

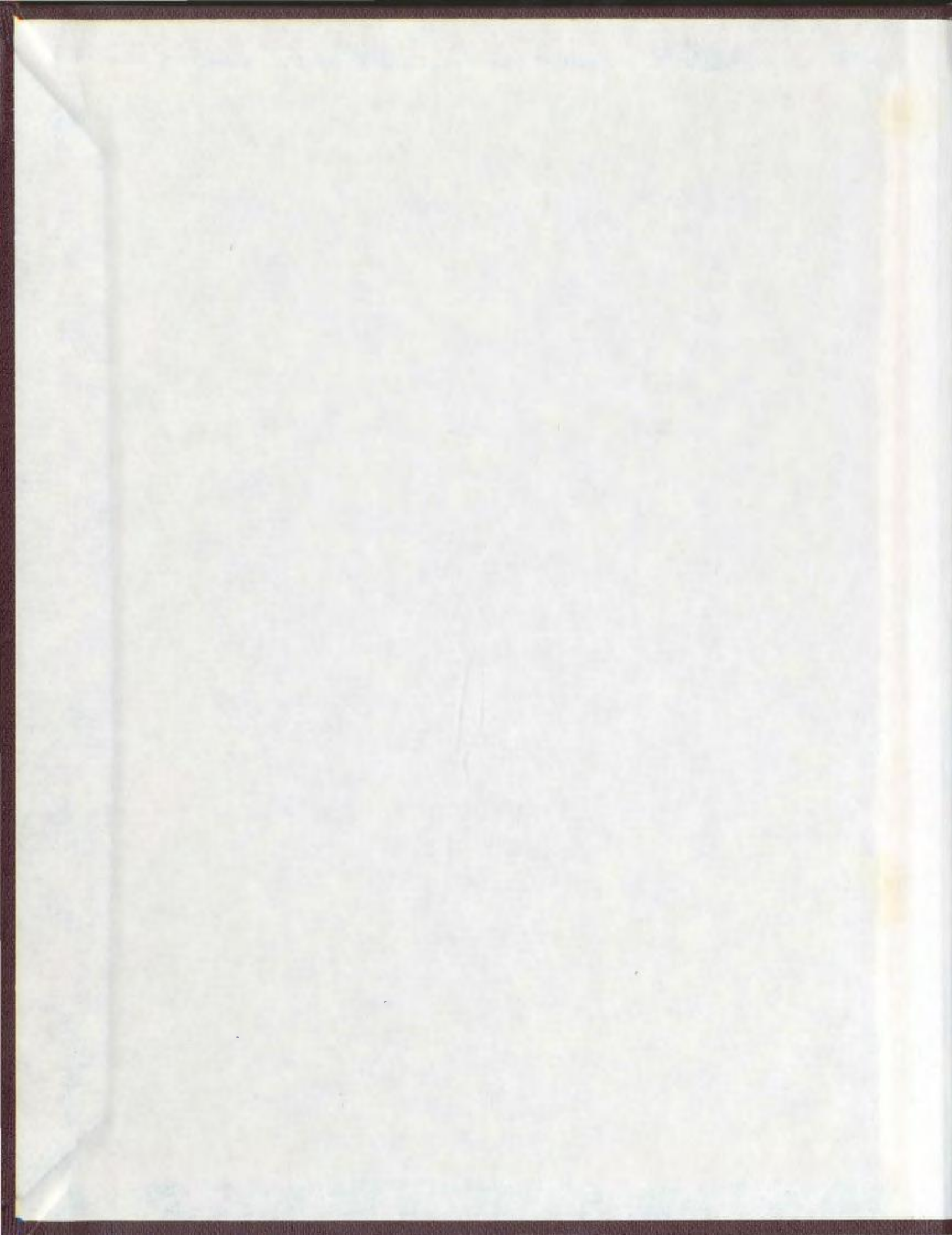
THE PARADOXICAL ENHANCE-  
MENT OF CLASSICALLY  
CONDITIONED PHYSIOLOGICAL  
RESPONSES IN HUMAN  
SUBJECTS

CENTRE FOR NEWFOUNDLAND STUDIES

**TOTAL OF 10 PAGES ONLY  
MAY BE XEROXED**

*(Without Author's Permission)*

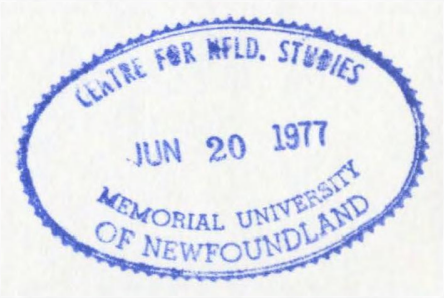
GORDON DAVID RODGERS





copy 1

101171





INFORMATION TO USERS

THIS DISSERTATION HAS BEEN  
MICROFILMED EXACTLY AS RECEIVED

This copy was produced from a microfiche copy of the original document. The quality of the copy is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

PLEASE NOTE: Some pages may have indistinct print. Filmed as received.

Canadian Theses Division  
Cataloguing Branch  
National Library of Canada  
Ottawa, Canada K1A 0N4

AVIS AUX USAGERS

LA THESE A ETE MICROFILMEE  
TELLE QUE NOUS L'AVONS RECUE

Cette copie a été faite à partir d'une microfiche du document original. La qualité de la copie dépend grandement de la qualité de la thèse soumise pour le microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

NOTA BENE: La qualité d'impression de certaines pages peut laisser à désirer. Microfilmée telle que nous l'avons reçue.

Division des thèses canadiennes  
Direction du catalogage  
Bibliothèque nationale du Canada  
Ottawa, Canada K1A 0N4

THE PARADOXICAL ENHANCEMENT OF CLASSICALLY CONDITIONED  
PHYSIOLOGICAL RESPONSES IN HUMAN SUBJECTS

by



Gordon David Rodgers, B.Sc.

A Thesis submitted in partial fulfillment  
of the requirements for the degree of  
Master of Science

Department of Psychology  
Memorial University of Newfoundland

January 1976

St. John's

Newfoundland

## ABSTRACT

This study investigated the phenomenon that, under some conditions, presentation of a Conditioned Stimulus (CS) alone produces an "enhancement" of the Conditioned Response (CR) instead of the well-known extinction effect. Three groups of five male undergraduates each received CS (slide presentations) - UCS (0.5 second burst of white noise) pairings on a 76 per cent irregular reinforcement schedule. Group treatments differed by the number of CS-alone presentations given immediately after conditioning (either 4, 12 or 30). CS-alone and UCS-alone control groups were also used. At two extinction sessions, a week apart, the magnitude (a change of at least 100 ohms between 0.9 and 5.0 seconds after CS onset) and latency of the Galvanic Skin Response (GSR) and change in Finger Pulse (FP) rate were recorded. The primary results showed: (a) a significantly higher GSR magnitude for the conditioning groups as compared to the control groups ( $F(4, 20) = 3.87, p < .025$ ); (b) the group with the greatest number of CS-alone presentations after conditioning was significantly different from the other experimental groups of GSR magnitude ( $F(2, 20) = 7.11, p < .05$ ) and FP rate ( $p < .05$ ; Duncan's Multiple Range Test); (c) this same group did not extinguish after 60 CS-alone presentations while the other experimental groups extinguished after approx-

imately 20 CS-alone presentations; and (d) a significant Trial Block x Group interaction for the latency of the GSR was noted ( $F(20, 100) = 1.68, p < .05$ ). Some implications of these findings and parameters for future research are discussed.



## ACKNOWLEDGEMENTS

To Dr. David S. Hart, for direction, precision, determination to see the project through, but mostly for his patience. To Drs. Mowbray and Preston for their initial encouragement. To Drs. Hedegard, Zagorski, Skanes, and B.T. Revusky, for courteously allowing me to profit from their advice. To D. Morrison for help with the polygraph. To Wayne Gamberg for equipment arrangement. To GERALYN, Dena, Rolf, and Tom, my good friends, for constant help and encouragement.

TABLE OF CONTENTS

	PAGE
Abstract . . . . .	ii
Acknowledgements . . . . .	iv
Table of Contents . . . . .	v
List of Tables . . . . .	vi
List of Figures . . . . .	ix
INTRODUCTION . . . . .	1
Incidental Findings . . . . .	2
Direct Studies of Enhancement . . . . .	4
Clinical Implications of Paradoxical Enhancement . . . . .	9
Purpose of the Present Study . . . . .	11
METHOD . . . . .	13
Subjects . . . . .	13
Apparatus . . . . .	14
Procedure . . . . .	16
RESULTS . . . . .	21
Group Characteristics . . . . .	21
Adaptation Trials . . . . .	23
Evidence for Conditioning . . . . .	28
Evidence for Enhancement . . . . .	30
Extinction . . . . .	33
Latency . . . . .	38

	PAGE
DISCUSSION . . . . .	40
References . . . . .	46

LIST OF TABLES

TABLE NO.	PAGE
1. Analysis of Variance: Age of Students . . . . .	22
2. Duncan's Multiple Range Test of Differences Between Age Scores in the Five Groups . . . . .	22
3. Analysis of Variance: Extraversion- Introversion Scores (EPI, Form A) : . . . . .	24
4. Analysis of Variance: Neuroticism- Stability Scores (EPI, Form A) . . . . .	24
5. Analysis of Variance: Lie Scores (EPI, Form A) : . . . . .	25
6. Group Means on E, N and L-Scales . . . . .	25
7. Summary of Analysis of Variance: Magnitude of GSR (kohms), Group x Trials, Adaptation . . . . .	26
8. Summary of Analysis of Variance: Finger Pulse, Group x Trials, Adaptation . . . . .	26
9. Analysis of Variance: Magnitude of GSR (kohms), Probe Trials, Session 1 . . . . .	29
10. Analysis of Variance: Magnitude of GSR (kohms), First Block of Five Trials (Session 2). . . . .	29

TABLE NO.

PAGE

11.	Analysis of Variance: Finger Pulse, First Block of Five Trials (Session 2) . . . . .	29
12.	Summary of Analysis of Variance: Magnitude of GSR (kohms), Groups x Sessions x Trial Blocks . . . . .	31
13.	Summary of Analysis of Variance: Change in Finger Pulse, Groups x Sessions x Trial Blocks . . . . .	32
14.	Comparison of Group Means of Change in Finger Pulse Rate (Session 2) . . . . .	36
15.	Comparison of Group Means of Change in Finger Pulse Rate (Session 3) . . . . .	36
16.	Summary of Analysis of Variance: Latency of GSR in Seconds, Sessions x Trial Blocks x Groups . . . . .	39

LIST OF FIGURES

FIGURE NO.	PAGE
1. The Experimental Room . . . . .	17
2. Magnitude of GSR (kohms), Averaged Over Groups, for Adaptation Trials (N = 25) . . . . .	27
3. Magnitude of GSR (kohms) for Groups during Trial Blocks of Session 2 . . . . .	34
4. Magnitude of GSR (kohms) for Groups during Trial Blocks of Session 3 . . . . .	35
5. Change in Finger Pulse for Groups during Trial Blocks of Session 2 and Session 3 . . . . .	37



## INTRODUCTION

Under some conditions, presentation of a Conditioned Stimulus (CS) alone produces an enhancement of the Conditioned Response (CR) instead of the well-known extinction effect.

Napalkov (1963) has described the results of some of his experiments with dogs. The CS in these experiments was either a metronome or the flash of a lightbulb. The Unconditioned Stimulus (UCS), which was applied only once, was designed to "provoke the emotions of fear and rage in the experimental dogs" (p. 64). A number of UCS were used, including electric current, flashes of light and raising the dogs to the ceiling. The CS were then presented alone, repeatedly, at intervals of 3 to 5 minutes. Instead of weakening, it was observed that response strength increased with successive applications of the CS. The CR, a rise in blood pressure, increased from the first CS-alone level of 30 to 40 mm to a final level of 190 to 230 mm and persisted even after a period of five months.

The CR in this experiment had been "enhanced." That is, during traditional CS-alone extinction trials the strength of the CR has increased when it would be expected to decrease. This paradoxical effect has not been

thoroughly studied although the literature has several incidental and, more recently, direct findings of increased response strength during extinction trials.

#### Incidental Findings

Lichtenstein (1950), in order to observe the inhibition of feeding responses, shocked dogs while they were eating. He reports on "anxiety symptoms" of the dogs such as tremors, tics and startle responses. He observed that these, and other "symptoms," showed a "tendency . . . to become more marked from day to day, even when no shocks were being applied" (p. 21).

Dykman, Mack and Ackerman (1955), Dykman and Gantt (1958, 1960a, 1960b), and Galbrecht, Dykman and Peters (1960) have also used dogs in their studies and have made observations similar to those of Lichtenstein (1950) and Napalkov (1963). For example, Dykman et al. (1965) conclude that:

- In general, extinction was more upsetting than conditioning, and this finding is contrary to expectation. Apparently, to some dogs the threat is more traumatic than the presence of shock. The median number of "symptoms" during all conditioning phases was 5.0 and the median number during extinction was 13.0 (p. 222).

Solomon, Kamin and Wynne (1953) and Solomon and Wynne (1953, 1954) used dogs in studies of traumatic avoidance learning. These studies show increased responsiveness to the CS during extinction. This has led Solomon et al.

(1953) to conclude:

During ordinary extinction, with very short latencies of the instrumental act we see no emotional reactions, when the animals are later held in the presence of the CS by the glass barrier they demonstrate that the CS has maintained its capacity to elicit anxiety (p. 299).

During this period they also noted that the latency of the response to the CS gradually decreased.

Campbell, Sanderson and Laverty (1964) have observed similar phenomena with humans. In this study the CS was a 600 Hertz tone to which alcoholic subjects had previously been habituated. The UCS was a single period of temporary interruption of respiration by scoline. During this period of interrupted respiration, which lasted an average of 104.6 seconds, the alcoholics remained conscious and the tone CS was turned on. After respiration returned there was a rest period followed by extinction trials. Further extinction trials were given after one and three weeks.

The dependent measures were (a) the Galvanic Skin Response (GSR), (b) Heart Rate (HR), (c) muscle tension, and (d) respiration. These responses, rather than showing a decrease during extinction trials, showed a significant increase over time. The number of GSRs to the CS increased over time while the latency of responses to the CS decreased over time, an observation similar to that reported by Solomon and Wynne (1953, 1954) and Solomon et al. (1953). Campbell et al. (1964) conclude that "a shift in latency

is apparently prognostic of an inextinguishable response" (p. 636). Here, then, is an example of one trial learning with humans where repeated application of the CS-alone leads to enhancement of a CR rather than extinction.

#### Direct Studies of Enhancement

While the above mentioned studies reported paradoxical enhancement of a CR as incidental findings, there are several, more recent, direct studies of the effect.

Rohrbaugh and Riccio (1970), Rohrbaugh, Riccio and Arthur (1972) and Silvestri, Rohrbaugh and Riccio (1970) have investigated the parameters of this phenomenon with rats while Miller and Levis (1971) have observed increased avoidance behavior in humans after brief exposure to a phobic test stimulus.

Rohrbaugh and Riccio (1970) used, as an index of fear, the suppression of approach behavior to food and/or water. Rats, deprived of food and water, were placed in a square, wooden box (CS) where they received ten brief shocks (UCS), which were inescapable. In the one-hour interval after conditioning, rats were returned to the CS apparatus for either 0, 1/2, 5, 15 or 50 minutes during which time no shocks were administered and food and water were not available. During the test situation, food and water were available and latency of intake was measured as an indication of fear (i.e., the greater the fear, the

larger the latency before intake of food or water). The results showed a significant effect of exposure duration in that the 15- and 50-minute exposure groups were less fearful than the 5-minute groups. Other group differences approached significance, but the authors consider enhancement to be "suggested but not clearly demonstrated" (p. 212). A replication of this study using exposure groups of 0, 5 and 50 minutes again showed a significant treatment effect in that the rats in the 50-minute group were less fearful than those in the 0- and 5-minute groups which did not differ.

The second experiment in this series used the conditioned reinstatement procedure of Campbell and Jaynes (1966). Rats were shocked (UCS) in a black compartment (CS) of a test apparatus and not shocked in the white compartment of the apparatus. During a two-week retention period the rats were reexposed to the stimuli three times without being shocked. There were four exposure durations of 0, 30, 60 or 300 seconds. Retention of the fear response was tested in a spatial avoidance situation (i.e., amount of time spent on the safe side of the apparatus is an indication of amount of fear retained). The results of this experiment showed a significant treatment effect. The 30- and 60-second exposure groups spent more time on the safe side of the compartment (more fear) than the 0- and 300-second exposure groups, which showed normal extinction. The authors conclude that these results are evidence of enhancement, stating that

"circumstances exist in which conditioned anxiety is enhanced rather than extinguished by unreinforced exposure to fear stimuli" (p. 214).

Silvestri et al. (1970) conditioned rats to avoid one compartment of a two compartment box, the other compartment being "safe". Experimental subjects received either 5-, 60-, 300-, or 900-second presentations of the CS weekly, for three weeks. Mean spatial avoidance time spent on the safe side was used as an index of fear. A significant treatment effect was observed with the 5- and 60-second exposure conditions producing significantly greater spatial avoidance of fear cues than the 900-second group. In a modified replication of this experiment, exposure durations of 30-, 60- or 300-seconds were used and the 30- and 60-second exposure groups showed greater avoidance than the controls. This effect was curvilinear in that short exposures or long exposures produced less fear than exposures of intermediate duration. Silvestri et al. (1970) conclude that "brief exposure to the conditioned fear cues administered at periodic intervals following training are an effective means of maintaining fear retention" (p. 392).

The most recent work by Rohrbaugh et al. (1972) tried to demonstrate enhancement using the conditioned suppression paradigm of Hoffman and Fleshler (1961). Rats were divided



into groups receiving either 0-, 15-seconds, or 10-minutes of exposure to the CS, which was a tone, after conditioning. Groups were then tested and latency to drink was used as a measure of 'fear retention' (i.e., the longer the latency to drink, the more fearful the rat). Results, as predicted, showed a significant treatment effect with the 15-second group being most fearful. Again, curvilinearity was observed with short or long exposures producing less fear than exposures of intermediate durations. However, when the rats were tested a second time there were no significant group differences. A second measure, the median number of licks at a waterspout paralleled the results of the latency measure. The 15-second group licked the waterspout fewer times than the other groups on the first test trial, but on a second trial there was no significant difference between groups.

Increased avoidance behavior of humans to a phobic test stimulus after certain exposure times to that stimulus has been observed by Miller and Levis (1971). Forty high-school girls who admitted a fear of snakes and who would not touch a live snake in a pretest stage of the experiment, were assigned to visual exposure groups of 0, 15, 30 or 45 minutes. That is, after pretesting a girl would spend either 0, 15, 30 or 45 minutes exposed to a live snake depending on which group she was in. After this, a posttest

was given. The dependent measures were: a distance measure (i.e., how close the girl would approach a snake); a latency measure (i.e., how long it took to approach a snake); and an Adjective Check List which was completed at both pre and posttesting. No significant results were found on measures of latency or on the Adjective Check List. However, the distance measure yielded a significant result in that the girls in the 15-minute group were more fearful than girls in the other groups which were not statistically different from each other. The girls in the 15-minute group displayed more avoidance behavior than the girls in the other groups. There is evidence, then, that certain exposure times to a fearful stimulus may "prevent extinction and results in the conservation of a high level of fear" (Miller and Levis, 1971, p. 20).

These studies by Rohrbaugh and Riccio (1970), Rohrbaugh et al. (1972), and Silvestri et al. (1970) constitute the experimental investigations of the paradoxical enhancement of a CR. The study by Miller and Levis (1971), though not designed as a direct study of enhancement, closely parallels the methodology and results of the other investigators, except that they used humans.

These studies show that brief exposures to the CS alone can increase the amount of conditioned fear behavior shown by a rat or the amount of avoidance behavior shown by

a human. That is, rats or humans, under certain conditions will show more fear behavior than other rats or humans treated similarly except for the duration of exposure to a CS or a feared stimulus. Their results uniformly show that the enhancement effect is curvilinear as a function of duration of CS-alone presentation or duration of exposure to a feared stimulus. With very brief CS-alone or feared stimulus presentations or very long CS-alone or feared stimulus presentations, rats and humans do not exhibit as much fear behavior as groups between these two extremes. These studies show us that amount of exposure to a feared stimulus is a critical parameter in the study of the enhancement phenomenon.

Clinical Implications of Paradoxical Enhancement

Eysenck (1967, 1968) has postulated that the enhancement phenomenon has many implications for clinical research. For example, Eysenck (1967) suggested that the enhancement phenomenon may help to explain the genesis of phobic disorders. He stated:

Occasionally phobic patients are found in which the original, traumatic event is not immediately followed by a strong conditioned fear of the CS, but where this fear seems to grow in time so that exposure to the unreinforced CS does not seem to lead to extinction but rather to an increase in the severity of the conditioned response. Again, not all patients show spontaneous remission; a fair proportion either remain ill or even get worse with time, in spite of the fact that no further reinforcement (pairing of CS and UCS) occurs (p. 63).

Certainly, experimental evidence for enhancement would help to explain these observations.

Stampfl and Levis (1968) developed the "implosion" therapy. The basis of this therapy is that frequent sustained presentation of a strong CS which evokes a strong CR leads to quick extinction of the CR. If, however, an enhancement effect can be shown in humans then the duration of these CS presentations is critical because, contrary to expectation, under some conditions the fear behavior will actually increase.

Desensitization also involves the presentation of a CS, either imaginally or in vivo. Again, the duration of CS presentation is important. If enhancement is a real effect then CS presentations of certain durations would tend to increase rather than decrease fear behavior.

Eysenck (1968) has also pointed out that enhancement may have certain beneficial effects for aversion therapy. Noting that aversion therapy can be regarded as creating an experimental neurosis that is not maladaptive, he points out that this "neurosis" is subject to extinction. Enhancement "would seem to present us with a mechanism which would counteract extinction, and lead to a positive feedback preserving and even strengthening the encapsulated "neurosis" (Eysenck, 1968, p. 316).

Miller and Levis (1971) have implied that enhancement could have effects on the results of certain "analogue

therapy" studies because of the exposure to the feared stimulus during pretesting. Indeed, their study showed that certain durations of exposure to the feared stimulus increased avoidance behavior in humans.

The paradoxical enhancement effect, if it does exist, would seem to have theoretical, practical and methodological implications.

#### Purpose of the Present Study

Although there is a respectable literature pertaining to the phenomenon of enhancement, there are many deficiencies. Probably the most obvious of these deficits is the small amount of work done with humans and, although certain studies are suggestive, no work has been done to actually establish the paradoxical enhancement effect in humans.

Also, most studies have employed avoidance measures but few investigate the relevant parameters in a simpler paradigm as a more direct way to study the acquisition of anxiety. Secondly, studies that did not use avoidance measures but physiological measures such as blood pressure or the GSR had only one conditioning trial which is not the usual case in the classical conditioning paradigm.

The purpose of the present study was to establish the existence of the paradoxical enhancement effect in humans using a traditional classical conditioning paradigm.

A second purpose was to define the characteristics of this effect if it did exist. That is, is the relationship between enhancement and CS exposure curvilinear as reported in earlier studies, and is there a decrease in latency of the response as reported earlier?

The basic strategy was to condition students to respond to a slide presentation (CS) by pairing it with loud white noise (UCS). Immediately after conditioning a variable number of CS-alone presentations were given. At one week and two week intervals further CS-alone presentations were given and magnitude and latency of the GSR (CR) and Pulse Rate (CR) were measured.

The independent variable in the design was the number of CS-alone presentations after conditioning. The dependent variables were the magnitude and latency of the GSR and change in pulse rate during extinction trials during the two weeks following conditioning. It was expected that the occurrence of enhancement depends upon the number of CS-alone trials given after conditioning. Students with few CS-alone trials (short exposure) or many CS-alone trials (long exposure) are expected to extinguish normally, while students with an intermediate number of CS-alone trials are expected to have enhanced CRs.

Specifically, enhancement means the strength of the CR is increased or maintained during extinction trials.



It was hypothesized that: (a) Enhancement of a CR occurs in humans following unreinforced presentations of the CS under certain conditions, and (b) the occurrence of CR enhancement is a curvilinear function of the frequency of unreinforced presentations of the CS; few or many CS presentations results in extinction of the CR, presentations of an intermediate number result in an enhanced CR.

#### METHOD

##### Subjects

The subjects were twenty-five male undergraduates who were paid \$8 for their participation in the experiment. The average age of the subjects was 21.7 years, ranging from 16 to 30 years. The subjects were solicited through notices posted at conspicuous places around the University informing them that participants would be paid.

Subjects were given an information sheet describing the conditions of participation. These conditions were, (a) the subject was not eligible for participation if he had any "medically implanted devices" or if he had "bad or sensitive ears" or was currently seeing a doctor about his ears; (b) the subject agreed to wait until completion of the experiment before being paid; (c) the subject agreed to return at the same time, each week, for three weeks; (d) if the subject missed any of his weekly appointments, he would not be entitled to payment; and (e) if he wished

to participate, the subject had to complete a copy of the Eysenck Personality Inventory (EPI, Form A).

When a subject agreed to these conditions and completed the EPI, a time convenient for participation by the subject was agreed upon and a card with the time and dates of his appointments was given him.

Each experimental session of the first week was assigned a number from one to five corresponding to experimental conditions. Subjects selected a convenient session and thus unwittingly assigned themselves to one of the five experimental conditions. Groups one to three were experimental groups and groups four and five were control groups. In this way a total of five subjects was assigned to each group.

Of the first twenty-five subjects who wished to participate, four did not return for the first session and one did not return after the first session. Replacements were found for these subjects by the same procedure outlined above.

#### Apparatus

A Beckman Type R411 Dynograph was used to record (a) finger pulse, (b) Galvanic Skin Response (GSR), and (c) the occurrence of slide-change signals.

Finger pulse was obtained by means of a Motorola digital transducer and a Beckman Type 9853A coupler. The

5

GSR was obtained through Beckman Biopotential Skin Electrodes (area =  $0.6 \text{ cm}^2$ ; current density =  $16.7 \mu\text{A}/\text{cm}^2$ ) and a Beckman Type 9842 coupler. The occurrences of slide-change signals, which had previously been recorded on magnetic tape, were obtained by connecting a third channel of the dynograph to the output of Channel 1 of the Sony Model TC-252 tape recorder.

The output of Channel 1 of the tape recorder was also connected to a Kodak Carousel Sound Synchronizer which was in turn connected to a Kodak Carousel 800 slide projector. By this arrangement, slide-changes were controlled by signals recorded on the magnetic tape.

In addition, a second output of the tape recorder was connected to a Harmon-Kardon Model AX20 audio amplifier to further amplify the recorded white noise signal and feed it to a set of headphones.

The Conditioned Stimulus (CS) in this experiment was a two second slide presentation of two black circles of equal size, one above the other on a white background. The Unconditioned Stimulus (UCS), which was also recorded on magnetic tape, was a 0.5 second burst of loud white noise of 101 decibels (db) intensity (as measured by a General Radio sound level meter) which was presented to the subject through the headphones. Because the UCS and slide-change signals were recorded on separate tracks of the tape, a subject could not hear slide-change signals. When the CS and UCS were paired there was an interstimulus interval of

1.5 seconds, the CS lasting for 2.0 seconds and the UCS overlapping the CS for the last 0.5 seconds. During inter-trial intervals a neutral pale blue slide was shown and there was a background noise level of 60 db. A second tape, used for CS-alone presentations had a background noise level of 48 db. Programming and timing of the conditioning events was initially done using the Psychology Department Digital computer.

The conditioning session consisted of forty-eight trials, with randomly ordered intertrial intervals (ITI) of 30, 40 or 50 seconds for an average ITI of 40 seconds. The first seven trials were adaptation trials (CS-alone) and the remaining forty-one were acquisition trials (CS + UCS pairing) which were presented on a 76 per cent irregular reinforcement schedule. Unreinforced trials were at positions 20, 23, 27, 30, 33, 34, 37, 40, 43 and 46.

The CS-alone tape had forty-eight trials with inter-trial intervals identical to the conditioning tape. There were no UCS presentations on this tape.

All sessions were run in a darkened, sound-dampened room, 3.34 x 2.13 x 3.05 m (see Figure 1).

#### Procedure

When subjects reported for their first session they were seated in a reclining chair. The subject's right hand was then cleansed with rubbing alcohol and rubbed

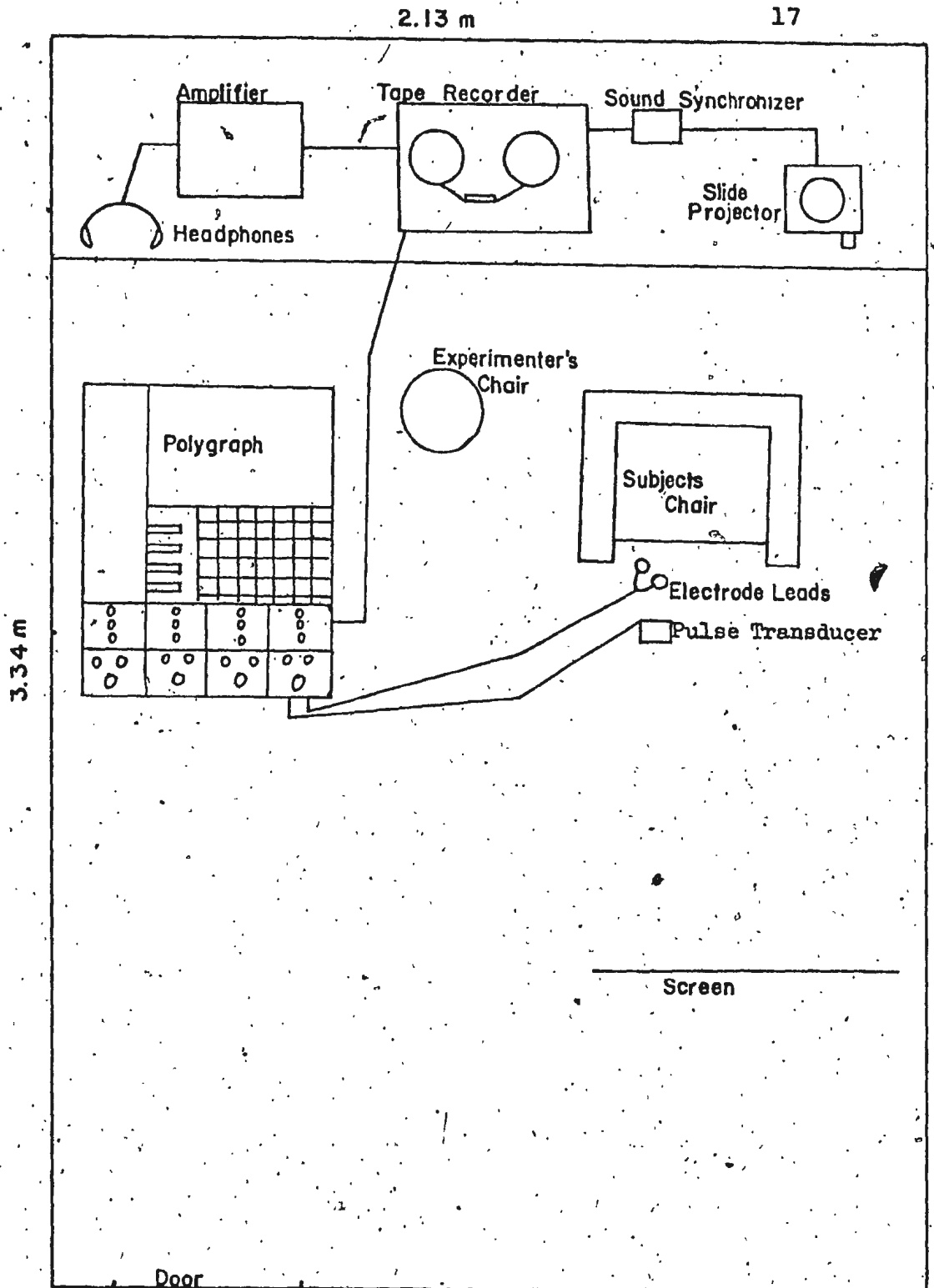


FIGURE 1. The Experimental Room.

briskly with a rough towel. The electrodes were then attached to the palm of the subject's hand by an adhesive cuff. The plethysmograph was then clipped to the subject's right index finger, the lights turned off and instructions read to the subject.

For the three experimental groups (X1, X2, X3) the instructions were:

Today you are going to watch slides and listen to certain sounds through these headphones. I will be observing your reactions to these stimuli through the polygraph. Relax as much as possible. Do not try to aid or inhibit your reactions. I will let you know when the session is over. Any questions?

Any questions, except those pertaining to the actual nature of the experiment, were answered. Then the polygraph, tape recorder and slide projector were turned on, the headphones placed on the subject and the session begun (CHART SPEED = 5 mm/sec and sensitivity adjusted to get the clearest signal from the subject).

All subjects in the three experimental groups received the same sequence of conditioning trials. One hundred and twenty seconds after the last trial on this tape, Group X1 was presented with the CS-alone four times, Group X2 was presented with the CS-alone twelve times, and Group X3 was presented with the CS-alone thirty times. The intertrial intervals were the same as during conditioning. Since each CS-alone slide presentation lasted 2 seconds, Group X1 was exposed to the CS for a total of



8 seconds, Group X2 was exposed to the CS for a total of 24 seconds, and Group X3 was exposed to the CS for a total of 60 seconds during the immediate postconditioning period.

The first control group (C1) was read the following instructions:

Today you are going to watch slides and listen to sounds through these headphones. First, you will watch slides and then I will turn the projector off and you will hear only sounds. Then I will turn the projector on again. I will be observing your reactions to these stimuli through the polygraph. Relax as much as possible. Do not try to aid or inhibit your reactions. I will let you know when the session is over. Any questions?

Questions were answered as before and the same general procedure was followed as with the experimental groups. However, after the seven adaptation trials, the slide projector was turned off so that subjects could hear the UCS but saw no CS (because no CS was administered and there were ten CS-only trials on the tape, no trials were counted during that period when a CS was usually administered. Consequently, the ITI was an average of 60 seconds rather than 40 seconds). Two minutes after the last trial, the slide projector was turned on and, to control for the manipulation of the experimental groups, two subjects received four CS-alone presentations, two subjects received twelve CS-alone presentations, and one subject received thirty CS-alone presentations. This was a UCS-only control group.

The second control group (C2) followed the same general procedure as the other groups except no reference to sound was included in the instructions. They were informed that, "Today you are going to watch slides." For this group, the CS-alone tape was used and after forty-eight presentations, two subjects were given four additional trials, two subjects twelve additional trials, and one subject given thirty additional trials, again, to control for the experimental manipulation. This was a CS-only control group.

After completion of the session, the apparatus was turned off, the lights turned on and the subject was detached from the polygraph. He was thanked and reminded of his appointment the following week.

For the second and third sessions the same procedure was followed for all groups. Each subject was attached to the polygraph as before and told that, "Today you are going to watch slides." The CS-alone tape was used and thirty CS-alone presentations were given to all groups at each session (CHART SPEED = 2.5 mm/sec, sensitivity adjusted to get the clearest signal from the subject). After the final session, subjects were debriefed, any questions answered and payment made.

The first session lasted about 55 minutes and the second and third sessions about 25 minutes each. The experimenter sat quietly behind the subjects during each

session to cope with any unforeseen problems.

The dependent measures in this study were the magnitude and latency of the GSR and change in pulse rate. Magnitude is defined as a change in base level which incorporates zero response. That is, if there is no response, a score of zero is averaged with the magnitude scores on other trials on which there was a response. For a response to be counted, there had to be a change of at least 100 ohms between 0.9 and 5.0 seconds after CS onset. Latency was recorded as the time between CS onset and response onset, if one occurred. The pulse rate change score was the difference between the number of finger pulses recorded in the 10 second interval preceding the CS and the number of finger pulses recorded during the 10 second interval immediately following the CS.

## RESULTS

### Group Characteristics

The mean age for Group X1 was 22 years, for Group X2, 20.8 years, for Group X3, 18.8 years, for Group C1, 23.8 years, and for Group C2, 23.2 years. The group differences were significant ( $F(4, 20) = 3.465, p < .05$ , see Table 1). Group X3 was younger than the control groups (C1 and C2), but none of the other differences were significant (Duncan's Multiple Range Test, see Table 2).

TABLE 1

ANALYSIS OF VARIANCE:  
AGE OF STUDENTS

Source	SS	df	MS	F
Between Groups	79.84	4	19.96	3.465*
Within Groups	115.20	20	5.76	
Total	195.04	24		

\*  $\underline{p} < .05.$ 

TABLE 2

DUNCAN'S MULTIPLE RANGE TEST OF DIFFERENCES  
BETWEEN AGE SCORES IN THE FIVE GROUPS

Group	Means	GROUP				Shortest significant range $\underline{p} < .05^*$
		C2	X1	X2	X3	
		23.2	22.0	20.8	18.8	
C1	23.8	0.6	1.8	2.0	5.0*	$R_2 = 3.172$
C2	23.2		1.2	2.4	4.4*	$R_3 = 3.334$
X1	22.0			1.2	3.2	$R_4 = 3.420$
X2	20.8				2.0	$R_5 = 3.495$

The mean score on the Extraversion scale of the EPI for Group X1 was 11.2, for Group X2, 11.6, for Group X3, 12.2, for Group C1, 12.8, and for Group C2, 13.4. On the Neuroticism scale the mean score for Group X1 was 6.2, Group X2, 14.2, Group X3, 11.0, Group C1, 7.6, and for Group C2, 8.1. On the L-scale mean scores for Group X1 were 3.2, Group X2, 3.0, Group X3, 1.2, Group C1, 1.0, and for Group C2, 2.0. The group differences on these scales were not significant yielding  $F$  ratios (4, 20) = 0.218, 1.786 and 1.888, respectively (see Tables 3, 4, 5 and 6).

#### Adaptation Trials

All subjects in all groups received seven adaptation trials at the beginning of the first session. A Group (5) x Trials (7) analysis of variance yielded no significant Group differences for either magnitude of the GSR,  $F(4, 20) = 1.34, p > .05$ , or change in number of Finger Pulses,  $F(4, 20) = 1.52, p > .05$ . A significant Trials effect,  $F(6, 24) = 2.65, p < .05$ , was found for the magnitude of the GSR (see Tables 7 and 8). The magnitude of the GSR decreased steadily during the adaptation trials (see Figure 2). No Trials effect was found for Finger Pulse ( $F(6, 24) = 1.44, p > .05$ ). These and subsequent analysis of variance were performed on an IBM 360 computer using the Balanova 5 (1968) computer program.

TABLE 3  
ANALYSIS OF VARIANCE:  
EXTRAVERSION-INTROVERSION SCORES (EPI, FORM A)

Source	SS	df	MS	F
Between Groups	15.76	4	3.94	0.218 <u>ns</u>
Within Groups	360.80	20	18.04	
Total	376.56	24		

TABLE 4  
ANALYSIS OF VARIANCE:  
NEUROTICISM-STABILITY SCORES (EPI, FORM A)

Source	SS	df	MS	F
Between Groups	194.16	4	48.54	1.786 <u>ns</u>
Within Groups	543.60	20	27.18	
Total	737.76	24		

TABLE 5  
ANALYSIS OF VARIANCE:  
LIE SCORES (EPI, FORM A)

Source	SS	df	MS	F
Between Groups	20.24	4	5.06	1.888 <u>ns</u>
Within Groups	53.60	20	2.68	
Total	73.84	24		

TABLE 6  
GROUP MEANS ON E, N AND L-SCALES

Scale	GROUP				
	X1	X2	X3	C1	C2
E	11.2	11.6	12.2	12.8	13.4
N	6.2	14.2	11.0	7.6	8.1
L	3.2	3.0	1.2	1.0	2.0

TABLE 7

SUMMARY OF ANALYSIS OF VARIANCE:  
MAGNITUDE OF GSR (kohms), GROUP x TRIALS, ADAPTATION

Source	SS	df	MS	F
Groups (G)	841.403	4	210.351	1.34311 <u>ns</u>
S	3132.29	20	156.615	
Trials (T)	403.805	6	67.3008	2.65545*
G x T	405.06	24	16.877	0.665919 <u>ns</u>
T x S	3041.33	120	25.3444	

\* $p < .05$ .

TABLE 8

SUMMARY OF ANALYSIS OF VARIANCE:  
FINGER PULSE, GROUP x TRIALS, ADAPTATION

Source	SS	df	MS	F
Groups (G)	20.1485	4	5.03713	1.52377 <u>ns</u>
S	66.1142	20	3.30571	
Trials (T)	17.2343	6	2.87238	1.44656 <u>ns</u>
G x T	53.0512	24	2.21047	1.11321 <u>ns</u>
T x S	238.279	120	1.98566	



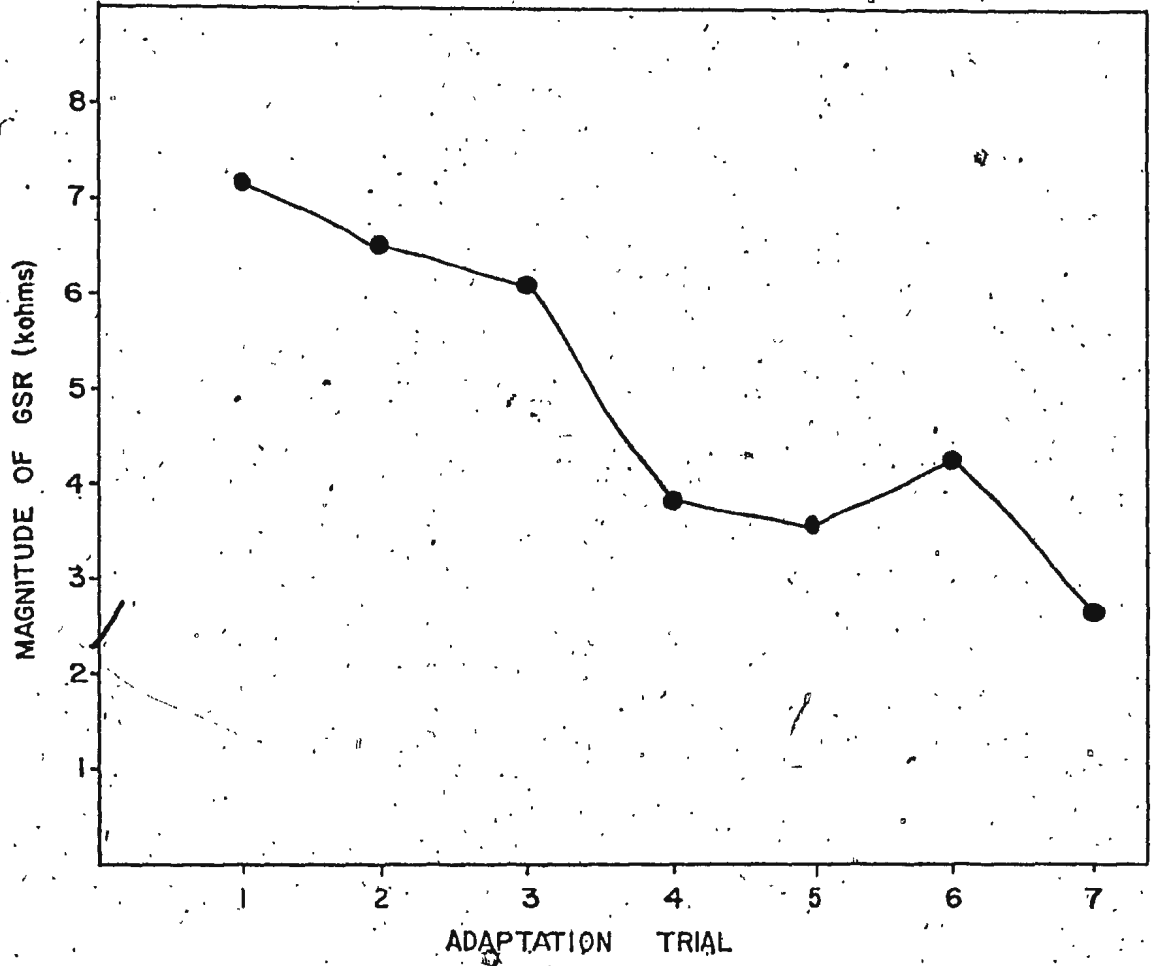


FIGURE 2. Magnitude of GSR (kohms), averaged over groups, for adaptation trials (n=25)

### Evidence for Conditioning

To determine whether conditioning had taken place in the experimental groups, the magnitude of the GSR during unreinforced trials of session one was compared to the magnitude of the GSR on the same trials in the CS-alone (C2) control group. The one-way analysis of variance was not significant ( $F(3, 16) = 2.81, p > .05$ ) (see Table 9). However, using the Scheffe test for a priori comparisons (Ferguson, 1966), a significant difference was indicated between the experimental and control groups ( $F(3, 16) = 3.63, p < .05$ ). The mean magnitude for Group X1, on the probe trials, was 4.268 kohms, for Group X2, 4.402 kohms, for Group X3, 10.432 kohms, and for Group C2, 1.372 kohms.

Further evidence of conditioning was sought by comparing GSR magnitudes and Pulse Rate changes observed in the first block of five extinction trials in session two. The group differences were significant for GSR magnitude ( $F(4, 20) = 3.87, p < .025$ ), but not Pulse Rate change ( $F(4, 20) = 2.80, p > .05$ ) (see Tables 10 and 11). The mean GSR magnitude for the control groups were shown to be different from the experimental groups' mean ( $F(4, 20) = 7.33, p < .01$ , Scheffe test for a priori comparisons). The mean GSR magnitude for Group X1 was 7.02 kohms, for Group X2, 8.98 kohms, for Group X3, 17.4 kohms, for Group C1, 5.88 kohms, and for Group C2, 2.12 kohms.

TABLE 9

ANALYSIS OF VARIANCE:  
MAGNITUDE OF GSR (kohms), PROBE TRIALS, SESSION 1.

Source	SS	df	MS	F
Between	217.54	3	72.51	2.81 ns
Within	412.67	16	25.79	
Total	630.21	19		

TABLE 10

ANALYSIS OF VARIANCE:  
MAGNITUDE OF GSR (kohms), FIRST BLOCK OF  
FIVE TRIALS (SESSION 2)

Source	SS	df	MS	F
Between	648.441	4	162.110	3.87888*
Within	835.862	20	41.7931	
Total	1484.303	24		

\* $p < .025$ .

TABLE 11

ANALYSIS OF VARIANCE:  
FINGER PULSE, FIRST BLOCK OF FIVE TRIALS (SESSION 2)

Source	SS	df	MS	F
Between	47.3600	4	11.8400	2.80569 ns
Within	84.3999	20	4.21999	
Total	131.7599	24		

### Evidence for Enhancement

It was predicted that during the CS-alone presentations of sessions two and three, Group X1, which received four CS-alone presentations after conditioning, and Group X3, which received thirty CS-alone presentations after conditioning, would extinguish normally, whereas Group X2, which received twelve CS-alone presentations after conditioning, would enhance.

Since a significant group difference was found for the magnitude of the GSR during the first five trials of session two and it was established that the experimental groups differed from the control groups, a comparison of the means of the experimental groups was made. Inspection of the means showed that Group X3 was apparently responding at a higher level than Groups X1 or X2. Scheffe's test for a posteriori comparisons showed that this difference was significant ( $F(2, 20) = 7.11, p < .05$ ). The mean for Group X3 was 17.4 kohms, and for Groups X1 and X2 combined, 8.00 kohms.

To further investigate the characteristics of responding during the two extinction sessions comparisons were made for GSR magnitude between groups in each extinction session and for each five-trial block average (i.e. a Groups (5) x Sessions (2) x Trial Blocks (6) analysis of variance; see Table 12). A similar analysis was made for change in Finger Pulse Rate (see Table 13).

TABLE 12

SUMMARY OF ANALYSIS OF VARIANCE:  
MAGNITUDE OF GSR (kohms),  
GROUPS x SESSIONS x TRIAL BLOCKS

Source	SS	df	MS	F
Groups (G)	1677.19	4	419.298	1.93226 <u>ns</u>
S	4339.97	20	216.998	
Sessions (S)	388.967	1	388.967	8.32029**
W x G	285.69	4	71.422	1.52777 <u>ns</u>
S x S	934.98	20	46.749	
Trials (T)	548.222	5	109.644	10.4089**
T x G	358.61	20	17.931	1.70219*
T x S	1053.37	100	10.534	
S x T	29.36	5	5.872	.476709 <u>ns</u>
S x T x G	319.24	20	15.962	1.29587 <u>ns</u>
S x T x S	1231.77	100	12.318	

\* $p < .05$ .

\*\* $p < .01$ .

TABLE 13

SUMMARY OF ANALYSIS OF VARIANCE:  
CHANGE IN FINGER PULSE,  
GROUPS x SESSIONS x TRIAL BLOCKS

Source	SS	df	MS	F
Groups (G)	217.179	4	54.2948	2.97888*
S	364.531	20	18.2265	
Sessions (S)	40.3331	1	40.331	5.68873*
S x G	63.033	4	15.758	2.22262 <u>ns</u>
S x S	141.80	20	7.09	
Trials (T)	8.58664	5	1.71733	0.196570 <u>ns</u>
T x G	108.579	20	5.42895	0.621412 <u>ns</u>
T x S	873.647	100	8.73647	
S x T	31.6267	5	6.32533	1.04657 <u>ns</u>
S x T x G	164.81	20	8.24030	1.36341 <u>ns</u>
S x T x S	604.39	100	6.04389	

\*p < .05.

Significant main effects for Sessions and Trials and a significant Trial Block x Group interaction was found for the magnitude of the GSR. The magnitude of the GSR decreased from the second session to the third session and also decreased across the six Trial Blocks (see Figures 3 and 4). These results would seem to reflect the extinction effect where "the gradual weakening and eventual disappearance of the CR . . . occurs if the CS is repeatedly presented without reinforcement" (Kimble, 1961, p. 324). The significant interaction may be accounted for by the experimental groups' gradual decline in response strength whereas the control groups do not show this decline.

For Finger Pulse, significant main effects were found for Groups and Sessions. Duncan's Multiple Range Test was applied to the group means of session two and session three. It revealed no group differences during session two, but during session three Group X3 was significantly different from all other groups, and Group X2 was significantly different from Group C1 (see Tables 14 and 15). Inspection of Figure 5 indicates that the mean Pulse Rate change for Group X3 was higher during session three than the Pulse Rate change of the other groups.

#### Extinction

The criterion for extinction of the experimental groups was two consecutive trial blocks, in any session,

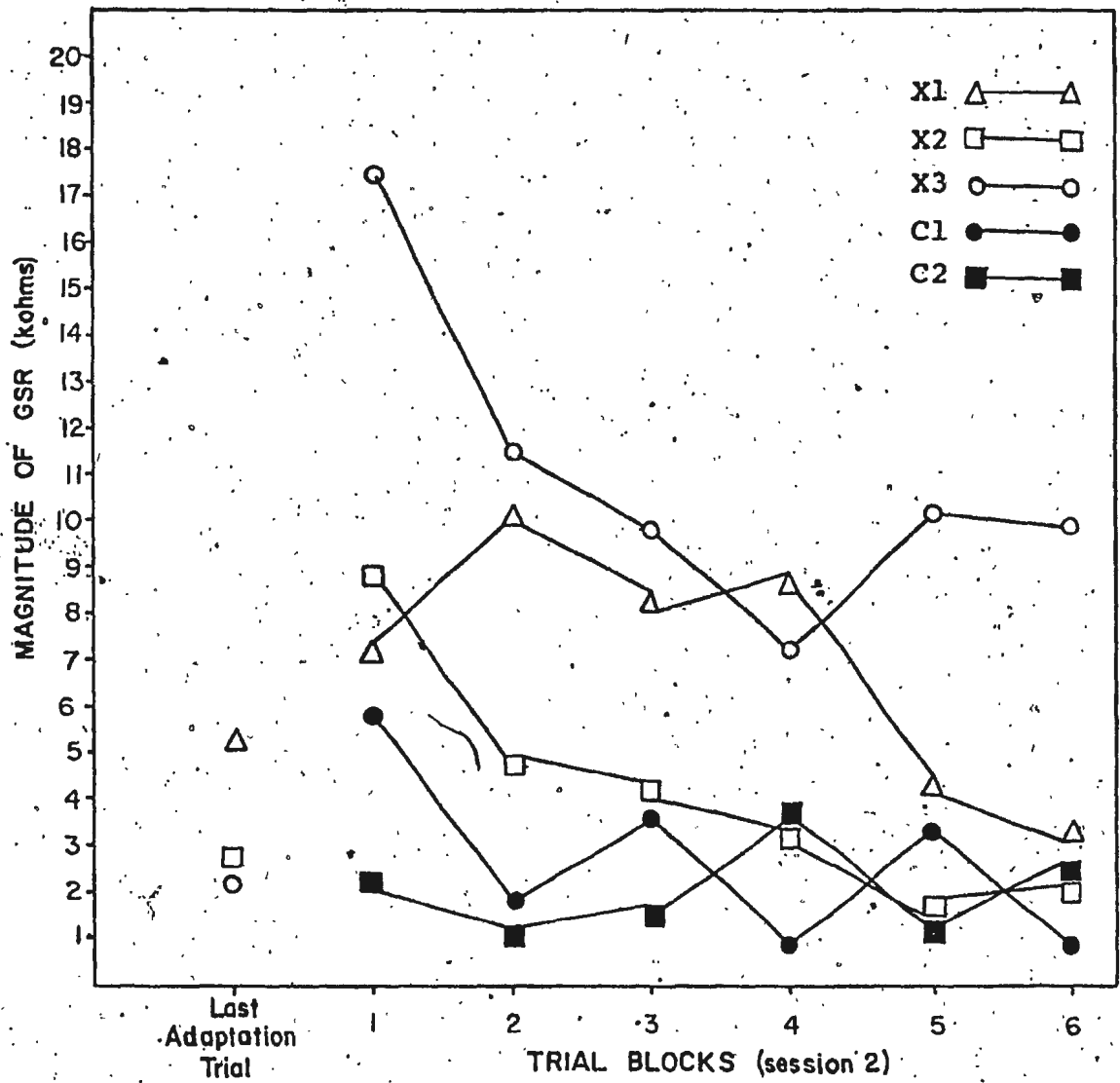


FIGURE 3. Magnitude of GSR (kohms) for groups during trial blocks of session 2.



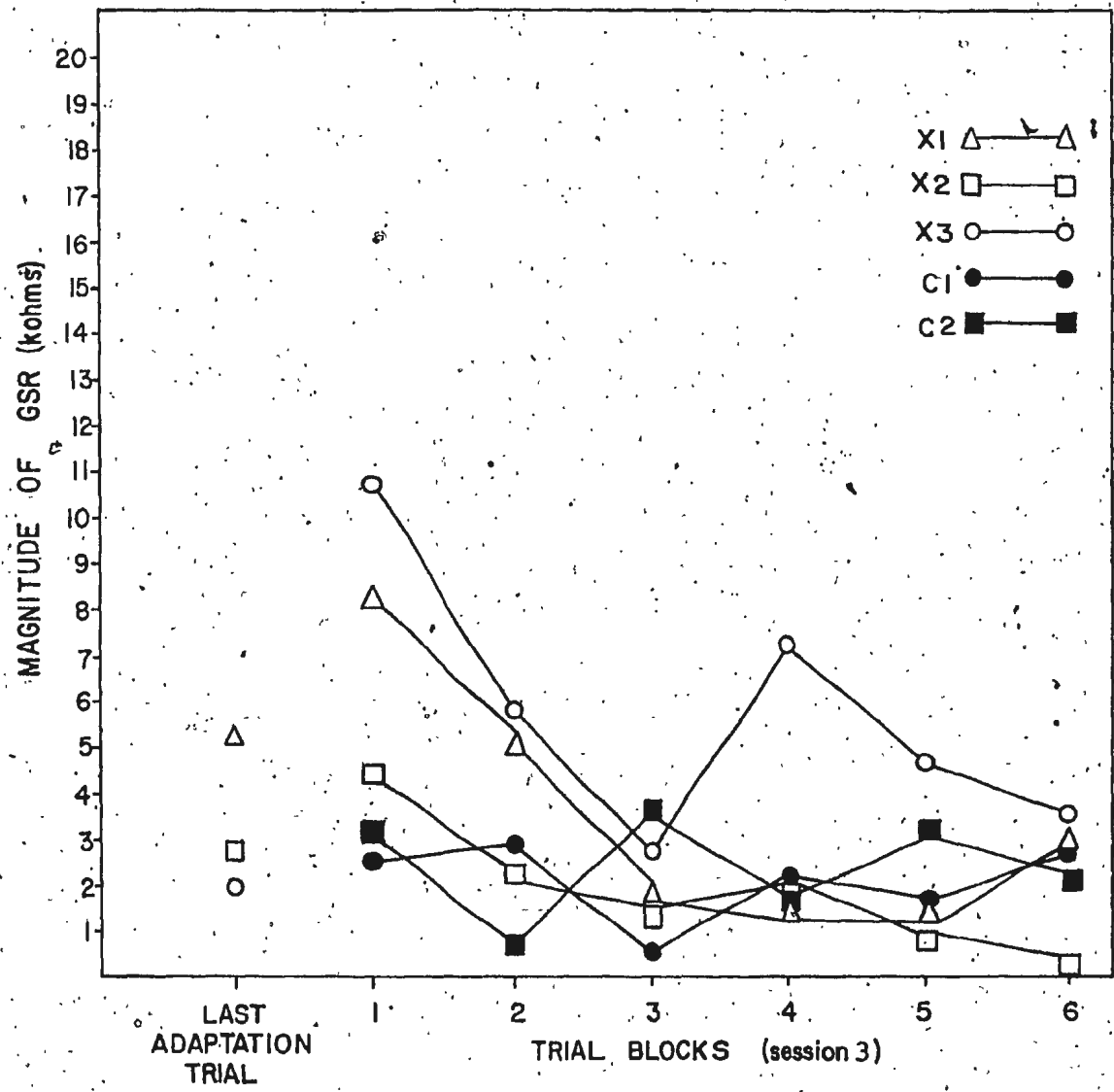


FIGURE 4 Magnitude of GSR (kohms) for groups during trial blocks of session 3.

TABLE 14  
COMPARISON OF GROUP MEANS OF CHANGE IN  
FINGER PULSE RATE (SESSION 2)

Group	Means	GROUP				Shortest range <sup>1</sup>	significant p < .05*
		X2	C2	C1	X1		
X3	0.57	0.84	0.84	1.28	1.51	R <sub>2</sub>	= 2.07
X2	-0.27		0	0.44	0.67	R <sub>3</sub>	= 2.17
C2	-0.27			0.44	0.67	R <sub>4</sub>	= 2.23
C1	-0.71				0.23	R <sub>5</sub>	= 2.28

<sup>1</sup>Duncan's Multiple Range Test.

TABLE 15  
COMPARISON OF GROUP MEANS OF CHANGE IN  
FINGER PULSE RATE (SESSION 3)

Group	Means	GROUP				Shortest range <sup>1</sup>	significant p < .05*
		X2	X1	C2	C1		
X3	1.07	1.88*	2.18*	2.68*	3.91*	R <sub>2</sub>	= 1.79
X2	-0.81		0.30	0.80	2.03*	R <sub>3</sub>	= 1.88
X1	-1.11			0.50	1.73	R <sub>4</sub>	= 1.93
C2	-1.61				1.23	R <sub>5</sub>	= 1.97

<sup>1</sup>Duncan's Multiple Range Test.

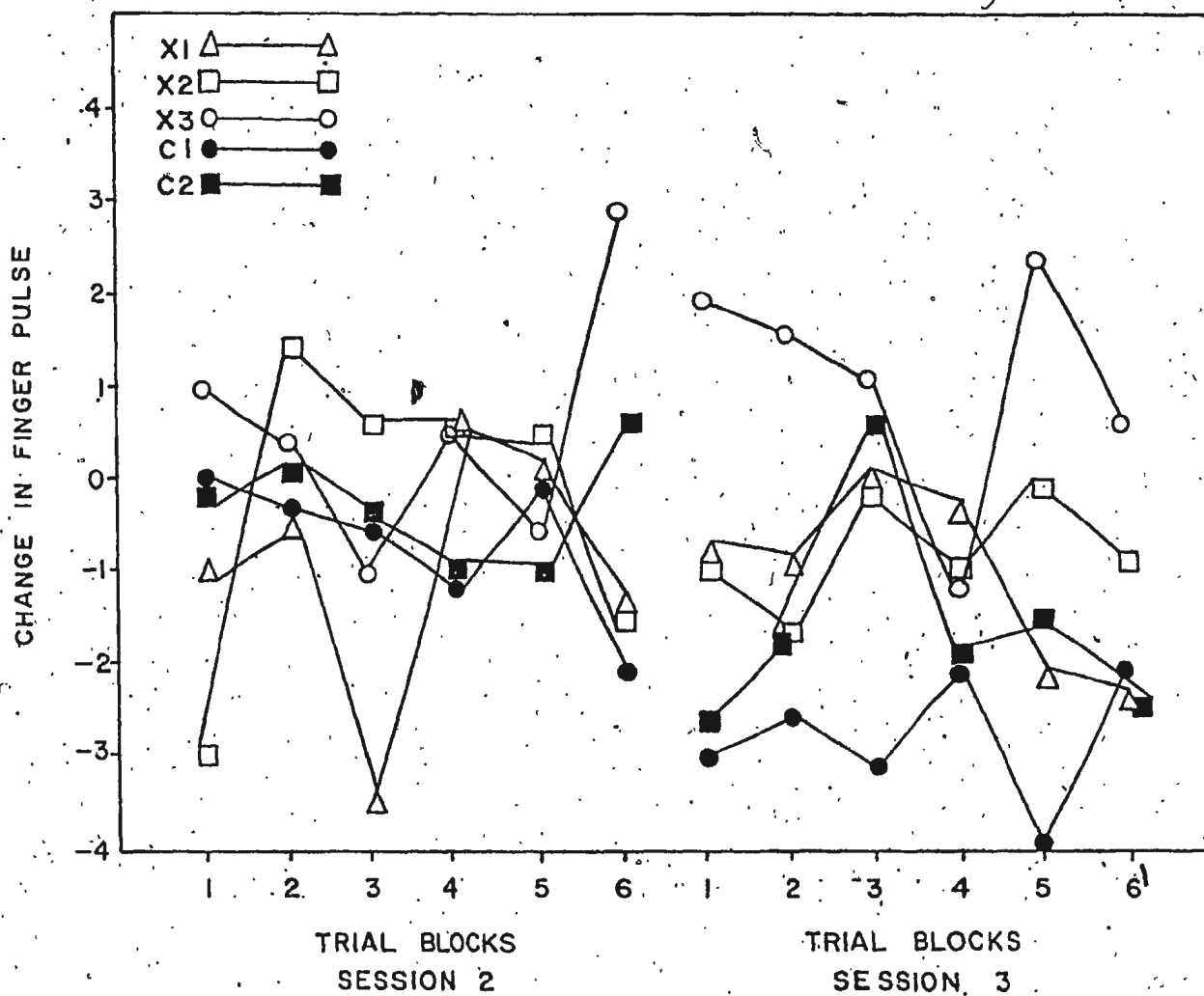


FIGURE 5. Change in finger pulse for groups during trial blocks of sessions 2 and sessions 3.

when the group's mean magnitude of GSR responding was equivalent to, or less than, the group mean on the last of the adaptation trials. These group means, for Group X1, X2 and X3, were, respectively, 5.24 kohms, 2.88 kohms and 2.14 kohms.

Using these criteria, investigation of Figures 3 and 4 shows that Groups X1 and X2 extinguish on Trial Blocks 5 and 6 of session two, that is, after twenty CS-alone presentations. Group X3, however, does not meet this criterion in either session two or session three during sixty CS-alone presentations.

#### Latency

It was expected that latency would decrease over trials if inextinguishable responses occurred. Analysis of this dependent variable using a Session (2) x Trial Block (6) x Group (5) analysis of variance, however, did not yield any significant main effects, but did yield a significant Trial Block x Group interaction ( $F(20, 100) = 1.68669$ ,  $p < .05$ , see Table 16). This significant interaction reflects a general latency increase in the conditioning groups with relatively little latency change in the control groups. As with a similar interaction for the magnitude of the GSR, these results would seem to reflect the extinction effect.

TABLE 16

SUMMARY OF ANALYSIS OF VARIANCE:  
 LATENCY OF GSR IN SECONDS,  
 SESSIONS x TRIAL BLOCKS x GROUPS

Source	SS	df	MS	F
Sessions (S)	3.18888	1	3.18888	1.56090 <u>ns</u>
S x G	12.1714	4	3.04286	1.48943 <u>ns</u>
S x S	40.8593	20	2.04297	
Trials (T)	7.34713	5	1.46943	1.57648 <u>ns</u>
T x G	31.4431	20	1.57216	1.68669*
T x S	93.2096	100	.932096	
Groups (G)	36.2852	4	9.07130	1.42967 <u>ns</u>
S	126.901	20	6.34503	
S x T	2.04556	5	.409113	< 1 <u>ns</u>
S x T x G	17.7694	20	.888472	1.12984 <u>ns</u>
S x T x S	78.6372	100	.786372	

\*p < .05.

## DISCUSSION

It is usually found that extinction trials decrease the strength of a CR to a CS (Kimble, 1961, p. 324). Paradoxical enhancement is said to have occurred when the opposite is found, i.e., extinction trials increase, or maintain, the strength of the CR. The results of this experiment provide partial support for the existence of the enhancement phenomenon.

It was predicted that the group which received the intermediate number of CS-alone presentations after conditioning (Group X2) would show enhancement. Though this prediction was not verified, the group with the greatest number of CS-alone presentations (Group X3), which usually would be expected to show less responsiveness to the CS during extinction trials, did, in fact, respond with greater strength both on measures of GSR and Finger Pulse. In addition, this group's GSR to the CS does not extinguish after sixty CS-alone trials, whereas the other experimental groups extinguish in session two after about twenty trials.

These results, taken together, would suggest that Group X3 is responding differently from the other experimental groups. It is maintaining its responsiveness to the CS (i.e., responses are stronger than at the preconditioning level) when it is the group in which least responsiveness is expected and this, by definition, is the paradoxical enhancement effect.

Though subjects were randomly assigned to experimental and control groups, the enhanced group was significantly younger than the control groups. This is most likely an irrelevant difference, but some consideration is necessary because it is the group that enhanced that differs and it could possibly be construed as a confounding variable accounting for at least part of the observed effects. There are two arguments against this. First, the enhanced group did not differ in age from the other experimental groups and, consequently, comparisons between experimental groups, which were germane to the enhancement hypothesis, were made on groups which did not differ in age. Secondly, and more important, the groups are not different on the personality dimensions of the EPI which have been shown (Franks, 1956) to be related to conditionability.

It was hypothesized that if enhancement occurred there would be a corresponding decrease in the latency of the GSR to the CS. This prediction was not confirmed, the results appearing to reflect an extinction effect. Though no explanation can be offered at this time, it seems possible that this variable may be affected by the strength of the UCS. For example, Campbell et al. (1964) report a shift in latency. However, the UCS (temporary interruption of respiration) in their study would appear to be much more traumatic than a half-second loud burst of white noise, which was the UCS in this research. Future research in

this area may show that with much stronger unconditioned stimuli, latency becomes a relevant, and significant, dependent variable.

A second line of investigation, regarding the curvilinearity of the paradoxical enhancement effect, could be neither supported nor rejected. Previous studies have shown that short or long exposure to the CS led to extinction, whereas intermediate exposure to the CS led to enhancement. In this study, the group with the longest exposure to the CS enhanced. This may be explained by the fact that, with no previous work in the area with humans, frequency of exposure to the CS was decided upon arbitrarily. Future work should concentrate on just what exactly constitutes "long," "short" or "intermediate" exposure for human subjects by including groups with more than thirty CS-alone presentations.

The results of this study suggests that a paradoxical enhancement effect exists in humans. The implications of this effect have been considered in the introduction and only a brief mention will be made here.

It has been suggested by Eysenck (1967, 1968), that the enhancement phenomenon may help to explain the genesis of phobic disorders. That is, post-traumatic exposure to a CS-alone is sometimes sufficient to develop a fear (CR) over a period of time.



Secondly, the two most popular behavior therapy models for use with phobic disorders, implosion therapy and systematic desensitization, both involve imaginal or in vivo presentations of the CS. It would be wise at this point to consider the durations of these exposures as it appears that some durations of exposure could hinder rather than help treatment. Indeed, Rachman (1966) and Rachman and Teasdale (1969) have reported that in some flooding procedures the anxiety to the feared stimulus is actually worsened. Watts (1974) has reported differential response to treatment in systematic desensitization depending on the duration of imaginal presentation of hierarchy scenes.

Thirdly, in those behavior therapies which rely on creating new responses which are not maladaptive (e.g., aversion therapy) knowledge of effective duration of CS exposure could help us to create stronger and longer lasting responses.

More interesting than these possibilities at the moment, however, is the actual experimental investigation of the enhancement phenomenon itself. Though this study suggests the existence of an enhancement effect, many questions are unanswered and many new questions are posed. With regard to the GSR one must consider whether latency, as found incidentally in previous studies, is a suitable indicator of enhanced responding? As previously mentioned, very intense, traumatic UCSs may be required before a shift in

latency occurs during unreinforced trials. Also, one must consider the other parameters of the GSR and to what extent these parameters of response, for example, duration and recruitment, are subject to enhancement. It seems reasonable to hypothesize that an enhanced fear response would last longer and take less time to reach a response peak than one that is less fearful and readily extinguished. The curvilinearity of the effect is another area of concern. First, if the effect exists, is there, in humans, as in animals, a curvilinear relationship between the occurrence of an enhanced CR and the frequency of presentation of the CS-alone? If so, what are the upper and lower limits which determine whether a response will extinguish or enhance? This question, in particular, would seem to be crucial to our understanding and even the demonstration of the effect. It may also be observed that there are no general limits, but that enhancement occurs at various points for various individuals. These are certainly topics worthy of investigation.

At a more experimental level there are some questions which would seem to be worth asking. In this study, duration of CS exposure was indirectly varied by varying the frequency of CS presentations. Would similar, or more conclusive, results be observed if, instead of varying frequencies, groups were given only one CS presentation of

varying durations? Then again, if the phenomenon of enhancement occurs in humans in a maladaptive way, how shall we deal with these responses? And again, are there individual differences related, perhaps, to personality dimensions which make some individuals susceptible to the enhancement effect and others not?

Indeed, it soon becomes apparent that enhancement is a very complex and little understood phenomenon which has many theoretical, practical and methodological implications. This study has suggested the existence of an enhancement effect in humans. However, it is readily seen that this effect poses a great number of important questions. Until more is known, immediate research in this area should concentrate on establishing the important contributing variables to paradoxical enhancement and on defining the characteristics of an enhanced response.

## REFERENCES

- Balanova 5, from York University, Institute for Data Analysis. Write up date 28/10/68.
- Campbell, B.A., & Jaynes, J. Reinstatement. Psychological Review, 1966, 73, 478-480.
- Campbell, D., Sanderson, R.E., & Lavery, S.G. Characteristics of a conditioned response in human subjects during extinction trials following a simple traumatic conditioning trial. Journal of Abnormal and Social Psychology, 1964, 68, 627-639.
- Dykman, R.A., & Gantt, W.H. Cardiovascular conditioning in dogs and humans. In W.H. Gantt (Ed.), Physiological Basis of Psychiatry. Springfield: C.C. Thomas, 1958.
- Dykman, R.A., & Gantt, W.H. Experimental psychogenic hypertensions; Blood pressure changes conditioned to painful stimuli (Schizokinesis). Bulletin of the Johns Hopkins Hospital, 1960, 107, 72-89.(a)
- Dykman, R.A., & Gantt, W.H. A case of experimental neurosis and recovery in relation to the orienting response. Journal of Psychology, 1960, 50, 105-110.(b)
- Dykman, R.A., Mack, R.L., & Ackerman, P.T. The evaluation of autonomic and motor components of the unavoidance conditioned response in the dog. Psychophysiology, 1965, 1, 209-230.
- Eysenck, H.J. Single trial conditioning, neurosis and the Napalkov phenomenon. Behavior Research and Therapy, 1967, 5, 63-65.
- Eysenck, H.J. A theory of the incubation of anxiety/fear responses. Behavior Research and Therapy, 1968, 6, 309-321.
- Ferguson, G.A. Statistical Analysis in Psychology and Education (2nd ed.). New York: McGraw-Hill, 1966.
- Franks, C.M. Conditioning and Personality. Journal of Abnormal and Social Psychology, 1956, 52, 143-150.

- Galbrecht, C.O., Dykman, R.A., & Peters, J.E. The effect of traumatic experiences on the growth and behavior of the rat. Journal of General Psychology, 1960, 50, 227-251.
- Hoffman, H.S., & Fleshler, M. Stimulus factors in aversive control: The generalization of conditioned suppression. Journal of the Experimental Analysis of Behavior, 1961, 4, 371-378.
- Kimble, G.A. Hilgard and Marquis' Conditioning and Learning. New York: Appleton-Century-Crofts, 1961.
- Lichtenstein, C.E. Studies of Anxiety: I. The production of a feeding inhibition in dogs. Journal of Comparative and Physiological Psychology, 1950, 43, 16-29.
- Miller, B.U., & Levis, D.J. The effects of varying short visual exposure times to a phobic test stimulus and subsequent avoidance behavior. Behavior Research and Therapy, 1971, 9, 17-21.
- Napalkov, A.V. Information process of the brain. In N. Wiener & J.C. Schade (Eds.), Progress of Brain Research (Vol. 2). Amsterdam: Elsevier, 1963.
- Rachman, S. Studies in desensitization - II. Flooding. Behavior Research and Therapy, 1966, 4, 1-6.
- Rachman, S., & Teasdale, J.D. Aversion Therapy: An appraisal. In C.M. Franks (Ed.), Behavior Therapy: Appraisal and Status. New York: McGraw-Hill, 1969.
- Rohrbaugh, M., & Riccio, D.C. Paradoxical enhancement of learned fear. Journal of Abnormal Psychology, 1970, 75, 210-216.
- Rohrbaugh, M., Riccio, D.C., & Arthur, A. Paradoxical enhancement of conditioned suppression. Behavior Research and Therapy, 1972, 10, 125-130.
- Silvestri, R., Rohrbaugh, M., & Riccio, D.C. Conditions influencing the retention of learned fear in young rats. Developmental Psychology, 1970, 2, 389-395.
- Solomon, R.L., Kamin, L.J., & Wynne, L.C. Traumatic avoidance learning: The outcome of several extinction procedures with dogs. Journal of Abnormal and Social Psychology, 1953, 48, 291-302.

Solomon, R.L., & Wynne, L.C. Traumatic avoidance learning. Psychological Monographs, 1953, 67, 4.

Solomon, R.L., & Wynne, L.C. Traumatic avoidance learning: The principles of anxiety conservation and partial irreversibility. Psychological Review, 1954, 61, 353-385.

Stampfl, T.G., & Levis, D.J. Implosive Therapy - A Behavioral Therapy? Behavior Research and Therapy, 1968, 6, 31-36.

Watts, F.N. The control of spontaneous recovery of anxiety in imaginal desensitization. Behavior Research and Therapy, 1974, 12, 57-59.







