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A primary purpose of the present investigation was to demonstrate that certain physiological and behavioral variables tend to change concordantly with experimentally-induced changes in activation and to show further that these same variables may be simultaneously influenced by other factors in an independent and specific manner. Activation level was altered by varying the difficulty as well as the incentive of the task. This was accomplished by having S watch a flashing light while: (a) maintaining a relaxed, quiescent state; (b) releasing a key in response to a flash; and (c) releasing a key under threat of shock. Heart rate, muscle tension, skin conductance, evoked cortical responses, and reaction times were recorded simultaneously for each of six Ss under all three conditions.

Changes in each of the physiological variables as a function of variation in activation were found to be statistically significant. In addition, changes between Ss and within sessions were found to be significant for each of the dependent variables. Although experimentally-induced changes in activation clearly affected each of the physiological variables, there were also differential changes within each over time. These results support the conclusion that the physiological responses do change concomitantly with changes in activation, but that other factors may sometime mask such changes. Thus, when using physiological variables as "indicants" of activation, other factors need to be controlled.

THE RELATION BETWEEN CENTRAL AND PERIPHERAL  
MEASURES IN A PERCEPTUAL-MOTOR TASK

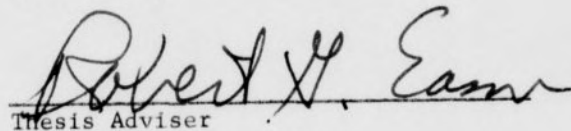
by

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TABLE OF CONTENTS

	PAGE
ACKNOWLEDGEMENTS . . . . .	iii
LIST OF TABLES . . . . .	v
LIST OF FIGURES . . . . .	vi
CHAPTER I. INTRODUCTION . . . . .	1
General Statement of Problem . . . . .	1
Evidence for the Existence of a General State of Activation . . . . .	2
Physiological "Indicants" of Activation . . . . .	3
Purpose of the Investigation . . . . .	9
Specific Statement of the Problem . . . . .	9
CHAPTER II. METHOD . . . . .	10
Subjects . . . . .	10
Experimental Design . . . . .	10
Procedure, Apparatus, and Data Recording . . . . .	10
CHAPTER III. RESULTS . . . . .	17
Findings Based on Variance Analysis . . . . .	17
Graphic Presentation of Results . . . . .	17
CHAPTER IV. DISCUSSION . . . . .	27
Experimentally-Induced Changes in Activation . . . . .	27
Within-Trial Changes . . . . .	29
CHAPTER V. SUMMARY . . . . .	32
BIBLIOGRAPHY . . . . .	34
APPENDIX . . . . .	39

LIST OF TABLES

TABLE	PAGE
1. VARIANCE ANALYSIS SUMMARY FOR EMG . . . . .	40
2. VARIANCE ANALYSIS SUMMARY FOR SKIN CONDUCTANCE . . . . .	41
3. VARIANCE ANALYSIS SUMMARY FOR HEART RATE . . . . .	42
4. VARIANCE ANALYSIS SUMMARY FOR ECP . . . . .	43
5. VARIANCE ANALYSIS SUMMARY FOR REACTION TIMES . . . . .	44

## LIST OF FIGURES

FIGURE	PAGE
1. Effects of activation on EMG during a 300-second response period . . . . .	18
2. Effects of activation on skin conductance during a 300-second response period . . . . .	20
3. Effects of activation on heart rate during a 300-second response period . . . . .	22
4. Effects of activation on ECP (superimposed tracings represent replications) . . . . .	24
5. Effects of activation on reaction times during a 300-second response period . . . . .	25



## CHAPTER I

## INTRODUCTION

General Statement of Problem.

The writings of Duffy (1951, 1957, 1962) and Freeman (1948) have long stressed the importance of an intensity dimension in behavior, variously referred to as activation. Physiological processes such as muscle tension, heart rate, skin conductance, the electroencephalogram (EEG), and the evoked cortical potential (ECP) have been employed experimentally in an attempt to measure this intensity variable, since they appear to reflect changes in the activation of the organism. These writers, relying mainly on the records of muscle tension (Duffy, 1932) and palmar conductance (Freeman, 1940), concluded from their experiments that there was a lawful relationship between a state of the organism called arousal, energy mobilization, or activation level and performance. In addition, they suggested the relationship might be described by an inverted U-shaped curve, implying an optimal level of arousal for the performance of a given task.

Considerable research effort has hence been directed toward ascertaining the exact relationship of activation to various physiological processes and performance. The ultimate aim is the establishment of relationships between physiological activity and performance sufficiently precise to permit accurate predictions about arousal, attention, other internal states, and performance. The physiological activity from which predictions are to be made are the general and specific patterns and levels of central and peripheral, autonomic and somatic, physiological activity.

Many studies have been reported which seem to support Duffy's hypothesis of a continuum in activation, reflected via the activity level of autonomic, somatic, and central nervous systems, as one progresses from situations of relatively little difficulty or little goal significance to greater difficulty or greater significance. However, Lacey and his co-workers (1958, 1959, 1963) have reported finding specific changes, particularly in heart rate, which imply that the various physiological events cannot be considered meaningful "indicants" of general activation. Hence, a primary objective in the present investigation was to obtain new information concerning the behavior of these systems in an effort to ascertain the conditions under which they do and do not co-vary.

#### Evidence for the Existence of a General State of Activation.

In many instances a close correspondence has been found among physiological measures with respect to the direction of change in response to effective stimulation. Malmo and Surwillo (1960) found that sleep-deprived Ss tracking under relatively high incentive conditions showed a progressive increase in skin conductance and respiration concomitant with a progressive fall in EEG alpha amplitude. Their findings were explained by saying that the sleep-deprived Ss in a demanding task situation had to exert more effort in order to perform well, than would ordinarily be the case in a rested state. Pinneo (1961) in a study of induced tension found that two EEG measures showed changes concordant with changes in heart rate, respiration rate, conductance, and muscle tension. Similarly, Malmo (1966) reported a significant decline in skin conductance, respiration, heart rate, and performance in a long session of tracking. MacNeilage (1966) found during the course of a testing session, concordant changes in EEG

alpha amplitude, heart rate, skin conductance, respiration, and muscle tension. Elliott (1964), in a study of kindergarten children and adults, recorded EEG, heart rate, skin conductance, and muscle tension. Comparing mean physiological levels during a low incentive reaction time performance to those obtained under the resting condition, he found significant mean changes in EEG, heart rate, skin conductance, and muscle tension between conditions. Davies and Krkovic (1965) recorded performance and physiological measures on an auditory vigilance task. The expectation was that over a long period of time, working on a boring task, activation level would decline. The results were in the expected direction. Level of performance and the physiological measures of activation showed concordant decline over the vigil. In addition, there was a close correspondence between the central measure (EEG) and the peripheral measure, skin conductance.

The results of these studies seem to indicate that activation is a quantifiable dimension, the physiological measures showing high intra-individual concordance.

#### Physiological "Indicants" of Activation.

Electromyogram (EMG). Level of muscle tension has been repeatedly shown to be a possible indicant of intensity of behavioral arousal. In numerous studies Malmö and his co-workers (1951, 1954, 1956, 1957) have demonstrated a gradient phenomenon from muscle tension, i. e., a progressive rise in tension from the beginning to the end of a task. Level of activation was calculated in terms of the steepness (slope) of the gradient. Malmö, Shagass, and Davis (1951) reported a gradient phenomenon from EMG during mirror tracing. Wallerstein (1954) found gradients in the frontalis

muscle while S was lying on a bed listening to verbal material. Surwillo (1956) demonstrated that increased incentive had the effect of raising the slope of the EMG gradient in a visual tracking experiment. Similarly, Stennett (1957a), while employing an auditory tracking task under four incentive conditions, reported that the most efficient tracking performance was associated with intermediate gradient steepness and intermediate levels of skin conductance. Both lower and higher levels of physiological functioning were found to produce inferior performance. All of the above findings were interpreted by Malmo and his co-workers (1951, 1954, 1956, 1957) to mean that the degree of motivation and, in general, activation level were reflected in the level of muscle tension.

Considerable research has been conducted by Eason and his co-workers (1959, 1961, 1963) concerning the relationship between muscle tension, performance, and general level of activation. Eason (1959) reported that EMG increased approximately linearly when a constant amount of force was applied to a dynamometer for a sustained period of time, the rate of increase being proportional to the absolute amount of force being applied. In addition he found that when S was instructed to apply a constant amount of pressure in the absence of visual feedback, naive Ss tended to hold muscle tension constant rather than physical force which declined exponentially. It was concluded that the surface EMG of muscles actively involved in a physical task constitutes a reliable index of the amount of effort voluntarily expended in order to perform a task.

In another study (Eason, 1963) it was found that increased task difficulty, through the reduction of target size being tracked, led to an improvement in tracking precision and a concomitant increase in EMG level

of skilled Ss up to a point. When target size was reduced beyond a certain value, however, performance deteriorated while EMG level increased further. This finding tended to support the hypothesis that an inverted U-shaped relation sometimes exists between activation level and performance. In later studies, however, Eason and Branks (1963) and Malmo (1966) reported that the U-shaped relation between activation level and performance is often due to the distracting effects of the stimuli responsible for higher activation. This was demonstrated by using a divided set technique, having S perform two tasks simultaneously. EMG level increased with amount of incentive, but performance on the two tasks improved or deteriorated in accordance with which of the two tasks the highest incentive was assigned. Thus it was concluded that in order to predict what happens to performance as activation level changes, one must also have knowledge of Ss attentional state.

Evoked Cortical Potential. Studies of activation, vigilance, or attention have indicated that the ECP increases in amplitude with an increase in these states. Eason and his co-workers (1964) reported that when Ss performed physical and mental tasks of varying degrees of difficulty (exerting sustained force on a hand grip device, memorizing digits, adding by 13's), there was a corresponding increase in the evoked potential patterns. In another study (Eason, Groves, & Bonelli, 1967) the evoked potentials recorded during an avoidant shock condition were found to be significantly larger than during three other conditions: no-response, response, and unavoidable shock. Reaction times were routinely shorter during the avoidant shock condition. These results were interpreted as indicating that the attentional and arousal effects of avoidant shock act together to substantially increase the amplitude of the evoked potential.

In an investigation by Haider, Spong, and Lindsley (1964) changes in evoked potentials during a vigilance task requiring visual discrimination and response were studied. Detection performance was used as a behavioral measure of attention and inattention. It was found that reduced attention, as measured objectively by signal detection, was paralleled by a corresponding reduction in amplitude of the evoked potential, suggesting that performance decrement could be attributed to changes in the observer's state of attention and, in general, his level of activation.

Additional reports by Garcia-Austt (1963), Spong and his co-workers (1965), and Donchin and Lindsley (1966) have emphasized the relationship of the evoked potential amplitude to the alertness of the S. Lansing, Schwartz, and Lindsley (1959) found a relationship between EEG activation and reaction time, with larger peak-to-peak amplitudes and shorter reaction times under the alert, as opposed to the non-alert, condition. The results of these experiments leave little doubt that the evoked potential is affected by states of alertness, attention, and, in general, activation level.

Skin Conductance. Generally speaking, skin conductance appears very sensitive to shifts in the kind of activity being performed or to sudden changes in environmental stimulation. For corroboration see previously cited studies (pp. 3 - 5).

Stennett (1957b), in a sleep deprivation experiment, studied the relationship between conductance and EEG. Three Ss were deprived of sleep for sixty hours and tracked at regular intervals throughout the vigil. Sleep deprivation, it was felt, should have the effect of raising level of physiological activity, reflected by higher conductance. The results were as predicted, i. e., conductance rose throughout the vigil concomitant with

a progressive fall in alpha amplitude.

The reference experiment in palmar conductance is the Freeman study (1940). Over a period of many days, one hundred paired measures of skin conductance and reaction times were taken from one S. The samples were taken on such a large number of occasions and at so many different hours that S was certain to be drowsy and alert, as well as many stages between the two extremes. Low conductance was found to be associated with long reaction times, and high conductance with short reaction times. The function commenced as a progressively rising one, though past a certain optimal range of conductance values, the curve took a downward turn, giving the appearance of an inverted U. The results of these studies seem to suggest that, in general, conductance is a sensitive indicator of changes in S's internal state as well as the stimulus situation.

Heart Rate. Considerable research has yielded inconsistencies in the direction of heart rate changes, with conflicting interpretations as to the role of these changes. For example, two opposing effects on heart rate have been found when anxiety is experimentally induced by signals of impending shock; sometimes there is an acceleration, at other times a deceleration. Notterman, Schoenfeld, and Bersh (1952) with trace conditioning found cardiac deceleration during a 6-second interval between the warning signal and shock. However, Lacey and Smith (1954) found an increase in heart rate when a verbal stimulus which had previously been associated with shock served as a warning signal. Both effects in different Ss have been observed by Zeaman and Wegner (1954).

Specific heart rate changes (bidirectional) have been reported by Lacey and his co-workers (1958, 1959, 1962, 1963) to be associated with

complex situations and to stimuli which appear to be a function of stimulus type. Noxious (cold pressor) or cognitive stimuli (mental arithmetic) resulted in a significant increase in heart rate, presumably associated with reduction in sensitivity to stimulation. It was proposed by the Laceys that the acceleration should facilitate rejection of the environment and should occur in situations where external distractions would interfere with problem solving. Stimuli defined as having high input characteristics (visual attention and empathetic listening) should produce a decrease in cardiac rate, presumably associated with increased sensitivity to stimulation, and occur when a situation requires attention. These findings were replicated by Obrist (1963). In addition, it is proposed that these cardiac changes function to modify organism-environment interaction via afferent feedback control of central nervous system activity, such that a deceleration acts to facilitate environmental intake while an acceleration acts to inhibit environmental intake (Lacey, 1959).

Under some circumstances, heart rate shows a decrease that is paradoxical because it is accompanied by an increase in other autonomic variables such as skin conductance. In a study by Lacey and his co-workers (1963) heart rate reactions were determined by comparing the average of the twelve fastest cardiac cycles during one minute of a rest condition with the corresponding average during one minute of an experimental condition. In this investigation, heart rate decelerations and increased conductance were found in situations where the major demand on the S was to receive environmental inputs. Such findings as these have led the Laceys to argue that these variables should never be used as "indicants" of activation, since other factors are known to affect them independently.



#### Purpose of the Investigation.

There were several kinds of information the writer intended to obtain from this study. A primary concern was to demonstrate that so-called "indicants" of activation tend to change concordantly when changes in activation are experimentally induced, and to show further that these same physiological variables may be simultaneously influenced by other factors in an independent and specific manner. A particular interest was to determine how the heart rate variable would behave with respect to the other "indicants" of activation in view of the work of Lacey and associates. Also of interest was the manner in which the evoked potential relates to the other measures, since it had not been previously recorded simultaneously with the other "indicants".

#### Specific Statement of the Problem.

Activation level or the amount of effort exerted in the performance of a given task was varied by altering the difficulty as well as the incentive of the task that S was required to perform. This was accomplished by having S watch a flashing light while: (a) maintaining a relaxed, quiescent state (low arousal); (b) releasing a key in response to a flash, thereby giving a reaction time (moderate arousal); (c) reacting under conditions in which shock could be avoided if reaction times were short enough (high arousal).

Four physiological variables (ECP, muscle tension, heart rate, and skin conductance) and one behavioral variable (reaction time) were recorded simultaneously for each of six Ss under all of the above conditions. Thus, via the behavior of these variables, information was obtained concerning the functioning of the autonomic and central nervous systems.

## CHAPTER II

## METHOD

Subjects.

Six male University of North Carolina at Greensboro psychology graduate students between the ages of 21 and 28 were used as subjects.

Experimental Design.

Each subject participated in six experimental sessions, two sessions per day for three days. A session lasted approximately 30 minutes and consisted of three trials, one each for the low, moderate, and high conditions. A trial lasted for six and one-half minutes and was followed by a five-minute rest. During the course of the experiment, each trial was replicated six times with each S serving as his own control. The order in which the Ss were subjected to the experimental conditions was counter-balanced within and across sessions by means of a latin square.

Procedure, Apparatus, and Data Recording.

S was placed in an electrically-shielded, semi-darkened room during the recording session. A Grass Model 7 Polygraph equipped with the necessary preamplifiers was used to record the various physiological events. The EEG was recorded with a Grass 7P5 from which the ECP was obtained by summing the amplified signals with a Mnemotron 400 B Computer of Average Transients (CAT); Skin conductance was recorded with a Grass 7P1; the Electrocardiogram (EKG) with a Grass 7P4; and the EMG with a Grass 7P5 for the unintegrated record, a 7P3 for the integrated record.

Reaction times were measured with a Hewlett Packard Electronic Counter and were manually recorded.

EEG and ECP Recording System. Evoked cortical potentials were recorded monopolarly with commercial silver disc electrodes (8 mm. in diameter). To reduce skin resistance below 10,000 ohms, both the surface of the scalp and the earlobe were rubbed with electrode jelly. The recording (scalp) electrode was placed one inch above the inion and one inch to the right of the midline being held in place by a headband made of electricians plastic tape. The reference electrode, a commercial silver clip, was placed on the right earlobe. The electrode leads were connected to the EEG input terminals located just behind Ss head.

The cortical electrical responses which are evoked by the presentation of stimuli of abrupt onset such as light flashes are usually of low amplitude and therefore masked by the ongoing activity of the brain. The CAT, a special purpose digital computer, makes these responses observable by adding together the EEG activity for a specific time segment following a light flash. That electrical activity not related to the stimulus will average out whereas the activity time locked to the stimulus will grow progressively larger (see Eason, et. al., 1964). Thus, the evoked cortical potential was obtained by summing the electrical responses of the brain to 100 light flashes with a Mnemotron 400 B CAT, and a permanent record of the information was obtained with a Moseley X-Y Plotter (Model 2D-2). The CAT analysis time during all recording and calibration was .5 seconds.

The ECP data were quantified by calculating the average amplitudes for each S on each trial. This consisted of measuring the vertical peak-to-trough distance covered by the major deflections, summing, and taking an

average. A variance analysis was then performed on the amplitudes.

Skin Conductance Recording System. Prior to each recording session, dime-sized silver electrodes were chlorided by placing them in a saline solution through which an electric current was passed. They were then taped to the volar surface of the first and third fingers and the electrode leads were connected to a Grass 7P1 preamplifier which contained the necessary circuitry for balancing skin resistance. The polygraph channel was calibrated in such a manner that  $S_s$  resistance could be read directly in ohms from the oscillogram. Thirty-second intervals were then marked off on the skin resistance analog record. Measures of conductance were obtained by drawing a visually "best fit" line through each of the 30-second intervals. The distance from the baseline, which had a known ohm value, to the midpoint of the "best fit" line was measured in millimeters. The skin resistance values thus obtained were converted to conductance units (micro-mhos) by finding their reciprocals and multiplying by one million.

EKG Recording System. Heart rate was recorded by connecting the electrodes attached to the right ear and the left forearm to one of the input channels of the polygraph. A 7P4 Tacheograph preamplifier (specially designed heart rate counter) was calibrated by feeding in pulses separated in time by a known amount. The data were quantified by marking off 30-second intervals on the analog record and simply counting the number of beats per interval. These values were then doubled in order to present the data in terms of the standard beats per minute value.

EMG Recording System. Muscle action potentials were recorded from the forearm flexor muscles of the inactive arm (left). Commercial silver disc electrodes (8 mm. in diameter) mounted two inches apart in plastic

adapters were used to pick up muscle potentials. The skin was scrubbed and the electrodes were taped along the longitudinal axis of the muscles. The resistance at each electrode was always below 10,000 ohms. The electrode leads were connected to the EMG input terminals located just behind S's head.

The muscle action potentials were integrated over 30-second intervals with a pulse-frequency type electronic integrator and were recorded with a Presin Printing Counter (Model C3-30). The system was calibrated by feeding a 30 cps sine wave signal of varying voltage into the input of the electronic integrator and recording the number of pulses per 30-second interval generated by the signal. Calibration curves were then constructed and used to convert the experimental data into microvolts. The oscillograms of the unintegrated EMG were used to check for possible artifacts in the integrated EMG data.

Reaction Time Recording System. Reaction times were measured in milliseconds with a Hewlett Packard Electronic Counter and were manually recorded. Upon giving a verbal ready signal, S pressed down on the end of a fulcrum-mounted, non-conductive lever inserted through a small circular hole in the wall of the shielded room. The other end of the lever contacted a microswitch which was mounted outside the shielded room in order to prevent transients from appearing in the evoked potential record when the switch contacts opened and closed. When a light flash was presented, the CAT was automatically triggered and S raised his finger as quickly as possible from the lever. The response time was displayed on the electronic counter and was manually recorded.

Visual Stimulus Apparatus. One hundred light flashes were presented

during the course of a trial. These were generated with a Grass Ps-2 Photo-Stimulator mounted outside of the electrically-shielded room. The light was transmitted by a tubular light guide which passed into S's room. The tip of the light guide was mounted flush against the inner surface of an opaque screen shaped like a hemisphere. The S was seated in a chair in front of the screen with his chin on a head rest. The distance from S's eyes to the center of the screen was 40 cm. His arms were placed in a comfortable resting position on a flat surface with his right index finger on the reaction key. Foveal stimulation was accomplished by having S fixate on the center of the screen where the light flashes appeared. The photo-flash element was viewed by S through a Number 26 (red) Kodak-Wratten filter. The flash intensity of the photo-simulator was set at 4, well above S's threshold, and held constant throughout the experiment.

Photo-Electric Programmer. A programmed tape loop was used to regulate not only the electronic counter which printed out every 30 seconds, but also the flash rate of the photo-stimulator. This was done by placing strips of magnetic tape the same width (half-second duration) at certain intervals along the loop. On the average a light flash was presented every 4.0 seconds during a trial with the time between flashes varying randomly from 2.0 to 7.0 seconds.

Shock Apparatus. Two commercial silver disc electrodes (8 mm. in diameter) mounted two inches apart were attached to the inner side of S's left ankle. The leads were connected to the output of a Grass Stimulus Isolation Unit (Model SIU-4678) which was activated by a Grass S-8 Stimulator. The voltage level of the S-8 Stimulator was adjusted for each S in the initial recording session by having him indicate when it was unpleasant

enough that he would work hard to avoid it. On the average the voltage applied to the Ss was 350 volts; its duration, approximately 2 milliseconds (msec.).

At the beginning of the experiment, each S was asked to make 50 reaction times to the light flashes. From these 50 the three slowest responses were selected, and the fastest of these was used as a criterion for the presentation of shock. Thus, shock was administered to S whenever his reaction times were slower than the previously determined criterion. Generally not more than two or three shocks, if any, were applied during a trial for any S. The experimenter (E) administered the shock by manually closing a switch located outside the shielded room.

Experimental Precautions and Controls. Upon his first trip to the laboratory, S was given a 15-minute indoctrination to acquaint him with the equipment and to "adapt" him to the experimental situation. Prior to each trial S was told to assume a comfortable position and to avoid making excessive movements during the course of a trial. During each session a ground electrode was attached to the left earlobe to reduce the possibility of 60-cycle interference.

Between-trials there was a 5-minute rest and between-sessions a 15-minute rest; on both occasions S was allowed to come out of the shielded room. During the between-sessions break, the resistance of all electrodes was checked. After each rest S watched the flashing light for at least a minute in order to readapt him to the stimulus situation before the recording process was begun. Throughout the experiment the amplified output of a "white noise" generator was fed through a speaker into the electrically-shielded room in an attempt to minimize the probability of S being dis-

tracted by extraneous noises, whether they were generated inside or outside the laboratory.



## CHAPTER III

## RESULTS

Variance analyses were performed on each of the dependent variables (muscle tension, skin conductance, heart rate, ECP, and reaction time) to ascertain whether each changed significantly (a) with variation in activation level, (b) within sessions, i. e., across 30-second intervals, and (c) between sessions. Tests for first and second-order interactions among these variables and among Ss were also made. However, these were of secondary interest.

Findings Based on Variance Analyses.

Changes in each of the physiological variables as a function of variation in activation level were found to be statistically significant ( $p < .01$  in every case). The apparent changes in reaction time, however, were not significant and must therefore be attributed to chance. Changes between Ss and within sessions were significant at the .01 level for each of the dependent variables. The statistical analyses performed on each of the dependent variables are summarized in Tables 1 through 5 (see Appendix).

Graphic Presentation of Results.

EMG Level. The mean values obtained for each 30-second segment under each of the three activation conditions are shown in Figure 1. The significant increase in muscle tension level ( $p < .01$ ) is clearly reflected for all three levels of activation. Looking more closely at the curves generated under these conditions, it is clear that: (a) tension level, from

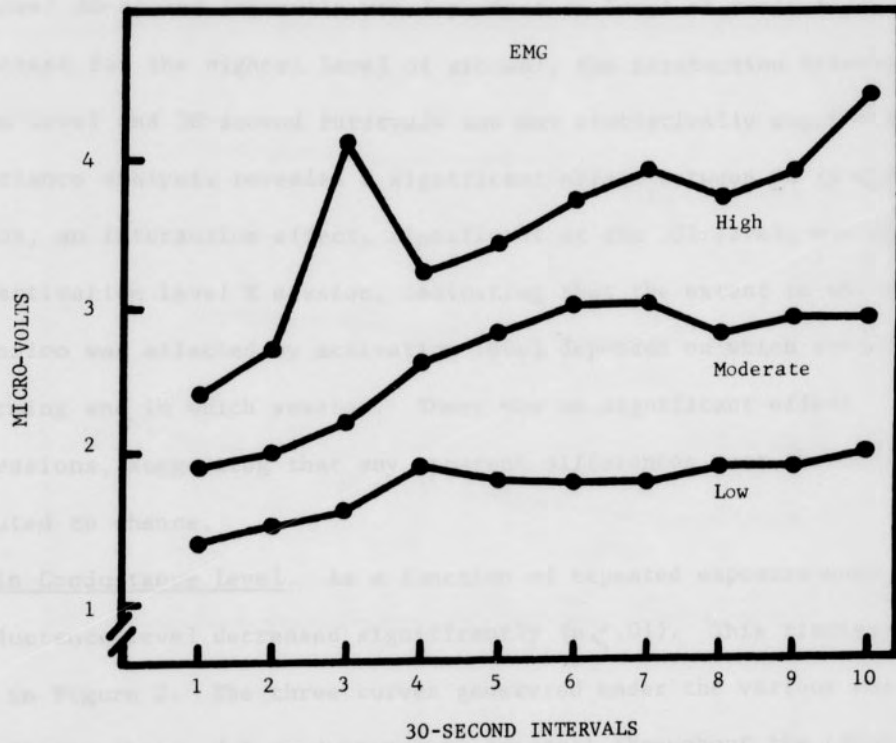


Figure 1. Effects of activation on EMG during a 300-second response period.

the beginning of the trial to the end, was highest under shock threat and continued to increase throughout the trial; (b) under moderate arousal, muscle tension was initially and consistently higher than in the relaxed condition and continued to increase; (c) a change in muscle tension also occurred under the relaxed condition. While it appears that the rate of increase over 30-second intervals was dependent on level of activation, being greatest for the highest level of arousal, the interaction between activation level and 30-second intervals was not statistically significant.

Variance analysis revealed a significant effect between  $S_s$  ( $p < .01$ ). In addition, an interaction effect, significant at the .01 level, was found for  $S_s \times$  activation level  $\times$  session, indicating that the extent to which muscle tension was affected by activation level depended on which subject was performing and in which session. There was no significant effect between sessions, suggesting that any apparent differences must therefore be attributed to chance.

Skin Conductance Level. As a function of repeated exposure over time, conductance level decreased significantly ( $p < .01$ ). This finding is reflected in Figure 2. The three curves generated under the various conditions indicate that: (a) conductance is greatest throughout the trial where  $S$  is performing under threat of shock, there being a gradual decline over time; (b) conductance is consistently greater under moderate arousal than low arousal and shows a gradual drop with repetitive stimulation; (c) the greatest decline appears to occur under the least activating condition wherein conductance level was initially the lowest. However, since there was no significant interaction between activation level and 30-second interval, the apparent difference in slope of the three curves must be attributed to

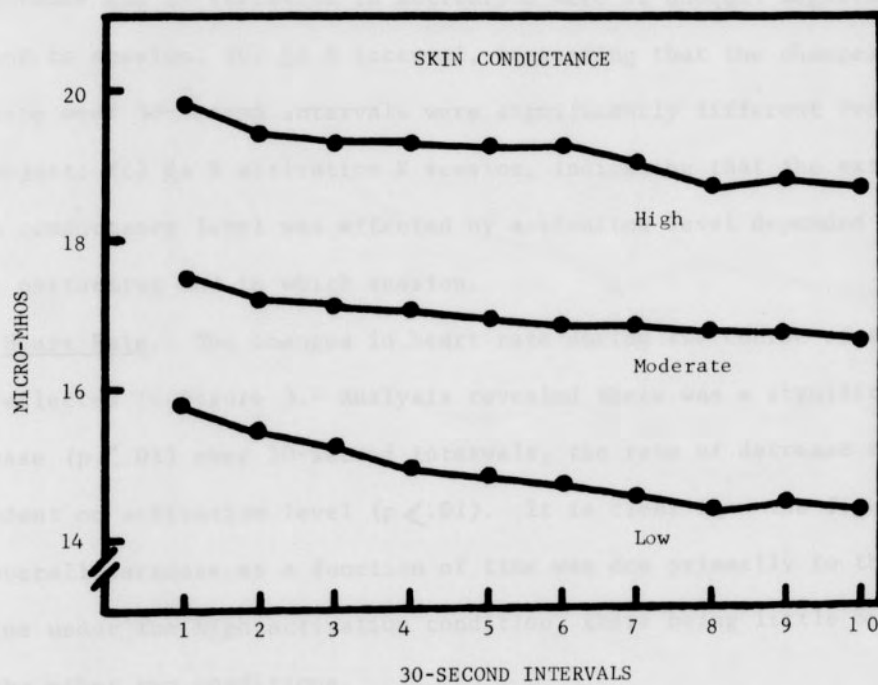


Figure 2. Effects of activation on skin conductance during a 300-second response period.

chance.

There were statistically significant changes in conductance levels between  $\underline{S}s$  ( $p < .01$ ). However, no effect was found between sessions. Variance analysis revealed several significant interactions ( $p < .01$  in every case): (a) Activation Level X session, indicating that the changes in conductance due to variation in activation were of unequal magnitude with respect to session; (b)  $\underline{S}s$  X interval, suggesting that the changes in conductance over 30-second intervals were significantly different from subject to subject; (c)  $\underline{S}s$  X activation X session, indicating that the extent to which conductance level was affected by activation level depended on which  $\underline{S}$  was performing and in which session.

Heart Rate. The changes in heart rate during the course of each trial are reflected in Figure 3. Analysis revealed there was a significant decrease ( $p < .01$ ) over 30-second intervals, the rate of decrease being dependent on activation level ( $p < .01$ ). It is clear from the figure that the overall decrease as a function of time was due primarily to the marked decline under the high activation condition, there being little or no change for the other two conditions.

Variance analysis revealed significant changes in heart rate between  $\underline{S}s$  ( $p < .01$ ) but not between sessions. In addition to the significant activation level X 30-second interval interaction, the following were also found to be significant at the .01 level: (a)  $\underline{S}s$  X activation level, implying that the changes in heart rate due to variation in activation were different from  $\underline{S}$  to  $\underline{S}$ ; (b)  $\underline{S}s$  X session, suggesting that the changes in heart rate due to within-session changes were significantly different from  $\underline{S}$  to  $\underline{S}$ ; (c)  $\underline{S}s$  X activation level X session, indicating that the extent to which heart

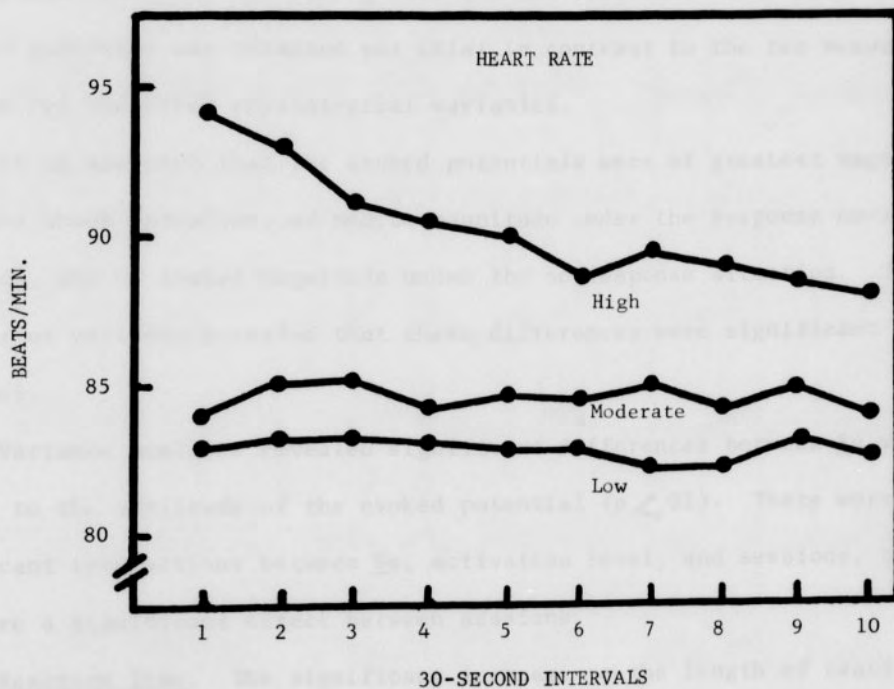


Figure 3. Effects of activation on heart rate during a 300-second response period.

rate was affected by activation level depended on which subject was performing and in which session.

Evoked Cortical Potential. Each of the six superimposed tracings shown in Figure 4 were based on the summation of 100 evoked responses to flashes presented during the course of a trial. These tracings represent the responses obtained for each S over the six sessions. Thus only one averaged potential was obtained per trial in contrast to the ten measures obtained for the other physiological variables.

It is apparent that the evoked potentials were of greatest magnitude under the shock situation, of medium magnitude under the response no-shock situation, and of lowest magnitude under the no-response situation. The analysis of variance revealed that these differences were significant at the .01 level.

Variance analysis revealed significant differences between Ss with respect to the amplitude of the evoked potential ( $p < .01$ ). There were no significant interactions between Ss, activation level, and sessions, nor was there a significant effect between sessions.

Reaction Time. The significant increase in the length of reaction times ( $p < .01$ ) as a function of time is illustrated in Figure 5. For the no-shock situation, reaction times increased from an average value of 265.9 msec. obtained during the first 30-second interval to 283.0 msec. for the last 30-second interval, an increase of 17.1 msec. For the shock condition, the initial reaction time was 250.6 msec., increasing to an average value of 267.7 msec. by the end of the trial, an increase of 17.1 msec.

Variance analysis indicated there were significant differences

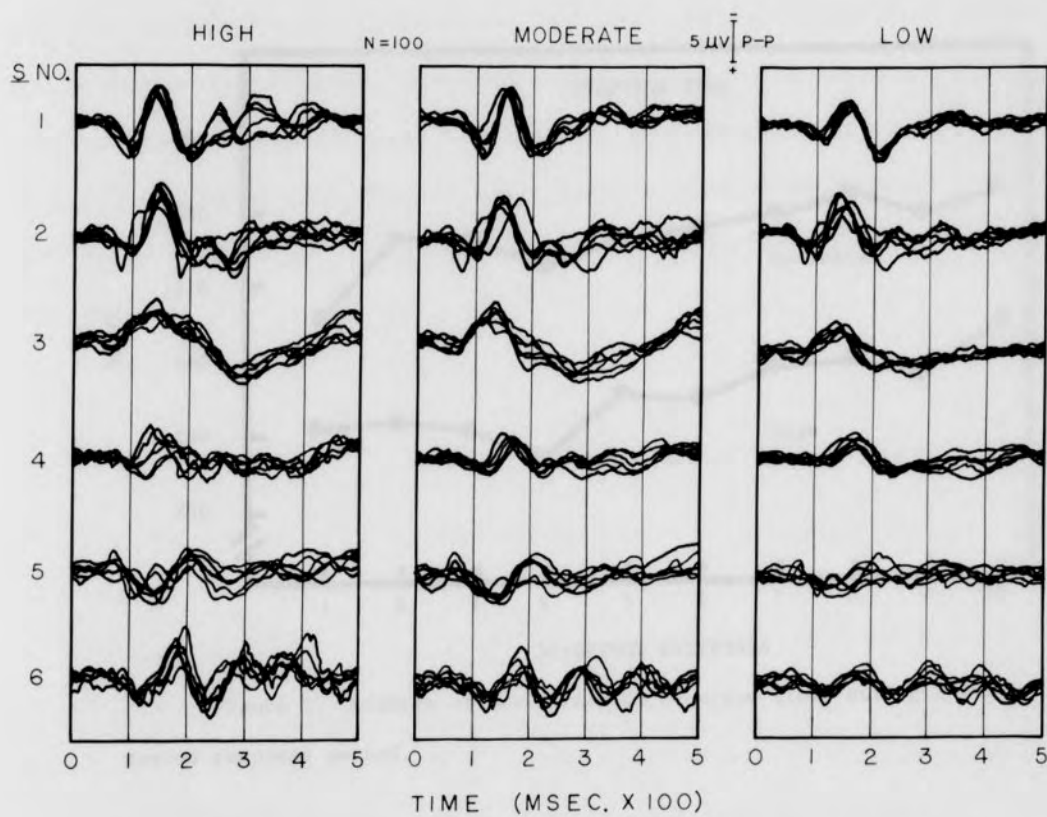


Figure 4. Effects of activation on ECP (superimposed tracings represent replications).



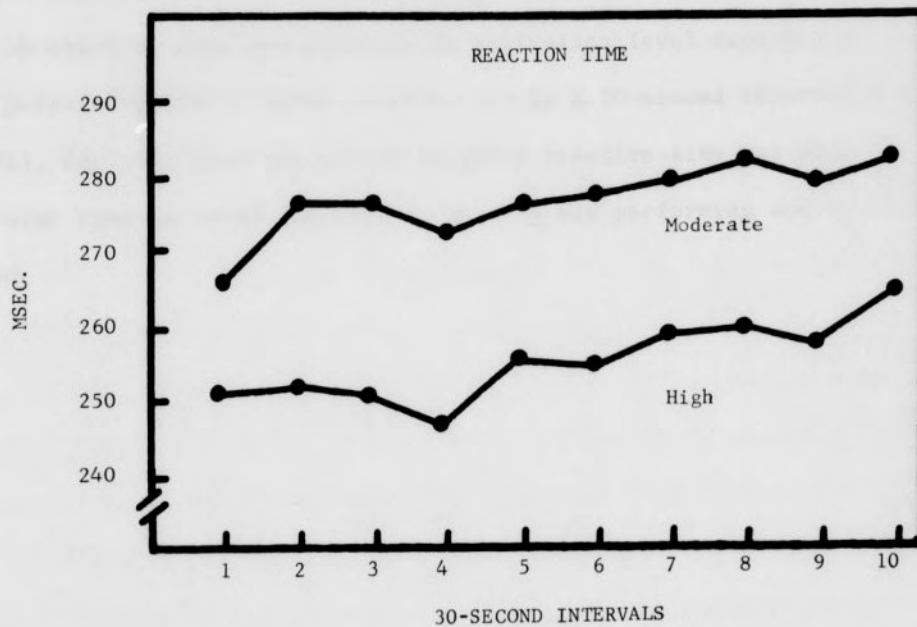


Figure 5. Effects of activation on reaction times during a 300-second response period.

among Ss in reaction times ( $p < .01$ ). However, there was no significant effect between sessions. The following interactions were found to be significant: (a) Ss X activation level X 30-second interval ( $p < .05$ ), implying that the affect of activation on reaction time depends on which S is reacting and on the particular time interval in which he is responding; (b) Ss X activation level X session ( $p < .01$ ), indicating that the extent to which reaction time was affected by activation level depended on which S was performing and in which session; (c) Ss X 30-second interval X session ( $p < .01$ ), implying that the extent to which reaction time was affected by a particular time interval depended on which S was performing and in which session.

## CHAPTER IV

## DISCUSSION

Experimentally-Induced Changes in Activation.

The finding in the present study of the effect of variation in activation level upon various physiological processes is consistent with the findings of others (Malmo & Surwillo, 1960; Pinneo, 1961; Davies & Krkovic, 1965; Malmo, 1966). In all of these studies, concomitant changes were found to occur in various physiological processes (i. e., muscle tension, heart rate, and skin conductance), and in the same direction, with changes in the demands of the stimulus situation.

The results tend to support Duffy's (1951, 1957, 1962) hypothesis that there appears to be a continuum of activation which is reflected in the activity level of several systems of the organism. It appears that as one goes from situations of relatively little difficulty or goal significance to greater difficulty or goal significance, there is a general enhancement of the activity level of neural and effector processes. In the more demanding task situation of the present study (the shock avoidance condition) S had to be more attentive, alert, and, in general, exert more effort in order to perform well. The changes in the energy requirements of the situation were reflected via increased autonomic, somatic, and central nervous system functioning. This interpretation is consistent with that offered by others (Duffy, 1957; Eason, 1959; Malmo, 1966).

The finding that heart rate behaved in a manner concordant with changes in muscle tension, skin conductance, and the ECP is at variance with

Lacey's (1959) proposal that heart rate is not a meaningful "indicant" of activation level. In situations requiring attention, Lacey would predict cardiac deceleration, the latter being associated with increased sensitivity to stimulation. The shock avoidance condition in the present investigation would seem to be just such a situation in that S could avoid unpleasant stimulation only if he was attentive to the flashing light and responded rapidly to it. However, the cardiac response was one of acceleration, in accordance with the increased activity level of the other physiological measures. This result suggests that increased cardiovascular activity has an excitatory and facilitative effect on cortical activity rather than an inhibitory effect as Lacey (1959) postulates. In conclusion, the findings indicate that heart rate is affected by activation level and does respond in a like manner with changes in skin conductance, muscle tension, and the ECP.

Numerous studies have demonstrated the relationship of the ECP, as a gross measure of cortical activity, to activation level and attention. In general, the amplitude of the ECP has been found to increase with an increase in these states (Garcia-Austt, 1963; Eason, Aiken, & White, 1964; Haider, Spong, & Lindsley, 1964). The present finding is consistent with those reported above. The amplitude of the evoked potential recorded during the shock avoidance condition was significantly larger than that obtained during the no-shock and no-response situations. Essentially this same result was obtained by Eason, Groves, & Bonelli (1967); in addition, they report that reaction times were significantly shorter during the shock condition. There was a tendency for reaction times to decrease in the present investigation, though not significantly so.

Since ECP's had not been previously recorded simultaneously with other

"indicants" of activation, its relationship to other measures was of particular interest. The results indicate that this central process behaves in the same manner as the peripheral events, skin conductance, muscle tension, and heart rate. As activation level increased, there was an increase in the amplitude of the evoked potential that occurred concomitantly with the increase in the periphery. Such findings reflect the interrelatedness of central and peripheral processes in activation.

#### Within-Trial Changes.

In the present investigation, a significant decrement in performance, heart rate, and skin conductance occurred concurrently with a progressive increase in muscle tension of the forearm flexors. Similar changes within sessions have been observed in these three physiological variables during the performance of verbal and perceptual-motor tasks (Eason, Harter, & Storm, 1964; Eason, Beardshall, & Jaffee, 1965). In these studies the differential changes over time were attributed to the relative degree of control that the autonomic and somatic nervous systems exerted on the three variables.

The fact that the two autonomic nervous system variables, heart rate and skin conductance, decreased during the course of a trial while reaction times increased seems to indicate progressive relaxation and lowered activation level. However, there were additional changes over time which were specific to each variable (Figures 1 - 5). From the time functions of skin conductance only, one might conclude that activation level progressively decreased during the course of the trial in like manner for all three experimental conditions, since the separation of the curves at any given time remained constant. However, a slightly different conclusion would have

to be drawn from the heart rate changes since there was a greater decline over time under the high activating condition than under the other two conditions. These differential changes over time in heart rate and skin conductance indicate that these variables are not simply "indicants" of general activation, but may be simultaneously affected by other variables in an independent and specific manner.

The progressive increase in muscle tension over time flatly contradicts the notion that peripheral measures reflect only changes in activation. The overall increase in muscle tension level, resulting from an increase in the activity level of the somatic nervous system primarily, has been explained in a similar study as a possible attempt to compensate for the detrimental effects of fatigue, boredom, feelings of discomfort, and increased difficulty in concentrating on the stimulus (Eason, Beardshall, & Jaffee, 1965). It has been previously demonstrated that muscle tension increases when one exerts more effort, whether to compensate for the detrimental effects of fatigue or a desire to perform better (Eason, 1959; Eason, & White, 1961; Eason & Branks, 1963).

The question as to what do we attribute the differential changes in the three variables overtime still remains to be satisfactorily answered. While experimentally-induced changes in activation clearly had an affect on each of the physiological variables, it does not account for the specific changes occurring within each over time. Hence, these variables can be regarded as "indicants" of activation only when all other factors are held constant, as Duffy indicates. However, to argue as Lacey does, that such variables should never be used as "indicants" of general activation because other factors are known to affect them independently, seems

unsubstantiated. When proper precautions and control of the experimental situations are maintained, changes in general activation level are reflected in all of the physiological measures utilized, as shown in the present study.

## CHAPTER V

## SUMMARY

A primary purpose of the present investigation was to demonstrate that certain physiological and behavioral variables tend to change concordantly with experimentally-induced changes in activation and to show further that these same variables may be simultaneously influenced by other factors in a differential manner. Secondary purposes of the study were to gain further information concerning the relationship of the heart rate variable and the ECP to other "indicants" of activation.

Changes in activation level were experimentally induced by having each of the six SS watch a flashing light while: (a) maintaining a relaxed, quiescent state (low arousal); (b) releasing a key in response to a flash (moderate arousal); and (c) releasing a key under threat of shock (high arousal). Heart rate, muscle tension, skin conductance, ECP, and reaction times were recorded simultaneously for each S under all three conditions.

The results indicated that the experimentally-induced changes in activation clearly affected each of the physiological variables ( $p < .01$ ). As the task situation became progressively more demanding, there was a general enhancement of the activity level of the autonomic and central nervous systems. Nevertheless, there were also differential changes within each over time. Heart rate, skin conductance, and performance declined significantly ( $p < .01$ ) concurrent with a progressive increase in muscle tension ( $p < .01$ ).



These findings support the conclusion that the physiological responses do change concomitantly with changes in activation, but that other factors may sometimes mask or modify these changes. Hence when using physiological variables as "indicants" of activation, proper precautions and control of the experimental situations must be maintained.

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TABLE 1  
 SUMMARY OF ANALYSIS RESULTS FOR THE

Level of Activation	df	MS	F	p
Between Subjects	119	1.10		
1. Activation Level	1	140.25	126	0.0001
2. 20-Second Interval	2	19.70	18	0.0001
3. Sessions	1	22.75	21	0.0001
4. Activation Level X 20-Second Interval	2	1.30	1	0.35
5. Activation Level X Sessions	2	47.50	43	0.0001
6. 20-Second Interval X Sessions	2	1.10	1	0.35
7. Activation Level X 20-Second Interval X Sessions	2	1.10	1	0.35
8. Within-Subject Error	119	1.10		
9. Error 1	119	1.10		
10. Error 2	119	1.10		
11. Error 3	119	1.10		
12. Error 4	119	1.10		
13. Error 5	119	1.10		
14. Error 6	119	1.10		
15. Error 7	119	1.10		
16. Error 8	119	1.10		
17. Error 9	119	1.10		
18. Error 10	119	1.10		
19. Error 11	119	1.10		
20. Error 12	119	1.10		
21. Error 13	119	1.10		
22. Error 14	119	1.10		
23. Error 15	119	1.10		
24. Error 16	119	1.10		
25. Error 17	119	1.10		
26. Error 18	119	1.10		
27. Error 19	119	1.10		
28. Error 20	119	1.10		
29. Error 21	119	1.10		
30. Error 22	119	1.10		
31. Error 23	119	1.10		
32. Error 24	119	1.10		
33. Error 25	119	1.10		
34. Error 26	119	1.10		
35. Error 27	119	1.10		
36. Error 28	119	1.10		
37. Error 29	119	1.10		
38. Error 30	119	1.10		
39. Error 31	119	1.10		
40. Error 32	119	1.10		
41. Error 33	119	1.10		
42. Error 34	119	1.10		
43. Error 35	119	1.10		
44. Error 36	119	1.10		
45. Error 37	119	1.10		
46. Error 38	119	1.10		
47. Error 39	119	1.10		
48. Error 40	119	1.10		
49. Error 41	119	1.10		
50. Error 42	119	1.10		
51. Error 43	119	1.10		
52. Error 44	119	1.10		
53. Error 45	119	1.10		
54. Error 46	119	1.10		
55. Error 47	119	1.10		
56. Error 48	119	1.10		
57. Error 49	119	1.10		
58. Error 50	119	1.10		
59. Error 51	119	1.10		
60. Error 52	119	1.10		
61. Error 53	119	1.10		
62. Error 54	119	1.10		
63. Error 55	119	1.10		
64. Error 56	119	1.10		
65. Error 57	119	1.10		
66. Error 58	119	1.10		
67. Error 59	119	1.10		
68. Error 60	119	1.10		
69. Error 61	119	1.10		
70. Error 62	119	1.10		
71. Error 63	119	1.10		
72. Error 64	119	1.10		
73. Error 65	119	1.10		
74. Error 66	119	1.10		
75. Error 67	119	1.10		
76. Error 68	119	1.10		
77. Error 69	119	1.10		
78. Error 70	119	1.10		
79. Error 71	119	1.10		
80. Error 72	119	1.10		
81. Error 73	119	1.10		
82. Error 74	119	1.10		
83. Error 75	119	1.10		
84. Error 76	119	1.10		
85. Error 77	119	1.10		
86. Error 78	119	1.10		
87. Error 79	119	1.10		
88. Error 80	119	1.10		
89. Error 81	119	1.10		
90. Error 82	119	1.10		
91. Error 83	119	1.10		
92. Error 84	119	1.10		
93. Error 85	119	1.10		
94. Error 86	119	1.10		
95. Error 87	119	1.10		
96. Error 88	119	1.10		
97. Error 89	119	1.10		
98. Error 90	119	1.10		
99. Error 91	119	1.10		
100. Error 92	119	1.10		
101. Error 93	119	1.10		
102. Error 94	119	1.10		
103. Error 95	119	1.10		
104. Error 96	119	1.10		
105. Error 97	119	1.10		
106. Error 98	119	1.10		
107. Error 99	119	1.10		
108. Error 100	119	1.10		

APPENDIX

\* Significant at .05 level

TABLE 1  
VARIANCE ANALYSIS SUMMARY FOR EMG

Source of Variation	df	MS	Error Term	F
I. Between Columns	179	8.30		
A. Activation Level	2	240.25	3A	52.67**
B. 30-Second Intervals	9	15.56	3B	4.92**
C. Sessions	5	22.78	3C	-----
D. Activation Level X 30-Second Intervals	18	1.94	3D	-----
E. Activation Level X Sessions	10	43.98	3E	1.43
F. 30-Second Intervals X Sessions	45	1.12	3F	0.59
G. Activation Level X Sessions	90	2.52	3G	1.13
II. Between Rows ( <u>Ss</u> )	5	176.06	3	32.72**
III. Rows X Columns	895	5.38		-----
A. <u>Ss</u> X Activation Level	10	45.61	3E	1.48
B. <u>Ss</u> X 30-Second Intervals	45	3.16	3G	1.41
C. <u>Ss</u> X Sessions	25	41.17	3E	1.34
D. <u>Ss</u> X Activation Level X 30-Second Intervals	90	2.30	3G	1.03
E. <u>Ss</u> X Activation Level X Sessions	50	30.81	3G	13.75**
F. <u>Ss</u> X 30-Second Intervals X Sessions	225	1.90	3G	-----
G. <u>Ss</u> X Activation Level X 30 Second Intervals X Sessions	450	2.24		-----
Total	1079			

\*\* Significant at .01 Level



TABLE 2  
 VARIANCE ANALYSIS SUMMARY FOR SKIN CONDUCTANCE

Source of Variation	df	MS	Error Term	F
I. Between Columns	179	35.71		
A. Activation Level	2	1,671.50	3A	41.37**
B. 30-Second Intervals	9	14.76	3B	4.94**
C. Sessions	5	337.38	3C	-----
D. Activation Level X 30-Second Intervals	18	0.71	3D	0.14
E. Activation Level X Sessions	10	116.98	3E	5.31**
F. 30-Second Intervals X Sessions	45	0.36	3F	-----
G. Activation Level X 30-Second Intervals X Sessions	90	0.35	3G	-----
II. Between Rows ( <u>Ss</u> )	5	6,139.74	3	243.16**
III. Rows X Columns	895	25.25		-----
A. <u>Ss</u> X Activation Level	10	40.30	3E	1.83
B. <u>Ss</u> X 30-Second Intervals	45	2.99	3G	1.83**
C. <u>Ss</u> X Sessions	25	802.39	3E	36.44**
D. <u>Ss</u> X Activation Level X 30-Second Intervals	90	0.52	3G	-----
E. <u>Ss</u> X Activation Level X Session	50	22.02	3G	13.51**
F. <u>Ss</u> X 30-Second Intervals X Sessions	225	0.55	3G	-----
G. <u>Ss</u> X Activation X 30-Second Intervals X Sessions	450	1.63		
Total	1079			

\*\* Significant at .01 level.

TABLE 3  
 VARIANCE ANALYSIS SUMMARY FOR HEART RATE

Source of Variation	df	MS	Error Term	F
I. Between Columns	179	111.34		
A. Activation Level	2	5,336.50	3A	8.94**
B. 30-Second Intervals	9	101.11	3B	5.52**
C. Sessions	5	785.40	3C	-----
D. Activation Level X 30-Second Intervals	18	35.06	3D	2.70**
E. Activation Level X Sessions	10	239.10	3E	1.27
F. 30-Second Intervals X Sessions	45	11.29	3F	1.01
G. Activation Level X 30-Second Intervals X Sessions	90	9.89	3G	0.99
II. Between Rows ( <u>Ss</u> )	5	13,168.40	3	234.90**
III. Rows X Columns	895	56.06		
A. <u>Ss</u> X Activation Level	10	597.10	3E	3.18**
B. <u>Ss</u> X 30-Second Intervals	45	18.33	3G	1.84
C. <u>Ss</u> X Sessions	25	1,033.12	3E	5.50**
D. <u>Ss</u> X Activation Level X 30-Second Intervals	90	12.96	3G	1.30
E. <u>Ss</u> X Activation Level X Sessions	50	187.70	3G	18.86**
F. <u>Ss</u> X 30-Second Intervals X Sessions	225	11.23	3G	1.13
G. <u>Ss</u> X Activation Level X 30-Second Intervals X Sessions	450	9.95		
Total	1079			

\*\* Significant at .01 level.

TABLE 4  
VARIANCE ANALYSIS SUMMARY FOR ECP

Source of Variation	df	MS	Error Term	F
I. Between Columns	17	0.66		
A. Activation Level	2	4.60	3A, B, C	27.06**
B. Sessions	5	0.16	3A, B, C	-----
C. Activation Level X Sessions	10	0.13	3A, B, C	-----
II. Between Rows ( <u>Ss</u> )	5	6.34	3	37.29**
III. Rows X Columns	85	0.17		
A. <u>Ss</u> X Activation Level	10	0.30		-----
B. <u>Ss</u> X Sessions	25	0.24		-----
C. <u>Ss</u> X Activation Level X Sessions	50	1.08		-----
Total	107			

\*\* Significant at .01 level.

TABLE 5  
VARIANCE ANALYSIS SUMMARY FOR REACTION TIMES

Source of Variation	df	MS	Error Term	F
I. Between Columns	119	1,771.88		
A. Activation Level	1	83,719.60	3A	1.63
B. 30-Second Intervals	9	1,944.30	3B	2.99**
C. Sessions	5	13,998.60	3C	2.19
D. Activation Level X 30-Second Intervals	9	294.60	3D	-----
E. Activation Level X Sessions	5	1,948.48	3E	-----
F. 30-Second Intervals X Sessions	45	286.14	3F	-----
G. Activation Level X 30-Second Intervals X Sessions	45	319.38	3G	1.21
II. Between Rows ( <u>S</u> s)	5	517,316.80	3	463.55**
III. Rows X Columns	595	1,116.01		
A. <u>S</u> s X Activation Level	5	51,398.70	3D, E, G	11.48**
B. <u>S</u> s X 30-Second Intervals	45	651.06	3D, F, G	1.98
C. <u>S</u> s X Sessions	25	6,403.38	3E, F, G	15.25
D. <u>S</u> s X Activation Level X 30-Second Intervals	45	403.73	3G	1.53*
E. <u>S</u> s X Activation Level X Sessions	25	21,811.36	3G	82.60**
F. <u>S</u> s X 30-Second Intervals X Sessions	225	380.35	3G	1.44**
G. <u>S</u> s X Activation Level X 30-Second Intervals X Sessions	225	264.03		
Total	719	264.03		

\* Significant at .05 level.

\*\* Significant at .01 level.