poorly time-locked responses obtained under the high alpha condition, averaged potentials containing components which were distinctly time locked to the probe stimulus were obtained under the low condition. There is a discernible positive component with a peak latency of approximately 110 msec. which is followed by three much larger components (negative, positive, negative) with peak latencies of 140-160, 200-220, and 240-260 msec., respectively. Even though there was considerable between-replication variability, such variability was much reduced under the low condition.

CHAPTER IV

DISCUSSION

Group Results

ALPHA LEVEL, EYE MOTOR ACTIVITY, EMG, AND SC

Alpha Level

The group results for alpha activity (depicted in Figure 1) provide further evidence that individuals can in fact demonstrate differential control of alpha activity with auditory feedback, either producing or suppressing alpha as instructed. Such differential control was maintained throughout all four experimental sessions in the present study despite a statistically significant general increase in alpha activity level on the third trial of each session. The reason alpha level was higher on the third trial of each session is not entirely clear. Since this particular trial immediately followed a 10-minute rest interval, it is tempting to hypothesize that the subjects were at their lowest level of arousal during trial three because they had just had an opportunity to engage in a sustained period of relaxation midway through the session. One might argue that the subjects should have been just as relaxed on the first trial since at least 24 hours had passed since they last participated in a session. However, there is evidence to suggest that subjects tend to be relatively highly aroused at the beginning of an experimental session (Eason, Harter, & Storm, 1964).

The interaction between alpha level condition and changes within trials (across 1-minute intervals), manifested in terms of an increase

across trials under the low alpha condition while remaining essentially constant under the high condition, may reflect differential changes in arousal. One may infer that under the high condition alpha level was about as high as it could become at the outset of the experiment, since the subjects had had extensive practice generating alpha. Under the low condition the subjects may have become progressively less activated during the course of the trial due to fatigue, boredom, lack of sustained attention to the auditory stimulus, etc.

In view of the fact that an arousal explanation can account for the temporal changes across trials as well as the differential changes within trials, such an explanation gains some plausibility on grounds of consistency. However, it should be kept in mind that numerous other factors could be contributing to these effects, and this particular ad hoc explanation is at best only tentative. Further evidence for or against the arousal hypothesis may be deduced from the changes in the group physiological data reported below in this section and from the data obtained for individual subjects reported in the subsequent section.

Eye Motor Activity, EMG, and SC

It will be recalled that the analyses of the group physiological data revealed no significant changes with respect to the high and low alpha conditions even though the individual analyses revealed significant changes in some of the measures for certain subjects. Failure to obtain significant differences for the group data certainly does not prove that no differences exist and therefore cannot be taken as

evidence against the arousal hypothesis. Such failure, however, serves to remind us that even if there should be a common effect of an independent variable (in this particular experiment, alpha-level instructions) on one or more of these physiological variables, unless such an effect is particularly pronounced, it may be masked by idiosyncratic changes occurring for each subject. Such masking effects require unusually large amounts of group data to demonstrate statistical significance. This point receives additional impact when considered in the context of individual analyses discussed in the next section.

With respect to temporal changes, the group analyses revealed that only SC changed significantly. The changes were in terms of a progressive decrease within trials, a change across trials, and an interaction between trials and 1-minute intervals. The within-trial decrease is typical of SC and has been reported many times in the literature (e.g., Eason et al., 1964). It is interesting to note that the significant change in SC across trials (depicted in Figure 1) is similar to that described in the preceding section for alpha, in that SC also is highest on trial three following the mid-session break. However, the increase in SC on trial three suggests an increase, rather than a decrease, in arousal, at least in the autonomic nervous system, which is just the opposite of that which would have been predicted by an arousal hypothesis of alpha control.

Interactions Involving Subjects. The significant interactions involving subjects (see Table 1) indicate that the effect of the alpha level instructions, as well as the temporal effects, on the various

physiological variables varied markedly from subject to subject. Although two or more subjects occasionally showed similar changes in certain dependent variables, the effects for most subjects may best be described as being idiosyncratic, and their significance for voluntary alpha control will therefore be described in the next section.

AVERAGED EVOKED POTENTIALS

Although the evoked potential data were not subjected to a group analysis, due to the marked differences in waveform from subject to subject, the reader is being reminded of these data at this time in order that he may not overlook their potential importance for the investigation of voluntary control of alpha. The highly discernible and consistent differences manifested in the EPs obtained from each subject under the high and low alpha conditions provide information which may prove to be most helpful in understanding the neural activational state of the subject while engaged in the voluntary generation or suppression of alpha. The implication of the EP results for a cortical arousal hypothesis of alpha control will be discussed as a separate unit in the section which follows.

Individual Results

SUBJECT DIFFERENCES IN ALPHA CONTROL AND MEAN ALPHA LEVEL

Tangentially related to the arousal-attentional hypothesis of alpha control is the question of whether the ability to voluntarily alter one's alpha level is related to the spontaneous amount of alpha

activity involuntarily generated per unit time under some specified condition. The reader will recall that while the subjects in the present experiment differed in the degree of alpha change associated with the high-low conditions, such differences appeared to be essentially independent of each subject's mean alpha level as manifested during the course of the experiment (Table 3). Failure to find a relationship between mean alpha level and ability to voluntarily enhance or suppress alpha is consistent with results obtained by Beatty (1971), who failed to find a significant correlation between the two variables. Although negative findings can never prove conclusively the complete absence of a relationship, combined results of these two studies at least suggest that if such a relationship does exist, as some people have stated (Hord & Barber, 1971; Nowlis & Kamiya, 1970), it is at best a very weak one. Quite clearly, knowledge of the mean amplitude of alpha activity of individuals obtained under standardized conditions would be of little value in predicting the degree to which each individual could, with practice, voluntarily alter his alpha level.

Inasmuch as the between-subject differences in mean alpha level observed for the six subjects in the present study could have been due to the composite variations of numerous chemical, physical, biological, and behavioral factors, including variations in degree of general arousal and attentional state, it would be inappropriate to attribute such differences solely to the latter factor. Even if the variations did primarily reflect subject differences in arousal and attention,

there is no a priori reason to assume that a relatively highly aroused subject will generate a greater (or lesser) differential in alpha level between the high and low conditions than a less aroused subject. It is concluded, therefore, that an examination of the between-subject differences exhibited by the six subjects in voluntarily controlling alpha in relation to their mean alpha levels simply sheds no light on the question of whether such control is mediated through self-induced changes in arousal and attention. Hopefully, further examination of such voluntary control in relation to concomitant changes in eye motor activity, EMG, SC, and cortical evoked potentials will prove to be more enlightening.

CHANGES IN EYE MOTOR ACTIVITY, EMG, AND SC RELATED TO ALPHA LEVEL

Alpha Level

It will be recalled that consistent differences in the non-cortical electrophysiological measures recorded in the present study between the high and low alpha conditions were observed for particular subjects (see Table 7). In 7 of 18 opportunities the three measures of peripheral nervous system activity (eye motor activity, EMG, and SC) were greater during the low alpha condition. In only one case (eye motor activity for K.F.) was difference between high and low conditions found in the direction of greater activity during the high alpha condition, a likely chance occurrence at the .05 level, in view of the fact that there were 18 opportunities to obtain significance at this level. These findings strongly support the hypothesis that

the voluntary control of alpha is mediated in part by self-induced changes in arousal and/or attention.

Temporal Relationships

Examination of the temporal changes in alpha level and the various peripheral physiological measures lends no further substantive support for the arousal/attentional hypothesis of alpha control. Thus, it appears that in order to demonstrate a relationship between alpha level and peripheral physiological activity level an experimental situation must be contrived wherein large differences in alpha level are generated and sustained for a substantial period of time, as was done in the present experiment. Apparently, any temporal covariation related specifically to changes in arousal and attentional states is masked by other factors concomitantly affecting the various physiological variables.

Although temporal changes in alpha level and the various physiological variables shed no further light on the hypothesis that alpha control is mediated in part through self-induced alterations of arousal and attention, it is interesting to speculate as to why some of the temporal variations occurred. For example, the progressive increase in EMG level across 1-minute intervals for R.S. and P.S. under the low alpha condition (Figure 4C and E) is reminescent of so-called "EMG gradients" described by Bartoshuk (1955a, b) and interpreted by Eason and Branks (1963) as reflecting increasing voluntary effort. If their interpretation is applied to the present data, it appears that progressively more effort was exerted during the course of a trial under the

low condition in order to maintain the degree of self-induced cortical activation and general bodily arousal required to maintain alpha suppression throughout the trial. Applying the same interpretation to the high alpha condition one would have to assume that no change in effort was required to maintain a high level of alpha since EMG level remained essentially constant throughout the trial.

A second example, involving differential changes in eye motor activity across trials and sessions for two subjects (see J.K. and G.R. in Figure 3A and E). perhaps reflects an intensification of visual strategies in attempting to suppress alpha under the low condition while employing essentially the same strategy throughout each session under the high condition. Both of these subjects showed marked increases in eye motor activity across sessions with significant increases within trials under the low alpha condition for G.R. (Figure 3C and E), whereas neither showed any substantial changes under the high condition. It is interesting to note that the marked increase in eye motor activity across 1-minute intervals and sessions was not accompanied by a corresponding change in alpha level under the low condition, so it would appear that if the subjects were intensifying strategies involving the eyes, this intensification had little payoff in terms of improving voluntary alpha suppression.

AVERAGED EVOKED POTENTIALS

The evoked potentials for each subject differed markedly for the high and low alpha conditions, as illustrated in Figure 6. These data provide the strongest support for an arousal/attentional hypothesis of

alpha control. In general there was less variability in the superimposed tracings generated from one replication to the next under the low alpha condition. For individual subjects the EPs were more clearly defined under the low condition, reflecting more stimulus-locked activity in the clearer differentiation of earlier components. The one exception, K.F., whose EP waveforms were more highly articulated under the high than under the low condition, was also the only subject with a difference in the peripheral physiological measures opposite to that expected by an arousal hypothesis, and also showed relatively poor control of alpha as compared to the other subjects.

The EP data in the present study as interpreted in arousal/attentional terms directly concur with results, reported by Eason, Harter, and White (1969), of a study in which arousal level and attention were externally manipulated. Only one EP study has been found in which, as in the present study, subjects voluntarily induced changes in cortical arousal. Spilker, Kamiya, Callaway, and Yeager (1969), recording flash-evoked potentials from subjects voluntarily producing high and low alpha levels, found that two early components were usually greater in amplitude during periods of high alpha. Only one subject in the present study (P.S.) showed a comparable difference. This finding is probably related to this subject's strategy for controlling alpha. The Spilker et al. findings cannot be further extended since they presented no EP tracings and apparently recorded only a single EP from each of seven subjects in the high and low conditions (as compared to the 16 replications in each condition for each subject in the present

study). Furthermore they did not specify whether their subjects were in an eyes-opened or eyes-closed condition, a fact which makes their limited results impossible to interpret.

Summary and Conclusion

The present study was designed to examine the hypothesis that voluntary alpha control is at least partly mediated through self-induced changes in cortical activation level and bodily arousal. Changes in several physiological indicants of such activity were systematically examined, for subjects who voluntarily produced and suppressed alpha activity. In general the results of this experiment support the notion that the voluntary alteration of alpha activity is partly mediated through such self-induced changes. Physiological arousal was greater during the low alpha condition as measured by eye motor activity, EMG, and SC in 7 of 18 opportunities. The complexity of the association between the physiological response systems and alpha activity must be recognized, however, in view of the lack of direct and consistent covariation between the temporal changes in these physiological measures and the temporal changes in alpha. Further research is needed to establish what factors contribute to temporal variations in alpha level and the peripheral physiological variables.

Attentional and arousal factors have been viewed in the present study as representing concomitant, if not functionally equivalent, neural and behavioral states. Inasmuch as visually evoked potentials reflect visual attention and degree of cortical activation, the

consistent differences in the EPs found between the high and low conditions provide substantial evidence that subjects were more highly activated during the low alpha condition. Although the results of this study indicate that voluntary control of alpha is partly mediated through self-induced changes in cortical activation and bodily arousal, more specific factors affecting such control nevertheless need to be examined.

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Appendix of Additional Tables

Table 8

Summary of Group Analysis of Variance for Alpha

Source	MS	Error Term	df	F
1. H-L	2575.18	16	1	17.28**
2. Ses	58.60	17	3	1.93
3. Tr	33.24	18	3	8.65**
4. Min Int	4.15	19	2	1.02
5. H-L X Ses	10.31	20	3	1.66
6. H-L X Tr	2.10	21	3	.76
7. Ses X Tr	1.88	22	9	1.22
8. H-L X Min Int	14.28	23	2	12.45**
9. Ses X Min Int	1.78	24	6	1.41
10. Tr X Min Int	1.36	25	6	1.38
11. H-L X Ses X Tr	2.21	26	9	1.33
12. H-L X Ses X Min Int	.43	27	6	.60
13. H-L X Tr X Min Int	.29	28	6	.80
14. Ses X Tr X Min	.55	29	18	.76
15. H-L X Ses X Tr X Min Int	.29	30	18	.43
16. S X H-L	149.01	30	5	225.81**
17. S X Ses	30.23	30	15	45.81**
18. S X Tr	3.84	30	15	5.83**
19. S X Min Int	4.08	30	10	6.19**
20. S X H-L X Ses	6.22	30	15	9.43**
21. S X H-L X Tr	2.76	30	15	4.17**
22. S X Ses X Tr	1.54	30	45	2.33**
23. S X H-L X Min Int	1.15	30	10	1.74
24. S X Ses X Min Int	1.26	30	30	1.91**
25. S X Tr X Min Int	.99	30	30	1.49
26. S X H-L X Ses X Tr	1.66	30	45	2.51**
27. S X H-L X Ses X Min Int	.71	30	30	1.07
28. S X H-L X Tr X Min Int	.36	30	30	.54
29. S X Ses X Tr X Min Int	.72	30	90	1.09
30. S X H-L X Ses X Tr X Min Int	.66		90	

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

Table 9

Summary of Group Analysis of Variance for Eye Motor Activity

Source	MS	Error Term	df	F
1. H-L	254.47	16	1	.50
2. Ses	71.53	17	3	1.03
3. Trials	6.60	18	3	.36
4. Min Int	1.47	19	2	.56
5. H-L X Ses	6.01	20	3	.11
6. H-L X Tr	4.41	21	3	.71
7. Ses X Tr	5.77	22	9	1.20
8. H-L X Min Int	7.84	23	2	1.62
9. Ses X Min Int	4.20	24	6	2.15
10. Tr X Min Int	1.13	25	6	1.54
11. H-L X Ses X Tr	5.49	26	9	1.14
12. H-L X Ses X Min Int	1.79	27	6	1.83
13. H-L X Tr X Min Int	1.62	28	6	1.35
14. Ses X Tr X Min Int	1.41	29	18	.97
15. H-L X Ses X Tr X Min Int	1.18	30	18	.75
16. S X H-L	515.00	30	5	325.41**
17. S X Ses	69.73	30	15	44.06**
18. S X Tr	18.23	30	15	11.52**
19. S X Min Int	2.65	30	10	1.68
20. S X H-L X Ses	53.00	30	15	33.49**
21. S X H-L X Tr	6.19	30	15	3.91**
22. S X Ses X Tr	4.80	30	45	3.03**
23. S X H-L X Min Int	4.84	30	10	3.06**
24. S X Ses X Min Int	1.96	30	30	1.24
25. S X Tr X Min Int	.73	30	30	.46
26. S X H-L X Ses X Tr	4.81	30	45	3.04**
27. S X H-L X Ses X Min Int	.98	30	30	.62
28. S X H-L X Tr X Min Int	1.21	30	30	.76
29. S X Ses X Tr X Min Int	1.45	30	90	.92
30. S X H-L X Ses X Tr X Min Int	1.58		90	

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

Table 10

Summary of Group Analysis of Variance for EMG

Source	MS	Error Term	df	F
1. H-L	88.87	16	1	3.36
2. Ses	14.44	17	3	.76
3. Trials	3.04	18	3	1.98
4. Min Int	3.19	19	2	1.28
5. H-L X Ses	.17	20	3	.05
6. H-L X Tr	1.88	21	3	1.62
7. Ses X Tr	1.42	22	9	.60
8. H-L X Min Int	4.61	23	2	2.24
9. Ses X Min Int	.09	24	6	1.18
10. Tr X Min Int	.07	25	6	1.00
11. H-L X Ses X Tr	1.15	26	9	1.21
12. H-L X Ses X Min Int	.05	27	6	.51
13. H-L X Tr X Min Int	.02	28	6	.18
14. Ses X Tr X Min Int	.14	29	18	1.19
15. H-L X Ses X Tr X Min Int	.07	30	18	.93
16. S X H-L	26.43	30	5	352.45**
17. S X Ses	18.88	30	15	251.72**
18. S X Tr	1.54	30	15	20.56**
19. S X Min Int	2.50	30	10	33.39**
20. S X H-L X Ses	3.20	30	15	42.68**
21. S X H-L X Tr	1.16	30	15	15.48**
22. S X Ses X Tr	2.38	30	45	31.77**
23. S X H-L X Min Int	2.06	30	10	27.42**
24. S X Ses X Min Int	.08	30	30	1.06
25. S X Tr X Min Int	.07	30	30	.99
26. S X H-L X Ses X Tr	.95	30	45	12.66**
27. S X H-L X Ses X Min Int	.10	30	30	1.36
28. S X H-L X Tr X Min Int	.12	30	30	1.60*
29. S X Ses X Tr X Min Int	.12	30	90	1.56*
30. S X H-L X Ses X Tr X Min Int	.08		90	

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

Table 11

Summary of Group Analysis of Variance for SC

Source	MS	Error Term	df	F
1. H-L	.84	16	1	.75
2. Ses	14.71	17	3	.93
3. Trials	37.492	18	3	7.22**
4. Min Int	51.42	19	2	16.57**
5. H-L X Ses	.34	20	3	.91
6. H-L X Tr	.53	21	3	1.12
7. Ses X Tr	.41	22	9	.24
8. H-L X Min Int	.00	23	2	.00
9. Ses X Min Int	.31	24	6	.64
10. Tr X Min Int	.92	25	6	4.09**
11. H-L X Ses X Tr	.64	26	9	1.09
12. H-L X Ses X Min Int	.18	27	6	1.10
13. H-L X Tr X Min Int	.12	28	6	1.34
14. Ses X Tr X Min Int	.12	29	18	.47
15. H-L X Ses X Tr X Min Int	.21	30	18	1.07
16. S X H-L	1.11	30	5	5.65**
17. S X Ses	15.76	30	15	79.63**
18. S X Tr	5.19	30	15	26.22**
19. S X Min Int	3.10	30	10	15.67**
20. S X H-L X Ses	.37	30	15	1.87*
21. S X H-L X Tr	.47	30	15	2.38**
22. S X Ses X Tr	1.68	30	45	8.48**
23. S X H-L X Min Int	.18	30	10	.93
24. S X Ses X Min Int	.49	30	30	2.49**
25. S X Tr X Min Int	.22	30	30	1.13
26. S X H-L X Ses X Tr	.92	30	45	4.63**
27. S X H-L X Ses X Min Int	.17	30	30	.84
28. S X H-L X Tr X Min Int	.09	30	30	.47
29. S X Ses X Tr X Min Int	.25	30	90	1.28
30. S X H-L X Ses X Tr X Min Int	.20		90	

*p < .05
**p < .01

Table 12

Summary Analysis of Variance of Integrated Alpha for R.S.

Source	MS	Error Term	df	F
1. H-L	1857.55	5,8	1	229.72**
2. Ses	10.87	5,7,9	3	2.56
3. Trials	21.03	6,7,10	3	10.31**
4. Min Int	2.29	8,9,10	2	.61
5. H-L X Ses	9.31	11,12,13,14,15	3	4.95**
6. H-L X Tr	.32	11,12,13,14,15	3	.17
7. Ses X Tr	2.45	11,12,13,14,15	9	1.30
8. H-L X Min Int	6.24	11,12,13,14,15	2	3.32*
9. Ses X Min Int	4.40	11,12,13,14,15	6	2.34*
10. Tr X Min Int	2.29	11,12,13,14,15	6	1.22
11. H-L X Ses X Tr	3.45	15	9	1.90
12. H-L X Ses X Min Int	2.01	15	6	1.11
13. H-L X Tr X Min Int	.85	15	6	.47
14. Ses X Tr X Min Int	1.41	15	18	.78
15. Full Model	1.81		18	

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

Table 13

Summary Analysis of Variance of Integrated Alpha for G.R.

Source	MS	Error Term	df	F
1. H-L	181.93	5,6,8	1	233.24**
2. Ses	9.83	5,7,9	3	35.11**
3. Trials	3.63	6,7	3	9.31**
4. Min Int	.68	8	2	.30
5. H-L X Ses	.24	11	3	.41
6. H-L X Tr	.34	11	3	.59
7. Ses X Tr	.40	11	9	.69
8. H-L X Min Int	2.25	15	2	23.06**
9. Ses X Min Int	.11	15	6	1.12
10. Tr X Min Int	.03	15	6	.36
11. H-L X Ses X Tr	.58	15	9	5.96**
12. H-L X Ses X Min Int	.04	15	6	.40
13. H-L X Tr X Min Int	.10	15	6	1.07
14. Ses X Tr X Min Int	.20	15	18	2.07
15. Full Model	.10		18	

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

Table 14

Summary Analysis of Variance of Integrated Alpha for J.K.

Source	MS	Error Term	df	F
1. H-L	284.25	5,6,8	1	38.32**
2. Ses	56.50	5,7	3	11.97**
3. Trials	18.49	6,7	3	3.06
4. Min Int	6.12	8	2	.96
5. H-L X Ses	5.11	11	3	1.82
6. H-L X Tr	10.40	11	3	3.70
7. Ses X Tr	4.59	11	9	1.63
8. H-L X Min Int	6.39	15	2	12.15**
9. Ses X Min Int	.79	15	6	1.50
10. Tr X Min Int	1.10	15	6	2.08
11. H-L X Ses X Tr	2.81	15	9	5.34**
12. H-L X Ses X Min Int	.21	15	6	.39
13. H-L X Tr X Min Int	.21	15	6	.40
14. Ses X Tr X Min Int	.79	15	18	1.50
15. Full Model	.53		18	

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

Table 15

Summary Analysis of Variance of Integrated Alpha for P.S.

Source	MS	Error Term	df	F
1. H-L	466.74	5,8	1	37.43**
2. Ses	12.83	5	3	.78
3. Trials	1.86	6,7	3	1.74
4. Min Int	7.49	8,9	2	4.51*
5. H-L X Ses	18.44	11	3	15.11**
6. H-L X Tr	1.86	11	3	1.52
7. Ses X Tr	.80	11	9	.65
8. H-L X Min Int	3.53	12,13,14,15	2	11.20**
9. Ses X Min Int	1.04	12,13,14,15	6	3.29**
10. Tr X Min Int	.33	12,13,14,15	6	1.05
11. H-L X Ses X Tr	1.22	15	9	3.29*
12. H-L X Ses X Min Int	.40	15	6	1.09
13. H-L X Tr X Min Int	.25	15	6	.67
14. Ses X Tr X Min Int	.25	15	18	.68
15. Full Model	.37		18	

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

Table 16

Summary Analysis of Variance of Integrated Alpha for K.F.

Source	MS	Error Term	df	F
1. H-L	56.05	5,6,8	1	156.39**
2. Ses	88.63	5,7,9	3	127.58**
3. Trials	7.40	6,7,10	3	6.05**
4. Min Int	.88	9,10	2	.62
5. H-L X Ses	.24	11	3	.21
6. H-L X Tr	.68	11	3	.61
7. Ses X Tr	.82	11	9	.73
8. H-L X Min Int	.05	12	2	.07
9. Ses X Min Int	.73	12	6	1.03
10. Tr X Min Int	2.09	14	6	3.73*
11. H-L X Ses X Tr	1.12	15	9	4.42**
12. H-L X Ses X Min Int	.71	15	6	2.78*
13. H-L X Tr X Min Int	.27	15	6	1.08
14. Ses X Tr X Min Int	.56	15	18	2.20
15. Full Model	.25		18	

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

Table 17

Summary Analysis of Variance of Integrated Alpha for P.B.

Source	MS	Error Term	df	F
1. H-L	473.57	5,6,8	1	112.41**
2. Ses	31.13	5	3	3.87
3. Trials	.09	6	3	.04
4. Min Int	7.17	8,9,10	2	8.73**
5. H-L X Ses	8.05	11	3	6.01*
6. H-L X Tr	2.22	11	3	1.66
7. Ses X Tr	.50	11	9	.37
8. H-L X Min Int	1.45	15	2	2.46
9. Ses X Min Int	1.00	15	6	1.69
10. Tr X Min Int	.44	15	6	.74
11. H-L X Ses X Tr	1.34	15	9	2.27
12. H-L X Ses X Min Int	.61	15	6	1.04
13. H-L X Tr X Min Int	.40	15	6	.68
14. Ses X Tr X Min Int	.97	15	18	1.65
15. Full Model	.59		18	

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

Table 18

Summary Analysis of Variance of Eye Motor Activity for R.S.

Source	MS	Error Term	df	F
1. H-L	8.14	5,6,8	1	1.03
2. Ses	41.03	5,7	3	4.76*
3. Trials	45.70	6,7	3	9.12**
4. Min Int	.86	8,9,10	2	.50
5. H-L X Ses	16.46	11	3	4.34*
6. H-L X Tr	2.02	11	3	.53
7. Ses X Tr	6.01	11	9	1.59
8. H-L X Min Int	3.93	12,13,15	2	3.03
9. Ses X Min Int	1.72	15	6	1.35
10. Tr X Min Int	.95	15	6	.75
11. H-L X Ses X Tr	3.79	15	9	2.98*
12. H-L X Ses X Min Int	.98	15	6	.77
13. H-L X Tr X Min Int	.82	15	6	.65
14. Ses X Tr X Min Int	1.51	15	18	1.19
15. Full Model	1.27		18	

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

Table 19

Summary Analysis of Variance of Eye Motor Activity for G.R.

Source	MS	Error Term	df	F
1. H-L	18.03	5,8	1	1.43
2. Ses	113.72	5,7,9	3	9.25**
3. Trials	12.73	6,7,10	3	2.88
4. Min Int	5.35	8,9	2	.74
5. H-L X Ses	8.83	11,12,13,14,15	3	4.71**
6. H-L X Tr	1.87	11,12,13,14,15	3	.99
7. Ses X Tr	6.95	11,12,13,14,15	9	3.71**
8. H-L X Min Int	18.19	11,12,13,14,15	2	9.70**
9. Ses X Min Int	3.59	11,12,13,14,15	6	1.91
10. Tr X Min Int	1.90	11,12,13,14,15	6	1.01
11. H-L X Ses X Tr	2.96	15	9	1.38
12. H-L X Ses X Min Int	1.15	15	6	.53
13. H-L X Tr X Min Int	.93	15	6	.43
14. Ses X Tr X Min Int	1.61	15	18	.75
15. Full Model	2.15		18	

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

Table 20

Summary Analysis of Variance of Eye Motor Activity for J.K.

Source	MS	Error Term	df	F
1. H-L	1686.32	5	1	8.47+
2. Ses	194.07	5	3	.97
3. Trials	3.41	6,7	3	.70
4. Min Int	1.85	8,9	2	.54
5. H-L X Ses	199.16	11,12,13,14,15	3	171.20**
6. H-L X Tr	4.46	11,12,13,14,15	3	2.50
7. Ses X Tr	4.99	11,12,13,14,15	9	2.90**
8. H-L X Min Int	3.61	11,12,13,14,15	2	2.10
9. Ses X Min Int	3.30	11,12,13,14,15	6	1.92
10. Tr X Min Int	.49	11,12,13,14,15	6	.28
11.H-L X Ses X Tr	2.45	15	9	1.28
12. H-L X Ses X Min Int	1.81	15	6	.94
13. H-L X Tr X Min Int	1.47	15	6	.77
14. Ses X Tr X Min Int	1.21	15	18	.63
15. Full Model	1.92		18	

+$p < .07$
*$p < .05$
**$p < .01$

Table 21

Summary Analysis of Variance of Eye Motor Activity for P.S.

Source	MS	Error Term	df	F
1. H-L	22.19	5,6,8	1	6.37*
2. Ses	7.54	5,7	3	2.30
3. Trials	3.64	6,7	3	1.67
4. Min Int	.06	8	2	.02
5. H-L X Ses	5.88	11	3	3.32
6. H-L X Tr	1.47	11	3	.83
7. Ses X Tr	2.41	11	9	1.36
8. H-L X Min Int	2.91	15	2	8.39**
9. Ses X Min Int	.55	15	6	1.59
10. Tr X Min Int	.28	15	6	.81
11. H-L X Ses X Tr	1.77	15	9	5.11**
12. H-L X Ses X Min Int	.73	15	6	2.09
13. H-L X Tr X Min Int	.50	15	6	1.45
14. Ses X Tr X Min Int	.70	15	18	2.03
15. Full Model	.35		18	

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

Table 22

Summary Analysis of Variance of Eye Motor Activity for K.F.

Source	MS	Error Term	df	F
1. H-L	855.88	5,6	1	9.86*
2. Ses	52.31	5,7,9	3	6.98**
3. Trials	31.11	6	3	1.32
4. Min Int	4.01	8,9,10	2	2.02
5. H-L X Ses	16.74	11	3	1.02
6. H-L X Tr	23.49	11	3	1.44
7. Ses X Tr	7.05	11	9	.43
8. H-L X Min Int	.69	15	2	.31
9. Ses X Min Int	3.54	12,14,15	6	1.65
10. Tr X Min Int	.86	15	6	.38
11. H-L X Ses X Tr	16.34	15	9	7.28**
12. H-L X Ses X Min Int	1.05	15	6	.47
13. H-L X Tr X Min Int	2.69	15	6	1.20
14. Ses X Tr X Min Int	2.39	15	18	1.07
15. Full Model	2.24		18	

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

Table 23

Summary Analysis of Variance of Eye Motor Activity for P.B.

Source	MS	Error Term	df	F
1. H-L	238.94	5,8	1	15.45*
2. Ses	11.48	5	3	.48
3. Trials	1.18	6,7	3	.52
4. Min Int	2.59	8,9	2	1.57
5. H-L X Ses	23.96	11	3	10.74**
6. H-L X Tr	2.04	11	3	.91
7. Ses X Tr	2.36	11	9	1.06
8. H-L X Min Int	2.73	15	2	2.34
9. Ses X Min Int	1.29	15	6	1.10
10. Tr X Min Int	.32	15	6	.27
11. H-L X Ses X Tr	2.23	15	9	1.91
12. H-L X Ses X Min Int	.97	15	6	.84
13. H-L X Tr X Min Int	1.23	15	6	1.06
14. Ses X Tr X Min Int	1.24	15	18	1.06
15. Full Model	1.17		18	

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

Table 24

Summary Analysis of Variance of EMG for R.S.

Source	MS	Error Term	df	F
1. H-L	161.46	5,6,8	1	24.21**
2. Ses	66.44	5	3	12.35*
3. Trials	2.63	6	3	.61
4. Min Int	12.23	8	2	1.00
5. H-L X Ses	5.38	11	3	1.72
6. H-L X Tr	4.29	11	3	1.37
7. Ses X Tr	.83	11	9	.26
8. H-L X Min Int	12.17	15	2	105.74**
9. Ses X Min Int	.06	14	6	.19
10. Tr X Min Int	.18	14	6	.56
11. H-L X Ses X Tr	3.13	15	9	27.18**
12. H-L X Ses X Min Int	.13	15	6	1.09
13. H-L X Tr X Min Int	.22	15	6	1.92
14. Ses X Tr X Min Int	.32	15	18	2.82*
15. Full Model	.12		18	

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

Table 25

Summary Analysis of Variance of EMG for G.R.

Source	MS	Error Term	df	F
1. H-L	.02	5,6	1	.01
2. Ses	25.70	5,7	3	8.62**
3. Trials	2.99	6,7	3	.71
4. Min Int	.06	8,9,10	2	.42
5. H-L X Ses	5.02	11	3	4.18*
6. H-L X Tr	1.50	11	3	1.25
7. Ses X Tr	2.30	11	9	1.92
8. H-L X Min Int	.03	12	2	.13
9. Ses X Min Int	.09	12	6	.39
10. Tr X Min Int	.05	15	6	1.32
11. H-L X Ses X Tr	1.20	15	9	35.01**
12. H-L X Ses X Min Int	.23	15	6	6.61**
13. H-L X Tr X Min Int	.06	15	6	1.85
14. Ses X Tr X Min Int	.05	15	18	1.49
15. Full Model	.03		18	

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

Table 26

Summary Analysis of Variance of EMG for J.K.

Source	MS	Error Term	df	F
1. H-L	9.69	5,6,8	1	15.36**
2. Ses	3.05	5,7	3	.44
3. Trials	3.58	7	3	.41
4. Min Int	1.27	8,9,10	2	9.17**
5. H-L X Ses	1.37	11	3	2.98
6. H-L X Tr	.25	11	3	.54
7. Ses X Tr	8.78	11	9	19.09**
8. H-L X Min Int	.08	15	2	.53
9. Ses X Min Int	.23	15	6	1.47
10. Tr X Min Int	.07	15	6	.42
11. H-L X Ses X Tr	.46	15	9	2.97*
12. H-L X Ses X Min Int	.05	15	6	.31
13. H-L X Tr X Min Int	.20	15	6	1.30
14. Ses X Tr X Min Int	.20	15	18	1.31
15. Full Model	.16		18	

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

Table 27

Summary Analysis of Variance of EMG for P.S.

Source	MS	Error Term	df	F
1. H-L	41.34	5,8	1	26.20**
2. Ses	2.07	5,7	3	3.83*
3. Trials	.27	6,7	3	.79
4. Min Int	2.08	8	2	.84
5. H-L X Ses	.98	11	3	9.80**
6. H-L X Tr	.17	11	3	1.70
7. Ses X Tr	.40	11	9	4.00*
8. H-L X Min Int	2.47	15	2	64.21**
9. Ses X Min Int	.03	15	6	.84
10. Tr X Min Int	.02	15	6	.46
11. H-L X Ses X Tr	.10	15	9	2.54*
12. H-L X Ses X Min Int	.04	15	6	1.14
13. H-L X Tr X Min Int	.02	15	6	.40
14. Ses X Tr X Min Int	.03	15	18	.65
15. Full Model	.04		18	

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

Table 28

Summary Analysis of Variance of EMG for K.F.

Source	MS	Error Term	df	F
1. H-L	6.25	5,6	1	5.82
2. Ses	1.43	5	3	.01
3. Trials	.04	6,7	3	.09
4. Min Int	.06	8,9,10	2	1.18
5. H-L X Ses	1.72	11	3	3.51
6. H-L X Tr	.42	11	3	.86
7. Ses X Tr	.41	11	9	.84
8. H-L X Min Int	.04	15	2	.88
9. Ses X Min Int	.02	15	6	.50
10. Tr X Min Int	.07	15	6	1.58
11. H-L X Ses X Tr	.49	15	9	10.96**
12. H-L X Ses X Min Int	.06	15	6	1.39
13. H-L X Tr X Min Int	.02	15	6	.42
14. Ses X Tr X Min Int	.05	15	18	1.22
15. Full Model	.04		18	

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

Table 29

Summary Analysis of Variance of EMG for P.B.

Source	MS	Error Term	df	F
1. H-L	2.27	5,6,8	1	2.75
2. Ses	10.16	5,7	3	11.33**
3. Trials	1.24	6,7	3	1.69
4. Min Int	.01	8,9,10	2	.14
5. H-L X Ses	1.70	11	3	3.33
6. H-L X Tr	1.05	11	3	2.06
7. Ses X Tr	.63	11	9	1.23
8. H-L X Min Int	.11	15	2	2.01
9. Ses X Min Int	.06	15	6	1.18
10. Tr X Min Int	.06	15	6	1.21
11. H-L X Ses X Tr	.51	15	9	9.72**
12. H-L X Ses X Min Int	.06	15	6	1.09
13. H-L X Tr X Min Int	.10	15	6	1.88
14. Ses X Tr X Min Int	.06	15	18	1.17
15. Full Model	.05		18	

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

Table 30

Summary Analysis of Variance of SC for R.S.

Source	MS	Error Term	df	F
1. H-L	.07	5,6,8	1	.20
2. Ses	3.38	5,7,9	3	7.56**
3. Trials	2.61	6,7,10	3	6.40**
4. Min Int	5.51	8,9,10	2	18.80**
5. H-L X Ses	.16	11	3	.27
6. H-L X Tr	.57	11	3	.97
7. Ses X Tr	.54	11	9	.91
8. H-L X Min Int	.29	15	2	1.66
9. Ses X Min Int	.46	12,14,15	6	3.46**
10. Tr X Min Int	.13	15	6	.76
11. H-L X Ses X Tr	.59	15	9	3.37*
12. H-L X Ses X Min Int	.06	15	6	.36
13. H-L X Tr X Min Int	.09	15	6	.51
14. Ses X Tr X Min Int	.11	15	18	.64
15. Full Model	.17		18	

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

Table 31

Summary Analysis of Variance of SC for G.R.

Source	MS	Error Term	df	F
1. H-L	3.79	5,6,8	1	12.23**
2. Ses	16.49	5,7,9	3	15.41**
3. Trials	44.09	6,7,10	3	40.45**
4. Min Int	38.62	9,10	2	61.30**
5. H-L X Ses	.24	11	3	.11
6. H-L X Tr	.57	11	3	.25
7. Ses X Tr	1.61	11	9	.71
8. H-L X Min Int	.03	15	2	.12
9. Ses X Min Int	.68	15	6	2.40
10. Tr X Min Int	.58	15	6	2.03
11. H-L X Ses X Tr	2.28	15	9	7.97
12. H-L X Ses X Min Int	.25	15	6	.87
13. H-L X Tr X Min Int	.04	15	6	.15
14. Ses X Tr X Min Int	.44	15	18	1.55
15. Full Model	.29		18	

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

Table 32

Summary Analysis of Variance of SC for J.K.

Source	MS	Error Term	df	F
1. H-L	.89	5,6	1	3.35
2. Ses	20.91	5,7,9	3	18.69**
3. Trials	2.68	6,7	3	1.75
4. Min Int	11.91	9	2	31.23**
5. H-L X Ses	.31	11	3	.63
6. H-L X Tr	.22	11	3	.45
7. Ses X Tr	1.88	11	9	3.84*
8. H-L X Min Int	.01	15	2	.02
9. Ses X Min Int	.38	15	6	1.76
10. Tr X Min Int	.07	15	6	.31
11. H-L X Ses X Tr	.49	15	9	2.30
12. H-L X Ses X Min Int	.25	15	6	1.15
13. H-L X Tr X Min Int	.01	15	6	.07
14. Ses X Tr X Min Int	.13	15	18	.62
15. Full Model	.21		18	

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

Table 33

Summary Analysis of Variance of SC for P.S.

Source	MS	Error Term	df	F
1. H-L	.27	5,6,8	1	1.47
2. Ses	6.09	5,7,9	3	7.47**
3. Trials	.89	6,7	3	.85
4. Min Int	1.56	8,9	2	3.77
5. H-L X Ses	.15	11	3	.88
6. H-L X Tr	.27	11	3	1.59
7. Ses X Tr	1.29	11	9	7.59**
8. H-L X Min Int	.09	15	2	1.52
9. Ses X Min Int	.43	14	6	2.26
10. Tr X Min Int	.08	14	6	.42
11. H-L X Ses X Tr	.17	15	9	2.83*
12. H-L X Ses X Min Int	.07	15	6	1.22
13. H-L X Tr X Min Int	.08	15	6	1.33
14. Ses X Tr X Min Int	.19	15	18	3.09**
15. Full Model	.06		18	

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

Table 34

Summary Analysis of Variance of SC for K.F.

Source	MS	Error Term	df	F
1. H-L	1.31	5,6,8	1	2.56
2. Ses	4.42	5,7,9	3	9.40**
3. Trials	3.80	6,7,10	3	10.80**
4. Min Int	4.34	8,9,10	2	13.52**
5. H-L X Ses	1.01	11	3	1.31
6. H-L X Tr	.15	11	3	.19
7. Ses X Tr	.41	11	9	.53
8. H-L X Min Int	.30	12,13	2	1.88
9. Ses X Min Int	.29	12,14	6	2.52*
10. Tr X Min Int	.36	13,14	6	5.14**
11. H-L X Ses X Tr	.77	15	9	6.62**
12. H-L X Ses X Min Int	.25	15	6	2.16
13. H-L X Tr X Min Int	.07	15	6	.61
14. Ses X Tr X Min Int	.07	15	18	.60
15. Full Model	.12		18	

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

Table 35

Summary Analysis of Variance of SC for P.B.

Source	MS	Error Term	df	F
1. H-L	.09	5,6,8	1	.15
2. Ses	42.23	7	3	13.80**
3. Trials	9.38	6,7,10	3	4.72*
4. Min Int	4.99	8,9,10	2	8.17**
5. H-L X Ses	.33	11	3	.36
6. H-L X Tr	1.09	11	3	1.18
7. Ses X Tr	3.06	11	9	3.33*
8. H-L X Min Int	.21	15	2	.60
9. Ses X Min Int	.54	15	6	1.53
10. Tr X Min Int	.82	13,14,15	6	2.15
11. H-L X Ses X Tr	.92	15	9	2.26*
12. H-L X Ses X Min Int	.13	15	6	.38
13. H-L X Tr X Min Int	.29	15	6	.82
14. Ses X Tr X Min Int	.44	15	18	1.26
15. Full Model	.35		18	

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